AUTISM AND THE PSEUDOSCIENCE OF MIND

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Abstract. This paper critically examines the theory-of-mind-deficit explanation of autism—a cognitive explanation of autistic behaviour that has significantly influenced empirical research and philosophical discourse surrounding autism. However, the claim that autistics lack a theory of mind is false. Part of the purpose of this paper is to describe how. First, a theory-of-mind deficit is inadequate as an explanatory model. Second, prior research has demonstrated the empirical failures of experiments intended to measure theory-of-mind abilities. These facts together suggest that the science of theory of mind in the context of autism is bad science. I argue that it is pseudoscience. This view has important consequences for philosophers who uncritically invoke autism (*qua* theory-of-mind deficit) as a thought experiment.

Keywords — Autism, Theory of Mind, Pseudoscience, Demarcation, Neurodiversity, Double Empathy, Monotropism, Philosophy of Autism, Autistic Philosophy, Philosophy on the Spectrum

What to do with scholarship that denies autistic agency, denies autistic voice, denies autistic personhood?
— M. Remi Yergeau, "Occupying Autism"

1. Introduction

Empirical research on autism spectrum disorder (ASD) has aimed to elucidate the psychological or cognitive mechanisms underpinning autism's behavioural manifestations.[1] Such cognitive explanations are supposed to further an aetiological understanding of autism by positing an "intervening variable" between biological and

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[1]Throughout this paper, I use "autism" (and cognates) to refer to the actual thing in the world. In contrast, I use Autism Spectrum *Disorder*, or ASD, to refer to the (pathologised) medical labelling of autism and autistics. I take the former to be a metaphysical property denoting a certain subset of the population, whereas the latter refers to a social construct that attempts to describe that population (regardless of whether it does so successfully). Thus, I occasionally use deficit language when describing ASD and research on ASD without committing to a negative description of autistics themselves. At the same time, I use identity-first language (i.e., autistic person) instead of person-first language (i.e., person with autism) to reflect the average preference of the autistic community (see discussion in Sinclair (2013); Botha et al. (2021); Bradshaw et al. (2021); Taboas et al. (2022)); "autistics" can be read as a shorthand for "autistic persons" or "autistic individuals".

environmental factors and autistic behaviour (Frith et al., 1991; Morton and Frith, 1995a,b; Pellicano, 2011). In the past half-century, numerous such hypotheses have been forwarded, including the popular claim that autistics lack a theory of mind (Baron-Cohen et al., 1985).

Theory-of-mind-deficit explanations of autism have been of particular interest to philosophers in light of the normative and theoretical entailments of the possibility of agents who are "unable" to attribute mental states to others. This fact would have consequences for epistemology, the philosophy of mind, theories of meaning, and normative theory, among other things. Thus, it should be unsurprising that philosophers have used autism as a token thought experiment for philosophical inquiry.

However, despite the rate at which philosophers repeat the claim that autistics lack a theory of mind, this claim is false. Part of the purpose of this paper is to describe how. On the one hand, previous research has demonstrated that a theory-of-mind deficit is not an adequate explanatory model of autistic behaviour insofar as purported theory-of-mind deficits are neither unique to nor universal amongst autistics. At the same time, the theory-of-mind-deficit explanation of autism does not have solid empirical grounding insofar as experiments that claim to measure theory-of-mind differences between autistics and neurotypicals have failed to replicate, and proxies for measuring theory of mind lack convergent validity. These facts suggest that the "science" of theory of mind in the context of autism is, minimally, bad science. However, the situation is more subtle.

Rather than abandoning this research programme, scholars who endorse the theory-of-mind-deficit explanation of autism have engaged in question-begging, ad-hoc hypothesising, and goalpost-shifting in an attempt to salvage the explanation. This phenomenon occurs despite well-documented empirical failures of research claiming to test theory-of-mind deficits in autistic individuals. Indeed, it continues despite first-person testimony from autistics contradicting the claim that autistics lack a theory of mind. Moreover, this view persists even though theory-of-mind-deficit explanations of autism do little to serve the autistic community. Instead, such theories reinforce the "pathology paradigm" (Walker, 2021) and further entrench dominance hierarchies of the "typical" neurotype, thus recapitulating the dehumanisation and stigmatisation of autistics in society.

In considering the combination of poor scientific enquiry and the social dimension of autism research, I argue that experimental "evidence" for the theory-of-mind-deficit explanation of autism is not merely bad science; it is pseudoscience. The pseudoscientific features of this body of research are elucidated by exploring the following two questions:

(1) *Do tests of theory of mind measure theory of mind?*

(2) *What test could disprove the claim that autistics lack a theory of mind?*

The paper proceeds as follows. Section 2 describes autism and ASD. Section 3 outlines several theories that posit a cognitive explanation for observable autistic behaviour, particularly the theory-of-mind-deficit explanation. Section 4 outlines the scientific basis of experiments purporting to test theory-of-mind abilities in autistics, and Section 5 summarises the empirical failures of this research. Section 6 describes the demarcation problem and argues that the theory-of-mind-deficit explanation is pseudoscientific. Section 7 concludes by examining this argument's consequences for philosophers who uncritically invoke autism *qua* theory-of-mind deficit as a thought experiment in normative research.

2. Autism and ASD

Autism is a neurodevelopmental difference that affects how autistics relate to and interact with the environment and people around them. In contrast, *autism spectrum disorder* (ASD) is a medical (pathological) categorisation of those who exhibit certain sets of behavioural traits deemed deficits relative to the neurotypical majority.

The diagnostic criteria for ASD have changed significantly since the codification of behaviours observed by Grunya Ssucharewa (1926), Leo Kanner (1943), Hans Asperger (1944), Lauretta Bender (1954), and others in the early 20th-Century. At this time, ASD was described as a form of childhood schizophrenia (APA, 1952, 1958),[2] later given its own classification—"infantile autism" and "autistic disorder"—in the third iteration of the *Diagnostic and Statistical Manual of Mental Disorders* (APA, 1980, 1987).[3] In 1994 and 2000, we got an expansion of autism categories under the umbrella term "pervasive developmental disorders", which include autistic disorder, Asperger's, Rett's, childhood disintegrative disorders and pervasive developmental disorder – not otherwise specified (APA, 1994, 2000). This expansion implicitly categorises autism as a spectrum—a concept advocated for by Lorna Wing in the 1980s (Wing and Gould, 1979).

The conceptualisation of autism as a *spectrum* is codified by the DSM 5, which collapses the nosological variation of the DSM-IV and DSM-IV-TR back into a single label: *autism spectrum disorder*. The DSM 5 criteria for an ASD diagnosis requires persistent deficits in each of 3 areas of social communication and interaction (A1-A3), including

A1. Deficits in social-emotional reciprocity;

[2] Asperger (1944) uses the label "autistic psychopathy", but here too the label "autism" is derived from the concept of autism in schizophrenia.

[3] Note that the DSM, published by the American Psychiatric Association, is the primary nosological reference in North America; outside of North America, the *International Classification of Diseases* (ICD), published by the World Health Organisation, predominates.

A2. Deficits in nonverbal communicative behaviours used for social interaction; and

A3. Deficits in developing, maintaining, and understanding relationships.

In addition, individuals must display at least two of four types of restricted, repetitive behaviours (B1-B4):

B1. Stereotyped or repetitive motor movements, use of objects, or speech;

B2. Insistence on sameness, inflexible adherence to routines, or ritualised patterns of verbal or nonverbal behaviour;

B3. Highly restricted, fixated interests that are abnormal in intensity or focus; and

B4. Hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of the environment.

Levels of "severity" for each of (A1-A3) and (B1-B4) are rated based on support needs (APA, 2013).[4]

Thus, although the diagnostic criteria have shifted significantly with each iteration of the DSM, it should be clear that the current description of ASD still entrenches the "triad of impairments" model—social impairments, communication impairments, and restricted or repetitive behaviour or interests—which was introduced by Wing and Gould (1979). (Although "social impairments" and "communication impairments" are collapsed into a single "pillar" in the DSM 5.) One key thing to note is that a diagnosis of ASD is based purely on behavioural characteristics.

3. Cognitive Explanations of Autism

Since the early 1970s, owing to the experimental work of Hermelin and O'Connor (1967, 1970); Frith (1970, 1972), much research has aimed to elucidate the *psychological* or *cognitive* mechanisms underpinning the behavioural manifestations of ASD (Pellicano, 2011). Despite its genetic basis, no specific gene is associated with autism—recent studies have identified over 250 genes linked with autism, including some *de novo* variations (Fu et al., 2022)—nor are there any biological markers for autism. As such, researchers have focused on furthering our understanding of autism by positing *cognitive* explanations that seek to determine an "intervening variable" between biology and behaviour (Rutter, 1983; Frith et al., 1991; Morton and Frith, 1995a,b). See Figure 1.

[4]In addition, these behavioural characteristics should (C) be present in the early developmental period, (D) cause clinically significant impairment in social, occupational, or other important areas of current functioning, and (E) not be better explained by intellectual disability or global developmental delay.

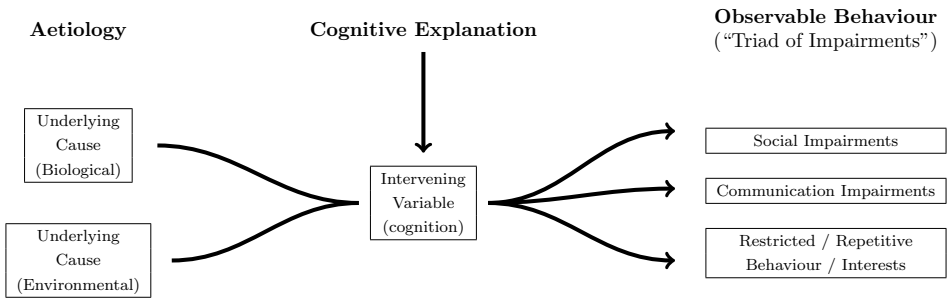

FIGURE 1. Cognitive-explanation model of ASD

To be explanatorily useful, a primary cognitive deficit must be "universal, specific, and necessary and sufficient to cause the symptoms of the disorder . . . in other words, the proximal cognitive cause of the behavioural symptoms of the disorder" (Pennington and Ozonoff, 1996, 57). In addition, the primary cognitive marker should have causal priority—meaning that it should be able to explain the earliest-emerging features of autism (Happé, 1994b; Boucher, 1996; Tager-Flusberg, 2001).

Numerous hypotheses concerning the cognitive "deficits" characterising ASD have been forwarded in the past half-century, including atypical "central" processes such as sequencing, concept formation and abstraction (Hermelin and O'Connor, 1970); core problems in language (Rutter, 1968); sensory and perceptual atypicality (Ornitz and Ritvo, 1968); disruption of "complex" information processing (Minshew et al., 1992, 1997); poor social responsiveness (Klin and Volkmar, 1993; Mundy and Sigman, 1989); and, impairments in interpersonal relatedness (Hobson, 1989, 1993, 2002), among many others.[5]

However, three particularly salient theories have been influential in directing research and conceptualisations of autism. The "theory of mind" hypothesis claims that autism is caused primarily by a specific inability to impute mental states to oneself and others (Baron-Cohen et al., 1985, 2000; Tager-Flusberg, 2007). The "executive dysfunction" hypothesis proposes that autistic behaviours are a result of a dysexecutive syndrome—a primary problem in the executive control of action (Hughes and Russell, 1993; Ozonoff et al., 1991a; Hill, 2004; Russo et al., 1998). And, "weak" central coherence theory posits that autistic individuals tend to focus on individual elements rather than wholes combined with an inability to integrate information into context (Frith, 1989; Frith and Happé, 1994; Happé and Booth, 2008; Happé and Frith, 2008). It is worth noting that each model posits a *single* cause at the cognitive level of analysis. This feature is reason alone to think that

[5]See further discussion in Pellicano (2011).

none of these hypotheses can be correct since autism is nothing if not heterogeneous.[6]

This paper focuses on the theory-of-mind explanation of ASD because, despite being false, it pervades popular descriptions of autistics. At the same time, some researchers have argued that these competing explanations are compatible insofar as, e.g., weak central coherence might be a facet of executive dysfunction (Frith, 2003) or theory-of-mind deficits might be caused by executive dysfunction (Frith and Happé, 1999; Happé, 2000; Glüer and Pagin, 2003). Importantly, all three theories are purported to entail some facts about an impaired theory of mind (Frith, 2003).

4. The Science of Theory of Mind

Despite its prevalence in the discourse surrounding ASD, theory of mind is a vexed concept. That said, one standard definition of theory of mind is the ability to *impute* or *attribute* mental states—e.g., emotions, intentions, desires, beliefs, etc.—to oneself or others (Dennett, 1978; Wimmer and Perner, 1983; Gallese and Sinigaglia, 2011). Early tests of theory-of-mind abilities involved so-called false-belief tasks, which are assumed to require, minimally, the ability to represent the mental states of others, understand that those mental states may differ from present experience—i.e., the *here* and the *now*—and distinguish others' mental states from one's own. All of these are taken to be standard "mindreading" or "mentalising" abilities.

Empirical "evidence" for the claim that autistics lack a theory of mind is given in a series of experiments, beginning in the 1980s, that utilise false-belief tasks to determine whether children (autistic or otherwise) can attribute false beliefs to another agent. One classic experiment, called the Sally-Anne test, uses the puppet-play paradigm (Wimmer and Perner, 1983) to probe belief attribution in children. The landmark study by Baron-Cohen et al. (1985), entitled "Does the autistic child have a theory of mind?"—a reference to Premack and Woodruff (1978)—led to the proposal that theory-of-mind deficits might explain the core features of autism (Baron-Cohen, 1993; Leslie, 1987, 1991; Frith et al., 1991; Baron-Cohen, 1995). However, it should immediately be apparent that such an explanation could *only* account for the *socio-communicative* aspects of ASD (A1-A3 above), but not the non-social aspects (B1-B4); this is discussed in more detail below.

[6] In light of this, some authors have proposed *multi*-deficit cognitive models of autism—see, e.g., Wing and Wing (1971); Goodman (1989); Bishop (1989); Pennington et al. (1997); others have proposed that the triad of impairments may be dissociated so that each impairment (separately inheritable) has a separate cause—see, e.g., Bolton et al. (1994); Piven et al. (1997). See also discussion in Pellicano (2011).

The children in the experiment are shown two puppets, called "Sally" and "Anne". The child watches Sally place a marble in a basket and then leave the scene. While Sally is away, Anne moves the marble from the basket to a box. Sally then returns to the scene, and the child is asked, "Where will Sally look for the marble". If the child answers "the basket", this is taken as evidence that the child is capable of attributing *to Sally* the false belief, THE MARBLE IS IN THE BASKET, despite that the child herself believes (truly) that the marble is in the box. If the child answers that Sally will look in the box, then this is taken as a failure to attribute a false belief to Sally, which is taken, in turn, as evidence for lack of theory of mind.

In Baron-Cohen et al.'s (1985) original study, the experimental design included a "typically-developing" group ($n_1 = 27$), a "Down's syndrome" group ($n_2 = 14$), and an autistic group ($n_3 = 20$). Each child is asked three control questions and one experimental question:

Naming Question	Which doll is Sally? Which is Anne?
Reality Question	Where is the marble really?
Memory Question	Where was the marble at the beginning?
Belief Question	Where will Sally look for the marble?

The *naming question* ensures that the children know which doll is which; this is important because if they think "Sally" refers to Anne, they would answer the *belief question* "incorrectly" by stating (correctly) that "Sally"—referring to *Anne*—would look in the box (false negative). The *reality question* is also an important control because if the child answers "correctly"—because she thinks that the marble is in the basket—then this would confound the results of the experimental question (false positive). The *memory question* is also an important control because if the child answers incorrectly—e.g., if she forgot the marble was in the basket and thinks that the marble was in the box at the start—then this would again confound the results of the experimental question (false negative). All of the subjects in all three groups answered the *naming*, *memory*, and *reality* questions correctly (Baron-Cohen et al., 1985, 42).

However, when posed with the experimental question—the *belief question*—85% of the typically-developing children and 86% of the children with Down's syndrome answered that Anne will look for the marble in the *basket*; in contrast, 80% of the autistic children answered that Anne will look for the marble in the *box*. These experimental results led to the assertion that "autistic children as a group fail to employ a theory of mind" (Baron-Cohen et al., 1985, 43), where such a failure is understood as an inability to represent mental states, which leads to an inability to impute beliefs to others, which causes a "grave disadvantage" in predicting others' behaviour (43).

Other experiments have been devised as proxies for testing theory-of-mind abilities, including second-order false-belief tasks (Baron-Cohen, 1989), which involve asking where some third party will think that Sally thinks the marble is; strange stories (Happé, 1994a; White et al., 2009), which involves explaining why a character in a short vignette might have said something that is not literally true; faux pas (Baron-Cohen et al., 1999), which involves asking subjects why a statement is unintentionally (socially) "wrong" or awkward; animated triangles (Abell et al., 2000), which involves describing what is happening in an animated clip containing geometric shapes; and reading the mind in the eyes (Baron-Cohen et al., 2001), which involves matching emotion and mental state descriptions to static images of the eye region of faces.

As a consequence of Baron-Cohen et al. (1985) and subsequent experiments, the view that autistics lack a theory of mind now pervades psychology. As Gernsbacher and Yergeau (2019) highlight, this view "is taught across a wide range of psychology textbooks. The assertion is argued by psychologists in state and federal court cases. The assertion is promoted by thousands of psychology articles" (2), the vast majority of which take the claim for granted. However, theory-of-mind deficits cannot adequately explain autism, and experimental evidence and autistic testimony both suggest that the theory-of-mind-deficit explanation of autism is false. The subsequent section summarises prior research on the inadequacy of the theory-of-mind-deficit theory of autism. Additional details can be found in Pellicano (2011), Gernsbacher (2018), and Gernsbacher and Yergeau (2019).

5. The Bad Science of Mind

In order for a putative cognitive model of autism to be genuinely explanatory, the explanatory features that the model proposes should: (1) be universal, or near universal, among autistics; (2) be unique to autistics (i.e., not present in individuals with other developmental conditions); (3) show causal precedence; (4) show explanatory power (i.e., the incidence and severity of the deficit should be directly related to the behavioural characteristics in each of the three domains).[7]

As noted above, 80% of autistic children in the experiment conducted by Baron-Cohen et al. (1985) failed the false-belief task. This result, of course, logically implies that 20% of the autistic children *passed* the false-belief task. Subsequent studies have had highly variable failure rates for autistics on several tasks intended to measure theory-of-mind capacities, ranging from 85% (Reed and Peterson, 1990) to 45% (Prior et al., 1990), with at least one study reporting only 10% of autistic

[7]See discussion in, e.g., Rutter (1983); Pennington and Ozonoff (1996); Boucher (1996); Happé (1994b); Tager-Flusberg (2001).

participants as failing the task (Dahlgren and Trillingsgaard, 1996). Therefore, alleged theory-of-mind deficits are not universal to autistics, and it is highly unlikely that they play any significant *causal* role in explaining the development of autism (Pellicano, 2011). Since failure on false-belief tasks (and other purported measures of theory-of-mind abilities) are not universal among autistics, the theory-of-mind deficit hypothesis fails the universality condition.[8]

At the same time, failure on false-belief tasks is also not *unique* to autistics, as many other populations of children fail these tasks.[9] Gernsbacher and Yergeau (2019) highlight that "the more atypical the child, the more likely they are to fail false belief tasks" (103). Perhaps most importantly, children with no social or emotional disability but specific language impairments also fail false-belief tasks (Miller, 2001). This fact makes good sense because studies have shown that false-belief tasks depend highly on linguistic ability (Milligan et al., 2007) and that syntax and semantics contribute to false belief understanding (Slade and Ruffman, 2005).

Linguistic analysis highlights the syntactic complexity of the belief question on some false-belief tasks since these sentences "exhibit sentential complement constructions, in which a complement clause is embedded in the matrix clause" (Gernsbacher and Frymiare, 2005, 6). As it happens, vocabulary alone predicts performance on false-belief tasks more accurately than whether a participant is autistic (Loukusa et al., 2014; Norbury, 2005). Because theory-of-mind tasks rely heavily on complex language and because ASD, by diagnostic definition, involves communicative differences, it is unsurprising that autistic participants perform less well than non-autistic participants (when they do). Furthermore, because autistics vary in their communicative abilities, it is unsurprising that autistic people vary in their false-belief task performance. Thus, it should be uncontroversial that failure on false-belief tasks—often interpreted as a failure of mentalising abilities associated with theory of mind—is not unique to autistics.

Furthermore, neurotypical children do not pass false-belief tasks, on average, until age 4. However, certain autistic behaviours—including atypicality in social responsiveness and reciprocity, gaze behaviour, joint attention, and imitation—may be noticeable around the age of 18-24 months (Dawson and Adams, 1984; Volkmar et al., 1987; Mundy and Sigman, 1989; Klin et al., 1992). This observation

[8]See further discussion in Ozonoff et al. (1991b); Bailey et al. (1996); Beversdorf et al. (1998); Bauminger and Kasari (1999); Buitelaar et al. (1999); Charman (2000); Pellicano (2011); Boucher (2012); Gernsbacher and Yergeau (2019) and citations in Gernsbacher (2018).

[9]For example, deaf or blind children (Peterson and Siegal, 1995, 1999; Russell et al., 1998; Brown et al., 1997; Minter et al., 1998; Tager-Flusberg, 2001; Green et al., 2004), children with particular language impairments (Miller, 2001; Loukusa et al., 2014; Norbury, 2005), Down's syndrome (Zelazo et al., 1996), Williams syndrome (Lo et al., 2013), cerebral palsy (Dahlgren, 2002; Caillies et al., 2012), Parkinson's (Saltzman et al., 2000), Fragile X (Cornish et al., 2005), epilepsy (Raud et al., 2015), and more (Benson et al., 1993; Yirmiya et al., 1996; Zelazo et al., 1996; Benson et al., 1993; Payne et al., 1995; Reidy et al., 2013; Rasmussen et al., 2009).

implies that theory of mind does not have causal precedence for explaining autism. This fact has led some researchers to *broaden* the definition of "theory of mind" to include precursors, such as eye-gaze detection and shared attention (Baron-Cohen, 1994, 1995). However, this broadening of criteria leads to a circularity whereby the earliest behavioural signs of autism are *defined* as components of a theory-of-mind deficit (Hughes and Leekam, 2004; Pellicano, 2011). Rather than being responsive to empirical evidence, this redefinition threatens to make the statement "autistics lack a theory of mind" only trivially true.

Thus, purported theory-of-mind deficits are neither unique to nor universal in autistics, and the theory-mind-deficit explanation of autism lacks causal precedence and explanatory power. From these facts, it follows that the theory-of-mind-deficit explanation is inadequate as an cognitive model of autism.

In addition to being a poor causal model, Gernsbacher and Yergeau (2019) thoroughly document key empirical failures of tests that purport to measure theory of mind. Besides the lack of specificity (uniqueness) and universality with regard to autistics' failing theory-of-mind tasks mentioned above, many of the tasks proposed to assess theory of mind fail to converge. For example, "strange stories" fails to correlate with "reading the mind in the eyes", "animated triangles", and "faux pas" tasks, particularly when language comprehension is controlled.[10] Similarly, "reading the mind in the eyes" fails to correlate significantly with the "faux pas", "animated triangles", "false belief", and other theory-of-mind tasks.[11] Moreover, false-belief tasks can fail to correlate significantly with one another.[12] Gernsbacher and Yergeau (2019) suggest that the lack of convergent validity among theory-of-mind tasks undermines the degree to which these tasks actually measure theory of mind in participants—i.e., construct validity.

At the same time, a lack of theory of mind should entail difficulty with several distinct social abilities—e.g., social attention, cooperation, anticipation, persuasion, deception, avoidance, etc. However, studies have demonstrated that autistics of all ages can understand others' intentions, goals, and desires.[13]

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[10]See Spek et al. (2010); Ahmed and Miller (2011); Scherzer et al. (2012); Vetter et al. (2013); Hollocks et al. (2014); Wilson et al. (2014); Chen et al. (2017); Lukito et al. (2017) and discussion in Gernsbacher (2018); Gernsbacher and Yergeau (2019).

[11]See Ozonoff et al. (1991a); Bora et al. (2005); Spek et al. (2010); Ahmed and Miller (2011); Duval et al. (2011); Gooding and Pflum (2011); White et al. (2011); Scherzer et al. (2012); Li et al. (2013); Hollocks et al. (2014); Chen et al. (2017); Lukito et al. (2017), and discussion in Gernsbacher (2018); Gernsbacher and Yergeau (2019).

[12]See Charman and Campbell (1997); Hughes (1998); Duval et al. (2011), and discussion in Gernsbacher (2018); Gernsbacher and Yergeau (2019).

[13]See, e.g., Aldridge et al. (2000); Carpenter et al. (2001); Russell and Hill (2001); Kerr and Durkin (2004); Ponnet et al. (2005); Sebanz et al. (2005); Hubert et al. (2007); Liebal et al. (2008); Colombi et al. (2009); Falck-Ytter (2010); Channon et al. (2011); McAleer et al. (2011); Vivanti et al. (2011); Fitzpatrick et al. (2013); Berger and Ingersoll (2014); Forgeot d'Arc et al.

Finally, reproducing studies' results is one of the cornerstones of scientific inquiry. Nonetheless, the findings of many highly-cited studies have failed to replicate. Contrary to the findings of Baron-Cohen et al. (1985), subsequent studies found no statistically significant differences between autistic and non-autistic groups on first-order false-belief tasks.[14] Gernsbacher and Yergeau (2019) enumerate similar failures to reproduce the experimental results of second-order false-belief tasks (Baron-Cohen, 1989) and strange stories tasks (Happé, 1994a).

All this research together suggests that the theory-of-mind-deficit theory of autism is *bad science*. (Of course, this has not prevented outlandish claims about autistics lacking a theory of mind.) In the next section, I argue that the theory-of-mind-deficit explanation of autism is not merely bad science but *pseudoscience*.

6. The Pseudoscience of Mind

Following the fundamental issue at the heart of demarcating science from pseudoscience (Fuller, 1985, 331), we can ask: Are beliefs about autistics' purported lack of a theory of mind epistemically warranted? Although there is disagreement about general principles for demarcating science and pseudoscience, there is more agreement on individual cases (Hansson, 2021). Although I will examine alternative criteria below, I begin with the classic Popperian approach to demarcation which suggests that for a hypothesis to be scientific, it must be *falsifiable*—in the sense that "statements or systems of statements . . . must be capable of conflicting with possible, or conceivable observations" (Popper, 1962, 32). In this case, a *theoretical* sentence is falsifiable just in case it logically contradicts some *empirical* sentence that describes a logically possible event that it would be logically possible to observe (Hansson, 2021). In this sense, *good* science is supposed to be risky. So, good scientific theories consist of highly-falsifiable statements that have been well-tested and, thus far, not falsified.

However, as has already been noted, even the earliest research shows that many autistic subjects pass tests that purport to measure theory-of-mind abilities. On the assumption that neurotypicals passing these tasks implies that they have a functioning theory of mind, it should presumably follow that autistics passing these tasks implies they have a functioning theory of mind. Indeed, early on in this research, Happé (1994a) notes that the success of autistic subjects at false-belief tasks "could be regarded as genuine proof of their possessing a theory of mind" (130). On the falsifiability criterion for demarcating science from pseudoscience,

(2016); Green et al. (2017); Cole et al. (2018); Li et al. (2019) and discussion in Gernsbacher and Yergeau (2019).

[14] See Oswald and Ollendick (1989); Dahlgren and Trillingsgaard (1996); Yirmiya and Shulman (1996); Yirmiya et al. (1996); Russell and Hill (2001); Moran et al. (2011); Fitzpatrick et al. (2013), and discussion in Gernsbacher (2018); Gernsbacher and Yergeau (2019).

theory-of-mind-deficit theory of autism is indeed scientific—insofar as the claim that autistics lack a theory of mind is, in principle, falsifiable. It just happens that this is also *bad* science insofar as the theoretical sentences comprising the theory-of-mind-deficit theory of autism have been repeatedly contradicted by empirical statements of fact—i.e., *falsified*.

Unfortunately, the state of inquiry surrounding theory of mind and autism is more pernicious than this. Although Happé (1994a) admits that the success of autistic subjects at false-belief tasks "could be regarded" as evidence against the theory-of-mind deficit in autistics, she continues: "Alternatively, their success could be seen not as proof of theory-of-mind ability but rather as evidence of the 'hacking out' of some strategy for solving the tasks" (130). Thus, researchers create new measures when existing measures fail to support the claim that autistics lack a theory of mind.[15] Some have argued that non-autistic clinical groups may fail false-belief tasks for reasons other than a "genuine" representational deficit (Baron-Cohen, 2000; Tager-Flusberg, 2001). Researchers have previously argued that although *some* autistics could pass first-order false-belief tasks, they still failed at (more difficult) *second*-order false-belief tasks (Baron-Cohen, 1989). This practice underscores the moving goalposts of the theory-of-mind explanation of autism.

There is a dilemma hidden here, which can be summarised as follows. If we accept that tasks which purport to measure theory-of-mind ability are good proxies, then the theory-of-mind-deficit explanation of autism is falsifiable (so perhaps scientific) but also falsified. Hence, the researchers who persist with attempts to demonstrate a lack of theory of mind in autistics are acting unscientifically. On the other hand, if the explanation for why tasks that purport to measure theory-of-mind abilities fail to do so is because they are not good proxies, or if contrary evidence is explained away by defining autism as involving a lack of theory of mind, then the statement "autistics lack a theory of mind" becomes unfalsifiable and hence pseudoscientific.

Let us examine the second horn first. We have already seen that studies have demonstrated that autistics can understand others' intentions, goals, and desires—prototypical theory-of-mind abilities. At the same time, autistics sometimes fail tasks that purport to measure theory of mind. These two facts suggest that these tasks do not actually measure theory-of-mind abilities; hence, they are bad proxies. (Recall that many such tasks are better indicators of linguistic ability than theory-of-mind ability.)

[15]This is a stunning example of Goodhart's Law: when a measure becomes a target, it ceases to be a good measure.

Furthermore, several of these tasks lack a ground truth because the "correct" answers to the experimenter's questions are context-relative. For example, the reading-the-mind-in-the-eyes task presupposed that emotions can be read from static images, but emotions are highly dependent upon context and culture (Pugh et al., 2021). Considering the Sally-Anne test, researchers assume that the "correct" answer is that the marble is in the box; however, autistics have underscored competing explanations, the plausibility of which highlight the lack of objective truth about the matter. For example, one commentator wonders whether the results might differ if the dolls were presented as children rather than adults. This question reflects a belief, which some children might hold, that adults simply know things, and hence Sally would know that the marble is in the box (Blackburn et al., 2019).

Part of the point worth highlighting here is that autistic ways of thinking often differ from neurotypical ways of thinking. Indeed, the Sally-Anne test suggests that even if the participant could not know something in a given situation (e.g., if she were in Sally's position), this does not imply that someone else would not be able to know something in the same situation. Again, knowledge here is highly context-dependent, and an individual might have different reasons for believing something in light of having, e.g., different background knowledge. Consider, for example, that Anne *always* moves Sally's marble from the basket to the box. Suppose also that Sally and Anne are good friends—i.e., Sally knows that Anne is always up to some mischief. Hence, when Sally leaves the scene and comes back, she might think, "Anne probably moved my marble again", and look in the box rather than the basket.

This rationalisation might sound utterly ad hoc; however, it is worth noting that all the co-authors of Baron-Cohen et al. (1985) frequently refer to Anne as "naughty Anne" in writing (Frith, 1989, 1991, 1998, 2001, 2008; Frith and Happé, 1999; Leslie, 1992; Baron-Cohen, 2003a). It is unclear whether this is the language that the experimenter uses in practice; however, if it is, then there is no reason why a subject might not pick up on this and consider that in her answer.[16] Rather than lacking a theory of mind, such an analysis suggests that autistics are more adept at such tasks as they do not "jump to conclusions", in the way that the (neurotypical) experimenters clearly expect the subject to do. Thus, autistics have aptly highlighted that the "correct" answer to the Sally-Anne task is not "Sally will look for the marble in the basket"; a better answer is something akin to, "It is very likely that Sally will look for the marble in the basket, but it is not impossible

[16]As far as I am aware, there are no scripts or transcripts from the original experiment in 1985; however, in several video recordings demonstrating the Sally-Anne task, the experimenter does indeed refer to Anne as "naughty Anne" or "tricky Anne".

that she will look in the box, or maybe in some other place, or she may not look for the marble at all" (Blackburn et al., 2019).

Researchers interested in measuring theory of mind in autism have typically ignored the possibility that autistics' passing these tasks demonstrates theory-of-mind abilities.[17] This, of course, is despite ample evidence to the contrary and also despite the irrefutable position that neurotypicals' success on these tasks demonstrates theory of mind. For example, Bloom and German (2000) suggest that

> Some [autistic individuals] fail the false belief task because they lack the capacity to acquire a theory of mind. In contrast, [typically-developing] 3-year-olds might fail the false belief task because of general task demands, because they don't have a grasp of false belief, or both. But [the typically-developing children] surely have a "theory of mind", in the general sense of having a sophisticated ability to reason about the mental states; this is precisely why they differ from autistic individuals in the social, communicative, and imaginative domains. (B29)

Hence, in practice, theory-of-mind abilities are taken for granted in neurotypicals, whereas theory-of-mind deficits come to *define* autism. Barnbaum (2008) goes so far as to suggest that the "whole point of theory of mind deficits is that the lack of theory of mind is a fundamental deficit that is characteristic of autism: If he did not have a compromised theory of mind, he would not be autistic" (Barnbaum, 2008, 160). Thus, the claim that "autistics lack a theory of mind" becomes analytic; hence, this statement is unfalsifiable. Once researchers have decided that autistics lack a theory of mind, no evidence could prove this false. The catch-22 here is that a lack of a theory of mind is taken to imply a lack of a theory of one's own mind (Carruthers, 1996). Hence, autistic testimony to the effect "I have a theory of mind" can be waved away on this account. For example, in response to an autistic subject's first-personal report about his inner experience, Frith and Happé (1999) write, "Very little of this description seemed believable" (13).[18] This is pseudoscience.

Now let us examine the first horn of the dilemma. Suppose that tasks purporting to measure theory-of-mind abilities are good proxies. In this case, the claim "autistics lack a theory of mind" has been falsified. Nonetheless, researchers persist in repeating the claim, devising increasingly challenging experiments to attempt to prove it.

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[17] See, for example, the ad hoc explanations given by Happé (1994a,b, 1995); Frith et al. (1994); Tager-Flusberg (2001); Baron-Cohen (2006), and the criticism of this logic in Gernsbacher and Yergeau (2019).

[18] One might reasonably wonder how the authors could know this.

Sometimes, in the history of science, practising scientists do not abandon a theory in light of falsifying empirical evidence. Sometimes, in the history of science, this is okay. For example, Newtonian celestial mechanics is falsified by the precession of the perihelion of Mercury. However, despite empirical evidence contradicting the theory, researchers did not abandon Newtonian mechanics; instead, they formulated new explanations that would allow them to hold on to the theory while explaining away the empirical observations that contradicted it—e.g., the existence of a hitherto unobserved planet could affect the perihelion of Mercury in such a way that the observations maintain consistency with Newtonian mechanics.

On Popper's (1962) criterion, one might think that this is unscientific. However, Lakatos (1970) contends that scientists were right not to abandon Newtonian mechanics. In this case, the theory was particularly *useful*, so there was good pragmatic reason not to abandon it despite falsifying evidence. Furthermore, this is often how science works when considering the sociology of scientific practice and a pragmatic epistemology of science (Waters, 2019). Hence, Popper's falsifiability criterion for demarcating science and pseudoscience is too restrictive.

Lakatos's (1970) notion of *methodological* falsification posits a sequence of theories, called a *research programme*, which includes a shared "hard core" in addition to auxiliary hypotheses. The auxiliary hypotheses connect the programme's hard core to the empirical world (via predictions) while also "protecting" the hard core, making it effectively irrefutable. In this case, the combination of the hard core and the auxiliary hypotheses are subject to empirical tests; therefore, a *programme*, on the whole, is (in principle) falsifiable. When an empirical prediction turns out to be false, science progresses by retaining the hard core of the programme and constructing new auxiliary hypotheses. In this case, a research programme is called *progressive* if it is both *theoretically* progressive—meaning that the hard core plus auxiliary hypotheses predict novel empirical facts—and *experimentally* progressive—meaning that some of the novel empirical facts predicted by the theory can be tested. Here, "novelty" means that a prediction is not furnished by a previous theory in the sequence and is not predicted by competing theories (or conventional wisdom).

Thus, theoretical progressiveness requires that each new theory in a research programme (a sequence of theories) should have excess empirical content over and above its predecessor. In contrast, a research programme is *degenerating* just in case it either does not predict novel facts (theoretically degenerating) or none of the novel facts it predicts can be tested (experimentally degenerating). Hence we can demarcate good and bad science (or genuine and pseudoscience) as follows. A

sequence of theories is good science if it is progressive and bad if it is degenerating; furthermore, a research programme may degenerate so much as to become pseudoscience.[19]

By analogy, just as scientists did not abandon Newtonian mechanics despite falsifying evidence, we might think that researchers have not abandoned the theory-of-mind-deficit explanation of autism despite falsifying evidence. The question, then, is whether this is warranted.

It is easier to demarcate the components of a research programme in historical cases than contemporary ones because we have the benefit of hindsight. For example, when considering the precession of the perihelion of Mercury, we can clearly differentiate the hard core—Newtonian mechanics—from the auxiliary hypotheses. In the case of the theory-of-mind-deficit explanation of autism, however, it is less clear whether a lack of theory of mind constitutes the research programme's hard core or an auxiliary hypothesis. If the former, then the theory-of-mind-deficit explanation of autism *should* be protected from falsification. However, the hard core of a research programme is not typically empirical. For example, Newtonian mechanics can be used to predict the movement of celestial bodies, but it will not tell you anything about what the night sky actually looks like. (To know this, one must go into the world and observe the stars.) This is why the auxiliary hypotheses serve to connect the hard core to the empirical world by positing statements that are testable and so require empirical observation. A lack of theory of mind would have empirical consequences, so it appears to be an auxiliary hypothesis.

Of course, it may be the case that Newtonian mechanics, as the hard core of a research programme, does have *some* empirical content which happens to be highly abstract. At the same time, it is probably more difficult to clearly separate the "purely" theoretical versus empirical components of psychology that it is with theoretical physics, insofar as any psychological theory is bound to have some empirical content. Notwithstanding, it seems more apt to describe the theory as an auxiliary hypothesis in a research programme whose hard core involves some broad generalisations of psychology—e.g., that there are such things as mental states, that mental states depend on brain states, that some generalisations about them can be explained genetically whereas others require an environmental basis, etc. Such a hard core presumably consists of the sorts of claims that guide psychological model-building in general. A specific claim about a cognitive explanation in a specific branch of psychology—such as the theory-of-mind-deficit explanation of autism—is far too narrow to serve as part of a hard core of a scientific research programme, in Lakatos' sense.

[19]Note that the distinct questions of whether something is scientific or pseudoscientific and whether something is good or bad science are collapsed into a single axis on this account.

Furthermore, for this horn, we assumed that tests of theory-of-mind abilities are good proxies—i.e., they *actually* measure theory of mind to some degree. It follows that this auxiliary hypothesis has been repeatedly falsified in light of the empirical evidence discussed in Section 5. Hence, the facts predicted by the theory have failed to be borne out, implying that the theory is experimentally degenerating. As it happens, the theory is also *theoretically* degenerating insofar as it lacks excess empirical content. Once again, novelty is time-relative on this account. So, even if the predictions furnished by the theory-of-mind-deficit explanation of autism were novel in 1985, the explanation is no longer theoretically progressive.[20] Thus, it is somewhat over-determined that theory-of-mind-deficit explanations of autism constitute a degenerating research programme.

Both horns of the dilemma lead to the same conclusion. Hence, beliefs about autistics' purported lack of a theory of mind are not epistemically warranted. This research programme is not merely bad science; it is pseudoscientific.

At the same time, however, autistic researchers have proposed alternative explanations to the theory-of-mind-deficit explanation of autism. For example, *monotropism* theory suggests that autistics' interests direct attention more strongly than in non-autistics (Murray et al., 2005). This view accounts for autistic inertia, sensory differences, social differences, and focused interests inherent to autistics without invoking pathologising, deficit-based language (Murray, 2018). Similarly, the *double empathy problem* (Milton, 2012; Milton et al., 2022) argues that the apparent social and communicative difficulties observed in autistics that have led to the claim that they lack a theory of mind are actually due to a reciprocal lack of understanding and bidirectional differences in communication style, social-cognitive characteristics, and experiences between different neurotypes (Crompton et al., 2020c). Hence, mismatches in communication styles can contribute to autistic social difficulties (Davis and Crompton, 2021). These proposals constitute good science insofar as they are falsifiable and they generate novel predictions. Recent research on intra- and inter-neurotype information transfer has provided empirical evidence supporting the double-empathy problem (Crompton et al., 2020a,b,c), the findings of which are inconsistent with the social-cognitive deficit narrative of autism.

[20]Attempts have been made to re-invigorate the theory's novelty, thus salvaging the claim that autistics lack a theory of mind. For example, admitting that a theory-of-mind deficit could only account for the social aspects of ASD, Baron-Cohen (2002, 2003b, 2010) has embedded "mind-blindness" within a larger theory dubbed the "extreme male brain" theory of autism (sometimes called the "empathising-systematising (E-S)" theory of autism). However, it is not obvious that sexing the brain makes the theory-of-mind-deficit explanation of autism better. Instead, it trades one falsehood for another. Rippon (2020) aptly refers to this theory as "neurotrash". See Krahn and Fenton (2012a); Sample (2013); Ridley (2019) for lucid criticisms.

7. The Normativity of Mind

Much of the research discussed in the previous sections might be called "primary" insofar as it includes on-the-ground experiments and novel data. When considering how philosophers engage with autism, the literature is typically secondary—philosophers usually cite the primary experimental literature rather than conducting experiments themselves. Given the apparent contingent of researchers in the primary literature that are inexplicably wedded to the claim that autistics lack a theory of mind in some form, this implies that the claims are repeated by philosophers. And, indeed they are: philosophers referencing this literature simply take for granted that autistics lack a theory of mind. Beginning from this assumption, this secondary (philosophical) literature proceeds to draw out the logical entailments of this "fact".

For example, we have already seen that if autistics lack a theory of mind, then autistics lack a theory of their own minds—i.e., they lack self-consciousness (Frith and Happé, 1999)—which in turn implies that they lack (first-personal) epistemic authority (Carruthers, 1996) or they lack the property of *moral personhood* (Warren, 1973). Furthermore, if autistics lack the property of moral personhood, then they presumably do not have full and equal moral rights (Warren, 1973), or they are not deemed members of the moral community (Benn, 1999)—i.e., those individuals with whom others share moral obligations.

These considerations seem to empower neurotypicals to enact epistemic injustices toward autistics on the assumption that they do not know any better.[21] The in-built testimonial injustice following from a lack of theory of mind reinforces the view since no evidence to the contrary—e.g., autistics uttering propositions like "I do not lack a theory of mind"—needs to be taken seriously. The exclusion of autistics from the moral community also raises questions about autistics' right to life, which can be used to justify research funding on the prevention of autism via genetics—i.e., eugenics programmes which are couched in the language of a "cure".[22]

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[21] As a salient example of this brand of ableism, in 2023, several states in the USA passed bills that restrict gender-affirming care, mentioning autism by name as a *justification*. For example, the state of Georgia's S.B.140 says that because gender dysphoria is "often comorbid with other mental health and developmental conditions, including autism spectrum disorder", certain surgical procedures for the treatment of gender dysphoria in minors should be prohibited (Georgia General Assembly, 2023). The logic here is that autistics do not know themselves; thus, when an autistic says, "I am trans", they can be ignored. Among other things, these laws provide further evidence in favour of the claim that ableism is the packaging with which transphobia is delivered (Smilges, 2022a). See also Smilges (2022b, 2023).

[22] Several reviews have shown that a majority of research funding in the field of autism is devoted to "basic science", including genetics and other "risk factors" (Singh et al., 2009; Krahn and Fenton, 2012b; Pellicano et al., 2014), although autistic communities have advocated for increased research on how public services can best meet the needs of autistics (Fletcher-Watson et al., 2017; den Houting and Pellicano, 2019; Roche et al., 2021).

At the same time, if autistics are not members of the moral community, then advocating for eugenics to eradicate autism may be taken to be defensible (Barnbaum, 2008).

Examining a different line of entailments, if autistics lack a theory of mind, then if theory of mind is a subset of empathy, as some have claimed (Baron-Cohen, 2002, 2003b, 2009, 2011; Baron-Cohen and Wheelwright, 2004), then it logically follows that autistics lack empathy (Chapman et al., 2006; Knickmeyer et al., 2006; Chura et al., 2010; Auyeung et al., 2010a,b; van Honk et al., 2011). If autistics lack empathy, then they cannot live the "good life"—at least on those accounts of the good life that require empathy (Nussbaum, 2006). Indeed, some philosophers have explicitly argued that "living an autistic life is not, *ceteris paribus*, as good a human life as that child's life had he not been born autistic" (Barnbaum, 2008, 149). Similarly, if autistics lack a theory of mind, and if theory of mind is one of the quintessential abilities that makes humans human (Baron-Cohen et al., 1985), it logically follows that autistics are not fully human.[23]

Moreover, if autistics lack a theory of mind, a community of autistic persons is impossible (Barnbaum, 2008).Although some are more obvious or explicit than others, all of the above claims are equally dehumanising to autistics.[24] It should be clear at this point that much philosophical work on autism falls under ethics, philosophy of mind, philosophy of psychology, or philosophy of medicine and bioethics (Bölte and Richman, 2018). That said, the upshot of an analysis of pseudoscientific approaches to autism research is that there are obvious ways in which the philosophy of science can positively benefit autism research and, indeed, benefit autistics. Importantly, however, this research must avoid succumbing to the current standard of research-based violence, which the autistic scholar Monique Botha defines as "a form of systemic violence perpetuated through societal systems" (Botha, 2021, 4), such as, for example, the academy.[25]

8. Conclusion

The theory-of-mind-deficit explanation of autistic behaviour is inadequate as a model insofar as a failure on tasks intended to measure theory-of-mind abilities is neither universal amongst nor unique to autistics, and theory-of-mind abilities lack causal precedence and explanatory power. At the same time, these tests lack

[23]I am not aware of any philosopher who actually *draws* this consequence, but it is a logical entailment *had* by claims that philosophers do make.

[24]In this case, dehumanisation can be variously defined as the denial of full humanness to others (Haslam, 2006), the denial of specific traits which are said to unite all humans or separate humans from non-human animals (Haslam, 2006), the denial of a group's ability to experience complex emotions (Leyens et al., 2000), the exclusion of a group from moral boundaries (Opotow, 1990), or the denial of a group's community or identity (Kelman, 1973). See the discussion in Botha (2021).

[25]See also the discussion of *epistemological violence* in Teo (2010).

convergent validity, implying that theory of mind (in the context of autism) lacks construct validity. The results of studies of theory-of-mind abilities often contradict the popular conception that autistics lack a theory of mind. Taken together, these facts should uncontroversially suggest that the "science" of theory of mind is bad science.

Moreover, the theory-of-mind-deficit explanation of autism, I have argued, has all the hallmarks of a degenerating research programme. These characteristics move the programme from merely bad science to genuine pseudoscience. To some extent, this should be unsurprising, insofar as autism, through history, has been no stranger to pseudoscientific claims—for example, "refrigerator mothers" cause autism (Bettelheim, 1967); "vaccines cause autism", etc. There is also a highly predatory industry predicated on offering cures and treatments for autism, including forcing autistic children to drink bleach, receive bleach enemas, undergo chelation, or be subjected to ABA therapy. The pseudoscientific features of the theory-of-mind-deficit explanation of autism are particularly pernicious when we consider that philosophers often uncritically assume that autistics lack a theory of mind. More than an idle thought experiment, the repetition of this view by philosophers serves to further stigmatise and dehumanise autistics while further entrenching violent dominance hierarchies.

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