Received: 11 December 2023 | Revised: 18 December 2023 | Accepted: 19 December 2023

DOI: 10.1111/acps.13653

Acta Psychiatrica Scandinavica WILEY

INVITED EDITORIAL

Embracing evolutionary theories of autism: Implications for psychiatry

1 | BACKGROUND

Autism is one of most common disabilities, with recent estimates that 1 in 36 children[1] have an autism diagnosis and late diagnoses in adults also increasing. The DSM-5 diagnostic criteria for autism now include sensory sensitivities alongside the long-established criteria of social and communication challenges and repetitive and restrictive behaviours and interests. Most clinicians no longer view autism as something to be cured or treated, instead providing support for the challenges that autistic people experience and treatments or interventions for any co-occurring conditions, while respecting their unique differences and strengths. As the prevalence of autism, who is being diagnosed with autism, and clinical understanding of autism are changing, so are perspectives on what it means to be autistic.

One field aiming to shift scientific perspectives on how we think about autism is evolutionary psychiatry,[2] a relatively new discipline that now has its own special interest group within the Royal College of Psychiatrists in the UK as well as dedicated textbooks. Like the related field of evolutionary medicine, evolutionary psychiatry sets its focus beyond biomedical causes (e.g., genetic and environmental factors), instead asking the "ultimate" question of *why* neurodevelopmental and psychiatric conditions have persisted in the human population over time. At first glance, autism presents an evolutionary paradox[3]: it is a highly heritable condition that affects "reproductive success" yet is relatively prevalent. Evolutionary psychiatry proposes possibilities that move us away from viewing autism as a disorder or disease to one that considers the role of crucial adaptations that have shaped the evolution of the human brain and our complex cognitive abilities. In this article, we consider several evolutionary explanations of autism, the supporting evidence, and how embracing an evolutionary perspective could influence clinical practice in psychiatry.

2 | AUTISM: EVOLUTIONARY ADVANTAGES AND TRADE-OFFS?

Most evolutionary theories conceptualise autism as a specialised neurocognitive profile characterised by a trade-off between costs (social cognition) and benefits (non-social cognition). The empathising-systemizing theory[4] was the first to suggest this, framing autism as enhanced motivation to systemize (the ability to analyse the variables in a system and derive the underlying rules that govern it) paired with challenges in empathising (the ability to imagine another person's thoughts and feelings and respond with an appropriate emotion). The link between autism and occupations that require a high level of systemizing ability[5]—such as science, technology, engineering, and mathematics—provides further support for the theory that autism reflects a brain specialised in rule-based thinking. Similarly, the "diametric model" of autism[6] highlights enhancement in diverse cognitive domains including visuo-spatial skills, reading ability, and local processing, with co-occurring reductions in imagination, shared attention, and personal agency (with, interestingly, opposite patterns observed for psychotic spectrum conditions). Broadly, these two theories view autism as reflecting enhanced development of the analytical brain, with the trade-off of reduced efficiency for processing social information. An updated theory from one author of the diametric model views autism as reflecting "enhanced, but imbalanced, components of intelligence,"[7] supported by recent findings of overlap between the genetics of autism and aspects of cognition.

Autism, like many conditions of interest to evolutionary psychiatry, is highly heritable. It is thought that thousands of genetic variants contribute to the likelihood of autism; notably, most of these genetic variants individually have very small effects and are common in the general population.[8] As a result, studies often add together autism-associated genes to create an index referred to as a polygenic score. While polygenic scores for autism are, by definition, associated with autism likelihood, they are also associated with various aspects of cognitive ability in the general population[9] and brain development.[10] Furthermore, there is evidence for positive selection of autism-related genes, implying that possessing autism-related genes (although not necessarily being autistic) was adaptive during human evolution.[11] The possibility that the same genes that underlie autism are those that played a crucial role in the development of the human brain and our unique cognitive abilities can help resolve

the above-mentioned paradox of why heritable conditions perceived as disabilities could be maintained at high prevalence rates.

Given the link between autism and analytical cognition, as reflected by enhanced systemizing, positive selection for autism-related genes could have been the foundation of the leap in cause-effect thinking that led to humankind's unique ability to innovate.[12] In the words of autistic inventor and advocate Temple Grandin: "What would happen if the autism gene was eliminated from the gene pool? You would have a bunch of people standing around in a cave, chatting and socializing and not getting anything done."[13]

3 | CAN EVOLUTIONARY THEORY EXPLAIN WHY AUTISM IS MORE COMMON IN MALES?

Evolutionary theory may also be useful for understanding the sex difference in the prevalence of autism. While it is now recognised that autism often presents differently in girls and women, that screening protocols may be biased toward recognising autism in males, and that girls and women are more likely to "camouflage" autism, males are still more likely to receive an autism diagnosis.[14]

The prenatal sex steroid theory of autism proposes that elevated levels of sex steroid hormones interact with other factors (e.g., genetic variants) to disproportionately increase autism likelihood among males.[15] Gonadal sex differentiation during development, which is determined by the sex chromosomes, results in higher average levels of prenatal androgens in males compared to females. High sex steroid hormone levels during pregnancy may be a unique adaptation that has arisen during human evolution, as peak levels exceed than those in non-human primates despite differences in body weight.[16] These hormones are believed to play crucial roles during brain development, particularly in terms of cortical expansion and synaptogenesis. Recent experiments involving human stem cell-derived brain organoids show that androgens enhance neuronal development during formation of the cortex,[17] a brain region associated with many higher cognitive functions. Together, these lines of evidence suggest that human-specific evolutionary adaptations in the steroid hormone system have resulted in high levels of both androgens and oestrogens which, in turn, are associated with increased autism likelihood in males.[18] Further research is needed to explore the role of sex-differential biological factors, including sex hormones and sex-linked genes, in human development and evolution and their potential links to autism.[19]

Another commonly used framework in evolutionary psychiatry is that an extreme of an adaptation may manifest in a way that resembles a disorder. While an analytical form of cognition such as systemizing is a strength at a certain level and may underlie technical aptitude, hyper-systemizing could result in the challenges experienced by some autistic people.[20] Specifically, hyper-systemizing may manifest as intense focus on details and patterns, rigid adherence to routine, feeling overwhelmed by too much detail, and distress when routines are interrupted. As males, on average, score higher on measures of systemizing than females, the tipping point of when high levels of systemizing become "too much of a good thing" could contribute to the higher rates of autism in males.

4 | FUTURE DIRECTIONS IN EVOLUTIONARY PSYCHIATRY

In its present state, evolutionary psychiatry mainly exists as an academic discipline, applying theory to understand how evolutionary processes have shaped the human mind and how certain psychiatric conditions might be better understood as adaptations or byproducts of our evolutionary history. However, psychiatry is a dynamic field with best practices adapting in response to changes in our understanding of conditions, their underlying causes, and the best way to provide support. Although applying evolutionary psychiatry theory in clinical practice is a new frontier, early efforts suggest that it is one worth exploring. Clinicians who adopt a holistic approach could help autistic people navigate challenges while directing them how to focus their strengths, such as excellent attention to and memory for detail, pattern-recognition skills, and ability to focus on one topic in great depth.

One of few randomised controlled trials to put evolutionary psychiatry thinking into practice presented participants with a history of depression with one of two sets of videos: the first explained depression as a disease with known biopsychosocial risk factors, while the second described depression being an evolutionarily conserved signal that serves an adaptive function in specific situations.[21] Participants who viewed the second set of videos later reported lower self-stigma and stronger beliefs about their agency to reduce depressive symptoms. Whether greater awareness of evolutionary theories could have similar benefits for autistic people remains to be seen.

Perhaps most obviously, evolutionary psychiatry provides a scientific basis supporting that autism reflects variation in the human brain and thinking, rather than defects that need to be fixed, and supporting shifts in social attitudes. In many ways, the aims of evolutionary psychiatry are shared with those of the neurodiversity movement,[22] which advocates that the differences associated with autism should be recognised and respected as a

normal part of human diversity. For clinicians, understanding the strengths and challenges of autism through the lens of evolution could lead to providing support and accommodations that enable neurodivergent individuals to thrive in different environments. As progress in biomedical understanding of autism has not had a transformative effect on clinical practice, evolutionary psychiatry may present a new framework for understanding autism that is still founded in science.

PEER REVIEW

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/acps.13653.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

Tanya L. Procyshyn
Alex Tsompanidis
Simon Baron-Cohen

Autism Research Centre, Department of Psychiatry, University of Cambridge, Cambridge, UK

Correspondence
Tanya L. Procyshyn, Autism Research Centre, Department of Psychiatry, University of Cambridge, Cambridge, UK.
Email: tlp30@cam.ac.uk

ORCID

Tanya L. Procyshyn https://orcid.org/0000-0003-1266-6697

REFERENCES

1. Maenner MJ, Warren Z, Williams AR, et al. Prevalence and characteristics of autism spectrum disorder among children aged 8 years—autism and developmental disabilities monitoring network, 11 sites, United States, 2020. *MMWR Surveill Summ*. 2023;72:1-14.
2. Abed R, Brüne M, Wilson DR. The role of the evolutionary approach in psychiatry. *World Psychiatry*. 2019;18:370-371.
3. Keller MC, Miller G. Resolving the paradox of common, harmful, heritable mental disorders: which evolutionary genetic models work best? *Behav Brain Sci*. 2006;29:385-404.
4. Baron-Cohen S. Autism: the empathizing–systemizing (E-S) theory. *Ann N Y Acad Sci*. 2009;1156:68-80.
5. Ruzich E, Allison C, Chakrabarti B, et al. Sex and STEM occupation predict autism-spectrum quotient (AQ) scores in half a million people. *PloS One*. 2015;10:e0141229.
6. Crespi B, Badcock C. Psychosis and autism as diametrical disorders of the social brain. *Behav Brain Sci*. 2008;31:241-261.
7. Crespi BJ. Autism as a disorder of high intelligence. *Front Neurosci*. 2016;10:300.
8. Grove J, Ripke S, Als TD, et al. Identification of common genetic risk variants for autism spectrum disorder. *Nat Genet*. 2019;51:431-444. doi:10.1038/s41588-019-0344-8
9. Clarke TK, Lupton MK, Fernandez-Pujals AM, et al. Common polygenic risk for autism spectrum disorder (ASD) is associated with cognitive ability in the general population. *Mol Psychiatry*. 2016;21:419-425. doi:10.1038/mp.2015.12
10. Khundrakpam B, Vainik U, Gong J, Al-sharif N. Neural correlates of polygenic risk score for autism spectrum disorders in general population. *Brain Commun*. 2020;2(2):fcaa092.
11. Prakash A, Banerjee M. Genomic selection signatures in autism spectrum disorder identifies cognitive genomic tradeoff and its relevance in paradoxical phenotypes of deficits versus potentialities. *Sci Rep*. 2021;11:10245.
12. Baron-Cohen S. *The Pattern Seekers: How Autism Drives Human Invention*; 2020.
13. Grandin T. *The Way I See it: A Personal Look at Autism & Asperger's*; 2008.
14. Hull L, Petrides KV, Mandy W. The female autism phenotype and camouflaging: a narrative review. *Rev J Autism Dev Disord*. 2020;7:306-317.
15. Baron-Cohen S. The extreme male brain theory of autism. *Trends Cogn Sci*. 2002;6:248-254.
16. Czekala NM, Benirschke K, McClure H, Lasley BL. Urinary estrogen excretion during pregnancy in the gorilla (*Gorilla gorilla*), orangutan (*Pongo pygmaeus*) and the human (*Homo sapiens*) 1. *Biol Reprod*. 1983;28:289-294.
17. Kelava I, Chiaradia I, Pellegrini L, Kalinka AT, Lancaster MA. Androgens increase excitatory neurogenic potential in human brain organoids. *Nature*. 2022;602:112-116.
18. Baron-Cohen S, Tsompanidis A, Auyeung B, et al. Foetal oestrogens and autism. *Mol Psychiatry*. 2019;25:2970-2978. doi:10.1038/s41380-019-0454-9
19. Werling DM. The role of sex-differential biology in risk for autism spectrum disorder. *Biol Sex Differ*. 2016;7:58.
20. Baron-Cohen S, Ashwin E, Ashwin C, Tavassoli T, Chakrabarti B. Talent in autism: hyper-systemizing, hyper-attention to detail and sensory hypersensitivity. *Philos Trans R Soc B Biol Sci*. 2009;364:1377-1383.
21. Schroder HS, Devendorf A, Zikmund-Fisher BJ. Framing depression as a functional signal, not a disease: rationale and initial randomized controlled trial. *Soc Sci Med*. 2023;328:115995.
22. Kapp SK, Gillespie-Lynch K, Sherman LE, Hutman T. Deficit, difference, or both? Autism and neurodiversity. *Dev Psychol*. 2013;49:59-71.

How to cite this article: Procyshyn TL, Tsompanidis A, Baron-Cohen S. Embracing evolutionary theories of autism: Implications for psychiatry. *Acta Psychiatr Scand*. 2024;149(2):85-87. doi:10.1111/acps.13653