Neurotypicality: Has It Lost Its Meaning and Is It Leading to Misclassification?

A Response form ION Global to Autism Spectrum Disorder: Has It Lost Its Meaning and Is It Leading to Misdiagnosis?

Abstract

The increasing heterogeneity encompassed by the diagnosis of autism has prompted important questions about whether autism remains a sufficiently coherent category for research and clinical practice. This paper applies equivalent scrutiny to the category against which autistic people are frequently compared: the “neurotypical” population.

Who, precisely, is neurotypical? Does the category include a person with intellectual disability, epilepsy, cerebral palsy, depression, anxiety, acquired brain injury or substantial physical disability? What about people with unusually high or low cognitive abilities, atypical sensory processing, significant differences in executive functioning or social cognition, but no recognised neurodevelopmental diagnosis? If these individuals remain neurotypical, how much neurological, cognitive, psychological and functional variation can the category encompass before its own explanatory meaning becomes uncertain?

The question becomes particularly revealing when applied to descriptions such as “profound autism”. If a non-autistic person has profound intellectual disability, epilepsy, minimal spoken language and substantial physical support needs, we would not describe that person as “profoundly neurotypical”. Why, then, should equivalent characteristics necessarily make an autistic person more profoundly autistic?

This is not merely a semantic problem. When autism becomes an umbrella explanation for intellectual, psychological, behavioural or physical differences, diagnostic overshadowing can result. Symptoms requiring independent investigation may instead be attributed to autism, potentially delaying appropriate diagnosis and treatment.

There is also an epistemological question. Autism has historically been researched predominantly by non-autistic researchers, with autistic people positioned as the population being observed and “neurotypical” people frequently positioned as the reference population. What happens when the research lens is reversed? What might neurominority researchers discover by studying neurotypicality itself?

This paper proposes that human variation among researchers is not merely a question of representation. Different neurological, cognitive and experiential perspectives may generate different questions, perceive different patterns and challenge different assumptions. When those perspectives are able to interact meaningfully, they expand the collective intelligence available to scientific inquiry.

Rather than asking only whether autism has become too heterogeneous, we should therefore examine the conceptual and epistemological models against which autism is being judged.

The problem may not be that autism encompasses too much human variation. It may be that we continue to assume the existence of a neurologically homogeneous human norm that has never adequately been demonstrated.

Introduction

Recent debate has questioned whether the contemporary autism spectrum encompasses people with such different characteristics, abilities, support requirements and co-occurring conditions that the category risks losing clinical or scientific specificity. (Frith, 2026; Lord et al., 2022).

This is a legitimate scientific question. Categories used in science and medicine require sufficient explanatory, predictive or practical value to remain useful.

There is, however, an important asymmetry in how this question is usually approached.

The heterogeneity of autistic people is examined extensively. Differences in intellectual ability, language, adaptive functioning, mental and physical health, genetics, sensory processing, age of diagnosis and support requirements are analysed in determining whether autistic people properly belong within one category.(Frith, 2026; Lord et al., 2022).

The corresponding heterogeneity of the population described as “neurotypical” receives considerably less scrutiny.

Yet autism does not exist in conceptual isolation. Much autism research, clinical reasoning and public discourse depends upon an implicit comparison between autistic people and people assumed to represent typical neurological development.
If heterogeneity threatens the validity of one category, equivalent heterogeneity should raise questions about the validity of the other.

Who is actually neurotypical?

Consider a hypothetical group of 1,000 people who do not meet diagnostic criteria for autism.

- Among them may be people with depression, anxiety disorders, obsessive-compulsive disorder, eating disorders and histories of psychological trauma.
- Some may have epilepsy, cerebral palsy, acquired brain injuries or neurodegenerative disease.
- Some may have intellectual disabilities. Others may have exceptionally high measured intelligence.
- Some will experience substantial sensory sensitivities without meeting criteria for autism.
- Some will have unusually strong or weak executive functioning, working memory, emotional regulation, social cognition, attentional control or language processing.
- Others may have neurological or cognitive characteristics for which no diagnostic category currently exists.

Are all these people neurotypical?

If they are, the category encompasses enormous neurological, cognitive and behavioural heterogeneity.

If they are not, where precisely should its boundaries be drawn?
The purpose of asking this question is not to suggest that intellectual disability, physical disability, mental illness or neurological disease necessarily changes a person's neurotype. Quite the opposite.

The question exposes how poorly specified the category of neurotypicality itself can be. What precisely must be "typical" for somebody to qualify as neurotypical?
And typical relative to what?

The "profoundly neurotypical" test

The conceptual problem becomes particularly visible when we consider the language of profound autism. (Lord et al., 2022; Pukki et al., 2022).

Consider two people, both with profound intellectual disability, epilepsy, minimal spoken language and substantial physical support needs.
One meets diagnostic criteria for autism. The other does not.

The first may be described as having "profound autism", with intellectual, communicative and physical support needs contributing to the perception of the severity of their autism.

But how should we describe the second person?

Are they "profoundly neurotypical"?

The expression sounds immediately strange.

We intuitively recognise that profound intellectual disability, epilepsy, limited speech and physical disability do not constitute a more extreme form of neurotypicality.

Why, then, should the same characteristics necessarily constitute a more extreme form of autism?

This is more than a question of terminology. It concerns what our classifications are intended to classify.

Autism describes a particular configuration of characteristics. Intellectual disability describes another dimension of human variation. Epilepsy is a neurological condition.

Physical disability introduces another dimension. Communication ability and support requirements introduce still others.

These characteristics can interact profoundly in an individual's life. Interaction, however, does not make them conceptually interchangeable.

If we would not classify a non-autistic person's intellectual, physical or communication disability as a degree of neurotypicality, we should question whether the same disabilities in an autistic person should be classified as a degree of autism.(Pukki et al., 2022; Eicher, Ne'eman & Quackenbush, 2026, preprint).

Perhaps the difficulty is not that the autism spectrum has become too broad.

Perhaps we are asking a single classification to carry information about too many different dimensions of a human being.

Autism should not become the totality of the phenotype

This distinction matters because the term autism can gradually acquire explanatory responsibility for almost every characteristic of an autistic person.

An autistic person's communication difficulties can become interpreted as autism; their intellectual disability can become absorbed into autism; their sensory experiences, distress and unusual behaviours can all become explained through autism; and even physical symptoms may be interpreted through the same lens. The result is that distinct experiences, conditions and support needs risk being collapsed into a single diagnostic explanation rather than being recognised and understood in their own right..

Yet we do not expect neurotypicality to explain the totality of a non-autistic person.

This produces a fundamental asymmetry.

The minority neurotype risks becoming an explanation for the person, while the majority neurotype remains merely one largely invisible aspect of the person.

A more precise conceptual architecture would separate dimensions that are currently too easily collapsed together.

Autism may tell us something about a person's neurotype, while intellectual disability, physical disability, communication profile, mental and physical health conditions, and support requirements each tell us something different about that person.
These dimensions interact, but none should automatically become a proxy for all the others.

When autism becomes the explanation: diagnostic overshadowing and patient safety (NICE, 2014; NICE, 2021).

The consequences of this conceptual imprecision are not confined to scientific classification or language.

They can become matters of patient safety.

Diagnostic overshadowing occurs when symptoms or characteristics are attributed to an existing diagnosis rather than appropriately investigated as potentially arising from another condition.

For autistic people, this presents a particular risk.

A change in behaviour may indicate pain, reduced communication may reflect illness, distress may have a physiological cause, and a new sensory experience may warrant neurological investigation (NICE, 2014; NICE, 2021).

Changes in eating, sleeping, movement or emotional regulation may have causes requiring independent clinical assessment.

An autistic person's inability to describe symptoms in the manner expected by healthcare professionals does not make those symptoms manifestations of autism. (NICE, 2014; NICE, 2021).

NHS England specifically recognises diagnostic overshadowing as a risk for autistic people. NICE guidance similarly requires clinicians to consider coexisting physical, neurological, neurodevelopmental and mental-health conditions alongside autism.

The underlying principle is important: the existence of an autism diagnosis should not terminate clinical curiosity about other explanations.

Consider again two people presenting with the same new physical symptoms.

One is autistic. One is not.

For the non-autistic person, clinicians must search for an explanation.

For the autistic person, an explanation may appear already to be available.

That asymmetry can be dangerous.

Autism should therefore neither exclude other diagnoses nor absorb them.
The distinction between autism and co-occurring conditions is not merely taxonomic precision. In some circumstances, maintaining that distinction may be essential to appropriate medical investigation and treatment. (NICE, 2014; NICE, 2021).

Neurotypicality as a residual category

There is a further asymmetry.

Autism has diagnostic criteria.

Neurotypicality does not.

There is no internationally recognised diagnostic assessment through which an individual can be positively identified as neurotypical.

In research, the category may effectively mean that a participant does not have autism, or does not have one of a specified number of excluded diagnoses.

This creates an important epistemological difference.
Autistic people must demonstrate sufficient characteristics to enter their category.

Neurotypical people may enter theirs through the absence of sufficient evidence to remove them from it.

Neurotypicality can consequently become a residual category: everyone remaining after particular recognised forms of neurological difference have been excluded.

But absence of diagnosis is not evidence of neurological typicality.

Some individuals may have unrecognised neurodevelopmental differences. Others may possess significant neurological variation that falls below current diagnostic thresholds. Still others may differ substantially from population averages without experiencing impairment and therefore have no reason ever to encounter diagnostic services.

The supposedly neurotypical population may therefore contain extensive human variation hidden by the classification itself.

The problem of the modelled human

Underlying this debate is a broader conceptual question.

When we describe someone as neurologically “typical”, typical relative to what?

Human beings vary across numerous dimensions: attention, sensory processing, sociability, communication, emotional regulation, executive functioning, memory, pattern recognition, motor coordination and many others.

Societies nevertheless construct ranges of functioning regarded as sufficiently typical.

People whose characteristics fall within those ranges generally pass unnoticed.

People whose characteristics fall sufficiently outside them may acquire diagnostic classifications.

The resulting categories can then easily be mistaken for natural boundaries between fundamentally different kinds of humans.

But another interpretation is possible.

What we call neurotypicality may represent the range of human neurological variation that currently fits sufficiently comfortably within our model of the expected human.

Autism and other minority neurotypes may represent recurring configurations of human variation that fall sufficiently outside those expected ranges to become socially or clinically visible.(Pellicano & den Houting, 2022).

From this perspective, diagnostic categories can remain extremely valuable. They can identify patterns, facilitate research, provide access to support and enable people with shared experiences to understand themselves and one another.

But the usefulness of a category does not establish the existence of a homogeneous neurological population on either side of its boundary. (Pellicano & den Houting, 2022).

The asymmetry of heterogeneity

This leads to an important methodological issue.

When substantial differences are identified among autistic people, those differences are frequently interpreted as evidence of heterogeneity within autism.

When substantial differences occur among non-autistic people, they are more readily understood as ordinary individual variation.

The same phenomenon can therefore acquire different conceptual significance depending upon which side of the diagnostic boundary it occurs.

An autistic person with unusual sensory processing contributes to the heterogeneity of autism.

A non-autistic person with unusual sensory processing may simply be an individual.

An autistic person with exceptionally high intelligence and another with profound intellectual disability may be presented as evidence that the autism spectrum encompasses people who are too different to belong to a meaningful single category.

Yet the non-autistic population also encompasses people across the full range of intellectual ability without this being considered evidence that neurotypicality has become scientifically meaningless.

Why should diversity within the minority category require taxonomic explanation while diversity within the majority category is accepted as human variation?

Human variation does not make autism meaningless

Recognising that autistic characteristics occur within the wider range of human variation does not mean that everybody is “a little autistic”, nor does it make autism an arbitrary classification.

Many human characteristics are distributed across populations while particular configurations of those characteristics remain meaningful. Headache provides a simple analogy. Most people experience headaches at some point in their lives. Migraine is also associated with headache, but it is not adequately described as simply having more headache. It involves a recognisable configuration of characteristics that may include particular forms of pain, sensory sensitivity, nausea, cognitive effects and other neurological phenomena. The fact that individual characteristics associated with migraine can also occur among people who do not experience migraine does not invalidate migraine as a meaningful phenomenon.

The same distinction matters when considering autism. Sensory sensitivity, difficulties with executive functioning, intense focus, repetitive behaviour, social differences, communication differences or a preference for predictability are not necessarily unique to autistic people. Non-autistic people may experience any of these characteristics.

That does not establish that autism is simply an arbitrary point on a continuum of otherwise identical human experience.

The relevant question is whether particular characteristics recur in configurations, degrees and interactions that produce a sufficiently recognisable neurological and lived experience for the concept of autism to retain explanatory and practical value. Human characteristics can be continuous while configurations of those characteristics remain meaningfully distinguishable.

This distinction is particularly important when autistic people describe their own experience. First-person experience should not be treated as scientifically infallible, but neither should it be treated as scientifically irrelevant. Autism research is attempting, among other things, to understand a phenomenon experienced by autistic people. Their observations therefore constitute part of the evidence that theories of autism must be capable of explaining.

A theory that struggles to accommodate substantial and recurring descriptions of autistic experience has an explanatory problem of its own.

This does not place lived experience above other forms of evidence. It requires something more scientifically demanding: that evidence from genetics, neuroscience, psychology, clinical observation, behaviour and lived experience be considered together when evaluating what autism is and what explanatory value the category retains.

The recognition of human neurological variation therefore need not invalidate autism. It may instead allow us to ask a more precise question: what particular configurations of human neurological variation does autism identify, and what can autistic people themselves tell us about those configurations that observation from outside cannot?

Are we asking the wrong question?

The question “Has autism lost its meaning?” may therefore begin one level too late.

A preceding question is required:

What model of human neurological variation makes autism a coherent category in the first place?

If autism is being evaluated against an assumed neurotypical norm, that norm requires the same scientific scrutiny as the category being compared with it. (Pellicano & den Houting, 2022).

We might discover that neither “autistic” nor “neurotypical” describes a homogeneous neurological population.

That would not necessarily make autism meaningless.

It would suggest instead that categorical diagnosis and human variation are performing different functions.

Clinical categories identify recurring configurations of characteristics associated with particular experiences, needs or outcomes.

Human variation describes the wider biological reality within which those configurations occur.

Confusing the two creates an expectation that diagnostic categories should correspond to discrete kinds of humans, they may not.

From neurotypicality to majority and minority neurotypes

One alternative is to move away from an implicit binary between autistic and neurotypical humans and towards a model based upon human neurological variation.
Within such a model, autism could be understood as a minority neurotype: a recurring configuration of human neurological characteristics occurring in a minority of the population.(Pellicano & den Houting, 2022).

Other neurodevelopmental configurations could similarly constitute minority neurotypes.

The remainder of the population would not need to be presumed neurologically identical.

People whose neurological characteristics fall sufficiently within the currently modelled ranges of human functioning could instead be understood as belonging to majority neurotypes.

This distinction matters.

“Neurotypical” risks implying the existence of neurological typicality.

“Majority neurotype” makes a more limited claim: that a person’s neurological configuration falls sufficiently within ranges represented by the majority population and accommodated by systems constructed around that population.

Neither minority nor majority neurotypes need to be internally homogeneous. Variation should be expected within both.

Implications for autism research

This reframing has practical consequences.

Researchers comparing autistic and non-autistic populations should describe precisely what their comparison group represents rather than assuming neurological typicality.

If participants have only been screened for autism, “non-autistic comparison group” may be scientifically more accurate than “neurotypical controls”.

Where neurological, psychiatric, intellectual, physical, sensory or cognitive characteristics are relevant to the research question, these should be considered in both autistic and non-autistic populations.

Most importantly, variation should be investigated symmetrically.
If intellectual ability, sensory processing, psychiatric diagnoses, communication characteristics or executive functioning are considered important sources of heterogeneity among autistic participants, researchers should ask whether equivalent variation within comparison populations also influences their findings.

Otherwise, characteristics of the minority population risk being interpreted as evidence of diagnostic instability while comparable characteristics of the majority population disappear statistically into an assumed norm.

This is not merely a matter of terminology.

The way comparison populations are conceptualised affects the questions research asks, the variables it measures and the conclusions it draws. (Pellicano & den Houting, 2022).

Reversing the research lens: neurotypicality as an object of study

There is another asymmetry within the production of knowledge itself.

For much of the history of autism research, non-autistic researchers have observed autistic people, defined the questions to be investigated, developed the instruments through which autistic characteristics are measured, interpreted autistic behaviour and compared autistic participants with populations described as neurotypical. (Bottema-Beutel et al., 2021; Pellicano & den Houting, 2022).

The direction of observation has overwhelmingly been from the presumed neurological majority towards the neurological minority.

What might we learn if we reversed the research lens?

There is no scientific reason why neurotypicality should remain principally the unexamined reference population against which minority neurotypes are studied.

Neurominority researchers could investigate neurotypicality as a phenomenon in its own right.

Such research might begin with surprisingly fundamental questions.

- What is neurotypicality?
- Which characteristics define it?
- How heterogeneous are people classified as neurotypical?
- How stable are those characteristics across cultures, environments and stages of life?
- What are the characteristic strengths and challenges associated with majority neurotypes?
- How do people of majority neurotypes communicate with one another and with people
- whose neurotypes differ from theirs?
- Are behaviours regarded as socially intuitive among neurotypical people universally intuitive, or are some learned conventions that become largely invisible because they are shared by the majority?
- How effectively do neurotypical people interpret people whose communication and sensory experiences differ substantially from their own?
- And how much of what has historically been described as impairment within autism represents an intrinsic characteristic of the autistic individual, rather than difficulty occurring at an interface between different neurotypes?

The purpose would not be to pathologise neurotypical people.

Nor would it be to claim that neurominority researchers possess inherently superior insight into majority neurotypes.

It would be to apply the principle of epistemic symmetry.

If majority-neurotype researchers can study minority neurotypes, minority-neurotype researchers can study majority neurotypes.

Different neurological perspectives may generate different questions, notice different patterns and challenge different assumptions.

Research associated with the double empathy problem provides an instructive example.

Social difficulties between autistic and non-autistic people need not always be understood as deficits located exclusively within autistic individuals. Research has increasingly examined what occurs between people of different neurotypes rather than locating the entire explanation within one person. (Milton, 2012; Crompton et al., 2020a; Crompton et al., 2020b).

Instead of asking only, “What is different about this autistic person?”, research can also ask, “What is happening at the interface between these two differently functioning human beings?”

The implications extend beyond autism.

Researchers inevitably bring perceptual and conceptual assumptions to the phenomena they study. Some assumptions may be particularly difficult to perceive when they are shared by most members of the research community.

A majority neurotype may therefore possess an unusual scientific characteristic: some of its features may become invisible precisely because they are regarded as normal.

Neurominority researchers may help make some of those assumptions visible.

This suggests a broader principle:

No population should exist solely as the observer while another exists solely as the observed.

Autistic people should participate meaningfully in autism research, including in determining research priorities, questions, methodologies and interpretation. (Fletcher-Watson et al., 2019; Pellicano & den Houting, 2022).

But epistemic equality requires something further.

Neurominority people should also be researchers of human beings generally.

Human variation and collective intelligence in research

There is a broader scientific issue underlying this discussion.

Research is itself a human system.

The questions researchers choose to ask, the phenomena they notice, the hypotheses they construct, the methods they develop and the interpretations they make are influenced by the perspectives through which they encounter the world. (Hong & Page, 2004).

Human variation within research therefore has significance beyond representation. Different neurological, cognitive and experiential perspectives may make different aspects of the same phenomenon visible.

They may generate different questions, detect different patterns, challenge different assumptions and offer alternative interpretations of the same evidence. (Hong & Page, 2004).

No individual researcher, regardless of expertise, can perceive a complex human phenomenon from every possible perspective.

The purpose of bringing human variation into research is therefore not to suggest that one neurotype possesses superior knowledge to another.
It is to increase the range of human intelligence from which the research process can draw.

Variation alone, however, is insufficient.

A research team can contain substantial human variation while continuing to reproduce a narrow set of assumptions.

If researchers with different perspectives are present but the research system privileges one established way of framing questions, interpreting evidence or determining what constitutes legitimate knowledge, the additional intelligence available to the system may never influence its conclusions.

Human variation needs effective interfaces through which different perspectives can interact, challenge one another and influence the direction of inquiry. It is through that interaction that variation can contribute to collective intelligence. (Hong & Page, 2004).

This has particular relevance to autism research.

If autism is predominantly conceptualised, investigated and interpreted from within majority-neurotype perspectives, some assumptions about autism may be repeatedly examined while assumptions about neurotypicality remain largely invisible.

Reversing the research lens is therefore not an argument for replacing neurotypical researchers with neurominority researchers.

It is an argument for widening the collective intelligence through which human neurological variation is investigated.

Neurominority researchers studying neurotypicality may ask questions that have historically received little attention. Majority-neurotype researchers may, in turn, perceive aspects of minority neurotypes that are less visible from within those experiences.

Researchers with different cultural, intellectual, physical, social and experiential perspectives may reveal still other dimensions.

No single perspective is sufficient. The interaction between them is where additional scientific insight may emerge.

This suggests a broader proposition:

Human variation within research is not simply a question of who is represented. It influences the range of human intelligence available to the scientific process.
If research does not draw upon human variation, it restricts the collective intelligence from which it can learn. This raises a question that deserves considerably greater attention within research itself.

We routinely examine diversity and heterogeneity within the populations being researched.

How often do we examine the diversity and heterogeneity of the people doing the researching?

The observer is part of the research system.

If the observer population is comparatively narrow, the questions asked, assumptions challenged and patterns perceived may also be narrower than they would be if a wider range of human variation participated meaningfully in the production of knowledge.

This is particularly important when the subject of research is human variation itself.

Conclusion

The heterogeneity of autism deserves serious scientific examination.

But scientific consistency requires us to subject the category against which autism is compared to equivalent scrutiny.

If autistic people differ substantially from one another, this does not by itself demonstrate that autism has become meaningless.

Human beings differ substantially from one another.

The relevant scientific question is whether autism identifies a sufficiently recurring and consequential configuration of human characteristics to provide explanatory, predictive, clinical or practical value.

That question cannot be answered simply by comparing a heterogeneous autistic population with an assumed homogeneous neurotypical population.

We first need to establish whether such a homogeneous population exists.

We should also be cautious about asking autism to communicate information that properly belongs to other dimensions of the individual.

Intellectual disability is not necessarily a degree of autism.

Physical disability is not necessarily a degree of autism.

Communication ability is not necessarily a degree of autism.

Co-occurring medical conditions are not necessarily manifestations of autism.

Support needs are not necessarily a measure of autism.

If we would not describe a non-autistic person with profound intellectual and physical disabilities as “profoundly neurotypical”, we should examine carefully what we mean when equivalent characteristics contribute to describing an autistic person as “profoundly autistic”.

The issue is not merely one of language.

When autism absorbs characteristics and symptoms that require independent understanding, diagnostic overshadowing can follow. The consequences can include delayed investigation, inappropriate treatment and potentially serious harm. (NICE, 2014; NICE, 2021).

But there is another form of overshadowing worth considering. By concentrating scientific attention on the heterogeneity of autistic people, we may have allowed the heterogeneity of the presumed neurotypical population to remain comparatively unexamined.

And by concentrating research on what makes minority neurotypes different, we may have left the neurological majority functioning as an assumed reference point rather than an object of scientific inquiry in its own right.

Reversing the research lens offers one way of testing those assumptions. Neurominority researchers studying majority neurotypes would not make science inherently more objective, nor should neurological identity be treated as conferring particular scientific authority.

Rather, researchers approaching the same phenomena from different neurological perspectives may perceive different patterns, ask different questions and challenge different assumptions. (Hong & Page, 2004).

That makes human variation among researchers relevant not only to equality or representation, but potentially to the quality of the research itself.
If human systems learn through the intelligence available to them, research is no exception.

A research system that draws on a narrow range of human perspectives necessarily limits the range of human intelligence from which it can learn.

The challenge, therefore, is larger than determining where the diagnostic boundaries of autism should lie. It is to reconsider the model of humanity against which those boundaries are being drawn, the assumptions embedded within that model, and whose intelligence participates in examining them.

Perhaps, therefore, the provocative question is not only whether autism has lost its meaning.

It is whether neurotypicality ever possessed the coherent scientific meaning we have assigned to it.

And perhaps the way to find out is finally to study it.

References

Bottema-Beutel, K., Kapp, S. K., Lester, J. N., Sasson, N. J. & Hand, B. N. (2021). Avoiding ableist language: Suggestions for autism researchers. Autism in Adulthood, 3(1), 18–29. https://doi.org/10.1089/aut.2020.0014

Crompton, C. J., Ropar, D., Evans-Williams, C. V. M., Flynn, E. G. & Fletcher-Watson, S. (2020a). Autistic peer-to-peer information transfer is highly effective. Autism, 24(7), 1704–1712. https://doi.org/10.1177/1362361320919286

Crompton, C. J., Sharp, M., Axbey, H., Fletcher-Watson, S., Flynn, E. G. & Ropar, D. (2020b). Neurotype-matching, but not being autistic, influences self and observer ratings of interpersonal rapport. Frontiers in Psychology, 11, 586171. https://doi.org/10.3389/fpsyg.2020.586171

Eicher, T., Ne'eman, A. & Quackenbush, J. (2026). Genomic, transcriptomic, and regulomic analyses do not support profound autism as a distinct biological category. bioRxiv preprint 2026.06.01.729392. https://doi.org/10.64898/2026.06.01.729392

Fletcher-Watson, S., Adams, J., Brook, K., Charman, T., Crane, L., Cusack, J., Leekam, S., Milton, D., Parr, J. R. & Pellicano, E. (2019). Making the future together: Shaping autism research through meaningful participation. Autism, 23(4), 943–953. https://doi.org/10.1177/1362361318786721

Frith, U. (2026). Autism spectrum disorder: has it lost its meaning and is it leading to misdiagnosis? Psychological Medicine. https://www.cambridge.org/core/journals/psychological-medicine/article/autism-spectrum-disorder-has-it-lost-its-meaning-and-is-it-leading-to-misdiagnosis/AF834026D004D42B1963BEA515495AC6

Hong, L. & Page, S. E. (2004). Groups of diverse problem solvers can outperform groups of high-ability problem solvers. Proceedings of the National Academy of Sciences, 101(46), 16385–16389. https://doi.org/10.1073/pnas.0403723101

Lord, C., Charman, T., Havdahl, A., Carbone, P., Anagnostou, E., Boyd, B., et al. (2022). The Lancet Commission on the future of care and clinical research in autism. The Lancet, 399(10321), 271–334. https://doi.org/10.1016/S0140-6736(21)01541-5

Milton, D. E. M. (2012). On the ontological status of autism: the 'double empathy problem'. Disability & Society, 27(6), 883–887. https://doi.org/10.1080/09687599.2012.710008

National Institute for Health and Care Excellence (NICE). (2014). Autism: Quality standard QS51, Quality statement 2: Assessment and diagnosis. https://www.nice.org.uk/guidance/qs51/chapter/quality-statement-2-assessment-and-diagnosis

National Institute for Health and Care Excellence (NICE). (2021). Autism spectrum disorder in adults: diagnosis and management (CG142; updated 2021). https://www.nice.org.uk/guidance/cg142/chapter/recommendations

Pellicano, E. & den Houting, J. (2022). Annual Research Review: Shifting from 'normal science' to neurodiversity in autism science. Journal of Child Psychology and Psychiatry, 63(4), 381–396. https://doi.org/10.1111/jcpp.13534

Pukki, H., Bettin, J., Outlaw, A. G., Hennessy, J., Brook, K., Dekker, M., Doherty, M., Shaw, S. C. K., Bervoets, J., Rudolph, S., Corneloup, T., Derwent, K., Lee, O., Rojas, Y. G., Lawson, W., Gutierrez, M. V., Petek, K., Kaar, M., Munday, K., … Walker, N. (2022). Autistic perspectives on the future of clinical autism research. Autism in Adulthood, 4(2), 93–101. https://doi.org/10.1089/aut.2022.0017