Prepulse reactivity in prepulse inhibition.

Truchanowicz, Ewa G

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Ewa G. Truchanowicz

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Abstract

Prepulse inhibition (PPI) is a popular paradigm in sensorimotor gating research. In healthy individuals the weak lead stimulus (i.e., the prepulse) presentation results in a reduction in the startle probe (pulse) elicited response. The motor responses to the prepulses (prepulse reactivity, PPER) were until recently largely ignored in PPI research. There are conflicting reports about prepulse reactivity and startle response modification (SRM) associations; and personality factors relevant to SRM have not been previously examined in prepulse reactivity context.

Healthy participants were drawn from university student and staff population. Three paradigms were used: unpredictable stimulus onset, predictable stimulus onset and conscious stimulus processing. The stimuli consisted of 80, 85 & 90dB prepulses and 115dB startle probe separated by 140ms inter-stimulus interval (onset to onset asynchrony). The inter-trial intervals varied between the studies. Startle responses were measured as eye blinks and recorded using surface EMG. All motor responses were quantified according to the same set of rules.

Prepulse-elicited motor responses reliably appeared in all the studies and were distinct from spontaneous EMG. Some PPER characteristics exhibited stimulus intensity dependence further proving PPER validity as stimulus-driven response. Prepulse reactivity exhibited significant associations with startle response modification. PPER was a stable tendency; individuals either consistently responded to the weak lead stimuli or did not.

Two types of startle response modification appeared under the conditions assumed to elicit maximal inhibition only: classical inhibition (as expected) and paradoxical prepulse facilitation. These appeared in motor responses and in conscious stimulus processing. The propensity towards the paradoxical prepulse facilitation was reduced by efficient prepulse inhibition.

PPER and SRM had limited associations with personality factors, sex, or age. The predictable stimulus onset paradigm however highlighted the associations of the defensive startle response and its modification with fear and anxiety. Increased emotionality, regardless of its valence, proved detrimental to sensorimotor gating.

Declarations and statements

DECLARATION

This work has not previously been accepted in substance for any degree and is not being concurrently submitted in candidature for any degree.

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Date 7/12/2010

STATEMENT 1

This thesis is the result of my own investigations, except where otherwise stated. Where correction services have been used, the extent and nature of the correction is clearly marked in a footnote(s).

Other sources are acknowledged by footnotes giving explicit references. A reference section is appended.

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STATEMENT 2

I hereby give consent for my thesis, if accepted, to be available for photocopying and for inter-library loan, and for the title and summary to be made available to outside organisations.

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Tables and Figures

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Definitions and Abbreviations:

Prepulse – lead stimulus, weak stimulus presented before the startle probe

Startle probe – also called 'pulse', an intense burst of sound, the stimulus eliciting startle response

Startle response – motor response elicited by the startle probe

PPER – prepulse reactivity, prepulse-elicited motor response

SRM – startle response modification

PPI – prepulse inhibition

PPF – prepulse facilitation

IRM- stimulus intensity ratings modification

PPIPSI – prepulse inhibition of the perceived stimulus ratings

PPFPSI- prepulse facilitation of the perceived stimulus ratings

PP – prepulse and pulse trials

PA – prepulse alone trials

1 Introduction

1.1 Startle response and prepulse inhibition

Startle response and prepulse inhibition

In an unpredictable environment possibly populated by predators and competing conspecifics it is imperative for an organism's survival to be able to do two things: (1) react swiftly to threats; and (2) efficiently process information relevant to its existence. Sudden onset, intense stimuli elicit a startle response, which is a defensive response designed to elicit an instant reaction to a dramatic change in the environment. However, it is not efficient to process and act upon all sensory information derived from the constant scanning of environment. Therefore a degree of discrimination is required as to what is admitted for further processing leading to response generation.

Sensorimotor gating ability, as indexed by prepulse inhibition (PPI), is one example of such discrimination in the context of startle response. The startle response is inhibited when weak lead stimulus precedes the startle probe (Hoffman & Searle, 1965) and has been demonstrated across a variety of species (D. L. Braff, Geyer, & Swerdlow, 2001; Burgess & Granato, 2007; Frost, Tian, Hoppe, Mongeluzi, & Wang, 2003; Geyer, Krebs-Thomson, Braff, & Swerdlow, 2001) allowing for cross-species comparisons of factors affecting the startle response and its modification. Prepulse inhibition in humans was first systematically researched by Graham (Graham, 1975) and the inhibition of the startle response was attributed to the protection of the ongoing prepulse processing. The lead stimulus presentation is assumed to trigger two processes: (1) prepulse detection; and (2) prepulse processing protection (Graham, 1975, 1980, 1992). Stronger activation of the prepulse processing mechanism leads to stronger startle response inhibition. Prepulse inhibition at short inter-stimulus intervals has been interpreted as measure of protection of processing, sensorimotor gating and early attentional processing (Filion, Dawson, & Schell, 1998). Despite the prominence of prepulse processing in the prepulse inhibition theory, prepulse processing per se had been until recently largely ignored.

Prepulse inhibition deficit as a hallmark of disorder

A number of neuropsychiatric disorders associated with sensorimotor gating deficits/disruptions, and specifically PPI deficits include: bipolar disorder (Perry, Minassian, Feifel, & Braff, 2001) [but increased PPI in BD females has also been reported (Gogos, van den Buuse, & Rossell, 2009)] , schizophrenia and schizotypal personality disorder (D. L. Braff, Geyer, & Swerdlow, 2001; Grillon, Ameli, Charney, Krystal, & Braff, 1992; Kumari & Sharma, 2002; K. Ludewig, Geyer, Etzensberger, & Vollenweider, 2002; K. Ludewig, Geyer, & Vollenweider, 2003), Huntington's disease (N. R. Swerdlow, Paulsen, et al., 1995), Tourette's Syndrome (N. R. Swerdlow, et al., 2001) , ADHD (Castellanos, et al., 1996) [but see (Feifel, Minassian, & Perry, 2009)], OCD (N. R. Swerdlow, Benbow, Zisook, Geyer, & Braff, 1993), Asperger's syndrome (McAlonan, et al., 2002), and Parkinson's Disease (Valls-Sole, Munoz, & Valldeoriola, 2004), although Leng and associates (Leng, Yee, Feldon, & Ferger, 2004) concluded that sensorimotor gating deficits in PD are due to a process distinct from that operating in schizophrenics. PPI is also deficient in psychosis-prone normal individuals (Kumari, Antonova, & Geyer, 2008; Schell, Dawson, Hazlett, & Filion, 1995; Simons & Giardina, 1992) [but Abel and colleagues (K. M. Abel, Jolley, Hemsley, & Geyer, 2004) reported lack of association between schizotypy and PPI]. PPI is low in people with schizotypal personality disorder (Cadenhead, Light, Geyer, McDowell, & Braff, 2002), in unaffected relatives of schizophrenia patients (Cadenhead, Swerdlow, Shafer, Diaz, & Braff, 2000; Kumari, Das, Zachariah, Ettinger, & Sharma, 2005) and in individuals with panic disorder (S. Ludewig, Ludewig, Geyer, Hell, & Vollenweider, 2002).

Schizophrenia related research remains the most common field of application of prepulse inhibition as the tool for investigating sensorimotor gating deficits per se and potential treatments. Sensorimotor gating, as indexed by PPI, contributes to cognitive integrity in healthy humans (Blumenthal, Schicatano, Chapman, Norris, & Ergenzinger, 1996; Graham, 1975). Although Swerdlow and colleagues found that PPI was not correlated with other measures of central inhibition (Stroop task, negative priming) in a sample of healthy participants, all three measures indicated reduced central inhibition in psychosis-prone individuals (N. R. Swerdlow, Filion, Geyer, & Braff, 1995).

Sensory flooding (Venables, 1960) is a model of schizophrenia consistent with deficits in sensorimotor gating demonstrated in schizophrenic patients. PPI is impaired in schizophrenics in comparison to controls across a range of prepulse intensities (Grillon, et al., 1992) and it has been demonstrated that inhibitory deficits in schizophrenics are not a result of stimuli detection failure (D. L. Braff, Grillon, & Geyer, 1992). PPI disruption in schizophrenic patients was found to be consistent with the 'protective hypothesis' (Csomor, et al., 2009), that is the ongoing prepulse processing protection from interruption by the subsequent pulse processing, as posited by Graham (Graham, 1975, 1980, 1992).

1.2 Prepulse processing in prepulse inhibition

Prepulse characteristics

Stronger and longer prepulses lead to higher percentage inhibition with a linear increase in PPI values (Blumenthal, 1995) although prepulse inhibition is determined by prepulse salience (against the background noise) rather than its absolute intensity (Franklin, Moretti, & Blumenthal, 2007). Whilst prepulse characteristics in terms of pure tone versus white noise, duration, bandwidth and intensity have been reported to dramatically affect the subsequent startle response modification (Blumenthal, 1996), a change in the pitch of prepulse stimuli has no effect on the startle response modification (Lipp & Siddle, 1998).

Prepulse characteristics are critical in demonstrating differences between healthy and schizophrenic participants (Blumenthal, Noto, Fox, & Franklin, 2006). Discrete white noise prepulses are most effective in eliciting PPI differences between healthy and schizophrenic participants (D. L. Braff, Geyer, Light, et al., 2001) and prepulse processing difficulty needs to be increased to demonstrate differences between these groups (Blumenthal, et al., 2006). Indeed some of the differential findings related to prepulse processing and its impact on startle response modification in the published studies can be attributed solely to procedural differences (related to prepulse characteristics) (Hsieh, Swerdlow, & Braff, 2006).

Prepulse-elicited motor responses

It has been known for some time that the weak lead stimuli are capable of eliciting motor responses (Blumenthal, 1988; Blumenthal & Goode, 1991). However, such responses were routinely ignored in the published studies of PPI until Dahmen and Corr (Dahmen & Corr, 2004) reported negative correlation between

prepulse-elicited motor responses and PPI in healthy humans, and recommended routine analysis of prepulse-elicited responses in studies using prepulse-inhibition paradigm. Prepulse reactivity has since then become a research question in its own right (N. R. Swerdlow, 2005; Yee & Feldon, 2005).

Prepulse-elicited responses are large enough to be detected and quantified (Csomor, Vollenweider, Feldon, & Yee, 2005) despite reports that such responses are not detectable in humans (N. R. Swerdlow, Sprock, & Braff, 2006). Prepulse reactivity increases in a monotonic fashion with increasing prepulse intensity, but only for individuals with above-median startle (95dB pulse) responses (Csomor, et al., 2006), Csomor and colleagues failed to find an effect of prepulse intensity on prepulse reactivity when all the participants were included.

Yee and colleagues investigated (Yee, Russig, & Feldon, 2004) whether the PPI disruptive effects of apomorphine are, indeed, due to impaired prepulse processing (Davis, et al., 1990) or due to impaired gating mechanism (D. L. Braff, et al., 1992) with no change in prepulse reactivity. They found that apomorphine enhanced prepulse reactivity whilst disrupting PPI. Haloperidol reversed both PPI disruption and enhanced prepulse reactivity induced by apomorphine. The authors (Yee, Russig, et al., 2004) suggested that prepulse reactivity might be a result of increased 'distractibility' with apomorphine treated mice reacting to stimuli that are normally ignored (processed less).

Apart from dopamine agonists, non-competitive N-methyl-, D-aspartate receptor antagonists, such as phencyclidine (PCP) or dizocilpine (MK-801) also disrupt PPI (Curzon & Decker, 1998; Martinez, Halim, Oostwegel, Geyer, & Swerdlow, 2000; Martinez, Oostwegel, Geyer, Ellison, & Swerdlow, 2000; Varty, Walters, Cohen-Williams, & Carey, 2001). Yee and colleagues (Yee, Chang, & Feldon, 2004) investigated PPI and prepulse reactivity in mice treated with dizocilpine and phencyclidine and found that at high doses both drugs severely disrupted PPI and attenuated prepulse-elicited responses. However at lower doses dizocilpine did not affect PPI and enhanced prepulse reactivity. Lower doses of PCP led to disruption of PPI, but did not significantly affect prepulse reactivity. Yee and colleagues (Yee, Chang, et al., 2004) concluded that PPI disruption can be associated with prepulse reactivity when the effects of apomorphine, dizocilpine and phencyclidine are considered together, but at the same time they asserted that prepulse processing (prepulse detection and prepulse reactivity) did not exert an overriding influence on

PPI magnitude (despite positive correlations between these). They concluded that the two processes are largely independent, and even though prepulse processing is crucial in triggering the gating process, prepulse detection or prepulse-elicited motor responses are not. This finding concurs with earlier reports from other authors (Postma, Kumari, Hines, & Gray, 2001; N. R. Swerdlow, J. M. Shoemaker, et al., 2002; N. R. Swerdlow, Talledo, et al., 2004) about the lack of direct relationship between prepulse reactivity and PPI.

Csomor and colleagues (Csomor, et al., 2009) reported that prepulse reactivity (PPER) in unmedicated schizophrenia patients was equal to their motor activity in 'no stimulus' trials. The same group also exhibited deficient PPI (in comparison to healthy controls) at 60ms, but not 120ms inter-stimulus interval. The authors concluded that PPI deficits detected in the schizophrenia patients were due to impaired prepulse perception or processing.

Attentional manipulation of prepulse processing

Directing attention towards the prepulses enhances the subsequent startle inhibition (Dawson, Hazlett, Filion, Nuechterlein, & Schell, 1993) and Filion and colleagues (Filion, Dawson, & Schell, 1993) found that both inhibition (at 120ms) and facilitation (at 2000ms) were enhanced by attended to, as compared to ignored prepulses. The same pattern of results was found in psychosis prone college students (Schell, et al., 1995). In pre-adolescent boys a similar pattern of results appeared, but the enhanced percentage inhibition at 120ms was not replicated across two separate sessions, whereas enhanced facilitation (4500ms) was (Hawk, Pelham, & Yartz, 2002). Extending the paradigm Heekeren and colleagues (Heekeren, Meincke, Geyer, & Gouzoulis-Mayfrank, 2004) directed attention towards both prepulse and pulse which resulted in enhanced PPI only at longer (240ms) intervals and had no effect on the shorter inter-stimulus-intervals (100ms).

Attending to prepulses increases probability of PPI, and probability of prepulse-elicited responses, but does not change other startle response or prepulse-elicited response characteristics (Acocella & Blumenthal, 1990). It can be argued that directing attention towards prepulses simply increases their salience, and the effects of prepulse salience change have been reported (Franklin, et al., 2007). Increased significance of the prepulse stimulus, induced by the need to judge its duration, enhances inhibition with short ISI and increases facilitation with long ISI (Bohmelt, Schell, & Dawson, 1999). The task of tone pitch judging also leads to

increased inhibition at short intervals and facilitation at long intervals (Jennings, Schell, Filion, & Dawson, 1996).

1.3 Individual differences pertinent to startle reflex, startle response modification and prepulse reactivity.

Personality models relevant to sensorimotor gating

Attention and emotion are both linked to personality. Certain personality characteristics make individuals more prone to direct their attention inwards (for example high anxiety) or experience high propensity towards approach or avoidance behaviour in response to different cues. Negative emotionality is generally associated with less likelihood of engagement in the situation, whereas positive emotionality usually indicates a high likelihood of exploration and engagement.

Different personality models are derived from a variety of data sources, some of them originate in animal research and pharmacological manipulations, and others are derived from collating data from self-reports and overt human behaviour. Partially due to their origin and partially due to different conceptual basis, the questionnaires measuring personality dimensions derived from the varied personality models reflect different aspects of the emotional and attentional processing and behaviour.

Sensorimotor gating is affected by both attention and emotion. Dopaminergic, adrenergic and serotonergic activities are implicated in schizophrenia, and personality traits (or states) underpinned by the relevant neurochemical substrates are all relevant. Several personality models (and questionnaires based on these models, see Appendix 2) include concepts pertinent to defensive behaviours, pathological information processing or affectivity present in disorders associated with sensorimotor gating deficits. Associations between personality factors and sensorimotor gating have been reported in published studies.

Personality effects on startle

Startle is enhanced in unpleasant or anxiety provoking situations (Grillon, Ameli, Foot, & Davis, 1993; Grillon, Ameli, Merikangas, Woods, & Davis, 1993), but decreased in social encounter (Britt & Blumenthal, 1993) despite increased state anxiety. Anticipatory anxiety leads to increased startle amplitude and decreased startle latency (Grillon, Ameli, Woods, Merikangas, & Davis, 1991). Highly fearful individuals demonstrate greater startle increase than participants with low fear scores

(Cook, Hawk, Davis, & Stevenson, 1991). Grillon and colleagues equated State Anxiety with fear (Grillon, Ameli, Foot, et al., 1993) and found that fear-potentiated, but not baseline startle, was elevated in high fear (i.e. high scores on State Anxiety subscale of the STAI) individuals. Trait Anxiety subscale of the STAI was not associated with any startle reflex differences between high and low scorers in their study.

Directing attention away from the startle probe decreases startle response amplitude (Silverstein, Graham, & Bohlin, 1981). Schlenker & Leary (Schlenker & Leary, 1982) posited that anxiety is attention directed inward, facilitating self-monitoring and self-presentation. It would therefore be expected that anxious individuals would in fact startle less, as their attention is directed away from the stimuli and towards self-monitoring. Indeed Nitschke and associates (Nitschke, et al., 2002) found that startle response was not enhanced in anxious (anxious apprehensive) participants anticipating exposure to unpleasant stimuli above startle magnitude increase in the rest of the participants in anticipation of an unpleasant image. However, emotion and attention can exert differential effects on the startle response (Patrick & Berthot, 1995) and whilst anxiety may reduce the startle by directing the attention away from the stimulus, task demands in study paradigm may direct the participant's attention towards the startle probe (for example in detection or rating tasks) and the two effects (emotion decreasing startle response and attention increasing it) may cancel each other out. Applying these principles to prepulse processing, directing attention towards the prepulses (for example in stimuli detection or intensity rating tasks) should result in larger motor responses, however the response size change might be nullified by the emotional state, i.e. highly anxious individuals would be directing their attention towards self-monitoring.

Personality effects on PPI

In Cloninger's personality model (Cloninger, Przybeck, & Svrakic, 1991) the trait of Novelty Seeking is underpinned by dopaminergic activity. Dopaminergic activity can also be measured by the resting blink rate and Swerdlow and colleagues (N.R. Swerdlow, et al., 2002) compared the two measures in relation to PPI. In healthy human males the resting blink rate was negatively correlated with PPI, but Novelty Seeking was not. The authors suggested that the lack of links between high NS scores and low PPI may reflect transient 'state' phenomena overriding the 'trait' tendencies; alternatively NS can be underpinned by dopaminergic receptors

irrelevant to PPI. Harm Avoidance (linked to adrenergic activity) and Reward Dependence (serotonergic activity) were also investigated, and neither of these traits was associated with PPI in the cited study (N.R. Swerdlow, et al., 2002). Hutchison and colleagues (Hutchison, Wood, & Swift, 1999) on the other hand found an inverse relationship between Novelty Seeking scale and lower PPI. Individuals high on sensation seeking, a concept similar to novelty seeking, exhibit less PPI (Alessi, Greenwald, & Johanson, 2003) and high motor activity in novel environments is associated with a trend for lower PPI in healthy humans (Alessi, et al., 2003).

Moreover PPI levels are associated with differences in specific polymorphisms affecting COMT (Quednow, et al., 2009; Quednow, Wagner, Mossner, Maier, & Kuhn), 5HT (2A) receptor (Maier, Mossner, Quednow, Wagner, & Hurlemann, 2008; Quednow, et al., 2009) and dopamine receptors D3 (Roussos, Giakoumaki, & Bitsios, 2008). Montag and colleagues (Montag, Hartmann, Merz, Burk, & Reuter, 2008) found no effect of polymorphism in genes coding for the D2 receptor, but the authors did not exclude the possibility of other subcomponents of the dopaminergic system exerting influence on PPI. Hong and colleagues (Hong, Wonodi, Stine, Mitchell, & Thaker, 2008) reported influence of neuregulin-1 (NRG-1) Arg38Gln SNP on PPI in healthy individuals and schizophrenics but Quednow and colleagues (Quednow, et al., 2009) did not replicate their findings. Vasopressin is implicated in prosocial behaviour and social cognitition, dysfunctions of which are notable in a number of disorders, and Levin and colleagues (Levin, et al., 2009) examined how arginine vasopressin 1a (AVPR1a) gene interacts with PPI and found longer alleles to be associated with higher PPI. The implicated genes account for only a small amount of variance in PPI. There have been no studies to date exploring possible genetics of PPER.

Increased DA levels occur in stress (Thierry, Tassin, Blanc, & Glowinski, 1976) or increased vigilance (Jouvet, 1975). It would be expected that highly fearful individuals would be experiencing more stress when exposed to the intense stimuli and highly anxious individuals would respond with increased vigilance, especially in the situation where the onset of the intense, unpleasant stimuli can be predicted. However, physical stress increases PPI (which ought to be disrupted by increased DA levels), but emotional stress reduces it (Pijlman, Herremans, Vend de Kieft, Kruse, & Van Ree, 2003).

Swerdlow and colleagues (N. R. Swerdlow, Bongiovanni, Tochen, & Shoemaker, 2006) reported that PPI is regulated by adrenaline and dopamine, and possibly interaction of both. These findings lend further support to the possible impact of personality characteristics underpinned by dopaminergic or adrenergic neural correlates as potential candidates for individual differences which ought to be considered in the context of sensorimotor gating research.

The scores on the BIS/BAS Scales are not related to percentage PPI in healthy individuals in an affectively neutral situation (Hawk & Kowmas, 2003), but Corr and colleagues (Corr, Tynan, & Kumari, 2002) reported a negative correlation between high scores on the BAS-Drive subscale, a measure of positive incentive motivation, and PPI.

Sex differences

Normal men exhibit higher PPI than normal women (D.L. Braff, Perry, Cadenhead, Swerdlow, & Geyer, 1995; Kumari, Aasen, et al., 2008; Kumari, Aasen, & Sharma, 2004; Rahman, Kumari, & Wilson, 2003; N. R. Swerdlow, Auerbach, et al., 1993; N. R. Swerdlow, Filion, et al., 1995; N. R. Swerdlow, et al., 1999). Other startle characteristics: startle reflex amplitude, latency and habituation are not significantly different between males and females (N. R. Swerdlow, Filion, et al., 1995). Kumari and colleagues (Kumari, et al., 2004) found that schizophrenic women have PPI levels comparable to healthy women, but PPI in schizophrenic males is severely reduced compared to healthy males. However, Braff and colleagues reported PPI deficits in female schizophrenic patients compared to healthy controls (D. L. Braff, Light, Ellwanger, Sprock, & Swerdlow, 2005).

Aasen and colleagues (Aasen, Kolli, & Kumari, 2005) found that the sex differences are not due to a simple reduction in inhibition percentage in females, but rather a shift towards processing the startle stimuli engaging prepulse facilitation (PPF) mechanism. They found that males indeed had higher PPI values, but females had higher PPF. The authors suggested that in females PPF may be a better gauge of sensorimotor gating deficits than PPI. Some studies report no effect of gender (K. Ludewig, Ludewig, et al., 2003) on PPI or PPF.

Sexual orientation in females (but not males) influences percentage PPI values (Rahman, et al., 2003) with homosexual females startle inhibition reaching values not significantly different from those of healthy males.

Menstrual cycle

The regular release of ova from the ovary is controlled by the interplay of physical, neural and endocrine mechanisms. All stages of the cycle, which is roughly four weeks in length from one oocyte release to the next, are associated with a plethora of hormonal releases. The cycle can be divided into two parts, separated by the point of ovulation when the mature follicle bursts releasing the ova. The first phase of the cycle is the follicular phase during which the ovarian follicle reaches its maturity; the second phase is the luteal phase, during which favourable conditions are maintained for the potential implantation of an early zygote. The first stage of the cycle is associated with estrogen dominance, and the second stage with progesterone dominance. If fertilization does not occur, the corpus luteum (the oocyte transformed by the action of anterior pituitary LH) degenerates after 10-14 days and the steroid levels are substantially reduced marking the end of the ovarian cycle. Estrogens have profound impact on mood and behaviour, but the underlying mechanisms of these effects are not well established. (Pocock & Richards, 2006)

Swerdlow and colleagues (N. R. Swerdlow, Hartman, & Auerbach, 1997) reported fluctuation in PPI in females across the menstrual cycle with higher PPI values in the follicular phase. The PPI reduction in luteal phase was most prominent at the point of mid-luteal elevation of estrogen and progesterone. Jovanovic and colleagues (Jovanovic, et al., 2004) reported PPI levels in follicular phase females equivalent to PPI levels in males. Kumari and colleagues (Kumari, et al., 2010) found that estradiol and progesterone elevation in the luteal phase of the menstrual cycle are linked to the lower PPI levels, but higher progesterone levels equal smaller PPI decrease in the luteal phase women. PPI is also lower in pregnant women (Kask, Backstrom, Gulinello, & Sundstrom-Poromaa, 2008) in the late stages of pregnancy when progesterone and estradiol levels are elevated, but returns to normal in post-partum.

Kumari and colleagues (Kumari, Aasen, et al., 2008) reported differences in the PPI levels in pre- and post-menopausal women in comparison to males. Whilst pre-menopausal women exhibit lower PPI and no differences in PPF in comparison to age-matched males, post-menopausal women have PPI levels similar to age-matched males, but increased levels of PPF.

Whilst investigating the effects of menstrual cycle on PPI Swerdlow and colleagues (N. R. Swerdlow, et al., 1997) found that in visuospatial priming

inhibition versus facilitation was linked to the menstrual cycle status. The follicular phase was associated with inhibition and luteal phase with facilitation. It is possible that the 'shift towards facilitation' reported by Aasen and colleagues (Aasen, et al., 2005) can be partially explained by the effects of hormones in the female participants. Indeed Kumari and colleagues (Kumari, et al., 2010) found increased PPI in the follicular phase and increased PPF in the luteal phase in healthy women.

The lack of menstrual cycle effects reported in some studies, or indeed the differential findings related to the menstrual cycle status of the female participants, can be attributed to either inaccurate menstrual cycle recording or the effects of age in the females. The hormonal shifts associated with female aging exert an effect on PPI and PPF levels in females, and can alter the findings in terms of the menstrual cycle effects or even the sex effects (Kumari, Aasen, et al., 2008; Kumari, et al., 2004).

Age

Age has no effect on PPI (Harbin & Berg, 1983, 1986; K. Ludewig, Ludewig, et al., 2003; N. R. Swerdlow, Filion, et al., 1995) in normal individuals and Ellwanger and colleagues (Ellwanger, Geyer, & Braff, 2003) found that PPI exhibits an inverted U-shape distribution with highest PPI at intermediate ages, but with no significant differences across the age groups. Their study refuted the assumption of general inhibitory decline associated with age. PPF is not affected by age either (K. Ludewig, Ludewig, et al., 2003) further demonstrating that age is not a critical factor affecting startle response modulation.

Baseline PPI as an individual difference

There is some evidence for PPI as a stable neurobiological marker (K. Abel, Waikar, Pedro, Hemsley, & Geyer, 1998; Cadenhead, Carasso, Swerdlow, Geyer, & Braff, 1999) and heritability of sensorimotor gating ability (N. R. Swerdlow, Krupin, et al., 2006; N. R. Swerdlow, Shoemaker, Auerbach, et al., 2004; N. R. Swerdlow, Shoemaker, Platten, et al., 2004). In humans over 50% of variance in PPI can be attributed to genetic factors (Anokhin, Heath, Myers, Ralano, & Wood, 2003). Several studies demonstrated heritability for PPI. The percentages differed subject to the onset to onset asynchrony values and ranged from 32 percent for PPI at 60ms inter-stimulus intervale (Greenwood, et al., 2007) to 58 percent for PPI at 120ms ISI (Anokhin, et al., 2003) A recent study reported 45 percent heritability for PPI at 60ms ISI, 33 percent for PPI at 120ms ISI and surprisingly no differences in PPI

between healthy controls, schizophrenia sufferers and their relatives (Hasenkamp, et al.). Baseline PPI is an individual difference useful in predicting individual tendencies in responding to treatment with substances affecting PPI (Feifel, 1999; Hutchison, Rohsenow, Monti, Palfai, & Swift, 1997). This suggestion was borne out in the study using clozapine (Vollenweider, Barro, Csomor, & Feldon, 2006) to enhance PPI in healthy individuals, where clozapine increased PPI levels only in participants with low baseline PPI levels. Swerdlow and colleagues (N. R. Swerdlow, Talledo, Sutherland, Nagy, & Shoemaker, 2006) suggested an existence of a low gating, antipsychotic-sensitive phenotype. Bitsios and colleagues (Bitsios, Giakoumaki, & Frangou, 2005) also found an effect of baseline PPI values on the outcomes of DA agonist treatment in healthy males, with DA agonists (pergolide and amantadine) disrupted PPI only in high-gating (high PPI) individuals. Swerdlow and colleagues (N. R. Swerdlow, A. Eastvold, et al., 2002) found no effect of direct and indirect dopamine agonists (bromocriptine, pergolide, amphetamine and amantadine) on PPI in healthy males, and this lack of effect could be due to the baseline PPI characteristics of the sample.

1.4 Research rationale

Although it has now been established that prepulse elicited motor responses (PPER) can be quantified in humans, their significance remains unclear, with conflicting results reported in animal and human research. The associations between PPER and individual differences have not been previously investigated. The conscious processing of prepulses beyond simple detection has not been previously probed and alternative forms of startle response modification, related to paradigm characteristics, have not been considered in the context of prepulse inhibition.

The main fulcrum of the thesis is prepulse reactivity per se: its prevalence in normal population and response characteristics. It has been demonstrated in published studies that PPER can be quantified despite the small response sizes. The studies contributing to this thesis are designed to further strengthen the argument that PPER is significantly different from spontaneous EMG and can be reliably measured in healthy human participants. Moreover the associations between PPER and SRM, especially PPI, are of special interest, since they formed the focus of controversy centered on PPER significance, or indeed existence, in sensorimotor gating research.

Furthermore some studies indicated associations between PPER and startle response modification, but the findings in animal and human research are mixed, with no clear cross-species translatable findings reported. The associations between PPER and SRM are examined in each and every study in this thesis to provide clarification in the area of PPER associations with SRM in healthy humans.

The conscious processing of the prepulse stimuli may exert some influence on the subsequent startle response modification if the processing extends beyond simple prepulse detection. Attention has been shown to increase prepulse inhibition in directed paradigms (in which attention is directed towards the prepulse), but the consequences of demands of prepulse intensity rating task have not been previously considered.

Several personality models contain personality factors constructs which are based on neural substrates. Some of these neural substrates are relevant to sensorimotor gating and specifically prepulse inhibition. For example dopamine is a well established culprit in the disruption of sensorimotor gating based on the evidence derived from pharmacological studies (see above) and serotonin is another neurochemical implicated in a number of neuropsychiatric disorders. The neurochemical dysfunctions and specific genetic mutations are present across a breadth of disorders indicating a common underlying disruption to normal functioning (Craddock, O'Donovan, & Owen, 2006). A number of personality models are based on biological substrates and the behavioural manifestations of the personality traits delineated within these models are presumed to reflect the underlying neurochemical activity. An example of such personality model is Cloninger's Trait and Character Inventory (described in detail in Chapter 2) and the trait of Novelty Seeking which is presumed to reflect dopaminergic activity. Individual differences have not been investigated in terms of their associations with prepulse reactivity, though some of the models used in the thesis have been considered in the context of prepulse inhibition, most commonly personality factors underpinned by putative dopaminergic activity have traditionally been popular choices for personality and sensorimotor gating research combination.

The associations between polymorphisms of serotonin receptors and PPI (see above) stipulate that personality traits putatively underpinned by serotonergic activity also ought to be considered in the context of startle response modification, and bearing in mind the differences in prepulse reactivity between schizophrenics and

healthy individuals, and the implications of both dopaminergic and serotonergic receptor dysfunctions in schizophrenia, these personality characteristics may exert some influence on PPER as well.

The assumption underpinning the implication of personality characteristics in sensorimotor gating is heritability of personality and the biological basis of the behavioural manifestations of the underlying neurochemical systems. The reason for the inclusion of personality models encompassing putative reflections of the dopaminergic and serotonergic systems is based on the genetic associations between these systems and PPI (or sensorimotor gating defined more widely), the implications of these systems in several neuropsychiatric disorders with sensorimotor gating deficits, and the definition of deficient sensorimotor gating ability per se as an endophenotype of schizophrenia.

The following assertions will be tested in the presented thesis:

1. PPER is significantly different from spontaneous EMG
2. Individual differences relevant to SRM are related to PPER
3. PPER has associations with startle response modification
4. SRM needs to be derived from each trial, not trial type means
5. SRM is related to individual differences
6. Conscious stimuli processing impacts on PPER and SRM
7. PPER and SRM are affected by stimulus onset predictability

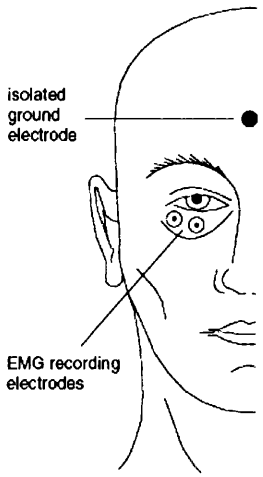
2 Eliciting, recording and quantifying the startle response in human participants

2.1 Introduction

In rodents and non-human primates the startle response is measured by whole body startle. Such an approach would be impractical in humans and a range of eyeblink measuring methods are available for human research (Blumenthal, et al., 2005). These techniques include potentiometric, photoelectric, vertical electrooculographic (vEOG) and magnetic search coil methods, all measuring eyelid movement. An alternative approach to measuring the eyeblink response is to trace the activity (action potentials) within the orbicularis oculi muscle generated during the startle response with either surface or needle EMG recording electrodes. Surface EMG recording offers the easiest solution in terms of the speed of electrode application, reduced electrode intrusiveness and participant discomfort. The majority of the publications in the area of human acoustic startle response use the surface EMG recording approach.

When muscle fibres contract a small voltage electric activity can be detected at the skin surface and the changes in this voltage constitute the EMG activity. The action potentials generated as the result of the startle response manifested in the orbicularis oculi activity are recorded by placing differentially amplified electrodes on the orbital part of the muscle and the ground electrode in an electrically independent location (e.g. mastoid or forehead; see Figure 2.1).

Figure 2.1 Electrode positioning for the surface eyeblink EMG recording (adapted from Blumenthal, Cuthbert et al 2005)



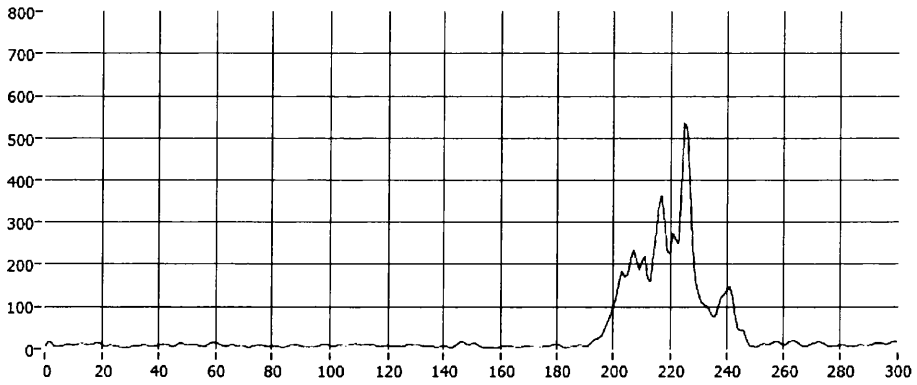
The quality of the EMG recording is compromised by high electrode impedances. Proper skin preparation is vital to obtain a good quality recording; however, with the modern Ag/AgCl electrodes, using abrasive gel is not necessary. The miniature electrodes (5mm in diameter) are enclosed in plastic casings and the conductivity between the skin surface and the electrode surface is provided by electrode gel. As can be seen in Fig. 2.1, the electrodes are placed below the eye which makes them even less intrusive to the participant. Laterality has no effect on the obtained recordings (Blumenthal, Cuthbert et al 2005), although some reports indicated a laterality effect in healthy, but not schizophrenic or schizotypal participants or their relatives (Cadenhead, et al., 2000). Recently Kumari and colleagues (Kumari, Fannon, Sumich, & Sharma, 2007) reported higher PPI levels in both healthy and schizophrenic individuals following a monaural prestimuli (prepulse) presentation, however the group differences in PPI levels (healthy participants had higher PPI levels) remained stable regardless of the prepulse presentation method.

The EMG signal is prone to contamination by, for example, proximal electrical sources, other physiological signals (e.g. ECG) or indeed movement artefacts. Loosely platting the electrode wires ensures that any electrical noise is picked up equally and will be removed effectively from the recorded signal by the common mode rejection application. The differential amplification technique relies on the subtraction of the signals recorded from the two different electrodes. This way any shared components are removed and the 'differential' between the electrode recordings is amplified. It is presumed that signal contamination from electrical noise or motion artefacts will be common to both electrodes, whereas the activity at the proximal locations will be electrodes specific and therefore easily differentiated from the shared components. The 'Holy Grail' of recording physiological phenomena is finding a method offering minimal signal distortion and high signal-to-noise ratio.

The recorded signals are filtered using analogue and digital filters. The low pass and high pass filtering ensures that noise contamination is reduced and the fidelity of the signal retained. Low pass filtering value means that all frequencies above it will be removed. For the eyeblink EMG low pass filter of 400-500Hz (assuming 1000Hz sampling rate) is recommended to include the EMG frequencies of interest. High pass filters reported in the literature range from 30Hz to 100 Hz.

Limiting the frequencies admitted for amplification has the advantage of reducing the possibility of including frequencies which are likely to result form environmental signal contamination (e.g. 50Hz frequency from electrical sources). One possible problem in recording any physiological signal is aliasing, which is recording a misrepresented waveform. Aliasing occurs when the sampling rate is too low and the peaks that ought to be recorded between the sampling points are therefore omitted from data capture and processing. Aliasing leads to a distorted waveform recording showing a much lower frequency of peaks (and consequently distorted maximum peak amplitude, latency and overall frequency). The final EMG waveform is subjected to rectification (conversion of the recorded values to absolute values, i.e. all values are positive, rather than positive and negative as they would be in the raw EMG waveform), smoothing (the peaks become smaller and shifted to the right, i.e. the latency would be distorted) and filtering (the waveform becomes more 'square' and acquires a steeper roll off compensating for the smoothing process by shifting the peaks to the left, i.e. changing the latency to a point closer to the original location compensating for the phase shift induced by the smoothing process). Figure 2.2 shows a typical EMG startle response waveform (rectified, smoothed and filtered). The components of interest are the size (peak response amplitude measured in micro volts) and the temporal characteristics (response onset and peak response latency, both measured in milliseconds) of the response.

Figure 2.2 EMG waveform recorded in pulse-alone trial showing pulse-elicited response (pulse stimulus presented at 160ms)



Detection of the EMG signal is only possible if it is larger or in a different frequency range than the background noise. The differential amplification reduces or removes contaminated elements. Some degree of signal contamination is likely to persist and hence a baseline comparison value is obtained for each admitted trial. This baseline value serves as a benchmark against which the responses are compared. The valid responses are derived from the EMG signal based on the specific criteria unique to the experimental paradigm. In the SDI SR-HLAB software these criteria comprise: baseline (set to a number of ms at the beginning of the recording window), response window (i.e., the length of which determines how far back from the peak response amplitude the beginning of the response is sought), rolling average (smoothes the EMG wave using the average of a specified number of neighbouring digital points), digital filter (compensates for using the rolling average by making the waves more square and thus correcting the temporal peak distribution), and response criterion (i.e., the number of standard deviations above the baseline, a *z* score). The scoring parameters are adjusted to reflect the trial events sequence in each study. The EMG signal is rectified (conversion into absolute values), amplified (the recorded values are increased), and filtered using analogue filter (low and high pass cut off values set prior to the recording) online. Subsequently the recorded EMG waveform is digitally smoothed and filtered. The statistical analysis is performed on the final outcome of the smoothing/filtering process.

2.2 Materials

In all the studies presented in this thesis, both questionnaire and physiological data were collected. Personality characteristics were measured using the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE), the BIS/BAS Scales (BIS/BAS), the State and Trait Anxiety Inventory (STAI), the Positive and Negative Affect Scale (PANAS), the Fear Survey Schedule (FSS) and the Temperament and Character Inventory (TCI). Demographic questionnaire was used to record age, sex, medication (including over the counter) and the time of last alcohol and nicotine intake. The menstrual cycle in females will not be reported, even though the self-reported first day of the menstrual cycle was recorded on the demographic questionnaire in all studies except for the first one, no reliable measures of ascertaining the true menstrual cycle status in were used. The questionnaires, their underlying theoretical models and the reasons for their inclusion are described in

detail below. The choice of the personality questionnaires was based on their relevance to sensorimotor gating and inclusion in previously published studies.

TCI

Cloninger's Temperament and Character Inventory (Cloninger, 1994; Cloninger, Svrakic, & Przybeck, 1993) contains seven dimensions of personality: four temperaments (Novelty Seeking, Harm Avoidance, Reward Dependence and Persistence) and three characters (Self-Directed Behavior, Cooperativeness and Self-Transcendence). Originating from the Tri-Dimensional Personality Questionnaire (TPQ) (Cloninger, 1986; Cloninger, et al., 1991), TCI benefitted from the addition of three 'character' factors and separating Persistence subscale form the Reward Dependence. The TCI contains the scales of Novelty Seeking (reflecting dopaminergic activity/ behavioural approach), Reward Dependence (serotonergic activity/ behavioural maintenance), Harm Avoidance (adrenergic activity/ behavioural avoidance) and Persistence (separated from Reward Dependence, presumed to also relate to serotonergic activity) and three character scales: Self-Directed Behavior, Cooperativeness and Self-Transcendence. A study utilizing genetic analysis confirmed the validity of the assumed neural substrates of both TCI temperaments and characters (Comings, et al., 2000).

The temperaments are most pertinent to sensorimotor gating, since malfunctions of the neural systems using the three neurotransmitters (dopamine, serotonin and adrenaline) have been implicated in schizophrenia (and other disorders associated with inhibitory deficits) and have been targets for pharmacological interventions. Moreover, the temperaments are posited to be purely biological in their nature, whereas the characters are more malleable to environmental influences and reflect re-coding of sensory information into abstract concepts contributing to the individual identity. Whereas the temperaments are automatic emotional reactions and habits, the characters can change the emotional experience. Attention and affectivity can impact on sensorimotor gating, and characteristics on levels other than biology can be beneficial explanations of differences in sensorimotor gating ability.

High and low scores on the TCI subscales translate into different, but not always opposing behavioural outcomes. Individuals with high Novelty Seeking scores are exploratory and curious, impulsive, disorderly, extravagant and enthusiastic, characteristics closely related to approach behaviour. Low scores on this scale denote an indifferent, reflective, frugal and detached, orderly and

regimented individual. High Reward Dependence scores indicate a sentimental and warm, dependent, dedicated and attached person. Low scores on Reward Dependence indicate someone who is practical and cold, withdrawn, detached and independent. Reward Dependence is associated with behaviour maintenance. High Harm Avoidance score reflects a worrying, pessimistic, fearful, doubtful and shy person. Low scores on this subscale are associated with a relaxed, optimistic, bold, confident, outgoing and vigorous individual. Harm Avoidance is associated with avoidance behaviour. A person scoring high on Persistence is hard working, industrious and will persist despite frustration. A low scorer on the Persistence subscale is inactive, indolent, modest, underachieving, quitting pragmatist who easily gives up. Persistence subscale was separated from the factor of Reward Dependence and also reflects behaviour maintenance.

Self-directedness expresses individual self-acceptance. Individuals with high scores on this character are mature, strong, responsible and reliable, purposeful, resourceful and effective, self-accepted and have habits congruent with long term goals. Low scores indicate an immature, fragile, blaming and unreliable, purposeless, inert and ineffective, self striving person with habits incongruent with their long term goals. Cooperativeness measures acceptance of other people. A person with high scores on this character subscale is socially tolerant, empathic, helpful, compassionate and constructive, ethical and principled. Low scores denote a socially intolerant, critical, unhelpful, revengeful and destructive and opportunistic person. Self-transcendence is a character denoting to what degree the person feels a part of the greater Universe. High scores on this scale indicate someone who is wise and patient, creative and self-forgetful and united with the Universe. Low scores are indicative of an impatient, unimaginative, self-conscious and proud individual who lacks humility.

O-LIFE

Schizophrenia, and more broadly psychosis, can be thought of as dichotomous from the 'normal', healthy state. An alternative is a model of continuum, starting from the point of super-efficient information processing and extending to a pathological or at least prodromal phenotype. Claridge (Claridge, 1990, 1997) conceptualized schizotypy as a personality trait expressing psychosis-proneness integrating the clinical symptoms and personality features.

The concept of schizotypy is based on the model of continuum from the perfectly accurate reality perception and healthy emotionality to the frank schizophrenic phenotype characterised by hallucinations, delusions and psychosis. According to this model every individual falls somewhere in between the two extremes in terms of their perceptions and interpretations of their sensory inputs and emotionality. The Oxford-Liverpool Inventory of Feelings and Experiences (Mason, Claridge, & Jackson, 1995) is a widely used and validated schizotypy measure incorporating several pre-existing scales related to psychosis proneness.

The O-LIFE does not measure a milder version of the clinical symptoms, but centres on quantifying four factors which are manifested in the schizophrenic phenotype and are present in the non-clinical population. These are Unusual Experiences, Cognitive Disorganisation, Introvertive Anhedonia and Impulsive Non-conformity. Unusual Experiences is a factor encompassing a range of abnormal perceptions and interpretations of the sensory inputs. The questions probing such experiences are akin to descriptions of hallucinatory experiences. Cognitive Disorganisation is linked to information processing, attentional difficulties and misinterpretation of the sensory input. Introvertive Anhedonia centres on depression-like feelings and experiences including low mood states and passivity. The final factor of Impulsive Non-conformity covers psychopathy-like traits focussing on the emotional experience poverty and impulsivity.

Each of the O-LIFE subscales covers a different area with a potential for abnormality: tendency to misperceive and misinterpret the incoming information (Unusual Experiences), inability to process information efficiently (Cognitive Disorganisation), tendency towards negative emotionality (Introvertive Anhedonia) and tendency towards suspiciousness and paranoia (Impulsive Non-conformity).

FSS

Fear is different from anxiety (J. A. Gray & McNaughton, 2000; McNaughton & Corr, 2004) and is important in the context of defensive, orienting responses such as startle. Fear in the sense of crippling, clinical condition, can be found in a milder form in the non-clinical population in a manner akin to schizotypy (everyone is fearful to a degree, but only a few develop fears so intense that they affect their daily functioning).

Fear Survey Schedule (Wolpe & Lang, 1977) is a questionnaire derived from clinical data on fear eliciting factors and it measures individual sensitivity to the

listed fear-eliciting items. The questionnaire comprises five factors including Fear of Animals, Interpersonal Fear, Fear of Tissue Damage, Fear of Noises and Classical Phobias. The total score is an indication of the general 'fearfulness'. Fear of Animals measures the individuals fear experiences related to animal stimuli. Interpersonal Fear covers a range of fears elicited in the situations involving interaction (real or anticipated) with people. Fear of Tissue Damage covers fear experiences related to disease, injury, death and associated concepts (e.g. hospitals, doctors etc.). Fear of Noises maps individual fears related to auditory stimuli. Classical Phobias encompass fears related to the classical phobias as defined in the clinical tradition and practice, i.e. fears related to space, height, dirt etc. High scores on any of the scales indicate increased sensitivity to the stimuli within the class comprising the factor. The therapeutic use of the scale is the measurement of the stimuli desensitization (as the result of therapy), i.e. loss or reduction in the fear or any other unpleasant feelings elicited by the stimuli.

STAI

Subjective feelings of tension, apprehension and nervousness and autonomic nervous system arousal are the hallmarks of anxiety states. Anxiety can be a fleeting state or a more enduring tendency to experience the anxious states with the associated thoughts, feelings and sensations (and their physiological aspects).

Defensive behaviours are closely linked to anxiety and thus anxiety is a likely candidate for a factor impacting on both the startle response and PPI. Spielberger's (Spielberger, 1983) model of anxiety splits the anxiety concept into trait and state anxiety. State and Trait Anxiety Inventory (Spielberger, 1983) has been widely used in anxiety related research and has been extensively validated.

Trait anxiety maps individual differences in anxiety proneness and the free-floating anxious state. It also maps the frequency and intensity of the anxiety experiences in the past and thus indicates the probability and intensity of state anxiety in a threatening situation. State anxiety is related to the specific context and is a snapshot of the elevated or diminished anxiety levels at the given point in time. The questionnaire measures anxiety expressed at the cognitive level, and the individual needs to be able to monitor their emotional state adequately for their answers to accurately reflect their anxiety proneness.

PANAS

The emotional state is known to affect the startle response and PPI. The Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988) allows the individual to indicate the valence and degree (intensity) of their mood. The questionnaire items contain both positive and negative mood labels (adjectives) and the final score reflects the mood traits or states (subject to the instruction, general moods versus specific point in time moods). Positive and negative affect states may reflect heightened activity within the punishment or reward cue sensitive systems. It is presumed that even momentary moods reflect the general tendency towards one or the other type of mood.

High Positive Affect is associated with high energy, full concentration, and pleasurable engagement; low scores indicate sadness and lethargy. High Negative Affect reflects subjective distress and a variety of aversive mood states, such as anger, fear, guilt and nervousness; low scores translate into calmness and serenity.

BIS/BAS

The BIS/BAS Scales (Carver & White, 1994) measure two systems proposed in Gray's model of anxiety (J.A. Gray, 1982; J. A. Gray & McNaughton, 2000): the behavioural inhibition system (BIS): sensitive to cues of punishment, non-reward and novelty, and behavioural approach system (BAS): sensitive to cues of reward, non-punishment and escape from punishment. The two systems are presumed to relate to two distinct, orthogonal affective qualities, with BIS relating to negative affect and BAS relating to positive affect. High BIS scorers experience heightened negative affect in the presence of punishment cues, whereas high BAS scorers experience positive affect in the presence of reward cues.

The BIS/BAS Scales consist of the following subscales: BIS, BAS Fun, BAS Drive and BAS Reward Responsiveness. The BIS subscale measures the likelihood of behaviour avoidance or termination in the presence of punishment cues and subjective negative affect elicited by such cues. BAS Reward Responsiveness measures sensitivity to reward cues, BAS Drive measures the intensity and probability of approach behaviour, and BAS Fun measures the subjective positive affect derived from the approach behaviour. Principal component analysis in a large sample demonstrated that the four subscales neatly fit two factor solution of BIS and BAS (Jorm , et al., 1998).

The original model has been updated and simplified by McNaughton and Corr (McNaughton & Corr, 2004). The behavioural outcomes of the sensitivity to

reward or punishment cues remain the same, i.e. approach towards appetitive cues and avoidance of aversive cues. However, the mechanism leading to the approach or avoidance behaviours differs from the original proposal.

Anxiety and fear are separate entities, but they are underpinned by overlapping neural substrates which can be concurrently activated. Whether fear or anxiety are experienced depends on two dimensions, one categorical and one hierarchical. The categorical dimension is whether the animal can escape (defensive avoidance, fear) or not (defensive approach, anxiety). The second dimension is perceived distance to threat (defensive distance) and this dimension is graded. The closer the animal is to defensive response eliciting stimuli, the shorter the neural pathway leading to the response generation. Distant dangers result in engagement of the prefrontal cortex, whereas close danger reduce the stimuli processing to periaqueductal grey.

Fear has the function of moving the animal away from danger and includes the fight/flight/freezing options, whereas anxiety is active at times of approach/avoidance conflict and moves the animal towards the danger. Punishment (or danger) cues activate fight/flight/freeze system (FFFS), whereas reward cues activate behavioural approach system (BAS). The behavioural activation system (BAS) is involved in obtaining reward and BAS activation increases the likelihood of approach behaviour.

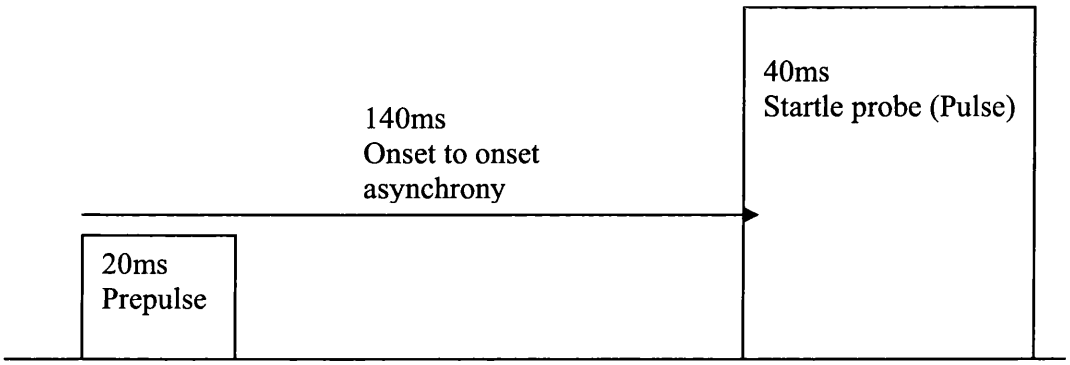
In situations of conflict between approach (appetitive cues, BAS) and avoidance (aversive cues, FFFS), the behavioural inhibition system (BIS) is activated. However, BIS does not function solely to inhibit the ongoing or imminent behaviour, but rather leads to conflict resolution and risk assessment, with a tendency to increase the likelihood of avoidance. The BIS activating conflict need not be approach and avoidance only, avoidance and avoidance or approach and approach conflicts can equally lead to BIS activation. BIS increases arousal and attention and it can initiate both approach and avoidance behaviour (to resolve the conflict).

The update of the BIS/BAS model outlined above does not invalidate the usefulness of the BIS/BAS Scales, indeed it offers a better understanding of at least one factor (BIS), although the fight/flight/freeze system (FFS) would need to be separated from the BAS factor to accurately reflect the new additions to the model.

2.3 Physiological data collection

Four types of white noise stimuli, with almost instantaneous (less than 1 ms) rise time, were used: pulse-alone (115dB; 40ms duration) and three discrete prepulses (80, 85 and 90dB; 20ms duration).The stimuli were presented binaurally via headphones over 70dB continuous white noise background. White noise, discrete prepulses are most effective in eliciting PPI and prepulse inhibition increases with increasing prepulse intensity (Blumenthal, 1995; Harbin & Berg, 1983; Hoffman & Searle, 1968). Figure 2.3 shows a diagram of the trial structure.

Figure 2.3 Trial structure



The stimuli were combined into eight different trial types (Table 2.1, Appendix 2): pulse-alone trial (70dB, 115dB), three prepulse and pulse trials (80dB, 115dB; 85dB, 115dB; 90dB, 115dB), three prepulse-alone trials (80dB, 70dB; 85dB, 70dB; 90dB, 70dB), and one 'blind' trial (70dB, 70dB). Lead (inter-stimulus) interval of 140 ms was used in all trials. The reason for selecting extended onset to onset asynchrony value was the need to differentiate clearly between the prepulse-elicited responses and startle responses. Since PPER has not been extensively investigated in terms of the waveform characteristics, it was safer to allow a longer gap between the stimuli presentation, at the same time the time lapse was not long enough to create a paradigm dramatically different from the commonly used 120ms ISI. The same stimuli intensities and lead interval were used in all the studies. PPI in humans reaches its maximum values at 120ms (D. L. Braff, et al., 1992; N. R. Swerdlow,

Paulsen, et al., 1995), although some studies report 60ms as the lead interval (inter-stimuli interval) leading to maximum PPI (Flaten, Nordmark, & Elden, 2005). Repeated presentation of prepulse stimuli does not affect PPI levels (Lipp & Krinitzky, 1998). The differential values of ISI for achieving the maximum PPI are the result of reporting differences in the published studies. Some authors report onset to onset asynchrony, whilst others report the gap between the end of the first stimulus presentation and the beginning of the second stimulus presentation.

The inter-trial intervals varied between the studies and are described in greater details in each study's method section. Trials were always presented in a fixed pseudorandom order.

Human EMG startle reflex testing system (SR-HLAB, San Diego Instruments, US) was used to deliver the acoustic stimuli and record, store and score (off line) the EMG response. The stimuli were delivered via headphones. Sound levels were measured using Precision Sound Level Meter (Type 2203; Bruel & Kjoer, Copenhagen). Sound levels were tested and calibrated monthly and rechecked before every study. EMG was recorded from the orbital part of the left orbicularis oculi. The Ag/AgCl electrodes were placed approximately 10mm (centre-to-centre) apart, with one electrode positioned medially beneath the pupil and the other laterally to the first one.

The reference electrode was placed on the forehead. Standard conducting gel (Signa gel, Ref 15-25, Parker Laboratories, US) was used. The amplifier gain was kept constant at 2.5. The EMG activity was recorded continuously with sampling rate of 1000Hz in all the studies bar the first one (250Hz rate used). The lower frequency cut off was set to 100Hz and the upper to 1000Hz (33Hz and 300Hz respectively for the first study).

The session begun with acclimation (to the background noise) followed by the presentation of the habituation stimuli (70dB, 115dB pulse alone trials) which were followed by the specified number of presentations of each stimulus type. The stimuli were presented in a fixed, pseudo-random order. If the study paradigm contained two separate parts counterbalancing was used.

EMG data were scored offline by the analytic program of the SR-HLAB unit (Software 6703-0009-B). The data were filtered and smoothed and each trial was visually inspected for the signs of corrupt EMG. Scoring parameters were chosen to maximize recognition of prepulse responses and minimize scoring spontaneous EMG

activation as valid responses. The analysis software used several parameters to determine if a response met the user defined validity criteria. The scoring parameters were adjusted to reflect the trial events sequence in each study, the details of the scoring criteria (baseline, response window, digital filter) are provided in the method section for each of the studies. The response criterion (i.e., the number of standard deviations above the baseline, a *z* score) was set to 2*SD* ($z > 2$) and was the same for all the studies.

2.4 Design

Participants were drawn from the University based non-clinical population and included both staff and students. Prior to any data collection the potential participants were informed of the exclusion criteria and provided their informed consent. Individuals with a self-declared history of head injury, neurological disorders, psychiatric disorders or drug abuse did not take part in any of the studies. In all studies (save the first one) smokers were excluded at the point of recruitment, in the first study they were excluded prior to any statistical analysis, since nicotine is known to affect PPI in rodents and humans (Baschnagel & Hawk, 2008; Della Casa, Hofer, Weiner, & Feldon, 1998; Evans, Gray, & Snowden, 2005; George, et al., 2006; Grillon, Avenevoli, Daurignac, & Merikangas, 2007; Hutchison, Niaura, & Swift, 2000; Kumari, Checkley, & Gray, 1996; Kumari, Cotter, Checkley, & Gray, 1997; Kumari, Toone, & Gray, 1997; M. Li, Mead, & Bevins, 2009; Postma, et al., 2006). The use of alcohol was also recorded (frequency and time of the last alcohol intake) since alcohol has been shown to affect PPI (Hutchison, et al., 1997) with reduction in participants with low baseline PPI and increase in those with high baseline PPI. Participants completed the self-report demographic and personality questionnaires and were exposed to all the stimuli (with 5-10 presentations of each stimulus type). A mixed design was used, with sex and personality measures as the between-participant factors and the eyeblink-eliciting stimuli as the within-participant factor. Details of the design for each study are described in the relevant sections. Each study was separately approved by the Ethics Committee within the Psychology Department of Swansea University.

2.5 Data Scoring and Statistical Analysis

All trials were visually inspected prior to statistical analysis to exclude any trials that included contamination of the eye blink response to the stimuli by spontaneous blinking. Stimuli-elicited response amplitude, onset, and peak latency values were derived from the data recorded in each trial. Stimuli-driven responses were defined by the scoring parameters presented in the physiological data collection section above. The prepulse and pulse parts were scored separately in every trial. Stimuli-elicited response probability was calculated by dividing the number of valid responses in the relevant part of each trial by the total number of trials (of each type). The response probabilities were calculated for each trial type separately. Stimuli-elicited responses were analyzed in terms of amplitude, onset, peak latency, and onset to peak latency.

Two approaches to deriving the 'averages' of the pulse-elicited responses in prepulse and pulse trials were possible. One method would have been to average the responses across all trials in the given trial type and then use that average value in the startle response modification formula. The other method involved calculating the startle response modification for each and every trial and then averaging across the conditions. The data in the presented set of studies were subjected to the second form of averaging, since it offered a more sensitive measure of the possible startle response modification.

Startle response modification (SRM) was calculated as a percentage reduction from the average startle response in pulse-alone trials (startle response) as compared to the responses in trials in which a prepulse preceded the pulse. Blumenthal and colleagues recommended proportion of the difference from control as the best method of quantifying startle response modification (Blumenthal, Elden, & Flaten, 2004).

SRM was calculated with the following formula:

$$SRM = \left[\frac{(PulseAloneAmplitude - \Pr epulseAndPulseAmplitude}{PulseAloneAmplitude} \right] * 100$$

Positive products of the SRM formula represented a reduction in the amplitude of startle response when a prepulse preceded the pulse. Such startle response reduction was termed prepulse inhibition (PPI) and all pulse-elicited

responses in prepulse and pulse trials were included, i.e. the $z > 2$ threshold was abolished for this type of SRM. This decision was motivated by the observation that the pulse-elicited responses in prepulse and pulse trials were often reduced to the level of EMG baseline, and would have been excluded from the statistical analysis thus obfuscating true SRM rates.

Negative products of the SRM formula were recoded as prepulse facilitation (PPF), a percentage increase in pulse-elicited response amplitude, however such responses had to exceed the $z > 2$ level to be included in the analysis. This approach was adopted to avoid inclusion of minor increases in the EMG activity which may have been due to spontaneous EMG activation, rather than a true response increase. Prepulse facilitation (PPF) occurs at very long (>500ms) (Graham, 1975; Hoffman & Searle, 1968) or very short (<37.5ms) (Plappert, Pilz, & Schnitzler, 2004) intervals. Previously prepulse facilitation at short intervals was reported only for lead stimuli and startle probes presented in different modalities (Blumenthal, 1999).

Figures 2.4 - 2.6 show EMG waveforms recorded from the same participant. Figure 2.5 shows a typical prepulse inhibition waveform and Figure 2.6. a prepulse facilitation waveform. Figures 2.5 and 2.6 also show prepulse-elicited responses. Prepulse-elicited responses (prepulse reactivity) reach easily detected and quantified amplitudes (Csomor, et al., 2005).

Figure 2.4 EMG waveform recorded in pulse alone trial showing pulse-elicited response (pulse stimulus presented at 160ms)

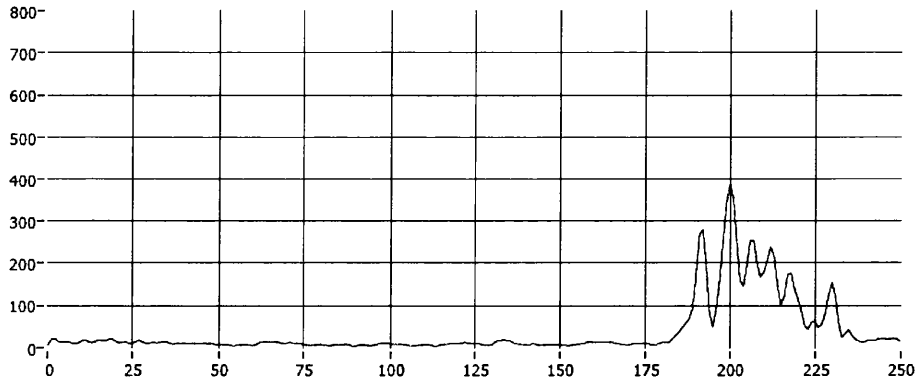


Figure 2.5 EMG waveform recorded in prepulse and pulse trial showing pulse-elicited response decrease (prepulse stimulus presented at 20ms, pulse stimulus presented at 160ms)

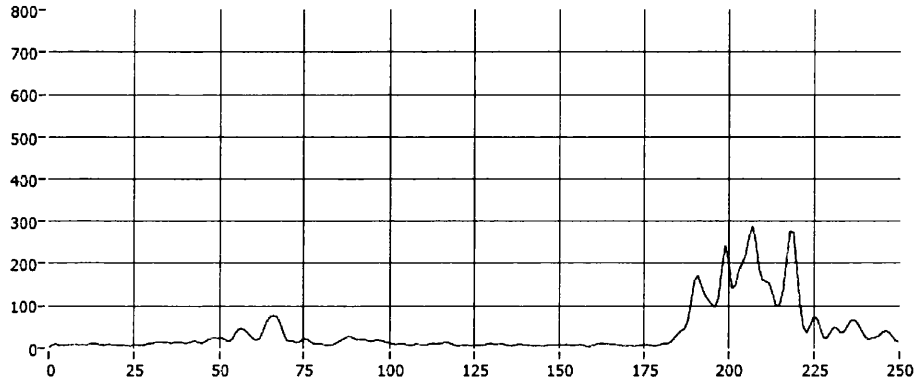
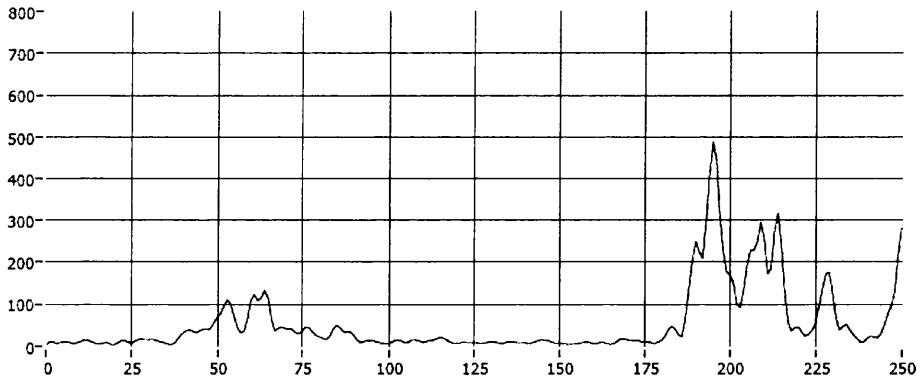


Figure 2.6 EMG waveform recorded in prepulse and pulse trial showing pulse-elicited response increase (prepulse stimulus presented at 20ms, pulse stimulus presented at 160ms)

.



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A conservative approach was adopted for the data analysis. The data was examined for the normality of distribution using Kolmogorov-Smirnov tests on each variable to ensure normal distribution of values. Outliers were excluded from further analysis. Following this treatment the data were normally distributed and parametric statistical tests of paired t-test, repeated measures ANOVA and correlations were used. The choice of the statistical approach was based on the methods employed in the publications in this research area and for the ease of effect comparison and procedure replication. Moreover prepulse reactivity is not a well established research

concept and caution was necessary in the choice of the statistical tests to avoid finding significant, but not robust effects using a more complex approach.

3 Prepulse reactivity, prepulse inhibition and paradoxical prepulse facilitation

3.1 Introduction

The relationship of prepulse processing to startle response modification remains unclear with conflicting reports of the significance (or indeed existence) of prepulse reactivity (PPER). The consequences of the prepulse-elicited response characteristics (for example peak values, length of the motor response) for the subsequent startle response modification are not well established. If prepulse reactivity has no effects on the subsequent startle response modification, then no significant associations should emerge between any dimension of prepulse reactivity (response size or temporal characteristics) and startle response modification.

Some of the published studies were criticized for their lack of 'no stimuli' and 'lead stimuli only' trials. The absence of 'no stimuli' trials makes it difficult to establish a baseline EMG activity values, and the lack of prepulse-alone trials has been criticized on the basis of subsequent startle response contaminating the PPER waveform. If prepulse-elicited responses are simply spontaneous EMG activation, then they should be no different from the EMG activity in the 'no stimulus' trials. If the subsequent startle probe presentation does not affect prepulse-elicited responses, then the prepulse-elicited responses in prepulse-alone, compared to prepulse and pulse trials should not be significantly different.

Prepulse reactivity is potentially another individual difference in a manner akin to PPI levels. Some individuals may be consistently responding to the lead stimuli while others fail to do so. Prepulse reactivity has not been previously investigated in the context of personality. Personality factors relevant to sensorimotor gating may exert some influence on prepulse reactivity in terms of response probability or other characteristics. If personality factors relevant to sensorimotor gating are not associated with prepulse reactivity, then the scores on such psychometric measures should bear no relationship to prepulse-elicited motor responses.

The main aims of the presented chapter are establishing the prevalence and characteristics of PPER in healthy humans and associations between PPER and

startle response modification. The impact of individual differences on PPER and startle response modification is also considered.

The following hypothesis will be tested:

1. PPER is significantly different from spontaneous EMG
2. PPER is not affected by the subsequent startle probe presentation
3. PPER impacts on startle response modification
4. PPER is a consistent tendency to generate motor responses to weak stimuli
5. Startle response modification is not limited to classical inhibition

6 Individual differences might be associated with PPER and SRM

3.2 Methods and materials

3.2.1 Participants

Fifty nine participants were recruited from university employees and postgraduate students (age range: 19-65 years, $M = 30$, $SD = 11$; 29 females, age range: 20-61, $M = 31$, $SD = 12$, 30 males, age range: 19-65, $M = 29$, $SD = 10$). Each was paid £3.00 for their participation.

Exclusion criteria comprised self-declared suboptimal hearing, tinnitus, drug abuse or psychiatric disorder history. The times of last nicotine and alcohol consumption were recorded and hearing acuity was tested at 40dB. Six participants responded in less than seven pulse alone trials (ten such stimuli presented in the main session) and were excluded as non-responders. Two participants had abnormally elevated baseline recordings (equipment failure) and one participant had adverse reaction to the intense startle probes and failed to complete the session. Ten smokers were excluded from further analysis since smoking is known to affect prepulse inhibition of the acoustic startle response (PPI).

The final sample size was thus reduced to 40 participants (age range: 19-65, $M = 31$, $SD = 12$; 19 females, age range: 21-61, $M = 32$, $SD = 13$, 21 males, age range: 19-65, $M = 30$, $SD = 12$).

3.2.2 Materials

Physiological and questionnaire data were collected (auditory stimuli and physiological data collection are described below). The Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE), The BIS/BAS Scales

(BIS/BAS), State and Trait Anxiety Inventory (STAI), Positive and Negative Affect Scale (PANAS), Fear Survey Schedule (FSS) and the Temperament and Character Inventory (TCI) were used to measure personality characteristics. Demographic questionnaire was used to record age, sex and the time of last alcohol and nicotine intake.

The majority of the self-report questionnaires (O-LIFE, TCI, FSS, BISBAS, STAI trait part) were completed prior to the arrival in the laboratory. Upon arrival, participants completed the PANAS, the state part of the STAI and a demographic questionnaire. The references for all these personality questionnaires are provided in Chapter 2 and the copies of the questionnaires are in Appendix 2.

3.2.3 Physiological data collection

Four types of white noise stimuli, with almost instantaneous (less than 1 ms) rise time, were used: pulse-alone (115dB; 40ms duration) and three discrete prepulses (80, 85 and 90dB; 20ms duration).The stimuli were presented binaurally via headphones over 70dB continuous white noise background.

The stimuli were combined into eight different trial types: pulse-alone trial (70dB, 115dB), three prepulse and pulse trials (80dB, 115dB; 85dB, 115dB; 90dB, 115dB), three prepulse-alone trials (80dB, 70dB; 85dB, 70dB; 90dB, 70dB), and one 'blind' trial (70dB, 70dB). Lead (inter-stimulus) interval of 140 ms was used in all trials. The inter trial interval ranged from 9-21s (mean = 12s). Trials were presented in a fixed pseudorandom order in ten blocks, each block containing all eight trial types presented once (86 trials total including 6 habituation trials) with the exception of 80dB, 115dB trial type presented 11 times and 85dB, 115dB trial type presented 9 times (programming error).

The details of the EMG recording are described in Chapter 2. The EMG activity was recorded continuously (sampling rate 250Hz) for 250ms starting at the trial onset. EMG data were low (300Hz) and high (30Hz) band pass filtered. The session begun with 3 minutes of acclimation (to the background noise) followed by six presentations of the 115dB stimuli (70dB background noise, 40ms duration, 5s inter-trial interval). The presentation of all the trial types in a fixed, pseudorandom order (see above) then followed. The session lasted approximately 30 minutes.

The scoring parameters were held constant and set to: baseline 20ms, response window 100ms, rolling average 3, Butterworth digital filter order 1 (pass frequency 124), response criterion 2. The only parameters changed for scoring prepulse or pulse part of trial responses were the start (21ms for prepulse-elicited response, 160ms for pulse-elicited response) and the end (140ms for prepulse-elicited response, 250ms for pulse-elicited response) of the analysis. Each trial was divided into two parts: the prepulse part and the pulse part. A response had to exceed the response criterion by 2*SD* ($z > 2$) to be classified as valid.

3.2.4 Design

Participants completed the self-report questionnaires and were exposed to all the stimuli. A mixed design was used, with sex and personality measures as between-participants factors, and stimulus-driven responses as within-participant factor.

3.2.5 Data Scoring and Statistical Analysis

All trials were visually inspected prior to statistical analysis to exclude any trials that included contamination of the eye blink response to the stimuli by spontaneous blinking. The response means to each trial type in terms of amplitude, onset and peak latency were derived for each individual and in all trial types.

In every trial the prepulse (20-120ms) and pulse (140-250ms) parts were scored separately. Prepulse-elicited response probability was calculated by dividing the number of valid responses in the prepulse part of each trial by the total number of trials (of each type). The response probabilities were calculated for the prepulse parts of each trial type separately (eight trial types, eight response probabilities). Prepulse-elicited responses were analyzed in terms of probability, amplitude, latency, onset, and duration.

Startle response modification calculation is described in Chapter 2.5. All pulse-elicited responses (including those with $z < 2$) in prepulse and pulse trials were admitted for the calculation of prepulse inhibition (PPI) since prepulse presentation can lead to absolute reflex suppression. A more conservative approach was adopted for prepulse facilitation (PPF) and only pulse-elicited responses exceeding the $z > 2$ criterion were accepted for further analysis.

The main aim of the presented study was systematic examination of the prepulse-elicited responses, associations between prepulse reactivity and startle response modification and the relationships between psychometric and physiological measures.

3.3 Results

3.3.1 Prepulse reactivity

Prepulse-elicited responses were assessed using the same criteria as pulse elicited responses (i.e. a valid exceeded z score > 2 in amplitude). Prepulse-elicited motor responses were small compared to the baseline startle responses or the inhibited pulse-elicited responses in PPI trials; however their amplitudes exceeded the set criterion of $z > 2$. Prepulse-elicited response frequency increased with increasing prepulse intensity. Thirty percent of the sample (12 out of 40) responded to 80dB prepulses, forty five percent (18 out of 40) to 85dB prepulses and sixty percent (24 out of 40) to 90dB prepulses.

3.3.1.1 Prepulse reactivity compared to spontaneous EMG activation

If prepulse-elicited responses were simply a spontaneous EMG activation, they would not be statistically different from the spontaneous EMG activation index (i.e. response probability in the absence of eyeblink-eliciting stimuli). Paired samples t-tests were conducted on the probability of prepulse-elicited responses and probability of responses in trials with no prepulse stimuli presented. Two trial types facilitated assessing the spontaneous EMG activation in the absence of prepulse stimulus: the prepulse part of the pulse alone trial (70dB, 115dB), and the prepulse part of the 'blind' trial (70dB, 70dB).

The probabilities of valid eyeblink responses were significantly higher at all prepulse intensities in all trials with prepulses compared to pulse-alone trials (70dB, 115dB) [prepulse and pulse trials: 80dB, $t(39) = 2.19$, $p = .03$; 85dB, $t(39) = 4.32$, $p < .01$; 90dB, $t(39) = 5.81$, $p < .01$; prepulse alone trials: 80dB, $t(39) = 3.34$, $p < .01$; 85dB, $t(39) = 3.98$, $p < .01$; 90dB, $t(39) = 6.11$, $p < .01$].

Comparison of valid response probabilities in prepulse and pulse trials against the probability of valid responses in the 'blind' (70dB, 70dB) trials, also indicated significant differences [prepulse and pulse trials: 80dB, $t(39) = 2.70$, $p = .01$; 85dB, $t(39) = 4.51$, $p < .01$; 90dB, $t(39) = 6.01$, $p < .01$; prepulse alone trials:

80dB, $t(39) = 3.91$, $p < .01$; 85dB, $t(39) = 4.30$, $p < .01$; 90dB, $t(39) = 6.26$, $p < .01$]. All the above results are in Appendix 3, Table 3.1.

3.3.1.2. Prepulse reactivity in prepulse alone and prepulse and pulse trials

Paired samples t-tests were conducted to compare spontaneous EMG activation amplitude and probability recorded in the prepulse part in pulse alone trials versus no stimuli 'blind' trials. The impact of the pulse presentation should be evident in the differences between baseline EMG activity in trials with and without the startle probe (pulse). The potential modulating effect of the subsequent startle probe presentation on prepulse-elicited responses was also assessed by gauging the differences in prepulse-elicited responses in trials with and without the startle probe presented (prepulse alone trials versus prepulse and pulse trials with the same prepulse intensity). The outcomes of the comparisons were not statistically significant (all p values not significant) demonstrating that the subsequent startle stimulus presentation did not affect prepulse-elicited response amplitude or probability (see Appendix 3, Table 3.2).

Paired-samples t-tests also revealed lack of statistically significant differences between the temporal characteristics of prepulse-elicited responses (onset, peak latency, onset to peak latency) in trials with and without the startle probe (Appendix 3, Table 3.3).

3.3.1.3. Prepulse intensity effects on prepulse-elicited responses

Prepulse intensity had a significant effect on prepulse-elicited response probabilities. More intense prepulses resulted in more frequent prepulse-elicited responses in prepulse alone [F (2,40) = 28.73, $p < .001$] and prepulse and pulse [F (2,40) = 22.12, $p < .001$] trials. Sex had no effect on prepulse-elicited response probabilities [F (2,40) = .67, ns and F (2,40) = 1.39, ns]. The effects of prepulse intensity on prepulse-elicited response probabilities are shown in Appendix 3, Figure 3.1.

Significant positive correlations [correlations ranging from $r(38) = .52$, $p < .01$ to $r(38) = .93$, $p < .01$] appeared for prepulse reactivity probability at all prepulse intensities in all conditions (Appendix 3, Table 3.4).

Prepulse-elicited response amplitude was also intensity dependent in prepulse and pulse trials [F (2,40) = 4.94, $p = .02$] and prepulse alone trials [F (2,40) = 5.59, p

=.01]. The more intense stimuli resulted in larger responses (Figure 3.2). Sex had no effect on the prepulse-elicited response amplitudes in either prepulse and pulse [F (2,40) = .01, ns] or prepulse alone [F (2,40) = 1.47, ns] trials.

Prepulse-elicited amplitude values were positively correlated across all the conditions, but only some associations reached significance [correlations ranging from $r(18) = .46, p = .04$ to $r(22) = .94, p < .01$] (Appendix 3, Table 3.5).

More intense stimuli led to faster prepulse-elicited response onset (Appendix 3, Figure 3.3). However the intensity effects were not statistically significant for prepulse-elicited response onset in prepulse and pulse trials [F (2,40) = 2.12, ns] and the intensity effect just reached significance in prepulse alone trials [F (2,40) = 4.34, p =.04]. Sex had no effect on prepulse-elicited response onsets in either prepulse and pulse [F (2,40) = 1.57, ns] or prepulse alone trials [F (2,40) = 1.15, ns].

Prepulse-elicited response onsets were positively correlated across all the conditions, though only some associations reached significance [correlations ranging from $r(18) = .51, p = .02$ to $r(9) = .83, p = .01$] (Appendix 3, Table 3.6).

Increased prepulse intensity resulted in lower peak response latency (Appendix 3, Figure 3.4). Intensity dependence was evident in the peak latency values in both prepulse and pulse trials [F (2,40) = 5.7, p = .01] and prepulse alone trials [F (2,40) = 11.01, p < .001]. Sex had no effect on peak latency in either prepulse and pulse [F (2,40) = 1.23, ns] or prepulse alone [F (2,40) = 1.43, ns] trials.

Peak latencies were positively correlated across all conditions, but not all the associations reached significance [correlations ranging from $r(16) = .50, p = .03$ to $r(10) = .89, p < .01$] (Appendix 3, Table 3.7).

Prepulse-elicited response onset to peak latency time was not intensity dependent in prepulse and pulse [F (2,40) = .19, ns] or prepulse alone trials [F (2,40) = 2.82, ns]. Sex had no significant effect on the onset to peak latency values in either prepulse and pulse [F (2,40) = 1.61, ns] or prepulse alone [F (2,40) = .15, ns] trials.

Despite the lack of significant effect the onset to peak latency values decreased with increasing prepulse intensity (Appendix 3, Figure 3.5) with prepulse intensity related reduction, but only in prepulse and pulse trials.

Positive correlations appeared for the onset to peak latency in all conditions, but as with the peak latency, only some of these reached significance [correlations ranging from $r(18) = .45, p = .04$ to $r(16) = .79, p < .01$] (Appendix 3, Table 3.8).

3.3.2 Startle response modification: PPI and paradoxical PPF

Prepulse presentation led to two types of startle response modification. All the participants exhibited the traditional prepulse inhibition, but in thirty seven percent of the sample (15 out of 40) the startle responses were increased, rather than decreased, following prepulse presentation in at least three trials in each prepulse and pulse trial type. Sixty-seven percent of the sample (26 out of 40) exhibited at least one instance of such startle response modification. This unusual modification of the startle response under the conditions presumed reliably to lead to maximal prepulse inhibition was dubbed 'paradoxical prepulse facilitation'. In majority of the published studies the inhibition values are derived from averaging the responses across trial types (collapsing data across trial types) and any facilitation becomes 'averaged out' across inhibition.

Prepulse inhibition probability and percentage were derived from all prepulse and pulse trials ignoring the response criterion ($z > 2$) since prepulse presentation can lead to 100% suppression of the startle response (i.e. no motor response above EMG baseline activity evident in response to the pulse presentation). Increased pulse-elicited responses in prepulse and pulse trials were treated more conservatively and only pulse elicited responses exceeding $z > 2$ were admitted for the analysis. The paradoxical nature of such startle modification stipulated caution in the inclusion of the responses to avoid interpreting spontaneous EMG modification (in prepulse and pulse trials) as a systematic and somehow meaningful change.

3.3.2.1 Prepulse inhibition (PPI): probability and percentage change

Increasing prepulse intensity led to increased probability [F (2,40) = 17.11, $p <$ .001] and percentage change [F (2,40) = 39.60, $p < .01$] of prepulse inhibition (see Appendix 3, Figures 3.6 – 3.7) although a U-shaped distribution appeared for PPI probabilities. Sex did not have a significant effect on either the probability [F (2,40) = .58, ns] or the percentage change in prepulse inhibition [F (2,40) = .05, ns].

3.3.2.2 Paradoxical prepulse facilitation (PPF): probability and percentage change

Startle response amplitude increases, rather than decreases following prepulse presentation were rare, and increasing prepulse intensity led to decreased probability of paradoxical prepulse facilitation [F (2,40) = 16.24, $p < .01$]. Sex did not have a significant effect on the probability [F (2,40) = .24, ns] of this type of startle

response modification. Increased startle responses in trials with 90dB prepulses were too rare to be entered into percentage change comparisons, and only 80db, 115dB and 85dB, 115dB trials were compared. Paired-samples t-test comparing the two conditions revealed no effect of prepulse intensity on the percentage response increase [t (40) = -.45, ns]. The effects of prepulse intensity on PPF probability and percentage are shown in Appendix 3, Figure 3.8 and Figure 3.9. Despite the lack of significant differences in PPF percentage at different prepulse intensities an inverted U-shaped distribution appeared.

3.3.2.3 Startle response modification: decreased and increased startle responses

The associations between the two startle response modification modes following prepulse presentation (decrease, PPI or increase, PPF) were analyzed to ascertain if they were mutually exclusive. The finite number of prepulse and pulse trials stipulated that, in terms of probability, an increased frequency of one modification type would lead to a decreased number of the other, unless neither type of startle response modification prevailed in the majority of cases.

Prepulse inhibition versus prepulse facilitation: probability

The more numerous were the instances of prepulse inhibition in the prepulse and pulse trials, the fewer were the trials with instances of prepulse facilitation. This relationship is expressed in the negative correlation between the probability and prepulse inhibition and probability of prepulse facilitation in prepulse and pulse trials [correlations ranging from $r(38) = -.41, p = .01$ to $r(38) = -.93, p < .01$], (Appendix 3, Table 3.9). Strong positive correlations within each startle response modification category (PPI or PPF) indicated consistency in the tendency to either decreased (PPI) or increased (PPF) startle responses following prepulse presentation [correlations ranging from $r(38) = .45, p = .01$ to $r(38) = .73, p < .01$], (Appendix 3, Table 3.9).

Prepulse inhibition versus prepulse facilitation: percentage values associations

There were no significant associations between percentage values of prepulse inhibition and prepulse facilitation. There were significant positive correlations within prepulse inhibition percentage change, indicating consistency in startle response percentage reduction [correlations ranging from $r(38) = .76, p < .01$ to $r(38) = .82, p < .01$], but no such systematic relationships emerged for prepulse facilitation (Appendix 3, Table 3.10).

Startle response modification percentage change versus probability

Significant negative correlations were present between PPI percentage change and probability of PPF, indicating that individuals with efficient startle response inhibition had a reduced chance of the alternative startle response modification (paradoxical PPF) [correlations ranging from $r(38) = -.39, p = .01$ to $r(38) = -.70, p < .01$], (Appendix 3, Table 3.11). PPI probability and PPF percentage change correlations failed to reveal any significant correlations (Appendix 3, Table 3.12).

3.3.3 Prepulse reactivity and startle response modification

Prepulse reactivity and startle response modification probability

The probability of prepulse inhibition in trials with the weakest 80dB prepulses was negatively correlated with the probabilities of prepulse-elicited responses to 85dB [prepulse and pulse trials: $r(38)= -.40, p = .01$] and 90dB [prepulse and pulse trials: $r(38)= -.39, p = .01$; pulse-alone trials $r(38)= -.40, p = .01$] prepulses.

Positive correlations were present between the probability of these (85dB and 90dB) prepulse-elicited responses and PPF following 80dB prepulse presentation [85dB prepulse and pulse trials: $r(38)= .36, p = .02$; 90dB prepulse and pulse trials: $r(38)= .37, p = .02$; 90dB pulse-alone trials: $r(38)= .39, p = .01$], (Appendix 3, Table 3.13).

In terms of response size there was only one significant positive correlation between responses to the mid-intense 85dB prepulses and PPF in trials with the weakest prepulses, but it appeared for a small number of participants only [$r(10)= .66, p = .02$], Appendix 3, Table 3.15.

Prepulse reactivity and startle response modification percentage

Individuals who were more likely to respond to 85dB and 90dB prepulses in prepulse and pulse trials had lower percentage PPI following 80dB prepulse presentation [prepulse and pulse trials: 85dB, $r(38)= -.33, p = .04$; prepulse and pulse trials: 90dB, $r(38)= -.34, p = .03$], (Appendix 3, Table 3.14). Lower percentage inhibition following the presentation of 85dB prepulses was also associated with increased probability of prepulse-elicited responses in 90dB prepulse and pulse trials [$r(38)= -.40, p = .01$] and prepulse-alone trials [$r(38)= -.37, p = .02$], (Appendix 3, Table 3.14).

Positive correlation emerged for prepulse reactivity in 80dB prepulse-alone trials and percentage startle response increase following 85dB prepulse presentation [$r(5)=$

.75, p = .04]. Increased probability of 90dB prepulse-elicited responses in prepulse-alone trials was positively correlated with percentage startle response increase in prepulse and pulse trials with the weakest, 80dB prepulses [r(13)= .61, p = .02], (Appendix 3, Table 3.14).

There were no significant correlations for PPER response size and SRM percentage change (Appendix 3, Table 3.16).

Prepulse reactivity peak latency and startle response modification

The impact of the temporal characteristics of the prepulse-elicited responses was also investigated. Extended processing of the prepulse may have had an effect on the subsequent startle response. No significant correlations appeared for prepulse-elicited response peak latency and startle response modification (of either type, PPI or PPF). Weak, but significant positive correlation appeared for 90dB prepulse-elicited response peak latency and PPI (percentage) in trials with 85dB prepulses [r(26)= .38, p = .04], (Appendix 3, Table 3.17). The lack of systematic associations between extended prepulse processing (later peak latency) and either startle response modification type implies limited influence of the duration of prepulse processing on the subsequent startle response modification.

Prepulse-elicited responses were manifested in a largely symmetrical waveform with a drop off following the peak response largely similar to the ascending trace preceding it. The onset-peak latency measured the lapse in time from the beginning of the response to the peak amplitude value. The symmetry of the waveforms allowed assessment of the lapse of time from the peak response to the point of return to the baseline EMG activity. The onset to peak latency values for the prepulse-elicited responses did not correlate with startle response modification (PPI or PPF) in terms of either probability or percentage change (Appendix 3, Table 3.17) indicating a limited impact of extended prepulse processing (in terms of motor response) on the subsequent startle response.

3.3.4 Individual differences in prepulse reactivity, PPI and PPF

Individual differences in terms of personality (and its consequences for attention, arousal and affectivity) were correlated with prepulse-elicited responses and spontaneous EMG activation in the absence of the experimental stimuli. Fear-potentiated startle is a well documented phenomenon, and highly anxious or fearful

individuals could have exhibited increased (or decreased) responsivity to the weak prepulses or indeed increased propensity towards spontaneous EMG activation.

Prepulse reactivity probability

Personality factors did not exhibit strong associations with motor response probabilities and only two weak correlations appeared. Novelty Seeking was positively correlated with the responses to the mid-intense, 85dB prepulses [$r(37)= .33$, $p = .04$], (Appendix 3, Table 3.18) and Tissue Damage subscale of the FSS scores were negatively correlated with probability of the weakest, 80dB prepulse – elicited responses [$r(37)= -.32$, $p = .04$], (Appendix 3, Table 3.18).

Prepulse reactivity amplitudes

Prepulse-elicited response amplitudes exhibited frequent significant correlations with the personality factors, indicating that the response size is a more sensitive measure than response probability in terms of individual differences.

High scores on Trait Anxiety were negatively correlated with the amplitude of spontaneous EMG activation in the 'blind' trials; however, such spontaneous EMG activation was a rare event, so any associations should not be overemphasized [$r(4)= -.91$, $p = .01$], (Appendix 3, Table 3.19). A negative association also appeared for Cognitive Disorganisation and the size of responses to the mid-intense, 85dB prepulses [$r(19)= -.55$, $p = .01$], (Appendix 3, Table 3.19). Novelty Seeking was negatively correlated with the amplitude of responses to the weakest, 80dB prepulses [$r(12)= -.58$, $p = .03$], (Appendix 3, Table 3.19). Reward Dependence was positively correlated with 85dB prepulse-elicited response amplitude [$r(19)= .56$, $p = .01$], (Appendix 3, Table 3.19). The total score on the FSS was negatively correlated with the spontaneous EMG activity in the 'blind' trials [$r(4)= -.88$, $p = .02$], (Appendix 3, Table 3.19). Interpersonal Fear subscale of the FSS was negatively correlated with spontaneous EMG activation both in the prepulse part of pulse-alone trials and in the 'blind' trials [$r(4)= -.88$, $p = .02$], (Appendix 3, Table 3.19). The Fear of Tissue Damage and Classic Phobias subscales of the FSS were also negatively correlated with the amplitude of the spontaneous EMG activation in the 'blind' trials [both correlations: $r(4)= -.82$, $p = .04$], (Appendix 3, Table 3.19). However, these correlations are based on small numbers of participants, therefore little can be inferred from them. The results listed above are summarized in Table 3.3.4.a.

Table 3.3.4.a Personality factors PPER amplitudes associations

Personality Factor	*Stimulus Type/Trial Type*	*Correlation*
Trait Anxiety	70dB/'Blind'	$r(4)= -.91, p = .01]$
Cognitive Disorganisation	85dB/Prepulse Alone	$r(19)= -.55, p = .01$
Novelty Seeking	80dB/Prepulse Alone	$r(12)= -.58, p = .03$
Reward Dependence	85dB/Prepulse Alone	$r(19)= .56, p = .01$
FSS Total Score	70dB/'Blind'	$r(4)= -.88, p = .02$
Interpersonal Fear	70dB/Pulse Alone	$r(7)= -.82, p = .02$
Interpersonal Fear	70dB/'Blind'	$r(6)= -.88, p = .02$
Fear of Tissue Damage	70dB/'Blind'	$r(6)= -.82, p = .04$
Classic Phobias	70dB/'Blind'	$r(4)= -.82, p = .04$

Prepulse reactivity onset

People scoring higher on the positive mood scale had faster onset of their spontaneous EMG activation preceding pulse presentation in pulse-alone trials [$r(7)= -.76$, $p = .02$], (Appendix 3, Table 3.20). Higher positive mood scores were also associated with faster onset of 90dB prepulse-elicited responses in prepulse and pulse trials $r(26)= -.38$, $p = .04$] (Appendix 3, Table 3.20). High scores on the BIS subscale of BIS/BAS were positively correlated with onset of spontaneous EMG activation in the 'blind' trials [$r(4)= .82$, $p = .05$], (Appendix 3, Table 3.20). High Introvertive Anhedonia scores were correlated with faster onsets of 80dB and 85dB prepulse-elicited responses [80dB, $r(17)= -.50, p = .03$; 85dB, $r(19)= -.45, p = .04$], (Appendix 3, Table 3.20). Reward Dependence was positively correlated with the onset of 80dB prepulse-elicited responses in prepulse and pulse trials [$r(12)= .63, p = .02$], 85dB prepulses in prepulse-alone trials [$r(19)= .62, p = .01$] and spontaneous EMG activation in 'blind' trials [$r(4)= .87, p = .02$], (Appendix 3, Table 3.20). The results are summarized in Table 3.3.4.b.

Table 3.3.4.b. Personality factors and PPER onsets associations

Personality Factor	*Stimulus Type/Trial Type*	*Correlation*
Positive Affect	70dB/Pulse Alone	$r(7)= -.76, p = .02$
Positive Affect	90dB/Prepulse and Pulse	$r(26)= -.38, p = .04$
BIS	70dB/'Blind'	$r(4)= .82, p = .05$
Introvertive Anhedonia	80dB/Prepulse Alone	$r(17)= -.50, p = .03$
Introvertive Anhedonia	85dB/Prepulse Alone	$r(19)= -.45, p = .04$
Reward Dependence	80dB/Prepulse and Pulse	$r(12)= .63, p = .02$
Reward Dependence	85dB/Prepusle Alone	$r(19)= .62, p = .01$
Reward Dependence	70dB/'Blind'	$r(4)= .87, p = .02$

Prepulse reactivity peak latency

Individuals with high Trait Anxiety scores had faster onset of the peak response amplitude (peak latency) for the weakest, 80dB prepulses [$r(12)= .75, p = .01$], and slower peak amplitude onset of the spontaneous EMG activity in the 'blind' trials [$r(4)= - .87, p = .02$], (Appendix 3, Table 3.21). High Negative Affect scores were negatively correlated with the peak latency of spontaneous EMG activation in the 'blind' trials [$r(4)= - .83, p = .04$] (Appendix 3, Table 3.21). There was a negative correlation between the Unusual Experiences score and the peak latency of spontaneous EMG activation in the 'blind' trials [$r(4)= - .92, p = .01$] (Appendix 3, Table 3.21). Scores on the Introvertive Anhedonia subscale were negatively correlated with peak latency of the responses to mid-intense, 85dB prepulses [$r(19)= - .52, p = .02$] (Appendix 3, Table 3.21). Another negative correlation appeared for Novelty Seeking and peak latency of the responses to the weakest, 80dB prepulses [$r(17)= - .47, p = .04$] (Appendix 3, Table 3.21). A positive association appeared for Harm Avoidance and peak latency of the responses to the weakest, 80dB prepulses [$r(12)= .60, p = .02$] (Appendix 3, Table 3.21). There were negative associations between the total score on the FSS and all the FSS subscales with the exception of Fear of Animals and peak latency of the spontaneous EMG activation in the 'blind' trials [correlations ranging from $r(4)= - .82, p = .04$ to $r(4)= - .90, p = .02$] (Appendix 3, Table 3.21). The results are summarized in Table 3.3.4.c

Table 3.3.4.c Personality factors and PPER peak latency associations

Personality Factor	*Stimulus Type/Trial Type*	*Correlation*
Trait Anxiety	80dB/Prepulse and Pulse	$r(12)= .75, p = .01$
Trait Anxiety	70dB/'Blind'	$r(4)= -.87, p = .02$
Negative Affect	70dB/'Blind'	$r(4)= -.83, p = .04$
Unusual Experiences	70dB/'Blind'	$r(4)= -.92, p = .01$
Introvertive Anhedonia	85dB/Prepulse Alone	$r(19)= -.52, p = .02$
Novelty Seeking	80dB/Prepulse Alone	$r(17)= -.47, p = .04$
Harm Avoidance	80dB/Prepulse and Pulse	$r(12)= .60, p = .02$
FSS Total Score	70dB/'Blind'	$r(6)= -.90, p = .02$
Interpersonal Fear	70dB/'Blind'	$r(6)= -.89, p = .02$
Fear of Tissue Damage	70dB/'Blind'	$r(6)= -.88, p = .02$
Fear of Noises	70dB/'Blind'	$r(6)= -.82, p = .04$
Classic Phobias	70dB/'Blind'	$r(6)= -.87, p = .03$

Prepulse reactivity onset to peak latency

The third temporal characteristic of the prepulse-elicited responses was the time lapse from the onset of the response to the peak amplitude time (onset to peak latency values). State Anxiety was positively correlated with spontaneous EMG activation in the prepulse part of pulse-alone trials [$r(6)= .72, p = .04$], yet the onset to peak latency values for the spontaneous EMG activation in the 'blind' trials were negatively correlated with Trait Anxiety [$r(4)= -.90, p = .01$], (Appendix 3, Table 3.22).

The Unusual Experiences subscale scores were negatively correlated with the onset-peak latency values for the spontaneous EMG activation in the 'blind' trials [$r(4)= -.84, p = .04$] (Appendix 3, Table 3.22). The onset to peak latency values for 85dB prepulse-elicited responses in prepulse and pulse trials were negatively correlated with two other O-LIFE subscales, Cognitive Disorganisation [$r(19)= -.49, p = .02$], and Introvertive Anhedonia [$r(19)= -.48, p = .03$] (Appendix 3, Table 3.22).

The onset-peak latency values of the spontaneous EMG activation in the 'blind' trials were negatively correlated with the total FSS score [$r(4)= -.89, p = .02$], Fear

of Tissue Damage [$r(4)= - .83, p = .04$], and Interpersonal Fear [$r(4)= - .95, p = .01$], (Appendix 3, Table 3.22). There was also a negative correlation for Fear of Animals and onset-peak latency values for 90dB prepulse-elicited responses in prepulse and pulse trials [$r(26)= - .40, p = .03$], (Appendix 3, Table 3.22). The results are summarized in Table 3.3.4.d.

Table 3.3.4.d Personality factors and PPER onset to peak latency associations

Personality Factor	*Stimulus Type/Trial Type*	*Correlation*
State Anxiety	70dB/Pulse Alone	$r(6)= .72, p = .04$
Trait Anxiety	70dB/'Blind'	$r(4)= - .90, p = .01$
Unusual Experiences	70dB/'Blind'	$r(4)= - .84, p = .04$
Cognitive Disorganisation	85dB/Prepulse and Pulse	$r(19)= - .49, p = .02$
Introvertive Anhedonia	85dB/Prepulse and Pulse	$r(19)= - .48, p = .03$
FSS Total Score	70dB/'Blind'	$r(4)= - .89, p = .02$
Interpersonal Fear	70dB/'Blind'	$r(4)= - .95, p = .01$
Fear of Tissue Damage	70dB/'Blind'	$r(4)= - .83, p = .04$
Fear of Animals	90dB/Prepulse and Pulse	$r(26)= - .40, p = .03$

Startle response modification probability

PPI

There was a weak negative correlation between the BIS subscale and probability of PPI following the presentation of 90dB prepulses [$r(37)= - .33, p = .04$], and a weak positive correlation between the Classic Phobias subscale of the FSS and probability of PPI following the presentation of these most intense, 90dB prepulses [$r(37)= .32, p = .01$], (Appendix 3, Table 3.21).

PPF

Paradoxical prepulse facilitation was least frequent in trials with 90dB prepulses and a number of personality factors were positively correlated with this type of response. These factors included State Anxiety [$r(38)= 37, p = .02$], Negative Affect [$r(38)= .44, p = .01$], BIS [$r(37)= .40, p = .01$], and Cognitive Disorganisation [$r(37)= .39, p = .01$], (Appendix 3, Table 3.21).

Startle response modification percentage change

There were no significant associations between the personality factors and startle response modification percentage change (Appendix 3, Table 3.22).

3.4 Discussion

In the past, prepulse-elicited responses were largely ignored in prepulse inhibition literature, even though it has been demonstrated that weak stimuli are capable of eliciting motor responses. The non-clinical participants providing data for this study exhibited a clear propensity towards quantifiable stimulus-driven prepulse-elicited responses with intensity dependent response characteristics.

Prepulse reactivity, prepulse-elicited response characteristics and associations with startle response modification

Nearly one third of the sample had measurable motor responses to the weakest 80dB prepulses and double that number of participants responded to the strongest 90dB prepulses. The mid-intense 85dB prepulses elicited motor responses in nearly half of the group. The 'responses' referred to here are numerous and systematic stimulus-driven responses following prepulse presentation. The probability of prepulse reactivity was intensity dependent, with higher intensity increasing the likelihood of a motor response which adds considerable weight to the validity of prepulse-elicited responses. The probabilities of prepulse-elicited responses were positively correlated across all the conditions indicating consistency in prepulse reactivity proneness. Prepulse stimuli characteristics had some effect on the elicited responses with intensity dependent probability, amplitude, onset (but only in prepulse alone trials) and peak latency, but not onset to peak latency values. All dimensions of prepulse reactivity: probability, amplitude, onset, peak latency and onset to peak latency, were positively correlated across the three prepulse intensities and six trial types. Although not each and every correlation reached statistical significance, they were all positive, indicating consistent associations amongst the response characteristics.

The probabilities of quantifiable motor responses were significantly higher following prepulse presentation as compared to spontaneous EMG in the absence of stimuli (exceeding the continuous background noise).This further lends good support to prepulse reactions The comparison of EMG activity in the temporal space of prepulse in 'pulse alone' and 'no stimulus' ('blind') trials demonstrated that the subsequent startle probe presentation (in pulse alone trials) did not increase the

spontaneous EMG activation somehow encroaching on the prepulse reactivity analysis window. The characteristics of prepulse-elicited responses, such as probability, amplitude, onset, peak latency and onset to peak latency, were not significantly different in prepulse-alone and prepulse and pulse trials, thus the critique of the subsequent pulse (startle probe) presentation somehow 'contaminating' the prepulse-elicited response is not valid – at least, not involving the parameters used in this experiment.

All participants exhibited prepulse inhibition. The traditional approach to deriving response means for startle response modification calculations is to collapse the data across the trial types (for each participant). This 'averaging' of the responses removes response details in each and every trial and replaces these with a single value used to calculate startle response modification. When each and every trial is treated separately (and the modified startle responses are individually subtracted from the baseline startle value) it becomes apparent that inhibition is not as universal a process (following the lead stimulus presentation) as is commonly assumed. Majority of the sample exhibited at least one example of increased, rather than decreased startle response in prepulse and pulse trials. This unexpected increase in startle responses, under the conditions designed to lead to maximal inhibition was dubbed 'paradoxical facilitation'. The increased responses had to meet the response validity criteria of surviving the visual inspection for the signs of corrupt EMG and reaching $z > 2$ with the scoring criteria held constant, that is being the same as for the inhibited startle responses.

Increasing prepulse intensity led to increased probability of inhibition and decreased probability of paradoxical facilitation. Whilst prepulse intensity affected inhibition percentage values (higher prepulse intensity = higher percentage inhibition), it had no effect on the percentage change of the paradoxical facilitation. Whereas percentage values of prepulse inhibition were positively correlated at different prepulse intensities, the percentage values of the paradoxical PPF exhibited no systematic relationships. Prepulse inhibition probability was inversely related to the paradoxical facilitation probability, but the percentage values of these two modes of startle response modification were not related to each other. However, high PPI values were inversely related to probability of the paradoxical PPF, indicating that efficient sensorimotor gating as indexed by PPI reduced the chances of the alternative startle response modification. The probability of PPI exhibited no

associations with the scope of paradoxical facilitation (i.e. the facilitation percentage change). Moreover the U-shaped distribution of PPI probabilities was mirrored in the inverted U-shaped distribution of PPF percentages, further strengthening the argument for the interdependence of classical inhibition and paradoxical facilitation.

Individuals likely to respond to the mid- and most intense prepulses (85dB and 90dB) had lowered probability of prepulse inhibition following the weakest, 80dB prepulse presentation. Prepulse inhibition is prepulse intensity dependent in terms of both probability and percentage and this association implies that prepulse reactivity is inversely related to prepulse inhibition in less then optimal conditions (i.e., low prepulse intensity equal elevated prepulse processing difficulty with decreased signal to noise ratio). At the same time, increased probability of responding to these same prepulses was associated with increased risk of paradoxical facilitation following the weakest lead stimuli presentation. Combining these two patterns of associations leads to the conclusion that increased prepulse reactivity is related to decreased probability of inhibition and increased risk of paradoxical facilitation. Bearing in mind that high PPI values were inversely related to the paradoxical PPF probability, it is tempting to conclude that increased prepulse reactivity is a hallmark of increased risk of paradoxical facilitation and less efficient sensorimotor gating (as indexed by PPI). The inverse relationship between prepulse reactivity and prepulse inhibition concurs with the findings of Dahmen and Corr (Dahmen & Corr, 2004).

Individuals more likely to respond to the mid- and most intense prepulses also had lower percentage inhibition values following the weakest prepulses. People likely to respond to the most intense prepulses had lower percentage inhibition values following the mid- intense 85dB prepulses. Thus prepulse reactivity is associated with not only probability, but also percentage values (i.e. efficiency) of prepulse inhibition. Increased prepulse reactivity to the weakest prepulses was positively related to increased percentage of paradoxical PPF in the 85dB prepulse condition and increased prepulse reactivity to the most intense prepulses was positively related to increased percentage of paradoxical PPF in the 80dB prepulse condition. Once again, prepulse reactivity is shown to be associated with increased risk of the alternative startle response modification under the conditions designed to elicit maximal inhibition.

Extended prepulse processing (longer duration of the motor response) was not associated with ether PPI or the paradoxical PPF in any way. The assumption that extended prepulse processing is somehow 'protected' from the subsequent startle probe interference does not seem to be supported by this finding. However, prepulse processing may not necessarily manifest at the level of motor response, therefore Graham's (Graham, 1975) original explanation of the nature of PPI still holds despite the lack of prepulse reactivity effect on the subsequent startle response modification.

Individual differences, prepulse reactivity and startle response modification

Prepulse reactivity

The propensity towards prepulse reactivity is best measured by the likelihood of prepulse-elicited responses. Personality factors exhibited scant associations with such propensity. Only the Novelty Seeking subscale of the TCI was positively correlated with prepulse reactivity probability to the mid-intense 85dB prepulses and Tissue Damage subscale of the FSS was negatively correlated with probability of prepulse reactivity to the weakest 80dB prepulses. These correlations were significant, but weak. The lack of systematic associations between personality factors and prepulse reactivity probability indicates that the stable tendency to either respond to the prepulses or not is not related to the concepts measured by the personality factors (such as for example dopaminergic activity). Pharmacological studies with rodents demonstrated mixed effects of dopamine agonists and antagonists on prepulse reactivity (see Chapter 1). In the context of these findings, the lack of systematic associations between the physiological measures and personality factors based on putative biological (neurochemical) substrates is not altogether surprising. Elevated dopaminergic activity does not reliably lead to increased prepulse reactivity, hence the subtle individual variation in dopaminergic activity as expressed by the relevant personality traits may not be influential enough to map onto prepulse reactivity.

Prepulse reactivity amplitudes exhibited far more frequent associations with personality factors. Response size, rather than frequency, seems to be more closely associated with the outcomes of the psychometric measures. Trait anxiety was negatively correlated with the size of very infrequent responses to non-existent prepulses in the 'blind' trials. With few participants displaying such responses, too much emphasis should not be placed on this association. The same caution is

relevant to the negative correlations between prepulse reactivity in the absence of prepulse stimuli in the 'blind' trials and FSS Total Score, Interpersonal Fear, Fear of Tissue Damage and Classic Phobias. The moderate negative correlations between Cognitive Disorganisation and prepulse reactivity elicited by 85dB prepulses is more meaningful since cognitive deficits are prominent in schizophrenia and recent study by Csomor and colleagues (Csomor, et al., 2009) demonstrated less prepulse reactivity in non-medicated schizophrenics and PPI deficits, but not a direct relationship between the two measures. The moderate negative correlation of Novelty Seeking and response size in the 80dB trials indicates that increased dopaminergic activity is associated with smaller size of prepulse-elicited responses. The reduced size of the responses in individuals high on NS concurs with lower incidence of prepulse reactivity in schizophrenics as found by Csomor (Csomor, et al., 2009) since dopaminergic hyperactivity is one of the aspects of schizophrenia and personality measures encompassing behaviours presumed to be initiated by dopaminergic activity (for example exploration) are often used as a proxy measure. What is more, prepulse processing difficulty needs to be elevated to demonstrate the differences between healthy and schizophrenic individuals (Blumenthal, et al., 2006) and reducing the signal to noise ration by presenting prepulses only mildly elevated above the background noise mimics the increased prepulse processing difficulty. The appearance of this association in the weakest prepulse condition concurs with the findings from both papers cited above. Reward Dependence, a trait underpinned by serotonergic activity, was positively correlated with the response size in response to 85dB prepulses.

In terms of the speed of response (onset) positive affect was associated with faster spontaneous EMG onset in the pulse alone trials (but for few participants) and more substantially with faster responses to 90dB prepulses. 90dB prepulses are near-startle threshold intensity stimuli and they reliably lead to frequent motor responses and strong PPI. High emotionality is presumed to lead to reduced PPI, whether it is negative or positive (Corr, et al., 2002). High BIS, active at times of behavioural conflict, was associated with slower onset of spontaneous EMG in the no stimulus trials. In the absence of stimuli above the background level, motor responses are wasteful and maladaptive. McNaughton and Corr's update of the Gray's BISBAS model (McNaughton & Corr, 2004) predicts increased BIS activity at times of conflict and hyper activation of motor responses in the absence of meaningful stimuli

is a situation of conflict, where the information delivered for further processing is obscured or unclear (stimuli yes, respond; or stimuli no, refrain from responding) against the continuous background noise. Introvertive Anhedonia, a trait akin to depression, was associated with faster onset of responses to the weakest and mid-intense prepulses. Yet again a negative association between high emotionality, in this case negative in flavour, and response to the stimuli relevant to sensorimotor gating, was demonstrated. Reward Dependence on the other hand was associated with slower onset of prepulse reactivity in the weakest and mid-intense prepulse condition and at the same time (in a few cases only) it was also positively correlated with spontaneous EMG onset in the no stimulus trials. With opposing directions of the associations for the depression-like Introvertive Anhedonia and serotonin based Reward Dependence (and extending the analogy behavioural avoidance/passivity versus behavioural maintenance) prepulse reactivity exhibits consistency.

High Trait Anxiety was associated with slower peak latency in prepulse and pulse trials, but only for the weakest, 80dB prepulses. Since anxiety is associated with attention directed inward, and not towards the external stimuli, it makes sense that stimuli not much above the background level would take a longer time to be detected, processed and responded to. Negative emotionality, in the form of high Trait Anxiety and high Negative Affect, was on the other hand associated with faster onset of spontaneous EMG peak amplitude values in the no stimulus 'blind' trials. The same was true for the Unusual Experiences subscale. High emotionality is associated with impaired sensorimotor gating and the Unusual Experiences subscale measures the propensity towards abnormal sensory experiences and unusual interpretations of the incoming sensory information leading to delusions and hallucinations in the most extreme degree. One of the problems encountered in schizophrenia is the patient's inability to distinguish the source of the experienced sensations (i.e. to distinguish internally from externally originating sensory event). The spontaneous EMG activity in the absence of any stimuli above the continuous background noise can be an indicator of overestimating the significance of a misperceived variation in the background noise or an internally generated sensory event which is misinterpreted as originating exogenously.

Interestingly Introvertive Anhedonia, a trait associated with passivity and behavioural withdrawal, was associated with faster peak latency for the mid-intense prepulses. Sensorimotor gating is impaired in depression and bipolar disorder and

the faster peak latency in the mid-intense condition, which is only 1dB below the optimal prepulse intensity of 16dB above the background noise, would indicate a sooner termination of the prepulse-elicited motor response (and thus prepulse processing). It is known that schizophrenics have lower rates of prepulse reactivity and impaired sensorimotor gating, but a direct relationship between the two has not been yet firmly demonstrated (Csomor, et al., 2009).

Novelty Seeking, a trait associated with activity and behavioural approach, was associated with faster peak latency to the weakest prepulses. The weakest prepulses represent the lowest signal to noise ratio condition in this paradigm and the faster peak latency implies faster stimuli detection. Increased dopaminergic activity is associated with increased exploratory behaviour, and it may be that minute changes in the environment are more likely to attract the attention of individuals actively exploring its different aspects.

High scores on the scales measuring fear were consistently associated with faster peak latency of the spontaneous EMG activation in the no stimulus trials. This can be indicative of increased vigilance in highly fearful participants and an over-sensitive startle system, which in a manner similar to the misperceived variation in the background noise warrants a maladaptive significance to a momentary change in the sound characteristics.

Harm Avoidance was associated with slower peak latency in response to the weakest prepulses. This delay in prepulse processing may reflect prolonged processing of the weak stimuli. Harm Avoidance and Trait Anxiety were the only two personality factors associated with slower peak latency in response to the weakest 80dB prepulses. Both are composed of items related to caution in approach behaviour, avoidance behaviour and negative emotionality. Perhaps stimuli significantly, but not excessively different from the stable environment (in terms of signal to noise ratio) are strong enough to start the motor response, but the response is either delayed or takes a longer time from the point of detection to the maximal motor response generation.

The response duration could be inferred from the onset to latency values, as prepulse reactivity EMG waves were symmetrical with the roll off following the peak not substantially different from the ascending trace preceding it. Highly anxious individuals (state anxiety) had longer time lapse from the onset of the response to the peak size in spontaneous EMG preceding startle probe presentation. Individuals with

high Trait Anxiety and those prone to Unusual Experiences, had shorter durations of spontaneous EMG activity (above their baseline EMG) in no stimulus 'blind' trials. Since these individuals also presented faster peak latencies in this condition, it appears that high scores on these two personality factors are associated with shorter duration of the responses to non-existent (above background noise) stimuli (i.e. the onsets are not different, but the peak latency is reached faster leading to shorter response duration). Therefore it is not faster detection that is responsible for the sooner peak latency in these people, but shorter response duration. Negative associations between the onset to peak latency values and two other subscales of the O-LIFE have also appeared for Cognitive Disorganisation and Introvertive Anhedonia, but these were detected in responses to the mid- intense prepulses in prepulse and pulse trials. Fear was yet again apparent in its associations with the temporal characteristics of spontaneous EMG in the no stimulus trials, but more importantly a moderate negative correlation appeared for the Fear of Animals subscale and the onset to peak latency values for the most intense 90dB prepulse-elicited responses in prepulse and pulse trials.

Despite the absence of significant differences in the responses derived from prepulse alone as compared to prepulse and pulse trials, the associations between personality factors and physiological measures often appeared in one or the other trial type only, indicating some effects of the different trial structures (startle probe presence).

PPI

A weak negative correlation appeared for the BIS subscale and probability of PPI following the presentation of the most intense 90dB prepulses. This finding concurs with Corr and colleagues (Corr, et al., 2002) reporting that high emotionality leads to PPI impairments although Ludewig and colleagues (S. Ludewig & Ludewig, 2003) reported lack of PPI deficits in unipolar depression. Surprisingly no significant associations appeared for anxiety (state or trait) and PPI. Ludewig and colleagues (S. Ludewig, et al., 2002) reported that within a group of individuals suffering from panic disorder, the high state and trait anxious participants had lower PPI and it would be expected that highly anxious individuals would process the weak lead stimuli less efficiently (due to attentional deficits, attention directed inwards towards self-monitoring) and therefore have lower PPI. However, processing of emotional stimuli (affective picture, intended to invoke the desired emotional state,

does not always disrupt orienting to intense, exogenous stimuli (Tipples & Sharma, 2000); therefore the state emotionality may have weaker impact on the startle response and its modification than expected.

PPI reliably appeared in the 90dB condition, the most intense prepulse presentation led to most frequent and strongest (highest percentage) PPI. A weak positive correlation between the Classic Phobias subscale of the FSS and probability of PPI following the presentation of these most intense, 90dB prepulses is a bit of a puzzle. The Classic Phobias subscale comprises items traditionally associated with clinically defined phobias, and it is possible that the very intense 90dB prepulse, close to intensity values used in some studies as startle probe, captures the individual's attention and processing resources if it mimics the phobic stimulus characteristics or some context associated with the appearance of the phobic stimulus.

PPF

Paradoxical prepulse facilitation was least frequent in trials with 90dB prepulses and a number of personality factors positively correlated with this type of response are factors associated with negative affectivity. These factors included State Anxiety, Negative Affect, BIS, and Cognitive Disorganisation. The first three factors measure experiences and information processing tendencies associated with negative affect and behavioural avoidance, with BIS activating at the time of behavioural conflict resolution, it is interesting that individuals high on negative affect, known to be detrimental to PPI as demonstrated by Corr and colleagues (Corr, et al., 2002) were consistently more likely to engage the alternative and paradoxical startle response modulation mechanism. PPF is deficient in schizophrenics (K. Ludewig, Geyer, et al., 2003; Wynn, et al., 2004) though some studies report lack of differences between schizophrenia patients and controls (Kumari, et al., 2004), but this effect is sex-dependent and appears only after the participants' age is taken into consideration; otherwise schizophrenics exhibit lowered PPF.. The association of the paradoxical PPF with high scores on Cognitive Disorganisation subscale, and previously reported associations of PPI with cognitive integrity (see Chapter 1) point towards the importance of investigating PPF at different lead intervals to ascertain its true nature, i.e. is it a sensorimotor gating mechanism completely different to PPI, or the same mechanism only operating in the opposite direction under specific circumstances.

Startle response modification percentage change

There were no significant associations between the personality factors and startle response modification percentage change. The activation of the sensorimotor gating mechanism, rather than its efficiency, appears to be related to some personality characteristics.

Effects of sex and age

There were no associations between PPER or startle response modification and sex or age. This absence of associations with SRM concurs with the findings by Ludewig and colleagues (K. Ludewig, Ludewig, et al., 2003) who reported no effect of age or sex on PPI or PPF. However, this finding of the lack of the effect of sex may be due to the lack of information on the females' menstrual or hormonal status. Females in the follicular phase have PPI values approaching the values normally exhibited by healthy males, but these reduce dramatically in the luteal part of the cycle. The PPI values in females are also affected by their age and the associated status in relation to the menopausal hormonal changes.

Summary

Prepulse-elicited responses are stimulus-driven and significantly different from spontaneous EMG. Several prepulse-elicited response characteristics are stimulus intensity dependent. The subsequent startle probe (pulse) presentation has no effect on PPER. PPER amplitude and temporal characteristics (but not probabilities) are most sensitive to associations with individual differences. Increased PPER is associated with decreased probability of PPI, as is high emotionality. Some individuals display paradoxical prepulse facilitation (PPF) under the conditions designed to elicit maximal PPI. High PPI values are associated with lower probability of the paradoxical PPF, but high emotionality is associated with increased risk of such startle response modification.

Limitations and future recommendations

The main limitation of the presented study was the unequal number of presentations of each trial type, the effect of which was subsequently investigated in the follow up study with a smaller sample. The small size and rarity of the prepulse-elicited responses stipulate a larger sample size, although the effect of prepulse

intensity on the elicited response can be seen with as few as fifteen participants (Csomor, et al., 2005).

PPI and baseline startle are stable, neurobiological characteristics (Cadenhead, et al., 1999), and it would be interesting to see if prepulse reactivity is also a stable neurobiological marker, both in males and females, even though there was no sex effect on prepulse reactivity in the reported data.

Investigating prepulse processing at the cortical level would make it feasible to find out if some participants have differential prepulse processing, yet fail to exhibit prepulse-elicited motor responses. The individuals who tend to exhibit the paradoxical PPF could also be compared to the majority engaging in the classical inhibition (following the prepulse presentation in prepulse and pulse trials). Two studies investigating the EEG changes associated with prepulse-elicited responses have been conducted, but are not be included in this thesis.

It would be useful to ascertain whether the paradoxical startle response modification would also appear under the conditions associated with maximal facilitation (i.e. would paradoxical inhibition, and not the expected facilitation consistently appear in some individuals).

3.5 Comparison study: increased sampling rate and corrected stimuli presentation procedure.

3.5.1 Rationale for the study

The follow up study was conducted to check if the paradoxical facilitation encountered in the first study was a real effect, or a peculiar characteristic of that specific sample, even though neither the sex ratio, nor demographic characteristics or personality profiles were unusual. What is more, the programming error in the session definition of the first study resulted in some trials being presented more frequently than others (as opposed to ten presentations of each stimulus type). The follow up study also offered possible corroboration of PPER prevalence in normal participants.

3.5.2 Materials and methods

3.5.2.1 Participants

Fifteen participants were recruited from university employees and postgraduate students (age range: 18-58 years, $M = 34$, $SD = 13$; 11 females, age range: 18-58, M

= 36, *SD* =14, 4 males, age range: 18-31, *M* = 25, *SD*= 5). There was no payment for the participation.

Exclusion criteria comprised self-declared suboptimal hearing, tinnitus, drug abuse or psychiatric disorder history. The times of last nicotine and alcohol consumption were recorded and hearing acuity was tested at 40dB. Three participants responded in less than seven pulse alone trials (ten such stimuli presented in the main session) and were excluded as non-responders. Three smokers were excluded from further analysis since smoking affects prepulse inhibition of the acoustic startle response (PPI). The final sample size was thus reduced to nine participants (age range: 18-58, *M* = 36, *SD* = 15; 7 females, age range: 19-58, *M* = 41, *SD* = 15, 2 males, age range: 18-25, *M* = 21, *SD* = 5).

3.5.2.2 Materials

The same materials were used as in the main study

3.5.2.3 Design

The design of this study was identical to the main study, with the correction of each trial type being presented ten times.

3.5.2.4 Physiological data collection

The physiological data collection was the same as in the main study with the exception of the sampling rate increase to 1000Hz and the filter settings to 100Hz high pass and 1000Hz low pass. The recording window was increased to 1000ms.

3.5.2.5 Data scoring and statistical analysis

The data were treated in the same way as for the main study. The main points for comparison were: prepulse-elicited response probability and startle response modification (PPI and PPF) probability and percentage change. Only the probability of the prepulse-elicited responses had impact on the subsequent startle response modification and hence only the prepulse-elicited response probability was entered as the comparison factor in the analysis. The sample was too small to derive any meaningful conclusions about the individual differences and physiological responses relationships; the questionnaire data were used solely to ensure that the comparison sample did not have characteristics dramatically different from the main sample.

3.5.2.6 Results

3.5.2.7 Personality characteristics comparison

The comparison sample and the original sample were largely similar in their personality characteristics. The comparison of the questionnaire scores for both groups is presented in Appendix 3, Table 3.23. The scores on the FSS questionnaire were higher in the comparison group and when all the questionnaire scores were compared a significant difference between the two samples emerged [t (25) = -2.41, p =.02]. Removal of the FSS questionnaire scores from the analysis revealed lack of a significant difference between the two samples in the other questionnaire scores [t (19) = -1.68, ns].

3.5.2.8 EMG responses comparison

The comparison sample was different in two aspects: a larger proportion of the comparison sample exhibited prepulse-elicited responses [t(2) = -.5.10, p = .04] and a larger number of individuals exhibited the paradoxical PPF [t(2) = -.5.07, p = .04], (Appendix 3, Table 3.24). All participants in both samples exhibited PPI.

Prepulse-elicited response probability was similar in both groups [t(2) = -.1.11, ns]. Probability values were significantly different for PPI [t(2) = -.7.34, p = .02], but not for PPF [t(2) = 2.77, ns]. Startle response modification probabilities followed the rules of increase (PPI) or decrease (PPF) with increasing prepulse intensity (Appendix 3, Table 3.25).

Both PPI [t(2) = - 3.81, ns] and paradoxical PPF [t(2) = - 2.46, ns] percentage change values were higher in the comparison sample, but the differences were not statistically significant. The percentage change of the startle response modification in PPI increased with increasing prepulse intensity. A small, but systematic PPF percentage change values decrease occurred with increasing prepulse intensity in the main sample, whilst more chaotic value distribution appeared for the percentage change of the paradoxical PPF in the comparison sample (Appendix 3, Table 3.26).

3.6 Discussion

The follow up study demonstrated that prepulse reactivity can be detected even in the small sample size used here. The paradoxical prepulse facilitation appeared in this group as well, indicating that it was a real phenomenon and not a consequence of some unusual sample characteristics of the first study. A larger proportion of the comparison sample exhibited prepulse reactivity, but the prepulse-elicited response probability was similar in the two groups, and it was the probability (rather than any other characteristic) that was associated with the effectiveness of startle response modification (percentage inhibition or facilitation).

All participants in each sample exhibited PPI, the comparison sample showing larger, but not significantly different, percentage PPI than the main sample. The probability of PPI (as opposed to PPF or lack of startle response modification) was significantly higher in the comparison sample. Fewer individuals in the main sample (the first study) exhibited the paradoxical PPF, but its overall probability was higher (though not significantly higher), which indicates that the same individuals would frequently exhibit the paradoxical PPF. Neither probabilities, nor percentage changes were significantly different for the paradoxical PPF between the two samples.

The differences could be attributed to the increased sampling rate, which resulted in a more faithful representation of the EMG waveform, and hence more accurate response values derived from its decomposition. Such increased response representation fidelity may have led to increased sensitivity to the response nuances. The second explanation could be sample characteristics: the comparison sample was predominantly female and on average older than the original sample. Age may have an effect on prepulse reactivity, although such an effect did not appear in the analysis of the first, much larger sample. The comparison sample had significantly higher FSS scores; therefore the increased prepulse reactivity could be attributed to increased defensive response (startle response) activation in highly fearful participants, although no such correlation appeared in the main sample.

The origins of the differences in the responses of these two samples can not be securely attributed to procedural differences. Despite occasional differences, similar linear trends in the prepulse-elicited response probability and startle response modification characteristics were identifiable in both groups. It appears that the procedural error resulting in unequal number of presentations of each trial type did not have a significant impact on the measured variables.

4 Perceived stimulus intensity: a reliable replacement for the EMG recording?

4.1 Introduction

The usual measure of sensorimotor gating is EMG recording of stimulus-driven motor response. The motor response alone is not representative of gating ability deficits phenomenology (i.e. what it 'feels like' to experience sensory overloading) and conscious stimulus perception complements EMG recording as sensorimotor gating function measurement. Filtered-out stimuli are not admitted for further processing, thus consciously perceived stimuli are those that make it past the initial sensory gating. Conscious perception of the stimulus characteristics can be posited to reside between sensory filtering (gating) and the motor response elicited by the processed stimulus.

Swerdlow and colleagues (N. R. Swerdlow, N. Stephany, et al., 2002; N. R. Swerdlow, et al., 2005) proposed measuring the perceived stimulus intensity as a robust replacement for the EMG recording of startle response and startle response modification. The perceived stimuli intensity (PSI) is subject to inhibition following prepulse presentation (prepulse inhibition) resulting in prepulse inhibition of perceived stimuli intensity (PPIPSI). There are marked differences between the inhibition of perceived stimulus intensity and the motor response inhibition following the lead stimulus presentation though. Whereas PPI can reach the values of 100% inhibition (no startle response in prepulse and pulse trials), values of 15% are reported as the ceiling value for perceived stimuli intensity inhibition (PPIPSI).

A lack of prepulse detection impact on PPI has been reported previously (Postma, et al., 2001) and the study presented below extends the possible associations between prepulse processing and the subsequent startle response modification beyond the lead stimuli detection alone. Prepulse detection task is a simple 'yes' or 'no' classification, but the rating of stimuli intensity requires more attentional resources directed at the stimuli. If some individuals perceive the lead stimuli as more intense, then these individuals may be simply more sensitive to the acoustic stimuli (lower hearing threshold), or they may process the prepulse more (by, for example, paying more attention to the weak stimulus). Increased

intensity perception could be associated with increased detection rates; on the other hand, detection could a process completely separate from intensity rating. The consequences of increased or decreased prepulse intensity perception for the subsequent startle response modification are unknown.

Swerdlow and colleagues (Swerdlow, Stephany et al. 2005; Swerdlow, Blumenthal et al. 2007) demonstrated that intensity ratings adequately reflect startle probe loudness in prepulse and pulse trials. They have not tested the reliability of perceived stimulus intensity ratings for the weaker prepulse stimuli. If the weak lead stimuli are faithfully reflected in the intensity ratings, than the stimulus intensity effects should appear for the prepulse intensity ratings. The paradoxical prepulse facilitation, encountered in Chapter 3, should also appear in the stimuli intensity ratings.

Attention is known to affect startle response modification. If the stimulus intensity judgment activity enhances the attentional processing, than the startle response modification should be enhanced as the consequence of the intensity rating activity. Significant differences in the EMG signal in the rating part of the session would indicate either the rating activity contamination (for example, abnormal EMG waveforms leading to lowered valid response frequencies) or the attentional effects.

Swerdlow and colleagues (Swerdlow, Stephany et al. 2002) claims that perceived stimulus intensity is a robust measure, largely insensitive to individual differences. The authors reported lack of significant associations with novelty seeking, reward dependence or harm avoidance, and lack of effects of sex or menstrual cyclicity (N. R. Swerdlow, N. Stephany, et al., 2002; N. R. Swerdlow, et al., 2005). If that assertion is true, then personality factors relevant to sensorimotor gating, sex, or age should exhibit no significant associations with the perceived stimulus intensity.

The main aims of the study presented in this chapter centre on the relationships between conscious stimuli processing and parallel physiological responses. The possible consequences of the conscious stimuli processing for startle response modification are also investigated. The associations between individual differences and conscious stimuli processing (for example, intensity judgment biases) are also probed.

The following hypotheses will be tested in this study:

1. Prepulse stimuli ratings adequately reflect the stimuli intensity

2. Conscious stimuli perception is subject to the same laws of modification as the physiological responses

3. Conscious prepulse processing, both simple detection and intensity ratings, impacts on startle response modification

4. Individual differences exhibit associations with conscious stimuli processing

4.2 Methods and materials

4.2.1 Participants

Thirty three participants were recruited from university employees and postgraduate students (age range: 18-55 years, $M = 23.00$, $SD = 7.43$; 24 females, age range: 18-55, $M = 23.29$, $SD = 8.54$, 9 males, age range: 19-29, $M = 22.22$, $SD = 3.23$). Participants had a chance to win £100.00 in return for taking part in the study.

Exclusion criteria comprised self-declared suboptimal hearing, tinnitus, drug abuse, psychiatric disorder history, smoking, head injury or any neurological disorder. The times of last nicotine and alcohol consumption were recorded to ensure only non-smokers would take part. The medication use and menstrual cycle (where applicable) were recorded on the demographic questionnaire. The hearing acuity was tested at 40dB. Six participants responded in less than three pulse alone trials (five such stimuli presented in the main session) and were excluded as non-responders. Two participants were excluded on the basis of abnormal intensity ratings. One of these participants detected only two pulse alone stimuli and rated the weak prepulses as highest intensity stimuli and consistently marked two lines in single stimulus trials. The other one rated all stimuli as very weak and exhibited unusual startle response modification patterns at all prepulse intensities (pulse-elicited responses delayed beyond the scoring criteria cut off point).

The final sample size was thus reduced to 25 participants (age range: 18-55, $M = 22.27$, $SD = 6.95$; 18 femalse, age range: 18-55, $M = 22.53$, $SD = 8.08$, 7 males, age range: 19-29, $M = 21.57$, $SD = 2.23$).

4.2.2 Materials

The same materials were used as in the first study presented in Chapter 3. Intensity judgments were recorded on 10cm visual analogue scale (VAS). A separate sheet was used for each trial with two lines to enable recording of both prepulse detection and intensity judgments.

4.2.3 Physiological data collection

Four types of white noise stimuli with almost instantaneous (less than 1 ms) rise time were used: pulse (115dB; 40ms duration) and three discrete prepulses (80, 85 and 90dB; 20ms duration). The stimuli were presented binaurally via headphones over 70dB continuous white noise background.

The stimuli were combined into seven different trial types: pulse-alone trial (70dB, 115dB), three prepulse and pulse trials (80dB, 115dB; 85dB, 115dB; 90dB, 115dB) and three prepulse-alone trials (80dB, 70dB; 85dB, 70dB; 90dB, 70dB) Lead (inter-stimulus) interval of 140 ms was used in all trials. The inter trial interval ranged from 9-21s (mean = 14s). Trials were presented in fixed, pseudo-random order with five presentations of each trial type. The same seven trial types were used in both parts of the session which lasted approximately 30 minutes.

The details of the EMG recording are described in Chapter 2. The EMG activity was recorded continuously (sampling rate 1000Hz) for 1000ms starting at the trial onset. EMG data were high (100Hz) and low (1000Hz) band pass filtered. The session consisted of two parts: comparison EMG activity recording part and intensity rating part. The comparison EMG part of the session was presented first. The session begun with 2 minutes of acclimation (to the background noise) followed by six presentations of pulse-alone trial and then all the other trial types were presented in a fixed, pseudorandom order (see above). Six more pulse-alone stimuli were presented at the end of this part. The intensity judgment part followed. One minute of the background noise was presented first to demonstrate the far left end of the scale. Three pulse-alone stimuli were then presented to demonstrate the far right end of the scale. The participants marked one or both of the two lines (100mm) on the sheet provided for each trial. Each stimulus type was presented five times (thirty five trials presented in this part).The inter-trial

intervals were variable, as the next trial was not presented until the participant recorded their judgment.

The scoring parameters were held constant and set to: baseline 20ms, response window 100ms, rolling average 3, Butterworth digital filter order 3 (pass frequency 333), response criterion 2. The only parameters changed for scoring prepulse or pulse part of trial responses were the start (21ms for prepulse-elicited response, 160ms for pulse-elicited response) and the end (160ms for prepulse-elicited response, 270ms for pulse-elicited response) of the analysis. Prepulse-elicited and pulse-elicited responses were analyzed separately for each trial.

4.2.4 Design

Participants completed the self-report questionnaires and were exposed to all the stimuli (with 5 presentations of each stimulus type in each part of the session). A mixed design was used, with sex and personality measures as the between subjects factors and the eyeblink-eliciting stimuli driven responses (EMG in both parts of the session and intensity judgments) as the within-participant factors.

4.2.5 Data Scoring and Statistical Analysis

The EMG data were treated in the same way as for the first study (Chapter 3). The intensity judgment values represented the location of the marked point on the 100mm VAS (distance from the far left end which represented the background noise loudness). Modification of the perceived stimulus intensity was calculated using the formula given by Swerdlow et al (2002, 2005) and was the same as the startle response modification (SRM) formula given in Chapter 2, with trial intensity ratings replacing the amplitude values. The product of this formula is the reversal of intensity ratings relationship seen in 'raw' data, since the least different stimuli will be rated similarly and hence the difference (or percentage change) from the pulse-alone stimulus ratings will be low. The 'range-correction' formula was redundant for investigating the perceived stimulus intensity of the weak prepulses since pure reversal of the relationships found in the data would not have enhanced the findings.

Repeated measures ANOVA was used to ascertain the effects of prepulse intensity and sex on prepulse reactivity, startle response modification, and perceived stimulus intensity ratings.

4.3 Results

4.3.1 Intensity ratings reliability in representing stimuli intensity

4.3.1.1 Prepulse detection

Prepulse detection rates increased with increasing prepulse intensity in prepulse and pulse trials, and detection rates in prepulse-alone trials remained stable across all intensities (see Appendix4, Figure 4.1).

Even the weakest prepulses were detected by half of the sample and the detection rates increased with increasing prepulse intensity. Fifty two percent of the sample detected 80dB prepulses and fifty six percent of the sample detected 85dB and 90dB prepulses. The average detection rates increased from thirty eight percent for the 80dB prepulses to forty three percent for the 85dB and 90dB prepulses in prepulse and pulse trials. All participants detected all the prepulses in prepulse alone trials (hundred percent of the sample had hundred percent prepulse detection rate). There were no erroneous ratings for prepulses in pulse-alone trials. Table 4.1 in Appendix 4 shows the percentage of the sample who detected the prepulses at 80dB, 85dB, and 90dB and prepulse detection probability in all conditions.

Prepulse detection rates were significantly higher in prepulse-alone than in prepulse and pulse trials for prepulses of 80dB [$t(24) = -7.36$, $p < .01$], 85dB [$t(24) = -6.76$, $p < .01$], and 90dB [$t(24) = -5.80$, $p < .01$] (Appendix 4, Table 4.2). Prepulse intensity effects were not significant in prepulse-alone trials. There were no significant effects of prepulse intensity [$F(2, 25) = 1.93$, ns] or sex [$F(2, 25) = .03$, ns] on the detection rates in prepulse and pulse trials. All prepulses at all intensities were detected in prepulse-alone trials.

There were strong positive correlations between prepulse detection rates at all prepulse intensities ranging from $r(23) = .92$, $p < .01$ to $r(23) = .98$, $p < .01$ ((Appendix 4, Table 4.3). Prepulse detection rates correlations in prepulse and pulse trials only were calculated as all the participants had 100% prepulse detection rate in prepulse-alone trials.

4.3.1.2 Prepulse intensity judgments

Increased prepulse intensity ratings reflected the increased stimuli intensity (see Appendix 4, Figure 4.2 and Table 4.4) with a clear linear trend present for the intensity ratings of prepulses presented alone. Prepulse intensity ratings ranged from

just over eighteen millimeters (out of 100) to nearly twenty four millimeters in prepulse and pulse trials, and from nearly four to over eight millimeters in prepulse alone trials.

However, paired-samples t-test revealed lack of significant differences in the prepulse intensity judgments in prepulse and pulse as compared to prepulse-alone trials (Appendix 4, Table 4.5), although prepulses in prepulse and pulse trials were rated as louder than the prepulses in prepulse-alone trials at all prepulse intensities. The direction of the possible differences was not assumed, since the prepulse intensity ratings could have been prone to loudness assimilation effect in prepulse and pulse trials, yet prepulses presented alone could have been more prominent against the stable background noise.

Prepulse intensity effect appeared both in prepulse and pulse trials [F (2, 25) = 3.94, p = .03] and in prepulse-alone conditions [F (2, 25) = 21.01, p < .001]. Sex did not have a significant effect on the intensity ratings in prepulse and pulse trials [F (2, 25) = .24, ns], but in prepulse-alone trials the interaction of sex and prepulse intensity was significant [F (2, 25) = 4.76, p = .01].

Prepulse intensity judgments were highly correlated across all prepulse intensities in prepulse and pulse trials (correlations ranging from r(12) = .93, p <.01 to r(11) = .97, p = <.01), but the correlations in prepulse-alone trials were less systematic with only two adjacent prepulse intensities exhibiting significant correlations [80dB and 85dB, r(23) = .41, p <.01; 85dB and 90dB r(23) = .77, p <.01] (see Appendix 4, Table 4.6).

4.3.1.3 Prepulse detection and prepulse intensity judgments

Prepulse detection was associated with lower intensity ratings for most prepulses in prepulse and pulse trials [correlations between prepulse detection and prepulse intensity ratings for ranged from r(12) = -.53, p = .05 to r(11) = -.80, p = .01]. Higher prepulse detection rates were associated with higher intensity ratings for 85dB prepulses presented alone [at 80dB, r(23) = .46, p = .02; 85dB, r(23) = 42, p = .04; 90dB, r(23) = .42, p = .03] (see Appendix 4, Table 4.7).

4.3.2 Startle probe (pulse) intensity judgments

4.3.2.1 Startle probe detection

Pulse stimuli were detected by all the participants in pulse-alone and prepulse and pulse trials and the detection rates were nearing one hundred percent in all trial types (Appendix 4, Table 4.8).

4.3.2.2 Startle probe intensity judgments

The intensity ratings for the startle probes in prepulse and pulse trials reflected reduction in relation to the ratings of pulse-alone stimulus (see Appendix 4, Figure 4.3).

The intensity judgments for startle probes presented alone were always higher than startle probe intensity judgments in prepulse and pulse trials. These differences were significant for pulse intensity ratings in 85dB [$t(24) = 2.29$, $p = .03$] and 90dB [$t(24) = 2.39$, $p = .02$] prepulse and pulse trials (Appendix 4, Table 4.9). There was no significant effect of prepulse intensity [F (2, 25) = .78, ns] or sex [F (2, 25) = 1.38, ns]. Converting the raw pulse intensity ratings into percentage differences from the pulse-alone ratings for each participant ('range correction', Swerdlow et al. 2005) did not change the comparison outcome [F (2, 25) = .70, ns; sex F (2, 25) = 1.28, ns].

Fifty six percent (14 out of 25) of the sample judged the pulses in 80dB prepulse and pulse trials as more intense than pulse stimuli presented alone. The same was true for thirty six percent (9 out of 25) at 85dB prepulse intensity and forty percent (10 out of 25) at 90dB prepulse intensity. These figures reflect individuals who responded in this manner in three or more trials (out of the total five) in each trial type. If the frequency benchmark was lowered to one, then ninety two to ninety six percent of the participants provided increased pulse intensity ratings in prepulse and pulse trials at least once (24 at 80dB, 23 at 85dB, 24 at 90dB).

4.3.2.3 Prepulse detection and perceived stimulus intensity ratings modification

Increased detection of 80dB prepulses was associated with increased startle probe intensity ratings in these trials [$r(22) = .45$, $p = .03$]. Moderate positive correlations emerged for perceived pulse intensity facilitation in 90dB prepulse and pulse trials and prepulse detection at all prepulse intensities [80dB, $r(22) = .46$, $p = .02$; 85dB, $r(22) = .44$, $p = .03$; 90dB, $r(22) = .41$, $p = .05$] (Appendix 4, Table 4.10).

Increased detection of 80dB prepulses was associated with decreased probability of increased startle probe ratings following the weakest lead stimulus presentation [$r(23) = -.43$, $p = .03$]. No other associations emerged for prepulse detection and intensity ratings modification probabilities (Appendix 4, Table 4.11).

4.3.3 Personality and demographic factors

4.3.3.1. Personality factors and prepulse detection and intensity ratings

There were not many significant correlations between personality factors and either prepulse detection or intensity ratings (Appendix 4, Table 4.12). A moderate negative correlation emerged for the Unusual Experiences subscale of O-LIFE and 85dB prepulse detection [$r(23) = -.41$, $p = .04$]. A moderate positive correlation was present for the Reward Dependence subscale of TCI and 80dB prepulse detection [$r(23) = .43$, $p = .03$]. Moderate negative correlations were present between prepulse detection rates at all intensities and the Self-Transcendence subscale of the TCI [80dB, $r(23) = -.41$, $p = .04$; 85dB, $r(23) = -.52$, $p = .01$; 90dB, $r(23) = -.51$, $p = .01$].

In terms of intensity ratings the O-LIFE Introvertive Anhedonia subscale was positively correlated with 90dB prepulse intensity ratings in prepulse and pulse trials [$r(12) = .55$, $p = .04$]. Strong negative correlations emerged for the Reward Dependence TCI subscale and intensity ratings for 80dB [$r(11) = -.76$, $p = .01$] and 85dB [$r(12) = -.63$, $p = .02$] prepulses in prepulse and pulse trials. Strong negative correlations emerged for the Cooperativeness TCI subscale and prepulse intensity ratings in all prepulse and pulse trials [80dB, $r(11) = -.88$, $p < .01$; 85dB, $r(12) = -.87$, $p < .01$; 90dB, $r(12) = -.85$, $p < .01$]. A positive correlation appeared for the Fear of Noises subscale of FSS and intensity ratings of 85dB prepulses in prepulse and pulse trials [$r(12) = .58$, $p = .03$] (Appendix 4, Table 4.12).

4.3.3.2 Personality factors and perceived stimulus intensity ratings modification

Negative Affect was positively correlated with percentage PPIPSI at all prepulse intensities [80dB, $r(22) = .41$, $p = .05$; 85dB, $r(23) = .44$, $p = .03$; 90dB, $r(22) = .44$, $p = .03$] and with PPFPSI in 90dB [$r(23) = .44$, $p = .03$] prepulse and pulse trials. The Positive Affect subscale of PANAS was negatively correlated with pulse intensity ratings increase (PPFPSI) in 80dB [$r(21) = -.50$, $p = .02$] prepulse and pulse trials. Other personality factors were rarely associated with any intensity ratings modification. The TCI subscale of Reward Dependence was significantly

correlated with PPIPSI in 90dB [$r(22) = .64$, $p = .01$] prepulse and pulse trials and PPFPSI in 80dB [$r(21) = .44$, $p = .03$] prepulse and pulse trials. The Fear of Animals subscale of FSS was positively correlated with PPIPSI in 90dB [$r(22) = .45$, $p = .03$] prepulse and pulse trials (Appendix 4, Table 4.13).

The probabilities of the perceived stimulus intensity ratings modification were unrelated to the personality characteristics except for the Cooperativeness subscale and probability of the perceived stimulus intensity inhibition in trials with 90dB prepulses [$r(22) = .42$, $p = .04$], (Appendix 4, Table 4.14).

4.3.3.3 Demographic factors (sex and age) and prepulse detection and intensity ratings

There were no significant correlations between sex or age and prepulse detection or perceived prepulse intensity ratings (Appendix 4, Table 4.15)

4.3.3.4 Demographic factors and perceived stimulus intensity modification

Only age was significantly negatively correlated with the perceived stimulus intensity facilitation percentage change in 85dB prepulse and pulse trials (Appendix 4, Table 4.16). Older participants, displaying perceived pulse stimulus intensity ratings increase (PPFPSI) did so to a lesser degree in 85db prepulse and pulse trials [$r(21) = -.52$, $p = .01$].

There were no significant correlations between the demographic factors and the probability of one type of PSI modification versus the other (PPIPSI or PPFPSI) (Appendix 4, Table 4.17).

4.4 Data derived from the EMG recordings

The EMG recordings were compared across the two parts of the session to gauge the levels of habituation and to check if the intensity judgment activity contaminated the EMG signal in the second part of the session. If only habituation was suspected, then the direction of the change would be clear (decrease of the response amplitude and frequency with time), but the issue of the potential contamination of the EMG signal by the stimulus intensity rating activity dictated adopting non-directional approach to the possible changes in the EMG responses. Large differences between the responses recorded in the two session parts would have indicated an effect of the directed attention.

4.4.1 Prepulse reactivity

The comparison of prepulse-elicited response amplitudes in the two parts of the session (baseline EMG recording and intensity rating part) has just reached statistical significance for 90dB prepulse-elicited responses [$t(9) = 2.25$, $p = .05$] in prepulse and pulse trials. Other prepulse-elicited responses were not significantly different between the two parts of the session. Startle response amplitude was higher in the first part of the session, but the comparison failed to reach significance (unless one assumed habituation only and no other effect of the paradigm). This lack of significant differences is indicative of limited response amplitude habituation for either startle responses or prepulse-elicited responses (Appendix 4, Table 4.18).

Statistically significant differences for prepulse-elicited response probabilities (Appendix 4, Table 4.19) appeared for 85dB [$t(24) = 3.05$. $p = .01$] and 90dB [$t(24) = 2.19$. $p = .04$] prepulses in prepulse and pulse trials. The baseline EMG part of the session (presented first) was marked by higher probabilities of 85dB and 90dB prepulse-elicited responses in prepulse and pulse trials. No significant difference was observed in the proneness to a motor response in the absence of any prepulse stimulus in pulse-alone trials. The frequency of the startle responses was reduced, if one assumed a change in one direction only (habituation, no contamination by the rating activity).

4.4.2 Startle response modification

The percentage changes of prepulse inhibition (PPI) and prepulse facilitation (PPF) remained stable across the baseline EMG recording and intensity judgment parts of the session with no statistically significant differences in the values present in both parts (see Appendix 4, Table 4.20).

The probabilities of either prepulse inhibition (PPI) or prepulse facilitation (PPF) have not exhibited statistically significant differences across the two parts of the session either (Appendix 4, Table 4.21).

4.4.3 Motor responses and intensity ratings relationships

Correlations were calculated for prepulse detection rates, intensity ratings and prepulse-elicited amplitudes and probabilities to ascertain if people more likely to detect prepulses would rate them as more intense, and if their EMG responses (size and probability) would reflect the higher detection rates or increased stimulus intensity ratings. The prepulse-elicited response amplitudes and probabilities are taken from the baseline EMG part of the session and from the EMG recorded concurrently with the intensity rating activity in the intensity judgment part of the session.

4.4.3.1 Prepulses: relationships between the detection rates, prepulse intensity ratings and prepulse-elicited motor response

Individuals with larger motor responses to 90dB prepulses in the first part of the session (baseline EMG recording) were more likely to detect prepulses at all three intensities [80dB, $r(12) = .68$, $p = .01$; 85dB, $r(12) = .63$, $p = .01$; 90dB, $r(12) = .55$, $p = .04$]. The few individuals who exhibited motor responses to 85dB prepulses in the intensity judgment part of the session were more likely to detect prepulses at all intensities [80dB, $r(3) = .92$, $p = .03$; 85dB, $r(3) = .92$, $p = .03$; 90dB, $r(3) = .92$, $p = .03$].

There was a significant negative correlation between 80dB prepulse intensity ratings in prepulse and pulse trials and the amplitude of 80dB prepulse-elicited responses in prepulse and pulse trials in the baseline EMG recording part of the session, but not in the intensity judgment part [$r(2) = -.97$, $p = .03$]. Another significant negative correlation was also present for the 80dB prepulse intensity ratings in prepulse and pulse trials and 90dB prepulse-elicited responses in prepulse-alone trials in the first part (baseline EMG recording) part of the session [$r(3) = -.95$, $p = .01$], but not in the second (intensity judgment) part.

All the correlations listed above are in Appendix 4, Table 4.22.

Prepulse detection or intensity ratings exhibited very limited correlation with prepulse-elicited response probability in either part of the session (Appendix 4, Table 4.23). Only 80dB prepulse-elicited response probability in prepulse-alone trials in the judgment part of the session and intensity ratings of 80dB prepulses presented alone

were positively correlated. Individuals rating the weakest prepulses as more intense were more likely to exhibit 80dB prepulse-elicited responses in the second part of the session [$r(23) = .52, p = .01$].

4.4.3.2 Prepulse detection and startle response modification

There were no significant correlations between prepulse detection rates and percentage startle response modification percentage in the baseline EMG recording part of the session (Appendix 4, Table 4.24) and no significant correlations between prepulse detection rates and percentage startle response change in the intensity judgment part of the session either (Appendix 4, Table 4.25).

There were no significant correlations between prepulse detection and probabilities of startle response modification in the baseline EMG recording part (Appendix 4, Table 4.26) or intensity judgment part (Appendix 4, Table 4.27) of the session. [However, if the startle response modification was calculated using the traditional 'collapsing across trial types' approach then significant negative correlations appeared for 80dB prepulse detection rates and startle response modification probabilities following the presentation of 80dB [$r(23) = -.44, p = .03$] and 85dB [$r(23) = -.46, p = .02$] prepulses. Prepulse detection rates for 85dB prepulses and 90dB prepulses were negatively correlated with startle response modification probabilities following the presentation of 90dB prepulses [85dB, $r(23) = -.43, p = .03$; 90dB, $r(23) = -.43, p = .03$].

4.4.3.3 Perceived stimulus intensity modification and startle response modification comparisons: probabilities and percentage changes

The probabilities of either decreased (PPIPSI) or increased (PPFPSI) perceived stimulus intensity ratings (probabilities and percentage change), were correlated with startle response probability and percentage modification in the comparison EMG recording and intensity judgments (IN) parts of the session. The formula for calculating the intensity ratings modification was the same as the formula for calculating the startle response modification given in Chapter 2. 'Stimulus' in this context meant the 'pulse' in the prepulse and pulse trials.

Increased probability of facilitated perceived stimulus intensity ratings in 90dB prepulse and pulse trials was associated with increases in the EMG responses in 80dB prepulse and pulse trials (PPF percentage change) in the baseline EMG part

of the session [$r(6) = .71$, $p = .05$]. Increased probability of facilitated perceived stimulus intensity ratings in 80dB prepulse and pulse trials was positively correlated with reduction (PPI) in the EMG responses in the same part of the session [$r(22) = .42$, $p = .04$] and also in the intensity judgment part [$r(20) = .47$, $p = .03$]. The probability of inhibited perceived stimulus intensity ratings in 80dB prepulse and pulse trials was negatively correlated with reduction (PPI) of the EMG responses in 80dB prepulse and pulse trials, but only the in the intensity judgment part of the session [$r(20) = -.45$, $p = .02$].

Probability of inhibited perceived stimulus ratings in 90dB prepulse and pulse trials was positively correlated with the probability of overall startle response modification (before subdividing into inhibition and facilitation) [$r(23) = .48$, $p = .02$], however, the probability of increased stimulus intensity ratings in 90dB prepulse and pulse trials was negatively correlated with the probability of overall startle response modification [$r(23) = -.51$, $p = .01$]. All these correlations are in Appendix 4, Table 4.28.

There was a strong positive correlation between increased perceived stimulus intensity (PPFPSI) in 80dB prepulse and pulse trials and increased startle responses in the same trial type in the baseline EMG part of the session [$r(6) = .77$, $p = .03$]. Decreased perceived stimulus intensity (PPIPSI) in 85dB, 115dB and increased startle response (PPF) in the same trial type in the baseline EMG part of the session also exhibited a strong positive correlation [$r(6) = .83$, $p = .04$]. In the intensity judgment part of the session there was a strong positive correlation between increased perceived stimulus intensity (PPFPSI) in 80dB, 115dB trials and increased startle response in 85dB, 115dB trials [$r(6) = .83$, $p = .04$]. All the intensity ratings modification percentage change and EMG responses correlations are in Appendix 4, Table 4.28.

4.5 Discussion

Neuropsychiatric disorders are associated with multiple levels of malfunction differentiating sufferers from the healthy population and some of these malfunction levels are accessible to the individual's conscious perception. Whilst physiological responses can demonstrate the presence and the extent of associated deficits, they do not represent the 'what it feels like' level very well. Some pathological endophenotypes are characterized by information processing mechanisms disruptions

or deficits not accessible to conscious experience and these features are best measured without resorting to any type of self-report.

Caution is advised when translating conscious perceptions into underlying neural pathways or mechanisms since it has been shown that people are not very good at estimating their motor response magnitude (Blumenthal, et al., 1996).

The limitations of the subjective judgments as a measure of response characteristics are also demonstrated in the temporal judgments. Startle probe presentation leads to faster responses (shorter reaction time), but people's perception of the speed of action remains unchanged (despite the objective measures demonstrating otherwise). The physical task execution is speeded up by the presentation of startle probe, but the conscious assessment of the speed of action remains unchanged (Sanegre, Castellote, Haggard, & Valls-Sole, 2004) indicating a dissociation between the neural circuits for the task execution (affected by the startle presentation, a shorter reaction time) and those involved in the conscious, subjective assessment of one's response to the experimental stimuli (reaction time assessment not affected). Therefore direct or simple relationships between physiological measures and conscious perception of either the stimuli or one's responses are unlikely. Despite these limitations the comparison of conscious experience and motor responses offers additional insight into the nature of sensory and sensory-motor processing in healthy and clinical populations.

Prepulse inhibition of the perceived stimulus intensity was first reported in the 1930s (Peak, 1939), but the subjective startle probe loudness ratings (the pulse, in prepulse and pulse trials) do not significantly correlate with the EMG activity elicited by the same stimuli, and Swerdlow and colleagues (N. R. Swerdlow, et al., 2005) conceded that the two methods measure separate inhibitory processes. Separate neural pathways have been shown to underpin the actual startle response (and its modification) and perceived stimulus intensity (N. R. Swerdlow, N. Stephany, et al., 2002). Amantadine and bromocriptine have divergent effects on PPI and PPIPSI with amantadine abolishing PPIPSI whilst increasing PPI, and bromocriptine having no significant effect on PPI or PPIPSI [a finding different from previous studies on the effects of bromocriptine (Abduljawad, Langley, Bradshaw, & Szabadi, 1998) on

PPI]. These differential effects imply separate neural pathways for the two levels of inhibition (N. R. Swerdlow, N. Stephany, et al., 2002).

Conscious stimuli perception

Prepulse detection

The low intensity stimuli detection probed individual efficiency in sensory processing. Reliable prepulse detection was evident at all prepulse intensities in majority of the sample with no erroneous prepulse detection in pulse-alone trials. In prepulse-alone trials all prepulses at all intensities were detected, but the detection rates in prepulse and pulse trials were significantly lower. The short 120ms inter-stimulus interval led to increased difficulty in prepulse detection when intense pulses followed the prepulses. The intensity of the prepulse stimuli did not have a significant impact on the detection rates, even though prepulse detection rates did increase with increasing prepulse intensity in prepulse and pulse trials. Prepulse detection was robust and the attentional demands resulting from the short inter-stimulus interval had limited effect on the detection rates. A stable detection tendency across all prepulse intensities in both trial types (prepulse-alone and prepulse and pulse), was evident in the strong positive correlations between prepulse detection rates, but was investigated in prepulse and pulse trials only, since all prepulses were detected in prepulse-alone trials.

Increased prepulse detection was associated with lower prepulse intensity ratings, possibly indicating comparison of the weak prepulses against the intense pulses. Since the ratings were provided after each trial, the intense pulse, not the background noise, was the more prominent comparison factor. Higher prepulse detection rates in all prepulse and pulse trials were also associated with higher intensity judgments for prepulses presented alone, but this association was significant for the mid-intense, 85dB prepulses only. A greater hearing sensitivity in the individuals able to discern the prepulses from the intense pulses could explain this association, since such hearing sensitivity would also make the weak prepulses presented alone more prominent against the background noise. Since the hearing thresholds were not tested for each individual beyond ascertaining that they did not suffer a hearing impairment, this explanation remains speculative.

Prepulse detection at all prepulse intensities was positively correlated with increased perceived stimulus intensity ratings (PPFPSI) following the 90dB lead stimulus presentation and prepulse detection at 80dB prepulses was also positively

correlated with PPFPSI in trials with the weakest, 80dB lead stimulus. Prepulse detection has been previously shown to be unrelated to motor response inhibition, yet at the level of stimuli intensity perception prepulse detection is associated with increased ratings for stimuli which ought to be rated as less intense (ratings ought to be inhibited by the prepulse presentation). At the same time probability of increased startle probe intensity ratings was negatively associated with the weakest lead stimulus detection. Individuals likely to detect the weakest prepulses were less likely to increase the startle probe intensity ratings in the 80dB prepulse trials, but at the same time if they did engage the perceived stimuli intensity facilitation, they did so to a greater degree. Prepulse detection at the weakest prepulse intensity was thus associated with decreased probability, but increased degree of the perceived stimulus intensity facilitation. No other associations emerged for prepulse detection and intensity ratings modification.

Individuals more likely to detect the prepulses at all prepulse intensities had higher 90dB prepulse-elicited responses amplitude in prepulse and pulse trials in the baseline EMG (but not the intensity judgment) part of the session. A similar association also emerged for the detection of prepulses at all prepulse intensities and 85dB prepulse-elicited response amplitude (in prepulse and pulse trials) in the intensity judgment part of the session. These associations indicate interaction between prepulse-elicited motor responses as measured by the eyeblink EMG, and prepulse detection. Since the detection activity took place after the motor response was recorded, it is difficult to ascertain whether additional attentional resources improving the detection (or some other enduring individual characteristics increasing prepulse detection) led to the increased prepulse-elicited motor response, or whether the increased muscular activity (expressed in the increased amplitude) led to an improved detection.

However, individuals more likely to detect prepulses were not more likely to exhibit motor responses to such weak stimuli, and there were very limited associations between prepulse detection, prepulse intensity ratings and prepulse-elicited EMG responses. The lack of associations between prepulse detection and startle response modification largely concur with previously published findings (Postma, et al., 2001).

Perceived stimuli intensity ratings

Prepulse detection only required the participants to decide whether the trial contained one or two stimuli. Perceived stimuli intensity judgments for the weak lead stimuli required a more detailed assessment of the stimuli parameters. Increased prepulse intensity was adequately reflected in the perceived prepulse intensity ratings with a significant prepulse intensity effect in all conditions. The comparison of perceived prepulse intensity judgments in prepulse-alone and prepulse and pulse trials revealed lack of significant differences between these two prepulse presentation modes. Unlike the detection rates, the perceived prepulse intensity ratings were not affected to a significant degree by the subsequent pulse presentation in prepulse and pulse trial. However, the prepulses in prepulse and pulse trials were rated as louder, than identical intensity prepulses presented alone, suggesting a perceived stimuli intensity summation for the two stimuli presented in a quick succession. The presentation of the pulse stimuli (of one intensity) had the effect of stabilizing the perceived stimulus intensity ratings for the prepulses, with perceived prepulse intensity ratings being highly positively correlated across all prepulse intensities in prepulse and pulse trials. The correlations in prepulse-alone trials were less systematic, which seems counter-intuitive, since the prepulses presented alone (only one stimulus per trial to compare against the background noise) should have required less effortful loudness judgments. On the other hand the prepulse-alone loudness judgment could have been affected the trial type it was preceded by and it would be interesting to establish to what degree the acoustic neighbourhood affected the perception of the subsequently presented stimuli perception. The small number of trials within the loudness judgment session part did not permit investigating this question. An alternative explanation could be that the prepulses activated the transient detecting reflex, which facilitates stimulus detection, but not discrimination (as between prepulses and pulses) (Graham, 1992), hence the two processes: detection and intensity ratings, were independent.

Conscious stimuli perception and EMG data comparison

Prepulse intensity ratings

Stronger motor responses to the weakest 80dB prepulses were associated with decreased prepulse intensity ratings in prepulse and pulse trials, unlike the associations between prepulse detection and prepulse-elicited response amplitude, in which stronger responses were associated with higher prepulse detection. The

weakest (80dB) and the most intense (90dB) prepulses leading to increased EMG responses were rated as less loud. However, these associations only reached significance for the prepulse-elicited responses in the first part of the session. When measured concurrently with the intensity ratings, no such relationships emerged. It is possible that individuals with stronger EMG responses in the first part of the session would habituate more quickly to the prepulse stimuli and such habituation of the motor response would then also be manifested in the perceived stimuli intensity. Even though the comparison of the EMG response amplitudes across the two parts of the session did not indicate significant differences, habituation too small to reach statistical significance was nevertheless possible.

However, individuals rating the weakest prepulses as more intense were more likely to exhibit 80dB prepulse-elicited responses in the second part of the session, an association mimicking the positive prepulse-elicited response amplitude and prepulse detection. Higher intensity ratings for the weakest prepulses were associated with increased probability of a motor response to these weak lead stimuli. Responses to the weakest prepulses are rare, as compared to the higher intensity lead stimuli (Dahmen & Corr, 2004) and prepulse-elicited responses are subject to habituation (Dahmen & Corr, 2004). The increase in response probability in the second part of the session for individuals perceiving the weakest stimuli as more intense indicates the directed attention made the weak lead stimuli prominent compensating for habituation.

It remains a puzzle as to why increased EMG activity was associated with an improved detection, but reduced intensity ratings. One tentative explanation is that the detection activity is less prone to subtle variation in the individual ability, as it is a 'yes' or 'no' judgment type. The intensity rating activity calls upon an accurate perception of the stimuli loudness in the presence of constant background noise and in the context of preceding trials, and may have posed a degree of difficulty resulting in the decreased accuracy.

Startle probe (pulse) intensity judgments: intensity ratings modification and startle response modification

The larger contrast in the pulse stimuli loudness levels compared to the background noise made them easier to detect in comparison to the weak lead stimuli. All the participants detected the startle stimuli in all trial types. Unlike PPI, often leading to absolute startle response suppression, PPIPSI only reaches 15-20%

reduction (N. R. Swerdlow, Blumenthal, Sutherland, Weber, & Talledo, 2007). The intensity ratings for the pulse stimuli in prepulse and pulse trials reflected some inhibition, and the perceived stimulus intensity judgments for pulse stimuli presented alone were always higher than for pulse stimuli in prepulse and pulse trials. These differences were significant for pulse intensity ratings in 85db and 90dB prepulse and pulse trials, but failed to reach significance for the weakest 80dB prepulse and pulse. The weakest lead stimuli result in the lowest prepulse inhibition of startle response, and this finding resembles prepulse inhibition principles observed in motor responses. Prepulse intensity was expected to have an effect on the pulse intensity ratings based on the findings of Swerdlow and colleagues (N. R. Swerdlow, et al., 2005), but it did not have a significant effect in this sample.

In the previous chapter a paradoxical prepulse facilitation of the startle response was reported which was more likely to occur with weak prepulses and decrease in probability with increasing prepulse intensity (see Chapter 3). The intensity ratings also exhibited the paradoxical facilitation, which was most likely to occur with the weakest prepulses, and decreased in likelihood with increasing prepulse intensity. Lead stimuli which are continuous prepulses (ending when the startle stimulus is presented, no inter-stimulus interval present) lead to perceived stimuli intensity augmentation evident in the pulse ratings in such trials (N. R. Swerdlow, et al., 2007), such augmentation would concur with the phenomenon of fusion, present in the precedence effect, an inhibitory mechanism similar to PPI, but pertaining to conscious perception of sounds (L. Li & Yue, 2002). The modification of the perceived stimuli intensity may be better explained by the precedence effect, rather than frank prepulse inhibition. Precedence effect is the suppression of processing of the second sound, usually presented as an echo of the first sound, in terms of lack of interference produced by the presentation of the second sound in the processing of the original stimulus. The precedence effect is associated with much shorter inter-stimulus intervals than PPI, it remains a relevant explanation though, since the conscious perception of the stimuli may be associated with different temporal sequences than the motor responses, and it is possible that the inter-stimulus interval in the maximal PPI paradigm (120ms) is short enough in terms of attentional processing to succumb to fusion, hearing of one sound only instead of two when presented with two stimuli in quick succession. The 'stabilizing' effect of the pulse presentation on the perceived intensity ratings of the preceding prepulse supports this

possibility, since prepulse intensity ratings were similar across all prepulse intensities in prepulse and pulse, but not prepulse alone trials. It is possible that the paradoxical facilitation under the conditions of maximal inhibition indicates involvement of this alternative sound processing mechanism, perhaps due to a differential sequencing of the stimuli processing, which may be underpinned by an inappropriate neural activation. Studies investigating the cross-modal effects in perceived stimulus intensity modification did not report such paradoxical facilitation (N. R. Swerdlow, Geyer, M.A., Blumenthal, T.D., Hartman, P.L., 1999) and explanations other than frank PPI failure are viable for stimuli perception augmentation for the same-modality stimuli.

The comparison of the physiological responses and the intensity ratings indicated that individuals more likely to inhibit their perceived stimulus intensity as the result of the weakest 80dB prepulse presentation exhibited lower percentage inhibition of their concurrently recorded startle response. Paradoxically people more likely to rate the pulses following the weakest prepulses as louder than those presented alone (PPFPSI), were more efficient in inhibiting their startle response in the same trial type in both parts of the session. There is some evidence that reflex inhibition and augmentation are underpinned by separate neural pathways (Hoffman, Cohen, & Stitt, 1981). Individuals more likely to perceive the pulse stimuli following 90dB prepulses as louder than those presented alone (PPFPSI) were also less likely to modify their concurrently recorded startle response in the prepulse and pulse trials containing the weakest, 80dB prepulses. These opposite directions of perceived stimuli intensity and motor response modification changes demonstrated the lack of a simple relationship between the conscious stimuli processing and the motor responses asserted in the relevant studies (Swerdlow, Stephany et al. 2005; Swerdlow, Blumenthal et al. 2007). Higher startle response percentage reduction in trials with the weakest lead stimuli was associated with increased probability of reduced stimuli intensity ratings (PPIPSI) in trials with the most intense lead stimuli. Individuals with efficient inhibition (elicited by the weakest lead stimuli) were likely to decrease their ratings pulse loudness following the most intense prepulses, an association which demonstrates some association between efficient inhibition of the motor response and efficient inhibition of the perceived stimuli intensity. Moreover increased probability of PPFPSI in prepulse and pulse trials with the most intense

90dB prepulses was associated with increased startle response in the same trial type, but only in the first part of the session.

The comparison of the intensity ratings modification (IRM) percentage change and startle response modification revealed several associations. Individuals prone to increased startle response (PPF) in 85dB prepulse and pulse trials in the baseline EMG part of the session were prone to higher inhibition of the perceived stimulus intensity (PPIPSI) in the same trial type. However, a different relationship emerged for increased startle responses in 80dB prepulse and pulse trials in the baseline EMG part of the session and increased perceived stimulus intensity (PPFPSI) in 80dB, 115dB prepulse and pulse trials; increased startle responses in such trials were associated with increased perceived stimuli intensity. A similar association emerged in the intensity judgment part of the session where there was a strong positive correlation between increased perceived stimulus intensity (PPFPSI) in 80dB, 115dB trials and increased startle response in 85dB, 115dB trials. These associations point to a closer relationship between the more unusual, increased startle responses (PPF) and perceived stimulus intensity modification, than is the case for the more common inhibited startle response in prepulse and pulse trials.

Attentional effects

Directing attention towards the lead stimuli has the effect of increasing PPI and the presented study aimed to assess the effects of directed attention paradigm using the proxy measure of differences between the two session parts (baseline EMG recording-undirected paradigm, presented first; intensity judgments- attention directed towards the lead stimuli). The comparison of the EMG recordings in the two parts of the session (the baseline EMG recording and the intensity judgment part) demonstrated a limited degree of habituation for prepulse-elicited responses or startle responses with only 90dB prepulse-elicited response amplitudes being different across the two parts of the session. However, since the 90dB prepulse-elicited responses were the most numerous ones, it is possible that the lack of habituation of the prepulse-elicited responses is the result of the limited number of responses available for the comparison, rather than a true lack of habituation for prepulse-elicited responses at the other two prepulse intensities. Alternatively the reduction in the response amplitudes may have been too subtle to be detected by the statistical test used to gauge the changes. The probabilities of prepulse-elicited responses exhibited habituation across the two parts of the session with responses to 85dB and 90dB

prepulses being significantly more frequent in the first (baseline EMG) part of the session. Motor responses to 80dB prepulses are the least frequent (Chapter 3) and it is possible that, in a manner similar to the amplitude changes, there were not enough instances of such responses to support a meaningful comparison.

There were too few trials of each type presented in each session part to assess habituation within each session part separately. A comparison of the EMG recordings across the two session parts enabled an assessment of habituation and the effects of the differential protocol (undirected versus directed attention paradigm) for startle response modification. The differences in the startle modification percentage change in the two session parts did not reach significance, whether attributed to habituation or directed attention. However, as it has been shown that directed attention increases PPI it is possible that the lack of differences is a result of the increased attention compensating for startle response modification habituation. PPI habituation has been previously demonstrated (Dahmen & Corr, 2004) for PPI calculated in a manner equivalent to startle response modification (SRM) presented in this study (i.e. before splitting into decreased, PPI and increased, PPF startle response). The same lack of session part effect held for startle response modification probability. Neither habituation nor contamination or directed attention (in the intensity judgment part) affected the probability of one type of startle response modification versus the other (PPI or PPF). Apart from the effects of habituation or directed attention neither the prepulse-elicited responses, nor startle response modification were associated with evidence of the rating activity contaminating the EMG recording in the intensity judgment part of the session.

Individual differences

Corr and associates (Corr, et al., 2002) suggested that personality and PPI may be associated via a third variable- subjective intensity of stimuli. Assuming that high emotionality leads to subjective stimuli intensity amplifications, then individuals prone to negative emotionality, and specifically those with high negative state emotionality, should differ from the rest of the group in terms of PPI, baseline startle and possibly prepulse reactivity. What is more, their subjective perception of stimuli intensity should also be increased, due to increased arousal associated with some aspects of negative emotionality.

Prepulse detection

Personality factors did not exhibit strong relationships with prepulse detection. Individuals high on the Unusual Experiences subscale of the O-LIFE were less likely to detect mid-intensity 85dB prepulses, indicating some association between proneness to misinterpretation of the perceptual inputs across several domains. High scorers on the Reward Dependence subscale of the TCI (putatively underpinned by serotonergic activity), were more likely to detect the weakest 80dB prepulses. Such association would imply a role for the serotonergic pathways in sensitizing individuals to the presence of not easily distinguished stimuli. The moderate negative associations of prepulse detection (at all prepulse intensities) and Self-Transcendence are interesting, since there is some evidence that this character is linked to dopamine genes (Comings, et al., 2000) and dopaminergic activity is implicated in sensorimotor gating. However, studies published to date indicate that conscious prepulse detection is not associated with the subsequent startle response modification (but see 4.4.3.2 in this Chapter).

Prepulse intensity ratings

Prepulse intensity ratings exhibited mixed associations with personality factors. Individuals high on the Introvertive Anhedonia subscale of the O-LIFE rated the most intense 90dB prepulses as louder. The Introvertive Anhedonia subscale measured characteristics akin to depression, including passivity and withdrawal, and it seemed to have rendered individuals sensitive to perceiving the most intense 90dB prepulses as very loud. Individuals high on the Reward Dependence subscale of the TCI rated 80dB and 85dB prepulses as weaker. Interestingly enough these individuals were more likely to detect the weakest 80dB prepulses and yet they rated them as weaker. It could be expected that individuals high on the Fear of Noises subscale of FSS would rate all stimuli as more intense, but such positive correlation appeared only for the intensity ratings of 85dB prepulses in prepulse and pulse trials.

Intensity ratings modification

Personality factors exhibited several significant associations with the stimulus intensity ratings modification (IRM) percentage change. Individuals experiencing high Negative Affect (subscale of PANAS) were revealed to have higher percentage inhibition at all prepulse intensities, but higher percentage facilitation in 90dB prepulse and pulse trials. Facilitation (PPF) in 90dB trials was rare in terms of the motor response, with an inverse relationship between prepulse intensity and PPF

probability (though not percentage, also see Chapter 3). It is interesting that negative affect was associated with increased stimuli intensity perception, as if the negative affective state predisposed the individuals to sensitization, rather than inhibition as the result of a strong prepulse presentation in the perceptual (not motor) processing of the stimuli. In the study presented in Chapter 3 a significant positive association between facilitated startle response probability (following 90dB prepulse presentation) and negative affect was also found.

Other personality factors were rarely associated with intensity ratings modification. The TCI subscale of Reward Dependence was significantly correlated with PPIPSI in 90dB prepulse and pulse trials, and PPFPSI in 80dB prepulse and pulse trials, which is a consistent association since the strongest prepulses elicit highest percentage PPI and the weakest prepulses are most likely to lead to PPF, although prepulse intensity had been shown to lack a significant effect on the percentage change in PPF (Chapter 3). The Fear of Animals subscale of FSS was positively correlated with PPIPSI in 90dB prepulse and pulse trials, indicating that individuals high in such fear type are more likely to have efficient inhibition as the result of the presentation of the most intense 90dB prepulse.

The effects of sex and age

Unlike PPI, which is normally higher in males and exhibits sex specific differences in both healthy and clinical samples (see Chapter 1), prepulse inhibition of the perceived stimulus intensity (PPIPSI) was associated with a lack of differences between males and females. Previously this lack of the effect of sex was attributed to attention directed towards the pulse stimuli during the rating activity (Swerdlow, Stephany et al. 2005; Swerdlow, Blumenthal et al. 2007). Directed attention has been shown to increase PPI and such increase should equally apply to all the comparison groups and result in elevated PPI in the PSI paradigm. It is however difficult to see how it can be determined whether the attention was effectively directed towards the prepulses (as in previously published studies demonstrating the effects of attentional manipulation) or the pulses when the inter-stimulus interval is only 120ms and the ratings are supplied after the presentation of both stimuli. In the presented study older participants who rated the pulses preceded by 85dB prepulses as more intense then the pulses presented alone, did so to a lesser degree (smaller percentage increase) and since the 85dB prepulses were expected to elicit maximal PPI this finding lends some support to refuting the assumption of deficient inhibitory

mechanisms associated with ageing. No other demographic characteristics predisposed individuals to one type of PSI modification (intensity ratings modification; IRM) versus the other (PPIPSI or PPFPSI). Sex had no significant effect on prepulse detection, prepulse intensity ratings (apart from prepulses presented alone) or perceived stimulus intensity modification.

Summary

The weak lead stimuli intensity ratings adequately reflect the stimuli intensity with linear, stimuli intensity dependent changes. The rating activity does not contaminate the concurrent EMG recording. Both prepulse inhibition and paradoxical prepulse facilitation appears in the perceived stimulus intensity ratings (PPIPSI and PPFPSI). Personality and demographic characteristics have limited associations with PSI. Conscious stimuli intensity perception, including prepulse detection, displays a similar lack of meaningful associations with motor responses. The relationships between the physiological responses and the conscious stimuli intensity perception (or reporting of thereof) is problematic in terms of reflecting the prepulse-driven response modification processes and therefore perceived stimuli intensity cannot be recommended as a reliable and valid, replacement for EMG recording.

Limitations and future recommendations

Whilst perceived stimulus intensity modifications remains a valid research question in its own right and would no doubt yield interesting data in terms of its neural substrates and pharmacological manipulation consequences, it is a process similar to, but separate from the classic startle response modification as measured by the motor responses. Hence it cannot be equated with the same inhibitory process, albeit delayed by the need for conscious processing and rating response.

The rating activity invariably attracts the participants' attention to the prepulses and the consequences of such attentional manipulation are well documented, it is not possible to envisage a paradigm in which this attentional bias can be controlled for. The reliability of the ratings has to be questioned, even though the 'range correction' can correct for the individual's tendency to systematically underrate or overrate the stimuli intensity. However, the ability to express one perceptual dimension (auditory

experience) in terms of another (visual representation) may in itself be prone to individual differences.

In future the perceived stimulus intensity modification should be treated as a research question separate from the traditional startle response modification, and it would be interesting to compare the neural activity in the individuals efficient at detecting the prepulses and accurately assessing the perceived stimuli intensity, with those who do not detect the weak prepulses or rate the stimuli inconsistently. Using methods of intensity ratings recording other than visual analogue scales may also be helpful, although invariably all such methods, even sound intensity matching, are ridden with the problem of individual perception and accuracy differences.

It would be interesting to employ the paradigm from visual stimuli processing which uses reaction times and temporal order judgment. The prepulses and pulses are presented in close temporal proximity and it would be interesting to see how accurate individuals are in perceiving the correct order of stimuli presentation and how stimuli intensity affects these judgements and stimuli detection reaction times. There is some evidence that attentional resources are preferentially allocated to low intensity stimuli regardless of the formal instruction (Jaskowski & Verleger, 2000) as shown by faster RT to bright visual stimuli when presented in the space where dim stimuli are expected. Such expectation violation could also affect detection reaction times or temporal order judgments for auditory stimuli. It would be interesting to see how well an individual's assessment of when they blink to different intensity stimuli relates to the EMG recording; i.e. do some people show systematic biases (related to their personality characteristics or their stimuli-elicited responses).

5 Predictable trial onset effects on prepulse reactivity, prepulse inhibition and paradoxical prepulse facilitation.

5.1 Introduction

Prepulse inhibition occurs when a weak lead stimulus (prepulse) precedes the startle probe presentation. Startle probe in the acoustic startle response modification research is an intense burst of sound, aversive even at lower intensities. People's responses to aversive stimuli are altered if the onset of these stimuli can be anticipated. The change in the response resulting from the predictability of the aversive stimulus onset is termed 'preception' (Lykken, 1962). Predictable aversive stimulus onset can lead to a reduction (negative preception) or increase (positive preception) of the response. The inter-stimulus interval (ISI) of 120ms, commonly used in sensorimotor gating research, is long enough for attentional mechanisms to exert some influence (see Chapter 1) – during this time, the experimental participant knows that there is a high probability of the aversive, startle probe, stimulus . The startle probes are intense bursts of sound, perceived as unpleasant by most people; and the 120ms ISI is long enough for attention to be engaged, which can be problematic if it allows participants to anticipate the startle probe onset. Prepulse presentation reliably leads to startle probe presentation in paradigms which use prepulse and pulse trials only to index startle response modification, and contain no 'prepulse alone' trials (so, thereby, increasing the uncertainty of startle probe onset). It can be argued that prepulse presentation, with a stable temporal distance to the startle probe presentation, serves as a cue of the impending startle probe onset in such paradigms. In humans, the startle reflex can be both inhibited and facilitated by stimulus anticipation (Ison, Sanes, Foss, & Pinckney, 1990) and whilst negative preception (response reduction as the result of the aversive stimulus onset predictability) has received widespread attention, facilitation of the responses in the condition of predictability (positive preception) has been less well documented.

The first study presented in this chapter investigates the effects of short, predictable inter-trial intervals (ITI) on prepulse reactivity (PPER) and startle response modification (SRM). These two processes have not been investigated under conditions of predictability and it is possible that preception can be an alternative inhibitory mechanism or an additive effect. The first study is intended as a

foray into an unknown territory, as the effects of stimulus onset predictability on prepulse inhibition or facilitation have not been previously investigated. Therefore the aims of this study are circumscribed to whether PPER, PPI and the paradoxical PPF occur under the conditions of stimulus onset predictability and what effects the paradigm has on the associations between PPER and SRM. The second study focuses on the issue of preception as a stand-alone modification mechanism and compares startle responses in the condition of unpredictability with startle responses in the condition of predictability. If preception is an alternative startle modification mechanism then the startle responses elicited by startle probes in the predictable condition should be different from those elicited in the traditional, unpredictable paradigm. Startle responses are potentiated in the condition of uncertainty if a condition of certainty has also been presented (Grillon, Baas, Cornwell, & Johnson, 2006). In the second study preception is calculated as change in the response size when startle probes have predictable onset. Negative preception in this paradigm is expressed as a reduction in the startle response size (positive value), and positive preception leads to increased startle response sizes (negative value) in the predictable condition. The sign of the change indicates decrease or increase of the startle response as the result of predictable stimulus onset, and the size of the figure reflects the degree of this change.

The third study centres on the issue of startle response modification under the conditions of certainty. Increased temporal (when the stimuli may occur) or increased event uncertainty (what type of stimuli will be presented) increase PPF in long lead (long inter-stimulus interval) paradigms, whereas increased certainty decreases it (Graham, 1975). Bearing in mind the paradoxical PPF noted in the previous studies (Chapters 3 & 4), the third study examines whether the temporal (stable inter-trial interval) or event (prepulses always followed by pulses) certainty would increase the probability of the paradoxical PPF. If preception is an additive effect in the same direction as startle response modification, then the rates of startle response modification should be higher for prepulse and pulse trials presented at predictable inter-trial intervals (reduction due to predictability plus reduction due to prepulse inhibition or increase due to predictability plus increase due to prepulse facilitation).

Negative preception (reduction) of startle response modification means lowering the percentage prepulse inhibition or facilitation, and positive preception means elevation of the SRM percentage values.

Therefore the following outcomes are possible assuming an effect of preception:

High PPI + negative preception of the startle response = elevated PPI

High PPI + positive preception of the startle response = reduced PPI

High PPF + negative preception of the startle response = reduced PPF

High PPF + positive preception of the startle response = elevated PPF

However, the changes in SRM from the unpredictable to the predictable condition are calculated as percentage change from one condition to the other. Therefore a small figure indicates maintenance of the specific SRM between the two conditions, and a large figure denotes a dramatic change between the SRM levels in the two conditions. A positive number indicates that the percentage SRM in the predictable condition is smaller than in the unpredictable condition, and a negative number means a substantial increase in the SRM percentage in the predictable condition.

The impact of individual differences on preception was investigated in all three studies, although previous investigations (Taylor, 2004) demonstrated lack of associations between preception and anxiety disorders or depression.

The main aim of the presented chapter is establishing the effects of the short, predictable inter-trial intervals, and in consequence predictable stimulus onset, on PPER and SRM. The following hypotheses about the impact of stimulus onset predictability will be tested:

1. PPER will be different in all its characteristics (probability, amplitude, temporal features)
2. SRM will be different (type, probability, percentage change)
3. Individual differences will affect the scope of differences in PPER
4. Individual differences will affect the scope of differences in SRM

5.2 Predictable inter-trial interval effects on prepulse reactivity and startle response modification

5.2.1 Methods and materials

5.2.1.1 Participants

Twenty participants were recruited from university employees and postgraduate students (age range: 18-55 years, *M* = 24, *SD* = 8; 13 females, age range: 20-28, *M* = 24, *SD* = 3, 7 males, age range: 18-55, *M* = 26, *SD*= 13). Participants had a chance to win £100 in return for taking part. Exclusion criteria comprised self-declared suboptimal hearing, tinnitus, drug abuse or psychiatric disorder history. The times of last nicotine and alcohol consumption were recorded and hearing acuity was tested at 40dB. The participants were all non-smokers. Two participants responded in less than seven pulse alone trials (ten such stimuli presented in the main session) and were excluded as non-responders. The final sample size consisted of eighteen participants (age range: 18-55, M = 24, SD = 8; 11 females, age range: 20-27, M = 23, SD = 2; 7 males, age range: 18-55, M = 26, SD= 13).

5.2.1.2 Materials

The same materials were used as in the study presented in Chapter 3.

5.2.1.3 Physiological data collection

Physiological data collection was identical to the second study presented in Chapter 3 with the exception of 2 minutes acclimation time (as opposed to three) and predictable, constant inter-trial interval of 10s (as opposed to variable ITI of 9-21s).

5.2.1.4 Design

Participants completed the self-report questionnaires and were exposed to all the stimuli (with 10 presentations of each stimulus type in each condition). A mixed design was used, with sex and personality measures as the between subjects factors and the eyeblink-eliciting stimuli driven responses as the within-participant factor.

5.2.1.5 Data Scoring and Statistical Analysis

Data scoring and statistical analysis were identical to those presented in Chapter 3. The effects of brief, predictable inter-trial interval were investigated in terms of prepulse reactivity and startle response modification.

5.2.2 Results

Prepulse reactivity (PPER)

PPER prevalence

The majority of the sample exhibited prepulse-elicited responses and the probabilities of prepulse reactivity increased with increasing prepulse intensity (Appendix 5, Figure 5.1). In prepulse and pulse trials, sixty-one percent of the sample (11 out of 18) had 80dB prepulse-elicited responses; sixty-seven percent (12 out of 18) had 85dB prepulse-elicited responses and eighty-three percent (15 out of 18) had 90dB prepulse-elicited responses. In prepulse-alone trials, eighty-three percent of the sample (15 out of 18) had 80dB prepulse-elicited responses; sixty-seven percent (12 out of 18) had 85dB prepulse-elicited responses and ninety-four percent (17 out of 18) had 90dB prepulse-elicited responses (Appendix 5, Table 5.1). The probabilities of prepulse-elicited responses increased with increasing prepulse intensity in prepulse and pulse [80dB, 14%; 85dB, 26%; 90dB, 39%] and prepulse-alone trials [80dB, 17%; 85dB, 27%; 90dB, 42%], (Appendix 5, Table 5.1). Three participants (17% of the sample) exhibited spontaneous EMG activity in prepulse temporal space in pulse-alone trials, and seven participants (39% of the sample) did so in the no stimulus, 'blind' trials.

PPER probability

The probabilities of lead stimulus-driven motor responses were significantly higher then non-stimulus driven spontaneous responses in all trials where prepulses were presented (Appendix 5, Table 5.3). The results are summarized in Table 5.2.2.a below. Motor response probability increased with increasing prepulse intensity (Appendix 5, Figure 5.1) and there was a significant effect of prepulse intensity on response probability in prepulse and pulse trials [Greenhouse-Geisser correction applied, F (2,18) = 8.60, p = .004] and in prepulse-alone trials [F (2,18) = 9.02, p = .001]. Sex had no effect on prepulse-elicited response probability in either prepulse and pulse [F (2,18) = .27, ns] or prepulse-alone trials [F (2,18) = .21, ns]. There were

no significant differences between response probabilities in prepulse and pulse and prepulse alone trials (Table 5.2.2.b). The probabilities of prepulse-elicited responses were positively correlated across all prepulse intensities and both trial types [correlations ranging from $r(16) = .49, p = .04$ to $r(16) = .87, p < .01$] (Appendix 5, Table 5.4).

Table 5.2.2.a Comparison of prepulse-elicited response probabilities to spontaneous EMG activation [SP = startle probe alone; PP = prepulse and pulse trial; PA = prepulse alone trial; PPx, x = lead stimulus dB]

Trial Type	*t*	*df*	*p*
PP80- SP	2.67	17	**.02**
PP85 - SP	3.53	17	**< .01**
PP90 - SP	4.65	17	**< .01**
PA80 - SP	3.56	17	**< .01**
PA85 - SP	3.69	17	**< .01**
PA90 - SP	5.27	17	**< .01**
PP80 - B	2.58	17	**< .01**
PP85 - B	3.48	17	**< .01**
PP90 - B	4.74	17	**< .01**
PA80 - B	3.55	17	**< .01**
PA85 - B	3.47	17	**< .01**
PA90 - B	5.29	17	**< .01**

Table 5.2.2.b Probabilities of PPER in prepulse and pulse and prepulse alone trials

Trial Type	*t*	*df*	*p*
PP80 - PA80	- 1.05	17	ns
PP85 - PA85	- .34	17	ns
PP90 - PA90	-.68	17	ns

PPER amplitude

PPER amplitudes are shown in Appendix 5, Figure 5.2. PPER amplitude values in PP trials increased in a linear fashion with increasing prepulse intensity, but in PA trials a u-shaped distribution was found. However, prepulse intensity had no effect on the response amplitudes in either trial type [PP: F (2,18) = .85, ns; PA: F (2,18) = 2.01, ns]. Sex had no significant effect on the amplitudes either [PP: F (18,2) = .50, ns; PA: F (2,18) = .11, p = ns]. There were no significant differences in terms of response amplitude between PP and PA trials (comparison outcomes summarized in Table 5.2.2.c). The amplitudes of prepulse-elicited response were positively correlated across all prepulse intensities and all trial types, but only some of these associations reached significance [correlations ranging from $r(10) = .60$, $p = .04$ to $r(9) = .78$, $p < .01$] (Appendix 5, Table 5.5).

Table 5.2.2.c Comparison of response amplitudes in PP and PA trials

Trial Type	*t*	*df*	*p*
PP80 - PA80	-.15	10	ns
PP85 - PA85	1.60	10	ns
PP90 - PA90	-.17	13	ns

PPER onset

Prepulse-elicited responses had faster onset times with rising prepulse intensity (Appendix 5, Figure 5.3). However, prepulse intensity had no effect on the response onsets in either trial type [PP: F (2,18) = 2.30, ns; PA: F (2,18) = 2.43, ns]. Sex had no significant effect on the onset values either [PP: F (2,18) = 1.58, ns; PA: F (2,18) = .56, ns]. There were no significant differences in terms of response onset between PP and PA trials (comparison outcomes summarized in Table 5.2.2.d). The onsets of prepulse-elicited response were positively correlated across all prepulse intensities and all trial types, but only some of these associations reached significance [correlations ranging from $r(12) = .58$, $p = .03$ to $r(10) = .70$, $p = 01$] (Appendix 5, Table 5.6).

Table 5.2.2.d PPER onsets in prepulse and pulse and prepulse alone trials

Trial Type	*t*	*df*	*p*
PP80 - PA80	-.11	10	ns
PP85 - PA85	.45	10	ns
PP90 - PA90	1.55	13	ns

PPER peak latency

The peak latency was decreasing with increasing prepulse intensity (Appendix 5, Figure 5.4). There was a significant effect of prepulse intensity on PPER peak latency in prepulse and pulse, but not in prepulse alone trials [PP: F (2,18) = 7.08, p = .01; PA: F (2,18) = .88, ns]. Sex had no significant effect on the peak latency [PP: F (2,18) = .02, ns; PA: F (18, 2) = .23, ns]. There were no significant differences in terms of response peak latency between PP and PA trials (comparison outcomes summarized in Table 5.2.2.e). The peak latency values of prepulse-elicited response were positively correlated across all prepulse intensities and all trial types, but only some of these associations reached significance [correlations ranging from $r(9)$ = .67, p = .02 to $r(9)$ = .91, p < .01] (Appendix 5, Table 5.7).

Table 5.2.2.e Peak latency in prepulse and pulse and prepulse alone trials

Trial Type	*t*	*df*	*p*
PP80 - PA80	1.30	10	ns
PP85 - PA85	.54	10	ns
PP90 - PA90	-1.01	13	ns

Prepulse-elicited response onset to peak latency

The onset to peak latency values decreased with increasing prepulse intensity in prepulse and pulse trials, but increased with increasing prepulse intensity in prepulse alone trials (Appendix 5, Figure 5.5). However, prepulse intensity had no effect on the response onset to peak latency values in either trial type [PP: F (2,18) = .66, ns; PA: F (2,18) = .70, ns]. Sex had no significant effect on the onset to peak latency values either [PP: F (2,18) = 2.14, ns; PA: F (2,18) = 1.65, ns]. There were no significant differences in terms of response onset to peak latency between PP and PA trials (comparison outcomes summarized in Table 5.2.2.f). There were no

significant associations between the onset to peak latency values in the six conditions (Appendix 5, Table 5.8).

Table 5.2.2.f Onset to peak latency (index of response duration) in prepulse and pulse and prepulse alone trials

Trial Type	*t*	*df*	*p*
PP80 - PA80	1.02	10	ns
PP85 - PA85	.07	10	ns
PP90 - PA90	- 2.25	13	ns

Individual differences and PPER

The associations between the personality factors and PPER characteristics are presented in Appendix 5, Table 5.9 – Table 5.13. Personality factors and PPER probabilities exhibited few significant correlations (Table 5.2.2.g).

Table 5.2.2.g Personality factors and PPER probabilities (x) = N

Personality Factors	PP80	PP85	PP90	PA80	PA85	PA90
Unusual Experiences	$r = .58$, $p = .01$ (16)	$r = .53$, $p = .02$ (16)	ns	$r = .57$, $p = .01$ (16)	$r = .57$, $p = .01$ (16)	ns
Persistence	ns	$r = .59$, $p = .01$ (16)	ns	ns	r = .45, $p = .04$ (16)	$r = .57$, $p = .01$ (16)
Fear of Noises	ns	ns	ns	$r = .53$, $p = .02$ (16)	ns	ns

PPER amplitudes exhibited more numerous significant associations with personality factors (Table 5.2.2.h).

Table 5.2.2.h Personality factors and PPER amplitudes (x) = N

Personality Factors	PP80	PP85	PP90	PA80	PA85	PA90
BAS Fun	ns	ns	$r = .55$, $p = .04$ (12)	ns	$r = .66$, $p = .02$ (10)	ns
Unusual Experiences	ns	ns	$r = .61$, $p = .01$ (13)	ns	ns	$r = .67$, $p < .01$ (15)
Cognitive Dissonance	ns	ns	ns	$r = -.53$, $p = .04$ (13)	ns	ns
Persistence	ns	ns	$r = .76$, $p < .01$ (13)	ns	ns	ns
Self Transcendence	ns	ns	ns	$r = .61$, $p = .01$ (13)	ns	ns
Fear of Animals	ns	$r = -.63$, $p = .03$ (10)	ns	ns	ns	ns

PPER onsets and personality factors had a few significant associations (Table 5.2.2.i).

Table 5.2.2.i Personality factors and PPER onsets

Personality Factors	PP80	PP85	PP90	PA80	PA85	PA90
Positive Affect	ns	ns	ns	ns	ns	$r = .54$, $p = .02$ (15)
BAS Reward	$r = -.68$, $p = .02$ (9)	ns	ns	ns	ns	ns
Persistence	ns	ns	ns	ns	ns	$r = .57$, $p = .02$ (15)

There were some significant associations between PPER peak latency values and personality factors (Table 5.2.2.j).

Table 5.2.2.j Personality factors and PPER peak latency values

Personality Factors	PP80	PP85	PP90	PA80	PA85	PA90
BAS Reward	ns	ns	ns	r= - .54, p = .04 (13)	ns	ns
Persistence	ns	ns	ns	r = - .60, p = .02 (13)	ns	ns
Cooperation	ns	ns	ns	ns	r = .66, p = .02 (10)	ns
Classical Phobias	ns	ns	ns	ns	ns	r = .48, p = .05 (15)

The associations between personality factors and PPER onset to peak latency values (index of response duration) were as numerous as the associations with PPER amplitudes (Table 5.2.2.j).

Table 5.2.2.j Personality factors and PPER onset to peak latency values

Personality Factors	PP80	PP85	PP90	PA80	PA85	PA90
Trait Anxiety	ns	r = .71, p < .01 (10)	ns	ns	ns	ns
BAS Reward	ns	ns	ns	r = -.58, p = .02 (13)		
BIS	r = .68, p = .02 (9)	ns	ns	ns	ns	ns
Cognitive Dissonance	ns	ns	ns	ns	r = .63, p = .03 (10)	ns
Harm Avoidance	ns	ns	ns		r = - .62, p = .03	ns

					(10)	
Persistence	ns	ns	ns	$r = -.56$, $p = .03$ (13)	ns	$r = -.65$, $p < .01$ (15)
Self Transcendence	ns	ns	ns	$r = -.52$, $p = .05$ (13)	ns	ns
Fear of Animals	ns	ns	ns	ns	ns	$r = .48$, $p = .05$ (15)

Age was associated with PPER onset and onset to peak latency values in prepulse and pulse trials with the weakest, 80dB lead stimuli. Older participants had faster onset of such responses [r (9) = - .76, $p < .01$] and it took them longer to reach the peak amplitude [r (9) = .70, p = .02]. Sex did not exhibit any significant associations with PPER characteristics except PPER amplitude in 90dB prepulse and pulse trials [r (13) = - .63, p = .01] (Appendix 5, Tables 5.25- 5.29).

Startle Response Modification (SRM)

Prepulse inhibition (PPI): probability and percentage change

All participants had pulse-elicited responses in prepulse and pulse trials (Appendix 5, Table 5.2). A number of participants (5 out of 18, 28%) had motor responses to non-existent pulses in the no stimulus 'blind' trials and in trials with the weakest lead stimuli of 80dB. Some (3 out of 18, 17%) also had motor response to non-existent startle probes in the prepulse alone trials with mid-intensity, 85B prepulses and the strongest, 90dB prepulses. However, the small number of these individuals and a small number (usually only one) of such spontaneous EMG activation stipulate caution in inferring too much from these occurrences and even though these responses met the response validity criteria, their scarcity points towards spontaneous EMG fluctuation, rather than a meaningful trend. On the other hand it may be that a larger number of trial presentations would demonstrate individual differences in propensity towards generating such responses, almost as if in anticipation of the startling stimulus.

Probability of prepulse inhibition (PPI) increased with increasing prepulse intensity (Appendix 5, Figure 5.6). There was a significant effect of prepulse

intensity [F (18, 2) = 13.26, $p < .01$], but not sex [F (18, 2) = 1.51, ns] on PPI probability. There was only one significant association amongst PPI probabilities, a positive correlation for PPI probability following the weakest, 80dB prepulses and the strongest, 90dB prepulses [$r(16) = .47, p = .05$] (Appendix 5, Table 5.14). There were positive associations between PPI probabilities and percentage changes [correlations ranging from $r(16) = .48, p = .04$ to $r(16) = .74, p < .01$] (Appendix 5, Table 5.15). PPI probability was not associated with percentage change PPF at all [all correlations ns] (Appendix 5, Table 5.16).

Percentage values of PPI also increased with increasing prepulse intensity (Appendix 5, Figure 5.7). There was a significant effect of prepulse intensity [F (2,18) = 31.70, $p < .01$], but not sex [F (2,18) = .10, ns] on PPI percentage values. Percentage values for PPI displayed significant associations at all prepulse intensities [correlations ranging from $r(16) = .73, p < .01$ to $r(16) = .91, p < .01$] (Appendix 5, Table 5.16).

Paradoxical prepulse facilitation (PPF): probability and percentage change

The probability of paradoxical prepulse facilitation decreased with increasing prepulse intensity (Appendix 5, Figure 5.8). There was a significant effect of prepulse intensity [F (2,18) = 15.60, $p < .01$], but not sex [F (2,18) = 1.81, ns] on the probability of PPF. Only one significant association appeared for PPF probabilities, a positive correlation between PPF in 80dB prepulse and pulse trials, and PPF in 90dB prepulse and pulse trials [$r(16) = .55, p = .02$] (Appendix 5, Table 5.16).

PPF percentage change increased with increasing prepulse intensity (Appendix 5, Figure 5.9) but there was no significant effect of prepulse intensity on PPF percentage change [F (2,18) = 1.19, ns] and the effects of sex could not be calculated due to scarcity of the responses for comparison. PPF probabilities and PPF percentage changes exhibited no significant associations [all correlations p = ns] (Appendix 5, Table 5.17). PPF probabilities and PPI percentage change on the other hand exhibited significant negative correlations [correlations ranging from $r(16) = -.48, p = .05$ to $r(16) = -.73, p < .01$] (Appendix 5, Table 5.17).

Associations between PPI and PPF

The probabilities of the two startle response modification types were negatively correlated [correlations ranging from $r(16) = -.52, p = .03$ to $r(16) = .98$, $p < .01$] (Appendix 5, Table 5.14). In terms of percentage change associations stronger PPI was associated with weaker PPF [correlations ranging from $r(15) =$ -

.49, $p = .04$ to $r(15) = -.60$, $p = .01$] with the exception of PPI in 80dB prepulse and pulse trials and PPF in 85dB prepulse and pulse trials [$r(8) = .69$, $p = .03$] (Appendix 5, Table 5.15).

PPER and SRM

Based on the findings reported in the previous chapters, PPER probabilities and SRM probabilities and percentage changes were correlated, but no significant associations emerged for any of the variables. PPER amplitudes and SRM exhibited two significant associations both for 80dB prepulses in prepulse and pulse trials and probabilities of PPI following 90dB prepulse presentation [$r(9) = -.66$, $p = .03$] and PPF following 85dB prepulse presentation [$r(9) = .66$, $p = .03$]. PPER onset in prepulse and pulse trials with 80dB prepulses was negatively correlated with PPF probability in the same trial type [$r(9) = -.61$, $p = .05$]. Several significant associations emerged for PPER latencies and SRM probabilities. PPER latency in 90dB prepulse-alone trials was positively correlated with PPI in trials with 80dB prepulses [$r(15) = .56$, $p = .02$] and 90dB prepulses [$r(15) = .52$, $p = .03$], but negatively correlated with PPF probability in 80dB prepulse and pulse trials [$r(15) = -.61$, $p < .01$] and 90dB prepulse and pulse trials [$r(15) = -.52$, $p = .03$]. PPER latency in 85dB prepulse-alone trials was positively correlated with probability of PPI in 90dB prepulse and pulse trials [$r(10) = .58$, $p = .05$]. There was a positive association between response duration for PPER in 85dB prepulse-alone trials and PPI probability in 85dB prepulse and pulse trials [$r(10) = .77$, $p < .01$] and a negative association with PPF probability in the same trial type [$r(10) = -.90$, $p < .01$]. There was a positive association for PPF following 90dB prepulse presentation and duration of responses in 80dB prepulse alone trials type [$r(13) = .62$, $p = .01$]. There were positive associations between PPER duration in 85dB prepulse-alone trials and percentage values of PPI in 85dB prepulse and pulse trials [$r(10) = .67$, $p = .02$] and PPI in 90dB prepulse and pulse trials [$r(10) = .66$, $p = .02$]. All these results are in Appendix 5, Tables 5.18 – 5.22.

Individual differences and startle response modification: probabilities and percentage changes

There were only two significant associations between personality factors and probabilities of startle response modification and these were both for the Classical Phobias subscale of the FSS [PPI 80dB: $r(16) = .54$, $p = .02$ and PPF 80dB: $r(16) = -.61$, $p < .01$] (Appendix 5, Table 5.23). Only Fear of Animals subscale of the FSS

was significantly correlated with PPF percentage change in prepulse and pulse trials [PPF 80dB: r (15) = - .50, p = .04] (Appendix 5, Table 5.24)

There were no associations between SRM probabilities or percentage changes and age or sex (Appendix 5, Table 5.30 and 5.31).

5.2.3 Study 1 Discussion

Prepulse reactivity (PPER)

The majority of the sample exhibited prepulse reactivity and the proportions increased with increasing prepulse intensity. Few individuals exhibited spontaneous EMG activity in prepulse temporal space in pulse-alone and no stimulus 'blind' trials. Stimulus-driven response probabilities were significantly higher than spontaneous EMG activity for all prepulse intensities. The probabilities of stimulus-driven responses increased with increasing prepulse intensity, but there was no significant effect of intensity. There were no differences in probability between prepulse and pulse and prepulse-alone trials and significant positive correlations at all prepulse intensities indicated consistency in response generation across the six conditions.

In terms of response size the two trial types (prepulse and pulse and prepulse-alone) led to different prepulse intensity related outcomes. There was a linear increase in prepulse and pulse trials, but a U-shaped distribution of response amplitudes in prepulse-alone trials. Interestingly the smallest responses appeared in the 85dB prepulse-alone trials, the prepulse intensity closest to 86dB which is associated with maximal PPI. The linear increase in response size exhibited in prepulse and pulse trials indicates some stabilizing impact of the subsequent startle probe presentation, even though it is difficult to conceive how the subsequent event can affect the preceding one. There was no significant effect of prepulse intensity in either trial type though and no significant differences between prepulse and pulse and prepulse-alone trials.

The temporal characteristics of PPER presented a mixed picture. The response onsets were faster with increasing prepulse intensity in both trials types (PP and PA), but there was no effect of intensity and no differences between prepulse and pulse and prepulse-alone trials. Increasing prepulse intensity led to faster peak latencies, but the intensity effect appeared only in prepulse and pulse trials, not prepulse-alone trials. There were no differences between prepulse and pulse and

prepulse-alone trials in terms of peak latency values. The onset to peak latency values, an index of response duration, decreased with increasing prepulse intensity in prepulse and pulse trials, but increased with increasing prepulse intensity in prepulse-alone trials. There was no significant effect of prepulse intensity in either trial type though and no significant differences between the prepulse and pulse and prepulse-alone trials.

PPER and SRM

Unlike in the study with unpredictable stimulus onset (Chapter 3) PPER probability was not related to SRM probabilities and had no significant associations with SRM percentages either. However other PPER characteristics had several associations with mostly SRM probability, but also with SRM percentage change. The size of PPER in 80dB prepulse-alone trials was negatively correlated with probability of PPI following the strongest prepulse presentation and positively correlated with probability of PPF following the mid-intense prepulse presentation. This particular pair of associations resembles those found for PPER probability under the unpredictable conditions (Chapter 3). PPER onset following 80dB prepulse presentation was negatively correlated with PPF probability in the same trial type, a relationship opposite to that found in the unpredictable condition, where PPER was positively correlated with both probability and percentage change of PPF. Later onset of peak response in 90dB prepulse-alone trials was positively related to PPI probability following the weakest prepulse presentation and the strongest prepulse presentation and negatively associated with PPF probability in trials with weakest and strongest prepulses. Later onset of PPER in 85dB prepulse-alone trials was positively associated with PPI probability following 90dB prepulse presentation. Delayed onset of peak response leads to a shorter gap between the end of prepulse processing and the commencement of startle probe processing, and it would appear that individuals whose peak responses occur later are more likely to exhibit prepulse inhibition. Longer duration of responses in 85dB prepulse-alone trials was positively correlated with PPI probability and negatively correlated with PPF probability in the same trial type. Longer duration of PPER in 85dB prepulse-alone trials was also positively correlated with percentage values for PPI following 85dB and 90dB prepulse presentation, but longer duration of responses in 80dB prepulse alone trials was negatively associated with PPF probability following 90dB prepulse presentation. On the basis of these associations between PPER temporal features and

SRM it appears that under the conditions of predictable stimulus onset the extended prepulse processing in the form of later peak amplitude onset and longer onset to peak latency period exerts more influence than the frequency of PPER. It is interesting that the manipulation of the stimulus presentation in time, rather than its type or intensity, results in differential associations focused mainly on the temporal features of PPER.

Individual differences in PPER

There were few significant associations between personality factors and PPER. The probabilities of PPER were positively correlated with the Unusual Experience subscale of the O-LIFE for 80dB and 85dB prepulses in both prepulse and pulse and prepulse-alone trials. The scores on the Persistence subscale of TCI were positively correlated with response probabilities to 85dB prepulses presented alone and in prepulse and pulse trials and to 90dB prepulses presented alone. People scoring high on the Fear of Noises subscale of the FSS were more likely to respond to the weakest, 80dB prepulses presented alone.

The Unusual Experiences subscale appeared again in the associations with PPER response size where high scores were positively correlated with PPER responses to the strongest, 90dB prepulses in both trial types (PP and PA). However the scores on another O-LIFE subscale, Cognitive Disorganisation, were negatively correlated with response size in 80dB prepulse-alone trials. High scores on the Persistence subscale of the TCI had strong positive associations with PPER size in 90dB prepulse and pulse trials. The BAS Fun subscale of BISBAS was positively correlated with the size of responses in 90dB prepulse and pulse trials and 85dB prepulse-alone trials. High scores on the Fear of Animals subscale of the FSS were negatively correlated with the size of PPER in 85dB prepulse and pulse trials.

The individual differences had some associations with PPER temporal characteristics. The onset of PPER in 90dB prepulse-alone conditions was positively associated with Positive Affect and Persistence. The onset of PPER in 80dB prepulse and pulse trials was negatively correlated with scores on the BAS Reward subscale of the BISBAS. The peak latency values in prepulse-alone trials with 80dB prepulses were negatively correlated with scores on BAS Reward and Persistence subscales. High scores on the Cooperation subscale of the TCI were associated with higher peak latency values in 85dB prepulse-alone trials and high scores on the Classical Phobias subscale of the FSS had a positive association with PPER peak latency in 90dB

prepulse-alone trials, but this association has just reached significance. The onset to peak latency values in 85dB prepulse and pulse trials exhibited a strong, positive correlation with Trait Anxiety, indicating that highly trait anxious people have shorter duration of responses to the 85dB prepulses, prepulse intensity closest to 86dB which leads to maximal PPI. There were positive associations between Cognitive Disorganisation and PPER duration in 85dB prepulse-alone trials and Fear of Animals subscale and PPER duration in 90dB prepulse-alone trials. Negative associations emerged for Harm Avoidance and PPER duration in 85dB prepulse-alone trials, Persistence and PPER duration in 80dB and 90dB prepulse-alone trials, and Self-Transcendence and PPER duration in80dB prepulse-alone trials.

Amongst the demographic differences only age had some associations with PPER. Older participants had faster response onset in 80dB prepulse and pulse trials and it took them longer to reach the peak amplitude in such trials. The earlier response onset was associated with longer response duration in relation to age.

In this predictable paradigm people could anticipate the onset of the next stimulus and this anticipation might have affected the motor response rates, hence the Unusual Experience subscale, measuring individual's proneness to unusual processing of the exogenous and endogenous stimuli was associated with increased responsivity to the weakest and mid-intense stimuli, but not the strongest, least ambiguous (as compared to the background noise) stimuli of 90dB. The scores on this subscale were also associated with response sizes.

The Persistence subscale of the TCI has exhibited associations with response probabilities, sizes and temporal characteristics indicating that the behavioural propensities it measures and the neural substrates underpinning it (serotonergic activity) are prominent in generating PPER responses in the predictable paradigm.

Startle response modification: prepulse inhibition (PPI) and prepulse facilitation (PPF)

The probability and percentage values of PPI increased with increasing prepulse intensity and there was a significant effect of prepulse intensity for both. There were no associations between PPI probability and percentage change though. Whilst PPI percentage change exhibited some consistency with significant positive correlations at all prepulse intensities, this was not true for PPI probabilities.

The probability of PPF on the other hand decreased with increasing prepulse intensity, and there was a significant effect of prepulse intensity on PPF probability

but not percentage change. The probability of PPF was inversely related to PPI probability and also PPI percentage change, people with strong PPI (high percentage change) had lower chances of exhibiting PPF. Stronger PPI (high percentage change) was inversely associated with PPF, but a positive association emerged for PPI percentage change in the trials with weakest, 80dB prepulses and PPF in 85dB prepulse and pulse trials (86dB prepulses leading to optimal PPI values). This association indicates that individuals prone to efficient PPI following the weakest lead stimulus are also prone to increased maladaptive startle response modification in trials with mid-intense prepulses, which are close to prepulse intensity presumed to lead to maximal PPI.

There were very few associations between SRM and individual differences and none of the associations were particularly strong.

Summary

The short and predictable inter-trial intervals did not have a marked effect on PPER or SRM. Linear increases in the response size and probability (reflecting stimuli intensity) were exhibited for PPER in both prepulse and pulse and prepulse-alone trial types. It would appear that under the conditions of stimulus onset predictability (although this effect was limited by the presence of the 'no stimulus' trials) a stabilizing effect of the subsequent pulse presentation on response size has appeared in prepulse and pulse trials, whilst prepulses presented alone have led to responses represented by U-shaped curve.

The individual differences had limited associations with PPER, most frequent associations appearing for the Unusual Experiences subscale of O-LIFE and the Persistence subscale of the TCI.

Both PPI and PPF appeared in this paradigm and prepulse intensity effects were clearly discernible in the startle response modification probabilities. There was an inverse relationship between PPI and PPF probabilities and stronger PPI was associated with lowered probability and percentage of PPF. Individual differences did not exhibit any meaningful associations with SRM.

Limitations and recommendations

The main limitation of the presented study was the lack of true predictability, since some trials contained no stimuli '(the 'blind' trials) and the presentation of such empty trials disrupted the regular trial presentation pattern (a trial presented every 10s). There was no baseline recording with unpredictable inter-trial intervals to compare the responses obtained in the predictable paradigm. It is impossible to discern the effects of preception from the effects of possible conditioning. It would be useful to expand the paradigm by the inclusion of unpredictable condition and some measure of conditioning to ascertain each participant's susceptibility and control for its effects in calculating the possible impact of preception.

The main limitation of this study was the inclusion of 'no stimulus' blind trials, which disrupted the stimulus onset predictability. There was no baseline, unpredictable condition to compare the responses in the predictable conditions with either. Arguably, since some trials did not contain the startle probes, it was the prepulse onset only that was predictable and even that was limited by the inclusion of the 'no stimulus' trials.

5.3 Predictable inter-trial interval effects on startle response

5.3.1 Methods and materials

5.3.1.1 Participants

Twenty two participants were recruited from university employees and postgraduate students (age range: 18-24 years, M = 21, SD =1.75; 12 females, age range: 18 -24, M = 21, SD =1.61, 10 males, age range: 18-24, M = 21, SD= 2). The participants had a chance to win £100 in return for taking part. Exclusion criteria comprised self-declared suboptimal hearing, tinnitus, drug abuse or psychiatric disorder history. The times of last nicotine and alcohol consumption were recorded and hearing acuity was tested at 40dB. The participants were all non-smokers. One participant responded in less than seven pulse alone trials (ten such stimuli presented in the main session) and was excluded as non-responder and five participants had abnormal EMG waves (corrupt signal) and were also excluded. The final sample size consisted of sixteen participants (age range: 18-24, M = 20.5, SD = 1.86; 9 females, age range: 18-22, M = 20, SD = 1.42, 7 males, age range: 18-24, M = 21, SD= 2.42).

5.3.1.2 Materials

The same materials were used as in the study presented in Chapter 3.

5.3.1.3 Physiological data collection

Physiological data collection was identical to the second study presented in Chapter 3 with the following exceptions. The study consisted of two conditions: unpredictable condition and predictable condition. The unpredictable condition was identical to the protocol used in the second study in Chapter 3. The predictable condition consisted of sixteen startle probe trials presented at a fixed inter-trial interval of 15s (the mean inter-trial interval in the unpredictable condition was also 15s with variable ITI ranging from 9-21s).

Counterbalancing was used to correct for the effects of habituation and when the predictable condition preceded the unpredictable one, the first six trials were excluded from the analysis as 'habituation trials'. When the predictable condition

followed the unpredictable one, the last six trials were excluded from the analysis to avoid inclusion of excessively habituated responses.

5.3.1.4 Design

Participants completed the self-report questionnaires and were exposed to all the stimuli (with 10 presentations of each stimulus type in the unpredictable condition and 16 presentations of startle probe in the predictable condition). A mixed design was used, with sex and personality measures as the between subjects factors and the eyeblink-eliciting stimuli driven responses as the within-participant factor.

5.3.1.5 Data Scoring and Statistical Analysis

Data scoring and statistical analysis were identical to those presented in Chapter 3. The effects of brief, predictable inter-trial interval were investigated in terms startle response modification and startle response reduction. The formula for preception calculated percentage difference between the unpredictable and predictable conditions and was: [((unpredictable condition mean – predictable condition mean) / unpredictable condition mean) * 100]. A positive outcome of the formula indicated decreased startle responses, and a negative outcome increased startle responses in the condition of predictability.

5.3.2 Results

Startle responses in the unpredictable and predictable conditions

Paired samples t-test revealed lack of significant differences in the size of startle responses in the unpredictable compared to predictable condition (Appendix 5, Table 5.32). This lack of significant differences indicated absence of preception as a form of inhibition of the startle response in the predictable condition. Half of the sample exhibited increased, rather than decreased responses in the condition of predictability.

PPER and startle response change in the predictable condition

PPER has been shown to have some associations with PPI and it also exhibited some associations with startle response change in the predictable condition (preception). The probabilities of PPER in prepulse alone trials with the weakest [$r(14) = .52, p = .04$] , mid-intense [$r(14) = .60, p = .01$] , and strongest [$r(14) = .63, p < .01$] prepulses were positively correlated with startle response change

(Appendix 5, Table 5.33). It has to be borne in mind that a positive value in the percentage change means a reduction in the startle response size and a negative value indicates increased startle response sizes in the predictable condition.

The amplitudes of PPER in 85dB prepulse and pulse trials (leading to maximal PPI) were positively correlated with startle response change [$r(9) = .73, p = .01$] (Appendix 5, Table 5.34). The onset values of responses in this trial type was also positively correlated with startle response change [$r(9) = .67, p = .04$] (Appendix 5, Table 5.35). There were no significant associations between PPER peak latency values and startle response change in the predictable condition (Appendix 5, Table 5.36). The onset to peak latency values of PPER in prepulse alone trials with the strongest, 90dB prepulses were positively correlated with startle response change [$r(9) = .62, p = .04$] (Appendix 5, Table 5.37).

Startle response modification and startle response change in the predictable condition (preception)

The probability of prepulse inhibition in the trials with the most intense, 90dB prepulses was negatively correlated with startle response change in the predictable condition [$r(14) = -.70, p < .01$] (Appendix 5, Table 5.38). There was a positive correlation between preception and prepulse facilitation in the same trial type [$r(14) = .51, p = .05$] (Appendix 5, Table 5.38).

Percentage PPI in 90dB prepulse and pulse trials was also negatively correlated with preception [$r(14) = -.51, p = .05$] (Appendix 5, Table 5.39). Percentage PPF was not associated with preception.

Individual differences and startle response reduction in the predictable condition

Preception was positively correlated with Trait Anxiety [$r(14) = .57, p = .02$] but negatively correlated with Fear of Animals subscale of the FSS [$r(13) = -.65, p < .01$] (Appendix 5, Table 5.40). There were no significant associations between the demographic characteristics (sex, age) and startle response reduction in the predictable condition (Appendix 5, Table 5.41).

5.3.3 Discussion

Comparison of the startle response sizes in the unpredictable and predictable conditions demonstrated lack of significant differences indicating that preception has limited impact on startle response. It may be that the aversive stimuli do elicit

reduced or increased responses in circumstances of responses other than fast, defensive startle response.

Half of the sample exhibited increased, and the other half decreased responses in the condition of predictability. Preception can be negative (response reduction) or positive (response increase) therefore the appearance of increased, as well as decreased responses under the condition of predictability is not altogether surprising.

Increased PPER probability following the presentation of the weakest, 80dB prepulses (least frequent PPER) and strongest, 90dB prepulses (most frequent PPER) was positively related to negative preception (response reduction) in the condition of predictability. The amplitude and onset of PPER in trials with 85dB prepulses was also positively correlated with startle response reduction, and so was the duration (onset to peak latency) of PPER in trials with 90dB prepulses. It is worth noting that the increased size and later onset of the 85dB PPER was positively related to startle response inhibition in the predictable condition. Since the 85dB prepulses are closest in their intensity values to 86dB which leads to optimal PPI, the later onset and larger motor response elicited by the 85dB prepulses might indicate extended prepulse processing invading the temporal space of the subsequent startle probe.

The probability of PPI in the unpredictable condition following the presentation of 90dB prepulses was negatively correlated with startle response change in the predictable condition. PPI in the trials with 90dB prepulses is most likely to occur and reaches highest percentage values (PPI is prepulse intensity dependent), and both the probability and percentage of PPI in the trials with strongest prepulses were inversely related to startle response reduction in the condition of predictability. At the same time the probability of PPF in the trials with 90dB prepulses (least likely to occur) was positively related to startle response reduction. These results are surprising, since it could be expected that efficient PPI should be associated with efficient response reduction in the condition of predictability. On the other hand, when it comes to defensive startle response it may be that an increase, rather than decrease of the response is the adaptive strategy, since the sudden onset, intense stimuli usually alert the animal to danger, rather than reward.

Individual differences were not prominent or frequent in their associations with startle response reduction in the condition of predictability. Highly Trait Anxious individuals reduced their startle responses in the predictable condition to a greater degree, and individuals with high levels of Fear of Animals reduced their

startle responses less in the predictable condition. Highly anxious individuals may devote their attentional resources to scanning the environment and attending to their internal states, thus reducing resources available for generating the startle response. On the other hand people high on Fear of Animals may be anticipating an imminent attack, and hence increase their vigilance and the resources available to the orienting, defensive responses. No other individual differences or demographic characteristics were associated with the startle response change in the condition of predictability.

Summary

Both decreased and increased startle responses appeared under the condition of predictability and in terms of the defensive startle response, it is not entirely clear as to which response should be classified as 'adaptive'. The comparison of the startle responses in the unpredictable and predictable conditions demonstrated lack of significant differences between the two conditions. There were some surprising associations between startle response modification and startle response change in the predictable condition. Response reduction (negative preception) was inversely related to both probability and percentage of PPI following 90dB prepulse presentation and positively related to PPF in the same trial type. Highly anxious people have reduced startle responses in the condition of predictability, whereas people highly fearful of animals have their responses increased when they can predict the startle probe's onset.

Limitations and future recommendations

The half of the sample in whom the startle responses increased, rather than decreased in the predictable condition, may have led to the lack of significant differences in the startle response sizes in the unpredictable versus predictable condition. It would be worthwhile to increase the sample size and screen for the increase versus decrease in the startle response in the two conditions and to investigate in what other ways such individuals differ. The increase, versus decrease in the startle response under the conditions of predictability, could reveal differential anticipatory states (or more enduring trait-like tendencies) if investigated at the cortical level.

5.4 Predictable inter-trial effects on startle response modification

5.4.1 Methods and Materials

5.4.1.1 Participants

Twenty one participants were recruited from university employees and postgraduate students (age range: 19-23 years, M = 21, SD =1.28; 12 females, age range: 19 -23, M = 21, SD =1.41, 9 males, age range: 19-24, M = 21, SD= 1.11). The participants had a chance to win £100 in return for taking part. Exclusion criteria comprised self-declared suboptimal hearing, tinnitus, drug abuse or psychiatric disorder history. The times of last nicotine and alcohol consumption were recorded and hearing acuity was tested at 40dB. The participants were all non-smokers. One participant had unusual EMG across the entire recording session and excluded. The final sample size consisted of twenty participants (age range: 19-23, M = 21, SD = 1.30; 12 females, age range: 19-23, M = 21, SD = 1.41, 8 males, age range: 19-22, M = 21, SD= 1.18).

5.4.1.2 Materials

The same materials were used as in the study presented in Chapter 3.

5.4.1.3 Physiological data collection

Physiological data collection was identical to the second study presented in Chapter 3 with the following exceptions. The study consisted of two conditions: unpredictable condition and predictable condition. The unpredictable condition was identical to the protocol used in the second study in Chapter 3. The predictable condition consisted of ten presentations of each prepulse and pulse trials (3 prepulse intensities, 30 trials) presented at a fixed inter-trial interval of 15s (the mean inter-trial interval in the unpredictable condition was also 15s with variable ITI ranging from 9-21s). There were no pulse alone trials in the predictable condition since inclusion of the pulse alone trials would remove the predictability of prepulse presentation always leading to startle probe presentation in this condition. The exclusion of prepulse alone trials increased the predictability of startle probe onset with stable ITI and ISI values. The prepulses were presented every fifteen seconds (ITI) interval and the startle probes reliably followed the prepulses hundred and

twenty milliseconds (ISI) later. The baseline startle value for calculating startle response modification for each participant was derived from the unpredictable condition. Counterbalancing was used to correct for the effects of habituation.

5.4.1.4 Design

Participants completed the self-report questionnaires and were exposed to all the stimuli (with 10 presentations of each stimulus type in each condition). A mixed design was used, with sex and personality measures as the between subjects factors and the eyeblink-eliciting stimuli driven responses as the within-participant factor.

5.4.1.5 Data Scoring and Statistical Analysis

Data scoring and statistical analysis were identical to those presented in Chapter 3. The effects of brief, predictable inter-trial interval were investigated in terms of startle response modification. The effects of stimulus onset predictability on startle response modification were investigated by comparing the unpredictable and predictable condition. The formula for preception calculated percentage difference between the unpredictable and predictable conditions and was: [((unpredictable condition mean – predictable condition mean) / unpredictable condition mean) * 100]. A positive outcome of the formula indicated decreased SRM, and a negative outcome increased SRM in the condition of predictability.

5.4.2 Results

The effects of predictable ITI on PPER

There were no significant differences between PPER characteristics (probability, amplitude, onset, latency, onset to peak latency) in the two conditions (Appendix 5, Table 5.42 – 5.44).

The effects of predictable ITI on startle response modification

The probability of PPI following the presentation of 80dB prepulses was significantly higher in the predictable condition [$t19) = 2.34$, $p = 03$], but the probability of PPI following the presentation of 85dB prepulses was significantly lower [$t19) = - 3.58$, $p < .01$] in the condition of predictability (Appendix 5, Table 5.45). There were no significant differences in the percentage change PPI between the two conditions (Appendix 5, Table 5.46).

There were no significant differences in either probabilities or percentage changes of PPF in the two conditions (Appendix 5, Table 5.47 and Table 5.48).

PPER and preception in SRM

Preception in SRM was calculated as percentage change between the unpredictable and predictable conditions. PPER was derived from prepulse and pulse trials in the unpredictable condition, since only prepulse and pulse trials were used in the predictable condition. There were very few associations between PPER and preception in the SRM. PPER probability in 80dB prepulse and pulse trials was negatively correlated with preception for PPI in 85dB prepulse and pulse trials in the predictable condition [$r(18) = -.54$, $p = .01$]. There was a positive correlation between PPER latency in trials with 80dB prepulses and preception in PPI in 90dB [$r(13) = .56$, $p = .03$] trials. Duration of PPER in trials with 90dB prepulses was positively correlated with preception in PPF in trials with 85dB prepulses [$r(4) = .86$, $p = .03$]. All these associations are in Appendix 5, Table 5.49.

The associations between individual differences and startle response modification in the condition of predictability

Sparse associations appeared between the SRM differences and personality factors. PPI preception in trials with 80dB prepulses were positively correlated with the Fear of Noises FSS subscale [$r(16) = .51$, $p = .03$], and PPF differences in the trials with 80dB prepulses were positively correlated with Trait Anxiety [$r(9) = .63$, $p = .04$], but negatively correlated with the BAS Drive subscale of BISBAS [$r(9) = -.69$, $p = .02$] (Appendix 5, Table 5.50). There was a negative correlation between age and preception in PPF in trials with 85dB prepulses [$r(5) = -.80$, $p = .03$] (Appendix 5, Table 5.51). There were no other significant associations between the differences in startle response modification in the two session parts (two conditions) and individual differences.

5.4.3. Discussion

PPER

There were no significant differences in the PPER characteristics between the two conditions indicating that PPER is robust enough not to be affected by this paradigm variation.

PPER and startle response modification (SRM)

The probability of PPI following the presentation of the weakest 80dB prepulses (least likely to occur) was significantly elevated in the condition of predictability. On the other hand the probability of PPI in trials with 85dB prepulses was reduced under the condition of predictability. It is peculiar that the probability of inhibition following the weakest lead stimulus is increased and the probability of inhibition following the more intense prepulse, normally associated with increased probability, is decreased in the condition of predictability. At the same time there were no differences between the two conditions in terms of percentage change, and no differences for the paradoxical PPF probability or percentage change.

It has to be borne in mind that a small percentage change between the two conditions means maintenance of the startle response modification trend. A positive value means a much smaller startle response modification in the predictable part, which means reduction in the modification efficacy, and conversely a negative value indicates increase in the SRM in the condition of predictability. Increased probability of PPER in 80dB was associated with smaller preception (maintenance of SRM levels) in PPI in trials with 85dB. High latency values in 80dB trials were associated with large preception (decrease in SRM values) in PPI in trials with 90dB prepulses in the predictable condition. Duration of PPER in trials with 90dB prepulses was positively correlated with preception in PPF in trials with 85dB prepulses, indicating that longer responses to the strongest prepulses were associated with larger preception (bigger percentage change, smaller startle response modification) in the predictable condition.

Individual differences and SRM in the unpredictable and predictable condition

There were very few associations between individual differences and preception. The positive association of the Fear of Noises and preception in PPI in trials with 80dB prepulses indicated that individual fearful of noises were more likely to have a larger percentage change between the unpredictable and predictable condition. Highly Trait Anxious individuals were likely to have larger preception for PPF in 80dB trials (larger change between the unpredictable and predictable condition, lower values of PPF in the predictable condition. Individuals scoring high on the BAS Drive subscale of the BISBAS were likely to have lower preception values for PPF in trials with the weakest prepulses. Older participants were less

likely to modify the percentage rates of PPF in trials with 85dB prepulses (small preception values).

Summary

PPER was not affected by the predictable onset and no significant differences appeared for any of the PPER characteristics in the two conditions. The probability of PPI was elevated for the weakest prepulses in the predictable condition, but lowered for the mid-intensity ones. There were some associations between the PPER characteristics and SRM changes, but these were not frequent or systematic. The individual differences exhibited limited associations with preception in SRM.

Limitations and future recommendations

It would have been useful to have had a third condition, with variable inter-trials intervals (ITI) to provide a condition of true unpredictability. Inclusion of a condition with prepulse trials only (presented in a predictable manner) would have also been useful to ascertain whether the U-shaped distribution of response sizes in relation to prepulse intensity in prepulse alone trials was a paradigm artefact or a true tendency under the conditions of predictability.

5.5 General discussion

PPER

In sensorimotor gating paradigms, where prepulse presentation always leads to the startle probe presentation, a degree of predictability is present and it can be argued that if stimulus onset predictability is further elevated by limited range of inter-trial interval (ITI), or indeed a constant ITI, then favourable conditions for the appearance of preception occur. In the paradigms used in the studies presented above only one ITI was used, but in the first study not all prepulse presentations led to startle probe presentations thus reducing the predictability. In fact, it was the prepulse onset that was the most predictable aspect. Neither in this study, nor in the third study which contained only prepulse and pulse trials, with constant inter-stimulus interval (ISI) and constant ITI, was PPER affected by the predictability of stimulus onset. Preception does not exert a strong, if any effect on PPER.

In both studies in which the prepulse stimuli were presented, various PPER characteristics changed with increasing prepulse intensity, but these changes failed to reach significance.

The U-shaped distribution of response sizes in prepulse-alone trials when compared to the neat linear response size increase with increasing stimulus intensity in prepulse and pulse trials, points towards some stabilizing effect of the startle probe presentation. A similar effect appeared in Chapter 4 for prepulse intensity ratings, where the prepulses in prepulse and pulse trials were rated with a clear linear ratings increase (in line with stimulus intensity increase), but a more mixed pattern emerged for the ratings of prepulses presented alone.

Startle response modification

Both prepulse inhibition (PPI) and prepulse facilitation (PPF) appeared in the predictable conditions. There was a significant effect of prepulse intensity for probability and percentage change of PPI, but only for probability (not percentage change) of PPF. In a manner similar to the unpredictable paradigm in Chapter 3, people with strong PPI (high percentage change) were unlikely to exhibit PPF. It also emerged that individuals prone to efficient PPI following the weakest lead stimulus are prone to increased maladaptive startle response modification in trials with mid-intense prepulses (closest in intensity to 86dB presumed to lead to maximal PPI).

An effect of preception failed to appear in the second study which presented startle probes at predictable ITI. However, half of the sample exhibited increased, and half of it decreased startle responses under the condition of predictability, which leads one to conclude that perhaps the tendency to increase or decrease the startle response in the predictable condition is an individual difference akin to prepulse reactivity, whereby some individuals consistently respond to the prepulses and some do not. Preception can be assumed to have occurred in two separate ways for these two groups: negative preception for those individuals who reduced their startle responses and positive preception for those who increased them. The two opposing tendencies led to the ultimate lack of effect of predictability on startle response size in the condition of predictability.

There were some associations between PPER startle response changes in the condition of predictability in the second study. One surprising finding in the second study was a negative association between PPI elicited by the presentation of the

strongest prepulses and positive association with PPF in the same trial type, and startle response change in the condition of predictability. It may be that when it comes to defensive startle response an increase, rather than decrease of the response is adaptive since sudden onset, intense stimuli usually alert the animal to danger, rather than reward. In fact the association between high fear of animals and smaller reduction of the startle response in the predictable condition lends some support to the adaptive interpretation of the startle response increase. High Trait Anxiety was associated with greater reduction of the startle response in the condition of predictability, and bearing in mind the approach/avoidance conflict and the distance to threat as factors determining the animal's response to the potential danger, it may be that highly anxious individuals perceived the startle probes as signals of danger less imminent, than individuals scoring high on the Fear of Animals subscale.

When startle response modification was compared across the conditions of unpredictability and predictability in the third study, it emerged that the probability of PPI following the weakest lead stimulus presentation was increased, and the probability of PPI following the mid-intense lead stimuli of 85dB was decreased. Normally the probability and percentage of PPI increase in a prepulse intensity dependent manner, so this outcome highlights an impact of the predictable stimulus onset on startle response inhibition.

In the third study some associations emerged for PPER and SRM, but none of these occurred for PPER and SRM in the same trial type, therefore the impact of these associations on the startle modification process can be dismissed as at best limited.

Individual differences

Personality factors had very limited associations with either startle response modification in the predictable condition, preception in startle responses or startle response modification difference between the unpredictable and predictable conditions. There were some associations with PPER in the first study, with Unusual Experience and Persistence emerging as personality factors correlated with various aspects of PPER. People scoring high on the Fear of Noises were more likely to respond to the weakest prepulses in the first study and more likely to have a greater change of PPI in trials with the weakest prepulses from the unpredictable to the predictable condition.

Summary

Stimulus onset predictability had limited effects on any dimension of PPER. Both PPI and PPF appeared in the predictable onset condition and prepulse intensity had significant effects on probabilities of both types of SRM, but only on PPI percentage change. PPI following the weakest prepulse presentation was more likely in the predictable condition, whereas PPI following mid-intense prepulses was less likely, yet at the same time there were no significant differences in terms of percentage change at any prepulse intensity across the two conditions. There were significant negative associations between PPI probability and PPF probability, and PPI and PPF percentage changes, but not for PPI in the trials with the weakest stimuli (lowest PPI values) and PPF in trials with mid-intense stimuli (leading to maximal PPI). PPF remained stable across the two conditions in terms of both probability and percentage change. Strong PPI (large percentage change) was associated with less likelihood of PPF. The associations between PPER and SRM in the predictable conditions were different from those found in the unpredictable paradigm. Extended prepulse processing, rather than probability of PPER, was significantly and positively associated with PPI probability and percentage change. PPI was not enhanced by negative preception and startle probes with predictable onset were equally often inhibited as facilitated under the condition of predictability. Individual differences had limited effect on either PPER or SRM.

Limitations and future recommendations

The limitations of each study are listed above. In general terms it is difficult to dissociate the effects of stimulus onset predictability from the effects of startle response modification. The best approach would be to have a large sample, subdivided into inhibitors and facilitators according to their startle response modification in the condition of predictable startle probe onset. Their startle response modification in the condition of predictability could then be more easily dissected to attribute the modulating effects to either startle response modification (in terms of PPI or PPF) or preception.

6 General Discussion

The main purpose of the presented thesis was to probe different aspects of prepulse reactivity (PPER) in healthy humans. The relationships between prepulse reactivity and startle response modification were of special interest, though the impact of individual differences on prepulse reactivity and startle response modification was also important.

The participants in the presented studies were exposed to different types of paradigm designed to replicate aspects of the relevant published research and to extend the research question beyond startle response modification per se. The participants took part in sessions with uninstructed paradigm and unpredictable stimulus onset, in a session combining uninstructed paradigm with stimulus intensity ratings, and in sessions with predictable inter-trial intervals and stimulus onset. The intensity ratings constituted a proxy for the response size on the level of conscious processing. Some things were in common in the outcomes of all these paradigms: robust prepulse reactivity appeared, startle response modification also appeared (classical prepulse inhibition (PPI) and paradoxical prepulse facilitation (PPF)), and associations between prepulse reactivity and startle response modification were present. The individual differences yielded mixed results, and claims of their strong associations with the physiological responses cannot be made on the basis of the presented data. Nevertheless some of the associations between individual differences, especially anxiety and fear, were noteworthy and are reviewed below.

PPER

The healthy humans constituting the samples in the presented studies exhibited quantifiable, intensity dependent prepulse-elicited responses. Individuals differed in their propensity towards such motor responses elicited by the weak lead stimuli and tended to consistently either respond, or not respond to the prepulses. The motor responses elicited by the prepulse presentation were significantly different from spontaneous EMG activity and their probability and size increased with increasing stimulus intensity. Increased prepulse intensity also affected the temporal PPER characteristics causing earlier onset and peak latency, but not affecting the onset to peak latency values (an index of response duration). PPER characteristics were similar across different trial types, including extensive similarities between prepulse and pulse and prepulse-alone trials. PPER properties (probabilities, sizes,

temporal characteristics) held across the different paradigms, including predictable stimulus onset.

In the first experimental chapter (Chapter 3) the hypothesis that the subsequent startle probe presentation affects PPER was tested by comparing the PPER in prepulse and pulse and prepulse-alone trials, as well as the spontaneous EMG activity in the PPER temporal window, in trials with and without startle probes. The results demonstrated no effect of the subsequent startle probe presentation, but this was not true for the subsequently conducted investigations probing conscious stimulus processing and the effects of predictable stimulus onset. The subsequent startle probe presentation certainly led to significant differences in the conscious perception of prepulse intensity, with prepulses followed by startle probes being judged as significantly louder than identical intensity stimuli presented alone. Prepulses followed by startle probe presentation were intensity rated in a more consistent manner than those presented alone. The subsequent startle probe presentation also lowered prepulse detection rates. However, the ratings were provided after each trial, and so after the startle probe presentation, thus the participants processed both stimuli. Prepulse-elicited motor responses on the other hand occurred prior to the startle probe onset, therefore these differential findings are complementing, not contradicting each other. In the predictable paradigm the size of prepulse-elicited responses increased with increasing prepulse intensity in prepulse and pulse trials, but had a U-shaped distribution in the prepulse-alone trials implying some impact of the subsequent startle probe presentation. The smallest responses occurred to the mid-intense prepulses presented alone, prepulses of this intensity lead to maximal PPI, but again, this difference occurred under very specific circumstances (predictable stimulus onset) and it is not clear to what degree it should be attributed to the startle probe onset anticipation rather than the actual startle probe presentation.

PPER was also investigated in terms of relationships between conscious stimuli processing (sensory processing) and stimulus-driven motor responses (sensory-driven motor responses). Prepulse stimuli were reliably detected at all prepulse intensities and increased detection was associated with lower prepulse intensity ratings. Increased prepulse detection was associated with larger size of PPER, but prepulse detection was not directly related to prepulse-elicited responses or startle response modification, a finding concurring with published studies (Postma, et al., 2001).

SRM

All participants in all the studies exhibited prepulse inhibition. In each study increased, rather than decreased startle responses following prepulse presentation were also present and this increase was dubbed 'paradoxical' prepulse facilitation (PPF) since it appeared under the conditions presumed to lead to maximal PPI. This startle response increase cannot be attributed to positive preception (described in Chapter 5) since despite the stable inter-stimulus interval of 140ms in all the studies, not all prepulse presentations reliably led to startle probe presentation. Hence the startle response increase has to be attributed to a mechanism other than stimulus onset anticipation.

Classical prepulse inhibition increases in probability and percentage change (startle response modification degree) with increasing prepulse intensity, and the opposite was true for the probability, but not percentage change of the paradoxical PPF. Whereas PPI exhibited systematic associations across different prepulse intensities, PPF was characterised by lack of systematic associations amongst its characteristics (probability and percentage change). High PPI percentage values were inversely related to PPF probability indicating that efficient sensorimotor gating (as indexed by PPI) decreases the risk of the unusually increased startle responses in prepulse and pulse trials.

Both classical PPI and paradoxical PPF appeared for the perceived stimulus intensity ratings, but neither of these perceived stimulus intensity (PSI) ratings modifications followed the intensity dependent features found in the recording of the modified startle responses. Even though the weakest prepulses led to the least inhibited startle probe intensity ratings, there was no intensity effect for the probability or percentage of PPIPSI. Increased prepulse intensity led to decreased likelihood of PPFPSI. The relationships between perceived stimulus intensity and its modification and startle response modification were not straightforward. Inhibition (or facilitation) of the startle response was not always associated with inhibition (or facilitation) of the perceived stimulus intensity and certainly no one to one mapping of the physiological response versus conscious stimulus processing could be derived from the presented data set. Directing attention towards the stimuli (for the purposes of intensity rating) had no significant effect on startle response modification.

Under the conditions of onset predictability a complex set of results emerged. In a session where all trial types were presented at a stable inter-trial interval robust PPI, increasing with increasing prepulse intensity, appeared. The features of SRM in this paradigm closely mimicked SRM features found in the uninstructed paradigm used in the first two studies. PPI probability and percentage change increased with increasing prepulse intensity, and paradoxical PPF probability, but not percentage change, decreased with increasing prepulse intensity. Also stronger PPI was inversely associated with the chances of exhibiting PPF. However, individuals with efficient PPI following the weakest lead stimulus were prone to PPF in trials with mid-intense prepulses, so it may be that under the conditions of predictability response increase is a type of startle response modification as adaptive as response decrease.

The need to keep an open mind on the issue of classifying startle response decreases as the only adaptive type of response (reflecting efficient sensorimotor gating, and a well functioning organism) is further strengthened by the findings from the study exploring the effects of onset predictability on startle probes presented alone. It turned out that half of the sample increased, rather than decreased their startle responses under the conditions of predictability. Preception, the change in physiological response under the conditions of aversive stimulus predictability, can be negative (reduction) or positive (increase). What is more both probability and percentage change of PPI in trials with strongest prepulses (leading to highest PPI values as compared to the weaker lead stimuli) was inversely related to startle response reduction under the conditions of predictability. Yet the likelihood of PPF in such trials (very unlikely to occur) was positively associated with the startle response reduction. This complex pattern of results points towards the possibility that just as the sex differences in males and females were demonstrated to be caused by the shift in females towards propensity to engage PPF (Aasen, et al., 2005), so it seems that PPI is not the only possibility of adaptive startle response modification in healthy humans. The final study in the predictable stimulus onset set explored SRM under the conditions of predictability, but only prepulse and pulse trials were presented in the predictable condition. The stimulus onset predictability led to increased probability of PPI following the weakest lead stimulus presentation and decreased probability following the mid-intense prepulse presentation. There were no effects of predictability on PPF probability or percentage change. It would appear

that stimulus onset predictability increased the chances of PPI occurring in the trials with the weakest prepulses, a trial type in which the chances and percentage of PPI are normally much lower than at higher prepulse intensities.

PPER and SRM

In uninstructed paradigm extended prepulse processing (longer duration, higher onset to peak latency values) was not associated with startle response modification type, probability or percentage change. However, individuals more likely to exhibit PPER to the mid- and most intense prepulses had lowered probability of PPI, and increased probability of PPF following the weakest prepulse presentation. The lowered PPI probability in individuals reacting to prepulses concurs with published studies. Increased PPER was also associated with lower percentage values of PPI and increased percentage values of PPF. Thus PPER was associated with lower probability and percentage values of the classical PPI and increased probability and percentage values of the paradoxical PPF. A similar association appeared on the level of conscious stimuli processing where the weakest prepulse detection was associated with decreased probability, but increased degree of perceived stimulus intensity facilitation (PPFPSI). However, under the condition of stimulus onset predictability neither type, nor probability or percentage of SRM was associated with PPER probability.

A slightly different set of results emerged in the studies with predictable stimulus onset. In the first study which used all trial types presented in the uninstructed, unpredictable paradigm (Chapter 3), but presented at a predictable, stable inter-trial interval, PPER probability exhibited no associations with type, probability or percentage change of SRM. At the same time size and temporal characteristics of PPER were related to SRM. The size of PPER in trials with the weakest lead stimuli was negatively associated with PPI probability following the strongest prepulse presentation, an association mimicking the negative correlation between PPER to the weakest prepulses and probability of PPI following the strongest prepulse presentation found in the published studies (Dahmen & Corr, 2004) and in Chapter 3. Under the conditions of predictability the temporal features of PPER, reflecting the timescale of prepulse processing, had closer associations with SRM than the frequency of the prepulse-elicited responses.

In terms of startle response modification in response to startle probes with predictable onset increased PPER probability (in trials with the weakest and the strongest prepulses) was positively related to negative preception of the startle responses. Other PPER characteristics (amplitude and onset for 85dB PPER, and onset to peak latency values for 90dB PPER) were also positively associated with negative preception. PPER probability did not exhibit significant associations with SRM in the first study in the predictable stimulus onset studies set and the associations between the temporal PPER characteristics and SRM did not reach significance in the uninstructed, unpredictable paradigm. It would appear that PPER measured in the condition of unpredictability is related to reduction of the startle response under the conditions of predictability. This relationship between PPER and startle response reduction for startle probes with predictable onset was positive at all PPER dimensions, which is quite different from the negative association appearing for PPER and PPI in the uninstructed, unpredictable paradigm presented in Chapter 3.

The third study in the predictable stimulus onset set only employed prepulse and pulse trials to probe SRM differences induced by the stimulus onset predictability and under these conditions increased probability of 80dB PPER was associated with smaller change in PPI percentage following 85dB prepulse presentation. Again the temporal PPER features were important in this context, since high peak latency for 80dB PPER was associated with larger decrease in PPI values following 90dB prepulse presentation in the predictable condition and PPER duration in 90dB prepulse and pulse trials was positively related to decrease in PPF percentage change in trials with 85dB prepulses in the predictable condition.

Individual differences, prepulse reactivity and startle response modification

Individual differences and PPER

Both dopaminergic and serotonergic receptor polymorphisms are implicated in schizophrenia (see Chapter 1) and heritability of PPI has also been demonstrated (see Chapter 1). No analysis of heritability or associated genetic differences have been conducted to date in relation to PPER, but since PPER is affected by dopamine agonists (Yee, Russig, et al., 2004) and is lowered in schizophrenics (Csomor, et al., 2009), a group known to suffer from both dopaminergic and serotonergic system

abnormalities, it can be speculated that genetic differences affecting PPI may also play a role in PPER.

PPER probability

The first study presented in this thesis used uninstructed and unpredictable paradigm. Under such conditions PPER probability had limited associations with personality factors. Novelty Seeking, the TCI subscale reflecting dopaminergic activity, was positively correlated with PPER probability for the mid-intense prepulses. Since elevated PPER is associated with lower PPI, albeit a relationship only found for the weakest prepulse-elicited PPER and PPI following the strongest prepulse presentation, this association of elevated dopaminergic activity and elevated PPER is consistent with the dopamine known to be a disruptive agent for PPI (see Chapter 1). Another subscale of the TCI, Self-Transcendence exhibited moderate negative correlations with prepulse detection, but did so more consistently across all prepulse intensities. Although no direct associations between prepulse detection and SRM have been demonstrated, nevertheless prepulse detection is to a degree a hallmark of prepulse processing on the level of conscious stimulus perception. Self-Transcendence is related to dopaminergic activity and ,extending the network of explanations related to the dopaminergic activity based personality factors, elevated dopaminergic activity as implied by the high scores on the relevant subscales, is associated with increased PPER and decreased prepulse detection. Elevated dopaminergic activity is known to compromise PPI, therefore despite the apparent lack of direct associations between prepulse detection and PPI and limited associations between PPI and PPER, some effect of both factors should be suspected.

In the unpredictable paradigm High Tissue Damage scores were negatively correlated with prepulse reactivity in trials with the weakest prepulses. It is not clear why this specific subscale would make people less likely to respond to the weakest stimuli. Tissue Damage encompasses items related to illness, injury and their associated concepts and it would be expected that if these items are relevant to the generation of PPER, then significant associations should persist across the more intense stimuli intensity.

The Unusual Experiences were positively correlated with PPER probabilities for the weakest and mid-intense prepulses in the predictable paradigm, but was negatively correlated with prepulse detection of the mid-intense prepulses. This subscale encompasses the individual's proneness to hallucinatory and delusional

experiences, and the inability to correctly attribute the origin of a state/experience to exogenous or endogenous factors is one of the problems encountered by people prone to hallucinations and misinterpretations of their sensory inputs. Therefore this opposite direction of associations for the physiological processing of the sensory information (positive association) and conscious experience of the sensory input (negative association) are complementary and not contradictory.

The Persistence subscale of the TCI was positively associated with PPER elicited by the mid-intense and strongest prepulses in the condition of stimulus onset predictability. Reward Dependence was positively associated with the weakest prepulse detection. These two subscales are both based on serotonergic activity, since Persistence was separated from the Reward Dependence during one of the TCI revisions.

PPER amplitude

High scores on the Novelty Seeking subscale were negatively associated with PPER size, but only for the weakest lead stimuli in the unpredictable paradigm. Novelty Seeking did not appear as a factor significantly associated with PPER response size in any of the other studies. However, in the predictable paradigm anther TCI subscale underpinned by dopaminergic activity, namely Self-Transcendence had moderate positive associations with PPER size in prepulse-alone trials with the weakest lead stimuli.

Cognitive Disorganisation was negatively correlated with PPER size, but for the mid-intense prepulses presented alone in the unpredictable paradigm. In the predictable condition Cognitive Disorganisation reappeared in a negative association with PPER size in the trials with the weakest stimuli. Even though the associations appeared for different prepulse intensities, they were both negative, which indicates consistency in the associations between this subscale and PPER size.

Another O-LIFE subscale, the Unusual Experiences, was positively related to the size of responses to the strongest prepulses in both prepulse and pulse and prepulse-alone trials in the predictable paradigm. The strongest prepulses reliably elicited motor responses and it is possible that these intense prepulses were perceived by people high on the Unusual Experiences as startle probes.

In the unpredictable paradigm Reward Dependence was positively associated with the size of responses to the mid-intensity prepulses presented alone. In the predictable paradigm Persistence had strong positive associations with PPER elicited

by the strongest prepulses. At the same time Reward Dependence was negatively associated with intensity ratings for the weakest prepulses in prepulse and pulse trials. The positive association between personality factors underpinned by serotonergic activity and PPER size persisted across the unpredictable and predictable paradigms, but the outcome of the conscious prepulse processing was a negative association of the perceived stimulus intensity with Reward Dependence. This pattern of associations strengthens the argument that the motor responses and the conscious stimulus processing are completely independent processes.

High fear of noises was associated with increased ratings for the mid-intense prepulses and another FSS subscale, the Fear of Animals, was negatively correlated with PPER size elicited by mid-intense prepulses in the predictable paradigm. It is perfectly logical that people fearful of noises would rate them as louder; the interesting point is that the significant association appeared for the mid-intensity stimuli only. The same intensity stimuli elicited smaller responses in people highly fearful of animals.

For conscious prepulse processing Introvertive Anhedonia was positively associated with higher ratings for the strongest prepulses in prepulse and pulse trials and Cooperativeness had strong negative associations with prepulse intensity ratings at all prepulse intensities. Introvertive Anhedonia is a factor encompassing negative emotionality, whereas Cooperativeness is a character associated with positive emotionality. The opposing direction of the associations of the perceived stimulus ratings and these two factors with their differential dominant emotionality exhibits consistency. Positive emotionality is associated with lower perceived stimulus intensity, and negative emotionality is associated with higher perceived stimulus intensity. In a manner similar to the pattern for the serotonergic activity based personality factors listed above, BAS Fun, a factor associated with positive emotionality, was positively associated with PPER size in trials with mid-intense and strongest prepulses in the predictable condition, whilst Cooperativeness had strong negative associations with perceived prepulse intensity for all prepulses.

PPER onset

The associations between the individual differences and temporal features of PPER could only be compared between the unpredictable and predictable paradigm, since the reaction time was not measured for the intensity ratings. High Positive Affect was associated with faster PPER onset to the strongest prepulses in prepulse

and pulse trials in the unpredictable paradigm, but under the conditions of stimulus onset predictability the relationship was in the opposite direction for the strongest lead stimuli presented alone (high Positive Affect, slower PPER onset).

Introvertive Anhedonia was negatively correlated with PPER onsets in prepulse-alone trials with the weakest and mid-intense lead stimuli in the unpredictable paradigm. The negative emotionality associated with Introvertive Anhedonia (factor encompassing experiences akin to depression) was associated with faster PPER onset in the unpredictable paradigm, which is not consistent with the association between high Positive Affect and faster PPER onset in the same paradigm.

Reward Dependence was positively correlated with PPER onset to the weakest and strongest prepulses presented in the unpredictable paradigm and in the predictable paradigm Persistence was positively correlated with PPER onset in prepulse-alone trials with the strongest lead stimuli. Yet again the two related personality factors exhibited consistent association pattern across the two paradigms.

PPER peak latency

In the unpredictable paradigm people high on Trait Anxiety and Harm Avoidance reached their PPER peak latency later for the weakest prepulses in prepulse and pulse trials. Individuals high on Introvertive Anhedonia had earlier onsets of the peak amplitude for the mid-intense prepulses presented alone and those high on Novelty Seeking reached the peak amplitude faster in prepulse and pulse trials with the weakest lead stimuli.

These associations did not appear in the predictable paradigm. Positive associations emerged for peak latency values and Cooperativeness and Classical Phobias. BAS Reward and the Persistence subscale of the TCI were negatively related to the peak latency in prepulse-alone trials with the weakest lead stimuli.

PPER onset to peak latency

The more interesting personality factors and PPER onset to peak latency value associations in the unpredictable paradigm included negative associations between Cognitive Disorganisation and Introvertive Anhedonia and PPER duration in prepulse and pulse trials with mid-intensity prepulses. Fear of Animals was negatively related to PPER duration in trials with the strongest lead stimuli.

Cognitive Disorganisation reappeared in the associations in the predictable paradigm, but this time it was positively related to PPER duration in prepulse-alone trials with mid-intensity prepulses. Fear of Animals was positively associated with

PPER duration in prepulse-alone trials with the strongest prepulses. Both of these are in the opposite direction of the associations found in the unpredictable paradigm. Trait Anxiety was positively related to PPER duration in prepulse and pulse trials with the mid-intensity prepulses. Harm Avoidance was negatively related to PPER duration in mid-intensity prepulse-alone trials. Self-Transcendence and Persistence were both negatively related to PPER duration in prepulse-alone trials with the weakest lead stimuli, and Persistence was also negatively related to such trials with the strongest prepulses.

Age, sex, and PPER

There were no associations between age or and PPER in the unpredictable paradigm or at the level of conscious prepulse processing. Age was associated with faster onset of responses to the weakest lead stimuli, but positively associated with peak latency indicating that the older participants had faster commencement of the prepulse-elicited response but then it took them longer to reach the peak amplitude.

Individual differences and SRM

Some personality factors were related to startle response modification across the different paradigms. Bearing in mind that startle response is a defensive response, fear and anxiety was expected to exhibit numerous associations with the startle response modification.

Three of the FSS subscales exhibited meaningful associations with startle response modification. One of these was the Classical Phobias subscale of the FSS. People with high Classical Phobia scores were more likely to inhibit their startle responses in trials with the strongest prepulses presented at unpredictable intervals, more likely to inhibit the startle responses following the weakest prepulse presentation in trials with predictable stimulus onset, and less likely to facilitate their startle responses in this same trial type (weakest prepulse, predictable onset).

It would appear that the scores on the Classical Phobias subscale were associated withy propensity towards increased inhibition under the favourable conditions of the strongest lead stimulus, and increased probability of inhibition and decreased probability of facilitation following the weakest prepulse presentation in the predictable stimulus onset context. In the unpredictable stimulus onset paradigm increased prepulse intensity was associated with increased probability of inhibition

and decreased probability of facilitation. It would appear that stimulus onset predictability in combination with high Classical Phobia scores were associated with increased efficiency of the inhibitory mechanism activated by the weakest lead stimulus presentation.

Interestingly in the context of defensive responses another subscale of the FSS has appeared across the different paradigms, the Fear of Animals. It was negatively correlated with percentage facilitation of startle responses following the weakest prepulse presentation, and also negatively correlated with startle response reduction (negative preception) in the predictable condition. It had a positive association with percentage PPIPSI in trials with the strongest prepulses. People highly fearful of animals would increase their responses less following the weakest prepulse presentation (likely to lead to facilitation), they would reduce their startle responses less in the predictable condition (less effective negative preception), but would inhibit their perception of the startle probe intensity more following the strongest prepulse presentation. High Fear of Animals was associated with reduced impact of the alternative SRM, reduced impact of preception and increased efficacy of PSI inhibition following the strongest prepulse presentation. In other words, the alternative forms of inhibition were compromised, and the conscious processing exhibited elevated inhibition in association with high fear of animals.

The last FSS subscale related to startle response modification was the Fear of Noises. High scores on this subscale were associated with increased inhibition following the weakest lead stimulus presentation in the condition of predictable stimulus onset. All the studies used the same auditory stimuli, and the startle probe was an intense burst of sound, not pleasant by any standards. This aversive stimulus was presented at a predictable interval in the final study in the predictable stimulus onset set and prepulse presentations reliably lead to the startle probe presentation in the predictable condition. It is peculiar that the Fear of Noises would be associated only with increases of the PPI at the weakest prepulse intensity, since the weakest lead stimuli result in lowest percentage inhibition. The mid-intensity prepulses were closest to the intensity associated with maximal PPI, and the strongest prepulses were shown across all paradigms to lead to efficient startle response inhibition. Therefore it makes sense that under the condition of predictability, where all the lead stimuli served as a warning of the imminent startle probe presentation, the responses to the startle probes following the weakest prepulses were inhibited more by those more

fearful of the intense startle probe sound burst. This relationship looks as if the stimulus onset predictability compensated for the lead stimulus weakness (associated with decreased probability and percentage inhibition of the subsequent startle response).

Anxiety did not exhibit associations with preception in some published studies (Taylor, 2004). However, Trait Anxiety appeared in associations with startle response modification under the condition of predictability, a context in which anxiety and the anticipatory element of the paradigm (predictable aversive stimulus onset) were expected to interact. In the second study in the predictable stimulus onset set (Chapter 5) preception (reduction in the startle responses elicited by the startle probes presented alone at predictable interval) was positively correlated with Trait Anxiety. Preception in this study was calculated as the percentage change in the startle responses between the unpredictable and predictable onset conditions, therefore a large and positive value would indicate a larger decrease of the startle response size in the predictable condition. Therefore people high on Trait Anxiety were more likely to inhibit their startle response with predictable startle probe onset. However, high Trait Anxiety was associated with increased facilitation of startle responses following the weakest prepulse presentation in the predictable condition in the final study in the predictable stimulus onset set (prepulse and pulse trials only presented in the predictable condition). Since the first study in the predictable onset set did not yield associations with Trait Anxiety, these differential directions of associations with Trait Anxiety for startle probes presented alone and startle probes preceded by lead stimulus presentation, has to be attributed to differential associations of Trait Anxiety with preception alone (no lead stimuli, predictable onset as the preception elicited factor), as opposed to its associations with preception combined with SRM (predictable stimulus onset eliciting preception and lead stimulus presentation eliciting SRM). Following this assumption the first study, combining trials with possible effects of preception alone (startle probes presented alone) and trials with possible effects of both preception and SRM (prepulse and pulse trials), was unlikely to yield associations between the physiological responses and personality factors differentially associated with the modification of these responses.

Also in the unpredictable paradigm people high on BIS had lower probability of PPI following the strongest prepulse presentation. BIS is included under the rubric of

'anxiety' even though strictly speaking it is not an equivalent of either trait of state anxiety. However, it is activated at times of conflict and nothing is as anxiogenic as the need to discern between alternative behaviours in an uncertain context with a potential threat looming closer, or further away. It has been previously demonstrated (see Chapter 2) that anxiety and BIS covers related emotional, cognitive and behavioural tendencies.

One other subscale of the BIS/BAS exhibited significant associations with startle response modification in the predictable condition. In the final study in the predictable stimulus onset set (prepulse and pulse trials only presented in the predictable condition) people high on BAS Drive facilitated their responses less in the trials with the weakest lead stimuli. Considering all the personality factors significantly associated with startle response changes together in this particular paradigm the pattern of results is consistent. Individuals fearful of noises would heed the prepulse warning, which increased the chances of startle probe onset following it and engaged negative preception which resulted in an additive inhibitory effect (regular PPI elicited by the lead stimulus presentation further augmented by negative preception elicited by the lead stimulus as the 'imminent startle probe onset' warning). The opposing directions of the associations with PPF for the startle responses following the weakest prepulse presentation and Trait Anxiety and BAS Drive are also interesting, as they imply that the chronically anxious individuals engage positive preception under the conditions of predictability, whereas people high on BAS Drive, highly active and approach motivated individuals, engaged in negative preception of their facilitated response under the condition of the aversive stimulus onset predictability.

Conscious stimulus processing was associated with personality factors mainly in the form of impact of emotionality. Negative Affect was associated with increased inhibition percentage (of the perceived stimulus intensity, PPIPSI) at all prepulse intensities and with increased percentage facilitation (PPFPSI) following the strongest prepulse presentation (which should have led to strongest inhibition). Positive Affect on the other hand was negatively associated with percentage facilitation in trials with the weakest lead stimuli. Increased emotionality, whether positive or negative is supposed to lead to compromised PPI (Corr, et al., 2002) and it is surprising to see high negative affect having two opposite effects (increase of both inhibition and facilitation in trials with the strongest prepulses) on the

modification of the conscious perception of the stimulus intensity. It is even more puzzling that it was associated with increased PSI inhibition at all intensities and yet with increased PSI facilitation in the trials with the strongest prepulses (where highest PPI would be expected). The positive associations between high positive emotionality and PPF is less surprising bearing in mind the associations between high emotionality and lower PPI reported in published studies (Corr, et al., 2002).

Apart from factors measuring the mood and affectivity per se, Reward Dependence exhibited positive associations with percentage PPIPSI in trials with the strongest prepulses and with percentage PPFPSI in the trials with the weakest lead stimuli. At first glance this pair of associations seems contradictory until one considers that the strongest prepulses lead to increased probability of PPI and the weakest prepulses to increased probability of PPF, therefore Reward Dependence is consistently associated with the percentage of the PSI modification type most likely to occur following the two lead stimuli intensities. One other TCI subscale was associated with SRM probability, but not percentage. PPIPSI following the strongest prepulse presentation was positively correlated with Cooperativeness. It is difficult to interpret why more cooperative people would be more likely to engage inhibition in these trials. Inhibition is the most common form startle response modification following the intense prepulse presentation, and to some degree the principles of response modification as the result of lead stimulus presentation were transferable between the conscious stimulus processing and the physiological responses. However, Cooperativeness is a character, and so encompasses characteristics acquired primarily experientially by the individual, rather than being predominantly a reflection of the underlying neurochemical activity (which is true for the Temperaments).

Age, sex and SRM

There were no significant associations between ageor sex and SRM in the unpredictable stimulus onset paradigm. In conscious stimulus processing age was negatively associated with the degree of facilitation in trials with the mid-intense prepulses. Older participants had lower percentage values of the PPF in prepulse and pulse trials with the mid-intense lead stimuli. There were no other associations between any of the demographic characteristics and PSI modification (type, probability, percentage).

In the predicable stimulus onset set older participants facilitated their startle responses less under the condition of stimulus onset predictability in trials with the mid-intense lead stimuli. However there were no significant associations between the demographic characteristics (sex, age) and startle response reduction in the predictable condition for startle responses elicited by startle probes presented alone. The study which presented startle probes alone at a predictable interval yielded unambiguous indication of the limited impact of preception on startle responses.

The lack of the effect of sex on PPI in the unpredictable paradigm is at odds with the published studies (see Chapter 1) the majority of which report sex differences in the uninstructed paradigm. It can be attributed to sample characteristics, since the samples contained both males and females of varying ages. In females both the menstrual cycle status and their age (in relation to menopausal changes) affect the levels of PPI and PPF (Kumari, Aasen, et al., 2008).

Summary

PPER was significantly different form spontaneous EMG and was undoubtedly a stimulus-driven response in healthy humans. Some individual differences were associated with PPER but an overwhelming breadth of associations with personality factors relevant to sensorimotor gating (dopaminergic and serotonergic activity) or defensive responses (BIS, anxiety and fear) failed to materialise.

Significant associations between PPER and SRM have appeared with major differences in the associations patterns across the different paradigms. In the unpredictable paradigm PPER probability was the crucial characteristic displaying negative associations with PPI, a finding confirming published reports of such association. In the predictable paradigm the size and temporal features of PPER, and not probability, exhibited numerous associations with SRM. PPER was not affected by conscious stimuli processing in a paradigm with concurrent stimuli intensity judging and EMG response recording. PPER was not affected by predictable stimulus onset.

PPER characteristics were associated with numerous personality factors. Personality factors assumed to relate to dopaminergic activity exhibited few significant associations with PPER. On the other hand personality factors presumed to reflect serotonergic activity appeared in significant associations across the

different paradigms. Fear and anxiety appeared in the context of PPER, but more of the associations seemed to fall broadly into the categories of positive and negative emotionality propensities. Cognitive Disorganisation and Unusual Experiences were related to a variety of PPER characteristics. Both cognitive difficulties and dysfunctions in processing information are hallmarks of a number of neuropsychiatric disorders and sensorimotor gating deficits are pronounces in both the patients and their relatives (see Chapter 1). The associations between these two personality characteristics and PPER features in healthy samples demonstrate the value of PPER as a research question in both clinical and healthy samples, since PPER has been shown to be different in healthy and both medicated and unmedicated schizophrenics (Csomor, et al., 2009).

SRM exhibited two forms consistently appearing in all the paradigms presented in this thesis: classical PPI and paradoxical PPF. These two forms of SRM also appeared at the level of conscious stimuli processing as PPI and PPF of perceived stimulus intensity (PPIPSI and PPFPSI). Efficient PPI (high PPI percentage) was associated with diminished probability of the paradoxical PPF (in unpredictable and predictable paradigms). Predictable stimulus onset had numerous effects on startle response modification and startle response change as the result of preception.

Individual differences had limited associations with SRM in the traditional, unpredictable stimulus onset paradigm. Amongst the individual differences fear and anxiety were prominent in their associations with startle response changes under the condition of stimulus onset predictability. Classical Phobias, Fear of Animals and Fear of Noises had significant associations with startle response modification and preception in the predictable condition, as did Trait Anxiety. Elevated emotionality, in the form of high Negative or Positive Affect were associated with elevated inhibition and decreased facilitation of the PSI, a direction opposite to what would be expected for PPI, which is reduced in the context of high emotionality (regardless of its valence). This particular association further strengthened the notion that PSI is a paradigm complementary to, but not an alternative to the traditional EMG recording.

The lack of associations between SRM and affective characterisitics, be it trait or state, in terms of personality factors, could be due to the effect of measuring the startle response on the left side only. Some studies report the laterality effect

(Cadenhead, et al., 2000) whilst others purport no differences(Blumenthal, et al., 2005)

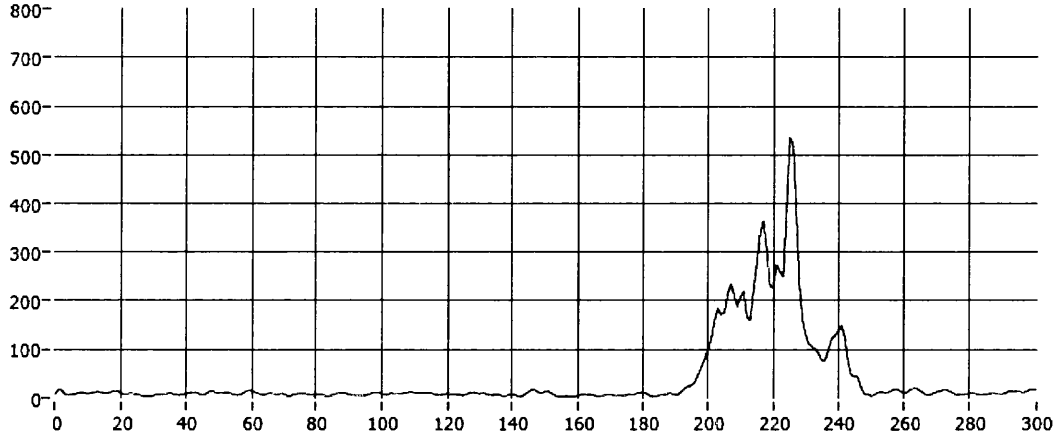
General Conclusion

PPER cannot be ignored in sensorimotor gating research. Individual propensity towards prepulse reactivity exhibits systematic associations with the subsequent startle response modification. PPER appears across different paradigms, proving it is a robust phenomenon. PPER appears on the level of motor response and on the level of conscious processing (as prepulse detection and perceived stimulus intensity).

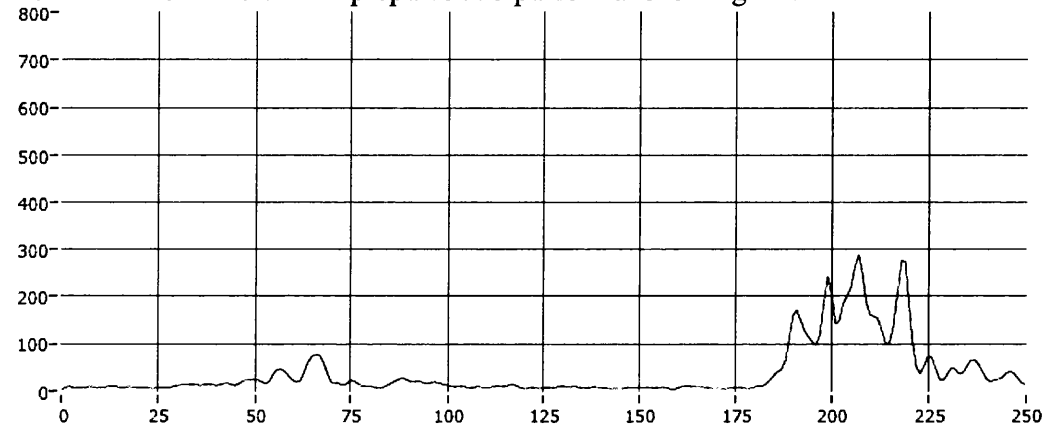
Individual differences have limited associations with PPER or SRM in the traditional paradigm with unpredictable stimulus onset, however, under the condition of stimulus onset predictability fear and anxiety come to the front, and in conscious stimulus processing few personality factors are related to the motor responses (PPER and startle responses) and their modification. Overall personality factors related to dopaminergic activity have very limited associations with PPER or SRM, but personality factors encompassing fear, anxiety and malfunctions of sensory information processing are more prominent in their relations with the physiological responses and conscious stimulus processing.

Appendix 1

Normal EMG waveform in pulse-alone trial (startle reaction)



Normal EMG waveform in prepulse and pulse trial showing PPI



Appendix 2

Part 1 Tables

Table 2.1 Stimuli types used in all the studies, the level above continuous background noise noted in the brackets

Trial Type	Prepulse Intensity	ISI (Inter-Stimulus Interval)	Pulse Intensity (Startle Probe)
Pulse Alone	70dB (0dB)	120ms	115dB (45dB)
Prepulse & Pulse	80dB (10dB)	120ms	115dB (45dB)
Prepulse & Pulse	85dB (15dB)	120ms	115dB (45dB)
Prepulse & Pulse	90dB (20dB)	120ms	115dB (45dB)
Prepulse Alone	80dB (10dB)	120ms	70dB (0dB)
Prepulse Alone	85dB (15dB)	120ms	70dB (0dB)
Prepulse Alone	90dB (20dB)	120ms	70dB (0dB)
'Blind' = No Prepulse & No Pulse	70dB (0dB)	120ms	70dB (0dB

Part 2 Questionnnaires

Individual differences questionnaires:

The demographic information questionnaire

The State and Trait Anxiety Inventory: STAI

The Temperament and Character Inventory: TCI

The BIS/BAS Scales: BIS/BAS

The Fear Survey Schedule: FSS

The Oxford-Liverpool Inventory of Feelings and Experiences: O-LIFE

The Positive and Negative Affect Schedule: PANAS

Participants Number:
Date:

Sex: M F

Age:

Smoker: Y N

If a smoker, at what time did you smoke your last cigarette?

How many cigarettes do you smoke per day?

When did you have your last alcoholic drink?

How often do you drink?

Please list all medication (including over the counter) that you are currently (within the last 48 hours) taking:

If female, please indicate the first day of your last period:

Spielberger Trait Anxiety 1983

A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you *generally* feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	ALMOST ALWAYS	OFTEN	SOMETIMES	ALMOST NEVER
1. I feel pleasant	(1)	(2)	(3)	(4)
2. I feel nervous and restless	(1)	(2)	(3)	(4)
3. I feel satisfied with myself	(1)	(2)	(3)	(4)
4. I wish I could be as happy as others seem to be	(1)	(2)	(3)	(4)
5. I feel like a failure	(1)	(2)	(3)	(4)
6. I feel rested	(1)	(2)	(3)	(4)
7. I am "calm, cool and collected"	(1)	(2)	(3)	(4)
8. I feel that difficulties are piling up so that I cannot overcome them	(1)	(2)	(3)	(4)
9. I worry too much over something that really doesn't matter	(1)	(2)	(3)	(4)
10. I am happy	(1)	(2)	(3)	(4)
11. I have disturbing thoughts	(1)	(2)	(3)	(4)
12. I lack self-confidence	(1)	(2)	(3)	(4)
13. I feel secure	(1)	(2)	(3)	(4)
14. I make decisions easily	(1)	(2)	(3)	(4)
15. I feel inadequate	(1)	(2)	(3)	(4)
16. I am content	(1)	(2)	(3)	(4)
17. Some unimportant thought runs through my mind and bothers me	(1)	(2)	(3)	(4)
18. I take disappointments so keenly that I can't put them out of my mind	(1)	(2)	(3)	(4)
19. I am a steady person	(1)	(2)	(3)	(4)
20. I get in a state of tension or turmoil as I think over my recent concerns and interests	(1)	(2)	(3)	(4)

The state anxiety questionnaire is the same, only 'how you generally feel' is replaced with 'how you are feeling right now'.

TCI

Below you will find statements people might use to describe their attitudes, opinions, interests, and other personal feelings. Each statement can be answered TRUE or FALSE. Read the statement and decide which choice best describes you. We would like you to fill out this questionnaire on your own – to answer you only need to circle either "T" or "F" after each question. Read each statement carefully, but don't spend too much time deciding on the answer. Please answer every statement, even if you are not completely sure of the answer. Remember there are no right or wrong answers – just describe your own personal opinions and feelings.

		True	False
1.	I often try new things just for fun or thrills, even if most people think it is a waste of time.	T	F
2.	I usually am confident that everything will go well, even in situations that worry most people.	T	F
3.	I often feel that I am the victim of circumstances.	T	F
4.	I can usually accept other people as they are, even when they are very different from me.	T	F
5.	I enjoy getting revenge on people who hurt me.	T	F
6.	Often I feel that my life has little purpose or meaning.	T	F
7.	I like to find a solution to problems so that everyone comes out ahead.	T	F
8.	I could probably accomplish more than I do, but I don't see the point in pushing myself harder than is necessary to get by.	T	F
9.	I often feel tense and worried in unfamiliar situations, even when others feel that there is little to worry about.	T	F
10.	I often do things based on how I feel at the moment without thinking about how they were done in the past.	T	F
11.	I usually do things my own way – rather than giving in to the wishes of other people.	T	F
12.	I generally don't like people who have different ideas from me.	T	F
13.	I would do almost anything legal in order to become rich and famous, even if I would lose the trust of many old friends.	T	F
14.	I am much more reserved and controlled than most people.	T	F
15.	I like to discuss my experiences and feelings openly with friends instead of keeping them to myself.	T	F
16.	I have less energy and get tired more quickly than most people.	T	F
17.	I seldom feel free to choose what I want to do.	T	F
18	I often consider another person's feelings as much as my own.	T	F
19.	I often avoid meeting strangers because I lack confidence with people I do not know.	T	F
20.	I like to please other people as much as I can.	T	F
21.	I often wish that I was smarter than everyone else.	T	F
22.	I am usually so determined that I continued to work long after other people have given up.	T	F
23.	I often wait for someone else to provide a solution to my problems.	T	F
24.	I often spend money until I run out of cash or get into debt from using too much credit.	T	F
25.	Often I have unexpected flashes of insight or understanding while relaxing.	T	F
26.	I don't care very much whether other people like me or the way I do things.	T	F
27.	I usually try to get just what I want for myself because it is not possible to satisfy everyone anyway.	T	F
28.	I have no patience with people who don't accept my views.	T	F
29.	I sometimes feel so connected to nature that everything seems to be part of one living organism.	T	F
30.	When I have to meet a group of strangers, I am more shy than most people.	T	F
31.	I am more sentimental than most people.	T	F
32.	I seem to have a "sixth sense" that sometimes allows me to know what is going to happen.	T	F
33.	When someone hurts me in any way, I usually try to get even.	T	F
34.	My attitudes are determined largely by influences outside my control.	T	F

35.	I often wish I was stronger than everyone else.	T	F
36.	I like to think about things for a long time before I make a decision.	T	F
37.	I am more hard working than most people.	T	F
38.	I usually stay calm and secure in situations that most people would find physically dangerous.	T	F
39.	I do not think it is smart to help weak people who cannot help themselves.	T	F
40.	I cannot have any peace of mind if I treat other people unfairly, even if they are unfair to me.	T	F
41.	People will usually tell me how they feel.	T	F
42.	Sometimes I have felt like I was part of something with no limits or boundaries in time and space.	T	F
43.	I sometimes feel a spiritual connection to other people that I cannot explain in words.	T	F
44.	I like it when people can do whatever they want without strict rules and regulations.	T	F
45.	I would probably stay relaxed and outgoing when meeting a group of strangers, even if I were told that they are unfriendly.	T	F
46.	Usually I am more worried than most people that something might go wrong in the future.	T	F
47.	I usually think about all the facts in detail before I make a decision.	T	F
48.	I often wish I had special powers like Superman.	T	F
49.	Other people control me too much.	T	F
50.	I like to share what I have learned with other people.	T	F
51.	I am usually able to get other people to believe me, even when I know that what I am saying is exaggerated or untrue.	T	F
52.	Sometimes I have felt my life was being directed by a spiritual force greater than any human being.	T	F
53.	I have a reputation as someone who is very practical and does not act on emotion.	T	F
54.	I am strongly moved by sentimental appeals (like when asked to help crippled children).	T	F
55.	I usually push myself harder than most people do because I want to do as well as I possibly can.	T	F
56.	I have so many faults that I don't like myself very much.	T	F
57.	I have too little time to look for long term solutions to my problems.	T	F
58.	I often cannot deal with problems because I just don't know what to do.	T	F
59.	I prefer spending money than saving it.	T	F
60.	I can usually do a good job of stretching the truth to tell a funnier story or to play a joke on someone.	T	F
61.	If I am embarrassed or humiliated, I get over it very quickly.	T	F
62.	It is extremely difficult for me to adjust to changes in my usual way of doing things because I get so tense, tired, or worried.	T	F
63.	I usually demand very good practical reasons before I am willing to change my old ways of doing things.	T	F
64.	I nearly always stay relaxed and carefree, even when nearly everyone else is fearful.	T	F
65.	I find sad songs and movies pretty boring.	T	F
66.	Circumstances often force me to do things against my will.	T	F
67.	I would rather be kind than to get revenge when someone hurts me.	T	F
68.	I often become so fascinated with what I'm doing that I get lost in the moment – like I'm detached from time and place.	T	F
69.	I do not think I have a real sense of purpose for my life.	T	F
70.	I often feel tense and worried in unfamiliar situations, even when others feel there is no danger at all.	T	F
71.	I often follow my instincts, hunches or intuition without thinking through all the details.	T	F
72.	Other people often think that I am too independent because I won't do what they want.	T	F
73.	I often feel a strong spiritual or emotional connection with all the people around	T	F

	me.		
74.	I usually try to imagine myself "in other people's shows", so I can really understand them.	T	F
75.	Principles like fairness and honesty have little role in some aspects of my life.	T	F
76.	I am better at saving money than most people.	T	F
77.	Even when most people feel it is not important, I often insist on things being done in a strict and orderly way.	T	F
78.	I feel very confident and sure of myself in almost all social situations.	T	F
79.	My friends find it hard to know my feelings because I seldom tell them about my private thoughts.	T	F
80.	I like to imagine my enemies suffering.	T	F
81.	I am more energetic and tire less quickly than most people.	T	F
82.	I often stop what I am doing because I get worried, even when my friends tell me everything will go well.	T	F
83.	I often wish I was more powerful than everyone else.	T	F
84.	Members of a team rarely get their fair share.	T	F
85.	I don't go out of my way to please other people.	T	F
86.	I am not shy with strangers at all.	T	F
87.	I spend a lot of time doing things that seem necessary but not really important to me.	T	F
88.	I don't think that religious or ethical principles about what is right and wrong should have much influence on business decisions.	T	F
89.	I often try to put aside my own judgments so that I can better understand what other people are experiencing.	T	F
90.	Many of my habits make it hard for me to accomplish worthwhile goals.	T	F
91.	I have made real personal sacrifices in order to make the world a better place – like trying to prevent war, poverty and injustice.	T	F
92.	I prefer to wait for someone else to take the lead in getting things done.	T	F
93.	I usually respect the opinions of others.	T	F
94.	My behavior is strongly guided by certain goals that I have set for my life.	T	F
95.	It is usually foolish to promote the success of other people.	T	F
96.	I usually like to stay cool and detached from other people.	T	F
97.	I am more likely to cry at a sad movie than most people	T	F
98.	I recover more quickly than most people from minor illnesses or stress.	T	F
99.	I often break rules and regulations when I think I can get away with it.	T	F
100.	I need much more practice in developing good habits before I will be able to trust myself in many tempting situations.	T	F
101.	I wish other people didn't talk as much as they do.	T	F
102.	Everyone should be treated with dignity and respect, even if they seem to be unimportant or bad.	T	F
103.	I like to make quick decisions so that I can get on with what has to be done.	T	F
104.	I am usually confident that I can easily do things that most people would consider dangerous (such as driving an automobile fast on a wet or icy road).	T	F
105.	I like to explore new ways to do things.	T	F
106.	I enjoy saving money more than spending it on entertainment or thrills.	T	F
107.	I have had personal experiences in which I felt in contact with a divine and wonderful spiritual power.	T	F
108.	I have had moments of great joy in which I suddenly had a clear, deep feeling of oneness with all that exists.	T	F
109.	Most people seem more resourceful than I am.	T	F
110.	I often feel like I am a part of the spiritual force on which all life depends.	T	F
111.	Even when I am with friends, I prefer not to "open up" very much.	T	F
112.	I think my natural responses now are usually consistent with my principles and long-term goals.	T	F
113.	I believe that all life depends on some spiritual order or power that cannot be completely explained.	T	F
114.	Often when I look at an ordinary thing, something wonderful happens – I get the feeling that I am seeing it fresh for the first time.	T	F

115.	I usually feel tense and worried when I have to do something new and unfamiliar.	T	F
116.	I often push myself to the point of exhaustion or try and do more than I really can.	T	F
117.	My will power is too weak to overcome very strong temptations, even if I know I will suffer as a consequence.	T	F
118.	I hate to see anyone suffer.	T	F
119.	If I am feeling upset I usually feel better around friends than when left alone.	T	F
120.	I wish I were better looking than everyone else.	T	F
121.	I love the blooming of flowers in the spring as much as seeing an old friend again.	T	F
122.	I usually look at a different situation as a challenge or opportunity.	T	F
123.	People involved with me have to learn how to do things my way.	T	F
124.	I usually feel much more confident and energetic than most people, even after minor illnesses or stress.	T	F
125.	When nothing new is happening, I usually start looking for something that is thrilling or exciting.	T	F

BIS/BAS Scales

Each item of this questionnaire is a statement that a person may either agree with or disagree with. For each item, indicate how much you agree or disagree with what the item says. Please respond to all the items; do not leave any blank. Choose only one response to each statement. Please be as accurate and honest as you can be. **Respond to each item as if it were the only item.** That is, don't worry about being "consistent" in your responses. Choose from the following four response options:

1 = very true for me
2 = somewhat true for me
3 = somewhat false for me
4 = very false for me

1. A person's family is the most important thing in life. ___
2. Even if something bad is about to happen to me, I rarely experience fear or nervousness. ___
3. I go out of my way to get things I want. ___
4. When I'm doing well at something I love to keep at it. ___
5. I'm always willing to try something new if I think it will be fun. ___
6. How I dress is important to me. ___
7. When I get something I want, I feel excited and energized. ___
8. Criticism or scolding hurts me quite a bit. ___
9. When I want something I usually go all-out to get it. ___
10. I will often do things for no other reason than that they might be fun. ___
11. It's hard for me to find the time to do things such as get a haircut. ___
12. If I see a chance to get something I want I move on it right away. ___
13. I feel pretty worried or upset when I think or know somebody is angry at me. ___
14. When I see an opportunity for something I like I get excited right away. ___
15. I often act on the spur of the moment. ___
16. If I think something unpleasant is going to happen I usually get pretty "worked up." ___
17. I often wonder why people act the way they do. ___
18. When good things happen to me, it affects me strongly. ___
19. I feel worried when I think I have done poorly at something important. ___
20. I crave excitement and new sensations. ___
21. When I go after something I use a "no holds barred" approach. ___
22. I have very few fears compared to my friends. ___
23. It would excite me to win a contest. ___
24. I worry about making mistakes. ___

Fear Survey Schedule

The items on the following page refer to things and experiences that may cause **fear or other, related unpleasant feelings**. Read **each item** and decide how much you are **disturbed by it**, then mark your response according to the following numerical scale:

0 = not at all
1 = a little
2 = a fair amount
3 = much
4 = very much

For example, if ***boating*** causes you to feel no fear at all, you would write 0 in the response box next to the item. Alternatively if ***boating*** causes you much fear, you would write 3 in the response box next to the item.

Use these numbers to indicate your response to the items 0 = Not at all, 1 = A little, 2 = A fair amount, 3 = Much, 4 = Very much					
		Response			*Response*
1.	Noise of vacuum cleaners		55.	Mice or rats	
2.	Open wounds		56.	Human blood	
3.	Being alone		57.	Animal blood	
4.	Loud voices		58.	Parting from friends	
5.	Dead people		59.	Enclosed places	
6.	Speaking in public		60.	Prospects of a surgical operation	
7.	Crossing streets		61.	Feeling rejected by others	
8.	People who seem insane		62.	Journeys by airplane	
9.	Being in a strange place		63.	Medical odors	
10.	Falling		64.	Feeling disapproved of	
11.	Automobiles		65.	Harmless snakes	
12.	Being teased		66.	Cemeteries	
13.	Dentists		67.	Being ignored	
14.	Thunder		68.	Darkness	
15.	Sirens		69.	Premature heart beats (missing a beat)	
16.	Failure		70.	Nude men	
17.	Entering a room where other people are already seated		71.	Nude women	
18.	High places on land		72.	Lightning	
19.	Looking down from high buildings		73.	Doctors	
20.	Worms		74.	Crippled or deformed people	
21.	Imaginary creatures		75.	Making mistakes	
22.	Receiving injections		76.	Looking foolish	
23.	Strangers		77.	Losing control of yourself	
24.	Bats		78.	Fainting	
25.	Journeys by train		79.	Becoming nauseous	
26.	Feeling angry		80.	Harmless spiders	
27.	People in authority		81.	Being responsible for decisions	
28.	Flying insects		82.	Sight of knives or sharp objects	
29.	Seeing other people injected		83.	Thoughts of being mentally ill	
30.	Sudden noises		84.	Taking written tests	
31.	Journeys by car		85.	Being with a member of the opposite sex	
32.	Dull weather		86.	Large open spaces	
33.	Crowds		87.	Dogs	

34.	Cats		88.	Germs	
35.	One person bullying another		89.	Being seen unclothed	
36.	Tough looking people		90.	Taking medicine	
37.	Birds		91.	Becoming sexually aroused	
38.	Sight of deep water		92.	Being punished by god	
39.	Being watched working		93.	Ideas of possible homosexuality	
40.	Dead animals		94.	Being dressed unsuitably (wearing wrong clothes for the occasion)	
41.	Weapons		95.	Ministers or priests	
42.	Dirt		96.	Hurting the feelings of others	
43.	Journeys by bus		97.	Kissing	
44.	Crawling insects		98.	Undertakers	
45.	Seeing a fight		99.	Police	
46.	Ugly people		100.	Fish	
47.	Fire		101.	Masturbation	
48.	Sick people		102.	Leaving home	
49.	Being criticized		103.	Physical examinations	
50.	Strange shapes		104.	Marriage	
51.	Being touched by others		105.	Insecticides	
52.	Being in an elevator		106.	Vomiting	
53.	Witnessing surgical operations		107.	Responsibility (being in charge)	
54.	Angry people		108.	Hospitals	

O-LIFE

Please Read the Instructions Before Continuing:

This questionnaire contains questions that may relate to your thoughts, feelings, experiences and preferences. There are no right or wrong answers or trick questions so please be as honest as possible.

For each question place a circle around either the "YES" or the "NO" in the correctly numbered space. Do not spend too much time deliberating any question but put the answer closest to your own. Please do not discuss the questionnaire with anyone who may also complete it as this may affected their answers. It is best completed in private, without the need to hurry.

1	Do you prefer reading to meeting people?	*YES*	*NO*
2	Do you often hesitate when you are going to say something in a group of people whom you more or less know?	*YES*	*NO*
3	Are you always willing to admit it when you have made a mistake?	*YES*	*NO*
4	Do you sometimes put off until tomorrow what you ought to do today?	*YES*	*NO*
5	Do you often overindulge in alcohol and food?	*YES*	*NO*
6	Do you often feel that people have it in for you?	*YES*	*NO*
7	Are the sounds you hear in your daydreams really clear and distinct?	*YES*	*NO*
8	Do you enjoy many different kinds of play and recreation?	*YES*	*NO*
9	Do your thoughts sometimes seem as real as actual events in your life?	*YES*	*NO*
10	Do you have many different hobbies?	*YES*	*NO*
11	Does it often happen that nearly every thought immediately and automatically suggests an enormous number of ideas?	*YES*	*NO*
12	When in a group of people do you usually prefer to let someone else be the centre of attention?	*YES*	*NO*
13	If you say you will do something do you always keep your promise no matter how inconvenient it might be?	*YES*	*NO*
14	Do you frequently have difficulty in starting to do things?	*YES*	*NO*
15	Has dancing or the idea of it always seemed dull to you?	*YES*	*NO*
16	When you catch a train do you often arrive at the last minute?	*YES*	*NO*

17	Is trying new food something you have always enjoyed?	*YES*	*NO*
18	Do you always wash before a meal?	*YES*	*NO*
19	Do you believe in telepathy?	*YES*	*NO*
20	Do you often change between intense liking and disliking of the same person?	*YES*	*NO*
21	Have you ever cheated at a game?	*YES*	*NO*
22	Are there very few things that you have ever really enjoyed doing?	*YES*	*NO*
23	Would you call yourself happy-go-lucky?	*YES*	*NO*
24	Do you at times have an urge to do something harmful or shocking?	*YES*	*NO*
25	Do you often worry about things you should not have done or said?	*YES*	*NO*
26	Are your thoughts sometimes so strong that you can almost hear them?	*YES*	*NO*
27	Do you usually take the initiative in making new friends?	*YES*	*NO*
28	Do your thoughts ever stop suddenly causing you to interrupt what you are saying?	*YES*	*NO*
29	Are you usually in an average sort of mood, not too high and not to low?	*YES*	*NO*
30	Do you often take on more activities than you have time for?	*YES*	*NO*
31	Would you take drugs which may have strange or dangerous effects?	*YES*	*NO*
32	Do you think you could learn to read other's minds if you wanted to?	*YES*	*NO*
33	When in a crowded room, do you often have difficulty in following a conversation?	*YES*	*NO*
34	No matter how hard you try to concentrate do unrelated thoughts always creep into your mind?	*YES*	*NO*
35	Are you easily hurt when people find fault with you or the work you do?	*YES*	*NO*
36	Do you stop to think things over before doing anything?	*YES*	*NO*
37	Have you ever felt that you have special, almost magical powers?	*YES*	*NO*
38	Are you much too independent to really get involved with other people?	*YES*	*NO*
39	Do you ever get nervous when someone is walking behind you?	*YES*	*NO*

40	Do ideas and insights sometimes come to you so fast that you cannot express them all?	*YES*	*NO*
41	Do you easily lose your courage when criticized or failing in something?	*YES*	*NO*
42	Can some people make you aware of them just by thinking about you?	*YES*	*NO*
43	Does a passing thought ever seem so real it frightens you?	*YES*	*NO*
44	Do you always practice what you preach?	*YES*	*NO*
45	Would you dodge paying taxes if you were sure you could never be found out?	*YES*	*NO*
46	Have you ever blamed someone for doing something you know was really your fault?	*YES*	*NO*
47	Are you a person whose mood goes up and down easily?	*YES*	*NO*
48	Does your voice ever seem distant or faraway?	*YES*	*NO*
49	Do you think having close friends is not as important as some people say?	*YES*	*NO*
50	Do you like doing things in which you have to act quickly?	*YES*	*NO*
51	Are you rather lively?	*YES*	*NO*
52	Do you feel at times that people are talking about you?	*YES*	*NO*
53	Are you sometimes so nervous that you are 'blocked'?	*YES*	*NO*
54	Do you find it difficult to keep interested in the same thing for a long time?	*YES*	*NO*
55	Have you ever insisted on having your own way?	*YES*	*NO*
56	Do you dread going into a room by yourself where other people have already gathered and are talking?	*YES*	*NO*
57	Have you ever felt that were communicating with someone telepathically?	*YES*	*NO*
58	Does it often feel good to massage your muscles when they are tired or sore?	*YES*	*NO*
59	Do you sometimes feel that your accidents are caused by mysterious forces?	*YES*	*NO*

60	Do you like mixing with people?	*YES*	*NO*
61	On seeing a soft thick carpet have you sometimes had the impulse to take off your shoes and walk barefoot on it?	*YES*	*NO*
62	Can you get a party going?	*YES*	*NO*
63	Do you often have difficulties in controlling your thoughts?	*YES*	*NO*
64	Do you feel that you cannot get 'close' to other people?	*YES*	*NO*
65	Do the people in your daydreams seem so true to life that you sometimes think they are real?	*YES*	*NO*
66	Do other people think of you as being very lively?	*YES*	*NO*
67	Are people usually better off if they stay aloof from emotional involvements with people?	*YES*	*NO*
68	Have you ever broken or lost something belonging to someone else?	*YES*	*NO*
69	Are you mostly quiet when you are with other people?	*YES*	*NO*
70	Can you just being with friends make you feel good?	*YES*	*NO*
71	Do you enjoy meeting new people?	*YES*	*NO*
72	Is your hearing sometimes so sensitive that ordinary sounds become uncomfortable?	*YES*	*NO*
73	Have you often felt uncomfortable when your friends touch you?	*YES*	*NO*
74	When things are bothering you do you like to talk to other people about it?	*YES*	*NO*
75	Do you ever have the sensation that your body or a part of it is changing shape?	*YES*	*NO*
76	Do you have many friends?	*YES*	*NO*
77	Are <u>all</u> your habits good and desirable ones?	*YES*	*NO*
78	Do you tend to keep in the background on social occasions?	*YES*	*NO*
79	Have you ever taken anything (even a pin or a button) that belonged to	*YES*	*NO*

	someone else?		
80	As a child were you ever cheeky to your parents?	*YES*	*NO*
81	Would being in debt worry you?	*YES*	*NO*
82	Have you ever felt when you looked in a mirror that your face seemed different?	*YES*	*NO*
83	Do you think people spend too much time safeguarding their future with savings and insurance?	*YES*	*NO*
84	Do you believe that dreams can come true?	*YES*	*NO*
85	Do you ever have the urge to break or smash things?	*YES*	*NO*
86	Do you often feel that there is no purpose to life?	*YES*	*NO*
87	Do things sometimes feel as though they were not real?	*YES*	*NO*
88	Do you worry about awful things that might happen?	*YES*	*NO*
89	Have you ever felt the urge to injure yourself?	*YES*	*NO*
90	Would it make you nervous to play the clown in front of other people?	*YES*	*NO*
91	Do you prefer watching television to going out with other people?	*YES*	*NO*
92	Have you ever felt that you might cause something to happen just by thinking too much about it?	*YES*	*NO*
93	Have you had very little fun from physical activities like walking, swimming, or sports?	*YES*	*NO*
94	Have you ever been late for an appointment or work?	*YES*	*NO*
95	Have you ever said anything bad or nasty about anyone?	*YES*	*NO*
96	Do you feel so good at controlling others that it sometimes scares you?	*YES*	*NO*
97	Are you easily distracted from work by daydreams?	*YES*	*NO*
98	Are you easily confused if too much happens at the same time?	*YES*	*NO*
99	Do you ever have a sense of vague danger or sudden dread for reasons that you do not understand?	*YES*	*NO*

100	Is it true that your relationships with other people never get very intense?	*YES*	*NO*
101	Do you feel that you have to be on your guard even with your friends?	*YES*	*NO*
102	Have you sometimes had the feeling of gaining or losing energy when certain people look at you or touch you?	*YES*	*NO*
103	When coming into a new situation have you ever felt strongly that it was a repeat of something that had happened before?	*YES*	*NO*
104	Do you worry too long after an embarrassing experience?	*YES*	*NO*
105	Do you love having your back massaged?	*YES*	*NO*
106	Do you consider yourself to be pretty much an average kind of person?	*YES*	*NO*
107	Have you ever taken advantage of someone?	*YES*	*NO*
108	Would you like other people to be afraid of you?	*YES*	*NO*
109	Have you ever thought you heard people talking only to discover that it was in fact some nondescript noise?	*YES*	*NO*
110	Have you occasionally felt as though your body did not exist?	*YES*	*NO*
111	Do you often feel lonely?	*YES*	*NO*
112	Do you often have an urge to hit someone?	*YES*	*NO*
113	Do you often experience an overwhelming sense of emptiness?	*YES*	*NO*
114	On occasion, have you seen a person's face in front of you when no one was in fact there?	*YES*	*NO*
115	Do you feel it is safer to trust nobody?	*YES*	*NO*
116	Is it fun to sing with other people?	*YES*	*NO*
117	Do you often have days when indoor lights seem so bright that they bother your eyes?	*YES*	*NO*
118	Have you wondered whether the spirits of the dead can influence the living?	*YES*	*NO*
119	Do people who try to get to know you better usually give up after a while?	*YES*	*NO*
120	Do you often feel 'fed up'?	*YES*	*NO*

121	Have you felt as though your head or limbs were somehow not your own?	*YES*	*NO*
122	Do you ever become oversensitive to light or noise?	*YES*	*NO*
123	When you look in the mirror does your face sometimes seem quite different from usual?	*YES*	*NO*
124	Do you nearly always have a 'ready answer' when people talk to you?	*YES*	*NO*
125	Do people who drive carefully annoy you?	*YES*	*NO*
126	Do you like telling jokes and funny stories to your friends?	*YES*	*NO*
127	Do you sometimes boast a little?	*YES*	*NO*
128	Are you very hurt by criticism?	*YES*	*NO*
129	Do you feel lonely most of the time, even when you're with people?	*YES*	*NO*
130	Would you call yourself a nervous person?	*YES*	*NO*
131	Can you usually let yourself go and enjoy yourself at a lively party?	*YES*	*NO*
132	Do you ever feel that your thoughts don't belong to you?	*YES*	*NO*
133	Do you ever suddenly feel distracted by distant sounds that you are not normally aware of?	*YES*	*NO*
134	As a child, did you do as you were told immediately and without grumbling?	*YES*	*NO*
135	Do you sometimes talk about things you know nothing about?	*YES*	*NO*
136	When you are worried or anxious do you have trouble with your bowels?	*YES*	*NO*
137	When in the dark do you often see shapes and forms even though there's nothing there?	*YES*	*NO*
138	Can you easily get some life into a rather dull party?	*YES*	*NO*
139	Do you often have vivid dreams that disturb your sleep?	*YES*	*NO*
140	Do you like plenty of bustle and excitement around you?	*YES*	*NO*

141	Have you sometimes sensed an evil presence around you, even though you could not see it?	*YES*	*NO*
142	Is it hard for you to make decisions?	*YES*	*NO*
143	Do you find the bright lights of a city exciting to look at?	*YES*	*NO*
144	Does your sense of smell sometimes become unusually strong?	*YES*	*NO*
145	Do you usually have very little desire to buy new kinds of food?	*YES*	*NO*
146	Are you often bothered by the feeling that people are watching you?	*YES*	*NO*
147	Do you ever feel that your speech is difficult to understand because the words are all mixed up and don't make sense?	*YES*	*NO*
148	Do you often feel like doing the opposite of what other people suggest, even though you know they are right?	*YES*	*NO*
149	Do you like going out a lot?	*YES*	*NO*
150	Do you feel very close to your friends?	*YES*	*NO*
151	Are you sometimes sure that other people can tell what you're thinking?	*YES*	*NO*
152	Do you ever feel sure that something is about to happen, even though there does not seem to be any reason for thinking that?	*YES*	*NO*
153	Do you often feel the impulse to spend money which you know you can't afford?	*YES*	*NO*
154	Are you easily distracted when you read or talk to someone?	*YES*	*NO*
155	Are you a talkative person?	*YES*	*NO*
156	Were you ever greedy by helping yourself to more than your share of anything?	*YES*	*NO*
157	Do everyday things sometimes seem unusually large or small?	*YES*	*NO*
158	Do you feel that making new friends isn't worth the energy it takes?	*YES*	*NO*
159	Have you ever taken the praise for something you knew someone else had really done?	*YES*	*NO*

PANAS

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word.
Indicate to what extent you **feel** this way **right now,** that is, at **the present moment.** Use the following scale to record your answers:

1 – very slightly or not at all

2 – a little

3 - moderately

4 – quite a lot

5 – extremely

Interested	
Distressed	
Excited	
Upset	
Strong	
Guilty	
Scared	
Hostile	
Enthusiastic	
Proud	
Irritable	
Alert	
Ashamed	
Inspired	
Nervous	
Determined	
Attentive	
Jittery	
Active	
Afraid	

Appendix 3

Part 1 Figures

Figure 3.1 PPER probabilities [SP = startle probe alone; PP = prepulse and pulse trial; PA = prepulse alone trial; PPx, x = lead stimulus dB]

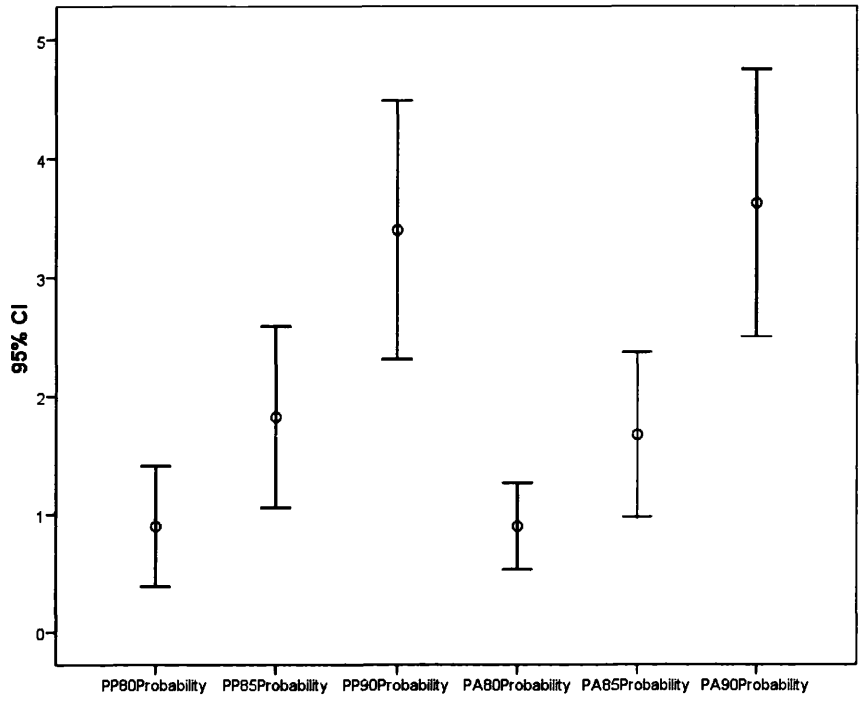


Figure 3.2 PPER amplitudes

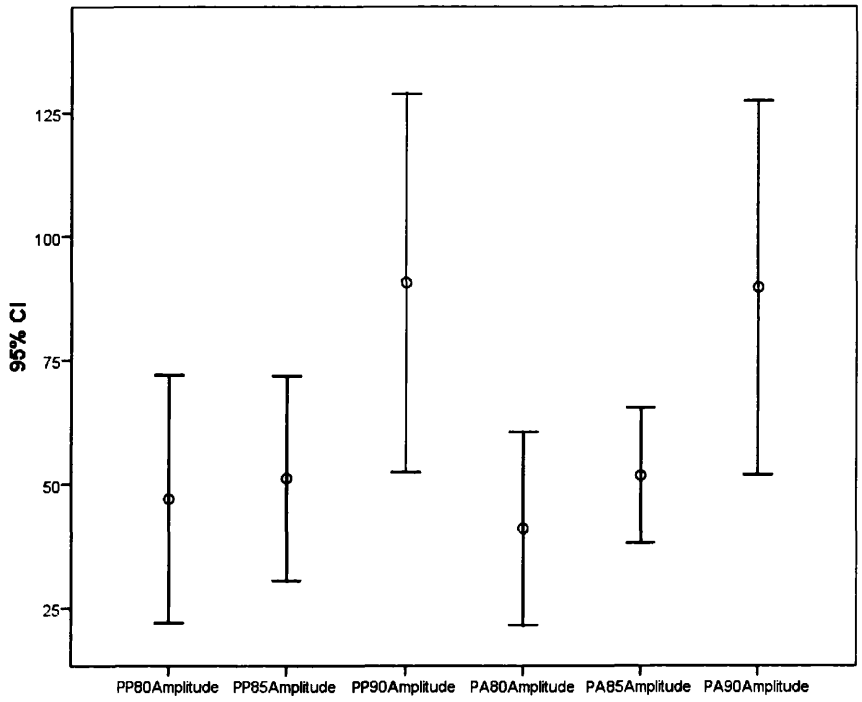


Figure 3.3 PPER onsets

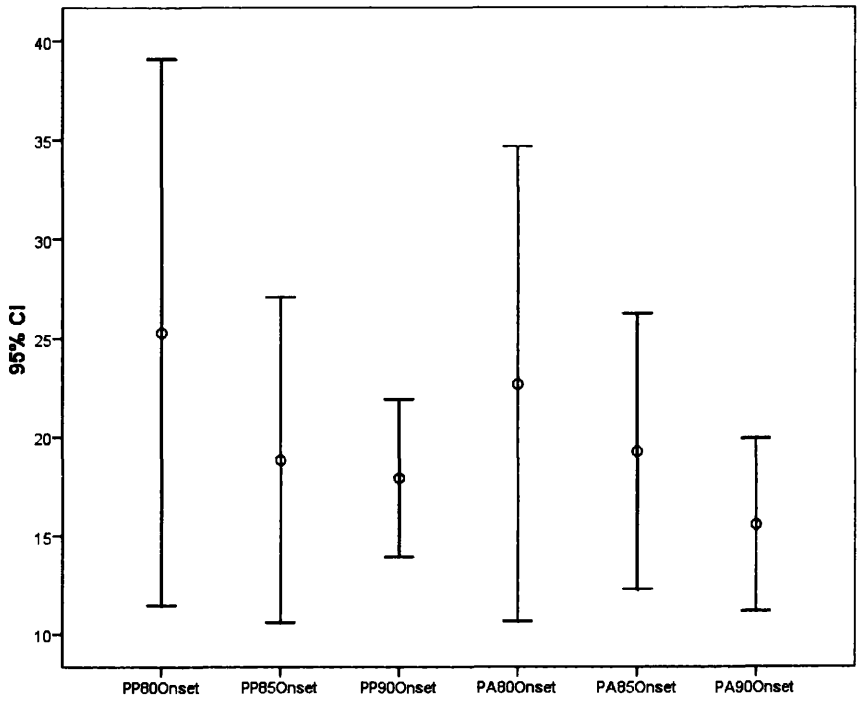


Figure 3.4 PPER peak latency

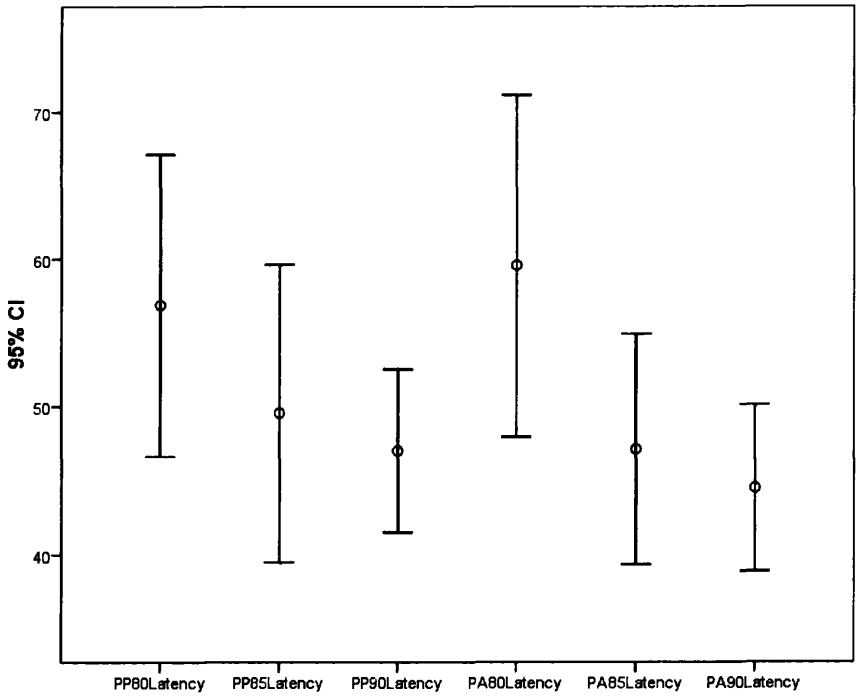


Figure 3.5 PPER onset to peak latency values

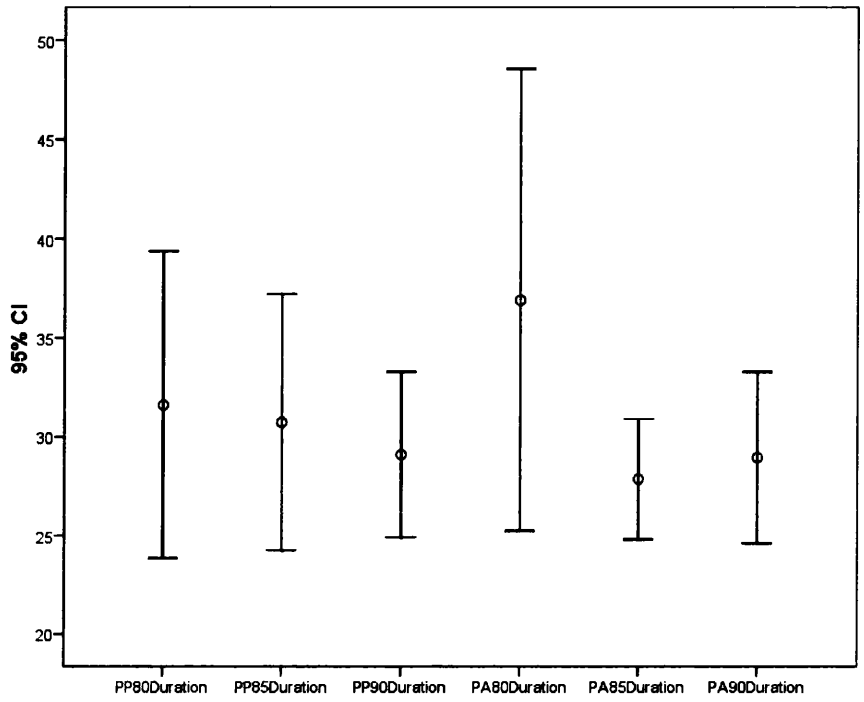


Figure 3.6 PPI probabilities

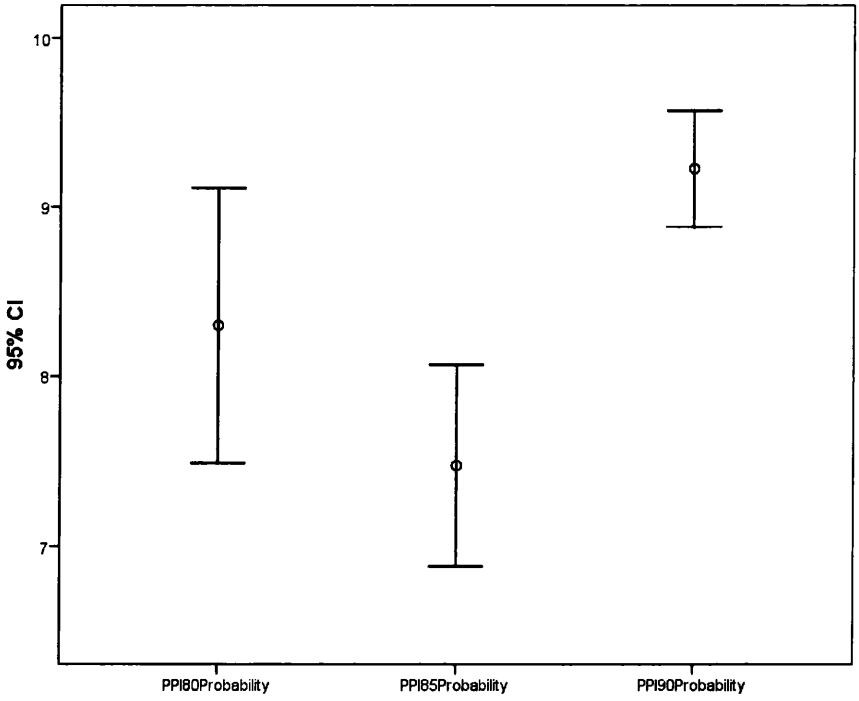


Figure 3.7 PPI percentages

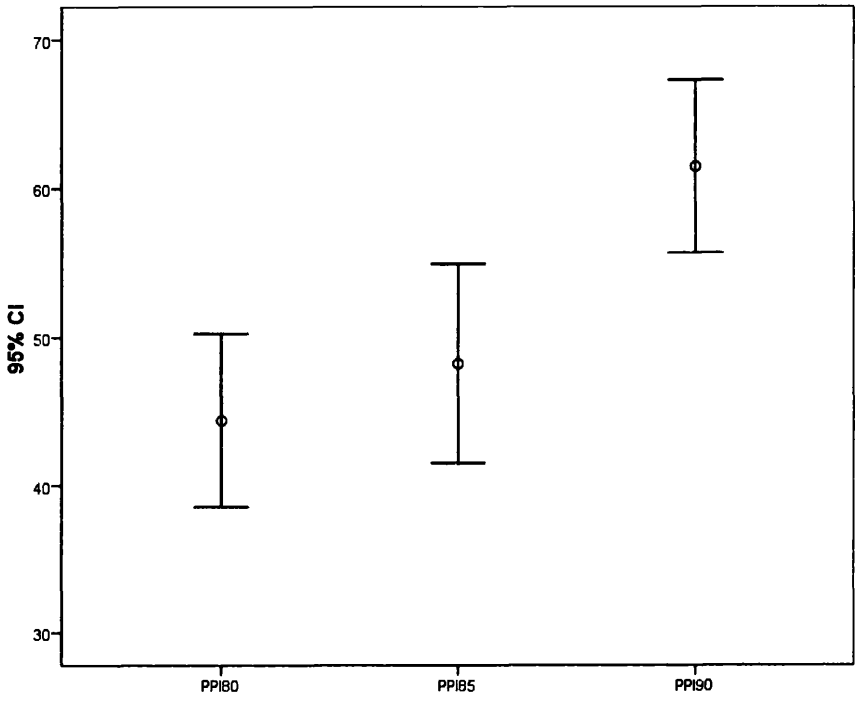


Figure 3.8 PPF probabilities

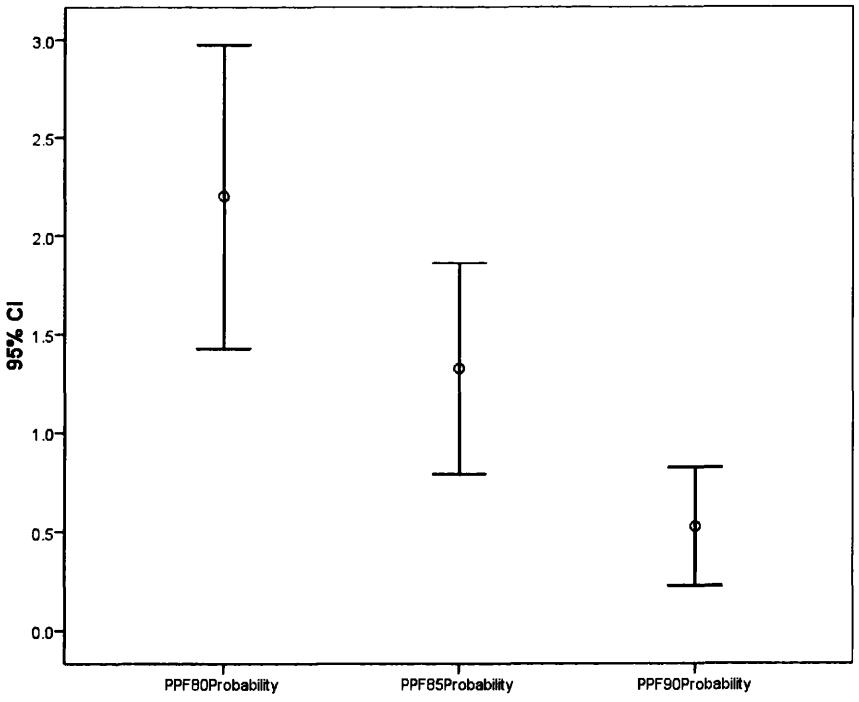
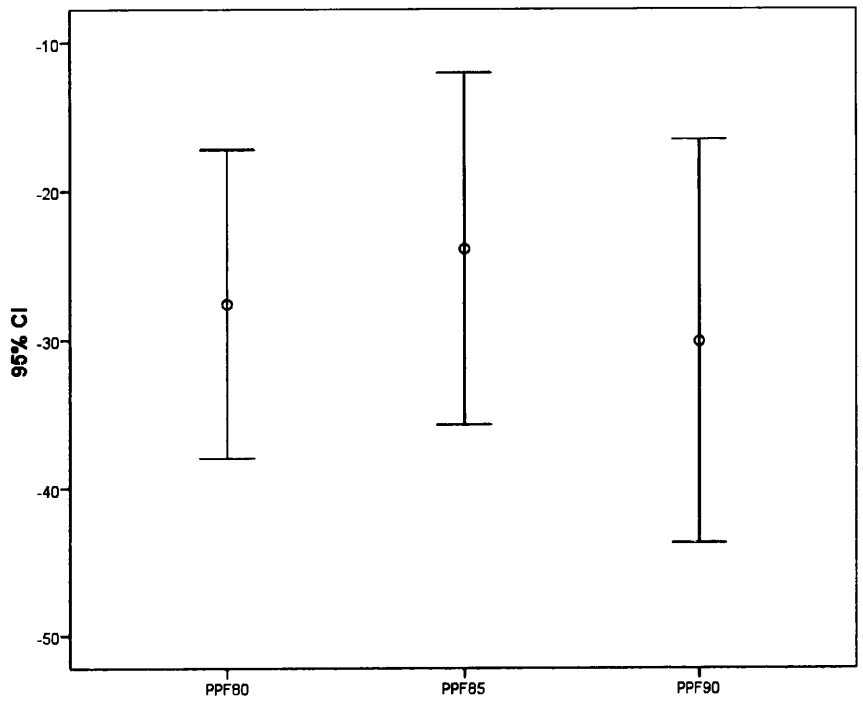


Figure 3.9 PPF percentages



Part 2 Tables

Table 3.1 Paired samples t-test results comparing prepulse-elicited response probabilities and spontaneous EMG activation probability in the absence of experimental stimuli (significant outcomes marked as **bold**) [SP=startle probe alone trial, PP= prepulse and pulse trial and PA= prepulse alone trial, B= no stimulus 'blind' trial; PPx, x = prepulse intensity]

Motor Response Probability	*M*	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
PP80- SP	.06	.16	.03	.11	.01	**2.19**	39	**.03**
PP85 - SP	.18	.26	.04	.26	.09	**4.32**	39	**< .01**
PP90 - SP	.31	.34	.05	.42	.20	**5.81**	39	**< .01**
PA80 - SP	.06	.12	.02	.10	.03	**3.34**	39	**< .01**
PA85 - SP	.14	.23	.04	.21	.07	**3.98**	39	**< .01**
PA90 - SP	.34	.35	.05	.45	.23	**6.11**	39	**< .01**
PP80 - B	.07	.16	.02	.02	.12	**2.70**	39	**.01**
PP85 - B	.19	.26	.04	.10	.27	**4.51**	39	**< .01**
PP90 -B	.32	.34	.05	.22	.43	**6.01**	39	**< .01**
PA80 - B	.07	.12	.01	.04	.11	**3.91**	39	**< .01**
PA85 - B	.15	.22	.03	.08	.224	**4.30**	39	**< .01**
PA90 - B	.35	.35	.05	.23	.46	**6.26**	39	**< .01**

Table 3.2 Paired samples t-test results comparing prepulse-elicited responses amplitude and probability in prepulse and pulse (pulse presented) and prepulse alone trials (pulse absent)

Motor Response	*M*	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
Amplitude SP – Amplitude B	32.50	49.52	28.59	-90.53	155.53	1.14	2	.37
Probability SP - Probability B	.010	.04	.07	-.01	.02	1.43	39	.16
Amplitude PP80- Amplitude PA80	-1.28	42.22	12.19	-28.10	25.55	-.10	11	.92
ProbabilityPP80 -Probability PA80	-.01	.11	.02	-.04	.03	-.49	39	.63
Amplitude PP85 - Amplitude PA85	-4.90	37.12	8.75	-23.36	13.55	-.56	17	.58
Probability PP85 - Probability PA85	.03	.14	.02	-.01	.08	1.56	39	.13
Amplitude PP90- Amplitude PA90	-2.47	16.69	3.41	-9.52	4.57	-.73	23	.47
Probability PP90 - ProbabilityPA90	-.02	.133	.02	-.06	.02	-1.07	39	.29

Table 3.3 Paired-samples t-test on the temporal characteristics of prepulse-elicited responses in prepulse and pulse and prepulse-alone trials

PPER	*M*	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
Onset SP - Onset B	-.33	6.06	3.03	-9.98	9.32	-.11	3	.92
Onset PP80 - Onset PA80	-4.19	27.38	7.90	-21.58	13.21	-.53	11	.61
Onset PP85 - Onset PA85	-1.57	14.09	3.32	-8.58	5.44	-.47	17	.64
Onset PP90 - Onset PA90	-.18	7.65	1.56	-3.41	3.05	-.12	23	.91
Peak Latency SP - Peak Latency B	-.41	32.18	16.09	-51.61	50.79	-.03	3	.98
Peak Latency PP80 - Peak Latency PA80	-9.20	25.26	7.29	-25.25	6.85	-1.26	11	.23
Peak Latency PP85 - Peak Latency PA85	.14	14.26	3.36	-6.95	7.23	.04	17	.97
Peak Latency PP90 - Peak Latency PA90	.34	7.67	1.57	-2.90	3.58	.22	23	.83
Onset-Peak SP - Onset-Peak B	-1.83	31.87	18.40	-81.00	77.33	-.10	2	.93
Onset-Peak PP80 - Onset-Peak PA80	-5.01	19.25	5.5	-17.24	7.22	-.90	11	.38
Onset-Peak PP85 - Onset-Peak PA85	1.71	9.59	2.26	-3.06	6.48	.76	17	.46
Onset-Peak PP90 - Onset-Peak PA90	.52	11.25	2.30	-4.23	5.27	.23	23	.82

Table 3. 4 Prepulse-elicited response probability correlations

PPER Probabilities		PP80	PP85	PP90	PA80	PA85	PA90
PP80	*r*	1	**.58(**)**	**.517(**)**	**.69(**)**	**.72(**)**	**.54(**)**
	p		**<.01**	**.01**	**<.01**	**<.01**	**<.01**
	N	40	40	40	40	40	40
PP85	*r*	**.58(**)**	1	**.79(**)**	**.54(**)**	**.84(**)**	**.84(**)**
	p	**<.01**		**<.01**	**<.01**	**<.01**	**<.01**
	N	40	40	40	40	40	40
PP90	*r*	**.52(**)**	**.79(**)**	1	**.58(**)**	**.83(**)**	**.93(**)**
	p	**.01**	**<.01**		**<.01**	**<.01**	**<.01**
	N	40	40	40	40	40	40
PA80	*r*	**.69(**)**	**.54(**)**	**.58(**)**	1	**.66(**)**	**.59(**)**
	p	**<.01**	**<.01**	**<.01**		**<.01**	**<.01**
	N	40	40	40	40	40	40
PA85	*r*	**.72(**)**	**.84(**)**	**.83(**)**	**.66(**)**	1	**.81(**)**
	p	**<.01**	**<.01**	**<.01**	**<.01**		**<.01**
	N	40	40	40	40	40	40
PA90	*r*	**.54(**)**	**.84(**)**	**.93(**)**	**.59(**)**	**.81(**)**	1
	p	**<.01**	**<.01**	**<.01**	**<.01**	**<.01**	
	N	40	40	40	40	40	40

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.5 Prepulse elicited response amplitudes correlations

PPER Amplitudes		PP80	PP85	PP90	PA80	PA85	PA90
PP80	*r*	1	.00	.49	.29	.42	**.63(*)**
	p		1.00	.12	.37	.19	**.04**
	N	14	12	11	12	11	11
PP85	*r*	.00	1	**.57(*)**	.36	.08	**.48(*)**
	p	1.00		**.01**	.19	.75	**.03**
	N	12	22	19	15	18	20
PP90	*r*	.49	**.57(*)**	1	.14	**.46(*)**	**.94(**)**
	p	.12	**.01**		.60	**.04**	**<.01**
	N	11	19	28	17	20	24
PA80	*r*	.29	.36	.14	1	.16	.28
	p	.37	.19	.60		.55	.27
	N	12	15	17	19	16	18
PA85	*r*	.42	.08	**.46(*)**	.162	1	.346
	p	.19	.75	**.04**	.550		.115
	N	11	18	20	16	22	22
PA90	*r*	**.63(*)**	**.48(*)**	**.94(**)**	.28	.35	1
	p	**.04**	**.03**	**<.01**	.27	.11	
	N	11	20	24	18	22	28

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.6 Prepulse elicited response onsets correlations

PPER Onsets		PP80	PP85	PP90	PA80	PA85	PA90
PP80	*r*	1	**.63(*)**	.55	.28	**.83(**)**	**.61(*)**
	p		**.03**	.08	.37	**.01**	**.04**
	N	14	12	11	12	11	11
PP85	*r*	**.63(*)**	1	**.56(*)**	**.60(*)**	.44	**.51(*)**
	p	**.03**		**.01**	**.02**	.07	**.02**
	N	12	22	19	15	18	20
PP90	*r*	.55	**.56(*)**	1	.31	.35	.40
	p	.08	**.01**		.22	.13	.05
	N	11	19	28	17	20	24
PA80	*r*	.28	**.60(*)**	.31	1	**.73(**)**	**.48(*)**
	p	.37	**.02**	.22		**.01**	**.04**
	N	12	15	17	19	16	18
PA85	*r*	**.83(**)**	.44	.35	**.73(**)**	1	**.65(**)**
	p	**.01**	.07	.13	**.01**		**.01**
	N	11	18	20	16	22	22
PA90	*r*	**.61 (*)**	**.51(*)**	.40	**.48(*)**	**.65(**)**	1
	p	**.04**	**.02**	.05	**.04**	**.01**	
	N	11	20	24	18	22	28

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.7 Prepulse-elicited response peak latency correlations

PPER Peak Latency		PP80	PP85	PP90	PA80	PA85	PA90
PP80	*r*	1	**.89(**)**	**.74 (**)**	.14	**.67(*)**	.44
	p		**<.01**	**.01**	.65	**.02**	.17
	N	14	12	11	12	11	11
PP85	*r*	**.89(**)**	1	.34	**.73(**)**	**.77(**)**	.17
	p	**<.01**		.15	**.01**	**<.01**	.46
	N	12	22	19	15	18	20
PP90	*r*	**.74(**)**	.34	1	**.57(*)**	.33	**.60 (**)**
	p	**.01**	.15		**.02**	.15	**.01**
	N	11	19	28	17	20	24
PA80	*r*	.14	**.73(**)**	**.57(*)**	1	.26	**.50(*)**
	p	.65	**.01**	**.02**		.33	**.03**
	N	12	15	17	19	16	18
PA85	*r*	**.67(*)**	**.77(**)**	.33	.26	1	.27
	p	**.02**	**<.01**	.15	.33		.22
	N	11	18	20	16	22	22
PA90	*r*	.44	.175	**.60(**)**	**.50(*)**	.27	1
	p	.17	.462	**.01**	**.03**	.22	
	N	11	20	24	18	22	28

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.8 Prepulse-elicited response onset to peak latency values correlations

PPER Onset-Peak Latency		PP80	PP85	PP90	PA80	PA85	PA90
PP80	*r*	1	-.359	-.024	.169	.075	.01
	p		.252	.944	.599	.827	.98
	N	14	12	11	12	11	11
PP85	*r*	-.36	1	**.56(*)**	-.08	**.79(**)**	**.45(*)**
	p	.25		**.01**	.78	**<.01**	**.04**
	N	12	22	19	15	18	20
PP90	*r*	-.02	**.56(*)**	1	.15	.27	.15
	p	.94	**.01**		.57	.25	.48
	N	11	19	28	17	20	24
PA80	*r*	.17	-.08	.15	1	-.14	.46
	p	.60	.78	.57		.60	.05
	N	12	15	17	19	16	18
PA85	*r*	.07	**.79(**)**	.27	-.14	1	.19
	p	.83	**<.01**	.25	.60		.39
	N	11	18	20	16	22	22
PA90	*r*	.01	**.45(*)**	.15	.46	.19	1
	p	.98	**.04**	.48	.05	.39	
	N	11	20	24	18	22	28

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.9 Startle response modification probabilities correlations

Probability		PPF 80	PPF 85	PPF 90	PPI 80	PPI 85	PPI 90
PPF 80	*r*	1	**.69 (**)**	**.51(**)**	**-.93(**)**	**-.71(**)**	**-.41(**)**
	p		**<.01**	**.01**	**<.01**	**<.01**	**.01**
	N	40	40	40	40	40	40
PPF 85	*r*	**.69(**)**	1	**.73(**)**	**-.70(**)**	**-.98(**)**	**-.60(**)**
	p	**<.01**		**<.01**	**<.01**	**<.001**	**<.01**
	N	40	40	40	40	40	40
PPF 90	*r*	**.51(**)**	**.73(**)**	1	**-.50(**)**	**-.76(**)**	**-.89(**)**
	p	**.01**	**<.01**		**.01**	**<.01**	**<.01**
	N	40	40	40	40	40	40
PPI 80	*r*	**-.93(**)**	**-.70(**)**	**-.50(**)**	1	**.71(**)**	**.45(**)**
	p	**<.01**	**<.01**	**.01**		**<.01**	**.01**
	N	40	40	40	40	40	40
PPI 85	*r*	**-.71(**)**	**-.98(**)**	**-.76(**)**	**.71(**)**	1	**.60(**)**
	p	**<.01**	**<.01**	**<.01**	**<.01**		**<.01**
	N	40	40	40	40	40	40
PPI 90	*r*	**-.41(**)**	**-.60(**)**	**-.89(**)**	**.45(**)**	**.60(**)**	1
	p	**.01**	**<.01**	**<.01**	**.01**	**<.01**	
	N	40	40	40	40	40	40

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.10 Startle response modification type percentage change correlations

Percentage Change		PPI 80	PPI 85	PPI 90	PPF 80	PPF 85	PPF 90
PPI 80	*r*	1	**.82(**)**	**.76(**)**	-.36	.38	.(a)
	p		**< .01**	**< .01**	.19	.40	.
	N	40	40	40	15	7	1
PPI 85	*r*	**.82(**)**	1	**.78(**)**	-.10	.07	.(a)
	p	**< .01**		**< .01**	.72	.89	.
	N	40	40	40	15	7	1
PPI 90	*r*	**.76(**)**	**.78(**)**	1	-.12	.33	.(a)
	p	**< .01**	**< .01**		.67	.47	.
	N	40	40	40	15	7	1
PPF 80	*r*	-.36	-.10	-.120	1	.18	.(a)
	p	.19	.72	.67		.73	.
	N	15	15	15	15	6	1
PPF 85	*r*	.38	.07	.33	.18	1	.(a)
	p	.40	.89	.47	.73		.
	N	7	7	7	6	7	1
PPF 90	*r*	.(a)	.(a)	.(a)	.(a)	.(a)	.(a)
	p	.	.	.	.	.	
	N	1	1	1	1	1	1

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.11 PPI percentage change and PPF probability correlations

Percentage		Probability PPF 80	Probability PPF 85	Probability PPF 90
PPI 80	*r*	**-.66(**)**	**-.47(**)**	**-.39(*)**
	p	**< .01**	**.01**	**.01**
	N	40	40	40
PPI 85	*r*	**-.70(**)**	**-.63(**)**	**-.62(**)**
	p	**< .01**	**< .01**	**< .01**
	N	40	40	40
PPI 90	*r*	**-.59(**)**	**-.59(**)**	**-.44(**)**
	p	**< .01**	**< .01**	**.01**
	N	40	40	40

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.12 PPI probability and PPF percentage correlations

Probability		Percentage PPF 80	Percentage PPF 85	Percentage PPF 90
PPI 80	*r*	-.33	.64	.(a)
	p	.23	.12	.
	N	15	7	1
PPI 85	*r*	.15	.67	.(a)
	p	.60	.10	.
	N	15	7	1
PPI 90	*r*	.12	.09	.(a)
	p	.68	.84	.
	N	15	7	1

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.13 Prepulse-elicited response probabilities and startle response modification probability

Probability		PP80	PP85	PP90	PA80	PA85	PA90
PPI 80	*r*	.01	**-.40(*)**	**-.39(*)**	-.07	-.31	**-.40(*)**
	p	.96	**.01**	**.01**	.67	.05	**.01**
	N	40	40	40	40	40	40
PPI 85	*r*	-.05	-.17	-.12	-.10	-.14	-.11
	p	.75	.290	.46	.55	.40	.50
	N	40	40	40	40	40	40
PPI 90	*r*	.07	-.13	-.14	-.13	-.04	-.11
	p	.65	.41	.37	.44	.78	.49
	N	40	40	40	40	40	40
PPF 80	*r*	-.01	**.36(*)**	**.37(*)**	.01	.24	**.39(*)**
	p	.96	**.02**	**.02**	.96	.14	**.01**
	N	40	40	40	40	40	40
PPF 85	*r*	.10	.21	.15	.12	.18	.13
	p	.54	.20	.36	.44	.27	.44
	N	40	40	40	40	40	40
PPF 90	*r*	-.08	.04	.15	.07	.04	.10
	p	.60	.80	.35	.65	.83	.54
	N	40	40	40	40	40	40

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.14 Prepulse-elicited response probability and startle response modification percentage change correlations

Percentage		PP80	PP85	PP90	PA80	PA85	PA90
PPI 80	*r*	.07	**-.33(*)**	**-.34(*)**	-.08	-.22	-.29
	p	.68	**.04**	**.03**	.63	.17	.07
	N	40	40	40	40	40	40
PPI 85	*r*	.04	-.26	**-.40(**)**	-.13	-.18	**-.37(*)**
	p	.79	.11	**.01**	.43	.26	**.02**
	N	40	40	40	40	40	40
PPI 90	*r*	.14	-.05	-.01	-.03	.07	-.03
	p	.40	.74	.93	.84	.66	.85
	N	40	40	40	40	40	40
PPF 80	*r*	.27	.51	.50	.38	.49	**.61(*)**
	p	.34	.05	.06	.16	.06	**.02**
	N	15	15	15	15	15	15
PPF 85	*r*	.15	.11	.55	**.78(*)**	<.001	.55
	p	.74	.82	.20	**.04**	.10	.20
	N	7	7	7	7	7	7
PPF 90	*r*	.(a)	.(a)	.(a)	.(a)	.(a)	.(a)
	p	.	.	.	.	.	.
	N	1	1	1	1	1	1

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.15 Prepulse-elicited responses amplitudes and startle response probabilities correlations

PPER Amplitude		PPF 80	PPF 85	PPF 90	PPI 80	PPI 85	PPI 90
PP80	*r*	.49	.58	.[a]	.21	.035	.44
	p	.40	.42	.	.48	.91	.11
	N	5	4	0	14	14	14
PP85	*r*	-.25	.17	.[a]	.08	.07	-.01
	p	.46	.75	.	.71	.76	.95
	N	11	6	0	22	22	22
PP90	*r*	.31	.10	.[a]	-.23	-.13	.02
	p	.30	.86	.	.23	.50	.93
	N	13	6	1	28	28	28
PA80	*r*	.18	.67	.[a]	-.06	-.31	.08
	p	.67	.22	.	.82	.20	.74
	N	8	5	0	19	19	19
PA85	*r*	**.66***	.40	.[a]	-.24	-.17	.09
	p	**.02**	.37	.	.28	.44	.69
	N	12	7	1	22	22	22
PA90	*r*	.21	-.02	.[a]	-.22	-.16	-.03
	p	.49	.97	.	.27	.43	.90
	N	13	7	1	28	28	28

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3. 16 PPER amplitude and SRM percentage change correlations

PPER Amplitude		Percentage PPF 80	Percentage PPF 85	Percentage PPF 90	Percentage PPI 80	Percentage PPI 85	Percentage PPI 90
PP80	*r*	.26	.20	-.40	.37	.27	.33
	p	.46	.57	.37	.19	.35	.25
	N	10	10	7	14	14	14
PP85	*r*	-.14	-.03	-.04	.13	.15	.28
	p	.57	.90	.92	.56	.51	.21
	N	19	16	11	22	22	22
PP90	*r*	.23	.23	.14	-.24	-.12	-.03
	p	.33	.35	.66	.22	.54	.89
	N	20	18	12	28	28	28
PA80	*r*	.26	-.28	-.11	.10	-.05	-.07
	p	.37	.34	.78	.69	.82	.78
	N	14	14	9	19	19	19
PA85	*r*	.29	-.17	-.14	-.26	-.02	-.27
	p	.24	.53	.67	.24	.91	.23
	N	18	17	12	22	22	22
PA90	*r*	.02	.26	.16	-.23	-.19	-.05
	p	.93	.28	.62	.24	.34	.81
	N	21	19	12	28	28	28

Table 3.17 PPER onset-peak latency values and startle response modification percentage and probability correlations

SRM		PP80	PP85	PP90
Percentage PPI 80	*r*	.08	.16	.06
	p	.78	.48	.74
	N	14	22	28
Percentage PPI 85	*r*	-.08	.12	-.02
	p	.79	.61	.90
	N	14	22	28
Percentage PPI 90	*r*	.02	.06	.09
	p	.95	.80	.65
	N	14	22	28
Percentage PPF 80	*r*	.73	.12	-.08
	p	.16	.73	.80
	N	5	11	13
Percentage PPF 85	*r*	.71	.36	.15
	p	.29	.48	.77
	N	4	6	6
Percentage PPF 90	*r*	.(a)	.(a)	.(a)
	p	.	.	.
	N	0	0	1
Probability PPI 80	*r*	.18	.06	-.12
	p	.54	.79	.54
	N	14	22	28
Probability PPI 85	*r*	.11	.01	-.09
	p	.71	.10	.65
	N	14	22	28
Probability PPI 90	*r*	.20	.20	.09
	p	.49	.36	.66
	N	14	22	28

Probability PPF 80	*r*	-.02	.01	.21
	p	.94	.96	.28
	N	14	22	28
Probability PPF 85	*r*	-.21	-.03	.07
	p	.47	.89	.73
	N	14	22	28
Probability PPF 90	*r*	.10	-.09	.06
	p	.72	.70	.76
	N	14	22	28

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.18 Personality factors and prepulse-elicited response probability correlations

Personality Factors		SP	PP80	PP85	PP90	PA80	PA85	PA90	B
State Anxiety	*r*	.04	.14	-.03	.01	.28	.10	-.07	-.22
	p	.80	.38	.85	.10	.08	.54	.68	.17
	N	40	40	40	40	40	40	40	40
Trait Anxiety	*r*	.12	.14	-.12	.04	.21	.01	-.03	-.16
	p	.47	.41	.47	.81	.20	.94	.86	.34
	N	39	39	39	39	39	39	39	39
Positive Affect	*r*	-.15	-.07	.03	.08	-.23	-.01	.10	.03
	p	.36	.65	.86	.62	.15	.94	.53	.83
	N	40	40	40	40	40	40	40	40
Negative Affect	*r*	-.09	.07	-.03	.15	-.01	.13	.04	-.15
	p	.58	.68	.85	.34	.10	.42	.80	.35
	N	40	40	40	40	40	40	40	40
BAS Drive	*r*	-.11	.14	.25	.12	-.06	.18	.11	.05
	p	.52	.39	.13	.45	.71	.26	.49	.76
	N	39	39	39	39	39	39	39	39
BAS Fun	*r*	-.11	.05	.18	.06	-.09	.19	-.01	-.18
	p	.48	.76	.26	.72	.60	.25	.10	.28
	N	39	39	39	39	39	39	39	39
BAS Reward	*r*	-.21	.10	.27	.21	-.27	.19	.18	-.09
	p	.19	.55	.10	.21	.10	.26	.27	.57
	N	39	39	39	39	39	39	39	39
BIS	*r*	-.12	-.01	-.02	.11	.06	-.08	.07	-.08
	p	.48	.94	.90	.52	.70	.63	.65	.62
	N	39	39	39	39	39	39	39	39
Unusual Experiences	*r*	.19	-.17	-.12	-.12	-.08	-.11	-.14	-.01
	p	.24	.30	.45	.48	.64	.51	.38	.10
	N	39	39	39	39	39	39	39	39
Cognitive Disorganisation	*r*	.15	.09	.06	.12	.18	.10	.17	-.16

	p	.37	.58	.70	.48	.27	.52	.31	.32
	N	39	39	39	39	39	39	39	39
Introvertive Anhedonia	*r*	.27	.09	.04	-.12	.08	-.06	.03	.14
	p	.09	.56	.80	.47	.63	.73	.86	.40
	N	39	39	39	39	39	39	39	39
Impulsive Non-conformity	*r*	.01	-.16	.04	.10	.05	.05	.08	-.12
	p	.98	.34	.82	.56	.75	.78	.62	.48
	N	39	39	39	39	39	39	39	39
Novelty Seeking	*r*	.01	.06	**.33(*)**	.25	.10	.25	.21	-.09
	p	.98	.70	**.04**	.12	.52	.12	.19	.58
	N	39	39	39	39	39	39	39	39
Harm Avoidance	*r*	.08	.14	-.08	.09	.23	-.02	.08	.03
	p	.64	.40	.62	.57	.16	.92	.64	.83
	N	39	39	39	39	39	39	39	39
Reward Dependence	*r*	-.10	.10	.19	.30	.10	.11	.23	.04
	p	.56	.56	.26	.06	.56	.50	.16	.83
	N	39	39	39	39	39	39	39	39
Persistence	*r*	-.14	.07	-.12	-.30	.02	-.20	-.26	-.11
	p	.40	.65	.46	.06	.90	.22	.11	.49
	N	39	39	39	39	39	39	39	39
Self-Directedness	*r*	.02	-.11	-.03	-.11	-.18	-.15	-.12	.28
	p	.89	.50	.85	.51	.27	.35	.48	.09
	N	39	39	39	39	39	39	39	39
Cooperativeness	*r*	.26	.16	.21	.12	.25	.09	.12	.17
	p	.10	.34	.21	.46	.12	.58	.47	.30
	N	39	39	39	39	39	39	39	39
Self-Transcendence	*r*	-.01	-.08	.08	.11	-.18	.03	.11	-.09
	p	.95	.61	.61	.51	.26	.87	.49	.60
	N	39	39	39	39	39	39	39	39
Personality Factors		SP	PP80	PP85	PP90	PA80	PA85	PA90	B

FSS Total Score	*r*	.29	-.30	-.14	-.12	-.27	-.19	-.15	.10
	p	.08	.07	.40	.46	.10	.24	.38	.53
	N	39	39	39	39	39	39	39	39
Fear of Animals	*r*	.14	-.20	-.22	-.23	-.28	-.25	-.29	.24
	p	.38	.22	.18	.15	.08	.12	.07	.14
	N	39	39	39	39	39	39	39	39
Interpersonal Fear	*r*	.26	-.23	-.17	-.13	-.24	-.18	-.10	-.04
	p	.11	.16	.29	.44	.14	.26	.53	.81
	N	39	39	39	39	39	39	39	39
Fear of Tissue Damage	*r*	.31	**-.32(*)**	-.05	-.04	-.23	-.15	-.08	.12
	p	.06	**.04**	.77	.79	.15	.35	.63	.47
	N	39	39	39	39	39	39	39	39
Fear of Noises	*r*	.14	-.15	-.10	-.17	-.20	-.14	-.14	.06
	p	.41	.35	.54	.29	.21	.38	.40	.70
	N	39	39	39	39	39	39	39	39
Classic Phobias	*r*	.27	-.25	-.11	-.12	-.16	-.16	-.14	.15
	p	.09	.12	.51	.45	.34	.33	.40	.35
	N	39	39	39	39	39	39	39	39

** ** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.19 Personality factors and prepulse-elicited response amplitude

Personality Factors		SP	PP80	PP85	PP90	PA80	PA85	PA90	B
State Anxiety	*r*	-.42	.30	-.23	.10	-.17	-.03	.19	-.27
	p	.23	.30	.30	.62	.50	.90	.341	.60
	N	8	14	22	28	19	22	28	6
Trait Anxiety	*r*	-.48	.28	-.32	-.16	-.36	-.15	.01	**-.91(*)**
	p	.28	.33	.15	.43	.13	.50	.97	**.01**
	N	7	14	21	28	19	21	27	6
Positive Affect	*r*	-.45	-.35	.08	-.01	.12	-.10	-.01	.33
	p	.26	.22	.73	.98	.64	.65	.94	.52
	N	8	14	22	28	19	22	28	6
Negative Affect	*r*	-.40	.04	.09	-.11	.07	-.23	-.03	-.80
	p	.32	.88	.69	.58	.79	.30	.87	.06
	N	8	14	22	28	19	22	28	6
BAS Drive	*r*	-.01	-.37	.36	.33	.08	.23	.29	.30
	p	.99	.20	.11	.08	.75	.32	.14	.56
	N	7	14	21	28	19	21	27	6
BAS Fun	*r*	-.42	-.24	.32	.22	-.25	.28	.23	.59
	p	.35	.40	.15	.26	.30	.21	.25	.22
	N	7	14	21	28	19	21	27	6
BAS Reward	*r*	.06	-.40	.40	.31	.10	.14	.22	.33
	p	.90	.15	.07	.11	.68	.54	.27	.52
	N	7	14	21	28	19	21	27	6
BIS	*r*	.39	-.22s	-.23	-.32	-.21	-.25	-.32	.39
	p	.38	.44	.32	.10	.38	.28	.11	.44
	N	7	14	21	28	19	21	27	6
Unusual Experiences	*r*	-.60	-.27	-.08	-.23	-.12	-.24	-.09	-.74
	p	.16	.34	.73	.24	.63	.30	.67	.09
	N	7	14	21	28	19	21	27	6

Cognitive Disorganisation	*r*	-.27	-.25	-.01	-.20	-.36	**-.55(**)**	-.22	-.31
	p	.56	.39	.98	.31	.13	**.01**	.26	.55
	N	7	14	21	28	19	21	27	6
Introvertive Anhedonia	*r*	.08	-.31	.09	-.08	-.21	-.37	-.17	.45
	p	.87	.28	.71	.70	.38	.10	.41	.37
	N	7	14	21	28	19	21	27	6
Impulsive Non-conformity	*r*	-.01	-.18	-.09	-.19	-.25	-.07	-.15	-.05
	p	.98	.54	.69	.33	.30	.76	.46	.92
	N	7	14	21	28	19	21	27	6
Novelty Seeking	*r*	.16	**-.58(*)**	.25	.06	-.36	.07	.14	.62
	p	.73	**.03**	.28	.76	.13	.76	.47	.18
	N	7	14	21	28	19	21	27	6
Harm Avoidance	*r*	.31	-.05	-.29	-.36	.05	-.36	-.28	-.47
	p	.50	.86	.21	.06	.83	.11	.15	.35
	N	7	14	21	28	19	21	27	6
Reward Dependence	*r*	.29	.49	-.01	.21	.26	**.56(**)**	.31	-.11
	p	.53	.07	.99	.29	.29	**.01**	.12	.83
	N	7	14	21	28	19	21	27	6
Persistence	*r*	-.38	-.10	-.12	.03	.04	.29	-.06	.51
	p	.40	.73	.59	.88	.87	.20	.77	.30
	N	7	14	21	28	19	21	27	6
Self-Directedness	*r*	.35	.10	.08	.06	.38	.40	.06	-.02
	p	.44	.74	.73	.77	.11	.07	.77	.96
	N	7	14	21	28	19	21	27	6
Cooperativeness	*r*	.21	.45	-.01	.24	.14	.25	.30	-.38
	p	.65	.10	.98	.21	.58	.27	.13	.46
	N	7	14	21	28	19	21	27	6
Self-Transcendence	*r*	-.51	-.08	.16	.20	.13	.16	.24	-.21
	p	.24	.79	.47	.31	.60	.48	.24	.69

	N	7	14	21	28	19	21	27	6
FSS Total Score	*r*	-.58	-.24	.01	-.29	-.04	-.15	-.21	**-.88(*)**
	p	.17	.41	.10	.13	.88	.52	.29	**.02**
	N	7	14	21	28	19	21	27	6
Fear of Animals	*r*	-.29	.17	-.11	-.08	-.01	-.04	.10	-.78
	p	.52	.55	.63	.70	.96	.86	.62	.07
	N	7	14	21	28	19	21	27	6
Interpersonal Fear	*r*	**-.82(*)**	-.29	.07	-.35	-.05	-.25	-.34	**-.88(*)**
	p	**.02**	.31	.75	.07	.84	.27	.08	**.02**
	N	7	14	21	28	19	21	27	6
Fear of Tissue Damage	*r*	-.53	-.28	-.01	-.29	-.04	-.12	-.11	**-.82(*)**
	p	.21	.32	.10	.13	.88	.61	.57	**.04**
	N	7	14	21	28	19	21	27	6
Fear of Noises	*r*	-.41	-.09	.24	-.17	.23	-.11	-.17	-.73
	p	.36	.75	.30	.39	.34	.65	.38	.10
	N	7	14	21	28	19	21	27	6
Classic Phobias	*r*	-.52	-.14	-.14	-.23	-.10	.08	-.16	**-.82(*)**
	p	.23	.63	.55	.24	.69	.72	.43	**.04**
	N	7	14	21	28	19	21	27	6

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.20 Personality factors and prepulse-elicited response onset correlations

Personality Factors		SP	PP80	PP85	PP90	PA80	PA85	PA90	B
State Anxiety	*r*	.30	.20	.15	.17	.03	.23	.26	-.41
	p	.42	.49	.50	.38	.90	.29	.18	.42
	N	9	14	22	28	19	22	28	6
Trait Anxiety	*r*	-.21	.47	.34	.11	-.01	.23	.28	-.12
	p	.62	.09	.12	.56	.98	.31	.16	.83
	N	8	14	21	28	19	21	27	6
Positive Affect	*r*	**-.76(*)**	-.38	-.20	-.07	.13	-.20	**-.38(*)**	-.31
	p	**.02**	.18	.36	.71	.61	.38	**.04**	.55
	N	9	14	22	28	19	22	28	6
Negative Affect	*r*	-.32	.02	.08	.03	-.10	.01	.11	-.49
	p	.41	.94	.73	.86	.67	.98	.59	.32
	N	9	14	22	28	19	22	28	6
BAS Drive	*r*	.64	-.39	-.06	.07	-.19	.09	-.02	-.36
	p	.08	.16	.79	.71	.45	.70	.93	.49
	N	8	14	21	28	19	21	27	6
BAS Fun	*r*	.19	-.42	-.21	-.17	-.24	.03	-.25	.04
	p	.65	.13	.37	.40	.32	.89	.21	.93
	N	8	14	21	28	19	21	27	6
BAS Reward	*r*	.61	-.23	.05	.15	-.04	-.01	-.07	.22
	p	.11	.43	.82	.45	.86	.95	.71	.68
	N	8	14	21	28	19	21	27	6
BIS	*r*	.49	.49	.36	-.01	.12	.27	.05	**.82(*)**
	p	.22	.07	.10	.94	.63	.24	.80	**.05**
	N	8	14	21	28	19	21	27	6
Unusual Experiences	*r*	-.21	.01	.05	.09	.01	-.07	-.09	-.46
	p	.62	.96	.81	.65	.97	.76	.66	.36
	N	8	14	21	28	19	21	27	6

Cognitive Disorganisation	*r*	-.21	.23	.35	.11	-.13	-.18	-.04	.47
	p	.61	.43	.12	.58	.59	.44	.83	.35
	N	8	14	21	28	19	21	27	6
Introvertive Anhedonia	*r*	.52	-.13	.26	-.13	**-.50(*)**	**-.45(*)**	.13	-.12
	p	.19	.64	.26	.50	**.03**	**.04**	.50	.82
	N	8	14	21	28	19	21	27	6
Impulsive Non-conformity	*r*	.05	.27	-.03	.03	.08	.10	-.08	.81
	p	.91	.36	.88	.87	.76	.67	.68	.05
	N	8	14	21	28	19	21	27	6
Novelty Seeking	*r*	.66	-.51	-.40	-.22	-.24	-.08	-.17	.15
	p	.07	.06	.07	.27	.33	.74	.39	.77
	N	8	14	21	28	19	21	27	6
Harm Avoidance	*r*	-.24	.47	.38	.07	.20	.17	.27	.08
	p	.57	.09	.09	.73	.41	.46	.18	.88
	N	8	14	21	28	19	21	27	6
Reward Dependence	*r*	-.01	**.63(*)**	-.04	-.07	.41	**.62(**)**	.07	**.87(*)**
	p	.99	**.02**	.85	.73	.08	**.01**	.74	**.02**
	N	8	14	21	28	19	21	27	6
Persistence	*r*	.36	.04	.25	-.12	.09	.31	-.05	.01
	p	.39	.89	.28	.53	.70	.17	.80	.99
	N	8	14	21	28	19	21	27	6
Self-Directedness	*r*	.20	-.09	-.08	-.14	-.04	-.01	.03	-.19
	p	.64	.75	.72	.49	.86	.97	.88	.72
	N	8	14	21	28	19	21	27	6
Cooperativeness	*r*	-.391	.15	-.19	.09	.08	.23	.07	-.08
	p	.338	.60	.40	.64	.73	.31	.72	.88
	N	8	14	21	28	19	21	27	6
Self-Transcendence	*r*	-.06	-.09	-.08	.25	.12	.01	.19	-.24

	p	.88	.75	.73	.20	.62	.99	.35	.65
	N	8	14	21	28	19	21	27	6
FSS Total Score	*r*	-.29	.04	.08	-.03	-.22	-.19	.08	-.22
	p	.48	.88	.71	.88	.36	.41	.69	.68
	N	8	14	21	28	19	21	27	6
Fear of Animals	*r*	.07	.08	-.02	.20	-.13	.05	.15	-.04
	p	.88	.80	.93	.30	.60	.82	.45	.94
	N	8	14	21	28	19	21	27	6
Interpersonal Fear	*r*	-.41	.02	.26	-.02	-.10	-.24	.04	-.02
	p	.31	.94	.25	.91	.68	.29	.85	.96
	N	8	14	21	28	19	21	27	6
Fear of Tissue Damage	*r*	-.36	-.06	-.14	-.13	-.25	-.20	.10	-.33
	p	.38	.82	.54	.52	.29	.38	.63	.52
	N	8	14	21	28	19	21	27	6
Fear of Noises	*r*	.04	.08	.42	-.01	.03	-.12	.06	-.23
	p	.93	.79	.05	.95	.92	.61	.75	.66
	N	8	14	21	28	19	21	27	6
Classic Phobias	*r*	-.35	.24	.11	.01	-.26	-.02	.14	-.39
	p	.40	.40	.62	.96	.28	.92	.50	.45
	N	8	14	21	28	19	21	27	6

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.21Personality factors and prepulse-elicited response peak latency correlations

Personality Factors		SP	PP80	PP85	PP90	PA80	PA85	PA90	B
State Anxiety	*r*	.54	.50	-.07	.19	-.09	-.12	-.21	-.79
	p	.13	.07	.74	.32	.71	.60	.28	.06
	N	9	14	22	28	19	22	28	6
Trait Anxiety	*r*	.40	**.75(**)**	.11	.33	-.03	-.11	-.17	**-.87(*)**
	p	.33	**.01**	.62	.09	.91	.63	.40	**.02**
	N	8	14	21	28	19	21	27	6
Positive Affect	*r*	-.55	-.51	.02	.09	.06	.12	.04	.24
	p	.12	.06	.92	.65	.82	.60	.85	.64
	N	9	14	22	28	19	22	28	6
Negative Affect	*r*	.09	.40	-.09	.05	-.36	-.06	-.18	**-.83(*)**
	p	.81	.16	.68	.80	.19	.79	.36	**.04**
	N	9	14	22	28	19	22	28	6
BAS Drive	*r*	.25	-.29	.03	-.17	-.23	.20	-.08	-.21
	p	.55	.32	.90	.38	.34	.38	.68	.69
	N	8	14	21	28	19	21	27	6
BAS Fun	*r*	.60	-.18	-.07	.02	-.12	.27	.10	.04
	p	.12	.54	.77	.91	.63	.24	.62	.93
	N	8	14	21	28	19	21	27	6
BAS Reward	*r*	.50	-.27	-.06	-.05	-.34	.19	-.01	-.21
	p	.21	.36	.79	.80	.15	.41	.99	.69
	N	8	14	21	28	19	21	27	6
BIS	*r*	.58	.37	.26	.07	-.12	.22	-.11	.36
	p	.13	.20	.25	.71	.61	.33	.59	.48
	N	8	14	21	28	19	21	27	6
Unusual Experiences	*r*	-.25	.28	-.08	-.14	-.22	-.28	-.37	**-.92(**)**
	p	.55	.33	.74	.48	.35	.22	.06	**.01**
	N	8	14	21	28	19	21	27	6

Cognitive Disorganisation	*r*	.19	.47	-.03	.07	-.25	-.27	-.37	-.48
	p	.66	.09	.90	.71	.31	.24	.06	.33
	N	8	14	21	28	19	21	27	6
Introvertive Anhedonia	*r*	.39	.22	-.10	.27	-.28	**-.52(*)**	-.31	-.23
	p	.34	.44	.66	.17	.24	**.02**	.11	.66
	N	8	14	21	28	19	21	27	6
Impulsive Non-conformity	*r*	.21	.25	-.03	.05	-.09	-.04	.15	-.06
	p	.62	.39	.90	.81	.70	.86	.47	.91
	N	8	14	21	28	19	21	27	6
Novelty Seeking	*r*	.58	-.45	-.30	-.09	**-.47(*)**	.09	-.27	.03
	p	.13	.10	.18	.63	**.04**	.70	.17	.96
	N	8	14	21	28	19	21	27	6
Harm Avoidance	*r*	.25	**.60(*)**	.16	.19	-.10	-.04	-.26	.17
	p	.55	**.02**	.49	.33	.69	.86	.18	.75
	N	8	14	21	28	19	21	27	6
Reward Dependence	*r*	-.01	.06	.22	-.23	.09	.30	-.07	.51
	p	.99	.84	.35	.24	.70	.18	.73	.30
	N	8	14	21	28	19	21	27	6
Persistence	*r*	.25	.07	.24	.14	.19	.08	.23	-.01
	p	.54	.82	.29	.48	.43	.73	.24	.99
	N	8	14	21	28	19	21	27	6
Self-Directedness	*r*	-.12	-.46	.13	-.20	.05	.34	.23	.13
	p	.77	.09	.58	.30	.83	.13	.25	.80
	N	8	14	21	28	19	21	27	6
Cooperativeness	*r*	-.44	-.27	.10	-.26	.15	.10	.09	-.14
	p	.27	.35	.66	.19	.54	.67	.64	.78
	N	8	14	21	28	19	21	27	6
Self-Transcendence	*r*	-.28	-.29	-.25	-.16	-.25	-.17	.03	-.63

	p	.50	.32	.27	.41	.30	.45	.89	.18
	N	8	14	21	28	19	21	27	6
FSS Total Score	*r*	-.36	.19	-.02	-.22	-.23	-.10	-.23	**-.90(*)**
	p	.38	.51	.92	.27	.34	.65	.25	**.02**
	N	8	14	21	28	19	21	27	6
Fear of Animals	*r*	-.01	.43	-.04	-.29	-.41	-.16	-.19	-.67
	p	.98	.12	.88	.13	.08	.49	.33	.15
	N	8	14	21	28	19	21	27	6
Interpersonal Fear	*r*	-.34	.19	.08	-.02	.01	-.09	-.16	**-.89(*)**
	p	.41	.51	.74	.93	.98	.68	.43	**.02**
	N	8	14	21	28	19	21	27	6
Fear of Tissue Damage	*r*	-.34	.02	-.17	-.32	-.32	-.02	-.28	**-.88(*)**
	p	.40	.95	.47	.09	.18	.92	.15	**.02**
	N	8	14	21	28	19	21	27	6
Fear of Noises	*r*	-.40	.08	.24	-.20	.06	-.17	-.20	**-.82(*)**
	p	.32	.80	.30	.31	.79	.47	.31	**.04**
	N	8	14	21	28	19	21	27	6
Classic Phobias	*r*	-.48	.21	.08	-.14	-.21	-.15	-.18	**-.87(*)**
	p	.23	.47	.74	.46	.40	.52	.36	**.03**
	N	8	14	21	28	19	21	27	6

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.22 Personality factors and prepulse-elicited response onset- peak latency values correlations

Personality Factors		SP	PP80	PP85	PP90	PA80	PA85	PA90	B
State Anxiety	*r*	**.72(*)**	.36	-.30	.07	-.13	-.37	-.35	-.72
	p	**.04**	.20	.17	.74	.61	.09	.07	.11
	N	8	14	22	28	19	22	28	6
Trait Anxiety	*r*	.52	.36	-.28	.23	-.02	-.28	-.32	**-.90(*)**
	p	.23	.20	.22	.25	.94	.22	.10	**.01**
	N	7	14	21	28	19	21	27	6
Positive Affect	*r*	-.39	-.18	.29	.13	-.09	.33	.30	.37
	p	.33	.54	.19	.51	.72	.13	.12	.47
	N	8	14	22	28	19	22	28	6
Negative Affect	*r*	.30	.45	-.24	.02	-.20	-.09	-.21	-.72
	p	.46	.11	.29	.90	.41	.70	.27	.10
	N	8	14	22	28	19	22	28	6
BAS Drive	*r*	.45	.11	.12	-.21	-.02	.19	-.05	-.10
	p	.30	.72	.60	.29	.94	.42	.80	.84
	N	7	14	21	28	19	21	27	6
BAS Fun	*r*	.65	.26	.17	.13	.16	.31	.25	.03
	p	.12	.36	.46	.51	.51	.18	.21	.95
	N	7	14	21	28	19	21	27	6
BAS Reward	*r*	.55	-.06	-.16	-.14	-.29	.24	.05	-.30
	p	.20	.85	.50	.46	.22	.30	.80	.56
	N	7	14	21	28	19	21	27	6
BIS	*r*	.58	-.12	-.09	.08	-.26	.10	-.12	.11
	p	.17	.68	.70	.70	.28	.66	.55	.83
	N	7	14	21	28	19	21	27	6
Unusual Experiences	*r*	-.20	.32	-.18	-.19	-.24	-.29	-.22	**-.84(*)**
	p	.67	.26	.43	.34	.33	.20	.26	**.04**
	N	7	14	21	28	19	21	27	6

Cognitive Disorganisation	*r*	.26	.30	**-.49(*)**	-.01	-.10	-.21	-.25	-.68
	p	.57	.29	**.02**	.98	.69	.35	.20	.14
	N	7	14	21	28	19	21	27	6
Introvertive Anhedonia	*r*	.34	.42	**-.48(*)**	.33	.30	-.35	-.33	-.20
	p	.46	.14	**.03**	.08	.22	.12	.09	.70
	N	7	14	21	28	19	21	27	6
Impulsive Non-conformity	*r*	.21	-.01	.01	.02	-.18	-.11	.17	-.34
	p	.65	.98	.99	.90	.45	.63	.40	.51
	N	7	14	21	28	19	21	27	6
Novelty Seeking	*r*	.52	.04	.08	.06	-.20	.16	-.09	-.02
	p	.23	.88	.74	.78	.40	.49	.67	.96
	N	7	14	21	28	19	21	27	6
Harm Avoidance	*r*	.33	.19	-.26	.13	-.33	-.15	**-.39(*)**	.16
	p	.47	.52	.26	.51	.17	.50	**.05**	.77
	N	7	14	21	28	19	21	27	6
Reward Dependence	*r*	.01	**-.65(*)**	.37	-.17	-.38	-.02	-.10	.25
	p	.98	**.01**	.10	.39	.11	.95	.62	.63
	N	7	14	21	28	19	21	27	6
Persistence	*r*	.26	.03	.03	.21	.09	-.10	.21	-.01
	p	.57	.92	.88	.28	.72	.67	.28	.99
	N	7	14	21	28	19	21	27	6
Self-Directedness	*r*	-.25	-.45	.29	-.10	.10	.42	.16	.21
	p	.59	.11	.20	.62	.67	.06	.44	.69
	N	7	14	21	28	19	21	27	6
Cooperativeness	*r*	-.69	-.49	.39	-.30	.05	-.02	.02	-.13
	p	.09	.07	.08	.12	.82	.92	.92	.81
	N	7	14	21	28	19	21	27	6
Self-Transcendence	*r*	-.29	-.23	-.26	-.31	-.39	-.21	-.11	-.60
	p	.53	.42	.25	.10	.10	.35	.59	.21

	N	7	14	21	28	19	21	27	6
FSS Total Score	*r*	-.33	.18	-.14	-.18	.02	-.01	-.23	**-.89(*)**
	p	.47	.54	.54	.36	.93	.97	.24	**.02**
	N	7	14	21	28	19	21	27	6
Fear of Animals	*r*	.04	.43	-.03	**-.40(*)**	-.26	-.23	-.25	-.71
	p	.93	.13	.91	**.03**	.27	.32	.20	.11
	N	7	14	21	28	19	21	27	6
Interpersonal Fear	*r*	-.27	.20	-.23	-.01	.12	.04	-.15	**-.95(**)**
	p	.56	.48	.32	.10	.62	.87	.46	**.01**
	N	7	14	21	28	19	21	27	6
Fear of Tissue Damage	*r*	-.33	.10	-.06	-.21	-.03	.10	-.29	**-.83(*)**
	p	.47	.74	.80	.28	.89	.67	.15	**.04**
	N	7	14	21	28	19	21	27	6
Fear of Noises	*r*	-.61	.01	-.20	-.17	.03	-.13	-.20	-.81
	p	.14	.99	.38	.37	.89	.58	.32	.051
	N	7	14	21	28	19	21	27	6
Classic Phobias	*r*	-.47	-.03	-.03	-.14	.09	-.17	-.24	-.80
	p	.28	.93	.88	.48	.70	.47	.24	.05
	N	7	14	21	28	19	21	27	6

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 3.23 Personality factors and startle response modification (SRM) probability

Personality Factors		PPI 80	PPI 85	PPI 90	PPF 80	PPF 85	PPF 90
State Anxiety	*r*	.10	-.20	-.21	-.05	.22	**.37(*)**
	p	.52	.20	.18	.78	.18	**.02**
	N	40	40	40	40	40	40
Trait Anxiety	*r*	.23	.02	.01	-.13	-.02	.12
	p	.15	.90	.95	.44	.90	.47
	N	39	39	39	39	39	39
Positive Affect	*r*	-.06	.23	.05	-.05	-.21	-.20
	p	.73	.14	.77	.77	.18	.22
	N	40	40	40	40	40	40
Negative Affect	*r*	.03	-.21	-.26	.02	.20	**.44(**)**
	p	.85	.20	.10	.92	.20	**.01**
	N	40	40	40	40	40	40
BAS Drive	*r*	-.20	-.09	-.14	.11	.14	.04
	p	.22	.60	.38	.50	.40	.80
	N	39	39	39	39	39	39
BAS Fun	*r*	-.04	-.01	-.09	-.09	.02	-.03
	p	.81	.94	.57	.59	.91	.88
	N	39	39	39	39	39	39
BAS Reward	*r*	-.13	.08	-.08	.15	-.04	.02
	p	.44	.64	.61	.36	.82	.92
	N	39	39	39	39	39	39
BIS	*r*	.11	.03	**-.33(*)**	-.11	-.04	**.40(*)**
	p	.50	.85	**.04**	.51	.80	**.01**
	N	39	39	39	39	39	39
Unusual Experiences	*r*	.17	.07	.08	-.12	-.06	.04
	p	.30	.69	.61	.48	.71	.81
	N	39	39	39	39	39	39
Cognitive Disorganisation	*r*	.03	-.21	-.21	.08	.19	**.39(*)**

	p	.87	.19	.19	.61	.25	**.01**
	N	39	39	39	39	39	39
Introvertive Anhedonia	*r*	.20	.12	.08	-.06	-.09	-.13
	p	.23	.47	.61	.71	.58	.43
	N	39	39	39	39	39	39
Impulsive Non-conformity	*r*	-.07	-.15	-.15	.03	.09	.28
	p	.66	.35	.35	.87	.57	.08
	N	39	39	39	39	39	39
Novelty Seeking	*r*	-.21	-.16	-.22	.13	.16	.13
	p	.19	.32	.19	.44	.34	.44
	N	39	39	39	39	39	39
Harm Avoidance	*r*	.05	-.17	-.12	-.01	.18	.26
	p	.74	.31	.45	.99	.27	.10
	N	39	39	39	39	39	39
Reward Dependence	*r*	-.08	.05	.10	.04	-.08	-.08
	p	.64	.77	.54	.78	.62	.62
	N	39	39	39	39	39	39
Persistence	*r*	.13	.06	.02	-.27	-.03	-.11
	p	.41	.69	.89	.10	.86	.52
	N	39	39	39	39	39	39
Self-Directedness	*r*	-.10	.17	.18	.06	-.16	-.29
	p	.56	.29	.26	.72	.33	.07
	N	39	39	39	39	39	39
Cooperativeness	*r*	.02	.05	.17	-.06	-.05	-.17
	p	.91	.77	.30	.72	.75	.29
	N	39	39	39	39	39	39
Self-Transcendence	*r*	-.09	-.02	.18	.13	-.01	-.12
	p	.59	.92	.28	.44	.93	.48
	N	39	39	39	39	39	39
FSS Total Score	*r*	.12	.26	.26	-.01	-.30	-.16

	p	.46	.11	.11	.96	.06	.33
	N	39	39	39	39	39	39
Fear of Animals	*r*	.26	.29	.30	-.13	-.30	-.24
	p	.11	.07	.06	.42	.07	.14
	N	39	39	39	39	39	39
Interpersonal Fear	*r*	.19	.21	.08	-.03	-.21	.03
	p	.25	.20	.63	.86	.20	.87
	N	39	39	39	39	39	39
Fear of Tissue Damage	*r*	.01	.22	.23	.05	-.29	-.17
	p	.96	.19	.15	.75	.07	.29
	N	39	39	39	39	39	39
Fear of Noises	*r*	.08	.12	.16	-.04	-.13	-.07
	p	.61	.46	.32	.80	.44	.67
	N	39	39	39	39	39	39
Classic Phobias	*r*	.05	.15	**.32(*)**	.01	-.20	-.22
	p	.76	.36	**.05**	.94	.21	.17
	N	39	39	39	39	39	39

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 3.24 Personality factors and startle response modification percentage change

Personality Factors		PPI 80	PPI 85	PPI 90	PPF 80	PPF 85	PPF 90
State Anxiety	*r*	.17	.08	.02	-.09	.05	.(a)
	p	.30	.64	.88	.74	.91	.
	N	40	40	40	15	7	1
Trait Anxiety	*r*	.07	.05	.09	-.27	.20	.(a)
	p	.68	.76	.56	.35	.70	.
	N	39	39	39	14	6	1
Positive Affect	*r*	-.05	-.15	.02	-.02	-.17	.(a)
	p	.76	.35	.91	.94	.71	.
	N	40	40	40	15	7	1
Negative Affect	*r*	.14	-.04	.16	-.37	-.20	.(a)
	p	.37	.82	.31	.17	.66	.
	N	40	40	40	15	7	1
BAS Drive	*r*	-.20	-.23	-.31	-.05	-.47	.(a)
	p	.23	.17	.05	.86	.35	.
	N	39	39	39	14	6	1
BAS Fun	*r*	-.20	-.10	-.12	-.16	-.71	.(a)
	p	.21	.57	.45	.58	.12	.
	N	39	39	39	14	6	1
BAS Reward	*r*	-.26	-.16	.05	.07	-.58	.(a)
	p	.11	.33	.74	.82	.22	.
	N	39	39	39	14	6	1
BIS	*r*	-.06	-.12	.12	.02	.17	.(a)
	p	.72	.47	.46	.95	.75	.
	N	39	39	39	14	6	1
Unusual Experiences	*r*	.20	.03	.02	-.24	.60	.(a)
	p	.22	.85	.88	.40	.21	.
	N	39	39	39	14	6	1

Cognitive Disorganisation	*r*	.02	-.10	.11	-.01	.09	.(a)
	p	.90	.54	.52	.96	.86	.
	N	39	39	39	14	6	1
Introvertive Anhedonia	*r*	.24	.18	.07	.20	.53	.(a)
	p	.15	.26	.68	.49	.28	.
	N	39	39	39	14	6	1
Impulsive Non-conformity	*r*	-.03	-.11	.07	.06	.42	.(a)
	p	.84	.51	.68	.85	.40	.
	N	39	39	39	14	6	1
Novelty Seeking	*r*	-.19	-.21	-.11	.02	-.15	.(a)
	p	.25	.20	.49	.94	.78	.
	N	39	39	39	14	6	1
Harm Avoidance	*r*	.12	.02	.08	-.03	.13	.(a)
	p	.46	.92	.61	.93	.81	.
	N	39	39	39	14	6	1
Reward Dependence	*r*	-.15	-.12	.01	.19	.38	.(a)
	p	.35	.47	.96	.51	.46	.
	N	39	39	39	14	6	1
Persistence	*r*	-.02	.01	-.19	.01	-.15	.(a)
	p	.90	.98	.23	.10	.77	.
	N	39	39	39	14	6	1
Self-Directedness	*r*	-.12	.01	-.11	.03	-.01	.(a)
	p	.47	.96	.50	.91	.99	.
	N	39	39	39	14	6	1
Cooperativeness	*r*	-.10	-.05	-.12	.06	.48	.(a)
	p	.53	.74	.48	.83	.33	.
	N	39	39	39	14	6	1
Self-Transcendence	*r*	-.04	-.16	.04	-.15	-.29	.(a)
	p	.79	.32	.79	.62	.58	.
	N	39	39	39	14	6	1

FSS Total Score	*r*	.03	.03	.06	-.38	.30	.(a)
	p	.87	.85	.70	.17	.56	.
	N	39	39	39	14	6	1
Fear of Animals	*r*	.19	.21	.15	-.46	.70	.(a)
	p	.26	.20	.35	.10	.12	.
	N	39	39	39	14	6	1
Interpersonal Fear	*r*	.05	.01	.07	-.24	.08	.(a)
	p	.78	.94	.66	.41	.88	.
	N	39	39	39	14	6	1
Fear of Tissue Damage	*r*	-.05	-.05	.01	-.40	.44	.(a)
	p	.75	.75	.96	.16	.39	.
	N	39	39	39	14	6	1
Fear of Noises	*r*	.11	.08	.03	-.37	.03	.(a)
	p	.50	.64	.85	.20	.95	.
	N	39	39	39	14	6	1
Classic Phobias	*r*	.07	.09	.07	-.45	.54	.(a)
	p	.66	.59	.67	.10	.27	.
	N	39	39	39	14	6	1

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 3.25 Personality factors comparisons

Personality Factors	*M* Original	*SD* Original	*M* Comparison	*SD* Comparison
State Anxiety	31.70	9.04	36.33	9.63
Trait Anxiety	36.54	8.68	44.55	9.84
Positive Affect	28.97	7.20	29.89	5.11
Negative Affect	12.22	3.30	12.44	3.72
BAS Drive	10.64	2.65	9.22	2.77
BAS Fun	11.54	2.49	11.55	3.21
BAS Reward	17.15	1.91	16.78	2.86
BIS	20.97	2.86	22.44	4.27
Unusual Experiences	4.41	4.39	9.55	5.29
Cognitive Disorganisation	8.49	5.55	13.22	5.61
Introvertive Anhedonia	5.23	3.38	7.33	5.87
Impulsive Non-conformity	7.31	3.04	8.78	2.78
Novelty Seeking	8.97	2.59	10.11	3.79
Harm Avoidance	9.03	4.96	9.11	5.60
Reward Dependence	9.87	2.97	9.00	3.12
Persistence	2.8	1.65	2.78	1.79
Self-Directedness	19.31	4.67	16.56	3.28
Cooperativeness	21.46	2.94	19.44	3.61
Self-Transcendence	3.97	3.34	4.89	4.34
FSS Total Score	96.02	48.60	127.75	76.65
Fear of Animals	8.10	7.18	8.12	7.40
Interpersonal Fear	38.77	17.60	49.75	32.49
Fear of Tissue Damage	30.51	18.05	47.00	21.95
Fear of Noises	3.00	2.89	4.62	3.58
Classic Phobias	11.08	9.77	14.62	13.03

Table 3.26 Proportion of the sample displaying prepulse-elicited responses and startle response modification

Comparison Factor	Main Study	Comparison Study
PPER PP80	30%	44%
PPER PP85	45%	55%
PPER PP90	60%	67%
PPI 80	100%	100%
PPI 85	100%	100%
PPI 90	100%	100%
PPF 80	38%	55%
PPF 85	18%	30%
PPF 90	2.5%	11%

Table 3.27 Prepulse-elicited response probabilities and startle response modification probabilities

Comparison Factor	Main Study	Comparison Study
PPER PP80	8%	8%
PPER PP85	20%	21%
PPER PP90	36%	45%
PPI 80	75%	88%
PPI 85	83%	91%
PPI 90	92%	93%
PPF 80	20%	10%
PPF 85	15%	7%
PPF 90	5%	3%

Table 3.28 Startle response modification percentage change values

Comparison Factor	Main Study	Comparison Study
PPI 80	44%	48%
PPI 85	48%	59%
PPI 90	62%	72%
PPF 80	23%	43%
PPF 85	27%	64%
PPF 90	28%	35%

Appendix 4

Part 1 Figures

Figure 4.1 Prepulse detection rates

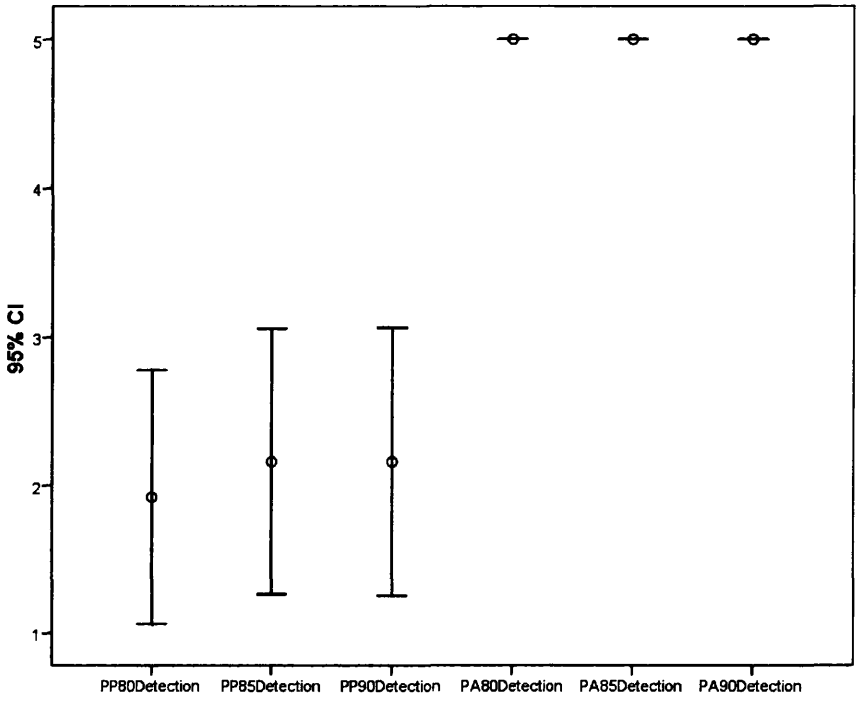


Figure 4.2 Prepulse intensity ratings

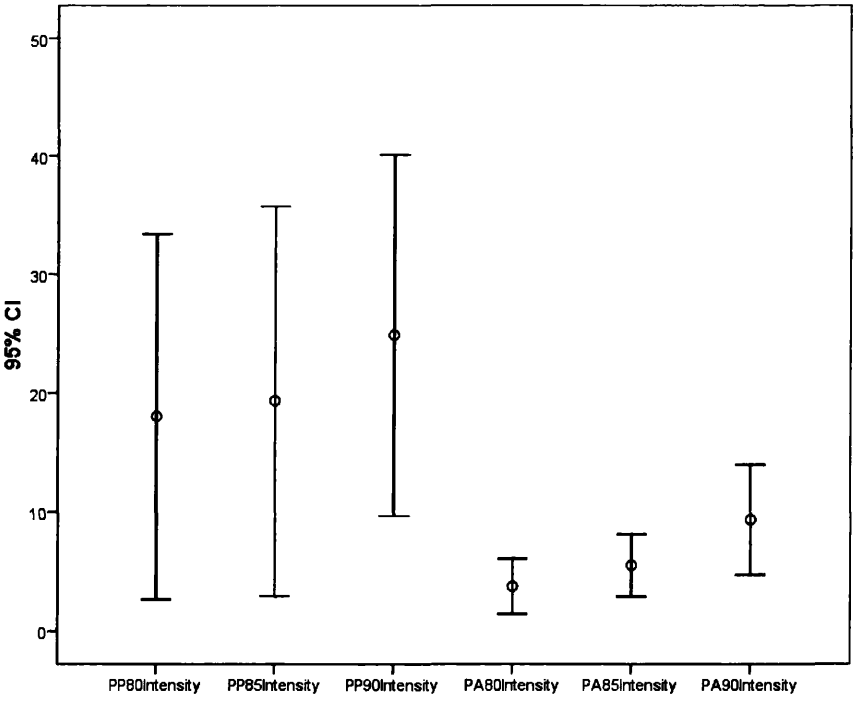
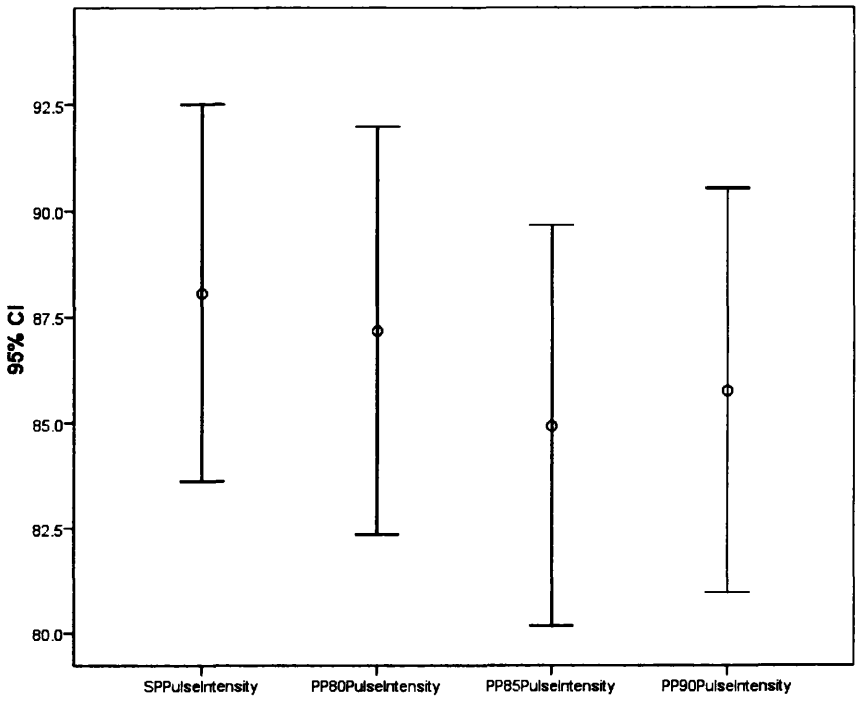


Figure 4.3 Pulse intensity ratings



Part 2 Tables

Table 4.1 Proportion of the sample detecting prepulses and prepulse detection probability [SP = startle probe alone; PP = prepulse and pulse trial; PA = prepulse alone trial; PPx, x = lead stimulus dB]

	Sample Proportion		Prepulse Detection	
Prepulse Intensity and Trial Type	n	(n/25)*100 = %	*M (SD)*	*(M* /5)*100 = %
PP80	13	52	1.92 (2.08)	38.40
PP85	14	56	2.16 (2.17)	43.20
PP90	14	56	2.16 (2.19)	43.20
PA80	25	100	5.00 (0)	100
PA85	25	100	5.00 (0)	100
PA90	25	100	5.00 (0)	100

Table 4.2 Paired-samples t-tests comparing prepulse detection rates (detection frequency) in prepulse and pulse and prepulse-alone trials (significant outcomes marked **bold**)

	Paired Differences					*t*	*df*	*p*
	M	*SD*	*SEM*	95% Confidence Interval of the Difference				
				Lower	Upper			
PP80 – PA80	-2.85	2.01	.39	-3.65	-2.05	**-7.36**	24	**< .01**
PP85 – PA85)	-2.63	2.02	.39	-3.43	-1.83	**-6.76**	24	**< .01**
PP90 – PA90)	-2.44	2.19	.42	-3.31	-1.58	**-5.80**	24	**< .01**

Table 4.3 Prepulse detection rates in prepulse and pulse trials

Prepulse Detection Rates		PP80	PP85	PP90
PP80	*r*	1	**.96(**)**	**.92(**)**
	p		**< .01**	**< .01**
	N	25	25	25
PP85	*r*	**.96(**)**	1	**.98(**)**
	p	**< .01**		**< .01**
	N	25	25	25
PP90	*r*	**.92(**)**	**.98(**)**	1
	p	**< .01**	**< .01**	
	N	25	25	25

** Correlation is significant at the 0.01 level (2-tailed).

Table 4.4 Prepulse intensity judgments (as marked on 100mm VAS)

Prepulse Intensity	*M (SD)*
PP80	18.04 (25.41)
PP85	18.72 (26.16)
PP90	23.71 (25.41)
PA80	3.70 (3.20)
PA85	4.51 (3.62)
PA90	8.42 (6.29)

Table 4.5 Paired-samples t-test comparing intensity judgments in prepulse and pulse with prepulse-alone trials

	Paired Differences					*t*	*df*	*p*
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
PP80 – PA80	14.27	26.23	7.28	-1.58	30.13	1.96	12	.07
PP85 – PA85	13.12	27.72	7.41	-2.88	29.12	1.77	13	.10
PP90 – PA90	14.21	28.93	7.73	-2.49	30.91	1.84	13	.09

Table 4.6 Prepulse intensity ratings correlations

Prepulse Intensity Ratings		PP80	PP85	PP90	PA80	PA85	PA90
PP80	*r*	1	**.97(**)**	**.95(**)**	-.14	-.37	-.46
	p		**< .01**	**< .01**	.64	.22	.11
	N	13	13	13	13	13	13
PP85	*r*	**.97(**)**	1	**.93(**)**	-.08	-.31	-.39
	p	**< .01**		**< .01**	.79	.28	.17
	N	13	14	14	14	14	14
PP90	*r*	**.95(**)**	**.93(**)**	1	-.18	-.43	-.49
	p	**< .01**	**< .01**		.55	.126	.07
	N	13	14	14	14	14	14
PA80	*r*	-.14	-.08	-.176	1	**.41(*)**	.39
	p	.64	.79	.55		**.04**	.06
	N	13	14	14	25	25	25
PA85	*r*	-.37	-.31	-.43	**.41(*)**	1	**.77(**)**
	p	.22	.28	.17	**.04**		**< .01**
	N	13	14	14	25	25	25
PA90	*r*	-.46	-.39	-.42	.39	**.77(**)**	1
	p	.11	.17	.075	.06	**< .01**	
	N	13	14	14	25	25	25

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 4.7 Prepulse detection rates and prepulse intensity ratings correlations

Prepulse Detection/Intensity Ratings		Detection PP80	Detection PP85	Detection PP90
Detection PP80	*r*	1	**.96(**)**	**.92(**)**
	p		**< .01**	**< .01**
	N	25	25	25
Detection PP85	*r*	**.96(**)**	1	**.98(**)**
	p	**< .01**		**< .01**
	N	25	25	25
Detection PP90	*r*	**.92(**)**	**.98(**)**	1
	p	**< .01**	**< .01**	
	N	25	25	25
PP80	*r*	**-.80(**)**	**-.75(**)**	**-.78(**)**
	p	**.01**	**.01**	**.01**
	N	13	13	13
PP85	*r*	-.50	**-.59(*)**	**-.73(**)**
	p	.07	**.03**	**< .01**
	N	14	14	14
PP90	*r*	**-.53**	**-.62(*)**	**-.68(**)**
	p	**.05**	**.02**	**.01**
	N	14	14	14
PA80	*r*	.01	-.04	-.05
	p	.96	.86	.79
	N	25	25	25
PA85	*r*	**.46(*)**	**.42(*)**	**.42(*)**
	p	**.02**	**.04**	**.03**
	N	25	25	25
PA90	*r*	.32	.26	.28
	p	.12	.21	.18
	N	25	25	25

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 4.8 Proportion of the sample detecting pulse stimuli and pulse detection probability

	Sample Proportion		Pulse Detection	
Trial Type	n	(n/25)*100 = %	*M (SD)*	*(M* /5)*100 = %
PP80	25	100	4.96 (.20)	99.92
PP85	25	100	5.00 (0)	100
PP90	25	100	4.96 (.20)	99.92
SP	25	100	5.00 (0)	100

Table 4.9 Paired-samples t-test comparison of pulse intensity ratings in pulse-alone and prepulse and pulse trials

Pulse Intensity Ratings	Paired Differences					*t*	*df*	*p*
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
SP - PP80	.89	4.47	.89	-.95	2.73	.99	24	.33
SP - PP85	3.14	6.85	1.37	.31	5.97	**2.29**	24	**.03**
SP - PP90	2.31	4.83	.97	.31	4.30	**2.39**	24	**.02**

Table 4.10 Prepulse detection and stimulus intensity ratings modification percentage change correlations

IRM Percentage		Detection PP80	Detection P85	Detection PP 90
PPIPSI 80	*r*	.12	.11	.08
	p	.59	.63	.70
	N	23	23	23
PPIPSI 85	*r*	-.34	-.35	-.38
	p	.11	.09	.06
	N	24	24	24
PPIPSI 90	*r*	-.24	-.24	-.23
	p	.25	.24	.27
	N	25	25	25
PPFPSI80	*r*	**.45(*)**	.33	.30
	p	**.03**	.12	.16
	N	24	24	24
PPFPSI85	*r*	.34	.27	.25
	p	.11	.22	.26
	N	23	23	23
PPFPSI90	*r*	**.46(*)**	**.44(*)**	**.41(*)**
	p	**.02**	**.03**	**.05**
	N	24	24	24

Table 4.11 Prepulse detection and intensity ratings modification probability correlations

IRM Probability		Detection PP80	Detection P85	Detection PP 90
PPIPSI 80	*r*	-.21	-.18	-.17
	p	.32	.40	.40
	N	25	25	25
PPIPSI 85	*r*	.[a]	.[a]	.[a]
	p	.	.	.
	N	25	25	25
PPIPSI 90	*r*	-.21	-.18	-.17
	p	.32	.40	.40
	N	25	25	25
PPFPSI80	*r*	**-.43***	-.36	-.30
	p	**.03**	.07	.14
	N	25	25	25
PPFPSI85	*r*	-.04	.07	.14
	p	.86	.73	.51
	N	25	25	25
PPFPSI90	*r*	.07	.15	.17
	p	.73	.47	.42
	N	25	25	25

Table 4.12 Personality factors and prepulse detection and perceived stimulus intensity ratings correlations

Personality Factors		Detection PP80	Detection PP85	Detection PP90	Intensity PP80	Intensity PP85	Intensity PP90	Intensity PA80	Intensity PA85	Intensity PA90
State Anxiety	*r*	-.07	-.08	-.02	-.33	-.37	-.12	.16	-.15	-.15
	p	.75	.72	.91	.28	.19	.69	.46	.48	.49
	N	24	24	24	13	14	14	24	24	24
Trait Anxiety	*r*	-.13	-.18	-.18	-.04	-.03	.09	.17	-.01	-.06
	p	.55	.40	.40	.89	.93	.76	.41	.97	.78
	N	25	25	25	13	14	14	25	25	25
Positive Affect	*r*	-.09	-.08	-.09	.02	-.02	-.14	-.27	-.17	-.18
	p	.67	.69	.67	.94	.94	.63	.19	.40	.39
	N	25	25	25	13	14	14	25	25	25
Negative Affect	*r*	-.01	-.17	-.16	.03	.01	.13	.27	-.03	-.06
	p	.65	.43	.43	.92	.74	.66	.19	.88	.77
	N	25	25	25	13	14	14	25	25	25
BAS Drive	*r*	-.04	.02	.06	-.05	.06	.06	-.13	.14	.19
	p	.83	.93	.76	.86	.83	.83	.52	.51	.36
	N	25	25	25	13	14	14	25	25	25
BAS Fun	*r*	-.01	.07	.09	-.09	.03	-.07	-.09	.12	.11
	p	.96	.73	.65	.77	.91	.81	.65	.57	.59
	N	25	25	25	13	14	14	25	25	25
BAS Reward	*r*	-.17	-.18	-.17	-.06	.07	.01	-.23	-.06	-.13
	p	.41	.40	.42	.84	.82	.99	.27	.77	.52
	N	25	25	25	13	14	14	25	25	25
BIS	*r*	.18	.10	.05	-.10	-.03	-.03	-.06	.01	.06
	p	.40	.63	.83	.75	.93	.91	.78	.99	.77
	N	23	23	23	12	13	13	23	23	23
Unusual Experiences	*r*	-.31	**-.41(*)**	-.38	-.22	-.21	-.24	.16	.21	.28
	p	.13	**.04**	.06	.47	.48	.41	.43	.31	.17
	N	25	25	25	13	14	14	25	25	25
Cognitive	*r*	-.09	-.17	-.18	.06	.11	.19	.08	.07	.03

Disorganisation										
	p	.66	.41	.40	.85	.72	.52	.72	.76	.89
	N	25	25	25	13	14	14	25	25	25
Introvertive Anhedonia	*r*	-.16	-.22	-.23	.49	.40	**.55(*)**	-.10	-.30	-.28
	p	.44	.29	.27	.09	.16	**.04**	.63	.15	.18
	N	25	25	25	13	14	14	25	25	25
Impulsive Non-conformity	*r*	.09	.14	.17	.14	.21	.19	.03	.39	.31
	p	.67	.52	.41	.64	.46	.51	.87	.05	.13
	N	25	25	25	13	14	14	25	25	25
Novelty Seeking	*r*	-.21	-.20	-.15	-.09	.06	-.04	-.06	.16	.25
	p	.30	.35	.47	.76	.84	.88	.79	.43	.22
	N	25	25	25	13	14	14	25	25	25
Harm Avoidance	*r*	.05	-.02	-.06	.30	.30	.41	.18	-.10	-.15
	p	.81	.92	.79	.32	.30	.15	.40	.63	.48
	N	25	25	25	13	14	14	25	25	25
Reward Dependence	*r*	**.43(*)**	.39	.38	**-.76(**)**	**-.63(*)**	-.53	.06	.12	.03
	p	**.03**	.06	.06	**.01**	**.02**	.05	.78	.58	.88
	N	25	25	25	13	14	14	25	25	25
Persistence	*r*	-.24	-.27	-.25	-.32	-.32	-.25	.24	-.18	-.20
	p	.25	.18	.22	.28	.27	.39	.25	.39	.33
	N	25	25	25	13	14	14	25	25	25
Self-Directedness	*r*	.02	.07	.08	-.21	-.20	-.23	-.21	-.09	-.06
	p	.92	.74	.70	.49	.49	.42	.31	.67	.78
	N	25	25	25	13	14	14	25	25	25
Cooperativeness	*r*	.01	-.03	.02	**-.88(**)**	**-.87(**)**	**-.85(**)**	.17	.20	.20
	p	.96	.87	.94	**p < .001**	**p < .001**	**p < .001**	.425	.35	.35

	N	24	24	24	13	14	14	24	24	24
Self-Transcendence	*r*	**-.41(*)**	**-.52(**)**	**-.51(**)**	-.09	-.06	-.13	.18	.06	.26
	p	**.04**	**.01**	**.01**	.76	.83	.65	.38	.79	.20
	N	25	25	25	13	14	14	25	25	25
FSS Total Score	*r*	-.18	-.29	-.34	-.07	.03	-.03	.20	-.02	-.06
	p	.42	.17	.11	.82	.92	.93	.35	.94	.80
	N	23	23	23	12	13	13	23	23	23
Fear of Animals	*r*	-.25	-.31	-.33	.23	.38	.28	-.13	-.15	-.15
	p	.24	.14	.10	.45	.18	.32	.55	.48	.47
	N	25	25	25	13	14	14	25	25	25
Interpersonal Fear	*r*	-.15	-.24	-.26	-.11	-.05	-.07	.20	-.05	-.04
	p	.49	.28	.23	.73	.87	.82	.35	.83	.85
	N	23	23	23	12	13	13	23	23	23
Fear of Tissue Damage	*r*	-.13	-.25	-.33	-.10	.05	-.07	.26	.11	.08
	p	.55	.23	.12	.75	.88	.81	.22	.60	.73
	N	24	24	24	12	13	13	24	24	24
Fear of Noises	*r*	-.16	-.24	-.29	.47	**.58(*)**	.49	.05	-.10	-.18
	p	.45	.25	.16	.10	**.03**	.08	.82	.62	.40
	N	25	25	25	13	14	14	25	25	25
Classic Phobias	*r*	-.01	-.12	-.15	-.11	-.05	-.05	.38	.11	-.06
	p	.99	.58	.48	.73	.86	.86	.07	.61	.76
	N	24	24	24	13	14	14	24	24	24

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 4.13 Personality factors and intensity ratings modification percentage change correlations

Personality Factors		PPIPSI 80	PPIPSI 85	PPIPSI 90	PPFPSI 80	PPFPSI 85	PPFPSI 90
State Anxiety	*r*	-.02	.07	.07	.11	-.15	.07
	p	.92	.74	.75	.62	.51	.74
	N	23	24	23	22	23	24
Trait Anxiety	*r*	.05	.19	.13	.09	.03	.19
	p	.82	.37	.54	.67	.87	.37
	N	24	25	24	23	24	25
Positive Affect	*r*	.02	.02	-.17	**-.50(*)**	-.25	.02
	p	.91	.93	.43	**.02**	.23	.93
	N	24	25	24	23	24	25
Negative Affect	*r*	**.41(*)**	**.44 (*)**	**.44(*)**	.11	.15	**.44(*)**
	p	**.05**	**.03**	**.03**	.61	.50	**.03**
	N	24	25	24	23	24	25
BAS Drive	*r*	-.26	-.10	.12	.29	.02	-.10
	p	.22	.62	.57	.17	.91	.62
	N	24	25	24	23	24	25
BAS Fun	*r*	-.17	.11	-.02	-.01	.09	.11
	p	.42	.62	.91	.95	.68	.62
	N	24	25	24	23	24	25
BAS Reward	*r*	-.01	.37	.19	.27	.15	.37
	p	1.00	.07	.38	.22	.50	.07
	N	24	25	24	23	24	25
BIS	*r*	-.29	-.14	.08	.15	.26	-.14
	p	.18	.54	.72	.51	.23	.54
	N	23	23	22	21	22	23
Unusual Experiences	*r*	.20	.25	-.06	-.02	-.30	.25
	p	.34	.23	.77	.93	.16	.23
	N	24	25	24	23	24	25
Cognitive Disorganisation	*r*	-.04	.20	.19	.02	-.02	.20
	p	.86	.35	.37	.93	.91	.35
	N	24	25	24	23	24	25
Introvertive Anhedonia	*r*	.05	-.05	-.10	.05	-.22	-.05
	p	.82	.81	.64	.82	.29	.81
	N	24	25	24	23	24	25
Impulsive Non-	*r*	-.02	.26	.03	-.16	.01	.26

conformity							
	p	.94	.20	.87	.47	.10	.20
	N	24	25	24	23	24	25
Novelty Seeking	*r*	-.01	-.03	.16	.20	-.03	-.03
	p	.97	.88	.44	.36	.88	.88
	N	24	25	24	23	24	25
Harm Avoidance	*r*	-.03	.17	.13	.13	.16	.17
	p	.91	.42	.53	.57	.45	.42
	N	24	25	24	23	24	25
Reward Dependence	*r*	-.15	-.06	**.64(**)**	**.44(*)**	.40	-.06
	p	.48	.78	**.01**	**.03**	.05	.78
	N	24	25	24	23	24	25
Persistence	*r*	.06	.21	.01	-.01	-.05	.21
	p	.76	.32	.96	.96	.80	.32
	N	24	25	24	23	24	25
Self-Directedness	*r*	.12	-.08	-.12	-.13	-.07	-.08
	p	.56	.71	.62	.55	.75	.71
	N	24	25	24	23	24	25
Cooperativeness	*r*	-.09	.16	.38	.04	.20	.16
	p	.68	.44	.08	.87	.35	.44
	N	23	24	23	22	23	24
Self-Transcendence	*r*	.13	.25	-.03	.01	-.36	.25
	p	.53	.22	.89	.98	.08	.22
	N	24	25	24	23	24	25
FSS Total Score	*r*	.16	.21	.40	.22	.14	.21
	p	.48	.34	.07	.31	.54	.34
	N	22	23	22	22	22	23
Fear of Animals	*r*	.09	.01	**.45(*)**	.34	.20	.01
	p	.69	.96	**.03**	.11	.34	.96
	N	24	25	24	23	24	25
Interpersonal Fear	*r*	.11	.19	.32	.20	.22	.19
	p	.64	.38	.15	.38	.31	.38
	N	22	23	22	22	22	23
Fear of Tissue Damage	*r*	.02	.13	.32	.13	.00	.13
	p	.93	.54	.14	.55	1.00	.54
	N	23	24	23	22	23	24
Fear of Noises	*r*	.22	.08	.15	.34	.02	.08
	p	.30	.72	.49	.11	.91	.72
	N	24	25	24	23	24	25

Classic Phobias	*r*	.04	.18	.27	.10	-.15	.18
	p	.86	.41	.21	.68	.49	.41
	N	23	24	23	22	23	24

Table 4.14 Personality factors and intensity ratings modification probability correlations

Personality Factors		PPIPSI 80	PPIPSI 85	PPIPSI 90	PPFPSI 80	PPFPSI 85	PPFPSI 90
State Anxiety	*r*	-.16	-.09	-.22	.21	.08	.31
	p	.45	.66	.31	.32	.71	.14
	N	24	24	24	24	24	24
Trait Anxiety	*r*	.15	.06	-.08	-.18	-.04	.08
	p	.47	.77	.70	.38	.84	.69
	N	25	25	25	25	25	25
Positive Affect	*r*	.08	-.18	-.15	-.03	.12	.17
	p	.70	.39	.47	.87	.55	.41
	N	25	25	25	25	25	25
Negative Affect	*r*	.39	.21	.05	-.39	-.22	-.09
	p	.05	.31	.82	.06	.30	.68
	N	25	25	25	25	25	25
BAS Drive	*r*	-.28	-.14	-.07	.21	.19	-.05
	p	.17	.50	.75	.32	.36	.82
	N	25	25	25	25	25	25
BAS Fun	*r*	-.11	-.03	.10	.06	.08	-.19
	p	.61	.88	.63	.76	.69	.36
	N	25	25	25	25	25	25
BAS Reward	*r*	.11	.05	.14	-.10	-.01	-.27
	p	.58	.80	.50	.62	.99	.20
	N	25	25	25	25	25	25
BIS	*r*	.11	.28	-.14	-.17	-.29	.05
	p	.62	.19	.53	.43	.18	.83
	N	23	23	23	23	23	23
Unusual Experiences	*r*	.18	.05	.03	-.10	-.02	.02
	p	.38	.80	.89	.64	.91	.93
	N	25	25	25	25	25	25
Cognitive Disorganisation	*r*	.23	.04	-.12	-.25	-.01	.10
	p	.27	.83	.55	.23	.95	.62
	N	25	25	25	25	25	25
Introvertive Anhedonia	*r*	-.12	-.14	-.11	.14	.17	.01
	p	.57	.49	.61	.49	.43	.94
	N	25	25	25	25	25	25
Impulsive Non-conformity	*r*	.25	.06	-.20	-.30	-.02	.13

	p	.22	.78	.35	.14	.91	.53
	N	25	25	25	25	25	25
Novelty Seeking	*r*	-.11	-.11	-.09	.08	.18	-.07
	p	.60	.60	.65	.70	.40	.73
	N	25	25	25	25	25	25
Harm Avoidance	*r*	.31	.14	-.05	-.36	-.13	-.01
	p	.13	.50	.82	.08	.52	.96
	N	25	25	25	25	25	25
Reward Dependence	*r*	.08	-.05	.06	-.08	.01	.03
	p	.72	.81	.78	.69	.99	.90
	N	25	25	25	25	25	25
Persistence	*r*	-.08	.27	.12	.08	-.31	-.06
	p	.71	.18	.58	.68	.13	.78
	N	25	25	25	25	25	25
Self-Directedness	*r*	-.25	-.16	.15	.31	.12	-.09
	p	.22	.43	.47	.13	.58	.66
	N	25	25	25	25	25	25
Cooperativeness	*r*	.16	.23	**.42(*)**	-.06	-.24	-.24
	p	.45	.27	**.04**	.78	.26	.27
	N	24	24	24	24	24	24
Self-Transcendence	*r*	-.04	.08	.17	.10	-.06	-.18
	p	.85	.71	.41	.64	.79	.37
	N	25	25	25	25	25	25
FSS Total Score	*r*	.25	.17	-.06	-.28	-.16	.03
	p	.25	.43	.77	.19	.45	.91
	N	23	23	23	23	23	23
Fear of Animals	*r*	.14	-.10	-.07	-.17	.11	-.01
	p	.49	.62	.75	.41	.60	.94
	N	25	25	25	25	25	25
Interpersonal Fear	*r*	.24	.30	-.05	-.27	-.28	.03
	p	.27	.16	.81	.20	.20	.89
	N	23	23	23	23	23	23
Fear of Tissue Damage	*r*	.20	.16	-.13	-.25	-.16	.07
	p	.34	.46	.55	.24	.44	.75
	N	24	24	24	24	24	24
Fear of Noises	*r*	.19	-.05	-.24	-.22	.08	.08
	p	.36	.81	.25	.28	.70	.70
	N	25	25	25	25	25	25
Classic Phobias	*r*	.10	.02	-.05	-.14	-.02	.02

	p	.63	.93	.83	.51	.91	.91
	N	24	24	24	24	24	24

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 4.15 Demographic factors (sex, age) and prepulse detection and intensity ratings correlations

Prepulse Detection/Intensity Ratings		Sex	Age
Detection PP80	*r*	.24	-.21
	p	.24	.30
	N	25	25
Detection PP85	*r*	.25	-.22
	p	.24	.29
	N	25	25
Detection PP90	*r*	.24	-.22
	p	.24	.30
	N	25	25
Intensity PP80	*r*	.05	-.36
	p	.86	.22
	N	13	13
Intensity PP85	*r*	.03	-.33
	p	.92	.24
	N	14	14
Intensity PP90	*r*	.02	-.44
	p	.93	.12
	N	14	14
Intensity PA80	*r*	-.23	-.30
	p	.26	.14
	N	25	25
Intensity PA85	*r*	.19	-.29
	p	.37	.16
	N	25	25
Intensity PA90	*r*	.32	-.22
	p	.12	.29
	N	25	25

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 4.16 Demographic factors (sex, age) and intensity ratings modification percentage change correlations

Intensity Ratings Modification Percentage		sex	age
PPIPSI 80	*r*	-.02	.39
	p	.92	.06
	N	23	23
PPIPSI 85	*r*	-.35	.07
	p	.09	.74
	N	24	24
PPIPSI 90	*r*	-.33	.36
	p	.10	.07
	N	25	25
PPFPSI 80	*r*	-.08	-.31
	p	.71	.14
	N	24	24
PPFPSI 85	*r*	.04	**-.52(*)**
	p	.86	**.01**
	N	23	23
PPFPSI 90	*r*	-.09	-.11
	p	.69	.61
	N	24	24

** Correlation is significant at the 0.01 level (2-tailed).)

* Correlation is significant at the 0.05 level (2-tailed).

Table 4.17 Demographic factors and PSI modification (IRM) probability correlations

Intensity Ratings Modification Probability		sex	age
PPIPSI 80	*r*	.01	.07
	p	.99	.74
	N	25	25
PPIPSI 85	*r*	-.24	.10
	p	.25	.64
	N	25	25
PPIPSI 90	*r*	-.06	.17
	p	.79	.41
	N	25	25
PPFPSI 80	*r*	-.01	-.03
	p	.95	.89
	N	25	25
PPFPSI 85	*r*	.25	-.10
	p	.24	.63
	N	25	25
PPFPSI 90	*r*	-.01	-.14
	p	.96	.49
	N	25	25

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 4.18 Motor response amplitudes in the comparison EMG recording and intensity judgment (IN) parts

PPER Amplitude	Paired Differences					*t*	*df*	*p*
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
PP80 – IN PP80	25.62	19.39	9.69	-5.22	56.47	2.64	3	.08
PP85 – IN PP85	10.167	16.82	8.41	-16.60	36.93	1.21	3	.31
PP90 – IN PP90	35.02	49.15	15.54	-.15	70.18	**2.25**	9	**.05**
PA80 – IN PA80	30.67	26.41	15.25	-34.93	96.26	2.01	2	.18
PA85 – IN PA85	8.03	31.36	14.02	-30.90	46.97	.57	4	.60
PA90 – IN PA90	7.97	16.10	6.57	-8.93	24.87	1.21	5	.28
SP – IN SP	22.32	59.06	11.81	-2.05	46.70	1.89	24	.07

Table 4.19 Motor response probabilities in the comparison EMG recording and intensity judgment parts

PPER Probability	Paired Differences					*t*	*df*	*p*
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
PP80 – IN PP80	-.12	.53	.10	-.34	.10	-1.14	24	.26
PP85 – IN PP85	.56	.92	.18	.18	.94	**3.05**	24	**.01**
PP90 – IN PP90	.44	1.00	.20	.03	.85	**2.19**	24	**.04**
PA80 – IN PA80	.12	.73	.14	-.18	.42	.83	24	.42
PA85 – IN PA85	.12	.60	.12	-.13	.37	1.00	24	.33
PA90 – IN PA90	.32	1.18	.24	-.17	.81	1.35	24	.19
SP – IN SP	.36	.99	.20	-.05	.77	1.81	24	.08

Table 4.20 Startle response modification percentage changes in the baseline EMG recording and the intensity judgment (IN) parts of the session

SRM Percentage	Paired Differences					*t*	*df*	*p*
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
PPI 80 – IN PPI 80	-2.53	16.16	3.37	-9.52	4.45	-.75	22	.46
PPI 85 – IN PPI 85	1.73	13.90	2.78	-4.00	7.47	.62	24	.54
PPI 90 – IN PPI 90	2.06	12.72	2.54	-3.19	7.31	.81	24	.43
PPF 80 - IN PPF 80	-22.66	12.89	7.44	-54.68	9.36	-3.04	2	.09
PPF 85 - IN PPF 85	-17.71	11.40	6.58	-46.03	10.61	-2.69	2	.11
PPF 90 - IN PPF 90	-7.22	13.54	7.82	-40.86	26.42	-.92	2	.45

Table 4.21 Startle response modification probabilities in the baseline EMG recording and intensity judgment (IN) parts of the session

SRM Probability	Paired Differences					*t*	*df*	*p*
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
PPI 80 – IN PPI 80	.08	1.35	.27	-.48	.64	.23	24	.77
PPI 85 – IN PPI 85	.04	.89	.18	-.33	.41	.22	24	.82
PPI 90 – IN PPI 90	.16	.94	.19	-.23	.55	.85	24	.40
PPF 80 - IN PPF 80	-.08	1.35	.27	-.64	.48	-.30	24	.77
PPF 85 - IN PPF 85	.16	.94	.19	-.23	.55	.85	24	.40
PPF 90 - IN PPF 90	-.08	.64	.13	-.34	.18	-.62	24	.54

Table 4.22 Prepulse detection rates, prepulse intensity ratings and PPER amplitudes in the baseline EMG recording and the intensity judgment (IN) session parts: correlations

PPER Amplitude		Prepulse Detected PP80	Prepulse Detected PP85	Prepulse Detected PP90	Ratings PP80	Ratings PP85	Ratings PP90	Ratings PA80	Ratings PA85	Ratings PA90
PP80	*r*	.77	.60	.45	**-.97(*)**	-.88	-.79	-.57	.47	.71
	p	.13	.29	.44	**.03**	.11	.21	.32	.42	.18
	N	5	5	5	4	4	4	5	5	5
PP85	*r*	.01	.04	.03	-.14	-.10	-.18	-.21	.24	.02
	p	.97	.91	.91	.74	.82	.66	.49	.43	.94
	N	13	13	13	8	8	8	13	13	13
PP90	*r*	**.68(**)**	**.63(*)**	**.55(*)**	-.49	-.47	-.62	.09	.27	.09
	p	**.01**	**.01**	**.04**	.27	.29	.14	.75	.34	.75
	N	14	14	14	7	7	7	14	14	14
PA80	*r*	.46	.24	.18	.07	-.37	-.37	.08	.31	.55
	p	.25	.57	.67	.93	.63	.63	.85	.45	.16
	N	8	8	8	4	4	4	8	8	8
PA85	*r*	.42	.43	.56	-.42	-.38	-.31	-.50	.29	.69
	p	.30	.28	.15	.40	.46	.54	.21	.48	.06
	N	8	8	8	6	6	6	8	8	8
PA90	*r*	-.01	-.06	-.07	**-.95(*)**	-.63	-.55	-.13	-.19	-.15
	p	.97	.84	.81	**.01**	.18	.26	.65	.49	.60
	N	15	15	15	5	6	6	15	15	15
IN PP80	*r*	.34	.39	.38	.14	-.23	-.17	-.44	-.20	-.12
	p	.36	.30	.31	.82	.71	.78	.23	.61	.75
	N	9	9	9	5	5	5	9	9	9
IN PP85	*r*	**.92(*)**	**.92(*)**	**.92(*)**	.(a)	.(a)	.(a)	-.49	.67	-.09
	p	**.03**	**.03**	**.03**	.	.	.	.40	.22	.89
	N	5	5	5	1	1	1	5	5	5
IN PP90	*r*	.09	.18	.21	-.49	-.92	-.37	-.03	-.18	-.22
	p	.80	.61	.56	.51	.08	.62	.92	.62	.54
	N	10	10	10	4	4	4	10	10	10
IN PA80	*r*	-.23	-.28	-.19	-.63	-.69	-.57	-.25	.20	.44

	p	.62	.55	.68	.37	.31	.43	.59	.67	.32
	N	7	7	7	4	4	4	7	7	7
IN PA85	*r*	.22	.39	.44	.57	.49	.66	.12	-.42	-.61
	p	.64	.39	.32	.43	.51	.34	.80	.35	.14
	N	7	7	7	4	4	4	7	7	7
IN PA90	*r*	.14	.26	.22	.21	.30	.13	-.46	.12	-.20
	p	.73	.54	.60	.73	.62	.84	.26	.77	.63
	N	8	8	8	5	5	5	8	8	8

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

a Cannot be computed because at least one of the variables is constant.

Table 4.23 Prepulse detection, intensity ratings and PPER probabilities in the baseline EMG and intensity judgment (IN) session parts

PPER Probability		Prepulse Detected PP80	Prepulse Detected PP85	Prepulse Detected PP90	Ratings PP80	Ratings PP85	Ratings PP90	Ratings PA80	Ratings PA85	Ratings PA90
PP80	*r*	.35	.37	.37	-.29	-.26	-.12	.03	-.05	.01
	p	.09	.07	.07	.34	.38	.67	.88	.80	.94
	N	25	25	25	13	14	14	25	25	25
PP85	*r*	.28	.25	.16	-.15	-.07	-.14	.04	-.17	-.07
	p	.17	.23	.45	.62	.81	.64	.85	.40	.72
	N	25	25	25	13	14	14	25	25	25
PP90	*r*	.05	.01	-.02	-.14	-.08	-.04	-.07	-.24	-.06
	p	.80	.97	.91	.64	.79	.88	.73	.25	.77
	N	25	25	25	13	14	14	25	25	25
PA80	*r*	-.09	-.10	-.05	-.29	-.31	-.04	.13	-.28	.01
	p	.68	.63	.82	.33	.28	.88	.55	.17	.97
	N	25	25	25	13	14	14	25	25	25
PA85	*r*	.27	.23	.18	-.22	-.16	-.17	.04	-.12	.11
	p	.19	.27	.38	.46	.59	.56	.86	.57	.60
	N	25	25	25	13	14	14	25	25	25
PA90	*r*	-.13	-.12	-.12	-.22	-.18	-.12	-.02	-.24	-.09
	p	.53	.57	.58	.46	.53	.68	.90	.25	.67
	N	25	25	25	13	14	14	25	25	25
IN PP80	*r*	.17	.18	.14	-.33	-.26	-.22	.22	.10	.05
	p	.43	.39	.49	.28	.38	.44	.28	.62	.80
	N	25	25	25	13	14	14	25	25	25
IN PP85	*r*	-.06	-.11	-.19	-.17	-.16	-.23	.20	-.07	-.10
	p	.77	.59	.37	.58	.59	.43	.34	.74	.62
	N	25	25	25	13	14	14	25	25	25
IN PP90	*r*	-.27	-.33	-.34	-.38	-.38	-.41	.29	-.04	.19
	p	.19	.11	.09	.20	.17	.15	.16	.84	.37
	N	25	25	25	13	14	14	25	25	25
IN PA80	*r*	-.05	-.15	-.18	.24	.30	.25	**.52(**)**	.16	.18

	p	.82	.48	.39	.42	.30	.39	**.01**	.44	.38
	N	25	25	25	13	14	14	25	25	25
IN PA85	*r*	.07	.06	.04	-.36	-.30	-.40	.24	.03	.32
	p	.73	.77	.87	.23	.29	.15	.25	.88	.12
	N	25	25	25	13	14	14	25	25	25
IN PA90	*r*	.02	-.01	-.05	-.16	-.14	-.19	.23	-.12	-.03
	p	.91	.10	.82	.60	.62	.52	.27	.55	.88
	N	25	25	25	13	14	14	25	25	25

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 4.24 Prepulse detection rates and percentage change startle response modification in the baseline EMG recording part of the session

SRM Percentage		Detection PP80	Detection PP85	Detection PP90
PPF 80	*r*	.03	.01	-.01
	p	.94	.98	.10
	N	8	8	8
PPF 85	*r*	.32	-.06	-.16
	p	.53	.91	.77
	N	6	6	6
PPF 90	*r*	-.09	-.27	-.26
	p	.88	.66	.68
	N	5	5	5
PPI 80	*r*	-.07	-.07	-.03
	p	.74	.73	.88
	N	24	24	24
PPI 85	*r*	-.01	-.01	.02
	p	.98	.97	.93
	N	25	25	25
PPI 90	*r*	-.09	-.11	-.09
	p	.67	.58	.68
	N	25	25	25

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 4.25 Prepulse detection rates and percentage change startle response modification in the intensity judgment part of the session (IN)

SRM Percentage		Detection PP80	Detection PP85	Detection PP90
IN PPF 80	*r*	.37	.34	.31
	p	.26	.31	.36
	N	11	11	11
IN PPF 85	*r*	.49	.40	.38
	p	.26	.38	.40
	N	7	7	7
IN PPF 90	*r*	.02	-.17	-.08
	p	.97	.74	.88
	N	6	6	6
IN PPI 80	*r*	.05	-.03	-.07
	p	.81	.88	.74
	N	24	24	24
IN PPI 85	*r*	.05	-.04	-.05
	p	.82	.85	.81
	N	25	25	25
IN PPI 90	*r*	-.07	-.14	-.14
	p	.75	.50	.50
	N	25	25	25

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 4.26 Prepulse detection rates and startle response modification probabilities in the baseline EMG recording part of the session

SRM Probability		Detection PP80	Detection PP85	Detection PP90
PPF 80	*r*	-.09	-.06	-.07
	p	.68	.78	.73
	N	25	25	25
PPF 85	*r*	.23	.20	.13
	p	.26	.33	.53
	N	25	25	25
PPF 90	*r*	.15	.15	.07
	p	.47	.48	.74
	N	25	25	25
PPI 80	*r*	-.02	-.02	.02
	p	.91	.94	.92
	N	25	25	25
PPI 85	*r*	-.21	-.17	-.11
	p	.32	.41	.60
	N	25	25	25
PPI 90	*r*	-.18	-.23	-.19
	p	.39	.27	.37
	N	25	25	25

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 4.27 Prepulse detection rates and startle response modification probabilities in the intensity judgment part of the session (IN)

SRM Probability		Detection PP80	Detection PP85	Detection PP90
IN PPF 80	*r*	.10	.13	.07
	p	.62	.52	.73
	N	25	25	25
IN PPF 85	*r*	-.19	-.14	-.16
	p	.37	.50	.43
	N	25	25	25
IN PPF 90	*r*	.13	.15	.11
	p	.54	.46	.59
	N	25	25	25
IN PPI 80	*r*	-.16	-.12	-.07
	p	.46	.56	.75
	N	25	25	25
IN PPI 85	*r*	-.05	-.05	.03
	p	.81	.83	.87
	N	25	25	25
IN PPI 90	*r*	-.28	-.30	-.26
	p	.18	.14	.20
	N	25	25	25

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 4.28 Perceived stimulus intensity modification probabilities and startle response modification percentage and probabilities in the baseline EMG and intensity judgment (IN) session parts

SRM Percentage		Probability PPIPSI80	Probability PPIPSI85	Probability PPIPSI90	Probability PPFPSI80	Probability PPFPSI85	Probability PPFPSI90
Percentage PPF 80	*r*	-.44	-.60	-.61	.59	.60	**.71(*)**
	p	.28	.11	.11	.13	.11	**.05**
	N	8	8	8	8	8	8
Percentage PPF 85	*r*	.24	-.40	-.63	.13	.40	.57
	p	.65	.44	.18	.81	.44	.23
	N	6	6	6	6	6	6
Percentage PPF 90	*r*	-.14	-.45	-.85	.14	.45	.85
	p	.82	.45	.07	.82	.45	.07
	N	5	5	5	5	5	5
Percentage PPI 80	*r*	-.40	-.13	-.04	**.42(*)**	.15	.03
	p	.05	.55	.85	**.04**	.48	.87
	N	24	24	24	24	24	24
Percentage PPI 85	*r*	-.28	-.11	.13	.30	.10	-.14
	p	.18	.59	.54	.15	.62	.49
	N	25	25	25	25	25	25
Percentage PPI 90	*r*	-.28	-.18	-.03	.29	.18	-.01
	p	.18	.40	.89	.16	.39	.97
	N	25	25	25	25	25	25
Probability PPF80	*r*	.21	.23	-.11	-.21	-.23	.13
	p	.32	.27	.61	.32	.28	.54
	N	25	25	25	25	25	25
Probability PPF85	*r*	.02	-.03	.09	-.06	.03	-.08
	p	.93	.88	.68	.79	.88	.71
	N	25	25	25	25	25	25
Probability PPF90	*r*	-.08	.11	.13	.12	-.10	-.07
	p	.71	.61	.54	.57	.62	.75
	N	25	25	25	25	25	25
Probability PPI80	*r*	-.17	-.18	.06	.16	.17	-.10
	p	.41	.40	.77	.45	.40	.64
	N	25	25	25	25	25	25
Probability PPI85	*r*	-.03	.01	-.09	.06	-.01	.08

	p	.87	.95	.65	.76	.95	.71
	N	25	25	25	25	25	25
Probability PPI90	*r*	-.01	-.23	-.21	-.01	.20	.16
	p	.98	.27	.32	.96	.35	.43
	N	25	25	25	25	25	25
IN Percentage PPF 80	*r*	.19	-.19	.03	-.19	.08	-.03
	p	.58	.58	.94	.58	.80	.94
	N	11	11	11	11	11	11
IN Percentage PPF 85	*r*	.45	-.07	-.15	-.45	-.04	.15
	p	.32	.88	.74	.32	.93	.74
	N	7	7	7	7	7	7
IN Percentage PPF 90	*r*	.38	-.54	-.65	-.48	.39	.57
	p	.46	.27	.16	.33	.44	.24
	N	6	6	6	6	6	6
IN Percentage PPI 80	*r*	**-.45(*)**	-.13	-.04	**.47(*)**	.11	-.04
	p	**.02**	.58	.85	**.03**	.62	.87
	N	22	22	22	22	22	22
IN Percentage PPI 85	*r*	-.19	-.20	-.10	.16	.20	-.02
	p	.42	.37	.67	.48	.40	.91
	N	21	21	21	21	21	21
IN Percentage PPI 90	*r*	-.35	.01	.08	.39	-.02	-.07
	p	.15	.96	.75	.11	.93	.79
	N	18	18	18	18	18	18
IN Probability PPF80	*r*	.00	.13	.13	.06	-.15	-.04
	p	1.00	.52	.52	.77	.46	.84
	N	25	25	25	25	25	25
IN Probability PPF85	*r*	.21	.20	.06	-.16	-.24	.01
	p	.31	.32	.76	.44	.25	.97
	N	25	25	25	25	25	25
IN Probability PPF90	*r*	.14	.20	.29	-.14	-.23	-.26
	p	.50	.33	.16	.52	.27	.22
	N	25	25	25	25	25	25
IN Probability PPI80	*r*	.00	-.02	.05	-.07	.04	-.14
	p	1.00	.92	.81	.74	.85	.49
	N	25	25	25	25	25	25

IN Probability PPI85	*r*	-.06	-.16	-.05	.06	.19	.02
	p	.77	.44	.82	.77	.36	.92
	N	25	25	25	25	25	25
IN Probability PPI90	*r*	-.02	-.06	-.18	.09	.09	.15
	p	.90	.76	.39	.68	.67	.47
	N	25	25	25	25	25	25

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 4.29 Perceived stimulus intensity ratings modification percentage change and startle response modifications percentage and probabilities in the baseline EMG and intensity judgment (IN) session parts

SRM		Percentage PPIPSI80	Percentage PPIPSI85	Percentage PPIPSI90	Percentage PPFPSI80	Percentage PPFPSI85	Percentage PPFPSI90
Percentage PPF 80	*r*	.21	.02	.27	**.77(*)**	.66	.65
	p	.62	.96	.56	**.03**	.07	.08
	N	8	8	7	8	8	8
Percentage PPF 85	*r*	.54	**.83(*)**	.49	.69	.03	.10
	p	.26	**.04**	.41	.13	.95	.85
	N	6	6	5	6	6	6
Percentage PPF 90	*r*	.74	.25	-.21	.51	.30	.31
	p	.26	.68	.78	.38	.63	.62
	N	4	5	4	5	5	5
Percentage PPI 80	*r*	-.01	-.07	.17	-.08	.09	.11
	p	.95	.76	.43	.72	.67	.60
	N	23	22	23	22	23	24
Percentage PPI 85	*r*	.13	.08	.21	-.12	.11	-.03
	p	.54	.71	.33	.58	.62	.87
	N	24	23	24	23	24	25
Percentage PPI 90	*r*	.29	.15	.23	-.03	.04	.01
	p	.17	.51	.28	.90	.84	.96
	N	24	23	24	23	24	25
Probability PPF80	*r*	-.37	-.27	-.37	.11	-.01	.11
	p	.07	.21	.07	.62	.10	.59
	N	24	23	24	23	24	25
Probability PPF85	*r*	-.14	.01	.04	-.04	-.15	.01
	p	.51	.94	.84	.85	.49	.10
	N	24	23	24	23	24	25
Probability PPF90	*r*	-.30	-.25	-.22	.22	.10	.15
	p	.15	.24	.30	.32	.66	.46
	N	24	23	24	23	24	25
Probability PPI80	*r*	.36	.22	.30	-.09	.05	-.07
	p	.08	.31	.16	.69	.80	.75
	N	24	23	24	23	24	25
Probability PPI85	*r*	.20	.02	.01	.06	.16	.02

	p	.35	.91	.94	.80	.46	.92
	N	24	23	24	23	24	25
Probability PPI90	*r*	.32	.18	.12	-.010	.04	-.12
	p	.13	.40	.57	.65	.86	.57
	N	24	23	24	23	24	25
IN Percentage PPF 80	*r*	-.08	-.13	-.15	.01	.41	-.19
	p	.84	.72	.67	.98	.24	.57
	N	10	10	10	10	10	11
IN Percentage PPF 85	*r*	.52	.35	.17	**.83(*)**	.05	-.44
	p	.23	.50	.75	**.04**	.93	.32
	N	7	6	6	6	6	7
IN Percentage PPF 90	*r*	.80	.78	.49	-.03	-.39	-.29
	p	.05	.06	.40	.96	.52	.58
	N	6	6	5	5	5	6
IN Percentage PPI 80	*r*	.22	.15	.35	-.02	.27	.19
	p	.35	.51	.11	.92	.23	.40
	N	21	20	21	20	21	22
IN Percentage PPI 85	*r*	-.01	.13	-.02	.03	.42	.06
	p	.98	.58	.92	.89	.06	.81
	N	20	20	20	19	20	21
IN Percentage PPI 90	*r*	-.11	-.03	-.08	.02	.01	.01
	p	.68	.92	.77	.95	.98	.10
	N	17	16	17	17	17	18
IN Probability PPF80	*r*	-.15	-.03	.04	.01	.20	.13
	p	.47	.90	.84	.96	.34	.55
	N	24	23	24	23	24	25
IN Probability PPF85	*r*	-.31	-.22	-.28	-.03	.17	-.01
	p	.13	.31	.19	.90	.43	.98
	N	24	23	24	23	24	25
IN Probability PPF90	*r*	-.39	-.20	-.17	-.05	-.05	-.04

	p	.06	.37	.44	.82	.81	.85
	N	24	23	24	23	24	25
IN Probability PPI80	*r*	.08	-.09	-.07	.03	-.08	-.03
	p	.71	.67	.74	.87	.72	.87
	N	24	23	24	23	24	25
IN Probability PPI85	*r*	.16	.09	.12	.09	.02	.15
	p	.45	.68	.56	.69	.91	.46
	N	24	23	24	23	24	25
IN Probability PPI90	*r*	.22	.07	-.02	.08	.26	.10
	p	.29	.73	.93	.70	.22	.63
	N	24	23	24	23	24	25

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Appendix 5

Part 1: Figures
Trial codes for all the figures: PPx = prepulse and pulse trials; PAx = prepulse-alone trials; x = prepulse intensity

Figure 5.1 PPER probabilities

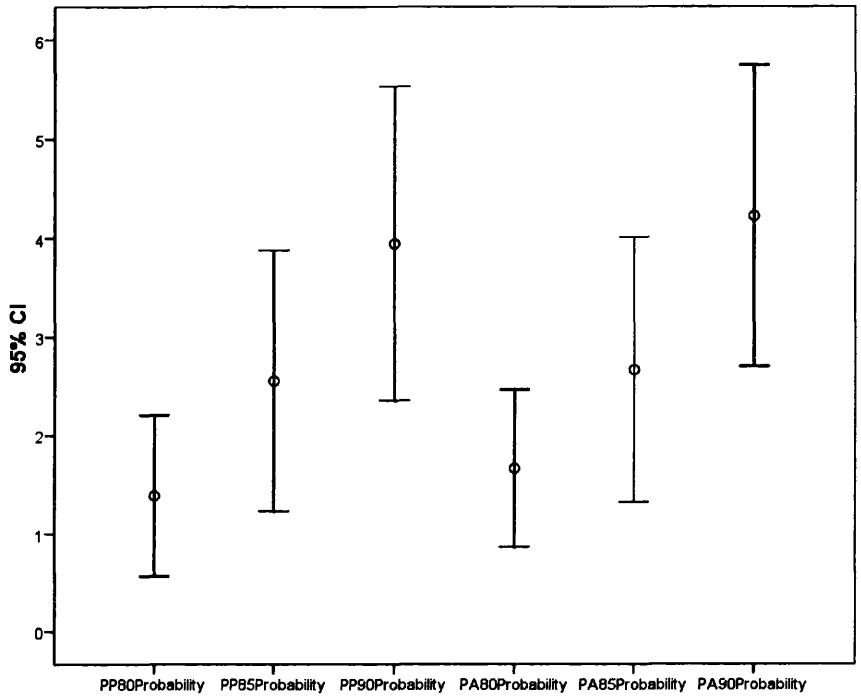


Figure 5.2 PPER amplitudes

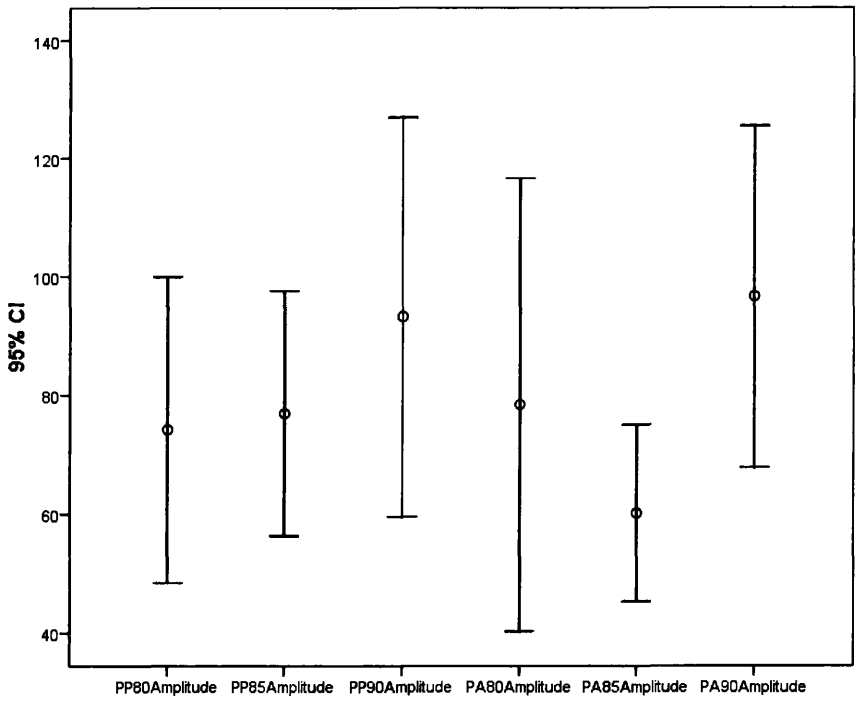


Figure 5.3 PPER onsets

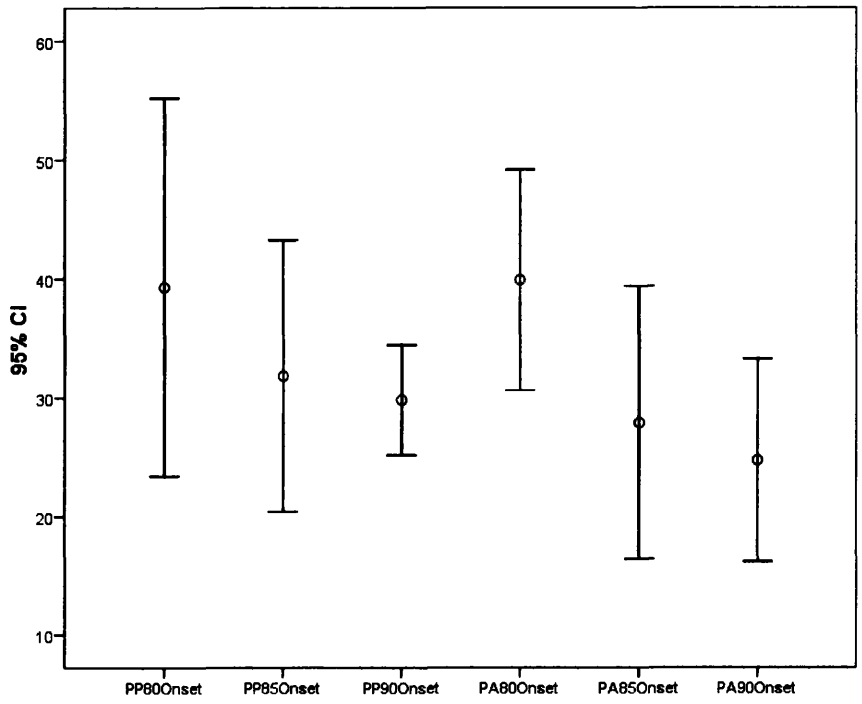


Figure 5.4 PPER peak latencies

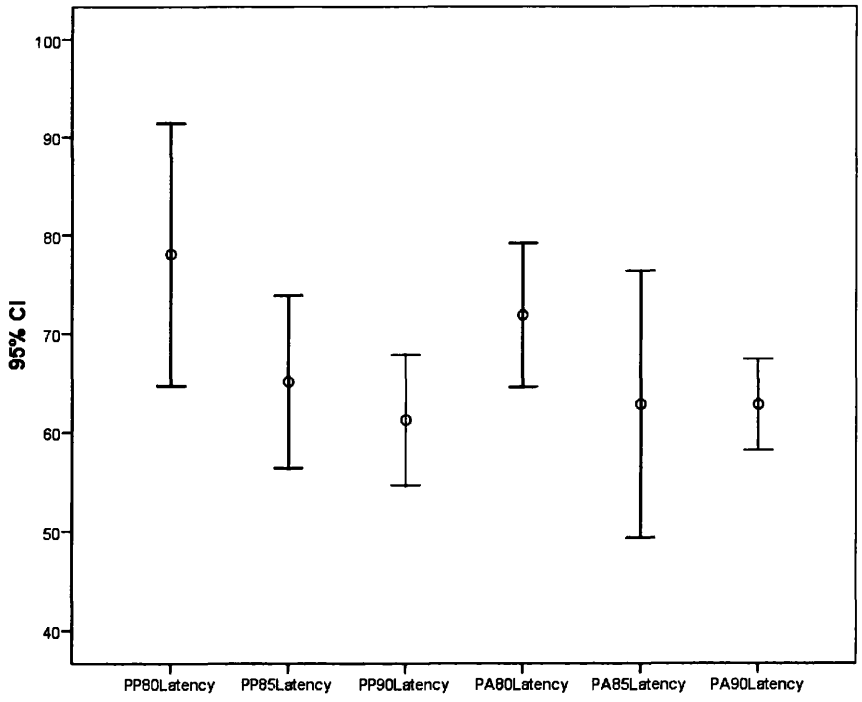


Figure 5.5 PPER response onset to peak latency (indexes response duration)

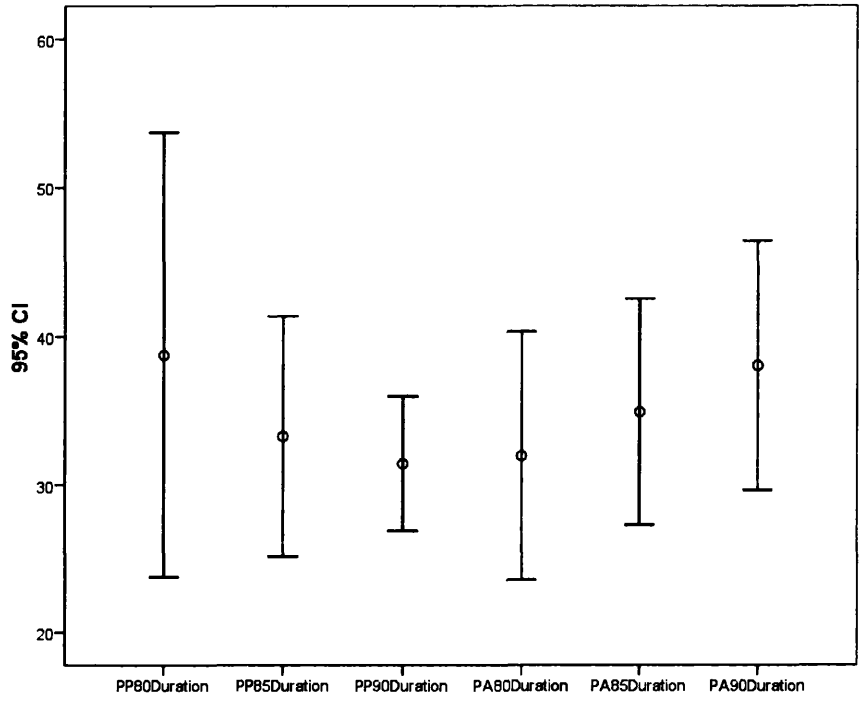


Figure 5.6 Prepulse inhibition probabilities at different prepulse intensities

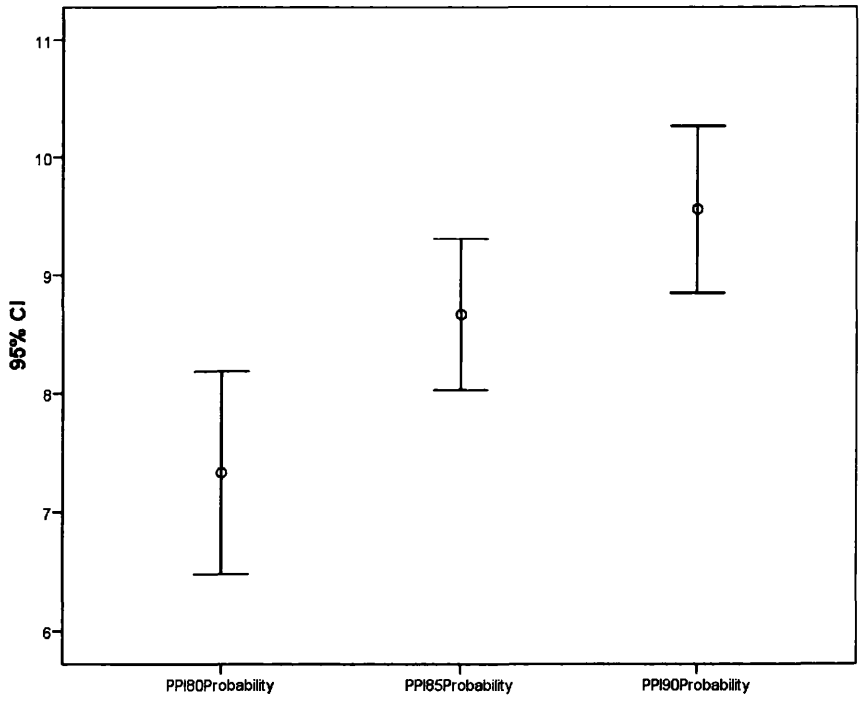


Figure 5.7 Prepulse inhibition percentage values at different prepulse intensities

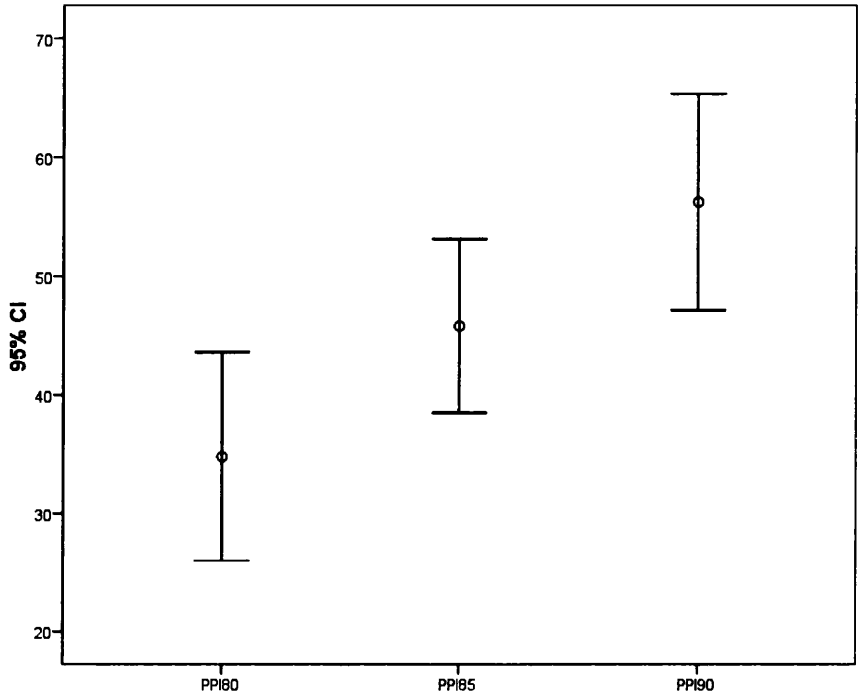


Figure 5.8 PPF probabilities at different prepulse intensities

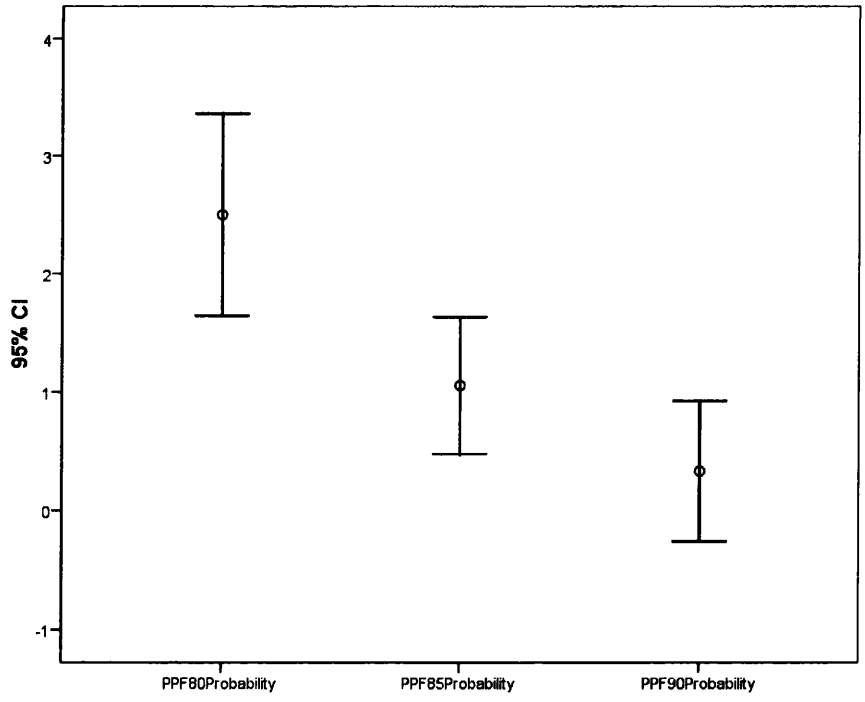
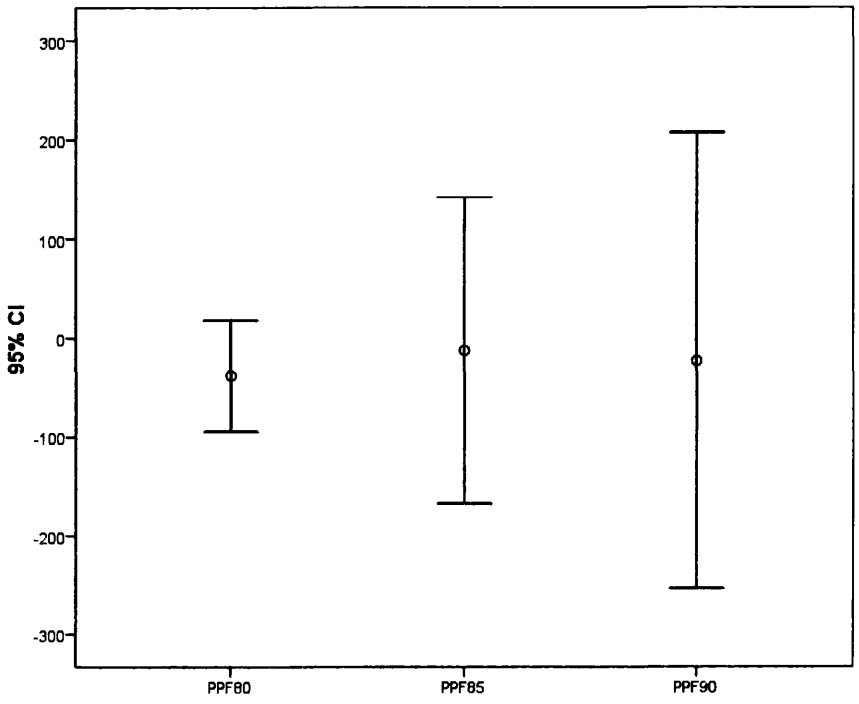


Figure 5.9 PPF percentage values at different prepulse intensities



Part 2: Tables

Study 1

Table 5.1 Motor response probabilities in all trial types [SP = startle probe alone; PP = prepulse and pulse trial; PA = prepulse alone trial; PPx, x = lead stimulus dB] in prepulse temporal space (presented stimuli: none in SP trials, prepulses in PP and PA trials, none in B trials)

Trial Type	Sample Proportion		Prepulse-elicited response probability	
	n	(n/18)*100 = %	*M (SD)*	*(M* /10)*100 = %
SP	3	17	.22 (.55)	2
PP80	11	61	1.40 (1.65)	14
PP85	12	67	2.66 (2.66)	26
PP90	15	83	3.94 (3.20)	39
PA80	15	83	1.67 (1.61)	17
PA85	12	67	2.67 (2.70)	27
PA90	17	94	4.22 (3.06)	42
B	7	39	.40 (.50)	4

Table 5.2 Motor response probabilities in all trial types in the startle probe temporal space (presented stimuli: pulse in SP trials, pulse in PP, none in PA trials, none in B trials)

Trial Type	Sample Proportion		Pulse-elicited response probability	
	n	(n/18)*100 = %	*M (SD)*	*(M* /10)*100 = %
SP	18	100	9.11 (.96)	91
PP80	18	100	8.94 (1.06)	89
PP85	18	100	8.72 (1.36)	87
PP90	18	100	8.61 (1.14)	86
PA80	5	28	.44 (.86)	4
PA85	3	17	.17 (.38)	2
PA90	3	17	.17 (.38)	2
B	5	28	.28 (.46)	3

Table 5.3 Paired samples t-test results comparing prepulse-elicited response probabilities and spontaneous EMG activation probability in the absence of experimental stimuli in prepulse temporal space (significant outcomes marked as **bold**) [SP=startle probe alone trial, PP= prepulse and pulse trial and PA= prepulse alone trial, B= no stimulus 'blind' trial; PPx, x = prepulse intensity]

Trial Type	*M*	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
PP80-SP	1.17	1.85	.44	.24	2.10	2.67	17	**.02**
PP85 - SP	2.33	2.81	.66	.94	3.73	3.53	17	**< .01**
PP90 - SP	3.72	3.39	.80	2.03	5.41	4.65	17	**< .01**
PA80 - SP	1.44	1.72	.41	.59	2.30	3.56	17	**< .01**
PA85 - SP	2.44	2.81	.66	1.04	3.84	3.69	17	**< .01**
PA90 - SP	4.00	3.22	.76	2.40	5.60	5.27	17	**< .01**
PP80 - B	1.00	1.64	.39	.12	1.82	2.58	17	**< .01**
PP85 - B	2.17	2.6	.62	.85	3.48	3.48	17	**< .01**
PP90 - B	3.55	3.18	.75	1.97	5.14	4.74	17	**< .01**
PA80 - B	1.28	1.53	.36	.52	2.04	3.55	17	**< .01**
PA85 - B	2.28	2.78	.66	.89	3.66	3.47	17	**< .01**
PA90 - B	3.83	3.07	.72	2.30	5.36	5.29	17	**< .01**

Table 5.4 The associations between PPER probabilities in all trial types

PPER		PP80 Probability	PP85 Probability	PP90 Probability	PA80 Probability	PA85 Probability	PA90 Probability
PP80Probability	*r*	1.00	**.76****	**.74****	**.76****	**.72****	**.49***
	p		**< .01**	**< .01**	**< .01**	**< .01**	**.04**
	N	18	18	18	18	18	18
PP85Probability	*r*	**.76****	1.0	**.86****	**.83****	**.87****	**.81****
	p	**< .01**		**< .01**	**< .01**	**< .01**	**< .01**
	N	18	18	18	18	18	18
PP90Probability	*r*	**.74****	**.86****	1.00	**.68****	**.75****	**.84****
	p	**< .01**	**< .01**		**.01**	**< .01**	**< .01**
	N	18	18	18	18	18	18
PA80Probability	*r*	**.76****	**.83****	**.68***	1.00	**.62****	**.67****
	p	**< .01**	**< .01**	**.01**		**.01**	**. < .01**
	N	18	18	18	18	18	18
PA85Probability	*r*	**.72****	**.87****	**.75****	**.62****	1.00	**.64****
	p	**< .01**	**< .01**	**< .01**	**.01**		**< .01**
	N	18	18	18	18	18	18
PA90Probability	*r*	**.49***	**.81****	**.85****	**.67****	**.64****	1.00
	p	**.04**	**< .01**	**< .01**	**< .01**	**< .01**	
	N	18	18	18	18	18	18

Table 5.5 The associations between PPER amplitudes in all trial types

PPER		PP80 Amplitude	PP85 Amplitude	PP90 Amplitude	PA80 Amplitude	PA85 Amplitude	PA90 Amplitude
PP80Amplitude	*r*	1.00	.01	.05	.58	.29	.37
	p		.99	.89	.06	.41	.26
	N	11	10	11	11	10	11
PP85Amplitude	*r*	.01	1.00	.33	.28	.07	**.60***
	p	.99		.30	.37	.84	**.04**
	N	10	12.00	12	12	11	12
PP90Amplitude	*r*	.05	.33	1.00	.22	**.78****	**.73****
	p	.89	.30		.46	**< .01**	**< .01**
	N	11	12	15.00	14	11	14
PA80Amplitude	*r*	.58	.28	.22	1.00	.06	.35
	p	.06	.37	.46		.85	.19
	N	11	12	14	15.00	11	15
PA85Amplitude	*r*	.29	.07	**.78****	.06	1.00	.26
	p	.41	.84	**< .01**	.85		.42
	N	10	11	11	11	12.00	12
PA90Amplitude	*r*	.37	**.60***	.73**	.35	.26	1.00
	p	.26	**.04**	**< .01**	.20	.42	
	N	11	12	14	15	12	17

Table 5.6 The associations between PPER onsets

PPER		PP80Onset	PP85Onset	PP90Onset	PA80Onset	PA85Onset	PA90Onset
PP80Onset	*r*	1.00	.63	.40	.49	.06	.43
	p		.05	.22	.12	.87	.19
	N	11	10	11	11	10	11
PP85Onset	*r*	.63	1.00	**.67***	**.60***	.56	.47
	p	.05		**.02**	**.04**	.07	.12
	N	10	12	12	12	11	12
PP90Onset	*r*	.40	**.67***	1.00	.51	.52	**.58***
	p	.22	**.02**		.06	.10	**.03**
	N	11	12	15	14	11	14
PA80Onset	*r*	.49	**.60***	.51	1.00	.21	.11
	p	.12	**.04**	.06		.54	.68
	N	11	12	14	15	11	15
PA85Onset	*r*	.06	.56	.52	.21	1.00	**.70***
	p	.87	.07	.10	.54		**.01**
	N	10	11	11	11	12	12
PA90Onset	*r*	.43	.47	**.58***	.11	**.70***	1.00
	p	.19	.12	**.03**	.68	**.01**	
	N	11	12	14	15	12	17

Table 5.7 The associations between PPER peak latencies in all trial types

PPER		PP80 Latency	PP85 Latency	PP90 Latency	PA80 Latency	PA85 Latency	PA90 Latency
PP80Latency	*r*	1.00	.63	.44	.52	.07	**.67***
	p		.05	.17	.10	.84	**.02**
	N	11	10	11	11	10	11
PP85Latency	*r*	.63	1.00	**.91****	.03	.59	**.78****
	p	.05		**< .01**	.92	.05	**< .01**
	N	10	12	12	12	11	12
PP90Latency	*r*	.44	**.91****	1.00	.25	**.68***	**.68****
	p	.17	**< .01**		.39	**.02**	**.01**
	N	11	12	15	14	11	14
PA80Latency	*r*	.52	.03	.25	1.00	.11	-.02
	p	.10	.92	.39		.75	.94
	N	11	12	14	15	11	15
PA85Latency	*r*	.07	.59	**.68***	.11	1.00	.55
	p	.84	.05	**.02**	.75		.06
	N	10	11	11	11	12	12
PA90Latency	*r*	**.67***	**.78****	**.68****	-.02	.55	1.000
	p	**.02**	**< .01**	**.01**	.94	.06	
	N	11	12	14	15	12	17

Table 5.8 The associations between PPER onset to peak latency values (index of response duration) in all trial types

PPER		PP80 Duration	PP85 Duration	PP90 Duration	PA80 Duration	PA85 Duration	PA90 Duration
PP80Duration	*r*	1.00	.60	.09	.03	-.16	.01
	p		.06	.79	.92	.67	.99
	N	11	10	11	11	10	11
PP85Duration	*r*	.60	1.00	.33	-.06	-.28	.10
	p	.06		.30	.86	.41	.75
	N	10	12	12	12	11	12
PP90Duration	*r*	.09	.33	1.00	.22	-.19	.42
	p	.79	.30		.46	.58	.13
	N	11	12	15	14	11	14
PA80Duration	*r*	.03	-.06	.22	1.00	-.29	.15
	p	.92	.86	.46		.39	.60
	N	11	12	14	15	11	15
PA85Duration	*r*	-.16	-.28	-.19	-.29	1.00	.08
	p	.67	.41	.58	.39		.81
	N	10	11	11	11	12	12
PA90Duration	*r*	.01	.10	.42	.15	.08	1.00
	p	.99	.75	.13	.60	.81	
	N	11	12	14	15	12	17

Table 5.9 Personality factors and PPER probabilities correlations

Personality Factors		PP80 Probability	PP85 Probability	PP90 Probability	PA80 Probability	PA85 Probability	PA90 Probability
State Anxiety	*r*	-.14	.03	.10	-.02	.02	-.04
	p	.57	.90	.70	.94	.94	.88
	N	18	18	18	18	18	18
Trait Anxiety	*r*	-.17	-.18	.01	-.12	-.05	-.11
	p	.49	.48	.95	.63	.84	.66
	N	18	18	18	18	18	18
Positive Affect	*r*	.13	.28	.30	-.01	.29	.45
	p	.60	.26	.22	.99	.25	.06
	N	18	18	18	18	18	18
Negative Affect	*r*	.06	.19	.08	.18	.16	.05
	p	.82	.45	.75	.48	.53	.83
	N	18	18	18	18	18	18
BAS Drive	*r*	.21	.11	.10	.16	-.07	.09
	p	.42	.69	.70	.53	.79	.73
	N	17	17	17	17	17	17
BAS Fun	*r*	.07	.28	.21	.23	.119	.45
	p	.79	.27	.42	.38	.649	.07
	N	17	17	17	17	17	17
BAS Reward	*r*	.23	.08	.09	.18	.00	.10
	p	.36	.75	.71	.46	1.00	.69
	N	18	18	18	18	18	18
BIS	*r*	.22	.23	.27	.16	.11	.19
	p	.39	.36	.27	.53	.66	.45
	N	18	18	18	18	18	18
Unusual Experiences	*r*	**.58***	**.53***	.36	**.57***	**.57***	.33
	p	**.01**	**.02**	.15	**.01**	**.01**	.18

	N	18	18	18	18	18	18
Cognitive Disorganisation	*r*	-.03	-.07	.04	.06	.03	-.02
	p	.91	.78	.88	.82	.90	.95
	N	18	18	18	18	18	18
Introvertive Anhedonia	*r*	-.03	.05	-.02	.13	.16	.07
	p	.89	.83	.93	.61	.52	.79
	N	18	18	18	18	18	18
Impulsive Non-conformity	*r*	.25	.16	.13	.46	.05	.25
	p	.31	.52	.59	.05	.83	.31
	N	18	18	18	18	18	18
Novelty Seeking	*r*	.39	.22	.20	.34	.12	.17
	p	.11	.39	.43	.17	.65	.49
	N	18	18	18	18	18	18
Harm Avoidance	*r*	.12	.19	.26	.23	.18	.23
	p	.64	.46	.29	.36	.46	.35
	N	18	18	18	18	18	18
Reward Dependence	*r*	.11	.11	.12	.18	-.10	.15
	p	.66	.65	.64	.47	.70	.55
	N	18	18	18	18	18	18
Persistence	*r*	.39	**.59***	.46	.42	**.49***	**.57***
	p	.11	**.01**	.06	.08	**.04**	**.01**
	N	18	18	18	18	18	18
Self-Directedness	*r*	-.07	-.05	-.04	-.14	-.12	-.07
	p	.79	.84	.87	.58	.66	.79
	N	17	17	17	17	17	17
Cooperativeness	*r*	.11	.20	.25	.01	.08	.11

	p	.67	.41	.32	.96	.76	.66
	N	18	18	18	18	18	18
Self-Transcendence	*r*	-.18	-.20	-.28	-.26	-.20	-.25
	p	.49	.41	.26	.30	.43	.31
	N	18	18	18	18	18	18
FSS Total Score	*r*	.34	.31	.29	.26	.24	.04
	p	.16	.22	.25	.31	.33	.86
	N	18	18	18	18	18	18
Fear of Animals	*r*	.30	.03	.17	-.02	.21	-.19
	p	.23	.91	.49	.94	.41	.45
	N	18	18	18	18	18	18
Interpersonal Fear	*r*	.24	.39	.38	.23	.38	.20
	p	.34	.11	.12	.35	.12	.44
	N	18	18	18	18	18	18
Fear of Tissue Damage	*r*	.29	.19	.15	.17	.08	-.10
	p	.24	.45	.56	.50	.74	.70
	N	18	18	18	18	18	18
Fear of Noises	*r*	.34	.40	.27	**.53***	.25	.36
	p	.17	.10	.27	**.02**	.31	.14
	N	18	18	18	18	18	18
Classic Phobias	*r*	.39	.23	.37	.23	.19	.17
	p	.11	.35	.13	.35	.44	.51
	N	18	18	18	18	18	18

Table 5.10 Personality factors and PPER amplitudes correlations

Personality Factors		PP80 Amplitude	PP85 Amplitude	PP90 Amplitude	PA80 Amplitude	PA85 Amplitude	PA90 Amplitude
State Anxiety	*r*	.21	.20	-.36	.28	-.51	.26
	p	.53	.52	.18	.31	.09	.32
	N	11	12	15	15	12	17
Trait Anxiety	*r*	-.07	-.28	-.49	-.49	-.53	-.16
	p	.83	.38	.06	.07	.08	.55
	N	11	12	15	15	12	17
Positive Affect	*r*	.05	.48	.31	.47	.26	.30
	p	.89	.12	.26	.08	.40	.24
	N	11	12	15	15	12	17
Negative Affect	*r*	.25	.25	-.12	.43	-.06	.32
	p	.45	.43	.67	.11	.86	.21
	N	11	12	15	15	12	17
BAS Drive	*r*	.03	.05	.52	.508	.28	-.03
	p	.94	.88	.05	.063	.38	.90
	N	10	12	14	14	12	16
BAS Fun	*r*	.12	.55	**.55***	.48	**.66***	.15
	p	.73	.06	**.04**	.08	**.02**	.58
	N	10	12	14	14	12	16
BAS Reward	*r*	-.19	-.02	.16	.03	.13	.05
	p	.58	.95	.57	.92	.70	.85
	N	11	12	15	15	12	17
BIS	*r*	.13	-.15	-.304	-.29	-.25	.37
	p	.71	.64	.27	.30	.44	.14
	N	11	12	15	15	12	17
Unusual Experiences	*r*	.15	.34	**.61***	-.04	.40	**.67****
	p	.66	.28	**.01**	.89	.20	**< .01**
	N	11	12	15	15	12	17
Cognitive Disorganisation	*r*	.17	-.12	-.35	**-.53***	-.31	.05
	p	.62	.71	.194	**.04**	.33	.86

	N	11	12	15	15	12	17
Introvertive Anhedonia	*r*	.30	-.11	.18	-.15	.07	.06
	p	.36	.73	.51	.59	.84	.80
	N	11	12	15	15	12	17
Impulsive Non-conformity	*r*	.48	.02	.22	.09	.55	.05
	p	.13	.95	.42	.75	.06	.84
	N	11	12	15	15	12	17
Novelty Seeking	*r*	.05	.24	.08	.27	.31	.24
	p	.88	.45	.77	.32	.32	.36
	N	11	12	15	15	12	17
Harm Avoidance	*r*	.28	.03	-.30	-.37	-.27	.23
	p	.41	.92	.28	.17	.40	.38
	N	11	12	15	15	12	17
Reward Dependence	*r*	.35	-.49	-.15	.14	.32	.07
	p	.29	.10	.60	.62	.32	.78
	N	11	12	15	15	12	17
Persistence	*r*	-.14	.20	**.76****	.31	.41	.45
	p	.68	.54	***<.01***	.26	.18	.07
	N	11	12	15	15	12	17
Self-Directedness	*r*	-.46	-.12	.01	.11	-.07	-.14
	p	.15	.72	.10	.70	.83	.61
	N	11	12	15	15	12	16
Cooperativeness	*r*	-.06	-.01	-.03	.28	-.08	.23
	p	.86	.98	.90	.31	.81	.38
	N	11	12	15	15	12	17
Self-Transcendence	*r*	.31	.13	.14	**.61***	-.41	-.20
	p	.35	.69	.63	**.01**	.19	.45
	N	11	12	15	15	12	17
FSS Total Score	*r*	.27	-.48	-.11	-.02	-.07	.30

	p	.41	.11	.68	.941	.98	.23
	N	11	12	15	15	12	17
Fear of Animals	*r*	-.38	**-.63***	-.32	-.49	-.32	-.19
	p	.24	**.03**	.24	.06	.30	.45
	N	11	12	15	15	12	17
Interpersonal Fear	*r*	.20	-.30	-.01	.05	-.03	.34
	p	.54	.34	.98	.84	.92	.18
	N	11	12	15	15	12	17
Fear of Tissue Damage	*r*	.35	-.46	-.18	-.02	-.01	.29
	p	.28	.12	.51	.92	.99	.25
	N	11	12	15	15	12	17
Fear of Noises	*r*	.58	-.13	.13	.01	.46	.37
	p	.05	.68	.62	.97	.13	.13
	N	11	12	15	15	12	17
Classic Phobias	*r*	.18	-.40	-.15	.08	.01	.12
	p	.59	.18	.57	.75	.97	.62
	N	11	12	15	15	12	17

Table 5.11 Personality factors and PPER onsets

Personality Factors		PP80 Onset	PP85 Onset	PP90 Onset	PA80 Onset	PA85 Onset	PA90 Onset
State Anxiety	*r*	.02	-.02	.14	.04	.19	.22
	p	.93	.93	.61	.87	.54	.38
	N	11	12	15	15	12	17
Trait Anxiety	*r*	-.16	-.41	-.33	-.04	-.01	-.10
	p	.62	.17	.22	.87	.99	.69
	N	11	12	15	15	12	17
Positive Affect	*r*	.35	.48	.47	.10	.36	**.54***
	p	.29	.11	.07	.71	.24	**.02**
	N	11	12	15	15	12	17
Negative Affect	*r*	-.04	.11	.04	-.12	-.01	.10
	p	.89	.72	.87	.65	.98	.69
	N	11	12	15	15	12	17
BAS Drive	*r*	-.05	.11	.33	.26	-.32	.01
	p	.87	.72	.23	.36	.29	.98
	N	10	12	14	14	12	16
BAS Fun	*r*	.17	.53	.26	.11	.28	.38
	p	.63	.07	.36	.70	.37	.14
	N	10	12	14	14	12	16
BAS Reward	*r*	**-.68***	-.30	-.22	.09	-.24	-.28
	p	**.02**	.33	.43	.72	.45	.27
	N	11	12	15	15	12	17
BIS	*r*	-.46	-.41	-.22	.02	.18	.06
	p	.14	.17	.41	.93	.56	.80
	N	11	12	15	15	12	17
Unusual Experiences	*r*	.16	-.09	.19	.29	-.04	-.08
	p	.62	.78	.48	.28	.87	.74

	N	11	12	15	15	12	17
Cognitive Disorganisation	*r*	.02	-.34	-.46	.06	-.02	-.20
	p	.94	.27	.08	.82	.93	.42
	N	11	12	15	15	12	17
Introvertive Anhedonia	*r*	.25	-.51	-.17	-.31	-.32	.10
	p	.45	.09	.53	.25	.30	.68
	N	11	12	15	15	12	17
Impulsive Non-conformity	*r*	.15	-.04	-.08	-.23	-.27	-.12
	p	.64	.89	.75	.39	.38	.64
	N	11	12	15	15	12	17
Novelty Seeking	*r*	-.10	.48	.11	.07	-.04	-.14
	p	.75	.11	.69	.78	.89	.57
	N	11	12	15	15	12	17
Harm Avoidance	*r*	.14	-.28	-.32	-.09	.21	.09
	p	.66	.37	.23	.74	.49	.71
	N	11	12	15	15	12	17
Reward Dependence	*r*	-.37	-.25	-.27	-.22	-.18	.08
	p	.25	.42	.32	.42	.55	.73
	N	11	12	15	15	12	17
Persistence	*r*	-.11	-.07	.28	.01	.23	**.57***
	p	.72	.82	.30	.98	.46	**.02**
	N	11	12	15	15	12	17
Self-Directedness	*r*	-.29	.23	.10	-.04	.31	.16
	p	.37	.47	.71	.88	.31	.53
	N	11	12	15	15	12	16
Cooperativeness	*r*	.01	.40	.34	.18	.48	.31
	p	.97	.19	.20	.50	.110	.21
	N	11	12	15	15	12	17
Self-Transcendence	*r*	.19	-.02	.08	.44	-.31	-.10
	p	.57	.93	.77	.10	.31	.69

	N	11	12	15	15	12	17
FSS Total Score	*r*	-.01	-.21	.18	.01	-.07	.05
	p	.96	.50	.51	.94	.82	.83
	N	11	12	15	15	12	17
Fear of Animals	*r*	-.15	-.13	.01	.10	-.18	-.25
	p	.64	.68	.94	.71	.56	.32
	N	11	12	15	15	12	17
Interpersonal Fear	*r*	.14	-.15	.27	.01	.14	.31
	p	.66	.62	.32	.96	.65	.22
	N	11	12	15	15	12	17
Fear of Tissue Damage	*r*	-.08	-.22	.14	.08	-.13	-.10
	p	.80	.49	.61	.76	.68	.68
	N	11	12	15	15	12	17
Fear of Noises	*r*	.18	-.11	-.10	-.30	-.01	.13
	p	.58	.71	.70	.26	.98	.59
	N	11	12	15	15	12	17
Classic Phobias	*r*	.11	.14	.24	-.06	-.14	.18
	p	.73	.64	.38	.81	.65	.48
	N	11	12	15	15	12	17

Table 5.12 Personality factors and PPER peak latencies

Personality Factors		PP80 Peak Latency	PP85 Peak Latency	PP90 Peak Latency	PA80 Peak Latency	PA85 Peak Latency	PA90 Peak Latency
State Anxiety	*r*	.34	.36	-.06	-.25	.15	.43
	p	.29	.24	.82	.36	.63	.08
	N	11	12	15	15	12	17
Trait Anxiety	*r*	.12	.18	-.04	-.15	-.19	.30
	p	.71	.57	.86	.58	.54	.23
	N	11	12	15	15	12	17
Positive Affect	*r*	.09	.36	.41	-.44	.48	.31
	P	.78	.24	.12	.09	.11	.21
	N	11	12	15	15	12	17
Negative Affect	*r*	-.10	.01	-.23	-.40	.09	.17
	p	.76	.95	.40	.13	.77	.50
	N	11	12	15	15	12	17
BAS Drive	*r*	.04	-.18	-.15	-.17	-.20	-.29
	p	.89	.57	.59	.55	.52	.27
	N	10	12	14	14	12	16
BAS Fun	*r*	.44	.39	.35	-.17	.13	.01
	p	.19	.20	.21	.55	.68	.95
	N	10	12	14	14	12	16
BAS Reward	*r*	-.36	-.26	-.27	**-.54***	-.30	-.34
	p	.27	.40	.31	**.04**	.34	.17
	N	11	12	15	15	12	17
BIS	*r*	.14	-.07	-.31	-.21	.12	.25
	p	.67	.82	.25	.44	.69	.32
	N	11	12	15	15	12	17
Unusual Experiences	*r*	-.37	-.48	-.17	-.25	-.16	-.26
	p	.25	.10	.52	.36	.60	.30

	N	11	12	15	15	12	17
Cognitive Disorganisation	*r*	.12	-.08	-.17	-.04	-.36	.18
	p	.72	.79	.54	.88	.24	.47
	N	11	12	15	15	12	17
Introvertive Anhedonia	*r*	.02	-.39	-.39	-.20	-.41	-.04
	p	.94	.20	.14	.47	.18	.86
	N	11	12	15	15	12	17
Impulsive Non-conformity	*r*	.15	-.04	-.16	-.19	-.51	-.22
	p	.64	.87	.56	.49	.09	.39
	N	11	12	15	15	12	17
Novelty Seeking	*r*	-.14	.05	-.01	-.12	.03	-.06
	p	.67	.85	.98	.64	.92	.81
	N	11	12	15	15	12	17
Harm Avoidance	*r*	.47	.01	-.11	.27	-.16	.10
	p	.13	.96	.69	.32	.62	.68
	N	11	12	15	15	12	17
Reward Dependence	*r*	-.15	-.23	-.42	-.18	-.17	-.02
	p	.64	.45	.11	.50	.58	.93
	N	11	12	15	15	12	17
Persistence	*r*	-.37	-.20	-.01	**-.60***	.39	.03
	p	.25	.51	.98	**.02**	.20	.88
	N	11	12	15	15	12	17
Self-Directedness	*r*	-.34	.06	.19	.20	.57	-.02
	p	.30	.84	.48	.46	.05	.93
	N	11	12	15	15	12	16
Cooperativeness	*r*	-.02	.28	.42	.29	**.66***	.27
	p	.93	.37	.11	.28	**.02**	.29
	N	11	12	15	15	12	17

Self-Transcendence	*r*	-.03	-.18	.03	-.18	-.11	-.07
	p	.92	.57	.89	.50	.73	.78
	N	11	12	15	15	12	17
FSS Total Score	*r*	-.03	-.01	-.02	.09	.06	.31
	p	.92	.98	.93	.73	.83	.21
	N	11	12	15	15	12	17
Fear of Animals	*r*	-.29	.14	.21	.21	.08	.24
	p	.37	.66	.44	.45	.78	.33
	N	11	12	15	15	12	17
Interpersonal Fear	*r*	.06	.21	.24	.08	.23	.41
	p	.85	.50	.37	.77	.46	.10
	N	11	12	15	15	12	17
Fear of Tissue Damage	*r*	.03	-.11	-.12	.20	-.02	.21
	p	.92	.72	.64	.46	.93	.40
	N	11	12	15	15	12	17
Fear of Noises	*r*	.07	-.14	-.18	-.01	-.13	.08
	p	.83	.65	.52	.94	.68	.76
	N	11	12	15	15	12	17
Classic Phobias	*r*	.03	.22	.04	-.13	.08	**.48***
	p	.92	.49	.87	.63	.78	**.05**
	N	11	12	15	15	12	17

Table 5.13 Personality factors and PPER onset to peak latencies (index of response duration)

Personality Factors		PP80 Duration	PP85 Duration	PP90 Duration	PA80 Duration	PA85 Duration	PA90 Duration
State Anxiety	*r*	.28	.39	-.21	-.26	-.02	.06
	P	.39	.20	.44	.33	.94	.79
	N	11	12	15	15	12	17
Trait Anxiety	*r*	.31	**.71****	.26	-.11	-.35	.35
	p	.35	**.01**	.33	.69	.25	.15
	N	11	12	15	15	12	17
Positive Affect	*r*	-.32	-.25	.03	-.49	.31	-.40
	p	.33	.43	.90	.06	.31	.11
	N	11	12	15	15	12	17
Negative Affect	*r*	-.04	-.17	-.31	-.27	.17	.01
	p	.90	.69	.25	.31	.58	.96
	N	11	12	15	15	12	17
BAS Drive	*r*	.10	-.32	-.47	-.33	.13	-.23
	p	.77	.30	.08	.24	.68	.39
	N	10	12	14	14	12	16
BAS Fun	*r*	.21	-.28	.15	-.22	-.19	-.43
	p	.54	.37	.60	.43	.54	.09
	N	10	12	14	14	12	16
BAS Reward	*r*	.46	.11	-.11	**-.58***	-.18	.06
	p	.14	.712	.68	**.02**	.57	.79
	N	11	12	15	15	12	17
BIS	*r*	**.68***	.45	-.15	-.21	-.05	.11
	p	**.02**	.13	.58	.43	.87	.65
	N	11	12	15	15	12	17
Unusual Experiences	*r*	-.53	-.37	-.39	-.46	-.23	-.10
	p	.08	.23	.14	.08	.47	.69

	N	11	12	15	15	12	17
Cognitive Disorganisation	*r*	.08	.34	.24	-.08	**-.63***	.39
	p	.80	.26	.38	.75	**.03**	.12
	N	11	12	15	15	12	17
Introvertive Anhedonia	*r*	-.27	.25	-.30	.05	-.26	-.16
	p	.41	.42	.27	.83	.41	.54
	N	11	12	15	15	12	17
Impulsive Non-conformity	*r*	-.04	.01	-.10	.01	-.50	-.02
	p	.90	.98	.71	.97	.09	.92
	N	11	12	15	15	12	17
Novelty Seeking	*r*	-.01	-.55	-.11	-.18	.12	.12
	p	.98	.06	.68	.52	.70	.62
	N	11	12	15	15	12	17
Harm Avoidance	*r*	.26	.37	.18	.32	**-.62***	-.03
	p	.42	.23	.51	.23	**.03**	.90
	N	11	12	15	15	12	17
Reward Dependence	*r*	.30	.08	-.22	.01	-.03	-.12
	p	.37	.78	.41	.99	.91	.63
	N	11	12	15	15	12	17
Persistence	*r*	-.20	-.11	-.27	**-.56***	.36	**-.64****
	p	.54	.72	.31	**.03**	.24	**.01**
	N	11	12	15	15	12	17
Self-Directedness	*r*	.03	-.22	.13	.22	.56	-.21
	p	.92	.47	.64	.42	.05	.42
	N	11	12	15	15	12	16
Cooperativeness	*r*	-.03	-.23	.16	.13	.47	-.16
	p	.91	.46	.56	.64	.12	.51
	N	11	12	15	15	12	17
Self-Transcendence	*r*	-.25	-.14	-.03	**-.52***	.28	.06
	p	.45	.64	.90	**.05**	.37	.80

	N	11	12	15	15	12	17
FSS Total Score	*r*	-.01	.26	-.20	.07	.23	.17
	p	.97	.41	.47	.79	.46	.49
	N	11	12	15	15	12	17
Fear of Animals	*r*	-.09	.30	.23	.11	.44	**.49***
	p	.78	.33	.41	.68	.15	**.05**
	N	11	12	15	15	12	17
Interpersonal Fear	*r*	-.11	.41	.02	.06	.21	-.04
	p	.73	.18	.93	.81	.51	.83
	N	11	12	15	15	12	17
Fear of Tissue Damage	*r*	.12	.16	-.28	.12	.15	.29
	p	.70	.60	.30	.66	.64	.25
	N	11	12	15	15	12	17
Fear of Noises	*r*	-.14	.01	-.10	.22	-.23	-.10
	p	.66	.98	.70	.42	.47	.69
	N	11	12	15	15	12	17
Classic Phobias	*r*	-.10	.03	-.18	-.07	.38	.15
	p	.75	.92	.52	.79	.22	.55
	N	11	12	15	15	12	17

Table 5.14 Correlations between PPI and PPF probabilities

Startle Response Modification		PPI80 Probability	PPI85 Probability	PPI90 Probability	PPF80 Probability	PPF85 Probability	PPF90 Probability
PPI80Probability	*r*	1.00	.16	**.47***	**-.97****	-.16	**-.52***
	p		.52	**.05**	**< .01**	.53	**.03**
	N	18	18	18	18	18	18
PPI85Probability	*r*	.16	1.00	.24	-.19	**-.93****	-.15
	p	.52		.35	.46	**< .01**	.54
	N	18	18	18	18	18	18
PPI90Probability	*r*	**.47***	.24	1.00	**-.50***	-.34	**-.98****
	p	**.05**	.35		**.03**	.17	**< .01**
	N	18	18	18	18	18	18
PPF80Probability	*r*	**-.97****	-.19	**-.50***	1.00	.19	**.55***
	p	**< .01**	.46	**.03**		.45	**.02**
	N	18	18	18	18	18	18
PPF85Probability	*r*	-.16	**-.93****	-.34	.19	1.00	.24
	p	.53	**< .01**	.17	.45		.33
	N	18	18	18	18	18	18
PPF90Probability	*r*	**-.52***	-.15	**-.98****	**.55***	.24	1.00
	p	**.03**	.54	**< .01**	**.02**	.33	
	N	18	18	18	18	18	18

Table 5. 15 Correlations between prepulse inhibition (PPI) and prepulse facilitation (PPF) percentage change at three prepulse intensities

Startle Response Modification		PPI80	PPI85	PPI90	PPF80	PPF85	PPF90
PPI80	*r*	1.00	**.81****	**.73****	**-.60***	**.69***	1.00**
	p		**< .01**	**< .01**	**.01**	**.03**	.
	N	18	18	18	17	10	2
PPI85	*r*	**.81****	1.00	**.91****	**-.49***	.29	1.00**
	p	**< .01**		**< .01**	**.04**	.41	.
	N	18	18	18	17	10	2
PPI90	*r*	**.73****	**.91****	1.00	-.46	.41	1.00**
	p	**< .01**	**< .01**		.06	.24	.
	N	18	18	18	17	10	2
PPF80	*r*	**-.60***	**-.49***	-.46	1.00	-.032	-1.00**
	p	**.01**	**.04**	.06		.93	.
	N	17	17	17	17	10	2
PPF85	*r*	**.69***	.29	.41	-.03	1.00	1.00**
	p	**.03**	.41	.24	.93		.
	N	10	10	10	10	10	2
PPF90	*r*	1.00**	1.00**	1.00**	-1.00**	1.00**	1.00
	p	.	.	.	.	.	
	N	2	2	2	2	2	2

Table 5.16 Correlations between PPI probabilities and PPI and PPF percentage changes

Startle Response Modification		PPI80	PPI85	PPI90	PPF80	PPF85	PPF90
PPI80Probability	*r*	**.48***	**.60****	.43	-.01	.54	1.00**
	p	**.04**	**< .01**	.08	.99	.11	.
	N	18	18	18	17	10	2
PPI85Probability	*r*	**.67****	**.74****	**.71****	-.38	.12	.[a]
	p	**< .01**	**< .01**	**< .01**	.14	.74	.
	N	18	18	18	17	10	2
PPI90Probability	*r*	.20	**.55***	**.58***	.09	.08	1.00**
	p	.43	**.02**	**.01**	.74	.81	.
	N	18	18	18	17	10	2

Table 517 Correlations between PPF probabilities and PPI and PPF percentage changes

Startle Response Modification		PPI80	PPI85	PPI90	PPF80	PPF85	PPF90
PPF80Probability	*r*	**-.48***	-.62**	-.44	-.03	-.44	-1.00**
	p	**.05**	**. < .01**	.06	.90	.21	.
	N	18	18	18	17	10	2
PPF85Probability	*r*	**-.57***	**-.73****	**-.66****	.31	-.04	.[a]
	p	**.01**	**< .01**	**< .01**	.23	.91	.
	N	18	18	18	17	10	2
PPF90Probability	*r*	-.17	**-.49***	-.51*	-.14	-.09	-1.00**
	p	.49	**.04**	.03	.59	.81	.
	N	18	18	18	17	10	2

Table 5.18 PPER probabilities and SRM probabilities and percentage changes

		PP80 Probability	PP85 Probability	PP90 Probability	PA80 Probability	PA85 Probability	PA90 Probability
PPI80Probability	*r*	.26	.14	.39	.21	.16	.15
	p	.29	.59	.11	.40	.51	.54
	N	18	18	18	18	18	18
PPI85Probability	*r*	-.16	-.25	-.28	-.34	-.07	-.43
	p	.53	.31	.26	.16	.80	.07
	N	18	18	18	18	18	18
PPI90Probability	*r*	.25	.30	.36	.26	.22	.13
	p	.31	.22	.15	.29	.38	.60
	N	18	18	18	18	18	18
PPF80Probability	*r*	-.30	-.17	-.41	-.23	-.18	-.18
	p	.23	.51	.09	.35	.48	.48
	N	18	18	18	18	18	18
PPF85Probability	*r*	-.10	.05	.08	.17	-.07	.29
	p	.68	.85	.75	.51	.79	.24
	N	18	18	18	18	18	18
PPF90Probability	*r*	-.25	-.28	-.35	-.28	-.20	-.13
	p	.32	.25	.15	.27	.42	.59
	N	18	18	18	18	18	18
PPI80	*r*	-.22	-.36	-.19	-.37	-.26	-.46
	p	.37	.14	.45	.13	.29	.06
	N	18	18	18	18	18	18
PPI85	*r*	.10	.07	.17	.02	.15	-.11
	p	.70	.78	.50	.94	.55	.67
	N	18	18	18	18	18	18
PPI90	*r*	-.02	-.01	.08	.00	.09	-.12
	p	.95	.99	.76	1.00	.72	.65

	N	18	18	18	18	18	18
PPF80	*r*	-.13	.10	.06	.09	-.13	.40
	p	.62	.70	.82	.74	.63	.11
	N	17	17	17	17	17	17
PPF85	*r*	-.24	-.51	-.22	-.21	-.54	-.31
	p	.51	.13	.53	.56	.10	.38
	N	10	10	10	10	10	10
PPF90	*r*	.[a]	.[a]	1.00**	1.00**	-1.00**	-1.00**
	p	.	.	.	.	.	.
	N	2	2	2	2	2	2

Table 5.19 PPER amplitudes and SRM probabilities and percentage changes

		PP80 Amplitude	PP85 Amplitude	PP90 Amplitude	PA80 Amplitude	PA85 Amplitude	PA90 Amplitude
PPI80Probability	*r*	.32	-.26	-.04	.18	-.23	.07
	p	.33	.41	.90	.51	.47	.80
	N	11	12	15	15	12	17
PPI85Probability	*r*	-.56	-.25	-.22	-.06	-.23	-.44
	p	.07	.44	.43	.82	.47	.08
	N	11	12	15	15	12	17
PPI90Probability	*r*	**-.66***	.20	.22	-.01	-.48	.30
	p	**.03**	.53	.42	.98	.12	.24
	N	11	12	15	15	12	17
PPF80Probability	*r*	-.36	.25	.03	-.20	.21	-.08
	p	.27	.43	.90	.47	.51	.74
	N	11	12	15	15	12	17
PPF85Probability	*r*	**.66***	.18	.02	.06	.18	.23
	p	**.03**	.57	.93	.82	.56	.38
	N	11	12	15	15	12	17
PPF90Probability	*r*	.[a]	.[a]	-.21	-.11	.50	-.29
	p	.00	.00	.45	.69	.10	.25
	N	11	12	15	15	12	17
PPI80	*r*	-.15	-.30	-.40	.38	-.40	-.45
	p	.65	.33	.14	.16	.20	.07
	N	11	12	15	15	12	17
PPI85	*r*	-.29	-.15	-.09	.28	-.33	-.10
	p	.39	.63	.76	.31	.30	.71
	N	11	12	15	15	12	17
PPI90	*r*	-.47	-.14	-.11	.12	-.32	-.16
	p	.14	.66	.70	.68	.31	.54
	N	11	12	15	15	12	17

PPF80	*r*	.43	.45	.19	.19	.19	.27
	p	.22	.16	.51	.51	.57	.31
	N	10	11	14	14	11	16
PPF85	*r*	-.03	-.08	-.24	-.63	-.04	-.45
	p	.95	.87	.53	.09	.92	.23
	N	6	7	9	8	8	9
PPF90	*r*	.[a]	.[a]	.[a]	.[a]	.[a]	1.00**
	p	.	.	.	.	.	.
	N	0	0	1	1	1	2

Table 5.20 PPER onsets and SRM probabilities and percentage changes

		PP80	PP85	PP90	PA80	PA85	PA90
PPI80Probability	*r*	.55	.33	.19	.26	-.01	.22
	p	.07	.29	.50	.34	.98	.39
	N	11	12	15	15	12	17
PPI85Probability	*r*	.01	.37	.18	.03	-.02	-.09
	p	.99	.23	.51	.89	.94	.74
	N	11	12	15	15	12	17
PPI90Probability	*r*	-.19	.45	.44	.35	.41	.30
	p	.56	.14	.09	.18	.17	.22
	N	11	12	15	15	12	17
PPF80Probability	*r*	**-.61***	-.39	-.23	-.23	-.02	-.24
	p	**.05**	.21	.40	.39	.94	.34
	N	11	12	15	15	12	17
PPF85Probability	*r*	.09	-.41	-.32	-.18	-.08	.03
	p	.79	.17	.24	.50	.80	.89
	N	11	12	15	15	12	17
PPF90Probability	*r*	.[a]	.[a]	-.12	-.37	-.32	-.27
	p	.00	.00	.64	.16	.29	.28
	N	11	12	15	15	12	17
PPI80	*r*	.13	.36	.30	.07	-.17	-.03
	p	.68	.24	.27	.78	.59	.88
	N	11	12	15	15	12	17
PPI85	*r*	.19	.48	.40	.13	.16	.18
	p	.55	.11	.13	.62	.61	.47
	N	11	12	15	15	12	17
PPI90	*r*	-.03	.28	.30	-.22	.23	.23
	p	.92	.36	.26	.41	.45	.36
	N	11	12	15	15	12	17

PPF80	*r*	.39	.32	-.11	.08	.29	.48
	p	.26	.32	.70	.77	.37	.06
	N	10	11	14	14	11	16
PPF85	*r*	.27	.14	.11	-.20	.03	-.01
	p	.60	.75	.76	.62	.93	.98
	N	6	7	9	8	8	9
PPF90	*r*	.[a]	.[a]	.[a]	.[a]	.[a]	1.000**
	p	.	.	.	.	.	.
	N	0	0	1	1	1	2

Table 5.21 PPER latencies and SRM probabilities and percentage changes

		PP80	PP85	PP90	PA80	PA85	PA90
PPI80Probability	*r*	.10	.19	.25	.09	.06	**.56***
	p	.76	.68	.36	.74	.84	**.02**
	N	11	12	15	15	12	17
PPI85Probability	*r*	-.37	.18	.23	.24	.40	.16
	p	.25	.55	.41	.38	.19	.52
	N	11	12	15	15	12	17
PPI90Probability	*r*	-.35	.30	.36	-.17	**.58***	**.52***
	p	.29	.33	.18	.53	**.05**	**.03**
	N	11	12	15	15	12	17
PPF80Probability	*r*	-.18	-.20	-.32	-.15	-.10	**-.61****
	p	.58	.51	.24	.59	.74	**.01**
	N	11	12	15	15	12	17
PPF85Probability	*r*	.48	-.09	-.23	-.18	-.56	-.16
	p	.13	.75	.41	.51	.05	.47
	N	11	12	15	15	12	17
PPF90Probability	*r*	.[a]	.[a]	-.06	.35	-.45	**-.52***
	p	.00	.00	.82	.20	.13	**.03**
	N	11	12	15	15	12	**17**
PPI80	*r*	-.05	.32	.25	.24	.16	.24
	p	.87	.30	.36	.37	.61	.35
	N	11	12	15	15	12	17
PPI85	*r*	-.22	.33	.44	.21	.50	.40
	p	.51	.28	.09	.43	.09	.10
	N	11	12	15	15	12	17
PPI90	*r*	-.38	.32	.29	.01	.56	.28
	p	.24	.30	.29	.98	.05	.27
	N	11	12	15	15	12	17

PPF80	*r*	.56	-.06	-.15	-.06	.05	.31
	p	.08	.85	.59	.82	.87	.23
	N	10	11	14	14	11	16
PPF85	*r*	-.03	-.02	-.07	.09	-.08	-.10
	p	.94	.96	.84	.82	.83	.79
	N	6	7	9	8	8	9
PPF90	*r*	.[a]	.[a]	.[a]	.[a]	.[a]	1.00**
	p	.	.	.	.	.	.
	N	0	0	1	1	1	2

Table 5.22 PPER onset to peak latency values (index of response duration) and SRM probabilities and percentage changes

		PP80	PP85	PP90	PA80	PA85	PA90
PPI80Probability	*r*	-.55	-.28	.11	-.12	.12	.16
	p	.07	.36	.68	.66	.69	.52
	N	11	12	15	15	12	17
PPI85Probability	*r*	-.35	-.28	.09	.19	**.77****	.23
	p	.29	.36	.74	.48	**p < .01**	.37
	N	11	12	15	15	12	17
PPI90Probability	*r*	-.09	-.26	-.01	-.44	.41	.02
	p	.78	.40	.98	.09	.17	.91
	N	11	12	15	15	12	17
PPF80Probability	*r*	.54	.28	-.15	.04	-.15	-.17
	p	.08	.36	.58	.87	.62	.49
	N	11	12	15	15	12	17
PPF85Probability	*r*	.34	.43	.03	-.02	**-.90****	-.18
	p	.30	.15	.89	.92	**p < .01**	.48
	N	11	12	15	15	12	17
PPF90Probability	*r*	.[a]	.[a]	.04	**.62***	-.32	-.07
	p	.00	.00	.86	**.01**	.30	.79
	N	11	12	15	15	12	17
PPI80	*r*	-.21	-.14	.01	.17	.55	.22
	p	.53	.66	.97	.54	.06	.37
	N	11	12	15	15	12	17
PPI85	*r*	-.43	-.28	.13	.09	**.67***	.09
	p	.17	.37	.64	.73	**.02**	.72
	N	11	12	15	15	12	17
PPI90	*r*	-.31	-.04	.04	.18	**.66***	-.06
	p	.34	.88	.86	.51	**.02**	.81

	N	11	12	15	15	12	17
PPF80	*r*	.09	-.44	-.07	-.12	-.39	-.29
	p	.78	.17	.79	.65	.22	.27
	N	10	11	14	14	11	16
PPF85	*r*	-.30	-.24	-.18	.23	-.33	-.08
	p	.55	.59	.63	.58	.42	.83
	N	6	7	9	8	8	9
PPF90	*r*	.[a]	.[a]	.[a]	.[a]	.[a]	-1.00**
	p	.	.	.	.	.	.
	N	0	0	1	1	1	2

Table 5.23 Personality factors and startle response modification probabilities

Personality Factors		PPI80 Probability	PPI85 Probability	PPI90 Probability	PPF80 Probability	PPF85 Probability	PPF90 Probability
State Anxiety	*r*	.32	-.01	.36	-.29	.12	-.40
	p	.19	.94	.13	.22	.63	.10
	N	18	18	18	18	18	18
Trait Anxiety	*r*	.32	-.11	.22	-.27	.25	-.29
	p	.19	.66	.36	.26	.30	.23
	N	18	18	18	18	18	18
Positive Affect	*r*	-.10	.06	-.09	.09	-.16	.15
	p	.68	.79	.70	.72	.51	.53
	N	18	18	18	18	18	18
Negative Affect	*r*	.17	.14	.28	-.14	-.10	-.29
	p	.49	.56	.25	.58	.68	.23
	N	18	18	18	18	18	18
BAS Drive	*r*	-.03	-.06	-.29	.04	-.10	.25
	p	.88	.98	.25	.85	.68	.32
	N	17	17	17	17	17	17
BAS Fun	*r*	-.23	-.04	-.35	.14	-.01	.36
	p	.37	.86	.16	.58	.95	.14
	N	17	17	17	17	17	17
BAS Reward	*r*	-.24	-.30	-.32	.33	.17	.30
	p	.33	.22	.18	.17	.48	.22
	N	18	18	18	18	18	18
BIS	*r*	.06	-.41	.27	.09	.35	-.30
	p	.98	.08	.26	.97	.15	.22
	N	18	18	18	18	18	18
Unusual Experiences	*r*	.03	-.36	-.09	.02	.18	.08
	p	.90	.13	.70	.93	.46	.75
	N	18	18	18	18	18	18

Cognitive Disorganisation	*r*	.31	-.30	.13	-.27	.39	-.21
	p	.19	.21	.59	.27	.10	.38
	N	18	18	18	18	18	18
Introvertive Anhedonia	*r*	.16	-.13	.11	-.16	.26	-.19
	p	.56	.59	.65	.50	.28	.43
	N	18	18	18	18	18	18
Impulsive Non-conformity	*r*	.11	-.19	-.27	-.15	.24	.19
	p	.66	.43	.27	.52	.32	.43
	N	18	18	18	18	18	18
Novelty Seeking	*r*	.03	-.01	-.17	-.08	-.12	.18
	p	.88	.95	.50	.72	.61	.46
	N	18	18	18	18	18	18
Harm Avoidance	*r*	.21	-.32	-.02	-.23	.32	.04
	p	.39	.19	.92	.35	.18	.98
	N	18	18	18	18	18	18
Reward Dependence	*r*	.10	-.28	-.05	-.01	.27	.04
	p	.68	.25	.82	.96	.26	.87
	N	18	18	18	18	18	18
Persistence	*r*	-.11	.00	.12	.14	-.17	-.08
	p	.65	1.00	.63	.57	.49	.74
	N	18	18	18	18	18	18
Self-Directedness	*r*	-.12	.25	.09	.13	-.36	.01
	p	.64	.32	.70	.59	.15	.95
	N	17	17	17	17	17	17
Cooperativeness	*r*	.11	.06	.28	-.11	-.18	-.17
	p	.64	.79	.25	.63	.46	.48

	N	18	18	18	18	18	18
Self-Transcendence	*r*	-.17	.24	.08	.20	-.22	-.07
	p	.47	.33	.73	.40	.37	.75
	N	18	18	18	18	18	18
FSS Total Score	*r*	.32	.04	.38	-.38	-.03	-.39
	p	.18	.89	.11	.18	.88	.10
	N	18	18	18	18	18	18
Fear of Animals	*r*	.34	.39	.25	-.37	-.36	-.23
	p	.16	.10	.31	.13	.14	.34
	N	18	18	18	18	18	18
Interpersonal Fear	*r*	.30	.05	.38	-.36	-.01	-.37
	p	.21	.82	.11	.14	.99	.12
	N	18	18	18	18	18	18
Fear of Tissue Damage	*r*	.22	-.03	.32	-.28	.01	-.33
	p	.36	.89	.19	.25	.95	.16
	N	18	18	18	18	18	18
Fear of Noises	*r*	.19	-.23	.17	-.24	.25	-.20
	p	.44	.35	.48	.39	.30	.41
	N	18	18	18	18	18	18
Classic Phobias	*r*	**.54***	.19	.30	**-.61****	-.19	-.32
	p	**.02**	.42	.22	**.01**	.43	.19
	N	18	18	18	18	18	18

Table 5.24 Personality factors and startle response modification percentage change

Personality Factors		PPI80	PPI85	PPI90	PPF80	PPF85	PPF90
State Anxiety	*r*	.24	.22	.26	-.05	-.09	1.00**
	p	.32	.37	.29	.84	.98	.
	N	18	18	18	17	10	2
Trait Anxiety	*r*	-.01	-.03	.04	-.05	.17	1.00**
	p	.96	.89	.86	.82	.62	.
	N	18	18	18	17	10	2
Positive Affect	*r*	.05	.075	.045	.13	-.23	-1.00**
	p	.98	.76	.85	.58	.51	.
	N	18	18	18	17	10	2
Negative Affect	*r*	.20	.29	.25	-.06	-.43	.a
	p	.40	.23	.30	.81	.20	.
	N	18	18	18	17	10	2
BAS Drive	*r*	.16	-.01	-.16	.07	-.06	-1.00**
	p	.54	.95	.52	.78	.86	.
	N	17	17	17	16	10	2
BAS Fun	*r*	-.13	-.07	-.18	.26	-.59	-1.00**
	p	.60	.76	.46	.32	.06	.
	N	17	17	17	16	10	2
BAS Reward	*r*	-.24	-.32	-.40	-.06	-.28	-1.00**
	p	.32	.18	.09	.81	.41	.
	N	18	18	18	17	10	2
BIS	*r*	-.42	-.33	-.29	.21	-.23	1.00**
	p	.07	.17	.24	.41	.52	.
	N	18	18	18	17	10	2
Unusual Experiences	*r*	-.45	-.26	-.36	.10	-.05	-1.00**
	p	.05	.28	.13	.69	.87	.
	N	18	18	18	17	10	2

Cognitive Disorganisation	*r*	-.29	-.23	-.23	.18	.16	.a
	p	.23	.34	.35	.47	.65	.
	N	18	18	18	17	10	2
Introvertive Anhedonia	*r*	-.16	-.16	.02	.21	.31	1.00**
	p	.51	.50	.93	.41	.38	.
	N	18	18	18	17	10	2
Impulsive Non-conformity	*r*	-.17	-.14	-.27	.12	-.26	-1.00**
	p	.47	.58	.27	.64	.46	.
	N	18	18	18	17	10	2
Novelty Seeking	*r*	.03	.10	-.06	.01	-.44	-1.00**
	p	.90	.66	.79	.96	.19	.
	N	18	18	18	17	10	2
Harm Avoidance	*r*	-.09	-.05	.02	.04	.35	1.00**
	p	.69	.84	.93	.85	.31	.
	N	18	18	18	17	10	2
Reward Dependence	*r*	-.08	-.15	-.17	.07	.04	1.00**
	p	.73	.53	.48	.98	.91	.
	N	18	18	18	17	10	2
Persistence	*r*	-.17	.04	.09	.11	-.22	-1.00**
	p	.49	.87	.69	.65	.53	.
	N	18	18	18	17	10	2
Self-Directedness	*r*	.28	.31	.38	-.22	.21	1.00**
	p	.27	.22	.12	.39	.54	.
	N	17	17	17	16	10	2
Cooperativeness	*r*	.25	.35	.29	-.22	.07	1.00**
	p	.31	.13	.23	.37	.83	.
	N	18	18	18	17	10	2
Self-Transcendence	*r*	.06	.01	-.06	-.01	-.35	-1.00**
	p	.78	.96	.81	.96	.31	.
	N	18	18	18	17	10	2

FSS Total Score	*r*	.09	.21	.14	-.18	-.20	1.00**
	p	.70	.39	.58	.47	.56	.
	N	18	18	18	17	10	2
Fear of Animals	*r*	.24	.36	.31	**-.50***	.04	1.00**
	p	.33	.13	.20	**.04**	.89	.
	N	18	18	18	17	10	2
Interpersonal Fear	*r*	.11	.28	.27	-.25	-.21	1.00**
	p	.65	.25	.27	.32	.54	.
	N	18	18	18	17	10	2
Fear of Tissue Damage	*r*	.04	.07	-.02	-.10	-.19	1.00**
	p	.85	.76	.92	.67	.59	.
	N	18	18	18	17	10	2
Fear of Noises	*r*	-.31	-.08	-.13	.18	-.25	1.00**
	p	.19	.74	.59	.48	.47	.
	N	18	18	18	17	10	2
Classic Phobias	*r*	.29	.37	.29	-.07	-.05	.a
	p	.23	.12	.23	.77	.87	.
	N	18	18	18	17	10	2

Table 5.25 Correlations between PPER probabilities and age

PPER		age
PP80Probability	*r*	-.37
	p	.12
	N	18
PP85Probability	*r*	-.38
	p	.11
	N	18
PP90Probability	*r*	-.44
	p	.06
	N	18
PA80Probability	*r*	-.19
	p	.44
	N	18
PA85Probability	*r*	-.43
	p	.07
	N	18
PA90Probability	*r*	-.37
	p	.12
	N	18

Table 5.26 Correlations between PPER amplitudes and age

PPER		age
PP80Amplitude	*r*	-.43
	p	.17
	N	11
PP85Amplitude	*r*	-.24
	p	.44
	N	12
PP90Amplitude	*r*	-.50
	p	.05
	N	15
PA80Amplitude	*r*	.07
	p	.79
	N	15
PA85Amplitude	*r*	-.21
	p	.49
	N	12
PA90Amplitude	*r*	-.37
	p	.14
	N	17

Table 5.27 Correlations between PPER onsets and age

PPER		age
PP80Onset	*r*	**-.76****
	p	**.01**
	N	11
PP85Onset	*r*	-.19
	p	.54
	N	12
PP90Onset	*r*	-.32
	p	.23
	N	15
PA80Onset	*r*	-.44
	p	.09
	N	15
PA85Onset	*r*	.41
	p	.18
	N	12
PA90Onset	*r*	-.06
	p	.81
	N	17

Table 5.28 Correlations between PPER peak latency values and age

		age
PP80Latency	*r*	-.20
	p	.54
	N	11
PP85Latency	*r*	.14
	p	.65
	N	12
PP90Latency	*r*	-.27
	p	.31
	N	15
PA80Latency	*r*	.17
	p	.54
	N	15
PA85Latency	*r*	.38
	p	.22
	N	12
PA90Latency	*r*	-.15
	p	.56
	N	17

Table 5.29 Correlations between PPER onset to peak latency values and age

		age
PP80Duration	*r*	**.70**
	p	**.02**
	N	11
PP85Duration	*r*	.39
	p	.21
	N	12
PP90Duration	*r*	-.01
	p	.95
	N	15
PA80Duration	*r*	.50
	p	.05
	N	15
PA85Duration	*r*	.06
	p	.84
	N	12
PA90Duration	*r*	-.04
	p	.86
	N	17

Table 5.30 Correlations between startle response modification probabilities and age

Startle Response Modification		age
PPI80Probability	*r*	-.11
	p	.64
	N	18
PPI85Probability	*r*	.30
	p	.21
	N	18
PPI90Probability	*r*	.15
	p	.53
	N	18
PPF80Probability	*r*	.01
	p	.94
	N	18
PPF85Probability	*r*	-.23
	p	.34
	N	18
PPF90Probability	*r*	-.13
	p	.60
	N	18

Table 5.31 Correlations between startle response modification percentage changes and age

Startle Response Modification		age
PPI80	*r*	.39
	p	.10
	N	18
PPI85	*r*	.28
	p	.24
	N	18
PPI90	*r*	.45
	p	.05
	N	18
PPF80	*r*	-.07
	p	.78
	N	17
PPF85	*r*	.34
	p	.32
	N	10
PPF90	*r*	1.00**
	p	.
	N	2

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Table 5.32 Comparison of the startle responses in the unpredictable (SP) and predictable (PSP) conditions

	M	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
SP - PSP	1.97	72.02	18.01	-36.41	40.34	.11	15	.91

Table 5.33 Startle response percentage change in the predictable condition and PPER probabilities correlations

		Startle Response Change
PP80Probability	*r*	.48
	p	.05
	N	16
PP85Probability	*r*	.33
	p	.20
	N	16
PP90Probability	*r*	.40
	p	.12
	N	16
PA80Probability	*r*	**.52***
	p	**.04**
	N	16
PA85Probability	*r*	**.60***
	p	**.01**
	N	16
PA90Probability	*r*	**.63****
	p	**.01**
	N	16

Table 5.34 Startle response percentage change in the predictable condition and PPER amplitudes correlations

		Startle Response Change
PP80Amplitude	*r*	.56
	p	.14
	N	8
PP85 Amplitude	*r*	**.73***
	p	**.01**
	N	11
PP90 Amplitude	*r*	.44
	p	.17
	N	11
PA80 Amplitude	*r*	.50
	p	.13
	N	10
PA85 Amplitude	*r*	-.14
	p	.70
	N	9
PA90 Amplitude	*r*	.47
	p	.13
	N	11

Table 5.35 Startle response percentage change in the predictable condition and PPER onsets correlations

		Startle Response Change
PP80Onset	*r*	.34
	p	.40
	N	8
PP85Onset	*r*	**-.67***
	p	**.02**
	N	11
PP90 Onset	*r*	-.53
	p	.09
	N	11
PA80 Onset	*r*	-.46
	p	.17
	N	10
PA85 Onset	*r*	-.52
	p	.14
	N	9
PA90 Onset	*r*	-.59
	p	.05
	N	11

Table 5.36 Startle response percentage change in the predictable condition and PPER peak latency values correlations

		Startle Response Change
PP80Latency	*r*	.64
	p	.08
	N	8
PP85Latency	*r*	-.35
	p	.28
	N	11
PP90Latency	*r*	.05
	p	.87
	N	11
PA80Latency	*r*	-.31
	p	.37
	N	10
PA85Latency	*r*	-.03
	p	.92
	N	9
PA90Latency	*r*	.09
	p	.78
	N	11

Table 5.37 Startle response percentage change in the predictable condition and PPER onset to peak latency values correlations

		Startle Response Change
PP80Duration	*r*	.69
	p	.05
	N	8
PP85Duration	*r*	.43
	p	.18
	N	11
PP90Duration	*r*	.56
	p	.06
	N	11
PA80Duration	*r*	.16
	p	.65
	N	10
PA85Duration	*r*	.46
	p	.20
	N	9
PA90Duration	*r*	**.62***
	p	**.04**
	N	11

Table 5.38 Startle response modification probability and startle response change in the predictable condition

		Startle Response Change
PPI80Probability	*r*	-.484
	p	.057
	N	16
PPI85Probability	*r*	-.446
	p	.084
	N	16
PPI90Probability	*r*	**-.695****
	p	**.003**
	N	16
PPF80Probability	*r*	.422
	p	.104
	N	16
PPF85Probability	*r*	.353
	p	.180
	N	16
PPF90Probability	*r*	**.507***
	p	**.045**
	N	16

Table 5.39 Startle response modification percentage change and startle response change in the predictable condition

		Startle Response Change
PPI80	*r*	-.38
	p	.14
	N	16
PPI85	*r*	-.44
	p	.08
	N	16
PPI90	*r*	**-.51***
	p	**.04**
	N	16
PPF80	*r*	-.42
	p	.21
	N	10
PPF85	*r*	.29
	p	.41
	N	10
PPF90	*r*	-.05
	p	.90
	N	7

Table 5.40 Personality factors and startle response change in the condition of predictability

		Startle Response Change
State Anxiety	*r*	-.11
	p	.66
	N	16
Trait Anxiety	*r*	**.57***
	p	**.02**
	N	16
Positive Affect	*r*	.06
	p	.82
	N	16
Negative Affect	*r*	.29
	p	.26
	N	16
BAS Drive	*r*	.25
	p	.34
	N	16
BAS Fun	*r*	-.30
	p	.24
	N	16
BAS Reward	*r*	-.11
	p	.67
	N	16
BIS	*r*	-.14
	p	.58
	N	16
Unusual Experiences	*r*	.12
	p	.65
	N	16

Cognitive Disorganisation	*r*	.30
	p	.25
	N	16
Introvertive Anhedonia	*r*	.47
	p	.06
	N	16
Impulsive Non-conformity	*r*	-.06
	p	.80
	N	16
Novelty Seeking	*r*	-.24
	p	.38
	N	15
Harm Avoidance	*r*	.26
	p	.33
	N	15
Reward Dependence	*r*	-.41
	p	.12
	N	15
Persistence	*r*	.17
	p	.54
	N	15
Self-Directedness	*r*	-.25
	p	.36
	N	15
Cooperativeness	*r*	-.39
	p	.14
	N	15
Self-Transcendence	*r*	.23
	p	.40
	N	15

FSS Total Score	*r*	-.19
	p	.49
	N	15
Fear of Animals	*r*	**-.65****
	p	**.01**
	N	15
Interpersonal Fear	*r*	.02
	p	.92
	N	15
Fear of Tissue Damage	*r*	-.22
	p	.42
	N	15
Fear of Noises	*r*	-.48
	p	.06
	N	15
Classic Phobias	*r*	-.01
	p	.95
	N	15

Table 5.41 Startle response change in the predictable condition correlations with age and sex

		Startle Response Change
sex	*r*	.05
	p	.84
	N	16
age	*r*	.34
	p	.18
	N	16

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Table 5.42 Comparison of PPER characteristics at 80dB in the predictable and unpredictable conditions (predictable – unpredictable)

	Paired Differences							
	M	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
PP80Probability	.70	2.53	.56	-.48	1.88	1.23	19	.70
PP80Amplitude	21.89	52.06	17.35	-18.12	61.90	1.26	8	21.89
PP80Latency	-7.11	9.72	3.24	-14.58	.36	-2.19	8	-7.11
PP80Onset	-4.82	20.85	6.95	-20.85	11.21	-.69	8	-4.82
PP80Duration	-2.28	19.53	6.51	-17.30	12.73	-.35	8	-2.28

Table 5.43 Comparison of PPER characteristics at 85dB in the predictable and unpredictable conditions

	Paired Differences							
	M	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
PP85Probability	.00	1.74	.39	-.81	.81	.00	19	1.00
PP85Amplitude	2.13	45.42	13.11	-26.72	30.99	.163	11	.87
PP85Latency	-9.14	29.34	8.47	-27.79	9.50	-1.08	11	.30
PP85Onset	-7.09	15.61	4.50	-17.01	2.82	-1.57	11	.14
PP85Duration	-2.05	22.07	6.37	-16.07	11.96	-.32	11	.75

Table 5.44 Comparison of PPER characteristics at 90dB in the predictable and unpredictable conditions

	Paired Differences							
	M	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
PP90Probability	-.60	2.47	.55	-1.76	.56	-1.08	19	.29
PP90Amplitude	-19.82	38.10	10.18	-41.82	2.17	-1.94	13	.07
PP90Latency	3.03	26.71	7.14	-12.38	18.46	.42	13	.67
PP90Onset	-2.90	12.27	3.27	-9.98	4.18	-.88	13	.39
PP90Duration	5.94	24.71	6.60	-8.32	20.21	.90	13	.38

Table 5.45 Prepulse inhibition probabilities in the predictable and unpredictable conditions

	Paired Differences							
	M	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
PPI80	1.35	2.58	.57	.14	2.55	**2.33**	19	**.03**
PPI85	-1.65	2.05	.46	-2.61	-.68	**-3.58**	19	**< .01**
PPI90	-.10	1.55	.34	-.82	.62	-.28	19	.77

Table 5.46 Prepulse inhibition percentage changes in the predictable and unpredictable conditions

	Paired Differences							
	M	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
PPI80	2.85	15.64	3.58	-4.68	10.39	.79	18	.43
PPI85	2.45	10.28	2.29	-2.35	7.26	1.06	19	.29
PPI90	3.37	12.36	2.76	-2.41	9.15	1.22	19	.23

Table 5.47 PPF probabilities in the predictable and unpredictable conditions

	Paired Differences							
	M	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
PPF80	.350	2.05	.46	-.61	1.31	.76	19	.45
PPF85	-.250	1.74	.38	-1.06	.56	-.64	19	.52
PPF90	.150	1.63	.36	-.61	.91	.41	19	.68

Table 5.48 PPF percentage changes in the predictable and unpredictable conditions

	Paired Differences							
	M	*SD*	*SEM*	95% Confidence Interval of the Difference		*t*	*df*	*p*
				Lower	Upper			
PPF80	-3.84	42.81	12.35	-31.05	23.35	-.31	11	.76
PPF85	-7.29	20.83	7.87	-26.56	11.97	-.92	6	.39
PPF90	-19.05	28.70	9.56	-41.12	3.01	-1.99	8	.08

Table 5.49 PPER in the unpredictable condition and startle response modification change differences between the unpredictable and predictable conditions

		PPI80 Preception	PPI85 Preception	PPI90 Preception	PPF80 Preception	PPF85 Preception	PPF90 Preception
PP80Probability	*r*	-.36	**-.54***	.13	.29	-.56	.21
	p	.12	**.01**	.58	.34	.18	.58
	N	19	20	20	12	7	9
PP85Probability	*r*	-.40	-.27	.01	.29	-.48	.26
	p	.08	.24	.96	.34	.26	.49
	N	19	20	20	12	7	9
PP90Probability	*r*	-.38	-.26	.06	.24	-.47	.29
	p	.10	.25	.77	.44	.28	.43
	N	19	20	20	12	7	9
PP80Amplitude	*r*	-.40	-.26	-.12	.22	-.26	-.08
	p	.15	.34	.64	.59	.67	.88
	N	14	15	15	8	5	6
PP85Amplitude	*r*	-.27	-.23	-.20	.13	.24	-.05
	p	.38	.43	.49	.77	.69	.93
	N	12	13	13	7	5	5
PP90Amplitude	*r*	-.39	-.45	.05	.29	-.77	-.50
	p	.11	.05	.82	.40	.07	.24
	N	17	18	18	10	6	7
PP80Latency	*r*	-.08	-.22	**.56***	-.10	-.27	-.42
	p	.78	.42	**.03**	.80	.65	.40
	N	14	15	15	8	5	6
PP85Latency	*r*	.25	.12	.10	-.52	.12	-.11
	p	.43	.68	.73	.22	.83	.85
	N	12	13	13	7	5	5
PP90Latency	*r*	-.13	.21	.05	-.21	.37	.08
	p	.61	.39	.82	.54	.46	.85

	N	17	18	18	10	6	7
PP80Onset	*r*	.13	.06	.15	-.33	.06	-.06
	p	.64	.82	.58	.42	.92	.90
	N	14	15	15	8	5	6
PP85Onset	*r*	.02	-.27	.17	-.35	-.09	-.24
	p	.93	.36	.57	.43	.88	.69
	N	12	13	13	7	5	5
PP90Onset	*r*	.03	-.28	.27	-.12	-.36	-.24
	p	.90	.25	.27	.73	.47	.59
	N	17	18	18	10	6	7
PP80Duration	*r*	-.18	-.25	.38	.28	-.35	-.34
	p	.52	.36	.15	.49	.56	.52
	N	14	15	15	8	5	6
PP85Duration	*r*	.34	.41	.01	-.54	.31	.08
	p	.27	.15	.98	.21	.60	.99
	N	12	13	13	7	5	5
PP90Duration	*r*	-.14	.46	-.21	-.05	**.86***	.30
	p	.57	.05	.39	.87	**.03**	.50
	N	17	18	18	10	6	7

Table 5.50 Personality factors and startle response modification percentage change differences between the unpredictable and predictable conditions

Personality Factors		PPI80 Preception	PPI85 Preception	PPI90 Preception	PPF80 Preception	PPF85 Preception	PPF90 Preception
State Anxiety	*r*	-.16	.08	.23	.09	-.18	.16
	p	.50	.72	.31	.77	.68	.67
	N	19	20	20	12	7	9
Trait Anxiety	*r*	.03	-.05	-.07	**.63***	.02	.51
	p	.89	.80	.77	**.04**	.97	.19
	N	18	19	19	11	6	8
Positive Affect	*r*	-.08	-.06	-.01	-.13	-.56	-.30
	p	.72	.78	.94	.65	.18	.34
	N	19	20	20	12	7	9
Negative Affect	*r*	-.23	-.07	.27	.04	-.49	.14
	p	.34	.75	.23	.88	.25	.70
	N	19	20	20	12	7	9
BAS Drive	*r*	-.05	.23	-.42	**-.69***	-.64	-.71
	p	.83	.35	.07	**.02**	.16	.05
	N	17	18	18	11	6	8
BAS Fun	*r*	.06	.19	.25	.11	-.29	-.13
	p	.80	.43	.30	.74	.57	.66
	N	17	18	18	11	6	8
BAS Reward	*r*	-.01	.29	.09	-.13	-.52	-.13
	p	.98	.23	.70	.63	.28	.79
	N	17	18	18	11	6	8
BIS	*r*	.24	.17	.13	.22	.02	.33
	p	.34	.49	.60	.50	.96	.41
	N	17	18	18	11	6	8

Unusual Experiences	*r*	.03	.21	.16	.19	-.38	-.14
	p	.88	.37	.50	.56	.45	.74
	N	18	19	19	11	6	8
Cognitive Disorganisation	*r*	-.03	.09	-.03	.17	.05	.29
	p	.90	.69	.89	.61	.91	.47
	N	18	19	19	11	6	8
Introvertive Anhedonia	*r*	-.12	.20	-.24	.41	-.05	.22
	p	.62	.40	.32	.20	.91	.58
	N	18	19	19	11	6	8
Impulsive Non-conformity	*r*	-.20	.21	.29	-.14	.69	-.13
	p	.40	.38	.21	.58	.12	.78
	N	18	19	19	11	6	8
Novelty Seeking	*r*	-.29	.05	.17	-.17	-.03	-.18
	p	.25	.98	.49	.73	.94	.65
	N	17	18	18	11	6	8
Harm Avoidance	*r*	.23	.09	-.26	.14	-.03	.34
	p	.37	.72	.29	.66	.94	.40
	N	17	18	18	11	6	8
Reward Dependence	*r*	-.13	-.08	-.05	-.29	-.08	-.33
	p	.61	.75	.83	.46	.86	.41
	N	17	18	18	11	6	8
Persistence	*r*	.22	.18	-.30	-.14	-.11	-.37
	p	.37	.46	.21	.69	.83	.36
	N	17	18	18	11	6	8
Self-Directedness	*r*	.06	-.14	-.03	-.53	-.12	-.43
	p	.80	.61	.89	.11	.81	.28

	N	14	15	15	9	6	8
Cooperativeness	*r*	.19	-.11	.17	-.33	-.19	-.20
	p	.45	.64	.50	.31	.71	.62
	N	17	18	18	11	6	8
Self-Transcendence	*r*	-.01	.31	.05	-.28	-.67	-.60
	p	.96	.20	.81	.39	.13	.11
	N	17	18	18	11	6	8
FSS Total Score	*r*	.32	.16	.33	.25	.23	.54
	p	.18	.51	.16	.44	.66	.16
	N	18	19	19	11	6	8
Fear of Animals	*r*	-.03	.23	.22	-.16	.13	.05
	p	.89	.33	.36	.62	.80	.89
	N	18	19	19	11	6	8
Interpersonal Fear	*r*	.26	.12	.26	.28	.29	.53
	p	.29	.61	.23	.40	.56	.17
	N	18	19	19	11	6	8
Fear of Tissue Damage	*r*	.28	.14	.26	.21	.28	.56
	p	.25	.55	.26	.53	.58	.14
	N	18	19	19	11	6	8
Fear of Noises	*r*	**.51***	.14	.43	.39	.17	.57
	p	**.03**	.55	.06	.22	.74	.13
	N	18	19	19	11	6	8
Classic Phobias	*r*	.31	.12	.27	.36	-.07	.50
	p	.20	.62	.26	.27	.89	.19
	N	18	19	19	11	6	8

Table 5.51 Startle response modification differences between the unpredictable and predictable conditions and age and sex

		sex	age
PPI80Preception	*r*	.11	.05
	p	.63	.81
	N	19	19
PPI85Preception	*r*	.09	-.21
	p	.69	.35
	N	20	20
PPI90Preception	*r*	-.32	-.03
	p	.16	.89
	N	20	20
PPF80Preception	*r*	.57	.28
	p	.05	.36
	N	12	12
PPF85Preception	*r*	.20	**-.80***
	p	.66	**.03**
	N	7	7
PPF90Preception	*r*	-.35	.20
	p	.35	.59
	N	9	9

Appendix 6

Table 6.1 Personality characteristics of all the samples

Personality Factors	*M*					
	3a Chapter 3	3b Chapter 3	4 Chapter 4	5a Chapter 5	5b Chapter 5	5c Chapter 5
State Anxiety	31.70	36.33	34.54	33.72	36.81	33.80
Trait Anxiety	36.54	44.55	44.40	40.11	45.31	44.47
Positive Affect	28.97	29.88	27.04	27.88	26.93	27.50
Negative Affect	12.22	12.44	12.32	12.88	13.31	11.80
BAS Drive	10.64	9.22	11.12	10.82	10.56	10.77
BAS Fun	11.53	11.55	11.36	12.29	13.00	13.38
BAS Reward	17.15	16.77	17.56	17.16	17.12	17.66
BIS	20.97	22.44	21.43	21.55	20.68	21.33
Unusual Experiences	4.41	9.55	11.00	7.27	9.68	9.63
Cognitive Disorganisation	8.48	13.22	14.04	11.22	15.25	11.94
Introvertive Anhedonia	5.23	7.33	7.72	6.27	5.50	6.26
Impulsive Nonconformity	7.30	8.77	8.80	8.33	10.25	8.31
Novelty Seeking	8.97	10.11	10.40	10.27	10.46	9.55
Harm Avoidance	9.02	9.11	10.20	3.83	10.53	9.94
Reward Dependence	9.87	9.00	9.40	10.77	10.46	9.88
Persistence	2.84	2.77	2.80	2.16	2.33	3.55
Self-Directedness	19.30	16.55	15.56	17.11	14.93	15.26
Cooperativeness	21.46	19.44	19.29	20.55	19.26	19.33
Self-Transcendence	3.97	4.88	5.80	8.83	4.00	4.55
FSS Total Score	106.02	127.75	123.91	100.83	100.66	109.63
Fear of Animals	8.10	8.12	12.28	7.83	9.80	8.10
Interpersonal Fear	38.76	49.75	49.56	38.33	38.33	43.89
Fear of Tissue Damage	30.51	37.00	36.70	34.61	29.06	34.89
Fear of Noises	3.00	4.62	4.60	2.61	3.06	3.42
Classical Phobias	11.07	14.02	14.08	12.05	14.06	13.31

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