

How social deficit models exacerbate the medical model: Autism as case in point.

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Abstract

This analysis argues that social deficit theories exacerbate the worst excesses of the medical model, a framework that attributes autism (in this example) as the cause of a person's functional impairment or disability, and empowers professionals and caregivers to treat autistic people's problems. Social deficit theories of autism generally conceptualise a deficit in understanding of others or motivation to relate to others as its primary cause.

Harms of the medical model heightened by these theories include dehumanisation that denies basic respect and dignity, pathologisation of neutral and positive differences, reductionism to a social disorder despite complex traits and sensorimotor underpinnings, and essentialism despite autism's fluid boundaries.

Proposed solutions include a more holistic and socially embedded classification system that recognises strengths and functional differences, more inclusion of autistic people in research and society, and practical strategies to help autistic and non-autistic people understand one another.

Introduction.

Deficits in reciprocity and relationships cannot lie in just one person – yet the autism diagnosis pathologises an autistic person's contribution to social processes (American Psychiatric Association [APA], 2013), essentially blaming an individual's autism for any problems in social communication and interaction. This stems from autism's origin and placement in the medical model, which locates deficits and functional impairment within autistic people and conceptualises them as directly and uniquely causing disability (Chown and Beardon, 2017; Kapp, 2013b). It views all differences related to the "disorders" diagnosed as dysfunctional, and ignores interpersonal and societal contributions to autistic people's challenges. This model empowers parents and professionals with the responsibility for treating individuals, regarded as submissive "patients" defined as "collections of organs that can malfunction or become infected" (Brickman *et al.*, 1982: p. 372).

The social model of disability, which recognises social construction of disability through oppression or access barriers, has helped to empower disabled people to resist practices of the medical model (Kapp, 2020; Kapp, 2013b). Nevertheless, global austerity measures since the Great Recession have threatened and overturned advances (Berghs, Atkin, Hatton and Thomas, 2019; Oliver, 2013). While mainstream autism researchers such as the main author of autism's leading diagnostic instruments, Catherine Lord (2012), acknowledge strengths associated with autism (Gillespie-Lynch, Kapp, Brooks, Pickens and Schwartzman, 2017), the diagnosis and its assessments continue to omit or pathologise them (APA, 2013). Social deficit theories continue to exacerbate the worst features of the medical model, pathologising the role of the people diagnosed in complex transactional and sociological dynamics. Arguably, no socially specific parts or "modules" of the mind or brain exist, but rather social dynamics always take place as part of broader processes within and between people (Kapp, 2013a). Indeed, the most recent revision of the *Diagnostic and Statistical Manual of Mental Disorders* added a social communication disorder diagnosis, but it suffers from a lack of evidence or utility (Tager-Flusberg, 2018). Lord, a member of the *DSM* workgroup responsible for this addition, admitted at the time of introduction: "The new social communication (pragmatic) disorder was created for political and health reasons ... *DSM-5* was not a scientific process ... the empirical evidence is NOT in support of social pragmatic disorder" (Insel, Lord and Tager-Flusberg, 2013). As a major communication scholar and autism researcher described, "Entry into the *DSM* ... has not changed anything: There are no new assessment tools, no clear diagnostic criteria, no stronger evidence for the existence of the condition and no innovative, effective interventions" (Tager-Flusberg, 2018). In contrast, autism commands fast-growing research attention, specific legislation and programmes for treatment or services, and a community of autistic people, relatives and professionals invested in the diagnosis. Therefore, this article focuses on autism as a case study for how social deficit models amplify the problems of the medical model.

Deficit-based, primarily social theories of autism amplify the medical model's dehumanisation, pathologisation, reductionism and essentialism, ironically demonstrating a lack of empathy by contributing to unnecessary marginalisation. Theories of a mechanism called theory of mind (the ability to attribute mental states, such as beliefs, intentions and emotions, to oneself and others) particularly deny agency to autistics, further privileging the power of non-autistics, and especially scientists and clinicians, to understand and act on behalf of us. Social motivation theory argues that autistic people lack desire for and enjoyment of interaction, despite evidence of our interest and empathy (Kapp, Goldknopf, Brooks, Kofiner and Hossain, 2019). Paradoxically, as an autistic autism researcher and psychologist, in this paper I mostly critique this and similar developments in the field on the basis of empirical evidence and outsiders' views rather than my and other self-advocates' lived experiences. A critical analysis of the literature supports the view that the "disability" of autistic people's atypical communication largely stems from the medical model's and society's oppressive practices. These harms of the medical model include dehumanisation that denies basic respect and dignity, pathologisation of neutral and positive differences, reductionism to a social disorder despite complex traits and sensorimotor underpinnings, and essentialism despite autism's fluid boundaries.

Dehumanisation

The medical model's separation of autism from the person and privileging of professionals dehumanises autistic people. Haque and Waytz (2012) argue that dehumanisation is inherent to medical settings because "mechanization" (such as objectification as an entity incapable of social responsiveness), "empathy reduction" and "moral disengagement" serve the "function" of helping practitioners efficiently

problem-solve. For example, they note that Cheng and colleagues (2007) found that inhibiting their natural impulse to feel patients' pain helps doctors maintain the cognitive self-regulation (including theory of mind) to perform their work. (The authors, do, however, recognise these phenomena as problems, and suggest solutions to empower patients and improve care.) Conversely, "deindividuating practices," "impaired patient agency" and provider-patient "dissimilarity" nonfunctionally dehumanise, but flow from the setting, relationship and practices (Haque and Waytz, 2012). Similarly, several studies have reported that medical students learn to keep this power distance from patients as a tacit part of their training (Michalec, 2012; Phillips and Clarke, 2012; Michalec and Hafferty, 2013). These articles all called this informal education the "hidden curriculum," a term that in most contexts refers to the structural forces that maintain the oppressive status quo against the disadvantaged (e.g. Apple, 1971). Meanwhile, in the context of autism this term is often applied to the social "rules" and norms to which most people more readily adapt and which clinical programs explicitly aim to teach (Myles, Trautman and Schelvan, 2004). Autistics perform better when allowed our natural tendency against social conformity (Yafai, Verrier and Reidy, 2014), such as in pursuing intense interests (Wood, 2019), and just approaches would help autistics navigate society on our own terms.

Social deficit theories have empowered scientists to adopt dehumanising rhetoric and practices toward autistics. The theory of mind hypothesis argues that autistics are all "mindblind" (Baron-Cohen, 1997), lacking a mental life, such as failing to understanding that we and others have minds (Baron-Cohen, Leslie and Frith, 1985; see also Yergeau, 2013). The landmark theory of mind paper on autism (Baron-Cohen *et al.*, 1985) was titled after and takes the definition of a seminal corresponding paper on chimpanzees. It asked: "Does the autistic child have a theory of mind?" (answering in the negative; Baron-Cohen *et al.*, 1985), while an earlier study asked "Does the chimpanzee have a theory of mind?" (answering in the affirmative; Premack and Woodruff, 1978). Furthermore, Baron-Cohen (2000) quoted Whiten (1993) as asserting that "a theory of mind is one of the quintessential qualities that makes us human." Whiten (2013) has since summarised that researchers have extended theory of mind abilities to – beyond other primates – goats, dogs and crows.

Beyond the serious ethical problems, this area of inquiry has notable scientific holes. While research on verbal autistics has demonstrated their theory of mind (if delayed or more effortful), the theory of mind hypothesis still is tested on language-impaired autistic people (Colle, Baron-Cohen and Hill, 2007). It postulates – without solid evidence – that "classic" autistics may view their parents as vending machines, and degrades them as "morality-negative" (Baron-Cohen, 2011; see Cohen-Rottenberg, 2011). This fails to make the least dangerous assumption of presuming competence (Donnellan, 1984), and instead further endangers autistics by publicising and lending credibility to myths that autistics are uncaring and prone to violence.

Furthermore, non-autistics are said to "mindread," but no one can (McGuire and Michalko, 2011). Instead, people infer another's mental state in part from personal experience; consequently empathic accuracy decreases as the perceiver and actor have greater differences in life experience (Hodges, Kiel, Kramer, Veach, and Villanueva, 2010). Indeed, the main symbol of autism is a stigmatising puzzle piece (Gernsbacher, Raimond, Stevenson, Boston and Harp, 2018), so the privileged neuromajority is not expected to understand autistics, but autistics must understand them – yet we are characterised as lacking reciprocity (Milton, 2012). Meanwhile, people – typically but especially autistic people (Davidson and Smith, 2009; White and Remington, 2018) – ascribe humanness to animals and inanimate objects, even

activating their brain regions when they do so in the same way that they do for fellow people (Epley, Schroeder and Waytz, 2013). Epley *et al.* (2013) also examined both dehumanisation and autism, but unfortunately neglected to tie autism to its history of dehumanisation, instead attributing it to a lack of social motivation.

The social motivation theory has been embroiled in some of the most systematic abuse against autistic people ever, yet has experienced a resurgence recently. A foundational description of autism includes profound emotional and physical isolation, of children withdrawn into their own world who experience social contact as a disturbance (Kanner, 1943). Kanner extended this social disinterest to the parents, objectifying them as “refrigerators which did not defrost” (Kanner, 1949: p. 425), and describing autism as the result of their cold neglect and deprivation. A generation later, Lovaas based his applied behavioural analysis-based “therapy” on the related idea that autistic children were so deficient in social motivation that he needed to use aversives such as electric shock, screams and slaps on them so that they would learn (Grant, 1965), an approach still defended by his students as an artefact of the times (Koegel, 2011; Smith and Eikeseth, 2011). Ironically, proponents of the latest instalment of social motivation theory argue that ABA can only do so much because now it tends to emphasises rewards, and they contend that autistics have deficits in reward-processing (Kohls, Chevallier, Troiani and Schultz, 2012).

Social deficit theories are quite destructive. These theories need not be totally deficit-based to dehumanise: the empathising-systemising theory, a revision of the theory of mind hypothesis (Baron-Cohen, 2009), objectifies autistics as systematic, unempathetic machines (Haslam, 2006).

The belief that autistics cannot understand themselves or others denies their agency, exacerbating the professional-knows-best position of the medical model (at least lip service is given to empowerment in medicine for patients generally; Aujoulat, d’Hoore and Deccache, 2007). It has helped to lend the historically most powerful voice in autism advocacy to parents, who have all too often used that power to put forward the false rhetoric of autism as an epidemic, to ensure a focus on children (Stevenson, Harp and Gernsbacher, 2011), and to employ pity and fear in representations (e.g., again, the puzzle piece, symbolising brokenness and heart-wrenching mystery; Waltz, 2012). In this climate (especially before the neurodiversity movement matured to expose many autistic people to compelling alternatives), some self-advocates internalise the messages of social deficit theories, describing themselves as “aliens” on the “wrong planet” (Broderick and Ne’eman, 2008).

While autistic people tend to demonstrate lower levels of implicit and explicit prejudice than non-autistic people (Kapp *et al.*, 2019), autistics tend to experience dehumanising attitudes from neurotypicals (Cage, Di Monaco and Newell, 2018). For example, Lin and colleagues (2012) categorised an autism organisation as a “mental health” rather than “people” charity, even though they classified a cancer organisation as a “people charity,” then claimed autistic participants demonstrated a lack of social motivation despite their relatively high donations to “mental health” charities. Representation of autistic people as less than human has been seen to justify abuse and violence, including “mercy killings” (Waltz, 2008), such as murders of autistic people by their parents that have drawn coverage sympathetic to the killers from the mainstream media and from relative-led autism advocacy organisations (Gross, 2012).

Pathologisation

The scientific method combines with the medical model to reduce autistics into a collection of deficits, whereas autism may bring inherent strengths that can function as selective advantages, depending on various factors (Russell *et al.*, 2019). Gernsbacher, Dawson and Mottron (2006) provide several examples of scientists' systematic representation of autistic people's useful strengths as dysfunctions. These include the pathologisation of enhanced performance in visuospatial tasks such as the Block Design and Embedded Figures Tests as indicating "weak central coherence" (Shah and Frith, 1993) "due to a central deficiency in information processing" (Shah and Frith, 1983: p. 618) – since revised to signify an atypical cognitive style toward attention to detail rather than necessarily a deficit toward processing gestalt (Happé and Frith, 2006). This subserves a pattern of confirmation bias in which researchers hypothesise deficits and interpret results to fit their ideas, even when this stretches reason (Gross, 2011). For example, Gross reported a study that found that the non-autistic controls laughed both genuinely and strategically, while the autistic participants tended to laugh only genuinely, which the authors interpreted as a social deficit (Hudenko, Stone and Bachorowski, 2009). As Gross discussed, viewing laughing only out of happiness as a deficit can lead to therapy to teach autistic children to fake laughter, hardly a useful or ethical intervention goal. While this is quite literally a laughing matter, figuratively it is far from it.

Researchers have since published several other studies pathologising autistics' laughter. One study found that autistic boys enjoy "humorous" scenes with "slapstick" comedy as much as typically developing boys, but the scientists problematised this in stating that because they rated the scenes as not requiring social cognitive or language skills, a deficit may have emerged from watching more complex humour (Weiss *et al.*, 2013). Furthermore, the autistic participants rated the "non-humorous" scenes – "which comprised the same characters and similar environments as the humorous scenes" (p. 428) – as funnier, which the authors said "suggested that the autistic children did not discriminate non-humorous from humorous stimuli as sensitively as the typically developing children did" (p. 423). Instead, considering that what qualifies as humorous depends on subjective preferences, and that the paper omits examples of the scenes to let readers decide the humorousness for themselves, the authors exhibited *insensitive* discrimination. Similarly, the authors reported "in autistic children, the outwards displays of emotion did not match their reports of subjective amusement" (p. 423), showing little tolerance for individual differences in expressions of body language. Another study found that autistic adults did not rank humour as highly as a personal strength as the control group, and – while agreeing humour can make life more enjoyable – did not share the controls' regard for it as important to a meaningful, satisfying, engaging life (Samson and Antonelli, 2013). Is it any wonder that, after a lifetime of marginalisation for inherently harmless, objectively neutral differences like one's sense of humour, autistics would learn to devalue such differences? Not doing so would fall out of the tendencies of stigmatised group members (Crocker and Major, 1989), so a deficit-oriented researcher might find fault with autistics for supposedly lacking awareness of or concern for reputation, as in Izuma *et al.*'s (2011) pathologisation of autistics for not giving more to charity in the presence of an observer; see Gross (2011) and Cage *et al.* (2013) for critique of that donation study.

This line of research also provides examples where a single-minded focus on autistics misses intersections with other groups. As with many autism studies, Weiss *et al.* (2013) only included boys in their sample, yet generalised the title and reporting, neglecting any discussion of girls or gender. Yet women and girls tend not to enjoy slapstick (Jorgensen, Quist, Steck, Terry and Taylor, 2008) or dark humour (Aillaud and Piolat, 2012) as much as their male counterparts, and tend to show more susceptibility to the social desirability bias (Chung and Monroe, 2003) – in this case, evaluating movies as funnier when encouraged to laugh (with the opposite effect seen in males; Leventhal and Mace, 1970). Gender differences in humour become socialised from an early age (Groch, 1974), and the form they take has historically varied

with the norms of the context (Kotthoff, 2006). Thus, girls may have produced different results in Weiss *et al.* (2013) had they participated, but the process of socialisation on gender and other effects presents a barrier to scientists interested in uncovering the “essence” of autism. Despite the bias of autism research and diagnoses toward males, autistic people often defy norms of gender (e.g. Bejerot & Eriksson, 2014) and sex (Bejerot *et al.*, 2012), and illustrate the social construction of gender (Jack, 2012).

Reductionism

Scientific research on people uses principles from the physical sciences like reductionism, but people are more complex than the sum of our parts. Despite some attempts to specify parts of the brain as social in both the general and autistic populations, the brain is a complicated system where large-scale networks serve multiple, general and overlapping roles (Sallet, Mars and Rushworth, 2012). Some have proposed that the temporal parietal junction only subserves theory of mind (accurately inferring others’ thoughts, beliefs, etc.; Saxe & Wexler, 2005), yet it also is involved in attention-switching (Decety and Lamm, 2007) and out-of-body experiences (Blanke and Arzy, 2005). Similarly, some describe the medial prefrontal cortex as a social information processing centre (Baron-Cohen, 2011), but it also contributes to executive functioning (e.g., attention, flexibility, memory) in both autistics and neurotypicals (Gilbert, Bird, Brindley, Frith and Burgess, 2008). The fusiform face area got its name because many scientists think it is only specialised for faces, yet it assists with visual recognition of familiar categories such as birds and cars (Xu, 2005). It also is not necessarily deficient in autistics, as scientists commonly believe (Baron-Cohen *et al.*, 2000), but simply not activated as often during interaction because of reduced eye contact (Dalton *et al.*, 2005). Some autism social deficit theories highlight the amygdala (e.g., Baron-Cohen *et al.*, 2000), yet this region is the flight-or-fight centre of the brain and relates to anxiety in both autistics and neurotypicals (Kleinhans *et al.*, 2010). Indeed, the tendency for autistics to have enlarged and hyper-activated amygdalas contributes to the avoidance of eye contact out of a sense of threat (Dalton *et al.*, 2005; Kleinhans *et al.*, 2010). Moreover, the superior temporal sulcus helps people interpret emotions, goals and intentions from observing actions, which requires sensory integration rather than a specific social module (Shih *et al.*, 2011).

Scientists have likewise attempted to reduce autism to ultimately a social disorder, trying to demonstrate that deficits originate in social functioning by studying the infant siblings of autistic people, with the understanding that a large minority of them will also meet criteria for autism. This line of research, however, has revealed repetitive behaviours and other atypical movement, as well as behaviours related to atypical sensory processing, as the earliest indicators of autism (Gallagher and Varga, 2015; Gliga, Jones, Bedford, Charman and Johnson, 2014; Rogers, 2009; Sacrey, Bennett and Zwaigenbaum, 2015).

Motor challenges in autistic people often become evident in infancy (Bhat, Galloway and Landa, 2012; Flanagan, Landa, Bhat and Bauman, 2012; Gernsbacher, Sauer, Geye, Schweigert and Goldsmith, 2008; LeBarton and Iverson, 2013; Leonard, Elsabbagh, Hill and the BASIS team, 2014; Leonard, Bedford *et al.*, 2014; Mulligan and White, 2012) and may form the earliest sign of autism (Lemcke, Juul, Parner, Lauritsen and Thorsen, 2013). These infants particularly seem to have challenges transitioning between movements (Mulligan and White, 2012), such as postures (Nickel, Thatcher, Keller, Wozniak and Iverson, 2013), so for example their heads may lag when pulled to sit (Flanagan *et al.*, 2012). Such challenges with timing, fluidity and coordination of movement (particularly fine motor delays in this case) may explain why the infants tend not to explore objects in their environment as much as most babies (LeBarton and Iverson, 2013; Mulligan and White, 2012; Soska and Adolph, 2014), and motor skills tend to fall further behind their typically developing peers as toddlers (Lloyd, MacDonald and Lord, 2013). Bhat, Landa and Galloway (2011: p. 1123) describe how motor delays can cascade into social communication difficulties: “slow or uncoordinated head and arm movements may limit effective and

timely head turning, reaching, pointing, giving, and showing that are key components of initiation and response to the social overtures of others, also known as *joint attention*,” a skill known to promote language (Adamson, Bakeman, Deckner and Romski, 2008). Indeed, motor delays predict communication (Bhat *et al.*, 2012) and expressive language (LeBarton and Iverson, 2013), face processing and social challenges (Leonard *et al.*, 2013) in infant siblings of autistics; most relevant to Bhat *et al.*'s (2011) account, *fine* motor delays predicted expressive language in the study by LeBarton and Iverson (2013).

These infants may act in a relatively passive way, not initiating activity as often as most babies because of motor delays (Wan *et al.*, 2012), and their parents may react in well-meaning ways to engage their child that, while not causing autism, present interactive difficulties for both communicators (Wan *et al.*, 2013). Many become more directive with these apparently disengaged infants, raising the intensity and frequency of how they command and instruct their child. This often involves more talking, touching and baby talk than used with typically developing infants and toddlers, which may overwhelm the child and slow learning (Cohen *et al.*, 2013). Autistic children benefit more from responsive, relationship-based support, which especially helps those with greater language impairment (Kapp, 2018). Furthermore, sensory processing in autistics exemplifies not only the sensorimotor underpinnings of autism, but also how individual differences may function as a strength or “deficit” depending on the context. McCleery *et al.* (2007) reported that infant siblings of autistic children exhibited almost twice as much sensitivity to black-and-white visual contrast as the control group. Indeed, infant siblings later diagnosed with autism show an inclination to visual (geometric) patterns (Pierce, Conant, Hazin, Stoner and Desmond, 2011) and enhanced perceptual sensitivity in their first year of life, yet can present a flood of sensation that may help to explain why they go on to display more negative affect and reduced responsiveness to cuddling in their second year (Clifford *et al.*, 2013). Typically, infants have an innate capacity to distinguish between a great variety of sensory inputs, but their perception begins to specialise in the stimuli around them (for example, one's native language), a process called *perceptual narrowing* (Lewkowicz and Ghazanfar, 2011). Children develop heuristics, or mental shortcuts, to familiar inputs to the extent that their prior experiences and expectations tend to filter out small changes in the environment that they see but do not consciously process, a phenomenon called *inattention blindness* (Lewkowicz and Ghazanfar, 2011; Simons and Chabris, 1999). Yet autistic children are less susceptible to this effect (Swettenham *et al.*, 2014), perceiving the world based more on the realities around them (Brock, 2012), possessing enhanced perceptual capacity that may promote talent or cause overload (Remington, Swettenham and Lavie, 2012).

These sensory differences, while potentially useful for brilliant careers (Happé and Frith, 2009), for many autistics also contribute to challenging differences with relationships and speech. Ben-Sasson and colleagues (2013) found that parenting stress grew and family life became affected by increased sensitivities in autistic toddlers, perhaps because the young children need structured routines and soothing stimuli that the families have not learned to accommodate. Moreover, autistic people, while often highly attuned to the auditory and especially visual senses when processed independently (Mottron, Dawson, Soulières, Hubert and Burack, 2006), tend to have difficulty with audiovisual integration (Stevenson *et al.*, 2014). Typically developing infants begin to show some mastery with this combination (Kushnerenko, Teinonen, Volein and Csibra, 2008), and after attending to the mouth to learn speech by lip-reading (Kubicek *et al.*, 2013; Tomalski *et al.*, 2013), their perceptual competence with speech enables them to turn their attention toward the eyes to access social cues (Kushnerenko *et al.*, 2013; Lewkowicz and Hansen-Tift, 2012). Conversely, infant siblings struggle with audiovisual speech integration (Guiraud *et al.*, 2012), and those later diagnosed with autism look less at the eyes and other inner facial features when people speak (Shic, Macari and Chawarska, 2014). Instead, looking at speaking mouths shows benefit for communicative competence from infancy through adulthood in autistic people (Elsabbagh *et al.*, 2014; Falck-Ytter, Fernell, Gillberg and Hofsten, 2010; Klin, Jones, Schultz, Volkmar and Cohen, 2002; Norbury *et al.*, 2009; Tenenbaum, Amso, Abar and Sheinkopf, 2014), because of the audiovisual

synchrony between speech sounds and lip motion (Klin, Lin, Gorrindo, Ramsay and Jones, 2009). Therefore, difficulties with audiovisual integration may help to explain why *high* eye contact in the first months of life predicts autism and developmental delays, and infants later diagnosed toward autism tend to make less eye contact as their typically developing peers increase their social attention to others' eyes (Kapp *et al.*, 2019). At least, these findings apply to the normative West, which has placed reduced eye contact in the autism's diagnostic criteria, whereas other cultures disapprove of eye contact between children and elders (Norbury and Sparks, 2013).

Essentialism

Although autistic people develop through a complex transaction of biologically-based domain-general underpinnings amid diverse cultural contexts, the drive of the medical model to mechanistically reduce autism to a social disorder results in scientific efforts to discover the essence of autism, as a discrete entity with specific causation to prevent or cure (Pellicano and Stears, 2011). The quest to prevent autism has led scientists to implicitly acknowledge the role of the social environment in contributing to the disablement of autistic people, through infant sibling studies that threaten to repeat autism's history by pathologising loving families. Autism is framed as so different and burdensome as to justify treatment studies on "presymptomatic" infants that seek to change their "developmental trajectory" (Webb, Jones, Kelly and Dawson, 2014), even though most will never meet criteria for autism (Ozonoff *et al.*, 2011). Therefore, programmes are designed to be general enough to be seen as helpful to any child. Ironically, this means parent training that loosely resembles the idea that parents are not doing enough for their child, even though the refrigerator mother hypothesis mobilised parents to steer autism research (Langan, 2011). This research also raises questions about *how* different scientists regard autism, but it seems that many think that sometime before age three, by which time toddlers become "outcomes" as autistic or not (as though this will determine functioning and quality of life; Messinger *et al.*, 2013), the brain has enough plasticity that the babies could avoid the essentialised fate of autism (Dawson, 2008).

Such attempts to constrain autism overlook the sheer complexity of socialisation. There is a growing recognition that social research in psychology and neuroscience often poorly measures what it attempts to study experimentally (Ames and Fletcher-Watson, 2010; Dziobek, 2012; Risko, Laidlaw, Freeth, Foulsham and Kingstone, 2012; Zaki and Ochsner, 2012). Social interaction involves (often live) contingencies with people, many times poorly represented in research by photos, avatars and other unrealistic substitutes (Ames and Fletcher-Watson, 2010; Dziobek, 2012; Risko *et al.*, 2012; Zaki and Ochsner, 2012). Sociality also entails long-term relationships, which experimental studies often fail to capture (Dunbar, 2014). People are affected by their social ecology, including culture and economic, political and religious systems (Oishi, Kesebir and Snyder, 2009). As Lord, the first author of the two autism diagnostic instruments considered the gold standard in the field, said, "...as somebody who's been trying to measure social behaviour for a long time, I don't think there's even one thing that is social behaviour. I think social behaviour is actually many different things. We do much less well quantifying social behaviour than we do lots of other things, even repetitive behaviour" (Insel, Lord and Tager-Flusberg, 2013). This acknowledges that while she has reduced autism's social and communication criteria to one statistical factor (Gotham, Risi, Pickles and Lord, 2007), autism may encompass more than we can understand now (Lord and Jones, 2012).

Clinical experiments and tools should account for the fact that autistics and others internalise their experiences and environments. In general, poverty, discrimination and stratified inequality often give rise to chronic stress (Goodman, 2013), which can cycle by impairing the ability to cope with stressful events

(Kim *et al.*, 2013), among other effects (Boyce, Sokolowski and Robinson, 2012; McEwen, 2013). Social norms vary by culture and context (Norbury and Sparks, 2013); yet most research is based on middle-class and wealthier people in the West, especially the US, the norms and traits of which clash with those of other peoples around the world (Henrich, Heine and Norenzayan, 2010). Autism diagnostic instruments' sensitivity to intercultural differences needs improvement (Lord, 2010), and people in low-income countries need access to clinical resources and social services (DeWeerd, 2013). Even in high-income countries like the US and UK, the diagnostic criteria may at times conflate core differences with coping mechanisms in response to stress. For example, repetitive motor movements, listed as a core symptom of autism, help with emotional self-regulation (Kapp *et al.*, 2019). Tightly followed routines, also listed as a core symptom, help autistics cope with executive functioning challenges (see Boyd, McBee, Holtzclaw, Baranek and Bodfish, 2009; Russell *et al.*, 2019). Furthermore, as with other stigmatised groups (Steele, 1997), stereotypes and mistreatment often lower self-concept and performance, and many autistics attempt to “pass” for “neurotypical” (Cage and Troxell-Whitman, 2019). Yet some cultures have less ableism and more acceptance, so the behaviours displayed and the views on them vary (Dyches, Wilder, Sudweeks, Obiakor and Algozzine, 2004).

Unsurprisingly, since autism is diagnosed as mainly a social deficit, and social matters have so many variables, autistic people exhibit high within-group diversity. Uneven skills (Jones *et al.*, 2009) and high intra-individual variability (an individual having different performances of or responses to the same task or activity; Geurts *et al.*, 2008; Haigh, 2018) are typical of autism. There is no single cause of autism (Happé, Ronald and Plomin, 2006) or dysfunctional neural or cognitive module (Gernsbacher and Frymiare, 2005). No distinctive universals apply to autistics (Brock, 2014). No valid subtypes or sub-diagnoses have been found, even though autism is considered to have relatively good reliability as a diagnosis (Mandy, 2018), in part because autistic people often alternate between the boundaries of subtype profiles as they gain skills or their contexts change (Wing, 2002; Wing, Gould and Gillberg, 2011). Thus, there have been numerous attempts to break up autism, whether by genes (Jeste and Geschwind, 2014), neurology (Pelphrey, Shultz, Hudac and Wyk, 2011), cognition (Happé and Ronald, 2008), behaviour (Mandy and Skuse, 2008), and so on; some refer to the “autisms” (Coleman and Gillberg, 2012).

This diversity does not negate the possibility that autism is a coherent syndrome (Rutter, 2014). Critics rightly point out that autism's diagnostic criteria have changed over time (Verhoeff, 2013), and that the criteria in part reflect what society sees at the time as abnormal (Verhoeff, 2012) – not unlike diagnostic debates of other disabilities (see Phillips *et al.*, 2012). Yet human development is fluid; even most well-established disabilities generally have significant heterogeneity, but the whole may be greater than and different from the sum of the parts (Rutter, 2014).

This fluidity also applies to the boundaries of autism; who is “disabled” or not by diagnostic conventions is often unclear. Most who meet criteria for autism lack a diagnosis (Brugha *et al.*, 2011; Kim *et al.*, 2011; White, Ollendick and Bray, 2011). For that matter, participants in the control groups of autism studies are not necessarily neurotypical or even non-autistic; one study that took the step of testing the control group for autism found that a whopping seven out of 35 met criteria (Henderson *et al.*, 2015). Moreover, those who have high traits of autism have similar genetic tendencies to those who apparently are autistic (Lundström *et al.*, 2012; Robinson *et al.*, 2011). Yet being considered “disabled” currently

requires clinical impairment, even though impairment can be affected by support and compensation (APA, 2013).

Conclusion

This analysis supports the need to move to an alternative classification system that recognises all features of autism, including those that can function as strengths, and the critical role support can play in autistic people's functioning (Chown and Leatherland, 2018; Kapp and Ne'eman, 2019). This could help autistic people understand themselves in a more accurate, holistic way, and help enable autistic people to receive appropriate support by recognising that an individual may function well in facilitative environments. A research version of the autism diagnosis that does not require functional impairment may help to test and develop this idea, to improve understanding of autistic people with comparable traits as others and who attain higher achievements (Rutter, 2011; Üstün and Kennedy, 2009; see Grandin, 2012; Perner, 2012; Santomauro, 2011).

Furthermore, participatory research that includes autistic people as equal partners or leaders – amid other means of social inclusion – helps to empower autistic people and may help generate the knowledge for such systems and social change (see Nicolaidis *et al.*, 2019). Autistic people have helped to develop useful theories for autism such as monotropism (Murray, Lesser and Lawson, 2005) and the enhanced perceptual functioning model (Mottron *et al.*, 2006) to explain how autistic people may think and learn, and the double empathy problem to explain the difficulties autistic and non-autistic people share in understanding one another (Milton, 2012; see also Heasman and Gillespie, 2018). We have also helped to extend existing theories such as that on sensory-movement differences (Kapp, 2013), an original account that comfortably sits in a special issue on autism and neurodiversity (Donnellan, Hill and Leary, 2010). Autistic people have demonstrated the critical, scientifically informed expertise many of us have on autism (Gillespie-Lynch *et al.*, 2017), in addition to the expertise any autistic person may have through lived expertise (Milton, 2014).

The nuanced account of autism in this analysis as having a foundation of sensorimotor and general (not socially specific) traits lends itself to many evidence-based support strategies and tools. Accepting autism improves others' ability to recognise and respond to autistic people's needs and interests, improving the relationship between autistic people and communication partners (see Kapp, 2018). Similarly, responsive rather than directive caregiving (e.g. imitation of an autistic child and following the child's lead: Gulsrud, Hellemann, Shire and Kasari, 2016; Nadel, 2015; see also Kapp, 2018) helps autistic people's development and relationships. Other strategies include not only encouraging lip-reading for learning to communicate (e.g. Tenenbaum *et al.* 2014), but also slowing down the presentation of facial expressions (Gepner, Deruelle and Grynfeldt, 2001; Lainé, Rauzy, Tardif and Gepner, 2011; Tardif, Lainé, Rodriguez and Gepner, 2007), using colour filters to enhance visual performance for both non-social things like written words and objects (Ludlow, Wilkins and Heaton, 2006; Ludlow, Wilkins and Heaton, 2008) and social cues (Ludlow, Taylor-Whiffen and Wilkins, 2012; Whitaker, Jones, Wilkins and Roberson, 2016), incorporation of song and music (Bhatara, Quintin, Fombonnen and Levitin, 2013; DePape, Hall, Tillmann and Trainor, 2012; Lai, Pantazatos, Schneider and Hirsch, 2012; Sharda, Midha, Malik, Mukerji and Singh, 2015), and augmentative and alternative communication methods like symbol-based apps on tablets (Almirall *et al.*, 2016; Kasari *et al.*, 2014). Sensorimotor differences can give rise to a variety of behaviors like lack of eye contact, and repetitive movements like rocking and hand-flapping,

that may serve as coping mechanisms that help individuals to self-regulate (e.g. Kapp *et al.*, 2019). Many people and theories make negative social interpretations about these behaviours, even though neurotypical people engage in these and other repetitive movements (Thelen, 1981, 1979; Jaswal and Ahktar, 2019). The growing neurodiversity movement likely helps people to recognise complexities like autistic people's differences from and commonalities shared with the rest of humanity, as it helps people view autism as a positive identity *and* disability that requires acceptance and support (Kapp, Gillespie-Lynch, Sherman and Hutman, 2013).

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