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Sección
Artículos de revisión

Modelos animales en la investigación sobre el autismo (y más allá): Una revisión narrativa

Animal Models in Autism Research (and beyond): A Narrative Review

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Abstract

This narrative review critically examines the use of animal models in behavioural psychology research focused on autism spectrum disorder (ASD). Through historical and conceptual analysis of animal experimentation, the article advances a critique of the 'as if' and 'similar to' definitions currently applied to these models and their associated methodologies. The analysis questions whether behavioural psychology experimentation aimed at studying neurodiversity from typically human complex conditions such as autism and schizophrenia meets the three fundamental criteria for ethical animal research: sound scientific basis, demonstrable usefulness, and minimization of suffering. By synthesizing perspectives from neuroscience, ethology, and philosophy of mind, the review argues that psychological-behavioural models of autism in animals are fundamentally problematic due to anthropomorphic fallacies, questionable validity, and significant ethical concerns. While genetic animal models may provide insights into specific biological mechanisms, the construction of 'autistic' animals through behavioural interpretation creates substantial scientific and moral challenges that undermine the translational value of such research and raise serious questions about its justifiability.

Keywords: autism; ASD; animal model; neurodiversity; behavioural psychology.

Resumen

Esta revisión narrativa examina críticamente el uso de modelos animales en la investigación en psicología conductual centrada en el trastorno del espectro autista (TEA). A través de un análisis histórico y conceptual de la experimentación con animales, el artículo presenta una crítica de las definiciones «como si» y «similar a» que se aplican actualmente a estos modelos y sus metodologías asociadas. El análisis cuestiona si la experimentación en psicología conductual destinada a estudiar la neurodiversidad a partir de afecciones complejas

típicamente humanas, como el autismo y la esquizofrenia, cumple los tres criterios fundamentales para la investigación ética con animales: base científica sólida, utilidad demostrable y minimización del sufrimiento. Al sintetizar las perspectivas de la neurociencia, la etología y la filosofía de la mente, la revisión sostiene que los modelos psicológico-conductuales del autismo en animales son fundamentalmente problemáticos debido a falacias antropomórficas, validez cuestionable y preocupaciones éticas significativas. Si bien los modelos genéticos animales pueden proporcionar información sobre mecanismos biológicos específicos, la construcción de animales «autistas» a través de la interpretación conductual crea importantes retos científicos y morales que socavan el valor traslacional de dicha investigación y plantean serias dudas sobre su justificación.

Palabras clave: autismo; TEA; modelo animal; neurodiversidad; psicología conductual.

1. INTRODUCTION

This article originated as a response to some objections that were raised about certain passages in a previous book on autism [1]. Specific criticism was received, which has instead been generalised, concerning animal models and research with animals, in particular concerning the definition of 'psychological' behaviour of the 'autistic mouse'. It must be emphasised from the outset that no one questions the relevance of animal models for understanding certain phenomena and for testing hypotheses for therapeutic intervention. The risk, however, is a 'as if' projection that evades the reality of the 'as if'. And perplexities in this direction are transversally expressed, as we shall see, by leading exponents of emotion psychology, genetics, pharmacology and connectomics. In formulating the reply, and in documenting it, illustrious precedents were found that - despite their extraordinary authority - have been significantly ignored in this regard. The motivation is simple: it is much more convenient and quicker to define an 'animal model of autistic behaviour' and proceed to experimentation, than to take into account a series of distinctions that would deprive research of this experimental tool, because they simply question the results from the ground up. Unable to counter them, one ignores them and moves on.

2. OBJECTIVES

The primary objective of this work is to critically examine the use of animal models in behavioural psychology research, with a specific focus on autism spectrum disorder. The analysis aims to: 1) Evaluate the theoretical foundations of 'psychological' versus 'genetic' animal models; 2) assess the validity and limitations of attributing complex human neuropsychiatric conditions to animal behaviours; 3) explore the ethical implications of behavioural experimentation on animals; and 4) synthesise historical, ethological, and philosophical perspectives on animal modelling.

3. METHOD

This narrative review was conducted through comprehensive searches of major academic databases including PubMed, Google Scholar, and PsycINFO. Search terms included "animal model autism," "autistic-like behaviour," "behavioural phenotyping rodents," "anthropomorphism ethology," "validity animal model," and related terms. The review incorporated historical texts alongside contemporary research to provide a comprehensive critical perspective. Key theoretical frameworks were examined through close reading of primary sources, with particular attention to foundational works in ethology and their relevance to current experimental practices. As a narrative review, the approach is interdisciplinary and seeks to integrate multiple perspectives rather than provide a systematic review of the literature.

4. IN DEFENCE OF THE MOUSE

A defence of the mouse may appear as a radical animalist position that disregards necessity. It does not. But it is good to clarify two things: whenever in history an attempt has been made to justify an act as 'due to necessity' there was

always something underneath that was never as edifying as the apparent stated motivation. Protocols for animal experimentation require such experimentation to be necessary, to minimise suffering, and to be based on sound theoretical foundations. Researchers presumably take every precaution to minimise suffering. Safety protocols should also be maintained at the highest level: one never knows what might happen if a bat treated with a Sars variant were to come out of a laboratory in China. When discussing experimenting on a 'genetically modified' mouse to see what happens with a drug that expresses the protein that that mouse does not produce because that gene has been suppressed, there is an adequate theoretical basis for trying this route. But when discussing 'psychological models of behaviour', the theoretical basis is at least more fragile than genetically controlled modification between several generations developed in a laboratory. This fragility does not justify the suffering - of a mouse, or of a fruit fly - which shows (according to the subjective interpretation of a researcher) a behaviour of isolation and lack of social interaction and is therefore likened to autism. That mouse is - if we take due account of its nature as a mouse - much more likely to be terrified, desperate and depressed. Nothing will tell us about autism. And this is what makes the result of the consequent research fragile. And it is this that makes it 'unhelpful' with respect to its stated aims.

A distinction of this kind was already made forty years ago by Eric Kandel, who can hardly be accused of being against animal experimentation. Kandel stated: *"In the course of the exposition, I preferred to go beyond the examination of animal studies to put forward hypotheses on human behaviour, in an attempt to emphasise two points that I consider fundamental for the future study of the cellular mechanisms of anxiety. The first is the power of experience over brain functioning, which it can modify by affecting synaptic efficiency and regulating gene expression. The second is the utility and potential of animal models in the study of anxiety. Unlike schizophrenia, which is not an exaggeration of a normal adaptive process and is therefore a typically human mental disorder, fear and anxiety express a universal adaptive mechanism, which is observed in both complex and simpler animals. There is good reason to believe that some cellular mechanisms of anxiety are also universal"* [2].

This quote is very important, for several reasons.

First, the difference between the animal 'genetic' model and the psychological and behavioural model becomes clear.

Then 'what' can be studied in the behavioural part, i.e. what Kandel calls 'a universal', is identified. And in this regard there is a specific passage that needs to be emphasised *"Unlike schizophrenia, which does not constitute the exaggeration of a normal adaptive process and is therefore a typically human mental disorder, fear and anxiety express a universal adaptive mechanism, which is observed in both complex and simpler animals."* The issue is very relevant.

We now know from the mapping of the multiplicity of genes involved that schizophrenia and autism are 'cousin', closely related connectopathies, involving many genes in common. But we also know that they are typically human. And what on a mental and psychological level is typically human, for better or worse, it makes no sense for it to be tested on an animal model of psychological behaviour. Be it autism - the case we started with - schizophrenia, or any other 'typically human mental disorder'. This modelling - as much as 'it would be nice to have it', insofar as it could also be useful - is a fiction of anthropomorphisation that removes much of the basis from the research that is based on it, and in the process creates enormous suffering for animals.

5. THE ANIMAL MODEL AND HUMAN PATHOLOGY

Appropriate animal models of human diseases are of fundamental importance for understanding their aetiology, pathogenesis and treatment. While models for diseases that have biological markers are easily defined, non-genetic animal models for neurobehavioural and neuropsychiatric disorders generally lack biological markers. It emerges that autistic-like behaviour is not easily defined because specific neurobehavioural characteristics in an animal do not exactly replicate human behaviour. Nevertheless, valid behavioural tests have been developed, especially in rodents, to measure human-like behavioural deviations. And genetic and environmentally induced models of behavioural deviations similar to those observed in human autism have been developed. These have made it possible to study the aetiological, pathogenetic and therapeutic aspects of autism (or rather the supposed autistic-like behaviour). Such patterns,

which mimic autistic-like behaviour, exist not only in rodents but also in primates and zebrafish. However, we should be cautious in our neurobehavioural evaluations to be sure that accurate models actually meet most of the clinical behavioural manifestations of human autism.

6. ANIMAL MODELS AND ANTHROPOMORPHISM

6.1. The essential and the questionable

Animal models and animal experimentation are essential for understanding mainly biological, biochemical, genetic, functional, physiological and neurophysiological mechanisms. They are almost indispensable; and precisely for this reason, support exists for all bioethical regulations aimed at minimising the suffering (which there is!) of other living beings. And this is precisely because - unfortunately - it is essential and unavoidable research. On a concrete level, it is evident that a genetic deletion, a selective addition, a neuro-bio-chemical experiment, for example on mice, can give us answers - real and concrete - that nothing else - at present - can provide. What is questioned is the 'psychological' model (for example) of the 'autistic mouse' or the 'autistic zebra fish'. That is, that idea that the behavioural responses of a mouse or fish can be defined as autistic from a psychological-behavioural analysis point of view. No other animal has a human brain, and to the extent that other animals have worlds that overlap with ours, their worlds will function differently from ours.

6.2. The problem of consciousness and interpretation

This forms the answer to Thomas Nagel's question about what it feels like to be a bat: we simply cannot know [3]. It takes a bat brain, with a bat body, with the senses and inferences of a bat, to know what it feels like. A bat will never know what it feels like to be a man (or a monkey, or a dog) any more than we can know what it feels like to be a bat. And it is not a question of what consciousness is. The widespread use of analogy with human behaviour to scientifically demonstrate animal psychology, if not animal consciousness, derives in large part from Darwin and the social climate in which he lived. Darwin is one of those authors that everyone seems to know, whose theses seem to be of collective heritage, but very few take the trouble to read seriously themselves. A recent survey showed that 50 per cent of Americans do not believe in evolution, for example. Some time ago, while reviewing Jerry Fodor and Massimo Piattelli-Palmarini's book, What Darwin Got Wrong [4], it was noted that in over 250 pages, there was not a single quotation from Darwin, from any of his books. Worse still, if one thinks about Social Darwinism, one easily finds that nothing is further from Darwin's ideas. The classics should be read, perhaps one might actually discover something new. In 'The Origin of Man' [5] he observed that 'there are no fundamental differences between human mental faculties and those of higher mammals'. However, instead of categorising animal characteristics of the human mind, he treated animal minds in human terms. Writing about Darwin's difficulties, Elizabeth Knoll [6] argued that he was concerned about the sometimes hostile reception of his theories on evolution, and therefore hoped that this 'more light-hearted view' would lead people to welcome his theory more favourably.

The rampant recourse to anecdotes about the mental causes of animal behaviour continued into the 20th century, and was one of the factors that led to the birth of behaviourism and thus to the rejection of mental states as explanations of behaviour in psychology. In the mid-twentieth century, when the influence of behaviourists began to wane, anthropomorphic explanations of animal behaviour unfortunately made a comeback: mice no longer pressed the lever for reinforcement, but for 'pleasure'; and when in danger, they froze out of 'fear'. Terms that humans invented to describe their own kind of mental states are used widely, and often without discrimination, in science to explain animal behaviour. Some scientists are proud of their anthropomorphism. As LeDoux [7] put it: *"claims about animal consciousness, when based on intuitions and beliefs corresponding to common sense and tradition, seem correct; and when they are authoritatively reaffirmed by scientists, they are considered obviously factual, to the point that no reasonable person would dare question them."*

6.3. The persistence of anthropomorphic thinking

Today's anthropomorphism thrives largely because modern biology credited the idea that it is scientifi-

cally permissible to assign emotions and other human mental states to animals on the simple basis of similarities between their behaviour and ours. It must also be said that if we 'start with Darwin' and carefully read his - forgotten for about a century!) 'The Expression of the Emotions in Man and Animals' [8], he seems to be looking precisely for 'universals' (to which Kandel referred) and not for similarities and overlaps at all costs. John Stodart Kennedy [9] offers an account of why we are so prone to anthropomorphism: *"Anthropomorphic thinking [...] is inherent in us [...] it is culturally inculcated in us from very early childhood. It has also supposedly been "pre-programmed" into our hereditary make-up by natural selection, perhaps because it has proved useful in predicting and controlling animal behaviour."*

Our language has a strong anthropomorphic component, and consequently our concepts and thoughts also go in this direction. If Stodart Kennedy is right, anthropomorphism is part of human nature and is perhaps the reason why we all see 'human' emotions in our animals. Bertrand Russell famously said that 'all animals observed closely exhibited behaviour that confirmed the philosophy the observer believed before starting the observation'. Behaviours attributable to conscious states in other animals often find simpler cognitive or behavioural explanations that do not involve conscious control of the behaviour. Since we are profoundly anthropomorphic, it is often impossible not to appeal to the knowledge we have of our own minds to understand what infants or animals might consciously experience. Assuming that they are experiencing something similar to what we might experience in a similar situation is the appropriate moral reaction. But it is not appropriate as a scientific answer.

6.4. Challenging perspectives on animal models

It is also important here to cite a very authoritative argument that would seem at first sight to refute the thesis and support an 'autistic animal model'. Reference is made to the studies of Jaak Panksepp, and in particular an excerpt from his last work, published posthumously [10] is quoted:

"We have learned more about the fundamental neuronal nature of human emotions by studying the brains of laboratory rats than that of humans. [...] One of the main advantages of laboratory rats is their combination of docility, fecundity and rapid maturation, as well as the fact that they have all the organs and brain systems of humans. [...] Much of what we know about the personality of rats comes from breeding for extreme emotional traits. [...] In conclusion, yes, rats do have personalities. They do indeed exhibit complex personalities, including the expression of different levels of maternal caring behaviour, which in turn has a direct impact on the stress tolerance of the offspring. Rats also share the capacity for two types of attack behaviour: a predatory, silent attack, which is linked to the search system, and an attack behaviour aimed at defending oneself and one's resources, which is linked to the anger/collude system. [...] Rats possess a complex fear system, which is easily activated and intertwined with all the other emotional brain systems: search (curiosity), anger/collude (defence), care (maternal care), panic/anger (separation stress), and play (playful social interaction). [...] With the genetic revolution we now have the solid certainty that the recipe for life is very similar for all mammals, indeed for all species. [...] The view of simple genetic determinism has been replaced by the realisation that heredity is no longer as predetermined as once thought. Genetic science has finally revealed the joint roles of nature and culture in guiding who we are and who we can become. And, in personality theory, exaggerated biological reductionism must now be complemented by new forms of environmental relativism."

There is belief that mammals have 'a personality' (this too is anthropomorphism: we speak of personality to indicate a subjective behavioural tendency, even when speaking of animals other than people!) and they certainly have emotions. There is ample evidence that basic emotions and instincts are inherited by us, rather than 'other mammals having them'. A set of complex emotions are the emerging fruit of possessing a nervous system. As long as we speak of 'emotional mental states' and primary emotions and instincts, therefore, the reasoning is consistent with scientific findings. After all, it is unscientific to believe that something as essential for survival as emotions is only human. Evidence shows that it is man who has inherited them from universal emotions present since bacteria. The broader reasoning remains separate: for 'personalities' of a complex type (for which self-consciousness, self-reflexivity and a 'theory of mind' are required simultaneously: all together) in addition to a central nervous system,

something 'extra' is required, such as a human prefrontal cortex.

At present, it seems very difficult to argue that a mouse, no matter how evolved and complex, or a zebra fish, can have a complex as articulated as autism. This does not detract from the fact that the mouse model may offer an essential basis for understanding the effects on behaviour of gene deletion, or responses to the administration of a neuromodulator. However, it is not enough for a mouse to be depressed 'as if' it were a human being; it is not enough for a mouse to behave 'as if' it were autistic to define it as such 'on the level of the human condition'. Sebastian Seung [11] also spoke on the subject, referring to what he called connectopathies:

"Studying human mental disorders using animals is not an easy task. The rabies virus causes the same disease, whether it infects rabbits, dogs or people. But is there such a thing as an autistic or schizophrenic animal? We do not know whether such animals exist in nature. We are, however, trying to create them through genetic engineering. We insert defective genes associated with autism or schizophrenia into the genome of animals - usually mice - with the prospect of creating similar disorders. In theory, these creatures would be 'models' of human pathologies, approximations of the real thing. [...] The fact is that this strategy, a variation of Pasteur's, sometimes fails even in infectious diseases. [...] Similarly, it is not necessarily the case that inserting defective human genes into animals causes autism or schizophrenia. Perhaps similar but different genetic defects are necessary. [...] These indeterminacies have brought to light the problem of validating animal models for mental disorders, but it is unclear which criteria to use. Some researchers emphasise similarity of symptoms; the fact is that even in infectious diseases the criterion does not always work. Sometimes the same microbe infects animals as humans, but producing very different symptoms: an animal might tolerate the infection with minimal side effects. [...] And if human genes for autism or schizophrenia produced very different symptoms in mice, it would not necessarily mean that rodent models are useless. Incidentally, it could be argued that there is no point in comparing symptoms, because mental disorders involve exclusively human behaviour. Similarly, similarity of connectopathies could be a valid criterion for animal models of disorders such as autism and schizophrenia. But it is clear that for similarity to work, we would have to identify in animal models connectomies similar to those of autistic or schizophrenic patients. [...] Comparing connectomes is a different project from wanting to decode them. Connectionist memory theory proposes specific hypotheses. Conversely, connectionist theory is an open field. What if, without specific hypotheses, the search for connectomics is in vain? [...] If autism and schizophrenia turn out to be caused by connectopathies, it will be important to identify similar wiring defects in animal models: at that point, drugs could be proven effective in preventing or correcting these defects. But for this to become feasible, we will have to accelerate the technologies of connectomics to rapidly compare many animal brains."

As can easily be seen, no one questions the experimental relevance of animal models for understanding certain phenomena and for testing hypotheses for therapeutic intervention. The risk, however, is a 'as if' projection that evades the reality of the 'as if'. And misgivings in this direction are transversally expressed by leading exponents of emotion psychology, genetics, pharmacology and connectomics. Perhaps this should be taken seriously.

7. MURINO 'GENETIC' AND 'PSYCHOLOGICAL' MODEL OF AUTISM

The recent study by Zhao and colleagues [12] clearly highlights the limitations when switching from the genetic to the 'psychological' mouse model. The study aimed to highlight the correlations of brain structure with social behaviour in mice with duplication 15q11-13, an animal model of autism. Chromosome 15q11-13 duplication has been reported as one of the most frequent cytogenetic copy number variations in autism spectrum disorder, and a mouse model of paternal 15q11-13 duplication, called 15q dup mice, was generated. While previous studies have separately replicated some of the behavioural and structural phenotypes of the autism brain, the relationship between brain structure and behaviour has rarely been examined. In this study, Zhao and colleagues performed behavioural experiments related to anxiety and social behaviour and magnetic resonance imaging (MRI) using the same set of 15q dup and wild-type mice. The 15q-dup mice showed increased anxiety and a tendency towards alterations in social behaviour, as reported previously, as well as variability in socia-

bility. MRI analysis revealed that a lower sociability index correlated with a lower grey matter volume in the right medial entorhinal cortex. These findings may help to understand how variability in the behavioural phenotypes of autism manifests itself even in individuals with the same genetic background and to determine individual differences in the neuroevolutionary trajectory related to specific brain structures that underlie these phenotypes.

As the researchers themselves pointed out: *"... this study has several limitations. First, it should be noted that no consistent results have been observed in previous studies on 15q-dup mice ... These inconsistent results could be due to differences in age or in the number of animals used in each experiment. Secondly, all mice used in this study were anaesthetised and immobilised during the MRI scan one week prior to the behavioural experiments. It is possible that these treatments influenced our behavioural results, even though we established a one-week interval between MRI and behavioural experiments to allow for recovery."* But it is also possible that quite simply the psychological 'as if' model does not provide an adequate experimental model for a complex state such as human autism, however much the genetic model tends to be valid.

8. REREADING LORENZ FOR THOSE WHO WERE ABSENT

8.1. The foundational critique of laboratory conditions

Konrad Lorenz can be considered the founder of modern scientific ethology. It is worth remembering that ethology is not just a branch confined to the biological study of animals in nature. Leading psychologists and psychoanalysts, such as John Bowlby, include an ethological approach in its own right in the complex of analytical strategies. Precisely because of this bridge between the study of animals in nature and the analysis of human (and other) behaviour, it is useful to 'go back to Lorenz', in the same spirit in which it is necessary to go back to reading 'the classics'. Reference is made here to a single text, 'Ethology' (Lorenz, 1978/2011), in which a large number of studies and research papers are cited. As Lorenz pointed out, one must always bear in mind what side effect on the whole system, and thus what feedback on the subsystem under study, can be caused by an experimental intervention. This problem is all the easier to solve the more the part being studied has the character of a component in relative independence from the whole. Otherwise, and in general, it must be borne in mind that an experimentally provoked variation in state can only have an identical and predictable effect, in a second experiment, if the whole system is, at the two times when the intervention has taken place, in exactly the same state. If the system being studied is a relatively small organic subsystem, one may succeed in achieving experimentally the equality of all the circumstances present, but this method yields good results in special physiology. In contrast, the system of sensory performance and central nervous functions, which is responsible for the behaviour of higher animals, is one of the most complex systems we know of. It seems a naive idea to be able to achieve completely identical conditions and processes in an intact, healthy organism by placing it under constant, controllable external conditions. A higher animal needs innumerable, often quite complex, stimulating effects acting continuously in order to maintain its state of health and to be able to exhibit non-pathological behaviour. The 'controlled laboratory conditions', created to get an idea of the possible effects of stimuli, inevitably eliminate an unpredictable number of stimulating situations that are indispensable for the animal and at the same time offer an abnormal and chaotic amount of stimuli.

Of the research cited by Lorenz, in particular that of Anne Rasa, in her classic work on the spontaneity of aggressive behaviour in the coral fish Microspathodon chrysurus, showed experimentally how absolute environmental constancy causes a pathological decrease in general excitability. Already in the late 1970s, despite being among the most committed experimenters, observers and researchers of animal behaviour - or perhaps because he was - Lorenz warned: *"Anyone who has tried to keep higher animals in captivity, so that the monotony of captive conditions does not cause such a decrease in general irritability, has an idea of how indispensable the continuous change of non-specific environmental stimuli is. Considering the unpredictable changes caused in the behaviour of even the mildest semi-activity of higher animals, all attempts to 'control' environmental conditions appear futile. Precisely for this reason, many researchers, who are intimately aware of the systemic character of their object, even shy away from experiments"* (Lorenz, 1978/2011).

8.2. The divide between experimental and ecological approaches

On the subject of the piscological study of animal behaviour, Lorenz literally ruled that: *"Examining the most modern behavioural studies literature, one could almost get the impression that the attributes 'systemically oriented' and 'experimental' contradict each other and can never be applied simultaneously to the same research. Too many experimental ethologists, who work exclusively in the laboratory, as a large proportion of American psychologists do, are indeed quite gifted for experiments, but often have no idea of biology and ecology, whereas, conversely, some behavioural scholars with a well-established basis in biology and ecology devote themselves only to observation in nature and tend to reject experiments, especially under laboratory conditions"* (Lorenz, 1978/2011). One could retort - as is often done - that these considerations were valid 'in Lorenz's time', but this retort is in itself unfounded: animal nature has not changed in the last fifty years, nor have these levels of complex behaviour, and no experimental variation in the laboratory or technological innovation can circumvent these premises. But it is also easier to realise that - quite simply - many experimenters on autistic Lorenz mice and zebra fish have not read him.

Faced with this state of affairs, Lorenz in turn quoted the advice of Fritz Knoll (1921/1922/1926) more than fifty years earlier: the experimenter must, first of all, acquire a thorough knowledge of the general life habits of the animals to be studied. This can only be achieved by prolonged and rigorous observation in their natural environment and of the relevant fauna (or flora); only after such a preparation should one move on to the execution of an experiment... first of all, it will be good to carry out the planned experiments as far as possible in the natural locality... certain experiments, for which the original environment is not suitable, will be carried out in the open air in other places, more suited to the case. According to Lorenz, these indications apply, without limitation, to the study of behaviour in general. *"No common behavioural form of a common animal could be understood in any other way than in relation to the ecology of its species. These concepts even apply to the pathology of behaviour, since the pathological can only be defined by reference to ecological concepts"* (Lorenz, 1978/2011). It is a widespread error to consider the simplest cases as most frequent (and therefore always taken as examples in textbooks). A higher animal in its natural environment must always maintain a disposition to several different behavioural forms that are often mutually exclusive, and what it does is almost always a compromise between several different needs.

8.3. The complexity of associative learning

Wilhelm Wundt (1897/2009) by association means 'links between contents of consciousness that ... have the common character of involuntary processes of consciousness, i.e. which occur in a state of passive attention'. Associations arise when two processes are provoked once or more in the same succession and in a short interval of time. We therefore speak of a law of succedaneity and a law of contiguity; both apply to a very large number of the processes we are about to discuss, if not to all of them. The fusion or coupling of two psychic, and therefore also nervous, events that follow one another has the effect that the organism, as soon as the first event has arrived, 'waits' for the second, i.e. prepares itself for it: Pavlov's dog begins to emit saliva when it hears the sound of the bell it has associated with food. Those who study association processes in the laboratory offer the 'conditioned' stimulus to which they want to train the animal (e.g. the ringing of the bell) immediately before the 'unconditioned' stimulus (e.g. food) in time. According to Lorenz, regarding this man-made regularity, it is easy to forget that in natural conditions a regular and direct temporal succession of two or even more events occurs in only one case: when a causal link exists between them. The capacity for association is an adaptation: the similarity, indeed the equality of function, has led great thinkers to confuse these two processes, even though they occur at very different levels of integration of nervous processes. It is by no means a logical consequence that, if two complex stimulating situations have occurred two or more times 'in succession and in contiguity', they must also do so in other cases and forever. It is only the a priori necessity of our thinking to be deductive that leads us to assume that: *"perhaps it is also the artificiality of the 'constant and controllable experimental conditions' (the aspiration of every experimenter), which favours unnaturally rapid desensitisation. In certain cases, the limitation of afference linked to complex*

perceptual processes can convey very specific adaptive information, informing the organism not, as is usually the case, about what is not dangerous but, conversely, making it selectively alert to stimuli that threaten danger. It therefore assumes the role usually played by sensitisation" (Lorenz, 1978/2011).

If the dog is freed from the fetters with which it was restrained in Pavlovian experiments, as Howard Lidell (1954) did, it is immediately seen that not only its salivary secretion is activated, but a whole, very peculiar system of appetitive behaviour, that is, the system by which the dog begs for food from its master, as the wolf does from the older members of its pack: It runs towards the source of the stimulus, be it a bell or a metronome or some other object, and begs for food by wagging its tail and barking: these, as Hassenstein (1970) puts it, are *"behavioural elements that could not have been learned as such in the given experimental situation, not least because they were not possible at all in that situation".* The information, obtained by training, that certain situations of gustatory stimuli herald an illness, can only be had because of the consequences on the vegetative system, whereas other punitive stimuli can more easily be associated with behavioural modules other than feeding. Lorenz comments in this regard: *"such was the force of ideological prejudices against the fact that behaviour is phylogenetically programmed ... that the publication of Lidell's important observation was prevented"* (Lorenz, 1978/2011).

9. WHAT DOES IT FEEL LIKE TO BE AN OCTOPUS?

Deliberately paraphrasing Nagel, the argument about octopus is made with the help of Peter Godfrey-Smith (2016) because octopuses, cephalopods in general and even more so molluscs and invertebrates in general are widely the subject of experimental research. According to Godfrey-Smith: *"much of the animal research has been conducted on the assumption that all individuals of the same species will behave very similarly until they encounter different gratifications, and that in order to obtain the same small morsel of food they will continue to peck, run or pull a lever all day. [The experimenters wish] to work in this way because they are determined to use what they call 'objective and quantitative methods of study'. There is full agreement. However, octopuses - much more than rats and pigeons - have their own ideas*".

As numerous anecdotes show, octopuses have a certain ability to adapt to the special circumstances of an aquarium and interaction with humans. They are rather solitary animals. Often in the laboratory, octopuses are quick to understand how life works in their new condition. We know, for example, that octopuses in captivity are able to distinguish single individuals among the keepers, and behave differently with each of them. In 2010, an experiment confirmed that giant Pacific octopuses can indeed recognise individual humans, and are able to do so even if the latter are wearing identical uniforms (Anderson et al., 2010).

According to Stefan Linquist (Godfrey-Smith 2016) *"when you work with fish, they have no idea that they are in a tank, in an unnatural situation. With octopuses it's a different matter: they know very well that they are in this particular place, and that you are out there. All their behaviour is influenced by the knowledge that they are in captivity."*

The problem with the old experiments by behaviourists 'a la Skinner' on octopuses - which led them to believe that they were not at all intelligent - is that as they were conceived, they assumed that an octopus would be interested in repeatedly pulling a lever to get pieces of sardine, thereby picking up a large amount of a second-rate food. Rats and pigeons do things like that, whereas octopuses take a while to process each piece of food, probably cannot binge, and tend to lose interest. At least for some of them other research and testing activities are more interesting... like splashing patrons. To overcome the difficulty of motivating octopuses, some researchers used negative reinforcements such as electric shocks in a greater form than they would have done with other animals. During much of the early research, octopuses were not only subjected to electric shocks, but in many experiments parts of their brains were removed, or important nerves severed, just to see what they would do once they woke up. Until recently, they could even be operated on without the use of anaesthetics. As invertebrates, they were not protected by regulations against cruelty to animals. In recent years, especially in the European Union, they have also been introduced into the rules governing the treatment of animals in experiments, almost as if they were honorary vertebrates (Directive 2010/63/EU).

10. BEHAVIOURAL ASSESSMENT IN ANIMAL MODELS

The problem of clarity on this issue is very central and debated in the current neuroscientific context. If the model is wrong, unreliable or unverifiable, all research and derived results are to be questioned. Assuming therefore that appropriate animal models of human diseases are a cornerstone in the advancement of science and medicine, creating animal models of neuropsychiatric and neurobehavioural diseases such as autism requires the development of sufficient neurobehavioural measurement tools to translate human behaviour into measurable behavioural characteristics expected in animals. If possible, the severity of the symptoms should also be assessed. At least in rodents, neurobehavioural and neurological tests have been developed. As autism is characterised by a number of specific behavioural tendencies with significant severity, animal models of autistic-like behaviour must demonstrate the specific features, i.e. impaired social interactions, communication deficits and restricted and repetitive behavioural patterns, with association with various additional impairments such as somatosensory, motor and memory impairments. Therefore, an appropriate model must show the behavioural impairment of a number of neurobehavioural characteristics using an appropriate number of behavioural tests.

For the aetiological aspects, models were developed using immunogenic substances such as polyinosinic-polycytidylic acid (PolyIC), lipopolysaccharide (LPS) and propionic acid, or other well-documented immunogens or pathogens such as Mycobacterium tuberculosis. Another approach is the use of chemicals such as valproic acid, polychlorinated biphenyls (PCBs), organophosphate pesticides such as chlorpyrifos (CPF) and others. These substances were administered prenatally, generally after the main organogenesis period, or, especially in rodents, during early postnatal life. Furthermore, using modern methods of genetic manipulation, genetic models have been created of almost all human genetic diseases that manifest themselves as autism-like behaviour (e.g. fragile X, Rett syndrome, SHANK gene mutation, neuroligin genes and others). Ideally, we should not only evaluate the different behavioural modes affected by autism-like behaviour, but also assess the severity of behavioural deviations by means of an appropriate scoring system, as applied to humans. Three researchers set themselves this goal very recently: Asher Ornoy, Boniface Echefu and Maria Becker (2024).

In general, animal models for human disease must fulfil three basic values: apparent validity, when animals recapitulate the disease phenotype in a similar way to humans; aetiological validity (construct and relevance), when the patho-physiological processes in animals are similar to those that cause the disease in humans; and predictive validity (pharmacological sensitivity), when animals respond to drugs that are effective in treating the human disease (Nestler & Hyman, 2010). Often the causality of human diseases and disease in animal models are similar, as are the symptoms, complications and treatment. Thus, there are genetic and non-genetic animal models used to study almost all human diseases. The 2013 Diagnostic and Statistical Manual (DSM-5) provides standardised criteria for diagnosing autism (American Psychiatric Association, 2013). The diagnostic features associated with autism are a triad of impaired social interactions [1], verbal and non-verbal communication deficits, and restricted and repetitive behavioural patterns that may also be associated with somatosensory and special sense impairments. Careful phenotypical characterisation of animal models of autism is essential to ensure that they accurately summarise key features of the human disorder. This includes the assessment of behavioural, cognitive, social and communicative deficits that are relevant to human symptoms. If there are only a few behavioural changes or the behavioural tests have not been applied sufficiently to assess most of the typical autism-like behaviour, the similarity to human autism is incomplete. Modelling neuroevolutionary disorders such as autism in animals is challenging and complex because the aetiology and pathogenesis of autism are multifactorial and still unclear (Sarovic, 2021). A significant difficulty is that autism is currently diagnosed on the basis of a number of fundamental behavioural abnormalities rather than objective biomarkers (Frye et al., 2019).

There is a wide variety of studies describing different patterns of autistic-type behaviour in animals, particularly rodents. Researchers have generally used a variety of well-accepted behavioural tests that have demonstrated various autistic-type characteristics. In many models, however, only some of the typical behavioural characteristics have been assessed. In none of these studies were the behavioural deviations classified according to their severity, as required for the diagnosis of autism in humans. Many of the tests used in humans to di-

agnose autism use a scoring system for different behaviours with a gradual transition from normal to abnormal scores. The score generally also defines the severity of the symptoms. This is true for common autism diagnostic tools such as the Child Autism Rating Scale (CARS), the Autism Diagnostic Observation Schedule (ADOS), the Autism Diagnostic Interview-Revised (ADI-R) and other diagnostic tools (American Psychiatric Association, 2013). It should at least be considered appropriate that animal models of autism-like behaviour also demonstrate measures similar to those used in humans. However, autism-like behaviour is a combination of changes in several obligatory behavioural traits at different degrees of severity. It may therefore be important to use a sufficient number of behavioural tests to describe most of the autism-specific behavioural changes. Studies that use too few tests (e.g. only tests for social interaction and communication or repetitive behaviour and anxiety) should at least be considered insufficient for the correct identification of an appropriate autism-like behavioural pattern. These studies can be used, if anything, to define specific traits of autism-like behaviour; e.g., communication difficulties, repetitive behaviour, restricted interests, abnormal response to sensory stimuli and others, but not the complete diagnostic set of autism-like behaviour. To the best of knowledge, there is no accepted scoring system for defining autism-like behaviour in animal models, and most studies only demonstrate some of the behavioural deviations considered typical of autism-like behaviour, without assessing their severity (El-Kordi et al., 2013).

Especially in non-genetic 'autism-like' models, it is expected that the severity of autism-like behavioural changes will differ between offspring of the same treated mother. Thus, an accepted scoring system or at least a definition of severity, similar to that in humans, seems to be mandatory. This is apparently true for all animal models that mimic human neurobehavioural and neuropsychiatric diseases.

In their study, Ornoy, Echefu and Becker make a concrete proposal for an unambiguous (possibly shared and homogeneous) scoring system: *"Autism in humans is only defined if there are also changes in at least two non-communicative characteristics such as repetitive behaviour and restricted interests. Thus, in animal models, at least one of these behaviours must be abnormal. Similar scores from 0 to 3 should be used with 2 and 3 defining abnormal behaviour. Other associated features such as cognitive impairment (including memory and spatial learning), anxiety, impaired motor coordination or sensory impairment are not mandatory, but if tested, similar scores from 0 to 3 should be used for each test, with normal (0), mildly abnormal (1), moderately abnormal (2) and severely abnormal (3). At least one of these should be abnormal. […] To summarise, the minimum score defining autistic-like behaviour in all mandatory domains is 5 and the maximum is 12. If a model does not meet the minimum required score, it defines specific behaviours (e.g., social impairment, restricted interests, anxiety, etc.) but is not a complete model for autistic-type behaviour"* (Ornoy et al., 2024).

The - common sense - consideration is basically that defining a score for 'autism-like' behaviour would encourage all researchers to use at least four different behavioural tests to appropriately assess models for 'autism-like' behaviour. Using fewer tests will define individual 'autism-like' behaviours, but they are not models for most or all characteristics of 'autism-like' behaviour. It would also allow us to define the animals presenting 'autism-like' behaviour in the litter and to carry out the planned specific studies only on those presenting the lowest score. It would also allow a better evaluation of the possible benefits of the preventive and/or therapeutic modalities used in these models. Defining a score for 'autism-like' behaviour would encourage researchers to use sufficient behavioural tests to appropriately evaluate comprehensive models for 'autism-like' behaviour.

11. AN ETHOLOGICAL VIEW OF PSYCHOLOGY

Konrad Lorenz can certainly be considered the founder of modern scientific ethology. It is worth remembering that ethology is not just a branch confined to the study of biology and animals in nature. Leading psychologists and psychoanalysts, such as John Bowlby, incorporate a fully-fledged ethological approach into their analytical strategies.

Precisely because of this bridge between the study of animals in nature and the analysis of human behaviour (and not only), it is useful to "return to Lorenz", with the same spirit with which it is necessary to return to reading "the classics", those that everyone thinks they know but often only through second, third or fourth hand (and in-

terpretation). Reference is made here to a single text, "Ethology" (Lorenz, 1978/2011), which cites a large number of studies and research projects.

As Lorenz pointed out, we must always bear in mind what side effects an experimental intervention may have on the entire system and, therefore, what feedback it may have on the subsystem under study. This problem is easier to solve when the part being studied is relatively independent from the whole. Otherwise, and in general, it must be borne in mind that a change in state caused experimentally can have an identical and predictable effect in a second experiment only if the entire system is in exactly the same state at the two moments of intervention. If the system being studied is a relatively small, entirely organic subsystem, it may be possible to experimentally achieve the equality of all present circumstances, but this method yields good results in special physiology. The system of sensory performance and central nervous functions, which governs the behaviour of higher animals, is, on the contrary, one of the most complex systems we know. It seems undoubtedly naive to think that it is possible to obtain completely identical conditions and processes in a healthy, intact organism by placing it in constant and controllable external conditions (strictly controlled laboratory conditions). A higher animal needs countless stimulating effects, often quite complex, acting continuously, in order to maintain its state of health and be able to exhibit non-pathological behaviour. Controlled laboratory conditions, created to gain an idea of the possible effects of stimuli, inevitably eliminate an unpredictable number of stimulating situations that are essential for the animal and, at the same time, offer a quantity of abnormal and chaotic stimuli.

Among the research cited by Lorenz, in particular that of Anne Rasa, in her classic work on the spontaneity of aggressive behaviour in the coral fish Microspathodon chrysurus, she experimentally showed how, even in this low vertebrate, absolute environmental constancy causes a pathological decrease in general excitability. Already in the late 1970s, despite being one of the most committed experimenters, observers and researchers of animal behaviour – or perhaps precisely because he was – Lorenz warned: *"those who have tried to keep higher animals in captivity, so that the monotony of captivity does not cause such a decrease in general irritability, have an idea of how indispensable the continuous change of non-specific environmental stimuli is. Considering the unpredictable changes caused in the behaviour of higher animals even by the mildest semi-captivity, all attempts to 'control' environmental conditions appear futile. Precisely for this reason, many researchers, who are intimately aware of the systemic nature of their subject, even shy away from experiments."* With regard to the psychological study of animal behaviour, Lorenz literally stated that: *"examining the most modern literature on behavioural studies, one could almost get the impression that the attributes 'systemic' and 'experimental'contradict each other and can never be applied simultaneously to the same research. Too many experimental ethologists, who work exclusively in the laboratory, as do most American psychologists, are in fact very gifted at experimentation, but often have no idea about biology and ecology, while, conversely, some behavioural scientists with a well-established background in biology and ecology devote themselves only to observation in nature and tend to reject experimentation, especially in laboratory conditions."*

One could reply – as is often done – that these considerations were valid 'in Lorenz's time', but this reply is in itself unfounded: animal nature has not changed in the last fifty years, nor have these levels of complex behaviour, and no experimental variation in the laboratory or technological innovation can circumvent these premises. But it is also easier to realise that – quite simply – many of today's experimenters on autistic mice and zebrafish have not even read Lorenz.

Faced with this state of affairs, Lorenz in turn cited the advice of Fritz Knoll (1921/1922/1926) from fifty years earlier: the experimenter must, first of all, acquire a thorough knowledge of the general habits of the animals to be studied. This can only be achieved through prolonged and rigorous observation in their natural environment and of the relevant fauna (or flora); only after such preparation should one proceed to carry out an experiment… first of all, it is advisable to carry out the planned experiments, as far as possible, in the natural location… certain experiments, for which the original environment is not suitable, will be carried out outdoors in other places more appropriate to the case. According to Lorenz, these guidelines apply without limitation to the study of behaviour in general. *"No common behavioural pattern of a common animal can be understood except in relation to the ecology of its species. These concepts apply even to*

behavioural pathology, since pathology can only be defined by reference to ecological concepts."

It is a widespread mistake to consider the simplest cases (and therefore always taken as examples in textbooks) to be the most frequent. In nature, behavioural patterns with simple motivations are no more frequent than monohybrid bastards. A higher animal in its natural environment must always maintain a disposition towards several different behavioural patterns that are often mutually exclusive, and what it does is almost always a compromise between several different needs. Wilhelm Wundt (1897/2009) uses the word "association" to mean "connections between contents of consciousness that... have the common character of involuntary processes of consciousness, i.e. that occur in a state of passive attention". Associations arise when two processes are triggered one or more times in the same sequence and within a short interval of time. We therefore speak of a law of succession and a law of contiguity; both apply to a very large number of the processes we are about to discuss, if not all of them. The fusion or coupling of two psychic, and therefore also nervous, events that follow one another has the effect that the organism, as soon as the first event has occurred, "waits" for the second, i.e. prepares for it: Pavlov's dog begins to salivate when it hears the sound of the bell it has associated with food. Those who study association processes in the laboratory naturally offer the "conditioned" stimulus with which they want to train the animal (e.g. the sound of the bell) immediately before, in time, the "unconditioned" stimulus (e.g. food). With regard to this man-made regularity, it is easy to forget that in natural conditions, a regular and direct temporal succession of two or even more events occurs in only one case: precisely when there is a causal link between them. In the mountains of Armenia, semi-wild goats, as soon as they hear thunder, gallop at full speed towards caves; it is clear that this is a teleonomic behaviour pattern to avoid rain and cold. If these animals do the same when rocks are blown up with mines in the vicinity, as has been seen very often, this appears meaningless. The ability to make associations is an adaptation to the so-called "transformation of force", in other words, to the law of conservation of energy. In this, it is functionally analogous to the human capacity for deductive thinking. The similarity, or rather the equality of function, has led great thinkers to confuse these two processes, even though they occur at very different levels of nervous process integration. It is by no means a logical consequence that if two complex stimulating situations have occurred two or more times "in succession and contiguity", they must do so in other cases and forever. Only the a priori need for our thinking to be deductive leads us to assume this, and only rarely does it lead us astray! Perhaps it is also the artificiality of "constant and controllable experimental conditions" (the aspiration of every experimenter) that promotes unnaturally rapid desensitisation. In certain cases, the limitation of afference linked to complex perceptual processes can transmit very specific adaptive information, informing the organism not, as usual, about what is not dangerous, but, conversely, making it selectively attentive to stimuli that threaten danger. It therefore assumes the role that is usually exercised by sensitisation.

If the dog is freed from the restraints with which it was immobilised in Pavlovian experiments, as Howard Lidell (1954) did, it is immediately apparent that not only is its salivary secretion activated, but also an entire, very particular system of appetitive behaviours, i.e. the system by which the dog begs for food from its owner, as the wolf does with the older members of its pack. It runs towards the source of the stimulus, whether it is a bell, a metronome or another object, and begs for food by wagging its tail and barking: these, as Hassenstein (1970) says, are *"behavioural elements that could not have been learned as such in the given experimental situation, not least because they were not at all possible in that situation*". It cannot be ignored that the publication of Lidell's important observation, cited above, was prevented: such was the strength of ideological prejudices against the fact that behaviour is phylogenetically programmed. The information, obtained through training, that certain taste stimuli herald illness can only be obtained through its consequences on the vegetative system, while other punitive stimuli can be more easily associated with behavioural patterns other than those related to feeding.

12. AN ETHOLOGICAL SUGGESTION FOR REPETITIVE BEHAVIOUR

One of the characteristics of many autistic people is "repetitive behaviour". In reality, this is a very varied and heterogeneous series of behaviours characterised by repetitiveness, ranging from the ritualistic repetition of practices and ges-

tures to obsessive behaviours, 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, a suggestion was found that should be shared and highlighted, which should (strictly) be subjected to further investigation in the field of cognitive-behavioural 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. We know that in this way, starting from a linear course, a regulatory circuit has developed and that, as a result, completely new systemic properties of the nervous system have arisen, even though the pre-existing subsystems, far from being modified or even eliminated, have retained their previous performance and represent essential components in the newly formed regulatory circuit. 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. The function of such a system begins with an appetition, leads to the response of an innate triggering mechanism, and concludes in a final action that discharges the impulse or in a rewarding state of rest. Not only from a purely theoretical, bio-cybernetic point of view is it difficult to imagine a behavioural system that, through backward communication of success, produces a teleonomic improvement without these three members, but also through observation, we know of no behavioural system that can be modified teleonomically by learning based on success and that does not contain these three partial systems. Even in specific instinctive actions that cannot be modified by success or failure, a "feedback communication" apparatus sometimes operates, but this only serves to complete the behavioural module, without communicating anything to the organism about the teleonomy of success. The end of an instinctive movement is by no means always determined by the exhaustion of the specific potential for action, but is often determined by a mechanism that communicates that the action has been completed, such as that constituted, for example, by the proprioceptive afferents of the seminal vesicle in the copulation of the male chimpanzee. 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. To be able to do this, it must possess a large amount of genetic information about both its pupil and the environment in which he lives. The open programme of learning based on success and failure presupposes a highly complex sensory and nervous apparatus containing large amounts of genetically acquired information. The behaviourist school does not ask what is the minimum complexity that a nervous system must have in order to process feedback on success in order to teleonomically improve its function, nor does it consider the possibility that there may be more than one system capable of providing this service: all processes of this type are instead lumped together as conditioning. Behaviourists first of all reject the question of teleonomy with the same energy with which we reject that of teleology. Secondly, however, they hope to be able to formulate laws on learning (and on behaviour in general) of general value, without subjecting themselves to the effort of analysing the complicated physiological mechanism, the performance of which is behaviour and in particular learning. This hope is completely futile because, as cyberneticists well know, it is impossible to understand the performance of a complex information processing system, consisting of variously constructed and functioning parts, by controlling the inputs and statistically calculating the probability with which certain output values emerge from the system. As Mittelstaedt once jokingly said, it is like trying to understand how a ticket machine works without knowing anything about it other than the type of coin that goes in and the type of ticket that comes

out. Things are different if you know more not only about the parts that make up such a device, but also about the partial functions that each of them performs. Hassenstein (1970) proposed a new conceptual subdivision of learning processes in which he included, in his flow chart, the mechanisms recognised by ethologists as independently functioning units. The consistency of his approach, as well as its practical applicability to child education and animal training, leads us to consider it accurate and also testifies to the accuracy of the old ethological concepts.

Without wishing to draw more from these considerations than they explicitly state, we should ask ourselves what "feedback" repetitive behaviour provides, which cannot be trivialised as stereotypy, instinct or an automated mechanism. It is clear, even in the general heterogeneity of repetitive behaviours 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. The thesis – yet to be proven – is that observing repetitive behaviour 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.

13. CONCLUSIONS

The excuse of not knowing that animals suffer, because they have no souls, has allowed surgical experiments on live animals without anaesthesia for centuries. A whole theory has been built on their lack of soul and suffering to justify such experiments. When it became increasingly clear - even though average observation and common sense would have sufficed - that animals, even the apparently simplest ones, experience emotions, including pain and suffering, and experience complex states such as depression, despair and terror, our attitude gradually changed. Today, those who advocate the necessity of animal experimentation emphasise the valuable information and discoveries it can reveal. In this sense, the section on vivisection is being discussed. There is a tendency to consider only clinical and biological testing as 'true', deliberately ignoring and leaving out psychological and behavioural testing. This exclusion is so strong that there are practically no validated scientific studies on psychological suffering in animals subjected to behavioural experimentation, and those few that do exist are published by animal welfare associations, often only on empirical grounds, which - accused of being biased - do not even enter into the discussion. Experimentation based on behavioural analysis is advertised as harmless, without consequences. It gives impressive results at a very low cost. The point is that without validation as described in the last paragraph, experimenters can subjectively derive whatever they like from animal behaviour. An even more expansive perspective when considering not a model of autism or schizophrenia, but of an 'as if/similar to'. If we add to this the comments on behavioural animal experimentation highlighted by Lorenz, the prospect of the model's effectiveness falls even further, when it does not collapse altogether. In between are they, the animals. While we think about 'what 'normal' or 'autism-like' behaviour of a mouse should be like in a certain context, we keep them in a Plexiglas cage without shelter, out of their ethological environment, and consider them autism-like when they are simply terrified, terrified and desperate. The essential point is that while there is no certain proof of the existence of the autistic mouse, on which it is not possible to perform the same tests as an autistic person, there is certain proof of the psychological stress to which these animals are subjected. If there is no certain scientifically validated model of an autistic mouse, all the evidence of the resulting psychological and behavioural analysis is greatly weakened, and with it the expected and hoped-for results. This is confirmed to the contrary: if one knew with certainty how to 'create autism in the laboratory' (and not a how-if, which in terms of neurodiversity makes no sense) then one would know the cause of autism with certainty and this would be due to one or very few factors, which is ontologically at odds with the scientific evidence. The fable of the laboratory-created autistic animal is so beautiful - for what it would allow us to do - that we end up believing it and wanting to believe it. A path already taken by Skinner, Pavlov and Lysenko (in different forms) and disproved by Lorenz and almost all the psychology of the last sixty years. The point is that we want to believe it. The price of this tale is not as visible as vivisection, of course, but it is enormous: incredible suffering (we cannot put it into words, so

we deny it more easily) in over six hundred thousand mice every year, and at least as many birds, fish and octopuses. If the three basic criteria for animal experimentation are a sound scientific basis, a certain usefulness and the least possible suffering, it is argued that in behavioural psychology animal experimentation aimed at studying neurodiversity from typically human complex diseases and conditions such as autism and schizophrenia, all three elements are essentially missing.

REFERENCES

1. Di Salvo M. Autism Research between Psychology and Neuroscience. Cham: Palgrave Macmillan; 2024. ISBN: 978-3-031-68338-1 https://doi.org/10.1007/978-3-031-68338-1
2. Kandel ER. From metapsychology to molecular biology: A study of the mechanisms of anxiety. Am J Psychiatry. 1983;140(10):1277-93. https://doi.org/10.1176/ajp.140.10.1277
3. Nagel T. What Is It Like to Be a Bat? Philos Rev. 1974;83(4):435-50. https://doi.org/10.2307/2183914
4. Fodor J, Piattelli-Palmarini M. What Darwin Got Wrong. New York: Farrar, Straus and Giroux; 2010. ISBN: 978-0374288792.
5. Darwin C. The Descent of Man, and Selection in Relation to Sex. London: John Murray; 1871.
6. Knoll E. Dogs, Darwinism, and English sensibilities. In: Mitchell RW, Thompson NS, Miles HL, editors. Anthropomorphism, Anecdotes, and Animals. Albany: State University of New York Press; 1997. p. 12-21. ISBN: 978-0791431231.
7. LeDoux J. The Four Realms of Existence: A New Theory of Being Human. Cambridge: Belknap Press; 2023. ISBN: 978-0674245130.
8. Darwin C. The expression of the emotions in man and animals. London: John Murray; 1872.
9. Kennedy JS. The New Anthropomorphism. Cambridge: Cambridge University Press; 1992. ISBN: 978-0521417972. https://doi.org/10.1017/CBO9780511623451
10. Davis KL, Panksepp J. The Emotional Foundations of Personality: A Neurobiological and Evolutionary Approach. New York: W.W. Norton & Company; 2018. ISBN: 978-0393712310.
11. Seung S. Connectome: How the Brain's Wiring Makes Us Who We Are. Boston: Houghton Mifflin Harcourt; 2012. ISBN: 978-0547508184.
12. Zhao Z, Okada N, Yagishita S, Yahata N, et al. Correlations of brain structure with the social behavior of 15q11-13 duplication mice, an animal model of autism. Neurosci Res. 2024 Jul 10:S0168-0102(24)00100-7. https://doi.org/10.1016/j.neures.2024.07.009
13. Lorenz K. Ethology. Torino: Bollati Boringhieri; 2011. ISBN: 978-8833921921. (Original work published 1978).
14. Knoll F. Insekten und Blumen. Experimentelle Arbeiten zur Vertiefung unserer Kenntnisse über die Wechselbeziehungen zwischen Pflanzen und Tieren. 3 Bände. Wien: Selbstverlag; 1921-1926.
15. Wundt W. Outlines of Psychology. Ithaca: Cornell University Library; 2009. ISBN: 978-1112078007. (Original work published 1897).
16. Liddel H. Conditioning and Emotions. Scientific American. 1954;190(3):48-57. https://doi.org/10.1038/scientificamerican0354-48
17. Hassenstein B. Young animal and human being in view of comparative ethology. Stuttgart: Gentner; 1970. ISBN: 978-3872350228.
18. Godfrey-Smith P. Other Minds: The Octopus, the Sea, and the Deep Origins of Consciousness. New York: Farrar, Straus and Giroux; 2016. ISBN: 978-0374227760.
19. Anderson R, Mather J, Monette M, Zimsen S. Octopuses (Enteroctopus dofleini) Recognize Individual Humans. J Appl Anim Welf Sci. 2010;13(3):261-72. https://doi.org/10.1080/10888705.2010.483897
20. European Parliament, Council of the European Union. Directive 2010/63/EU of the European Parliament and of the Council of 22 September 2010 on the protection of animals used for scientific purposes. Official Journal of the European Union. 2010;L 276:33-79.
21. Ornoy A, Echefu B, Becker M. Animal Models of Autistic-like Behavior in Rodents: A Scoping Review and Call for a Comprehensive Scoring System. Int J Mol Sci. 2024;25(19):10469. https://doi.org/10.3390/ijms251910469
22. Nestler EJ, Hyman SE. Animal models of neuropsychiatric disorders. Nat Neurosci. 2010;13(10):1161-9. https://doi.org/10.1038/nn.2647
23. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th-TER ed. Arlington: American Psychiatric Publishing; 2022.
24. Sarovic D. A Unifying Theory for Autism: The Pathogenetic Triad as a Theoretical Framework. Front Psychiatry. 2021;12:767075. https://doi.org/10.3389/fpsyt.2021.767075

25. Frye RE, Vassall S, Kaur G, Lewis C, Karim M, Rossignol D. Emerging biomarkers in autism spectrum disorder: A systematic review. Ann Transl Med. 2019;7(23):792. https://doi.org/10.21037/atm.2019.11.53
26. El-Kordi A, Winkler D, Hammerschmidt K, Kästner A, Krueger D, Ronnenberg A, Ritter C, Jatho J, Radyushkin K, Bourgeron T, et al. Development of an autism severity score for mice using Nlgn4 null mutants as a construct-valid model of heritable monogenic autism. Behav Brain Res. 2013;251:41-9. https://doi.org/10.1016/j.bbr.2013.04.060
27. Nakatani J, Tamada K, Hatanaka F, Ise S, et al. Abnormal behaviour in a chromosome-engineered mouse model for human 15q11-13 duplication seen in autism. Cell. 2009;137(7):1235-46. https://doi.org/10.1016/j.cell.2009.04.024