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Two Brief Communications on Autism: The Relationship Between Testosterone and Stem Savants and an Ethological View of Repetitive Behavior

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Abstract

This commentary addresses two distinct aspects of autism spectrum condition. First, it critiques the proposed link between fetal testosterone levels and the emergence of autistic traits, arguing that such theories are reductive and risk overshadowing the profound heterogeneity and individuality of autistic people. The author suggests a more limited, theoretical connection between testosterone-driven neural development and a higher incidence of STEM savant skills in some autistic individuals. Second, the paper proposes an ethological perspective, inspired by Konrad Lorenz's work on learning and feedback, to interpret repetitive behaviors not as mere stereotypes but as purposeful actions providing essential positive feedback to the individual. The overarching theme is a plea for a nuanced, individual-centric approach to understanding autism that prioritizes personal experience over reductive biological determinism.

Keywords: Autism spectrum disorder, Testosterone, Savant syndrome, STEM, Repetitive behavior, Ethology, Individual differences, Simon Baron-Cohen, Konrad Lorenz

Testosterone, Mathematical Savants, and STEM Faculties in Autism

Robert Sapolsky is one of the leading experts in behavioral neuroscience, but first and foremost, he is a person of rare humanity (in the highest sense of the term) and one of the most brilliant minds who, in recent years, has illuminated reflections on free will and consciousness [1]. He had the extraordinary courage to write two volumes totaling over 1,700 pages (in the Italian version I read) that I consider essential reading for any judge or lawyer working in criminal law [1,2].

In his two most recent books, I came across some references to autism and felt they deserved a response. As always happens when trying to respond to a particularly profound author, they stimulate you in turn to a deeper reflection. If possible, here is mine, concerning the link between testosterone, mathematical savants, and STEM faculties in autism.

I found the reference to studies (in this case, I refer to Sapolsky 2017) particularly important, indicating that anomalies found in oxytocin and vasopressin increase the risk associated with the possibility of presenting disorders attributable to impaired sociality, with particular reference to the autism spectrum [2]. In this context, Sapolsky rightly observes that autistic people show reduced responses in the fusiform facial area [2]. Continuing the citation, it is highlighted that autistic people are associated with genetic variants that encode oxytocin and vasopressin and other non-genetic mechanisms that can silence the oxytocin receptor gene, as well as lower concentrations of the receptor itself [2]. In this context, he adds that these neuropeptides enhance social skills in some autistic people, for example by improving eye contact [2].

Clearly, I understand the brief reference in the broader context of the paragraph and chapter in which it is inserted. However, I wish to emphasize that, unfortunately, given the sensitivity of the topic, such a reference can be misleading in the context of a debate that is always highly active on autism.

In a 2011 book, Simon Baron-Cohen linked a mere statistical contingency—high testosterone levels—to the onset of autism, qualified as the "extreme male brain" as early as 2003 [3,4]. Perhaps in an attempt to recover, in 2020 the same author published a new book in which "he explains to us" how autism drives human invention, defining autistic people as "the geniuses of creativity" [5]. Both books report numerous citations of scientific studies, obviously extrapolated and connecting the data in the unequivocal direction of confirming their respective theses.

Personally, the only correlation I have found—moreover theoretical, entirely to be validated through serious investigation—might be the following: since testosterone contributes to developing a specific brain area that we know is specialized in logic and mathematical processing [6], this testosterone-related anomaly could (at most) explain a fair percentage of high-functioning autistic people in STEM areas as well as mathematical "savants." And that is all.

The problem with such a delicate topic as autism is that we often forget we are talking about people. Even in specialist journals, reviewers almost always ask me to change "autistic people" to "ASD," something I always refuse to do! It reminds me—and with this memory, I console myself—when a Jane Goodall article was rejected as "unscientific" because she called her primates by name instead of referring to them "with a number" [7].

In the extraordinary heterogeneity of the manifestation of the autistic complex, we encounter enormous facets and declinations. We can certainly state that no two autistic people are identical. One of the reasons (and here I resume Sapolsky 2023) why the debate on autism has been much more nuanced and heterogeneous compared to schizophrenia is precisely linked to the diagnostic process [1].

First, I wish to highlight a small error: Leo Kanner did not speak of refrigerator mothers. When he apologized to the families of autistic people, he did so on behalf of all of science and medicine in particular [8]. The response to Bettelheim—unlike what happened in the history of schizophrenia—did not wait until the late 1970s. In 1964, Bernard Rimland stated that autism was not a psychological manifestation caused by insensitive parents, a conviction widely held and popularized by Bettelheim [9]. Instead, Rimland suggested, autism was the result of biochemical "defects triggered by environmental assaults" [9]. He recognized that there might also be a genetic component that predisposes children to the disorder. He argued that autism could "be treated, or at least improved, with biomedical and behavioral therapies" [9]. From the very beginning, therefore, a real movement developed that sought to bring autism into the more coherent context of biology and medicine (on all this, see Di Salvo 2024a, in which I have extensively examined the path from Kanner's diagnosis to recent years) [8].

Unfortunately, the medical context of the second half of the 20th century was entirely focused on the "search for the single cause." An approach that has been dragged even into the era of genetics: the search for the single gene and the single variation. This approach and this research direction were certainly useful for Down syndrome and Fragile X syndrome. Autism and schizophrenia, however, are complex syndromes that do not have a single cause; suffice it to say that there are currently over 100 genes involved in autism [10]. But autism itself can be an overall example of its declination (in both of Sapolsky's books) of a chronological process that goes back in time, at least to fetal development and perinatal phases [1,2]. At least this is what emerges from a systematic review and an overall reorganization of the enormous body of studies from the last thirty years.

And just as an overall and extremely heterogeneous picture emerges in which objectively every individual is unique, the therapy also turns out to be unique and individual. For less severe and high-functioning situations, the support of cognitive-behavioral therapy can yield enormous results [11]. With a limited number of children and adolescents, interactive therapy with animals helps to greatly reduce states of anxiety and stress and is an objective facilitator of social integration; but in this specific case, we have the ultimate proof of extreme individuality: there are children who find interaction with horses miraculous, others with dogs, others with dolphins, and such interactions are absolutely subjective and not interchangeable.

An Ethological Suggestion for Repetitive Behavior

One of the characteristics of many autistic people is "repetitive behavior." In reality, this is a very varied and heterogeneous series of behaviors characterized by repetitiveness, ranging from the ritualistic repetition of practices and gestures to obsessive behaviors, and in some ways also includes rocking and circular paths or even "persistent observations."

When an analysis and a suggestion are profound, they stimulate reflection. In studying Lorenz's ethology, I found a suggestion that I would like to share and highlight, which should (strictly) be subjected to further investigation in the field of cognitive-behavioral psychology research. Lorenz states:

> "In contrast to most behaviourists, for us ethologists it is essential to ask why learning (apart from a few incorrect performances, from which important deductions can be drawn) always leads to an adaptation of behaviour, i.e. to an improvement in its teleonomic effect. We know that success encourages the animal to repeat the behaviour that leads to it and that failure produces the opposite effect. But where does the animal get its awareness of what success and failure are? We know that the triad consisting of appetitive behaviour, innate triggering mechanism and final action that discharges the impulse also appears in the animal kingdom as a closed programme that cannot be modified by learning; we also know that this occurs mostly in lower organisms and that learning through success or failure has evidently been added in a later evolutionary step... The origin of the regulatory circuit that communicates the success of a behavioural module backwards is unthinkable without assuming that a linear system already exists that can function even without this retroactive effect... In order to convey to the animal information about the success of the action just performed, in the sense of its teleonomic effect on the external world, communications from this external world are necessary. Our "innate teacher", who, in case of success, pats the organism on the shoulder and says, "Do it again", and, in case of failure, wields the corrective rod, must therefore receive information from the outside world." [12]

Without wishing to draw more from these considerations than they explicitly state, we should ask ourselves what "feedback" repetitive behavior provides, which cannot be trivialized as stereotypy, instinct, or an automated mechanism. It is clear, even in the general heterogeneity of repetitive behaviors in autistic people, that each of these repetitions (often exhausting and obsessive for an observer) must correspond to positive feedback, something that alleviates pain, solves a problem, or generates a proprioceptive benefit.

Questioning the specific and individual case can account for the underlying need and allow ample room for communication and intervention. My thesis—yet to be proven—is that observing repetitive behavior opens up a primary individual channel of communication and, at the same time, a unique window for understanding the individual "discomfort" of that autistic person.

Conclusions

These two brief communications are not intended to be a review or research article. They are two communications that aim to highlight two very interesting correlations. The aim is, if anything, to stimulate new research and theoretical insights that can lead us to a better interpretation of two central aspects of autistic behavior. They can be considered simple reflections, as such without any claim to be exhaustive or conclusive, but if they have even the merit of stimulating objections and refutations, they will have been useful and in line with the author's purpose. If, on the other hand, they lead to more fruitful results, they will have exceeded my personal expectations.

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