


COMMENTARY

Changing perspectives on autism: Overlapping contributions of evolutionary psychiatry and the neurodiversity movement

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Abstract

Perspectives on autism and psychiatric conditions are affected by a mix of scientific and social influences. Evolutionary psychiatry (EP) and the neurodiversity movement are emerging paradigms that reflect these distinct influences, with the former grounded in scientific theory and the latter driven by political and social principles. Despite their separate foundations, there is a significant overlap between EP and neurodiversity that has not been explored. Specifically, both paradigms reframe disorders as natural cognitive differences rather than disease; expand the concept of “normal” beyond that implied in modern psychiatry; focus on relative strengths; recognize that modern environments disadvantage certain individuals to cause functional impairment; emphasize cognitive variation being socially accommodated and integrated rather than treated or cured; and can help reduce stigmatization. However, in other ways, they are distinct and sometimes in conflict. EP emphasizes scientific explanation, defines “dysfunction” in objective terms, and differentiates heterogeneous cases based on underlying causes (e.g. autism due to *de novo* genetic mutations). The neurodiversity movement emphasizes social action, removes barriers to inclusion, promotes inclusive language, and allows unrestricted identification as neurodivergent. By comparing and contrasting these two approaches, we find that EP can, to some extent, support the goals of neurodiversity. In particular, EP perspectives could be convincing to groups more responsive to scientific evidence and help achieve a middle ground between neurodiversity advocates and critics of the movement.

Lay Summary

This paper introduces neurodiversity and evolutionary psychiatry and explores the ways in which they overlap and contrast. Both approaches emphasize strengths, question what we think of as “pathological,” reflect on the role of modern environments in disabling people, and could help destigmatize autism and other conditions. However, there are also notable differences: neurodiversity principles apply to any type of diagnosable mental condition, but evolutionary explanations are only suitable for certain conditions and individuals. Nevertheless, evolutionary psychiatry may be useful for convincing scientifically-minded people of the validity of the neurodiversity concept and movement.

KEYWORDS

autism, evolutionary psychiatry, neurodiversity, psychiatry, stigma

INTRODUCTION

In recent years, evolutionary psychiatry (EP) and neurodiversity have emerged as novel paradigms for reframing

cognitive states and traits historically diagnosed as disorders. At first glance, these two movements occupy distinct spheres: EP seeks to understand such conditions by examining their roots in human evolution, exploring theories

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related to potential adaptive functions and mismatches with the modern environment. The neurodiversity social movement advocates for the acceptance and celebration of cognitive differences, emphasizing that conditions including, but not limited to, autism and attention-deficit hyperactivity disorder (ADHD) reflect natural variation in human brains and behavior rather than pathology. Despite their differences, EP and neurodiversity share the overarching goal of broadening perspectives on cognitive differences in a way that leads to reconsideration of conditions typically diagnosed as psychopathology. In this article, we consider the ability of EP and neurodiversity to complement each other to better achieve understanding and support of differently-thinking people, with a particular focus on autism.

PRINCIPLES OF EP

Mainstream psychiatric research generally seeks underlying mechanistic causes of diagnosed conditions with intention to treat. Such causes may be biological (e.g., genetic mutations, alterations in brain structure or function, variation in neurochemistry) or environmental (e.g., stress). In contrast, EP emphasizes explanation at the “ultimate” level (Mayr, 1961), asking *why* the process of evolution by natural selection has resulted in these “proximate” causal mechanisms and their particular effects (Hunt et al., 2023; Nesse, 2023). Why do brains, genes, and stress predispose us to psychiatric conditions?

The paradox that psychiatric conditions are common, heritable, and (as perceived by some) harmful is widely recognized (Keller & Miller, 2006): Shouldn't natural selection have eliminated the responsible genetic variants? Why haven't humans evolved optimal mental health? EP researchers have proposed diverse explanations for the persistence of various psychiatric symptoms and conditions. For example, ADHD may reflect “evolutionary mismatch”: while increased activity levels and the tendency to rapidly switch attention may have been adaptive in the environments of our evolutionary past, these traits may be experienced as challenges in the more sedentary modern world. Moderate anxiety symptoms could have value as crucial, life-saving signals in the face of certain threats, and may be a misinterpretation of adaptation as disease (Nesse, 2019). One outcome of adopting an EP-oriented perspective is reconsidering certain presumed cognitive dysfunctions as differences and recognizing their potential functional adaptive value (although caution should be taken to avoid excessive evolutionary storytelling, Gould & Lewontin, 1979).

EP THEORIES OF AUTISM

Autism spectrum conditions (henceforth autism), which are characterized by a combination of social and non-social symptoms, present a complex case for EP

explanations given their high level of phenotypic and etiological heterogeneity and associated low reproductive success (Ploeger & Galis, 2011). Nevertheless, proposed EP explanations generally recognize the cognitive strengths shown by many autistic individuals, framing social challenges as costs offset by these abilities.

The clinical psychologist Simon Baron-Cohen has a long history of challenging the pathologizing of autism (Baron-Cohen, 2002), noting that autistic traits could be restated without negative connotations (e.g., “obsessive behavior” restated as “single-minded focus”) and calling for autism spectrum disorder to be renamed autism spectrum *condition* (Lai & Baron-Cohen, 2015). His empathizing-systemizing theory proposes that autism reflects an extreme position along a dimension of people- and system-focused cognition, resulting in strengths in understanding patterns and rule-based systems paired with challenges in social cognition. Support for this theory includes autistic people's strengths in understanding of folk physics (Baron-Cohen, Wheelwright, Spong, et al., 2001), high attention to detail (O'Riordan et al., 2001), and expertise in science, technology, engineering, and mathematics (STEM) (Baron-Cohen et al., 1999). Notably, individuals working in STEM-related domains score higher than average on self-report questionnaires assessing non-clinical levels of autism-related traits, implying such traits (which are, to some degree, heritable) also predispose one to technical ability (Baron-Cohen, Wheelwright, Skinner, et al., 2001; Greenberg et al. 2018). Baron-Cohen's book *The Pattern Seekers* (2020) develops a more fully fledged evolutionary theory, suggesting that individuals exceptionally skilled in systemizing were valuable in ancestral social groups, potentially playing roles as tool-makers, inventors, and knowledge experts (e.g., properties of plants, animal behavior).

Subsequent EP theories similarly propose that autism is related to cognitive specialization (Hunt & Jaeggi, 2022). Crespi and Badcock (2008) frame autism and schizophrenia as “diametrical disorders of the social brain,” with the former reflecting the hyperdevelopment of mechanistic cognition paired with underdevelopment of mentalistic cognition (and vice versa for psychotic spectrum conditions such as schizophrenia). They collate evidence of opposite patterns of phenotypic alterations between these conditions (e.g., in autism: enhanced visuospatial skills, increased local processing, reduced imagination; vice versa in psychotic spectrum conditions) alongside differences in neuroanatomy, neurological function, genetic variants, and levels of hormones and growth factors that may influence the differential trajectories of brain development that predispose individuals to autism or psychotic spectrum conditions. Subsequently, Crespi (2016) developed the “high intelligence imbalance” hypothesis more specific to autism, arguing that strong recent evolutionary pressures for increased intelligence in humans cause vulnerability to autism when certain aspects of intelligence are exaggerated. As empirical support, Crespi points to recent findings of overlap between

aspects of brain size, brain growth, genetic variants, and sensory and visuospatial abilities associated with autism and high IQ. Moreover, autistic people and people with a high IQ both typically show more focused attention, a more deliberate decision-making style, and greater interest in STEM. Notably, several cognitive strengths associated with autism are shared by the (non-autistic) relatives of autistic people (Gizzonio et al., 2014; Noland et al., 2010), further suggesting a shared genetic basis between autism and cognitive specialization.

Such EP hypotheses align with current genetic understanding of autism, namely that autism likelihood is influenced by more than a thousand genes with individually small effects. In the general population, polygenic scores for autism (i.e., an individual's collective "sum" of genes linked to autism) are positively correlated with cognitive ability (Clarke et al., 2016) and verbal-numerical reasoning and educational attainment (Hagenaars et al., 2016). In other words, individuals who carry more autism-related genes (yet are not autistic) tend to perform better on cognitive tests and be more successful in formal education. Studies examining specific genetic variants report similar patterns: Olduvai protein domain family gene (formerly *DUF1220*) copy number is linked to increased brain size, higher cognitive function (measured as higher IQ and mathematical aptitude), and higher autism likelihood (Sikela & Searles Quick, 2018). Taken together, these lines of evidence support an EP perspective that genes with pleiotropic effects related to brain development, intelligence, and autism were selected for as humankind found itself living in increasingly technologically complex societies.

From an evolutionary perspective, predisposition to autism could be explained as adaptation for increased human cognitive specialization, systemizing, and intelligence, with the most disabling cases of autism occurring as occasional costly by-products that are not adaptive in themselves. For a parallel proposed shift of perspective on autism and other cognitive differences independent of adaptive explanations, we turn to the contributions of the neurodiversity movement.

THE NEURODIVERSITY MOVEMENT

Neurodiversity, introduced in the milieu of the social model of disability (Shakespeare, 2006), now generally refers to the social movement with the goal of reconceptualizing common, lifelong mental conditions as differences to be accepted and integrated, rather than defects or diseases to be treated or cured (Kapp et al., 2020). Neurodiversity shares similarities with, and in some cases was explicitly inspired by, historical movements in antipsychiatry (Szasz, 1974) and disability activism, such as "Mad Pride" (Lewis, 2006). While the neurodiversity movement was initiated by autistic people, it has expanded to include numerous conditions broadly classified as mental disorders

(Nelson, 2020). Advocates generally seek recognition and acceptance of neurodiversity—diversity among minds—as a valuable part of natural human cognitive diversity (alike biodiversity), claiming that differences are harmfully pathologized and strengths of "neurodivergent" individuals are overlooked by the "neurotypical" majority. Attempts to cure psychiatric conditions are seen as equivalent to curing someone's sexuality or race; just as there is no "right" gender or race, there is no "normal" or "right" type of mind. Neurodiversity thus offers a social paradigm for reconceiving psychiatric traits as differences rather than diseases, promoting the need for acceptance and accommodation instead of cures or treatment. The movement also highlights how modern society is, to some extent, responsible for turning differences into disabilities. For example, the expectation of fluid social interactions in schools or workplaces may disable autistic people, and their disability could be prevented by removing this expectation.

To advocates, the argument for neurodiversity is founded on the explicit goal of social justice and biological explanations are only engaged with in the context of rejecting pathologization and emphasizing that disorder and dysfunction are contingent on contemporary environments (Chapman, 2021). Although biological and mainstream psychiatric explanations of neurodiversity are generally avoided or outright rejected by the movement, there is a history of speculation that neurodivergent conditions may have been integrated or better valued in the evolutionary past. For example, autistic advocate Temple Grandin mused: "Who do you think made the first stone spear? That wasn't the yakkity yaks sitting around the campfire. It was some Asperger sitting in the back of a cave..." (Weiss, 2010).

AREAS OF ALIGNMENT BETWEEN EP AND NEURODIVERSITY

Despite their common reference to neurodiverse strengths and emphasis on modern environments as responsible for creating disability, extended investigation into the areas of overlap and divergence between EP explanations and neurodiversity perspectives is lacking.

While it must be acknowledged that the neurodiversity movement and EP occupy distinct spheres—with the former driven by political and social principles and the latter by scientific theory—many of their goals and implications are shared (Figure 1). Specifically, EP and neurodiversity overlap in their: (i) reframing of traits currently diagnosed as mental disorders as natural cognitive differences rather than disease; (ii) expansion of the concept of "normal" beyond that implied in modern psychiatry; (iii) encouragement of understanding of psychiatric conditions in terms of relative strengths; (iv) recognition that modern environments unfairly disadvantage certain individuals, and so the environment is causing functional impairment; (v) emphasis on cognitive variation being

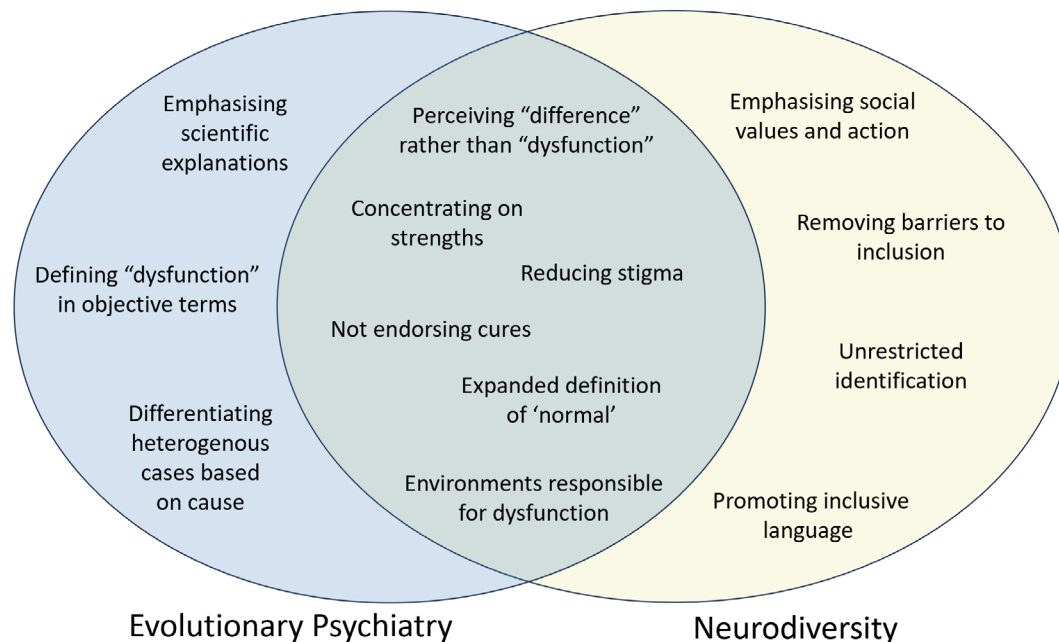


FIGURE 1 A Venn diagram displaying areas of overlap and difference between evolutionary psychiatry and neurodiversity.

socially accommodated and integrated rather than treated or cured; and (vi) potentially reducing stigmatization.

Both paradigms are concerned that the pathologizing of psychiatric conditions has justified billions of dollars in biomedical research focused on cures and causes with near zero success in improving outcomes for affected individuals (Insel, 2013). EP directs attention to different areas of research and approaches to inclusion aligned closely with the neurodiversity perspective. Despite the need for separation of scientific fact and social values (to avoid the “naturalistic fallacy”), EP explanations may play important roles fostering support for neurodiversity. Notably, historic political movements pushing for changes in treatment of minority groups have referenced scientific facts (Brookey 2002; Prontzos, 2019). Scientific justification could be particularly useful in convincing demographics unlikely to be swayed by purely social arguments. This includes the scientific research community, who still overwhelmingly research brain and genetic correlates searching for biomarkers, assuming the conditions to be true pathologies in need of cures. Evolutionary accounts instead encourage identifying correlated cognitive benefits of neurodiverse conditions, understanding the specific modern factors that exacerbate harms, and rejecting simple biomedical treatments if these traits are linked with adaptations. Published findings of strengths associated with various conditions, or genetic evidence that most cases of a particular condition are due to variants common in the general population—rather than “disease alleles”—could support the shared aim of EP and the neurodiversity movement in redefining what it means to have a psychiatric diagnosis. Wider support of EP could also increase participation of neurodivergent

individuals in scientific research—a concern raised by neurodiversity advocates (Fletcher-Watson et al., 2019)—as study designs go beyond reductionist biomedical models and embrace more holistic approaches to both understand the conditions and improve outcomes.

Social movements endeavoring to alter norms and language relate to what philosophers call “conceptual engineering”: the process by which existing concepts are assessed and improvements are proposed and implemented (Plunkett & Cappelan, 2020). The neurodiversity movement has clearly been engaged in a form of conceptual engineering, introducing the term “neurodiversity” and attempting to redefine what it means to be labeled as autistic, ADHD, dyslexic, and so forth. Given that medicalization of a state carries connotations of negative evaluation—something to be cured or prevented—the need to move away from pathology-oriented language is entirely understandable. While the neurodiversity movement’s conceptual engineering efforts are concerned with acceptance and not explanation, EP has the power to explain cognitive differences without pathologizing. Indeed, there is evidence that explanations of mental health conditions referring to stress, rather than neurological or genetic malfunction, result in lower stigmatization (Loughman & Haslam, 2018); that EP explanations lead to lower self-stigmatization in individuals with depression (Schroder et al., 2023); and that cognitive behavioral therapy can be enhanced by integrating evolutionary insights (Abrams, 2020), particularly by destigmatizing conditions or encouraging individuals to perceive potential strengths associated with their conditions. As the success of conceptual engineering is related to how well the new concept is adopted (Pinder, 2017), if the

communities that neurodiversity advocates communicate with are better convinced by scientific—and specifically EP—arguments, then adopting those hypotheses will help their goals.

In the context of autism, promotion of “strength-based” approaches are associated with improved life satisfaction (Duan et al., 2014); improved parental perceptions of their autistic children (Steiner, 2011); and improved hospitalization rates, employment/educational attainment, and intrapersonal outcomes such as self-efficacy and sense of hope (Tse et al., 2016). In education, approaches to utilizing strong autistic interests effectively are blossoming (Bianco et al., 2009; Campbell & Tincani, 2011; R. Wood, 2019). Neurodiversity Celebration Week, a yearly occurrence since 2018, specifically reaches out to schools and universities encouraging the recognition of talents and advantages of neurodivergent students (Palumbo, 2023). Neurodiversity has been raised as the next frontier for improving inclusion in the workplace (Salman, 2019), encouraging employers to incorporate neurodivergent employees by framing their strengths as a competitive advantage (Austin & Pisano, 2017; Curry, 2019; V. Wood, 2019). Suggesting that these approaches do work, multinational corporations like SAP are now specifically seeking neurodivergent employees through their Autism at Work program, and consulting firms like Auticon have been established to place autistic employees in IT roles. Although there are not yet large-scale studies on outcomes of such efforts, strengths-based perspectives seem to be increasing educational and employment opportunities for autistic people, potentially with subsequent effects of supporting mental health and general well-being. In allowing spaces to exist where individuals’ weaknesses are supported and their strengths incorporated, we may be replicating the ancestral environment that caused that very cognitive diversity to flourish.

AREAS OF DISAGREEMENT AND ONGOING DEBATE

Lastly, the areas where the EP and neurodiversity approaches diverge—and even actively conflict—must be noted. Neurodiversity’s social model allows extension of the “neurodivergent” label to any individual with any neurotype, rejecting any sense of “normal” or “health” to the traits under its umbrella (Walker, 2013). By contrast, one of EP’s fundamental scientific appeals is grounding the distinction between health and disorder with reference to evolutionary dysfunction (Hunt et al., 2022). This has been primarily forwarded by Wakefield’s (1992, 2015) “harmful dysfunction” definition of disorder, which claims that disorder exists wherever there is concurrent harm, defined by social and individual values, and dysfunction, defined by interruption of evolutionarily selected effects. This definition specifically aims to ground definitions of disorder in objective scientific facts,

ensuring that diagnoses of health and disease are not merely social.

As a result, EP inevitably subtypes the autism spectrum into multiple classes with distinct etiologies (Del Giudice & Haltigan, 2021; Hunt, 2023). While most cases of autism have no clear pathological cause, roughly 25% of cases (dependent on inclusion criteria) are described as syndromic autism, as they are associated with somatic abnormalities or a clear biomedical cause, such as genetic mutations (e.g., fragile X syndrome, 16p11.2 duplication) or fetal/neonatal damage (e.g., fetal valproate syndrome, obstetric complications) (Fernandez & Scherer, 2017). As these cases of autism are not evolutionarily paradoxical (Keller & Miller, 2006), they do not warrant explanations referencing adaptive traits. The EP approach could thus be perceived as callous and discriminatory by neurodiversity advocates who consider all cases of autism as examples of neurodiversity. Similarly, EP explanations are not required for cognitive differences arising from other *de novo* genetic causes like Down Syndrome, which often result in intellectual disability. While these conditions are all examples of neurodiversity and can be advocated for under that banner, any scientific approach to understanding cognitive divergence will discriminate between heterogeneous cases.

Scientific calls for discerning autistic subtypes are not unique to evolutionary approaches. Even within cases of nonsyndromic autism, high levels of heterogeneity continue to pose challenges to both research and clinical practice. Informed by clinical experience, Mottron and Gagnon (2023) propose more specific diagnostic criteria for what they term “prototypical autism.” In this model, there is a developmental trajectory from ages 2 to 5 where most children increasingly process language and information may in a socially biased manner. In the absence of this bias, children without neurodevelopmental impairment will develop interest in complex information independent of its social content, resulting in the more homogenous phenotype of prototypical autism. In this way, the authors frame prototypical autism as the non-dominant—but certainly not pathological—outcome of a dynamic biological system, similar to being left-handed. As described above, this rarer developmental trajectory could lead to the higher rates of specialized talents and strengths among “prototypical” autistic people of normal to high intelligence (e.g., Meilleur et al., 2015).

To some neurodiversity advocates suggestions that prototypical autism warrants evolutionary explanations and syndromic cases of autism do not may be unacceptably discriminatory. This is where the major distinction between the EP and neurodiversity paradigms emerge: while scientific justification of neurodiversity is constrained to empirical evidence, social norms can be extended indefinitely. As a matter of principle, every cognitive difference can be equally included under the “neurodiversity” banner—whether by self-identification (Aftab, 2021) or otherwise.

This had led to major criticisms that the neurodiversity movement makes “implausible claims about the distinction between difference and disorder” (Nelson, 2020). Indeed, some individuals are unhappy with their neurodivergent traits and prefer to label them as a disorder, not mere difference. Criticism has also come from parents and family members of severely disabled autistic individuals (e.g., Clements, 2019; Escher, 2023), as the claim of difference and not disease, strengths over weaknesses, and acceptance rather than cure is contradictory to their experience of loved ones faced with serious disability. Even Judy Singer, who coined the term “neurodiversity,” intended it to be applied to people with normal or high intelligence and thinks excessive inclusivity is unrealistic (Lutz, 2023). Neurodiversity advocates may argue that the core of this distinction should rest in the hands of the affected individuals, particularly whether they perceive their differences as impacting their well-being, which follows the general trend in contemporary psychiatry to diagnose mental states as disorder wherever there is substantial harm. However, this doesn’t provide a reliable scientific basis for attributing difference or disorder, leaving the disagreement to be resolved as a purely social matter.

Scientific approaches from EP could calm this debate by meeting this disagreement halfway, pushing back on assumptions of simple pathology dominant in contemporary psychiatry while making sense of stark differences by recognizing the heterogeneity within wide diagnostic labels like autism spectrum disorder. This is not to say that strength-based approaches, social accommodations, and greater acceptance are not valid for cases of neurodiversity with an identifiable genetic or developmental pathology; rather, EP adds nuance that helps resolves the conflation of diverse conditions under singular banners. While the “neurodiversity” term can encompass all biological causes of differences in minds, acknowledgment of distinct biological causes seems useful for reducing confusion and encouraging productive debate, which is sorely needed (Baron-Cohen, 2019).

Without acknowledgment of heterogeneity of forms of neurodivergence like autism, knowledge of certain causes, and varying levels of disability, critics of the neurodiversity movement may dismiss the *whole* neurodiversity paradigm’s validity. As this would unfairly exclude the vast majority of individuals for whom no such cause has been found, EP explanations might be critical. As both EP and neurodiversity continue to grow, it seems prescient to consider how the science may reinforce and support the social goals, as has been recognized as important in the context of challenging sexual prejudice (Bartoş et al., 2014) and racism (Donovan et al., 2019).

CONCLUSION

EP and neurodiversity are novel paradigms developing in parallel with different but complementary aims. While

EP stipulates scientific theory and neurodiversity stipulates social response, both ultimately advocate for broadening the definition of “normal,” rejecting cures, recognizing the role of the environment, and highlighting that differences can be strengths. Notably, scientific perspectives such as EP may provide powerful justification of the core ideas of neurodiversity: it could become not just good or socially desirable to agree with the principles of the neurodiversity movement, but an accurate and true understanding of evolutionary processes explaining cognitive differences captured under the umbrella of neurodiversity. Within an EP-believing world, we would expect shifts in how education, employment, and public spaces include neurodivergent people that are largely in line with what neurodiversity advocates ask for. How much neurodiversity advocates care about integrating scientific explanation into their advocacy is up to them; but of all the psychiatric paradigms, the evolutionary approach may be the one that makes the most sense.

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DATA AVAILABILITY STATEMENT

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

ETHICS STATEMENTS

No ethics statements are required, as no participants were involved.

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