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Improving the DSM-5 approach to cognitive impairment: developmental prosopagnosia reveals the need for tailored diagnoses

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Abstract

The Diagnostic Statistical Manual of Mental Disorders (DSM-5) recommends diagnosing neurocognitive disorders (i.e., cognitive impairment) when a patient scores beyond -1 SD below neurotypical norms on two tests. I review how this approach will fail due to cognitive tests' power limitations, validity issues, imperfect reliabilities, and biases, before summarising their resulting negative consequences. As a proof of concept, I use developmental prosopagnosia, a condition characterised by difficulties recognising faces, to show the DSM-5 only diagnoses 62-70% ($n1 = 61, n2 = 165$) versus 100% ($n1 = 61$) through symptoms alone. Pooling the DSM-5 missed cases confirmed the presence of group-level impairments on objective tests, which were further evidenced through meta-analyses, thus validating their highly atypical symptoms. These findings support a paradigm shift towards bespoke diagnostic approaches for distinct cognitive impairments, including a symptom-based method when validated effective. I reject dogmatic adherence to the DSM-5 approach to neurocognitive disorders, and underscore the importance of a data driven, transdiagnostic approach to understanding patients' subjective cognitive impairments. This will ultimately benefit patients, their families, clinicians, and scientific progress.

Keywords: diagnosis; neurocognitive disorders; prosopagnosia; single case analysis; mild cognitive impairment; major; subjective cognitive impairment, MCI; transdiagnostic.

1 **1. Introduction**

2 The Diagnostic and Statistical Manual for Mental Disorders fifth edition (DSM-5, APA,
3 2014) is considered the gold standard guidance for practitioners diagnosing mental disorders
4 in the United States. One section of the DSM-5 focuses on neurocognitive disorders which
5 can be graded as reflecting mild and major cognitive impairment due to a variety of causes,
6 including Alzheimer’s disease, Parkinson’s disease, HIV and traumatic brain injury (Sachdev
7 et al., 2014). Impairments are typically characterised by a reduction in cognitive or
8 behavioural functioning within, or across, six key domains, covering perceptual-motor
9 function, language, learning and memory, social cognition, complex attention, and executive
10 function (Sachdev et al., 2014). To diagnose impairment, the DSM-5 developers recommend
11 that a patient must score more poorly than one standard deviation below a neurotypical mean
12 on two cognitive or behavioural tasks (Sachdev et al., 2014).

13 It could be claimed that the DSM-5 has been helpful in providing formal, and
14 straightforward, standardised guidance to diagnose neurocognitive disorders. However, this
15 approach has been criticised partly because the liberal criterion of -1 SDs below a
16 neurotypical mean will result in mistaken diagnoses (Schultz, 2010, 2013; Wakefield, 2013),
17 i.e., sixteen percent of the normally distributed general population would be diagnosed as
18 abnormal on a single test, despite such individuals being cognitively intact. Thus, the DSM-5
19 in its current form poses risks to neurotypical patients by diagnosing impairments where none
20 exist.

21 However, we must not ignore the converse risks absolute cut-offs pose when patients are
22 erroneously rejected as cognitively intact, i.e., missed diagnoses. Such false negatives are
23 rarely highlighted in critiques of the DSM-5 approach to neurocognitive disorders (Schultz,
24 2010, 2013; Wakefield, 2013), despite missed diagnoses and pathologizing normality often
25 having a common cause, i.e., when patients’ diagnostic test performance distributions

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1 substantially overlap with neurotypicals. Numerous conditions associated with cognitive
2 impairments suffer problems with missed diagnoses, including Long COVID (Costas-Carrera
3 et al., 2022; Pihlaja et al., 2023) and dementia (Beishon et al., 2019; Potts et al., 2022), where
4 patients will often appear, at least from their cognitive test results, indistinguishable from
5 those absent of disease. It is therefore important for clinicians and researchers to understand
6 the risks associated with missed diagnoses, and how they can occur. This will help avoid
7 dogmatic thinking that the DSM-5 approach is infallible when assessing whether a patient is
8 impaired.

9 *Can developmental prosopagnosia reveal the DSM-5's limitations?*

10 To illustrate how the DSM-5 approach can result in missed diagnoses, I present the
11 case of developmental prosopagnosia (DP). This is a lifelong condition characterised by
12 severe difficulties recognising facial identity (Avidan & Behrmann, 2008; Bate et al., 2014;
13 Behrmann & Avidan, 2005; Behrmann et al., 2005; Bennetts et al., 2024; De Haan, 1999; De
14 Haan & Campbell, 1991; Duchaine & Nakayama, 2006; Halder et al., 2024; Maw et al.,
15 2024; McConachie, 1976; Thomas et al., 2009), affecting 1.88-6% of the general population
16 (Burns, 2023; Burns et al., 2022; Gray et al., 2017; Kennerknecht et al., 2006; Kennerknecht
17 et al., 2008). It can have a substantial negative impact upon peoples' interpersonal, romantic
18 and professional relationships, causing fear, anxiety and low self-confidence (Dalrymple et
19 al., 2014; Yardley et al., 2008). While the causes of DP are unclear, it does run in families
20 suggesting a possible genetic component (De Haan, 1999; Duchaine, Germine, et al., 2007;
21 Grueter et al., 2007; Kennerknecht et al., 2008; Lee et al., 2010), which may account for the
22 wide range of neural atypicalities they exhibit (Behrmann et al., 2007; Behrmann & Plaut,
23 2013; Burns et al., 2013; 2014; Fisher et al., 2016, 2017; Fisher et al., 2020; Fox et al., 2011;
24 Furl et al., 2011; Jiahui et al., 2018; Lohse et al., 2016; Manippa et al., 2023; Righart & de

1 Gelder, 2007; Rivolta et al., 2014; Rosenthal et al., 2017; Song et al., 2015; Thomas et al.,
2 2008; Towler et al., 2012; 2018; 2016; Van den Stock et al., 2008).

3 I chose this group for the current paper first, because I have experience working with
4 them and the tests used to assess their problems. Second, these individuals suffer an
5 extremely high proportion of potentially missed diagnoses (i.e., up to 85%) when using a cut-
6 off of -2 SDs on two cognitive tasks of face processing (Bate, Bennetts, Gregory, et al., 2019;
7 Burns et al., 2022; Lowes et al., 2023). Owing to this, I had an *a priori* hypothesis (see Burns
8 et al., 2023) that problems in diagnosing would remain, even if we had used the more liberal
9 DSM-5 criteria for neurocognitive disorders.

10 While neurodevelopmental conditions like DP would not be included in the umbrella
11 term of neurocognitive disorders by the DSM-5 developers (Sachdev et al., 2014),
12 researchers have applied its principles to DP (DeGutis et al., 2023; Stumps et al., 2020).
13 Similarly, even though the updated DSM-5 stresses that clinicians must not rigidly follow its
14 recommended diagnostic cut-offs (DSM-5-TR Neurocognitive Disorders Supplement, APA,
15 2022), this is what some researchers have suggested the field adopt (DeGutis et al., 2023).
16 Moreover, an argument could be made that acquired prosopagnosia, typically onseting after
17 an observable brain injury (Barton et al., 2001; Behrmann & Plaut, 2014; Bornstein &
18 Kidron, 1959; Bukach et al., 2006; Bukach et al., 2012; Bukach et al., 2008; de Gelder &
19 Rouw, 2000; Humphreys et al., 2007; Marotta et al., 2001), *would* come under the umbrella
20 term of neurocognitive disorders. Importantly, this form suffers similar problems as DP
21 whereby cases can perform too well on face processing tasks (Burns et al., 2023; Fysh &
22 Ramon, 2022; Josephs & Josephs, 2024). Given acquired cases are exceptionally difficult to
23 recruit in large numbers, and similar cognitive task based diagnostic issues are present in both
24 groups, it seems reasonable to use DP in the present paper to demonstrate the limitations of
25 the DSM-5 approach to diagnosing.

1 *Reasons for Missed Diagnoses*

2 In this section, I present reasons why developmental prosopagnosia may go
3 undiagnosed through the DSM-5, building on prior work (Burns et al., 2023; Epihova &
4 Astle, 2024; Lowes et al., 2023; McIntosh & Rittmo, 2021; Volfart & Rossion, 2024). My
5 intention is to provide a basis upon which professionals dealing with other neurocognitive
6 disorders can reflect on, and scrutinize, the potential issues with cognitive and behavioural
7 tests they employ. This should encourage the adoption of a more accurate, data driven
8 approach for diagnosing and treatment, where researchers and clinicians recognise the
9 limitations of the DSM-5 method, i.e., we should attempt to validate self-reported complaints
10 with objective data, rather than using arbitrary cognitive task cutoffs to reject subjective
11 complaints.

12 Before I begin, I should mention some researchers believe self-identified DP cases do
13 not have the condition when their individual cognitive test scores miss diagnostic cutoffs.
14 While theoretically possible, I largely reject this hypothesis. In my experience, historically
15 missed cases describe qualitatively similar face recognition failures during interview as those
16 who do meet criteria, and exhibit quantitatively comparable symptoms (Burns et al., 2023).
17 Moreover, it is arguably easy to detect when you fail to recognise a familiar person during a
18 conversation, as it is patently obvious that they know who you are, but you do not recollect
19 them (Burns, 2023; Burns et al., 2023; Tsantani et al., 2021). Consider the types of
20 conversations you have with familiar people; they are intuitively different from those struck
21 up by strangers. As I suspect most, if not all, missed cases have DP, this review focuses on
22 why the DSM-5 fails to diagnose those with the condition.

23 This first reason why the DSM-5 fails is because researchers and clinicians do not
24 follow the guidance set out by those who develop diagnostic tests. This has been a consistent
25 issue in the DP literature since the Cambridge Face Memory Test (CFMT), an unfamiliar face

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1 memory task widely used to diagnose developmental prosopagnosia, first came into print
2 (Duchaine & Nakayama, 2006). Its developers reported that it failed to detect impairments at
3 the -2 SD level in 25% of DP cases, and 12.5% at -1 SD. As a result, the authors stated that
4 professionals should not solely rely on it for a diagnosis (Duchaine & Nakayama, 2006).
5 Despite this, researchers and clinicians have not heeded these warnings. If you read the
6 literature over the last 10-15 years, you will find impairment on the CFMT was essential for a
7 diagnosis in the majority of papers (Burns et al., 2023; DeGutis et al., 2023), and I must
8 admit to being guilty of this myself (Burns et al., 2017; Burns et al., 2017; Burns et al., 2014;
9 Wilcockson et al., 2020). Thus, even when developers of diagnostic tests highlight their
10 limitations, professionals will fail to acknowledge them. This will result in patients
11 erroneously told they do not have developmental prosopagnosia, simply because the tests and
12 cutoffs we enforce do not capture every patient's impairment.

13 Why do tasks like the CFMT fail to detect atypicality in every self-identified case?
14 One reason may be that such tests suffer imperfect ecological validity (Burns et al., 2023;
15 Ramon et al., 2019). This occurs if they fail to fully capture the problems a patient
16 experiences in the real world. Alternatively, such tasks may accurately reflect the problems
17 they suffer from, but fail to clearly detect the superior abilities of neurotypicals. In either
18 case, the performance distributions of DP and neurotypicals will overlap to such an extent
19 that they render the DSM-5's -1 SD cutoff ineffective for diagnostic purposes.

20 To illustrate how tests can potentially lack validity, let us consider the defining
21 characteristics of DP: consistent failures when attempting to recognise personally familiar
22 people, such as co-workers, friends and even family members. In an ideal world, it would
23 seem sensible to use these people in our diagnostic tests. However, this is exceptionally
24 impractical due to the consent requirements of all involved, and the time constraints on
25 researchers and clinicians who must create such tasks. As a solution, a famous faces test

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1 (FFT) is almost always used to aid a diagnosis (Bate & Tree, 2017; Burns et al., 2022;
2 Dalrymple & Palermo, 2016), where patients are required to recognise images of highly
3 familiar celebrities.

4 However, this test cannot be easily standardised given that people of different
5 cultures, different personal interests, and different age groups will be more familiar with
6 certain famous faces than others. This may partly explain the heterogeneity of 15-35% of
7 those who self-identify as suffering from developmental prosopagnosia failing to score below
8 -1 SD on this task (Bate et al., 2019; Burns et al., 2023; Lowes et al., 2023). When employed,
9 this cutoff simply removes the top end of the homogenous DP performance distribution when
10 plotted with those who do meet criteria (Figure 1). If we assume that they are all part of the
11 same DP group as the distributions suggest, then the -1 SD cutoff will inevitably exclude
12 many from a diagnosis.

13 Why might people with DP score above the DSM-5 cutoff on the famous faces test?
14 These tasks almost always use a single, still image of each celebrity. Maybe neurotypicals
15 rely more heavily on movement when recognising familiar faces in the real world than DP
16 cases. This means neurotypical performance when using photographs will be shifted down so
17 that many DP cases land above the -1 SD cutoff, despite their problems in real life. Another
18 reason may be that people with developmental prosopagnosia have more problems in real
19 world settings than is captured by a computer screen based famous faces test¹. This could
20 occur if the brain processes celebrities, or rather celebrity photographs, to some extent
21 differently from personal acquaintances (Herzmann et al., 2004; Ramon & Gobbini, 2018;

¹ Curiously, video clips of famous faces abolish the recognition impairments that are apparent when still images are used (Bennetts et al., 2015). Does normal performance on what is a more ecologically valid test due to the inclusion of movement, not suggest that cognitive tests fail to capture something with regards to DP cases' complaints? Maybe diagnosing DP cases through still image recognition impairments, we are removing those who are largely intact on this task, but who may suffer difficulties integrating identity related information when faces are moving. Anecdotally, DP cases who perform too well on cognitive tests to acquire a diagnosis have told me that they find moving faces particularly challenging in contrast to photographs.

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1 Taylor et al., 2009; Wiese et al., 2022), where the recognition of the former may be partly
2 intact in DP, while the latter is not. In any case, given that 15-35% of people with self-
3 identified prosopagnosia perform too well on famous faces tests (Bate et al., 2019; Burns et
4 al., 2022; Lowes et al., 2023), we must acknowledge such tasks' validity limitations can
5 theoretically cause missed diagnoses.

6 Cognitive tests also suffer imperfect test-retest reliabilities too, whereby a patient can
7 acquire a diagnosis one day, but then fail to gain one the next. To illustrate this, I reanalysed
8 data from Murray and Bate (2020) who retested DP cases days to months apart after an initial
9 assessment on the CFMT. Out of their 70 cases, 29% failed to replicate their initial diagnostic
10 status using the -1 SD cutoff, shifting from DP to neurotypical, or from neurotypical to DP
11 (Figure 2). Importantly, the CFMT has been discoverable from internet searches over the
12 years, so those suspecting that they may have the condition could have taken it prior to
13 formal testing. Given 80% of Bate and Murray's (2020) cases that crossed the -1 SD
14 threshold on their second attempt moved from potentially diagnosed to missed should give
15 cause for concern. This is because many DP cases will miss acquiring a diagnosis simply
16 because of their curiosity to seek out an initial online CFMT self-assessment prior to
17 contacting a clinician for testing. Thus, the DSM-5 will fail to diagnose many patients
18 because of imperfect test-retest reliabilities².

19 *Problems with two diagnostic tests*

20 It is important to note that scoring below -1 SD on a single cognitive test is not
21 enough to acquire a diagnosis in the DSM-5 (Sachdev et al., 2014). A patient must score
22 below -1 SD on *two* tests (Sachdev et al., 2014). However, this additional requirement is
23 especially problematic as it exacerbates the issue of missed diagnoses: maybe all cognitive

² Please note, Murray and Bate (2020) showed changing diagnostic status was also an issue when using the -2 SD cutoff.

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1 tests suffer the validity and reliability issues outlined above. If true, our ability to diagnose
2 any cognitive impairment will be constrained by the statistical power of the weakest test.
3 Power in this case is simply the percentage of our patient sample that scores below any
4 arbitrary cutoff (i.e., alpha) we have chosen for our two tests³ (McIntosh & Rittmo, 2021).
5 When the famous faces test requires a -1 SD cutoff, analysing data from recent papers
6 suggests its power ranges from 65-85% (e.g., Bate et al., 2019; Burns et al., 2022; Lowes et
7 al., 2023). The CFMT is the most widely used standardised DP diagnostic assessment in
8 combination with the FFT (Burns et al., 2023; DeGutis et al., 2023). However, it too misses
9 many cases, exhibiting power of only 50-79% at the -1 SD cutoff (e.g., Bate et al., 2019;
10 Burns et al., 2023; Lowes et al., 2023). If we require impairment on both the FFT and CFMT,
11 then power will likely decline further, with it impossible to achieve overall power to diagnose
12 higher than that provided by the lowest powered test.

13 To illustrate this, I plotted the FFT and CFMT data (Figure 1) from a large sample of
14 165 self-identified DP cases reported in Bate et al. (2019). Eighty-five percent met the -1 SD
15 diagnostic criteria on the famous faces test, while 79% met criteria on the CFMT. When we
16 required impairment on both tests, power fell below the lowest of the two, namely the CFMT,
17 with 70% diagnosed. Many people who self-identify as having DP will therefore never
18 acquire a diagnosis simply because of power constraints. Furthermore, modelling work has
19 shown testing additional control participants will have limited scope for improving power
20 (McIntosh & Rittmo, 2021).

21 Another issue with requiring impairment on two cognitive tests is that it misses cases
22 who are, objectively speaking, highly abnormal in terms of their single test score. We can see

³ My discussion of power limitations is comparatively brief compared to prior works on the subject in neuropsychology (McIntosh & Rittmo, 2021) and DP (Burns et al., 2023). I recommend both papers, but especially the first which provides a convincing demonstration of how cognitive tests can lack power.

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1 this in the bottom left panel of Figure 1. The FFT cut-off excludes cases who score more
2 poorly than -2 SDs on the CFMT, which is the two-tailed threshold for an individual's test
3 score being statistically significant in terms of atypicality. Despite these cases suffering
4 severe difficulties when learning new faces, the DSM-5 requirement of impaired on two tests
5 means they will never acquire a diagnosis. These problems appear even more pronounced
6 when we examine cases excluded by the CFMT, with many scoring between -2 to -7 SDs on
7 the FFT. Can we honestly state someone who scores almost -7 SDs on the FFT does not have
8 developmental prosopagnosia? I do not believe so. Especially when such cases may be spared
9 in learning unfamiliar faces over several seconds, as tested by the CFMT, but fail to
10 effectively recollect identity for long-term recognition, as is required in the FFT and daily
11 life. Thus, while adding a DSM-5 diagnostic option of impaired on a single task at the -2 SD
12 level may counter these issues, the data shows the current DSM-5 excludes many objectively
13 atypical DP cases.

14 Another problem with requiring two tests to diagnose neurocognitive disorders is that
15 only one of them may have perfect, or close to perfect, validity. In DP, this would likely be
16 some form of assessment that captures their failings to recognise personally familiar people
17 in the real world. Even if this hypothetical task could detect impairment at the -1 SD level in
18 all cases, no other task would arguably be as good. This is because the defining problems of
19 the condition are captured by this perfect test, and any other test will likely detect related,
20 albeit imperfect, peripheral aspects of their day-to-day difficulties. As a result, requiring
21 impairment on a second test will inevitably exclude patients from a diagnosis. This is
22 demonstrated in a hypothetical famous faces test (bottom right panel of Figure 1) capable of
23 detecting impairment in 100% of DP cases at the -1 SD level. Despite this perfect assessment,
24 overall power to diagnose a patient will be equal to that provided by the second test, namely
25 the Cambridge Face Memory Test (79%), with many atypical FFT cases missed. Thus, if one

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1 cognitive task is perfect for diagnosing, then we should simply use one, rather than
2 redundantly introducing a second as the DSM-5 recommends.

3 Relatedly, the DSM-5 only describes in general terms which two tests should be used
4 to diagnose impairments within each of its six cognitive domains (DSM-5, 2014). For
5 example, in the visual-motor category, face perception and/or recognition tasks are referred to
6 as potential candidates with few details beyond that (DSM-5, 2014). This risks clinicians and
7 researchers viewing perception (e.g., what's makes this face unique?), unfamiliar face
8 recognition (e.g., can I recognise a face that I have been briefly exposed to?), and familiar
9 face recognition (e.g., can I identify a personally known or famous face?) tests as equally
10 valid diagnostic tools. However, a famous faces test that controls for familiarity is currently
11 the best for detecting single case atypicality (e.g., Bate et al., 2019), and the closest that can
12 come to measuring the severity of symptoms described by those with DP (e.g., Bate et al.,
13 2019). Thus, if familiar face recognition is the cognitive construct that is impaired in this
14 group, then we should only use diagnostic tasks that measure this construct. While perception
15 and unfamiliar face recognition tasks may be useful for identifying subtypes of DP, they can
16 lack the validity and sensitivity of the famous faces test. Thus, the DSM-5 does not contain
17 sufficient details that enable clinicians and researchers to make informed choices about which
18 two diagnostic tasks are the most valid.

19 Another issue with the DSM-5 is that the strength of the relationship between the two
20 tests will introduce a unique bias into the types of patients we can diagnose. This is illustrated
21 in Figure 2, with our 100% powered hypothetical FFT, and a second hypothetical test that
22 only has around 50% power. When the correlation between the two is perfect (i.e., $r = +1$),
23 we will exclusively diagnose the lowest scoring participants on the FFT test. By contrast, if
24 there is no correlation between the two (i.e., $r = 0$), we will sample cases from throughout the
25 FFT distribution. It is important to note that the further the relationship moves from $r = +1$ to

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1 $r = 0$, the probability of excluding cases who are more severely impaired on the FFT
2 increases. Thus, while we are diagnosing a sample that reflects the breadth of the FFT
3 distribution when $r = 0$, we will also exclude many highly atypical cases from a diagnosis
4 too. The DSM-5 requirement of impaired on two tests will therefore introduce a correlation
5 dependent bias in the types of patients we can ever diagnose, study, and treat.

6 In summary, the DSM-5 approach to diagnosing cognitive impairment is extremely
7 limited due to issues in cognitive and behavioural testing. These include imperfect validities,
8 test-retest reliabilities, and a failure to acknowledge diagnostic tests' limitations. Similarly,
9 requiring impairments on a second task will further exclude objectively atypical cases. By
10 highlighting these pitfalls in DP, I encourage clinicians and researchers working in other
11 neurocognitive disorders to reevaluate their diagnostic methods. Only by doing so can we
12 hope to develop more sensitive, patient-centred, diagnostic approaches.

13 *Consequences of missed diagnoses*

14 While it is important to acknowledge why missed diagnoses occur, it is equally important
15 to recognise the myriad of negative consequences they create. For example, patients can
16 question their own lived-in experiences and sanity after being told there is nothing wrong
17 with them by a medical practitioner ([Au et al., 2022](#); [Eyal, 2022](#); [Wise, 2022](#)). This is a
18 frequent occurrence in diverse conditions linked to problems in cognition, such as Long
19 COVID ([Au et al., 2022](#); [Eyal, 2022](#)), dementia ([Nelson & O'Connor, 2008](#); [Rentz et al.,](#)
20 [2000](#); [Rentz et al., 2004](#)), electroconvulsive therapy patients ([Rose, 2022](#); [Rose et al., 2003](#)),
21 and DP ([Burns et al., 2022](#)). Without a formal diagnosis, patients will not be able to move
22 forward with insurance claims, impacting their ability to acquire support and treatment. This
23 will be particularly problematic if treatments are only effective at an early stage of disease, as
24 those missed from a diagnosis will not be treated in time. Also, an absence of a diagnosis will
25 block patients from legally protected, workplace related, reasonable adjustments to

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1 accommodate their loss of function. This means that missed diagnoses will severely impact
2 the lives of patients with neurocognitive disorders and their families.

3 The above could be described as patient focused issues, but missed diagnoses will also
4 negatively impact science. For example, if only the most extremely impaired cases who score
5 below -1 SD are diagnosed, then prevalence rates and effect sizes of impairments will never
6 be accurate, owing to missed cases being excluded from the top end of the disorder's
7 performance distribution. Such exclusions will undermine any epidemiological work that
8 seeks to assess the aetiology and outcomes of neurocognitive disorders, as missing cases will
9 potentially bias results and waste vast resources.

10 Missed cases will also impact neurocognitive models, because their absence risks altering
11 dissociations and associations to such an extent, they render the model's underlying evidence
12 base meaningless. To illustrate this, imagine a hypothetical diagnostic test of face recognition
13 has power of roughly 50% at the -1 SD level, and is correlated with an object recognition test
14 (Figure 3). The effect size of impairment in potentially diagnosed DP cases on our faces test
15 averages -1.8 SDs below a neurotypical mean, with a comorbid impairment of -.62 SD in
16 object recognition. These group level impairments suggest the two processes are to some
17 extent not dissociable. Thus, a cognitive model derived from this data shows overlapping
18 functionality between face and object recognition.

19 However, our 50% powered diagnostic test has excluded many DP cases. If we include
20 them, then we will find we overestimated our DP group's impairment on this task, as it is
21 now reduced to -1 SD. Importantly, the impairment in object recognition disappears. The
22 resulting cognitive model now favours a dissociation between these processes⁴. Moreover,
23 given object recognition is now intact, the correlation between faces and objects may be

⁴ Please note, excluded cases will only affect neurocognitive models if there is a relationship between face and object recognition. If there is no correlation (i.e., $r = 0$), then missed diagnoses will only alter the severity of group level face processing impairments in DP. By contrast, object recognition will not be affected.

1 explained by another process. For example, attention is frequently cited as the domain
2 general cause of such associations because face and object recognition are believed by many
3 to be reliant upon dissociable brain networks (Kanwisher, 2017; Kanwisher & Yovel, 2006;
4 McKone et al., 2007; although see Burns, Arnold, et al., 2019; Gauthier et al., 2017) Thus, if
5 attention were the cause of the relationship between face and object recognition, then adding
6 data from an attention task as a covariate should abolish the link. Alternatively, the
7 correlation could be due to domain general processes being utilised, but a dissociable face-
8 specific component being impaired, hence the lack of group level object recognition
9 impairments. Either way, exclusions similarly affect our cognitive model's assumptions.

10 The same problem would also be apparent when identifying abnormalities in structural or
11 functional MRI work. Imagine replacing object recognition in Figure 3 with the neural
12 activation in a brain region. This region's BOLD response is correlated with performance on
13 our hypothetical face recognition test used to diagnose DP. Again, as we excluded many of
14 our DP cases, we find this brain region exhibits a lower-than-normal BOLD response,
15 suggesting it is atypical in DP. When we add the missed cases, this atypicality disappears.
16 Our neural model therefore changes from this region seeming essential for face recognition,
17 to being of limited importance and appearing intact in DP. Remarkably, a recent study
18 showed DP cases exhibited reduced neural responsiveness across much of the brain (Guo et
19 al., 2018). However, as these atypicalities were shown in only a minority of the most
20 extremely impaired DP cases (i.e., diagnosed -2 SDs on two face processing tests), they may
21 as a population appear intact if we had included the full range of potential cases. Missed
22 diagnoses will therefore impact virtually every area of cognitive and neurocognitive science,
23 undermining any trust we have in the literature.

24 Failing to diagnose patients correctly also means treatments can erroneously appear
25 effective due to statistical artefacts. This is because when we only diagnose and treat a

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1 sample that perform the lowest on cognitive assessments, post-treatment improvements
2 identified through retests risk being nothing more than a regression to the mean (Barnett et
3 al., 2005; Finney, 2008; Morton & Torgerson, 2005). That is, patients who score
4 exceptionally poorly in the first instance to gain a diagnosis, will typically perform better on a
5 retest due to the simple fact that there is only one direction their scores can go post treatment.
6 Conversely, patients with the mildest impairments that fail to acquire a diagnosis may be the
7 most responsive to interventions due to their residual cognitive abilities. Unfortunately, as
8 they have been blocked from acquiring a diagnosis, they will never be included in clinical
9 trials that assess a treatment's efficacy. In summary, failed diagnoses will have a substantial,
10 negative impact upon patients, their families, and science.

Validating a Symptom-Based Approach to Diagnosing

12 Given the shortcomings of cognitive tests as diagnostic tools, we must explore viable
13 alternatives when substantial overlaps exist between the performance distributions of
14 neurotypicals and neurocognitive disorders, i.e., when the DSM-5 approach proves
15 ineffective at distinguishing between impaired versus intact cognitive abilities at the level of
16 the individual patient. One solution might be a symptom questionnaire if it were shown more
17 effective at differentiating a patient's complaints from neurotypicals. If so, we must validate
18 it. One way of accomplishing this would be to pool missed cases' cognitive task data together
19 to enhance power for detecting objective impairments at the group level. This will transform
20 missed patients' atypical levels of symptoms from mere subjective complaints, into validated
21 indices of underlying cognitive deficits. Moreover, the remaining patients' self-reported
22 complaints will have been validated at the -1 SD level on two cognitive tasks via the DSM-5
23 approach. Given these dual approaches to detecting impairment across the whole patient
24 sample, a symptom-based method, if more effective, would become a viable diagnostic
25 alternative to the DSM-5.

1 As a proof of concept, I assessed a large sample ($n = 61$) of self-identified developmental
2 prosopagnosia cases. I first quantified the proportion of these individuals that would fail to
3 acquire a diagnosis through the DSM-5, then sought to identify their group-level impairments
4 on multiple tasks to validate a symptom-based approach to diagnosing. Finally, I replicated
5 these deficits in a separately collected sample (Bate et al., 2019), before assessing all data
6 using meta-analyses. If missed cases exhibit deficits in face processing, it will confirm the
7 DSM-5 fails to diagnose objectively impaired DP. This would mean the one-size-fits-all
8 approach recommended by the DSM-5 does not always work, as it blocks people with
9 objective impairments from acquiring a diagnosis. Instead, we should tailor diagnostic
10 approaches to the specific cognitive impairments we are assessing.

11 **2. Methods**

12 **2.1. Transparency and Openness**

13 All data required to replicate our results is available on the Open Science Framework
14 (<https://osf.io/3x86n/>). As I do not own the copyright for the tasks, I do not make them
15 available. The PI20 is freely available in the original paper (Shah et al., 2015).

16 **2.2. Participants**

17 I report how I determined my sample size, all data exclusions, all manipulations, and all
18 measures in the study. The first DP sample comprised 62 cases whose ages ranged between
19 18-72 years old ($M = 41.5$, $SD = 14$), with nine males, and three who identified as neither
20 male nor female. All cases reported severe, lifelong troubles with faces with no obvious
21 historical reason for it being acquired (Burns et al., 2023). Due to COVID-19 restrictions, all
22 tests were carried out online. One DP case was excluded for failing two out of two attention
23 checks throughout the tasks. Another failed to move any faces during the Cambridge Face
24 Perception Test: i.e., 62-year-old female who made 96 errors on upright and inverted (Table
25 1). Presumably, she had a problem with her mouse on this task, so included her other data

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1 where possible. This left 61 DP cases for all assessments, except for the CFPT related
2 measures ($n = 60$). Data was not collected on participants' other cultural backgrounds (e.g.,
3 socioeconomic status; Applebaum et al., 2018).

4 Fifty-two controls participated: ages ranged 22-68 years old ($M = 38.8$, $SD = 11.3$) with
5 31 males. While there were many more males than females in this group, and women
6 typically recognise faces better than men (Herlitz & Lovén, 2013), there is a concern this may
7 result in an underestimation of face recognition difficulties in our DP sample. I ran
8 independent t -tests on the control data from all measures and found no significant gender
9 related effects [all $ps > .08$, $BF_{10} \leq 1$], thus do not include gender in any of my analyses.

10 Three control males reported lifelong troubles with faces, and scored highly atypical (i.e.,
11 significant at the one-tailed alpha threshold) on the prosopagnosia index questionnaire (74, 68
12 and 63), suggesting they likely have DP. I therefore excluded them from all analyses.

13 Power was difficult to estimate *a priori*, as it was unclear just how many cases would fail
14 to acquire a diagnosis using the DSM-5 approach, or what their level of impairment might be.
15 Given the DSM-5 diagnostic criterion is -1 SD on two cognitive tests, and the average level
16 of cognitive impairment in the self-identified DP cases is just below -2 SDs on the CFMT
17 (Burns et al., 2023), I presumed the group-based deficits in the DSM-5 missed cases would
18 be close to -1 SD. This is because their distribution was potentially skewed (i.e., being the
19 high performing tail-end of the normally distributed DP group), with most cases congregating
20 around this level. An effect size (*Cohen's d* = .8) was chosen based on this hypothesis. This
21 required 26 participants in the excluded DP group (i.e., those who failed to meet DSM-5
22 criteria) and 26 in the control group, to detect effects with an alpha of .05 and power of 80%.
23 A *post hoc* power analysis based on the 23 DP cases who were missed by the DSM-5
24 approach, and 48 controls, suggested that power was 87%, with 80% power to detect down to
25 *Cohen's d* = .72.

1 To avoid reducing power further, I do not make any corrections for multiple comparisons,
2 especially given these are difficult to recruit cases. However, to reassure readers the results
3 were not false positives, I largely replicated my findings in an additional DP group (Bate et
4 al., 2019). Moreover, I ran *p*-curve meta-analyses to ensure any impairments detected in
5 cases excluded from a DSM-5 diagnosis had evidential value (Simonsohn et al., 2014).

6 None of our experiments or hypotheses were formally pre-registered. While low-level
7 object recognition difficulties were not tested (e.g., Birmingham Object Recognition Battery:
8 BORB; Riddoch & Humphreys, 2022), no DP cases disclosed general problems with vision,
9 and when I historically included the BORB in DP testing, those with the condition did not
10 exhibit problems. This has been objectively confirmed across 200 cases in recent papers
11 (Bate et al., 2019; Lowes et al., 2023), with only one DP case exhibiting possible difficulty
12 (Lowes et al., 2023). Thus, despite some concerns about omitting such testing (Nørkær et al.,
13 2024), low-level vision problems are no more prevalent in DP than neurotypicals, meaning
14 that such tasks are unnecessarily onerous for DP testing in the absence of patient complaints.

15 **2.3. Procedure and Materials**

16 A battery of cognitive tests that have historically been used to diagnose developmental
17 prosopagnosia (Burns et al., 2023; DeGutis et al., 2023) were completed by all participants.
18 These included a 72-trial assessment of unfamiliar face memory (the Cambridge Face
19 Memory Test: CFMT; Duchaine & Nakayama, 2006), an unfamiliar face perception test
20 which included eight upright trials and eight inverted (the Cambridge Face Perception Test:
21 CFPT; Duchaine et al., 2007), and a validated, 30-trial familiar face memory test (i.e.,
22 Famous Faces Test: FFT) developed by my own lab (Burns et al., 2023). FFT scores were not
23 corrected for participants' familiarity (i.e., we did not provide celebrities' names afterwards
24 to adjust participants' possible scores based on these responses). Participants also completed

1 the Prosopagnosia Index questionnaire (Shah et al., 2015). All DP cases' individual scores
2 and full test details are presented in Supplementary Information here (<https://osf.io/3x86n/>).

3 The Bate et al. (2019) sample completed their own FFT, the CFMT and only the eight
4 CFPT upright trials. Bate et al.'s (2019) FFT differed from mine in a few ways. First, they
5 used two versions, one for participants aged under 35 years old, and one for participants aged
6 ≥ 35 ; faces were selected based on pilot work with these age groups. Both versions used 60
7 faces, and in contrast to my FFT, each participant's total possible correct scores were
8 adjusted by removing celebrities they were not familiar with by name. Full details of Bate et
9 al.'s (2019) methods can be found in their open access paper.

10 Ethical approval was granted by Edge Hill University Ethics Review Board, with all work
11 carried out in accordance with the 1964 Helsinki Declaration on human testing. All
12 participants gave informed consent, and for their anonymised data to be published.

13 **3. Results**

14 **3.1. The DSM-5 diagnoses 62% of DP cases, the symptom-based approach 100%**

15 The DSM-5 approach to neurocognitive disorders requires participants to score more
16 poorly than -1 SD from the neurotypical mean on two cognitive tests. DeGutis et al. (2023)
17 recommended that these should be two tests of face memory when diagnosing DP. I used this
18 guidance with the CFMT and FFT because they have historically been the two most widely
19 used tests to diagnose DP (Burns et al., 2023; DeGutis et al., 2023). This revealed a striking
20 38% of self-identified DP cases were excluded from a diagnosis. By contrast, 100% were
21 classified as atypical using the prosopagnosia index via a Crawford's *t*-test (Crawford &
22 Howell, 1998).

23 We wanted to assess whether the DSM-5 approach diagnosed DP cases who reported
24 more severe symptoms than those that were excluded. To ensure higher power to detect
25 potential symptom differences between the groups, and because all DP cases were highly

1 abnormal in their self-reported symptoms at the individual level (i.e., more than -2 SDs below
2 a neurotypical mean), I performed a between participants *t*-test on the DSM-5 diagnosed and
3 excluded DP cases' PI20 scores, i.e., I did not include control data. This revealed excluded
4 cases reported fewer problems with faces [$M = 79, SD = 8.17$] than those who acquired a
5 diagnosis using the DSM-5 approach [$M = 83.61, SD = 6.84, t(59) = 2.37, p = .021, Cohen's$
6 $d = .63$]. The DSM-5 approach therefore seems to capture some of the symptom differences
7 between those who are diagnosed and those that are not. Figure 4 illustrates the mean level of
8 z-score impairment for both the DP groups across all measures.

9 **3.2.Excluded DP cases are impaired in unfamiliar face memory**

10 To validate excluded cases' highly abnormal symptom complaints, we compared their
11 CFMT scores to the neurotypical group (Figure 4). This confirmed they were impaired in
12 unfamiliar face memory abilities [Excluded DP $M = 51.52$ trials correct, $SD = 8.39$;
13 Neurotypical $M = 59.7, SD = 9.8, t(69) = 3.43, p < .001, Cohen's d = .87$]. Thus, the DSM-5
14 approach to diagnosing neurocognitive disorders excludes DP cases who exhibit objective
15 difficulties with unfamiliar faces.

16 **3.3. Excluded DP cases are impaired in upright face perception**

17 The Cambridge Face Perception Test (Duchaine, Germine & Nakayama, 2007) is often
18 used in neuropsychological assessment to identify DP cases who suffer from the apperceptive
19 form of the disorder (Biotti & Cook, 2018), i.e., those that have difficulties telling faces apart
20 from one another. I used the CFPT to assess whether the DSM-5 approach would exclude DP
21 cases who were objectively impaired in face perception. Confirming this, they made
22 significantly more errors on this task [$M = 41.9$ errors, $SD = 12.02$] than the control group [M
23 $= 33.33, SD = 14.16, t(69) = 2.5, p = .015, Cohen's d = .64$]. This means we have validated
24 excluded cases' self-reported complaints in unfamiliar face memory and face perception.

25

1 **3.4.Excluded DP cases’ holistic perception abilities are impaired**

2 Holistic perception is characterised by the brain encoding an interaction between a face’s
3 features to create a salient, unitary percept (Burns & Wilcockson, 2019; Dal Lago et al.,
4 2023; 2024; Luo et al., 2017; Maw et al., 2023), with reduced abilities a suggested cause of
5 face processing difficulties in DP (Avidan et al., 2011; DeGutis et al., 2012; although see:
6 Biotti et al., 2019). I assessed whether excluded DP cases were impaired in holistic
7 perception using the corrected inversion scores from the CFPT as an index (Supplementary
8 Information). This revealed that excluded cases [$M = .69, SD = .53$] exhibited smaller
9 inversion effects in comparison to our controls [$M = 1.16, SD = .9, t(66.04) = 2.74, p = .008,$
10 *Cohen’s d = .58*]. We have therefore validated excluded DP cases’ symptom complaints via
11 three objective measures: CFMT, CFPT upright and CFPT Holistic Perception.

12 **3.5. Excluded DP cases exhibit impairments when judging famous faces**

13 DSM-5 excluded DP cases exhibited deficits in face perception and unfamiliar face
14 recognition. However, familiar faces, such as friends and celebrities, are thought processed in
15 a partially distinct way from unfamiliar faces (Ellis et al., 1979; Johnston & Edmonds, 2009;
16 Megreya & Burton, 2006). I therefore assessed the presence of familiar face impairments in
17 excluded cases using famous faces. This confirmed those who failed to acquire a diagnosis
18 through the DSM-5 [$M = 17.87$ trials correct, $SD = 4.75$] identified fewer celebrity faces in
19 comparison to controls [$M = 20.65$ correct, $SD = 6.2, t(55.34) = 2.08, p = .042, Cohen’s d =$
20 $.48$]. This means excluded DP cases exhibited objective impairments on all four measures of
21 face processing.

22 **3.6. Replication: DSM-5 excluded DP cases’ exhibit face memory impairments**

23 Bate et al. (2019) used the CFPT upright, CFMT and their own FFT to test 165 self-
24 identified DP cases. Strikingly, 30% of these individuals failed to meet the DSM-5 criteria
25 (Table 1). While slightly lower than the 38% I found with my sample, it largely replicated

1 this figure, i.e., a substantial number of potential DP cases will fail to acquire a diagnosis
2 using the DSM-5 method. Table 1 shows the mean z-scores for my data and Bate et al. (2019)
3 side by side for easy comparison. Importantly, analyses of Bate et al. (2019) replicated my
4 finding of missed cases exhibiting significant unfamiliar and familiar face memory
5 impairments [$ps < .013$], but did not corroborate the upright CFPT difficulties [$p = .24$].

6 **3.7. Meta-analysis support the existence of objective impairments in missed cases**

7 It is increasingly common for researchers to assess multi-experiment evidence presented
8 within their papers through a meta-analysis (e.g., [Alves et al., 2017](#); [Van Kuijk et al., 2018](#)).
9 This is an important antidote to the replication crisis psychology has faced in recent years, as
10 it can provide support that any studied effect is real ([Simonsohn et al., 2014](#)). The p -curve is a
11 widely used ([Simonsohn et al., 2014](#)) and effective ([Lakens, 2023](#)) meta-analysis technique
12 that only uses significant p -values. It is based on the fact that when true effects exist, p -values
13 will exhibit a right skew when plotted together; with the highest frequency of values
14 congregating around .01 ([Burns & Bukach, 2023](#); [Simonsohn et al., 2014](#)). By contrast, if the
15 null hypothesis is true, then p -values should appear flat (i.e., uniformly distributed).

16 I performed a p -curve using all significant p -values that confirmed missed cases'
17 objective cognitive impairments from mine and the Bate et al. (2019) samples. This meta-
18 analysis was significant [Full curve: $Z = -3.08$, $p = .001$; Half curve: $Z = -2.74$, $p = .003$], thus
19 supporting the proposal that DSM-5 missed cases' cognitive impairments contain evidential
20 value. Figure 5 illustrates the right-skewed distribution of p -values indicating they reflect a
21 real effect. As the holistic perception related p -value may not be entirely independent from
22 the CFPT upright p -value, I ran the analysis again with the former excluded: the p -curve
23 remained significant [Full curve: $Z = -2.92$, $p < .002$; Half curve: $Z = -2.83$, $p < .003$]. This
24 was replicated when I replaced the CFPT upright p -value with the CFPT holistic perception
25 p -value [Full curve: $Z = -3.13$, $p < .001$; Half curve: $Z = -3.18$, $p < .001$]. In summary, meta-

1 analyses confirm DSM-5 missed cases' objective impairments in face processing.

2 **4. Discussion**

3 The DSM-5 approach to diagnosing cognitive impairment requires a patient to score
4 beyond -1 SD on two objective tests of behaviour or cognition (Sachdev et al., 2014). I
5 outlined how this approach will result in missed diagnoses, using developmental
6 prosopagnosia as a proof of concept. Testing two large, independently collected samples, I
7 showed the DSM-5 excluded between 30-38% self-identified DP cases. Importantly, both
8 groups of missed cases exhibited impairments in familiar and unfamiliar face memory, with
9 one sample displaying face perception difficulties too⁵. These problems were further
10 confirmed through meta-analyses. The prosopagnosia index questionnaire proved more
11 effective as a diagnostic tool than the DSM-5, with the former identifying significant
12 atypicality in 100% of cases at the individual level, in contrast to the latter that ranged
13 between 62-70%. It is important to recognise that a diagnosis of DP based on a case's
14 symptoms is no longer a purely subjective measure, but reflects multiple, underlying
15 objective impairments. These deficits were found using the DSM-5 approach for individual
16 cases at the -1 SD threshold on two tests, or when that failed, at the level of the group in
17 excluded DP, thus validating the prosopagnosia index as a diagnostic tool (Figure 6).

18 This study is an important demonstration of how cognitive tests can fail to diagnose a
19 substantial proportion of objectively impaired patients. While I have shown this in one
20 clinically relevant population, it is frequent to find substantial overlaps in the performance
21 distributions of those who report cognitive impairments in other disorders versus those who
22 are neurotypical (Beishon et al., 2019; Burns et al., 2022; Costas-Carrera et al., 2022; Eyal,

⁵ There are a few potential reasons why the excluded DP cases from Bate et al. (2019) failed to exhibit perceptual impairments in contrast to my own: their DP group was several years older and contained proportionally fewer women, they excluded cases who were high in autistic traits, and they corrected famous face scores for familiarity which would have changed the types of cases diagnosed. Any, or all, of these differences may have contributed to the absence of perceptual impairments in the Bate et al. (2019) sample, although the direction of effect, while much smaller, was the same.

1 2022; Nelson & O'Connor, 2008; Potts et al., 2022; Rentz et al., 2000; Rentz et al., 2004).

2 This should pose as a warning to any clinician or researcher who tries to rigidly enforce a
3 diagnostic criterion, such as the DSM-5 cutoff of -1 SD on two cognitive tests, when it
4 clearly does not fit the patient population. Instead, I advocate the use of a transdiagnostic,
5 data driven approach, in which the whole range of the possible patient sample self-reporting
6 complaints are tested (Astle et al., 2022; Burns et al., 2023; Epihova & Astle, 2024), to
7 identify, and correct, deficiencies in diagnostic tests and cutoffs. Only by doing so, and
8 including them in our work, can we hope to improve our methods (Burns et al., 2023;
9 Epihova & Astle, 2024).

10 If a symptom-based approach is more effective at identifying a patient's atypicality in
11 daily life, then I recommend validating it, and using it, to replace the cognitive or behavioural
12 test-based approach proposed by the DSM-5. We can validate a symptom questionnaire, such
13 as the prosopagnosia index, by identifying group based, objective impairments in missed
14 cases on multiple tasks. By contrast, the remaining DP cases are already validated via the
15 DSM-5 approach at the level of the individual patient. One benefit of using a symptom-based
16 approach to diagnosing DP is that it may not be susceptible to the biases that face recognition
17 tests suffer from. For example, women are typically better at identifying still images of faces
18 in cognitive tests than men (Herlitz & Lovén, 2013; Wright & Sladden, 2003), and people are
19 typically better at recognising faces of their own ethnicity (Bate, Bennetts, Hasshim, et al.,
20 2019; Burns, Tree, et al., 2019; Childs et al., 2021; Estudillo et al., 2020; Meissner &
21 Brigham, 2001), and ages (Rhodes & Anastasi, 2012). Given that standardized cognitive tests
22 (e.g., CFMT, CFPT) almost always contain images of young adults, they will underestimate
23 face processing abilities of participants furthest away from these age groups (Burns, 2023).
24 While some may have concerns that a symptom-based approach is susceptible to malingering
25 patients (i.e., they can be easily faked), these issues are equally true of cognitive tests (Suhr et

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1 al., 2008; Suhr et al., 2021). Moreover, an argument could be made that DP cases failing to
2 meet DSM-5 criteria are highly unlikely to be malingerers, given the fact that they perform
3 *above* diagnostic cutoffs on cognitive tasks, i.e., malingerers would likely exaggerate
4 problems on the Famous and Cambridge Face Memory Tests.

5 Of course, it is theoretically possible a single cognitive test may detect atypicality in
6 every patient. Or maybe when using two tests, at least one will always detect a patient's
7 cognitive impairment at the -2 SD level. In such cases, I would recommend loosening the
8 DSM-5 criteria. For example, if we added an additional option to diagnose based on a single
9 test (i.e., -2 SD cutoff), then we would remedy the DSM-5's problems in missing highly
10 atypical cases. Similarly, incorporating patients' response times, in contrast to the historical
11 reliance on accuracy rates, improves cognitive tests' diagnostic sensitivity (Lowe et al.,
12 2023). Thus, small modifications can result in improvements to the current DSM-5 method.
13 Simply put, I would recommend using the most effective test for providing a diagnosis. This
14 would prove useful, in contrast to the self-reported symptom approach, when patients are
15 lacking awareness of their cognitive difficulties. The important message here for readers is
16 that not every cognitive impairment will be easily diagnosed by the one-size-fits-all approach
17 endorsed by the DSM-5, i.e., impaired at -1 SD on two tests. When you consider the
18 heterogeneity of patients' impairments, and the heterogeneity in cognitive tests' validities and
19 reliabilities, it seems implausible that such a rigid method will work ubiquitously. We must
20 tailor how impairments are diagnosed by assessing our best options within patient samples.
21 Only by using bespoke, rather than general, approaches can we improve patient support and
22 science.

23 I should add that there is some merit in the DSM-5 method. While I reject it as a
24 diagnostic approach in the context of DP, it did distinguish between self-reported symptoms
25 in those it diagnosed, versus those that it missed. This shows the DSM-5 can reflect cases'

1 differing levels of symptoms. There is a great deal of interest in the face recognition literature
2 as to whether people have insights into their cognitive abilities (Bobak et al., 2019; Estudillo
3 & Wong, 2021; Gehdu et al., 2023; Gray et al., 2017; Livingston & Shah, 2018; Matsuyoshi
4 & Watanabe, 2021; Nørkær et al., 2023; Oishi et al., 2024; Palermo et al., 2016; Shah et al.,
5 2015; Ventura et al., 2018). I have shown those with developmental prosopagnosia exhibit
6 accurate insights into the existence of their objective impairments, and their severity, given
7 the graded symptom levels between the DP cases on either side of the DSM-5 cutoff. This
8 rejects suggestions that excluded cases are misinterpreting their face recognition abilities, or
9 that they are suffering from a failure of meta-cognition (Arizpe et al., 2019; De Haan, 1999;
10 DeGutis et al., 2023).

11 Another benefit of the symptom-based approach is that it is much shorter (i.e., a couple
12 minutes) than the battery of cognitive tests we typically ask DP cases to complete (e.g., at
13 least 40-60 minutes). This means a symptom questionnaire, once validated as a diagnostic
14 tool, can save patients and clinicians valuable time in clinical settings. Also, there are no
15 standardised cognitive assessments for DP in all ethnicities as cognitive tests used to
16 diagnose DP are frequently geared towards Caucasian samples (e.g., Duchaine & Nakayama,
17 2006; Duchaine, Yovel, et al., 2007). This makes them exclusionary given there are
18 substantial performance variations when recognising faces from other ethnicities, e.g.,
19 Caucasians will often exhibit problems recognising Asian faces (e.g., Bate et al., 2019; Childs
20 et al., 2021; Meissner & Brigham, 2001). A symptom-based approach should, in theory,
21 negate these issues to some extent.

22 It is important to note some believe symptoms alone should never be used when
23 diagnosing cognitive impairment (DeGutis et al., 2023; DeHaan, 1999; Nørkær et al., 2024),
24 and that the solution to missed diagnoses is to develop more sensitive experimental tests
25 (DeGutis et al., 2023). I have shown here that pooling missed cases' data reveals group level

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1 impairments that are not otherwise detectable using the DSM-5. Thus, when a patient is
2 diagnosed through the symptom-based approach, we can be confident their atypical
3 symptoms reflect underlying objective impairments on multiple experimental measures
4 (Figure 6). I agree cognitive tests need to be improved in their sensitivity, validity, and
5 reliability, but such improvements will further validate the symptom-based approach.
6 Imagine we develop a cognitive testing battery that matches the PI20's sensitivity, i.e.,
7 identifying all cases as atypical at the -2 SD level. Why would we use such time-consuming
8 tasks in overstretched clinical practices when we have a validated, and rapid, symptom
9 questionnaire at our disposal? If we must wait for cognitive tests to improve, we will only
10 perpetuate the problems outlined in the Introduction, and block 30-38% of objectively
11 impaired developmental prosopagnosia cases from a diagnosis. Without the symptom-based
12 approach, such individuals will be unable to access essential treatments, support, and legal
13 protections in the workplace.

14 I must acknowledge there may be limitations to a symptom-based approach. For example,
15 prior work has shown, albeit not with the prosopagnosia index, that symptom questionnaires
16 can be susceptible to pathologizing normal behaviours in one culture over the other (Norbury
17 & Sparks, 2013). However, given the vast numbers of cases missed when using cognitive
18 tests in DP, potential cross-cultural issues in symptoms are, in my opinion, likely to have a
19 much smaller impact in terms of missed diagnoses. Also, the PI20 (Sun et al., 2021)
20 outperforms the CFMT (Murray & Bate, 2020; Wilmer et al., 2010) in test-retest reliabilities,
21 meaning that a patient's diagnostic status is less susceptible to changing from one day to the
22 next, in contrast to a symptom-based approach. Despite this, it is recommended that a team
23 independent of the scale developers assess such questionnaires, to remove potentially
24 redundant items (Boateng et al., 2018). While this has been done to some extent with the
25 PI20, the new scale was designed to improve the detection of neurotypical face recognition

1 abilities, not developmental prosopagnosia symptoms (Bobak et al., 2019). This means there
2 may be some benefits from further PI20 refinement. However, in our sample of 61 DP cases,
3 it performed exceptionally well, identifying 100% of cases as suffering atypical levels of
4 prosopagnosia symptoms beyond the neurotypical -2 SD cutoff, i.e., no self-identified DP
5 cases were erroneously reporting symptoms in the neurotypical range.

6 In summary, I have shed light on the limitations of the DSM-5 approach to diagnosing
7 neurocognitive disorders, using developmental prosopagnosia as a compelling case in point.
8 The conventional DSM-5 criterion of scoring below -1 SD on two objective tasks excludes a
9 significant percentage (i.e., 30-38%) of individuals who report severe problems in daily life.
10 By introducing a symptom-based approach, we have identified excluded DP cases'
11 complaints as significantly atypical in all instances, and validated them through their
12 underlying objective impairments. This offers a more comprehensive and patient-centred
13 perspective on diagnoses, acknowledging the limitations of cognitive tests. These findings,
14 although focused on developmental prosopagnosia, serve as a crucial reminder to clinicians
15 and researchers that diagnostic criteria must be tailored to the unique characteristics of the
16 patient population. Embracing a data-driven approach through such cases' suspected issues
17 can lead to a more effective diagnostic method and improve the accuracy of assessments. If a
18 symptom-based approach demonstrates superior effectiveness in identifying atypicality in
19 daily life, as we have shown here, then it should replace the DSM-5 method. By doing so, we
20 can enhance the diagnostic process, making it more inclusive, unbiased, and ultimately, more
21 reflective of real-world cognitive functioning.

22

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4

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7

8 **7. Conflicts of interest/Competing interests**

9 No conflicts/competing interests to report.

10

11 **8. Ethics approval**

12 Ethical approval was granted by Edge Hill University Ethics Review Board.

13

14 **9. Consent to participate**

15 All participants provided informed consent to participate.

16

17 **10. Consent for publication**

18 All participants gave consent for their anonymized data to be published.

19

20 **11. Availability of data and materials**

21 The data required to replicate my Results is available on the Open Science Framework

22 (<https://osf.io/3x86n/>)

23

24

25

1 **12. Code availability**

2 I do not own the copyright for many of the tests used so do not make them available, but I
3 thank Brad Duchaine for providing the CFMT and CFPT. The PI20 can be found in the paper
4 it was first reported on (Shah et al., 2016).

6 **13. Authors' contributions:**

7 I am responsible for all aspects of this paper.

9 **14. References**

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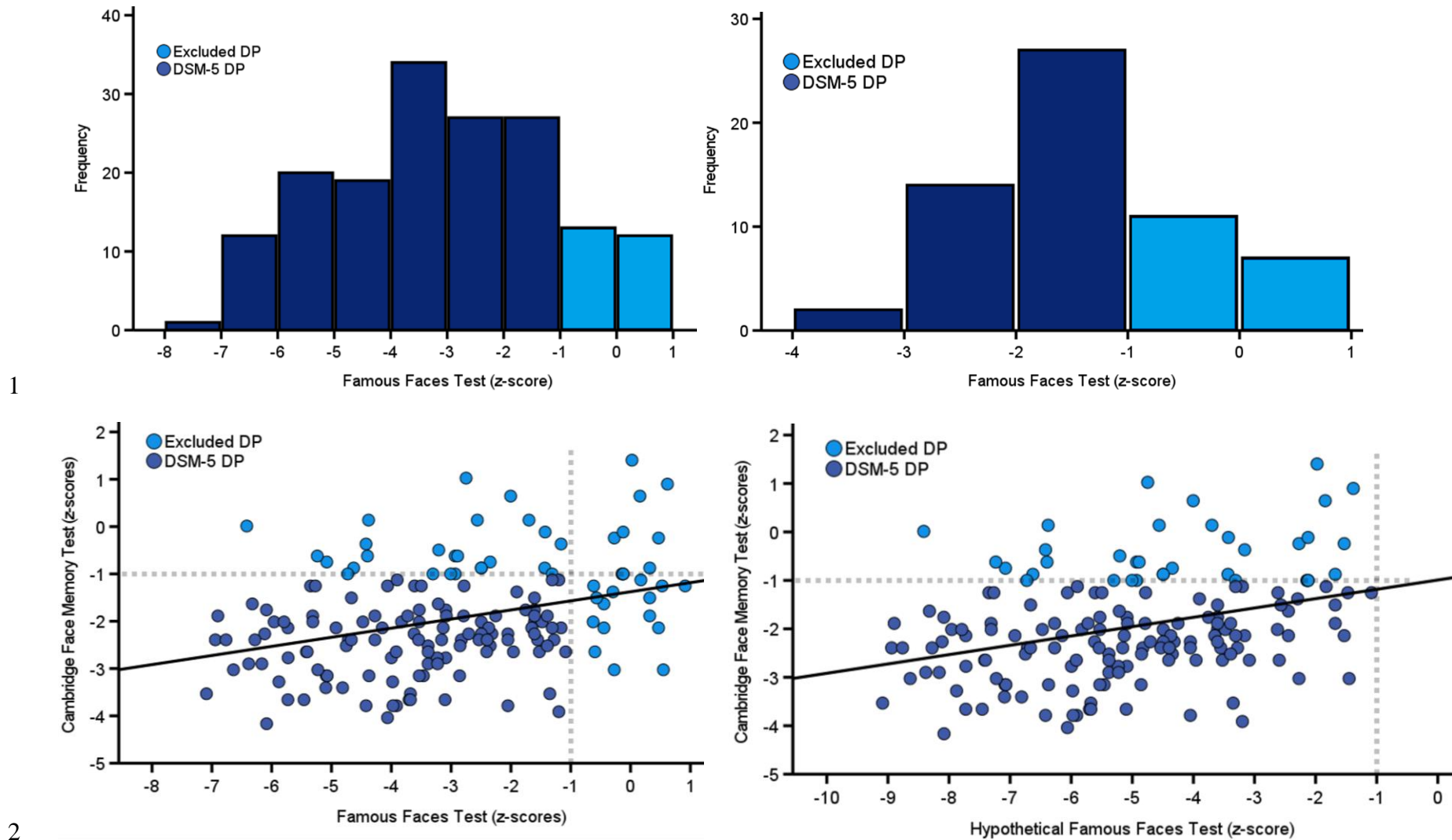
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HEADER: DSM-5 LIMITATIONS

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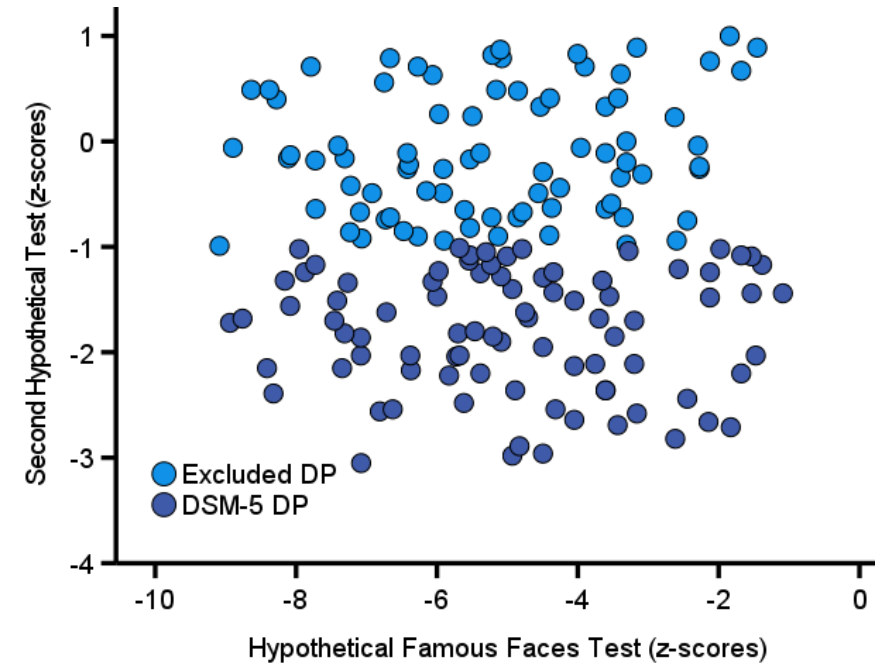
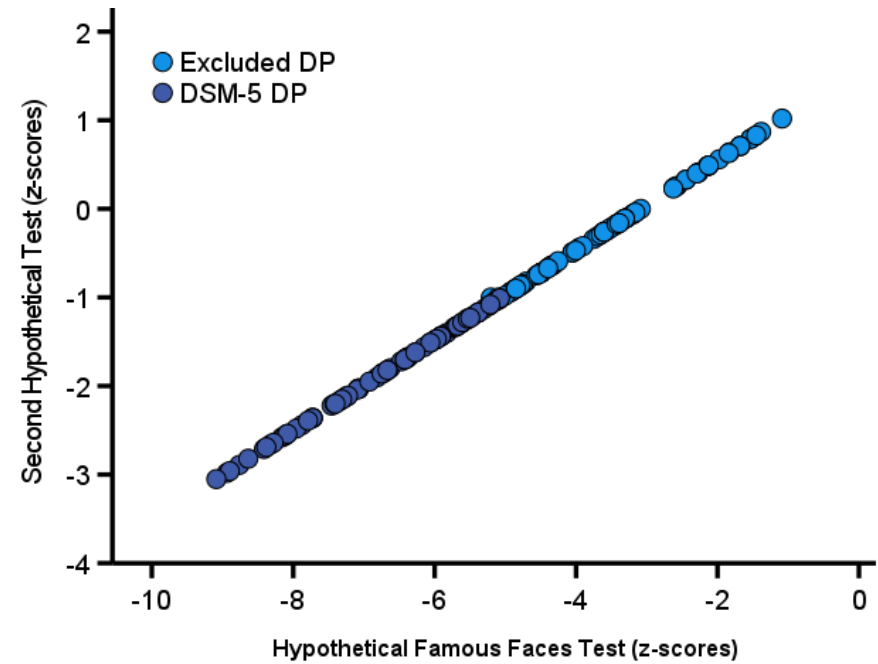
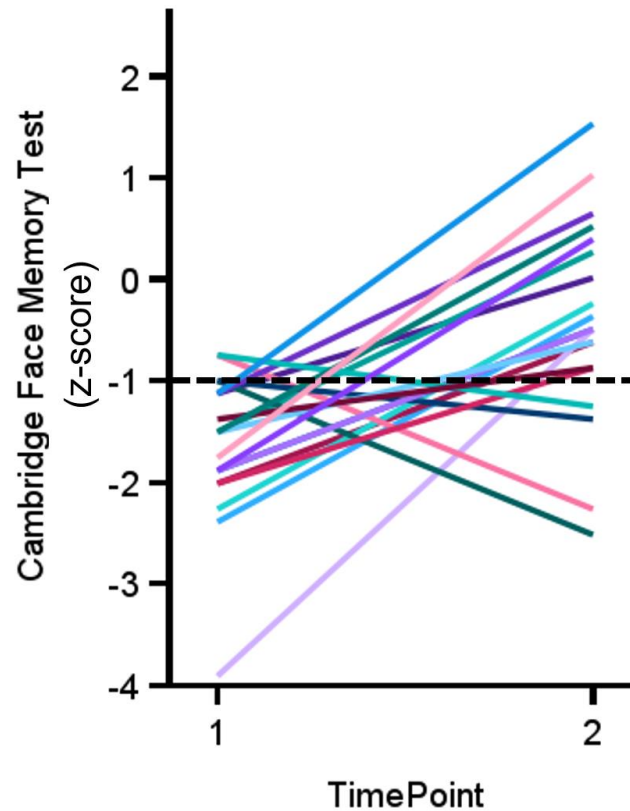
HEADER: DSM-5 LIMITATIONS



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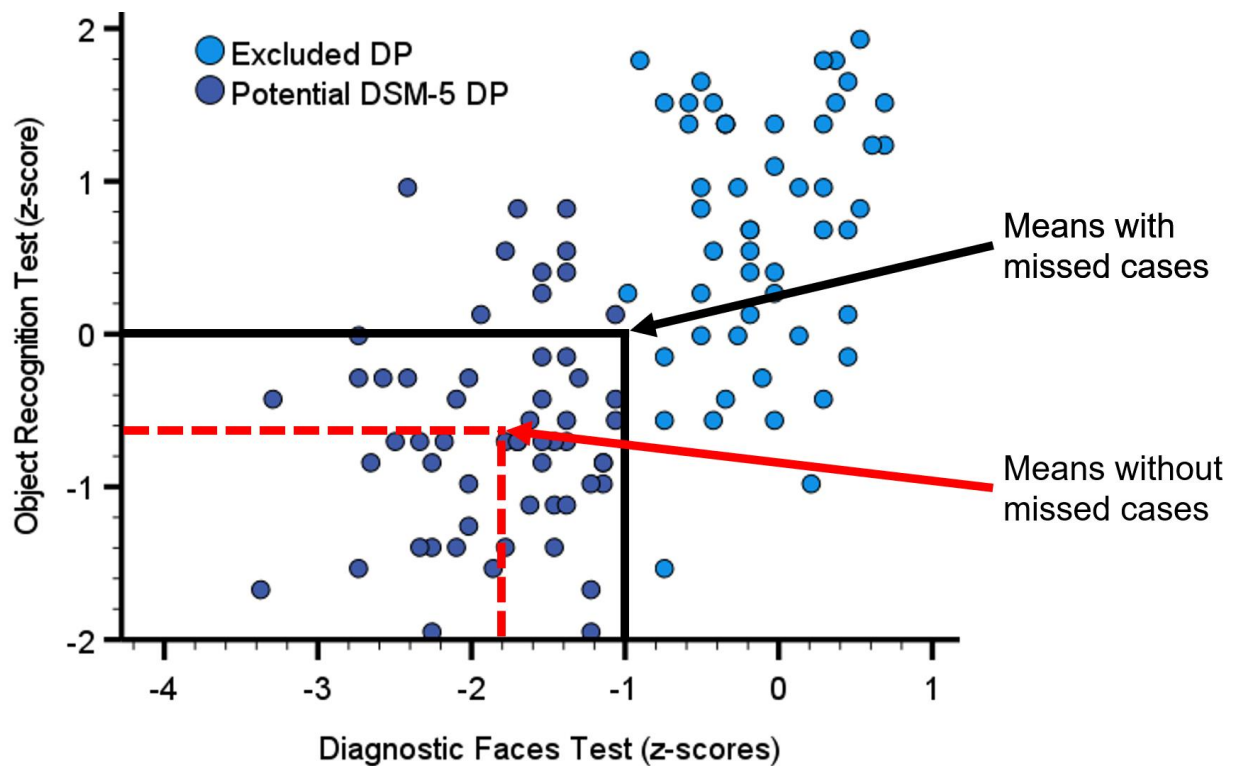
Figure 1. The top left and right panels demonstrate the -1 SD cutoff removes the top end (light blue) of the DP FFT performance distribution, with 15% (top left: Bate et al., 2019) and 30% (top right: Burns et al., 2022) failing to meet criteria. In the bottom left panel, using Bate et al. (2019) data, the -1 SD cutoff on FFT (x-axis) has power to detect impairment in 85% of cases, while CFMT (y-axis) has 79% power. Requiring deficits on both means power can never be greater than the weakest of the two, with only 70% of self-identified DP cases (dark blue) meeting DSM-5 criteria and 30% excluded (light blue). In the bottom right panel, we present a hypothetical famous faces test that has perfect sensitivity, i.e., 100% power at the -1 SD level. Unfortunately, as we require impairment on a second imperfect test that has 79% power (i.e., the CFMT), we will only ever diagnose at this rate. This is despite some of the DP cases scoring almost -9 SDs on the FFT. Please note, I simply subtracted -2 from the Bate et al. (2019) famous faces test data to create this hypothetical data.



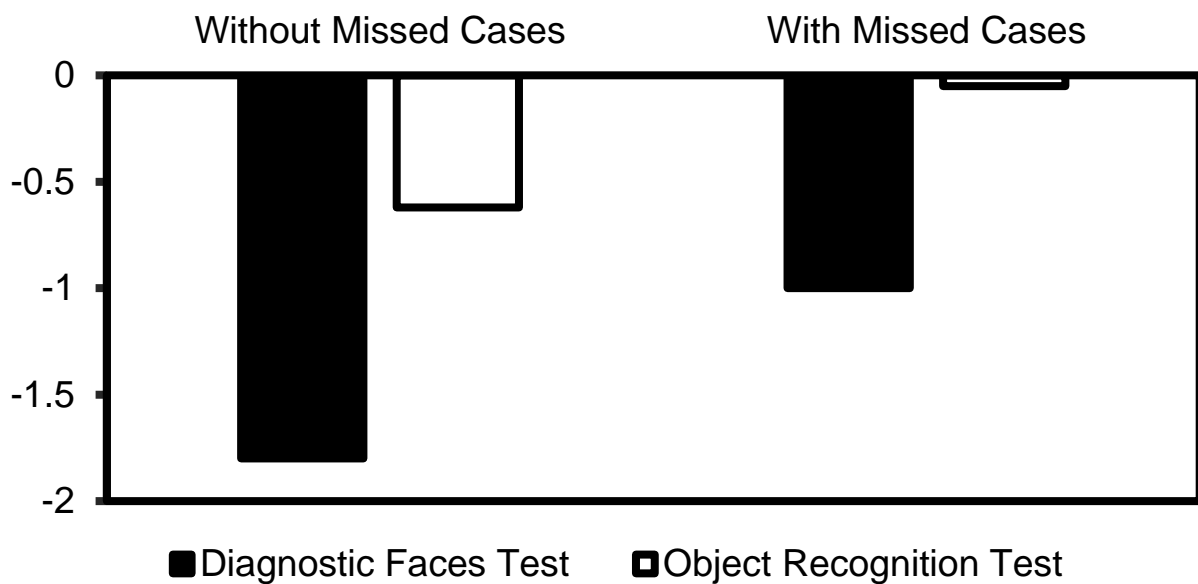
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Figure 2. Top left panel illustrates how imperfect CFMT test-retest reliabilities will result in changes to potential diagnostic status (black dashed line represents -1 SD below neurotypical mean) from the first assessment timepoint to the second in 20 DP cases (Murray & Bate, 2020). Top right panel demonstrates how a perfect correlation ($r = +1$) between our two hypothetical diagnostic tests will result in only the most severely impaired FFT DP cases receiving a diagnosis. In the bottom right panel, where there is no correlation ($r \approx 0$), the diagnosed cases will be sampled throughout the FFT distribution. Thus, the strength of the relationship between the two cognitive tests will produce unique biases in the types of cases we can diagnose and study. Please note, the FFT has 100% power and the second test roughly 50% in both plots on the right.

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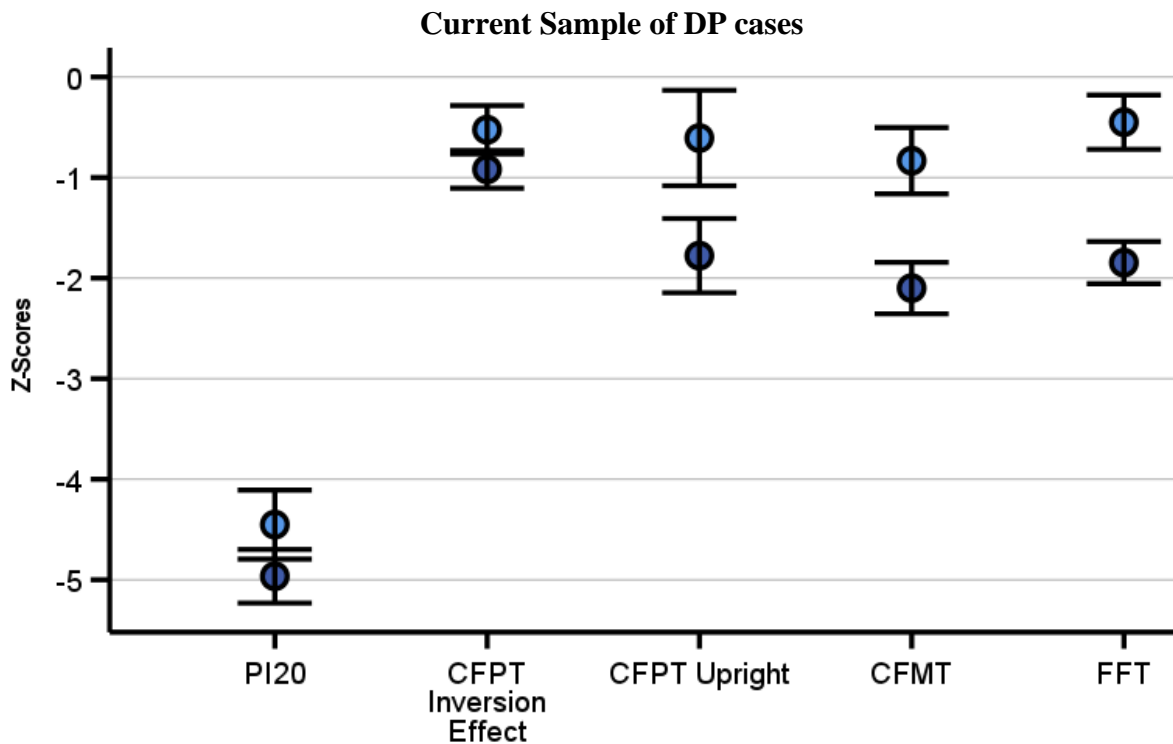
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4 **Figure 3.** In the top panel, we present a hypothetical diagnostic faces test (y-axis) that is
 5 correlated with a hypothetical object recognition test (x-axis). The -1 SD cutoff excludes
 6 roughly 50% of potential DP cases on this single test (light blue circles). When we plot the
 7 mean z-scores in the bottom panel, you can see that without the missed cases, DP is
 8 associated with face *and* object recognition impairments. This supports a cognitive model
 of shared underlying processes for the two abilities. However, when we include the DP
 cases who have been excluded from a potential DSM-5 diagnosis due to the -1 SD cutoff,
 we find estimates of face recognition impairments in this group becoming milder, and
 object recognition deficits disappear. We now create a cognitive model that dissociates
 these two abilities. These issues will also be apparent in neurocognitive models if we use
 replace object recognition with neuroimaging measures, such as an fMRI BOLD response.
 Please note, I only used one diagnostic test here. These problems will likely become more
 pronounced with the introduction of a second diagnostic test as the DSM-5 requires.

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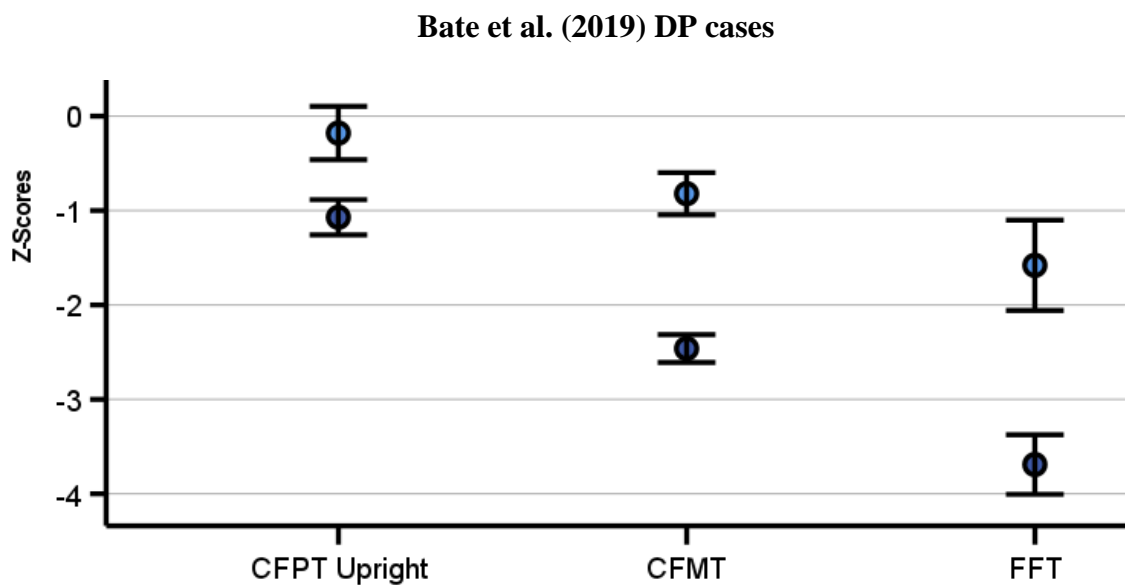
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Figure 4. DSM-5 missed DP cases are objectively impaired on multiple cognitive measures. In the top panel, DP cases excluded from a DSM-5 diagnosis (light blue circles) exhibited highly atypical prosopagnosia symptoms (i.e., PI20) and objective impairments on every cognitive task related measure. These deficits validate their subjective complaints. Please note, the DSM-5 DP group’s mean z-scores are plotted for context (dark blue): all were significantly milder on all measures [all $ps < .022$]. In the bottom panel, the excluded DP cases from Bate et al. (2019) were impaired on the CFMT and FFT. While the CFPT upright z-scores were not significantly different from controls, they were in the same direction (i.e., impaired) as my DP sample. Please note, Bate et al. (2019) FFT impairments were likely much larger than my sample because the former controlled for participants’ familiarity with the faces, while I did not.

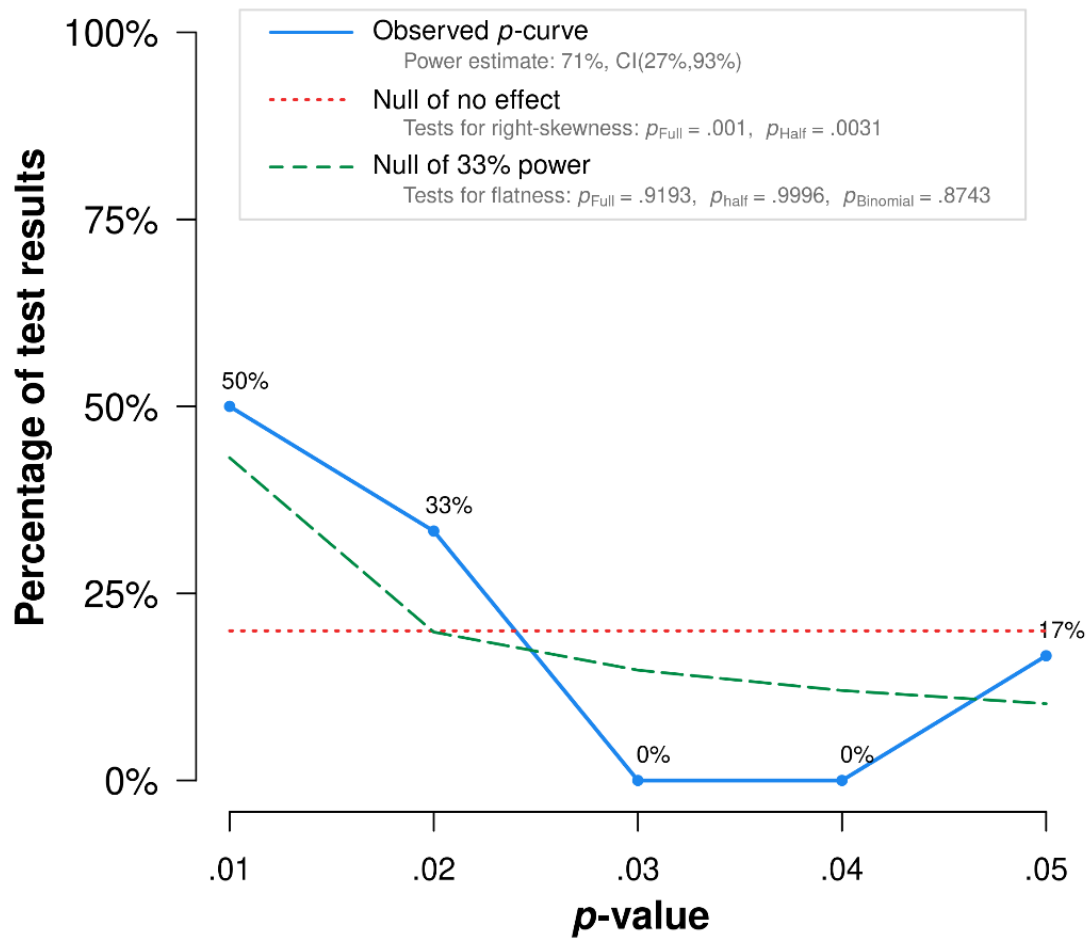
HEADER: DSM-5 LIMITATIONS

DSM-5	Current Sample	Bate et al.
Missed Cases		(2019)
% of Total DPs	38%	30%
CFPT Upright	-.6* (.85)	-.18 (.91)
CFPT Holistic	-.52* (.52)	N/A
CFMT	-.83* (.86)	-.82* (.94)
FFT	-.45* (.77)	-1.58* (1.91)
PI20	-4.45* (.91)	N/A

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Table 1. Percentage of DSM-5 missed DP cases (top row) and their mean impairment z-scores from the current sample and Bate et al. (2019). While my DSM-5 missed cases were impaired on all measures, Bate et al.'s (2019) cases were only impaired on CFMT and FFT. Please note, Bate et al. (2019) DPs did not complete the PI20 or inverted portion of the CFPT. Asterisks indicate significant impairments relative to neurotypical controls at <.05, SDs are in brackets.

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3 **Figure 5. Meta-analyses support cognitive impairments in excluded DP cases.** All *p*-
 4 values in support of missed cases' objective cognitive impairments from my sample and Bate
 5 et al. (2019) exhibit a right-skew distribution, with the *p*-curve analysis statistically
 6 significant. This means that missed cases' complaints are validated by objective impairments
 7 in face processing.

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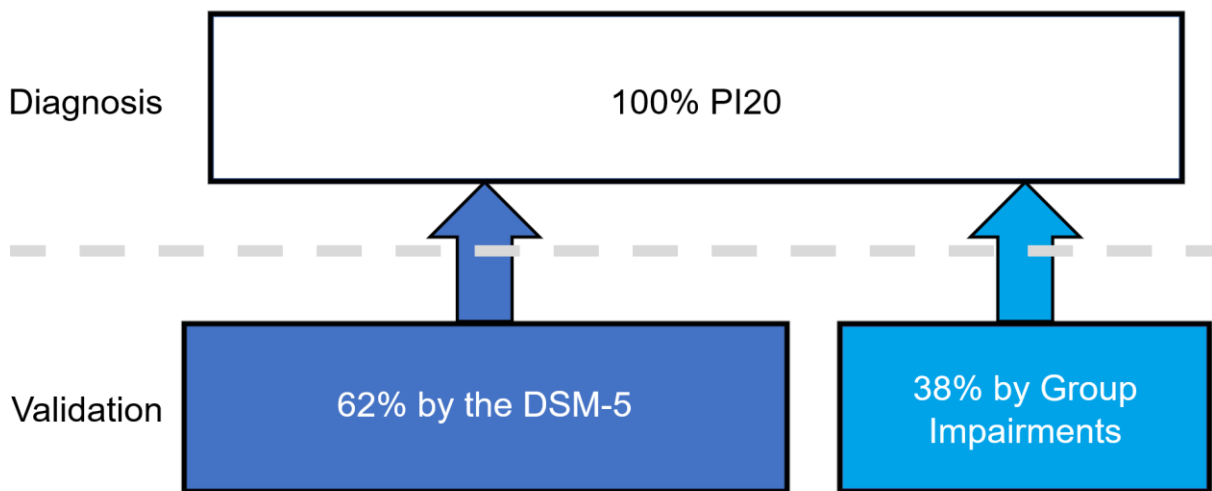
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2 **Figure 6. How the symptom-based approach to diagnosing is validated by objective**
3 **impairments in DP.** The PI20 diagnoses 100% of DP cases as atypical in their symptoms
4 (top). Sixty-two percent of these self-identified DP cases were validated as objectively
5 impaired at the -1 SD level on the FFT and CFMT, with the remaining 38% validated through
6 deficits at the group level in their FFT, CFMT, CFPT upright and Holistic Perception
7 measures. Thus, when a DP case reports atypical symptoms via the prosopagnosia index
8 questionnaire, we can be confident that they reflect underlying cognitive impairments.
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