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The importance of involving experts-by-experience with different psychiatric diagnoses when revising diagnostic criteria

Abstract

Philosophers of science have recently called for experts-by-experience to be involved in revising psychiatric diagnoses. They argue that experts-by-experience can have relevant knowledge which is important for considering potential modifications to psychiatric diagnoses. I show how altering one diagnosis can impact individuals with a different diagnosis. For example, altering autism can impact individuals diagnosed with Attention Deficit Hyperactivity Disorder and Schizoid Personality Disorder through co-morbidity and differential diagnostic criteria. Altering autism can impact the population making up the diagnosis of Attention Deficit Hyperactivity Disorder and Schizoid Personality Disorder which can then influence which behaviour each diagnosis covers and which parts of the causal structure each diagnosis covers. Expanding one diagnosis can mean, if differential diagnostic criteria are present, that individuals who have a different diagnosis, or would one day have been diagnosed with that different diagnosis, are instead now diagnosed with the expanded diagnosis. Alternatively, if two diagnoses can be co-morbid then expanding one diagnosis can mean individuals who have a different diagnosis can now also receive the expanded diagnosis. Changing these can then impact the adequacy of symptoms formulated to cover behaviour and causal mechanisms formulated to cover the causal structure. This means experts-by-experience representing a range of diagnoses should be involved in decisions over modifying diagnoses they do not have. For example, Attention Deficit Hyperactivity Disorder individuals should be involved in decisions to modify autism. I discuss practical issues relating to how much say people with a range of diagnoses should have in relation to modifying a diagnosis which they do not have. We should employ a principle of equality whereby the level of say individual with one diagnosis should have on another diagnosis should be equally reciprocated.

Keywords

Experts-by-experience; revising diagnostic criteria; participatory research; psychiatric diagnoses, collaborative research

Competing interests

Competing interests: The author(s) declare none

1. Introduction

Individuals with psychiatric diagnoses and scholars in disability studies have a long history of calling for experts-by-experience to be involved in psychiatric research. Recently, philosophers of science have called for experts-by-experience to be involved in revising diagnostic manuals (Bueter 2019; Bueter 2021; Tekin 2022). They argue that experts-by-experience can have relevant knowledge which is important for considering potential modifications to psychiatric diagnoses. Whilst I agree to this, I will outline an important consequence of their position. I argue that this means we need to include individuals representing a wide range of diagnoses even when only considering modifying a single diagnosis. For example, when considering modifying the diagnosis of Autism Spectrum Conditions (autism) we do not just need to involve individuals diagnosed with autism but we need also include other individuals who have diagnoses like Attention Deficit Hyperactivity Disorder and Schizoid Personality Disorder. This is because modifying one diagnosis can impact people with a different diagnosis.

Modifying the diagnostic criteria can lead to an expansion or a contraction. An expansion is where there are more ways to meet the diagnostic criteria through adding a symptom or lowering the diagnostic threshold. A contraction is where there are less ways to meet the diagnostic criteria through removing a symptom or increasing the diagnostic threshold. Expansions and contractions can then impact people with other diagnoses. An individual diagnosed with Schizoid Personality Disorder (SPD) or Attention Deficit Hyperactivity Disorder (ADHD) might exhibit some symptoms on the diagnostic criteria for autism but not enough to meet the diagnostic criteria for autism. If autism expands through changing the diagnostic threshold for autism or adding symptoms to autism could mean that an individual diagnosed with SPD or ADHD who previously did not meet the diagnostic criteria for autism might now do so. Similarly, if the diagnostic criteria of autism contracts through removing a symptom or increasing the diagnostic threshold then someone who currently meets the diagnostic criteria of autism and SPD or ADHD might now only meet the diagnostic criteria of SPD or ADHD.

This can change the population which makes up the diagnosis of SPD or ADHD (as I will outline, this works in different ways depending upon the presence of differential diagnostic criteria). Changing populations can then influence which behaviour each diagnosis covers and which parts of the causal structure each diagnosis covers. Changing these can then impact the adequacy of symptoms formulated to cover behaviour and causal mechanisms formulated to cover the causal structure. I outline how changing populations can cause redundancies to arise. A redundancy is where a symptom or causal mechanism previously covered something relevant but no longer does so after the change in population. Changing populations can also cause inadequate coverage to arise. Inadequate coverage is where there was no need to cover something but after the population changes new symptoms or causal mechanisms are needed to adequately cover it. This is not only an epistemic issue but can also have important practical and ethical consequences.

Since modifying one diagnosis can impact individuals who have other diagnoses, I will argue that individuals representing a range of diagnoses should be included in decisions to modify a single diagnosis. I consider multiple models incorporating individuals with a range of psychiatric diagnoses, suggesting that both consultation and collaboration models should be employed. I also outline various practical questions about exactly how much say people with one diagnosis should have in modifying a different diagnosis. I suggest there is an irreducible practical element to these questions and reasonable individuals are likely to hold divergent views. However, I suggest that there should be a principle of equality whereby the degree of involvement of, say, autistic individuals in modifying schizophrenia should be the same as the degree of involvement that schizophrenic individuals have in modifying autism.

In this article I focus upon psychiatric diagnoses rather than somatic diagnoses. Revising psychiatric diagnoses is much more controversial than revising somatic diagnoses and there is a

much greater call for experts-by-experience in relation to revising psychiatric diagnoses than experts-by-experience in revising somatic medicine. The reason why there is a much greater call is likely because the causal basis of psychiatric diagnoses is far worse understood than diagnoses in somatic medicine, giving greater room for value decisions. Also, I believe that we should consider how altering any particular psychiatric diagnosis can affect any other psychiatric diagnosis. All psychiatric diagnoses should be considered. However, as I outline in relation to Opioid Use Disorder, there could be case by case exceptions though this would require specific bioethical arguments.

2. Involving Experts-By-Experience in Psychiatric Research

An expert-by-experience is someone who can draw upon their own experience to generate relevant knowledge and has relevant input upon value questions. The basic idea of involving experts-by-experience is that they have relevant knowledge which other people lack and those other people cannot gain that knowledge except through directly or indirectly engaging with experts-by-experience. Also, they are interested parties because they can be impacted by many aspects of psychiatry. This knowledge comes from their lived experience (Bueter 2019, 1071; Friesen et al 2019, 55; Johnson, Barrett & Sisti 2103, 340; Stein & Philips 2013, 2; Tekin 2022, 1170). For example, an autistic individual has a particular lived experience of having various symptoms, the process of being diagnosed as well as potentially accessing support services, facing discrimination and taking medication. Individuals with lived experience can draw upon that lived experience when considering what issues, they face and what solutions might alleviate those issues. This helps establish what should be studied. They can also draw upon lived experience when considering what data is relevant to the question, how best to obtain that data and how to interpret it. This means that experts-by-experience should not merely be restricted to answering questions posed by non-diagnosed academics but instead should be involved in setting the question, designing the study and interpreting the results.

There are multiple areas where experts-by-experience in psychiatry can contribute to psychiatric or psychological research (for discussion see Bueter 2021; Johnson, Barrett & Sisti 2013; Tekin 2022). Areas include improving services, therapies and medication, and critiquing and developing theoretical claims. This article does not comment upon any of these areas and instead focuses on the role of experts-by-experience in revising psychiatric diagnoses.

To set up this discussion I shall outline how diagnostic criteria for psychiatric diagnoses work. Most countries employ the DSM (Diagnostic and Statistical Manual) or ICD (International Classification of Disease). Both diagnostic manuals have near identical psychiatric content (the ICD, unlike the DSM, also covers somatic diagnoses). Each diagnostic manual contains hundreds of psychiatric diagnoses. All those diagnoses have diagnostic criteria which are rules for deciding who should receive the diagnosis. This consists of a list or multiple lists of symptoms plus rules about how many symptoms are needed from that list or multiple lists. DSM and ICD psychiatric diagnoses are categorical whereby an individual either meets or fails to meet the criteria for the psychiatric diagnoses. An individual either exhibits equal to or more than the number of symptoms which the diagnostic criteria specify as required or they exhibit less than that number of symptoms. When following the DSM or ICD an individual who passes that threshold of number of symptoms on the diagnostic criteria should receive the diagnosis whilst an individual who does not pass the threshold should not receive the diagnosis.

I now outline Bueter's and Tekin's arguments for experts-by-experience being involved in setting diagnostic criteria. Bueter notes that decisions are needed when reformulating psychiatric diagnoses. The immense complexity of underlying causes and our highly limited understanding of their interactions leads to very high levels of underdetermination. Decisions involving value judgements which “exceed the available data” (Bueter 2019, 1067) will be required. Those

judgements can be enhanced by incorporating experts-by-experience. Bueter writes that "[p]atients' input is undervalued because they do not qualify as scientific experts, even though many of the questions at hand are either irreducible to empirical evidence or can profit from additional first person knowledge" (2019, p.1071). Bueter argues experts-by-experience can draw upon first person knowledge to inform on potential modifications to diagnostic criteria and it is wrong to ignore that first person knowledge. Similarly, Tekin outlines how experts-by-experience should be involved in revising diagnostic criteria, arguing that participation is "necessary for objectivity in psychiatry" (Tekin 2022). Tekin argues that lived experience has an objective element to it. She develops a notion named Participatory Interactive Objectivity to argue objectivity will be increased by drawing upon lived experience when revising diagnostic criteria.

Bueter draws upon epistemic injustice (2019, p.1071) and Tekin draws upon feminist epistemology (2022, p.1167) to provide theoretical foundations for their positions. There is a clear epistemic component whereby lived experience is taken as providing some type of knowledge that is relevant for questions of modifying psychiatric diagnoses. There is also a value component whereby people with a condition can provide input upon the most practically or ethically beneficial way to formulate their diagnosis. Bueter and Tekin mention multiple areas where value decisions are required, such as medicalisation (Bueter 2019, p.1069), the reaction of others to their illness (Tekin 2022, p.1173), self-conception (Tekin 2022, p.1173) and relationship to resources that can produce good quality of life (Tekin 2022, p.1173).[1]

Bueter outlines multiple areas where experts-by-experience should be involved in revising diagnostic criteria. She argues that experts-by-experience can provide input on "setting *diagnostic thresholds*" (2019, p.1069 emphasis original). Setting diagnostic thresholds relates to how many symptoms on the diagnostic criteria should be required to receive a diagnosis. Additionally, she argues for input on "*the accuracy of diagnostic criteria*" (2019, p.1070 emphasis original). Accuracy relates to whether there are symptoms which should not be on the diagnostic criteria but which are currently present. Finally, she argues for input on the "*incompleteness* of diagnostic criteria" (2019, p.1070 emphasis original). Incompleteness of diagnostic criteria relates to whether there are symptoms which should be on the diagnostic criteria but which are currently absent.

I believe that Bueter and Tekin are correct but I shall not provide arguments to support their positions. Rather, I assume Bueter and Tekin are correct and then show how their arguments lead to an important practical consequence which they do not discuss.

All these areas relate to expanding or contracting psychiatric diagnoses. Expansion is where there is an increase in the number of combination of symptoms which meet the diagnostic criteria. This can occur through adding a symptom to the diagnostic criteria or lowering the diagnostic threshold (the number of symptoms required to receive the diagnosis). A diagnosis which needed five of nine symptoms to meet the diagnostic threshold would now need five of ten symptoms if a symptom was added, or would only need four of nine symptoms if the diagnostic threshold was lowered. Imagine an individual exhibited four of the symptoms listed on a diagnostic criteria but five symptoms are needed to receive that diagnosis. If a new symptom was added to the diagnostic criteria which the individual exhibited then they would now qualify for the diagnosis. Alternatively, if the diagnostic threshold was lowered, whereby only four symptoms rather than five are required, then the individual would now meet the diagnostic criteria. A contraction is where there is a decrease in the number of combinations of symptoms which meet the diagnostic criteria. Removing a symptom or increasing the diagnostic threshold does this. A diagnostic criteria requiring five of nine symptoms would now need five of eight symptoms if a symptom was removed, or would need six of nine symptoms if the diagnostic threshold was increased. Imagine an individual exhibited five

1 Drawing a line between the epistemic and values could be problematic. Philosophers of science disagree about whether epistemic values and non-epistemic values are separate or interlinked, and in what places it is legitimate to use each type. It is not clear to me where Bueter and Tekin draw this line. I believe that all points of my discussion will be relevant reguardless of where the line is drawn, though others will disagree about how I categorise these points as epistemic or as values.

symptoms of the diagnostic criteria and the diagnostic threshold is five symptoms. If a symptom was removed from the diagnostic criteria and the individual exhibited that symptom then they would only exhibit four of the required five symptoms so no longer meet the diagnostic criteria. Alternatively, if the diagnostic threshold was increased to six symptoms then they would no longer meet the diagnostic criteria.

I now explore a consequence not outlined by Bueter or Tekin. Expanding or contracting a psychiatric diagnosis can impact individuals with other diagnoses. For example, expanding autism can mean someone who has another diagnosis may now also meet the diagnostic criteria for autism. Contracting autism can mean an individual who is diagnosed with autism and another diagnosis might no longer meet the diagnostic criteria for autism and now only meets the diagnostic criteria for the other diagnosis. If differential diagnostic criteria are present, some people who previously met the diagnostic criteria for one diagnosis now meet the diagnostic criteria of autism whilst a contraction of autism can mean people who previously fit the diagnostic criteria of autism now instead meet the diagnostic criteria of that other diagnosis. Similarly, if differential diagnostic criteria are not present, expanding autism can mean people who previously just had a diagnosis of ADHD are now co-morbid with autism, whilst contracting autism can mean people who are co-morbid with another diagnosis and autism now only fit that other diagnosis.

Modifying one diagnosis can leave symptoms and causal mechanisms of a different diagnosis having a redundant element or leave them providing inadequate coverage. I shall refer to this terminology of redundancy and inadequate coverage in what follows. Redundancy is where an aspect of a symptom or causal mechanism did describe something relevant but no longer does so. Before a redundancy occurs, the population covered by a psychiatric diagnosis exhibited a set of behaviour that was adequately covered by a symptom or covers an area of the causal structure which was adequately covered by a causal mechanism. After the population changes, the population exhibits a different set of behaviour which no longer needs covering by that symptom or covers a different area of causal structure which no longer needs covering by the causal mechanism. As such, the symptom or causal mechanism is redundant. Inadequate coverage is where there was no need for a particular symptom or causal mechanism to cover a set of behaviour or area of the causal structure but now that behaviour or area of the causal structure needs be covered by that symptom or causal mechanism. Before inadequate coverage occurs, the population exhibits a set of behaviour which is being adequately covered by a symptom or covers an area of the causal structure which is adequately covered by a causal mechanism. After the population changes there is a change in the behaviour exhibited which then means existing symptoms inadequately cover that behaviour or a different area of the causal structure is covered which means currently formulated causal mechanism are inadequate. Redundancies and inadequate coverage can also result in negative practical and ethical consequences. I will outline these after I spend the bulk of this paper showing how redundancies and inadequate coverage can arise.[2]

In what follows I shall only focus upon expanding rather than contracting. I only have space to focus upon one of these because there are multiple implications to expansion which I highlight with six detailed examples. I will highlight where contractions occur but I do not have sufficient space to discuss whether they generate the exact same problems as expansions.

3. Overlapping Psychiatric Diagnoses

There are very strong overlaps between different psychiatric diagnoses (Hyman 2010, p.167; Kirk

2 Note that I am not discussing Hacking's looping effect (Hacking 1995, p.370). Hacking argues that people can change their behaviour after being diagnosed because they have a new self-understanding. As such, if expanding or contracting one diagnosis impacts another diagnosis then people with the impacted diagnosis may have a different self-understanding which results in different behaviour. In this paper I will show how modifying one diagnosis can impact another diagnosis even if the behaviour of individuals covered by the impacted diagnosis remains identical.

Cohen & Gomery 2015, p.69). Two different psychiatric diagnoses can share one or multiple symptoms in common. Also, two different diagnoses can have different symptoms but those symptoms have significant overlapping elements whereby both symptoms manifest in ways with significant similarities.

The manner in which changes to one psychiatric diagnosis can impact individuals with other diagnoses works differently depending upon whether there are differential diagnostic criteria or not. These are rules which specify which diagnosis an individual should receive when they meet the diagnostic criteria for two particular diagnoses which means an individual who meets the diagnostic criteria for both diagnoses only receives one of those diagnoses. The diagnostic manual specifies which diagnosis they receive, such as how an individual who meets the diagnostic criteria for autism and SPD only receives the diagnosis of autism. Alternatively, two diagnoses can be co-morbid, meaning an individual who meets the diagnostic criteria for both diagnoses receives both diagnoses.

To provide strong and detailed empirical examples I will show how modifications to autism can impact SPD, which has differential diagnostic criteria with autism, meaning only autism should be diagnosed if an individual meets both diagnoses. I will then later show how modifications to autism can impact ADHD, which can be co-morbid with autism, meaning one individual can receive both diagnoses. Note that I pick these examples to highlight how differential diagnostic criteria and co-morbidity work; whether these particular diagnoses should have a relationship of differential diagnostic criteria or co-morbidity is not a question I comment upon. Neither do I discuss whether the general principle of differential diagnostic criteria is justified: I discuss it since it is present in the DSM and because I am aware of no empirical data showing the degree that experts-by-experience support or object to different diagnostic criteria, whether in general or in relation to specific diagnoses.

4. Differential Diagnostic Criteria

To highlight differential diagnostic criteria I shall draw upon autism and schizoid personality disorder (SPD). In relation to autism, the DSM portrays autism as having "[p]ersistent deficits in social communication and social interaction... [and] [r]estricted, repetitive patterns of behaviour, interests, or activities" (APA 2013, p.50). In relation to SPD, the DSM portrays SPD as "a pervasive pattern of detachment from social relationships and a restricted range of expressions of emotions in interpersonal settings" (APA 2013, p.655). According to the DSM, autism and SPD are demarcated by autism having more pronounced social abnormalities and more stereotyped behaviour (APA 2013, p.655). However, there are differential diagnostic criteria between each diagnosis. The DSM and ICD specify that if an individual meets the diagnostic criteria of both diagnoses then they should be diagnosed as autistic and should not be diagnosed as SPD (APA 2013, p.59). The justification for this is not stated in the DSM. I will investigate the consequences of differential diagnostic criteria because it is present in the DSM and because I have no data upon what proportion of autistic individuals or SPD individuals support or object to the presence of this differential diagnostic criteria. Having outlined the relationship between autism and SPD, I now show how modifying the diagnostic criteria for autism can alter who is eligible to receive a diagnosis of SPD.

Autism can be expanded (more ways to meet the diagnostic criteria) whereby the diagnostic threshold is lowered or symptoms are added to the diagnostic criteria. Expanding autism could mean that an individual who is currently diagnosed as SPD goes from not meeting the diagnostic criteria for autism to meeting the diagnostic criteria for autism. Imagine an SPD individual is one symptom away from qualifying for the diagnosis of autism. If the number of symptoms required for the diagnosis of autism was reduced by one then they would now meet the diagnostic criteria for

autism. Alternatively, imagine a symptom was added to the diagnostic criteria of autism and the SPD individual exhibits that symptom then they would also now meet the diagnostic criteria of autism. In either case the individual now meets the diagnostic criteria for autism and SPD. However, when differential diagnostic criteria are applied the individual should be diagnosed as autistic and not as SPD. Therefore, if we followed the DSM and ICD, then this individual should be re-diagnosed as autistic and should no longer be considered to be diagnosed with SPD. Some individuals may resist re-diagnosis, still considering themselves to be instances of SPD and would consider themselves not to be autistic. However, regardless of what currently diagnosed individuals do, future undiagnosed individuals who would effectively be in this situation would now be diagnosed as autistic. A currently undiagnosed individual who has not yet encountered a psychiatrist and exhibits all the same symptoms as an individual who resists re-diagnosis would be simply diagnosed as autistic.

This means that individuals who are diagnosed with SPD and future individuals who would be diagnosed with SPD under current approaches can be affected by decisions to modify autism. I will outline two different areas in which it can have an effect. These points relate to SPD individuals who still meet the criteria for SPD and who do not meet the diagnostic criteria for autism after autism has been expanded. These would be the remaining SPD individuals who still meet the diagnostic criteria for SPD, in contrast to SPD individuals who are re-diagnosed as autistic and future individuals who would be diagnosed with autism rather than SPD.

Firstly, by altering which individuals make-up the population of people diagnosed with SPD, the statistical frequency of any particular behaviour exhibited by the total population of SPD changes. To make this argument I follow the framework outlined by Fellowes (2021) by drawing upon the distinction between *behaviour* and *symptoms*. A behaviour is the specific activity someone exhibits at a particular moment. A symptom is a generalised description that abstracts away from particular individuals and their contexts. For example, a behaviour would be a particular person feeling a particular uncomfortable sensation due to a particular light source. In contrast, the symptom of sensory issues covers many different people feeling different levels of uncomfortable sensation due to a wide variety of sounds, smells, textures and lights. For example, any particular individual diagnosed with SPD will spend an amount of time per day engaging in solitary hobbies. We could combine the time spent by every SPD individual to produce an average. If the population of SPD changes then the average level will almost certainly change (be that new average higher or lower by a millisecond or multiple hours per day).

Changing the frequency of behaviour may impact symptoms. Symptoms can be constructed from behaviour in multiple ways. Relevant factors for constructing symptoms include balancing factors like making symptoms sufficiently general, making symptoms sufficiently accurate and limiting the causal factors which the symptom covers (Fellowes 2021, p.4510-4512). One relevant consideration is the frequency of the behaviour. For example, if a behaviour is considered statistically significant within multiple high quality studies then there may be good reason to ensure that a symptom covers that behaviour. If, however, the population we consider to make-up SPD changes then the frequency of the behaviour changes. If changing the population sufficiently reduced the frequency of the behaviour then it might be considered too infrequent to be worth covering with a symptom.

As a potential example, the relationship between desiring to socialise and autism is not very well conveyed by the diagnostic criteria of autism. The DSM-5 diagnostic criteria mention “absence of interest in peers” (APA 2013, p.50). This effectively says there is a lack of desire for socialising with peers but it does not demarcate between two different types of desires. Firstly, an autistic individual might have no interest in socialising no matter the quality of potential social interactions. Secondly, an autistic individual might in principle desire to socialise but shows no interest in socialising with anyone they know because they find all of them boring or all those people are hostile to the autistic individual. The autistic individual does, however, desire to socialise with

people who would provide high quality social interactions. Most autistic individuals seem to fall into the latter group so there might be good reason to reformulate the symptom as 'absence of interest in peers *despite in principle desiring high quality social relationships*'. However, a minority of autistic individuals appear to fall into the former group of actually in principle having no desire to socialise (Chevallier et al 2012, p.1508). So there might also be good reason to add the symptom 'absence of interest in peers *because of an in principle lack of desire to socialise, regardless of who they socialise with'*. These two changes would make the diagnostic criteria more precise, being clear about how autism can manifest in two quite different ways that is not covered by simply stating there can be an "absence of interest in peers" (APA 2013, p.50).

However, making these modifications would have significant impact upon SPD individuals. One symptom of SPD is "[n]either desires nor enjoys close relationships, including being part of a family" (APA 2013, p.653). This appears to be a common aspect of SPD whereby most SPD individuals genuinely do not desire to socialise. As such, by adding the symptom 'absence of interest in peers *because of an in principle lack of desire to socialise, regardless of who they socialise with'* to autism would mean significantly more individuals who currently meet the diagnostic criteria for SPD would now meet the diagnostic criteria for autism. Given differential diagnostic criteria, some of them would be rediagnosed as autistic and future individuals who are currently undiagnosed would now be diagnosed as autistic. Having changed the population of SPD a particular behaviour of SPD now becomes more frequent within the population of SPD. Under current diagnostic approaches some SPD individuals do genuinely desire to socialise, they have typical levels of social intuition, but they get very little feeling from socialising. This is analogous to how they can get very little feeling from sexual activity despite having sexual desires (APA, 2013, p.653). Within the remaining population of SPD after autism has been expanded a greater proportion of SPD individuals would have a desire to socialise but cannot obtain any satisfaction from socialising due to a lack of feeling. This creates a situation of inadequate coverage whereby there is now stronger reason for this behaviour to be covered by a specific symptom of SPD. Modifying autism changes the population of SPD, which then changes the frequency of particular behaviours in SPD, which then means current symptoms of SPD start providing inadequate coverage.

Secondly, changing the population of SPD then influences how SPD correlates with causes. Psychiatric diagnoses typically only have a very loose connection with causes. Individuals who receive the same diagnosis will typically have significantly diverse causes (in relation to autism see Petrolini & Vicente 2022, p.11; Weiskopf 2017, p.178). Additionally, most causes associated with a particular diagnosis are typically present in individuals who do not meet the diagnostic criteria for that diagnosis (Cuthbert & Insel, 2013, p.3; Kendler 2010, p.1291). The fit between causes and diagnoses is one of loose statistical correlation. Those statistical correlations will change if the population which makes up the diagnosis changes. As such, if autism is expanded in a manner which means some SPD individuals were rediagnosed or future currently undiagnosed individuals were now diagnosed with autism rather than SPD then the population of SPD would change. This would then alter the correlations between individuals making up the new population of SPD and any causal factor.

Tekin and Bueter do not mention that experts-by-experience should provide input upon areas of causation. However, it seems that we need value decisions when formulating psychiatric diagnoses and some of these relate to causation (Kendler, Zachar & Craver 2011, p.1149; Poland 2014, p.35). For example, since the statistical correlation between a diagnosis and a causal factor varies dependent upon the population making up the diagnosis the diagnosis could be split into multiple narrower diagnoses which would likely increase the statistical correlation between some of those causal factors and each new diagnosis. It seems plausible that diagnosed individuals should be involved in deciding which type of correlation is desirable.

I will highlight this by drawing upon the distinction between the *causal structure* of the world and *causal mechanisms* (Boone & Puccini 2016, p.696; Overton 2011, p.943). The causal

structure is what occurs causally at any given moment, covering every single causal interaction taking place. In contrast causal mechanisms are abstracted away from the causal structure, removing much of the details to create generalised claims. For example, the causal mechanism of a hippocampus is a part of the brain that allows navigations of an environment. However, the hippocampus can take many different forms, such as being present in a mouse, a human and a whale. Additionally, any two humans will have some variance in brain structures and will encounter at least slightly different environments throughout their life. Thus all the specific causal happening in any one person relates to the causal structure whereas a causal mechanism is an idealisation which misses out on some of the specific details of the causal structure. One way to understand this distinction between the causal structure and causal mechanisms is that of tokens and types. An area of the causal structure is a token whilst the causal mechanism is an idealised type which misses some details present in tokens (Boone & Puccinini 2016, p.693; Overton 2011, p.947).

Changing the population of SPD then also changes the frequency that any part of the causal structure occurs within the population of SPD. This works in an analogous manner to how changing the population of SPD changes the frequency of the behaviour exhibited by SPD individuals. For example, any particular gene will undergo a specific interaction with the environment a particular number of times within the population of SPD. Changing the population of SPD changes the exact number of times that specific interaction occurs. Since causal mechanisms abstract away parts of the causal structure there needs be a decision over what parts of the causal structure should be associated with the mechanism and which should be abstracted away. This works in an analogous manner to how decisions are needed over which behaviour a symptom should cover and which behaviour should be abstracted away. Darden outlines twelve relevant constraints in formulating mechanisms, including four temporal constraints of order, rate, duration and frequency (2002, p.358). Potentially, any of these twelve constraints may be relevant if a population changes but I shall focus here upon statistical frequency. If covering statistically frequent parts of the causal structure is one basis for formulating a mechanism then the mechanism itself might need changing as a population changes. Changing the population of SPD changes the area of the causal structure covered by SPD. This then changes the frequency of any given part of the causal structure. One part of the causal structure might have a high level of frequency in the population of SPD before autism expands and then might have a lower level of frequency after autism expands. Aspects of the mechanism might no longer cover areas of the causal structure which occur with sufficient statistical regularity. As such, a causal mechanism associated with SPD might cover all relevant parts of the causal structure before autism expands but now covers an area which is no longer relevant after autism expands. The causal mechanism associated with SPD now has a redundancy.

To provide a potential example, both autism and SPD are considered to have the causal mechanism of theory of mind deficits. This is where an individual has difficulty consciously or unconsciously seeing the views of other individuals.[3] However, experimental evidence suggests that theory of mind deficits work slightly differently in each diagnosis. A demarcation can be made between cognitive and affective theory of mind. “Cognitive ToM [Theory of Mind] reflects the understanding of someone's beliefs, while affective ToM requires the empathic appreciation of someone's emotional state” (Booules-Katri et al 2019, p.3378). The causal mechanism of theory of mind deficits seems to have both a cognitive and an affective component. A recent study compared both types of theory of mind in autistic individuals and in a combined group of schizoid personality disorder and schizotypal personality disorder individuals.[4] The study found that autistic individuals

3 Theory of mind deficits are sometimes conceptualised as an internal module in the mind that breaks down. More sophisticated approaches see social understanding as being related to social communities and interactions with the external environment. As such, I take the sources I draw upon here as making scientific claims about cognition and emotion without then assuming this simply entails the dysfunction of an internal module.

4 Schizotypal personality disorder is a diagnosis which overlaps heavily with schizoid personality disorder. They both share many symptoms in common but schizotypal personality disorder also has symptoms covering magical thinking and odd beliefs (APA 2013, p.655).

faced greater difficulties with affective theory of mind compared to the combined schizoid schizotypal group (Booules-Katri et al 2019, p.3378). Meanwhile, autism and the combined schizoid schizotypal group seem to have similar difficulties with cognitive theory of mind. This suggests there are significant differences in the area of the causal structure covered by the mechanism of theory of mind deficits and these correlate with each diagnosis. There is a significantly stronger correlation between theory of mind deficits covering parts of the causal structure relating to difficulties reading emotions in autism compared to what occurs in SPD individuals.

These correlations can change if the population changes. Imagine autism was expanded whereby the symptom of "struggles to read the emotions of other individuals" was added to the diagnostic criteria. Currently, the diagnostic criteria only mention "difficulties in social-emotional reciprocity" (APA 2013, p.50) and "reduced sharing of interests, emotions, or effect" (APA 2013, p.50) rather than specifically reading of emotions. This would then impact SPD. Difficulty reading emotion appears to be rare within SPD but experimental evidence suggests it is present (Booules-Katri et al 2019, p.3378). Some SPD individuals who struggle reading emotions would be re-diagnosed as autistic (as would future undiagnosed individuals be diagnosed as autistic rather than SPD). This means that struggling to read emotion would now be rarer in the remaining population of SPD compared to before autism expanded. This could be a case where the causal mechanism covers a redundant element. If struggling to read emotions becomes sufficiently rare within SPD individuals then the general mechanism of theory of mind deficits has a redundant part, covering difficulties reading emotions even though this behaviour may be immensely rare or non-existent in SPD individuals after autism expands.

I have shown three consequences of expanding the diagnosis of autism for individuals with SPD. Also, both the points can work in the opposite way if the diagnostic criteria of autism were contracted whereby some individuals who currently met the diagnostic criteria for both autism and SPD would now only meet the diagnostic criteria for SPD but I do not have room to explore this. Modifying autism can mean symptoms or causal mechanisms associated with SPD can start having a redundant element or start having inadequate coverage.

5. Co-morbidity

My argument works differently when two psychiatric diagnoses can be co-morbid. This is where an individual who meets the diagnostic criteria for two particular psychiatric diagnoses should receive both diagnoses. An example of this which I will draw upon is how autism and ADHD can be co-morbid. The DSM describes ADHD as "[a] persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development" (APA 2013, p.59). Each diagnosis has many overlapping features (Mayes et al 2012, p.277; Taurines et al 2012, p.115). It has been estimated that approximately 1 in 8 ADHD youths also have co-morbid diagnoses of autism whilst approximately forty to seventy percent of autistic children have co-morbid diagnoses of ADHD (Antshel & Russo 2019, p.2).

If the diagnostic criteria for autism is expanded then some individuals who are currently diagnosed with ADHD but not autism will now be diagnosed with both ADHD and autism. This does not reduce the number of individuals diagnosed with ADHD but it does mean ADHD is no longer as central to the individual when assessed from a psychiatric perspective. When an ADHD individual is diagnosed with autism they move from being an instance of ADHD to an instance of both ADHD and autism.

The epistemic consequences of this vary depending upon whether studies exclude co-morbid individuals. Sometimes studies which aim to study a single diagnosis will exclude individuals who are co-morbid. A study which recruits ADHD individuals might exclude anyone diagnosed with both ADHD and autism (for discussion see Taurines et al 2012, p.125). This is typically because

researchers wish to study a single diagnosis and believe the presence of other diagnoses within the cohort will distort the results. I will not comment upon the justification of including or excluding co-morbid individuals from studies. I will outline both options because both are done in scientific research and because I am aware of no empirical data indicating what portion of experts-by-experience support or reject excluding co-morbid individuals from studies.

I start by considering excluding co-morbid individuals from studies (and later consider not excluding co-morbid individuals). This can impact how symptoms are formulated. Earlier, I drew upon a distinction between behaviour and symptoms. I argued that changes to a population can influence which behaviour is exhibited by that population which can then influence which symptoms are formulated. The same is true in relation to excluding individuals due to co-morbidity. The total population of ADHD will exhibit a certain level of a particular behaviour. If researchers excluded co-morbid individuals then the level of behaviour is instead based upon all individuals who are not co-morbid. If autism is expanded then some ADHD individuals will become co-morbid with autism. Consequently, the population of all ADHD individuals who are not co-morbid with autism changes after autism expands. This then almost certainly changes the level that any particular behaviour is exhibited. This can then influence which behaviour is considered statistically significant.

As a possible example of this, both ADHD individuals and autistic individuals often struggle in social situations. However, they seem to struggle for different reasons.

> "The social difficulties of individuals with ASD [autism] appear more due to the absence of positive behaviours (e.g., social approach, eye contact) rather than the presence of negative behaviours... Conversely, the social difficulties of individuals with ADHD are more likely due to the presence of negative behaviours such as interrupting and intruding on conversations" (Antshel & Russo 2019, p.3).

Autistic individuals struggle to know implicit typical social conventions whereas ADHD individuals seem to know those social conventions. Rather, for ADHD individuals, other behaviour such as low inhibition causing them to interrupt the typical flow of conversations makes socialising difficult. However, this demarcation only seems to hold once co-morbid individuals are excluded. Given how many ADHD individuals are also diagnosed with autism many ADHD individuals will exhibit low social intuition. Since there is no sharp cut off between low social intuition and standard social intuition, there will be ADHD individuals who exhibit lower than average levels of low social intuition but not sufficiently low to be considered to exhibit the symptom on the diagnostic criteria of autism. Now imagine that autism is expanded whereby the threshold for being considered to exhibit low social intuition is increased. That is, people whose level of social intuition is higher than the current threshold for autism but lower than standard levels of social intuition in the non-clinical population would now be considered to exhibit a symptom of this modified autism. A greater number of ADHD individuals would also be diagnosed with autism. Within the remaining ADHD individuals who are not diagnosed with autism, the level of low social intuition becomes lower once autism expands. This could create a redundancy. For example, one symptom on the diagnostic criteria of ADHD currently reads "[o]ften does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction" (APA 2013, p.59). This could be understood to have a redundant element because both low inhibition of ADHD *and* a level of low social intuition could be playing a role here before autism expands whereas after autism expands, inhibition may only be the relevant element.

Secondly, this general point is also applicable to causes. If autism were expanded then more ADHD individuals would now be diagnosed with autism, altering the population of ADHD individuals who are not diagnosed with autism which then alters the correlation between that population and any particular causal factor. As a possible example of this, both ADHD individuals and autistic individuals are considered to have the causal mechanisms executive dysfunction (a

mechanism posited by cognitive psychology relating to problems with "inhibition, cognitive shifting, planning, working memory, and concept formation" (Antshel & Russo 2019, p.3)). However,

> "ADHD and ASD have their own specific profile of executive dysfunction... individuals with ADHD appear to struggle most clearly with inhibition, the ability to withhold a pre-potent response, and planning/problem solving, while those with ASD [autism] struggle most with cognitive flexibility, which requires holding and switching between multiple perspectives rapidly" (Antshel & Russo 2019, p.4)

though cognitive inflexibility is still present in ADHD (Roshani et al 2020, p.3). The ADHD profile is based upon the current population making up ADHD. If some of those individuals were now diagnosed with autism after autism expands then the remaining ADHD population who are not diagnosed with autism would occupy a different area of the causal structure. In relation to cognitive flexibility, the diagnostic checklist of autism in the DSM currently mentions "inflexible adherence to routines... e.g.... rigid thinking patterns" (APA 2013, p.50). Imagine the diagnostic criteria was made slightly broader to cover, to quote Antshel & Russo again, "[difficulties] holding and switching between multiple perspectives rapidly" (2019, p.4). Adding this symptom to autism might mean more ADHD individuals who have greater problems with cognitive flexibility would be diagnosed with autism. This might mean the ADHD population who are not diagnosed with autism might have the same total level of inhibition but lower total levels of cognitive flexibility. This could mean the mechanism of executive dysfunction has a redundancy when applied to ADHD. ADHD might no longer cover areas of the causal structure associated with cognitive inflexibility which would then leave the mechanism of executive dysfunction with a significant redundant element when applied to ADHD.

My next two points relate to not excluding individuals based upon co-morbidity. The population I discuss here is all ADHD individuals regardless of if they are also diagnosed with autism. When an individual is considered as both an instance of ADHD and autism then there is the question of whether behaviour is understood to be a manifestation of a symptom associated with ADHD, a symptom associated with autism or a combination of the two. ADHD individuals who are not diagnosed with autism may exhibit a behaviour which is understood to be an instance of a symptom of ADHD. If autism expands and some of those individuals become diagnosed with autism then some behaviour may now be understood to also or instead be an instance of a symptom associated with autism. A symptom of ADHD was considered to adequately describe the behaviour before autism expands but after autism expands the behaviour is fully or partly described instead by a symptom of autism. If this occurred to a sufficiently high degree then we might start thinking the symptom of ADHD has a redundancy. The behaviour remains the same but if a symptom of ADHD is no longer, or significantly less, describing that behaviour then the symptom of ADHD may have a redundancy.

The symptoms of ADHD can also start exhibiting inadequate coverage when autism expands. As a possible example, co-morbid individuals do not simply exhibit symptoms of two independent diagnoses but rather their symptoms can present differently.

> "[P]eople with ASD+ADHD do not simply exhibit the socio-affective and communicative problems typical of ASD *plus* the hyperactivity, impulsivity, and inhibition-related problems typical of ADHD. Rather, the co-occurrence of both conditions has been found to be correlated with the appearance of typical features of each condition at a higher degree of severity" (Petrolini & Vicente 2022 p.14)

The symptoms cannot be added onto a person but instead present in a manner that is different to their individual components. The symptoms of ADHD need be formulated in a manner whereby

they can manifest differently if an individual is diagnosed with autism. However, which behaviours result from the combination of ADHD and autism can change if autism expands. Some behaviour exhibited by ADHD individuals are unrelated to ADHD, unrelated to autism and unrelated to the combined effect of ADHD and autism. It is incidental rather than something which needs covering by a symptom of ADHD. There is no need for the symptoms of ADHD to accommodate that behaviour because it is simply not considered to be a manifestation of any of these. However, once autism expands there are more ways in which the symptoms of ADHD and the symptom of autism can have a combined effect. More behaviour can now be seen to stem from the combination of these diagnoses because one of those diagnoses has expanded. As such, a particular behaviour can be seen as unrelated to ADHD, autism or their combined effect before autism expands. After autism expands that behaviour can be understood to arise from the combined effect of ADHD and autism. As such, a symptom of ADHD can start exhibiting inadequate coverage since the symptoms of ADHD needs be formulated in a manner whereby it can manifest differently when an ADHD individual is diagnosed with autism.

These points are also applicable to causes. Drawing upon the distinction between the causal structure and causal mechanisms discussed earlier, the area of the causal structure covered by ADHD does not change if some ADHD individuals become diagnosed with autism. The same individuals are still considered ADHD, regardless of if they are diagnosed with autism, so the area of the causal structure covered by ADHD remains the same. However, it raises the question of whether a particular part of the causal structure is covered by a mechanism associated with ADHD, a mechanism associated with autism or mechanism associated with both. As such, part of the causal structure might be considered an instance of a causal mechanism associated with ADHD before autism expands and then is considered an instance of a causal mechanism associated with autism after autism expands. If this happened sufficiently, then the causal mechanism associated with ADHD might be considered to have a redundancy.

Expanding autism can also mean causal mechanisms associated with ADHD can start exhibiting inadequate coverage when not excluding co-morbid individuals. Like some symptoms, some causal mechanisms associated with ADHD and autism can have a combined effect whereby they present differently to their component parts (Antshel & Russo 2019, p.4). A part of the causal structure which was previously considered incidental to causal mechanisms associated with ADHD might now start being seen to arise from the combined effect of causal mechanisms associated with ADHD and causal mechanisms associated with autism once autism expands. Whereas before causal mechanisms associated with ADHD had no need to account for that part of the causal structure, we might now need ensure the causal mechanism associated with ADHD can, when in combination with causal mechanisms associated with autism, account for that part of the causal structure. As such, expanding autism can mean a causal mechanism associated with ADHD can start exhibiting inadequate coverage.

All these four points can work in reverse if autism were contracted whereby there are less ways of meeting the diagnostic criteria. This would result in less individuals who are diagnosed with ADHD also being diagnosed with autism. There would now be more individuals who are just diagnosed with ADHD which could have implications when excluding or not excluding co-morbid individuals.

All this means that modifying one diagnosis could potentially impact individuals with a different diagnosis. Modifying autism can create redundancies or inadequate coverage for ADHD.

6.0 Value decisions

As outlined earlier, Bueter and Tekin not only cover an epistemic element to experts-by-experience involvement but also outline a value element. Experts-by-experience should have input upon value

questions relating to values employed in reformulating psychiatric diagnoses. I show now that redundancies and inadequate coverage in relation to symptoms and causes can be practically and ethically consequential.

A redundancy or inadequate coverage has the epistemic impact of symptoms and causal mechanisms inaccurately describing a particular population. This by itself has important practical and ethical consequences. Inaccurate descriptions will likely reduce self-understanding of people with the psychiatric diagnosis, and reduce understanding of the general public, the media and medical professionals towards individuals with that psychiatric diagnosis. An obvious solution would be to then reformulate existing symptoms or causal mechanisms or formulate new symptoms and causal mechanisms which are no longer inaccurate. This by itself would reduce the inaccuracies which could then reduce the negative practical and ethical implications. However, as I now outline, this might leave symptoms or causal mechanism practically or ethically less beneficial compared to how they were before the population of the diagnosis was altered. That being, if autism expands and SPD faces redundancies or inadequate coverage then the newly formulated symptom or causal mechanism formulated in response can be of less practical or ethical value compared to the symptoms and causal mechanism they replaced. I now show how this can occur in three different ways.

Firstly, it can alter ease of understanding. Imagine that when a redundancy occurs the behaviour or the areas of the causal structure which no longer needs coverage was relatively easy to understand whereas the remaining behaviour or remaining part of the causal structure which still needs covering is relatively hard to understand. The same can occur through inadequate coverage whereby the existing behaviour or area of the causal structure is relatively easy to understand but the new behaviour or new area of the causal structure which needs covering after a change in population is relatively difficult to understand. This means that before the change in population symptoms or causal mechanisms were relatively easy to understand but the symptoms or causal mechanisms which replaced them following a redundancy or inadequate coverage are relatively difficult to understand. After removing the redundancy or inadequate coverage the diagnosis is now associated with harder to understand symptoms or causal mechanisms compared to beforehand.

Secondly, it can alter the association with negative connotations. Many traits associated with psychiatric diagnoses are negatively valued. This could be thought of as, depending upon stance taken towards notions of disability or disorder, simply a matter of societal prejudice or could be understood as some traits being genuinely bad to have. Imagine that when a redundancy occurs the behaviour or area of the causal structure which was present in the population but no longer is present largely had few negative connotations. Also, imagine that when adequate coverage occurs the new behaviour or new area of the causal structure which is now associated with the population has many negative connotations. This means that before the population changes due to another diagnosis expanding a symptom or causal mechanism had relatively few negative connotations but the symptom or causal mechanism which replaced them had significantly more negative connotations. After removing the redundancy or inadequate coverage the diagnosis is now associated with a symptom or causal mechanism which has more negative connotations compared to beforehand.[5]

Thirdly, it can affect relationship to resources. Imagine that a symptom or causal mechanism was being researched heavily by scientists or received a lot of helpful medical, therapeutic or social support. Imagine this symptom or causal mechanism received a significant level of resources. Then imagine that a redundancy or inadequate coverage occurs and the reformulated or replacement symptoms or causal mechanisms receives a lot less resources compared to the previously formulated symptoms or causal mechanisms. For example, imagine that a symptom which received a lot of

5 It is easy to see how symptoms can have negative connotations but this is also true of causal mechanisms. For example, people typically consider biological causes to be more negative than psychological ones. Before the change in population the diagnosis might have formulated biological and psychological causes whereas to remove the redundancy or inadequate coverage only the biological mechanism remains.

resources is considered present in both autism and SPD but after autism expands, that symptom is now redundant in SPD. To remove the redundancy a new symptom is formulated which is considered present in SPD whereas the previous symptom which received a lot of resources is no longer considered present in SPD. Autistic people still receive lots of resources whereas SPD no longer do so. This means that after removing a redundancy or inadequate coverage the total level of resources applied to the diagnosis has been reduced.

Ease of understanding, negative connotations and level of resources all seem like important issues for value decisions. If people with psychiatric diagnoses should have input upon value issues then they should have input upon decisions that could lead to symptoms or causal mechanisms being harder to understand, having negative connotations and having less resources applied to them. Additionally, these three areas can be applied to the specific values Bueter and Tekin mention. Self-understanding and reaction of others to the diagnosed individual (Tekin 2022, p.1173) can be easily influenced by how easy symptoms and causal mechanism are to understand, whether they have negative associations and the level of resources. Medicalisation (Bueter 2019, p.1069) is obviously influenced by negative associations but ease of understanding and level of resources can also have an influence. Finally, relationship to resources that can produce good quality of life (Tekin 2022, p.1173) has obvious relationship to my discussion of level of resources.

All this shows significant practical and ethical consequences to ways of formulating symptoms and causal mechanisms. There is a significant value element and if we follow Bueter and Tekin then experts-by-experience should play a role in these value decisions. Also, I have shown how modifying one diagnosis can impact the population of another diagnosis. It can create inadequate coverage and redundancies. These can be ignored, which creates the problems that some symptoms or causal mechanisms associated with a diagnosis have significant inaccuracies. Alternatively, we can reformulate or replace the symptoms and causal mechanisms but we could face a situation where some or all the ways of reformulating could have negative practical or ethical consequences.

A particular danger is that expansions of one diagnosis occur in a manner that covers some of the more valued behaviour or areas of the causal structure associated with another diagnosis. This effectively transfers some of the more valued aspects from the diagnosis that is not being modified to the diagnosis that experts-by-experience have been involved in modifying. In relation to contractions, a contraction can mean that less valuable aspects of the diagnosis which is being modified by experts-by-experience are transferred to another diagnosis. Under these situations, reformulating or replacing a symptom or causal mechanism in response would likely lead to formulating symptoms or causal mechanisms with lower practical or ethical value. The diagnosis being modified by experts-by-experience ultimately ends up with more practically or ethically beneficial symptoms or causal mechanisms whereas another diagnosis ultimately ends up with less practically or ethically beneficial symptoms or causal mechanisms.

7.0 Broadening experts-by-experience involvement

If we accept Bueter's and Tekin's claims then my argument shows that when revising diagnostic criteria individuals with a wider range of diagnoses need to be involved in addition to those who actually have the diagnosis under consideration. For example, my argument shows that autistic people should not just be considered the sole interested party when it comes to questions of modifying autism. It can also impact people with other diagnoses. Additionally, there could be situations where autistic individuals wished to expand or contract autism but SPD or ADHD individuals did not desire the resulting consequences for their diagnoses. I now consider ways of dealing with this issue.

One way to accommodate this impact of modifying one diagnosis upon other diagnoses is awareness. For example, autistic individuals who are involved in modifying autism should have awareness of potential impacts upon people with other diagnoses. They should try and think through what other diagnoses could be impacted and consider whether those implications are positive or negative. However, autistic individuals do not have lived experience of SPD or of just being ADHD. If lived experience is important for accurate knowledge then it is unclear how autistic individuals could have sufficient knowledge to reliably make these decisions. It would also create a significant power imbalance between autistic people and people with those other diagnoses.

If we accept the general principle that people with psychiatric diagnoses should be involved in revising psychiatric diagnoses then SPD and ADHD individuals should be involved. I now consider ways of doing this. There are three main models for experts-by-experience involvement (for discussion of each model see Happell et al 2018, p.1010; Rose & Kalathil 2019, p.2; Sangill et al 2019, p.799). The first model is *consultation*. Non-diagnosed academic researchers conduct all the research and then present their findings for feedback from experts-by-experience. A second model is *collaborative*. Experts-by-experience are involved in research at all stages. This includes deciding what to research, how to research it, deciding how to interpret the results and deciding how to turn the interpreted results into theories or diagnoses. A third model is *user-controlled*. Only experts-by-experience conduct research. Non-diagnosed academic researchers are excluded. Neither Bueter nor Tekin endorse user-controlled (I also reject this). Bueter seems to endorse a combination of the first two models. Bueter writes that "a mixed model of online feedback and integration of patient representatives with different perspectives into the DSM-revision staff might be the best approach" (2021, p.4775). Some experts-by-experience should have a collaborative role by actively participating alongside psychiatrists in the revisions process whilst many other experts-by-experience can be involved more indirectly through a consultation role. Tekin also desires a collaborative role, writing that "[p]articipants in the DSM revision process must be invited to argue with each other and to ferret out the sources of their disagreements. The clinically trained experts and the patients, i.e., experience-based experts, can scrutinize each other's hypotheses and evidential reasoning" (2022, p.1174). Tekin does not comment upon accompanying it with a consultation role for other experts-by-experience. Whilst incorporating experts-by-experience through a collaborative model would seem ideal it is also a lot easier to radically increase the number and diversity of experts-by-experience involved by also employing a consultation model. As such, I endorse using both a consultation and collaboration role when revising diagnostic criteria.

We could incorporate individuals with a range of diagnoses through a consultation approach. That being, autistic individuals and academic researchers collaborate together upon deciding which modifications to make to autism and then consult SPD and ADHD individuals. This gives SPD and ADHD individuals an opportunity for feedback, stating whether any changes to the population of these diagnoses have any negative epistemic or value consequences. This approach is certainly preferable to simply ignoring people with other diagnoses or the awareness approach listed above. However, this approach contains a very significant power imbalance. Having gathered the views of SPD and ADHD individuals, the autistic individuals and the academic researchers are under no obligation to then implement any of the views by SPD and ADHD individuals. If there are conflicting desires then, since SPD and ADHD individuals have no decision making role, the autistic individuals and academic researchers can decide to entirely or almost entirely favour the views of autistic people. Also, autistic individuals might not understand the epistemic and value claims made by SPD and ADHD individuals because they lack the lived experience of those conditions. Autistic individuals might see the advantages to modifying autism but cannot adequately understand the disadvantages for other diagnoses. As such, I believe this consultation approach is inadequate (though it can be used to supplement collaborative approaches).

A more suitable approach is collaboration. Autistic, SPD and ADHD individuals should all collaborate upon decisions to modify autism (and similarly autistic individuals should collaborate

with SPD and ADHD individuals upon decisions to modify SPD and ADHD). SPD and ADHD individuals need to have a literal seat at the table when discussions occur around modifying autism and need to be part of making the decisions upon what modifications are made. Anything less than this can mean autistic individuals can make changes to SPD and ADHD without SPD and ADHD individuals having been adequately represented.

Implementing this raises important practical questions. Firstly, which diagnoses should be represented when decisions over modifying autism are made? The most obvious answer is basing inclusion upon level of overlap. Diagnoses which overlap more with autism should be included whereas diagnoses which overlap less should not be. This still leaves significant choice over where that threshold should be. This question seems to have an irreducible practical element and reasonable individuals might hold different views upon this. I feel that at least schizophrenia, borderline personality disorder, attention deficit hyperactivity disorder, schizoid personality disorder, schizotypal personality disorder and obsessive-compulsive disorder all have sufficient overlap with autism to warrant inclusion in discussions upon modifying autism.

Secondly, what degree of involvement should individuals with other diagnoses have? Imagine that fifty autistic individuals were involved in decisions about modifying autism. Should we also have fifty or ten schizophrenic individuals involved? Should it vary depending upon the degree of overlap whereby there are ten schizophrenic individuals involved but only five OCD individuals are involved? I believe that there is an irreducible practical element to these questions. Rational people can have different views upon these matters. I feel that a ratio of one to five for each diagnosis with significant overlap and one to ten for each diagnosis with lower overlap could be workable. I would consider schizophrenia, borderline personality disorder, attention deficit hyperactivity disorder, schizoid personality disorder, schizotypal personality disorder to have significant overlap and obsessive-compulsive disorder to have lower overlap with autism.

Thirdly, at what stage should individuals with other diagnoses be involved? Should they be present at the initial stage of considering possible modifications to autism, doing scientific research relating to understanding those modifications or at the stage of making decisions about what changes to implement? Again, this seems to have an irreducible practical element. I believe individuals with other diagnoses should be involved in a research capacity of considering the implications for their diagnoses and in deciding what changes to implement.

I have made potential suggestions in relation to these practical issues and, given the irreducible practical elements, many other suggestions would serve well. However, I believe there should be an overarching principle of equality of influence. The way in which we implement these practical issues should be the same for all diagnoses. That being, if schizophrenic individuals have a significant say in modifying autism then autistic individuals should have a significant say in modifying schizophrenia. Similarly, if schizophrenic individuals have little decision making input about autism then autistic individuals should only have limited input on schizophrenia. Unless there are very strong practical reasons then we should not have a situation where autistic people can have significant input on another diagnosis but people with other diagnoses cannot have significant input on autism. This general principle should be applied to number of people involved and in relation to areas of involvement. Whatever stance is taken it should be applied in a manner that respects equality among different diagnoses.

A potential example of very strong practical reasons for overruling this equality principle might be Opioid Use Disorder. Whilst most psychiatric diagnoses are correlated with early death, people diagnosed with Opioid Use Disorder have an especially high risk of early death. Perhaps the elevated risk associated with Opioid Use Disorder gives reason to significantly reduce or even eliminate involvement of individuals with other diagnoses when considering modifying the diagnostic criteria of Opioid Use Disorder. Perhaps this would be justified even if modifications to Opioid Use Disorder made other diagnoses scientifically worse or had negative value implications for other diagnoses. This is an example of the type of important bioethical questions which need asking on a

case-by-case basis when deciding if the equality principle can be violated.

To implement a principle of equality would require a level of formal organisation. There would need be a means of distributing resources to people with different diagnoses and recording level of involvement of people with different diagnoses. For example, the DSM committee could invite experts-by-experience to collaboratively engage in the DSM revision process in a manner that respects equality whereby the same number of schizophrenic individuals are invited into collaborative discussions on modifying autism as autistic individuals are invited into collaborative discussions on modifying schizophrenia. The DSM committee should also promote online consultation equally. The number of advertisements placed on the number of, say, internet forums inviting schizophrenic individuals to feedback on changes to autism would be the same as occurs in relation to inviting autistic people to feedback on changes to schizophrenia. The DSM committee cannot control the number of people with each diagnosis who respond to online consultations but they can provide equal weighing to the results of each online consultation. Also, funding bodies should fund things like fellowships and workshops which involves experts-by-experience research in a manner that would be spread among a variety of diagnoses to attain equality.

8.0 Conclusion

Tekin and Bueter convincingly argue that experts-by-experience should be involved in modifying the diagnostic criteria of psychiatric diagnoses. These philosophers do not specify which specific experts-by-experience should be involved in modifying diagnoses. I have shown that their position entails that individuals with other diagnoses than the one being modified should also be involved.

The problem arises from how diagnoses overlap with one another. As such, expanding autism could result in individuals who have alternative diagnoses now meeting the diagnostic criteria of autism. This can have important implications which take a different form depending upon the existence of differential diagnostic criteria or whether two diagnoses can be co-morbid. Expanding one diagnosis can mean, if differential diagnostic criteria are present, that individuals who have a different diagnosis, or would one day have been diagnosed with that different diagnosis, are instead now diagnosed with the expanded diagnosis. Alternatively, if two diagnoses can be co-morbid then expanding one diagnosis can mean individuals who have a different diagnosis can now also receive the expanded diagnosis. I also briefly mentioned the issue of diagnoses contracting but I did not have space to explore contracting in detail.

Modifying one diagnosis can mean that the symptoms or causal mechanisms associated with another diagnosis can start having a redundant element or start exhibiting inadequate coverage. Modifying one diagnosis can change the population of another diagnosis through differential diagnostic criteria. It also can change the level of co-morbidity in a population making up another diagnosis. This is both relevant when studying individuals who are not co-morbid and when studying individuals who are co-morbid. I highlighted how redundancies can arise because a symptom or causal mechanism might have previously covered something that was relevant but no longer is relevant after the population changes. I also highlighted how inadequate coverage can arise because a symptom or causal mechanism which covered all relevant elements no longer does cover all those relevant elements after the population changes. I have highlighted how these can have important ethical and practical consequences. Symptoms and causal mechanisms can be less ethically and practically useful. This can occur because the symptom or causal mechanism has inaccuracies due to the redundancy or inaccurate coverage. Also, removing the redundancy or inaccurate coverage through reformulating or replacing the symptoms or causal mechanisms may leave them practically or ethically worse compared to before the redundancy or inadequate coverage occurred.

This means that individuals with a range of diagnoses should be involved in decisions to modify a particular diagnosis. I outlined both consultation and collaboration as the best models. I

then outlined practical issues relating to how much say people with a range of diagnoses should have in relation to modifying a diagnosis which they do not have. I made some possible suggestions but suggested there is an irreducible practical element to these questions. However, we should employ a principle of equality whereby the level of input individuals with one diagnosis should have on another diagnosis should be equally reciprocated.

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References

APA. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed). Washington, DC: American Psychiatric Association.

Antshel, Kevin, M. & Russo, Natalie. (2019). Autism Spectrum Disorders and ADHD: Overlapping Phenomenology, Diagnostic Issues, and Treatment Considerations. *Current Psychiatry Reports* 21(34), 1-11.

Booules-Katri, Terez-Maria., Pedreño, C., Navarro, Jose-Blas., Pamias, Montserrat. & Obiols, Jordi, E. (2019). Theory of Mind (ToM) Performance in High Functioning Autism (HFA) and Schizotypal–Schizoid Personality Disorders (SSPD) Patients. *Journal of Autism and Developmental Disorders* 49, 3376–86.

Boone, Worth. & Puccinini, Gualtiero. (2016). Mechanistic Abstraction. *Philosophy of Science* 83, 686-97.

Bueter, Anke. (2019). Epistemic Injustice and Psychiatric Classification. *Philosophy of Science* 86, 1064-74.

Bueter, Anke. (2021). Public epistemic trustworthiness and the integration of patients in psychiatric classification. *Synthese* 198, 4711-29.

Chevallier, Coralie., Grèzes, Julie., Molesworth, Catherine. Berthoz, Sylvie. & Happé, Francesca. (2012). Brief Report: Selective Social Anhedonia in High Functioning Autism. *Journal of Autism and Developmental Disorders* 42, 1504–09.

Cuthbert, Bruce. N., & Insel, Thomas, R. (2013). Towards the future of psychiatric diagnosis: the seven pillars of RDoC. *BMC Medicine* 11(126), 1-8.

Darden, Lindley. (2002). Strategies for Discovering Mechanisms: Schema Instantiation, Modular Subassembly, Forward/Backward Chaining. *Philosophy of Science* 69, 354-65.

Fellowes, Sam. (2021). How autism shows that symptoms, like psychiatric diagnoses, are 'constructed': methodological and epistemic consequences. *Synthese* 199, 4499–22.

Friesen, Phoebe., Lignou, Sapfo., Sheehan, Mark. & Singh, Ilina. (2019). Measuring the impact of participatory research in psychiatry: How the search for epistemic justifications obscures ethical considerations. *Health Expectations* 24(1), 54-61

Hacking, Ian. (1995). The Looping Effects of Human Kinds. In Sperber, Dan., Premack, David. & Premack, Ann, James (Eds.), *Causal cognitions: a multi-disciplinary debate* (pp.351-83). New York: Oxford University Press.

Happell, Brenda., Gordon, Sarah., Bocking, Julia., Ellis, Pete., Roper, Cath., Liggins, Jackie., Platania-Phung, Chris. & Scholz, Brett. (2018). Mental Health Researcher's' Views About Service user Research: A Literature Review. *Issues In Mental Health Nursing* 39(12), 1010-16.

Hyman, Steven. E. (2010). The diagnosis of mental disorder: the problem of reification. *Annual Review of Clinical Psychology* 6, 155-79.

Johnson, Rebecca, A., Barrett, Marna, S. & Sisti, Dominic, A. (2013). The Ethical Boundaries of Patient and Advocate Influence on DSM-5. *Harvard Review of Psychiatry* 21(6), 334-44.

Kendler, Kenneth. S. (2010). Advances in Our Understanding of Genetic Risk Factors for Autism Spectrum Disorders. *American Journal of Psychiatry* 167(11), 1291-93.

Kendlar, Kenneth, S., Zachar, Peter., & Craver, Carl. (2011). What kinds of things are psychiatric disorders?. *Psychological Medicine* 41, 1143-50.

Kirk, Stuart. A., Cohen, David. & Gomory, Tomi. (2015). DSM-5: The Delayed Demise of Descriptive Diagnosis. In Steeves Demazeux. & Patrick Singy (Eds.), *The DSM-5 in Perspective* (pp.63-81). Netherlands, Springer.

Mayes, Susan, Dickerson., Calhoun, Susan L,. Mayes, Rebecca D. & Molitoris, Sarah. (2012). Autism and ADHD: Overlapping and discriminating symptoms. *Research in Autism Spectrum Disorders* 6(1), 277-85.

Petrolini, Valentina. & Vicente, Agustin. (2022). The challenges raised by comorbidity in psychiatric research: The case of autism. *Philosophical Psychology* 35(8), 1234-63.

Poland, Jeffrey (2014). Deeply Rooted Sources of Error and Bias in Psychiatric Classifications. In Kincaid Harold. & Jacqueline Sullivan A (Eds.), *Classifying Psychopathology* (pp.29-64). Cambridge: MIT Press.

Overton, James, A. (2011). "Mechanisms, Types, and Abstractions. *Philosophy of Science* 78, 941-54.

Rose, Diana. & Kalathil, Jayasree. (2019). Power, Privilege and Knowedge: the Untenable Promise of Co-production in Mental "Health". *Frontiers in Sociology* 4(57), 1-11.

Roshani, Fereshteh., Piri, Reza., Malek, Ayyoub., Michel, Tanja, Maria. & Vafaee, Manouchehr, Seyedi. (2020). Comparison of cognitive flexibility, appropriate risk-taking and reaction time in individuals with and without adult ADHD. *Psychiatry Research* 284, 1-6.

Sangill, Carina., Buus, Niels., Hybholt, Lisbeth. & Berring, Lene, Lauge. (2019). Service user's actual involvement in mental health research practices: A scoping review. *International Journal of Mental Health Nursing* 28, 798-15.

Stein, Dan, J. & Phillips, Katherine, A. (2013). Patient advocacy and DSM-5. *BMC Medicine* 11(13), 1-5.

Taurines, Regina., Schwenck, Christina., Westerwald, Eva., Sasche, Micheal., Siniatchkin, Micheal. & Freitag, Christine. 2012) ADHD and autism: differential diagnosis or overlapping traits? A selective review. *ADHD Attention Deficit and Hyperactivity Disorders*. 4, 115–39.

Tekin, Şerife. (2022). Participatory Interactive Objectivity in Psychiatry. *Philosophy of Science.* 1-20. doi:10.1017/psa.2022.47.

Weiskopf, D, A. (2017). An Ideal Disorder? Autism as a Psychiatric Kind. *Philosophical Explorations* 20(2), 175-90.