Full title: Revisiting the case for ‘feral’ humans under the light of the human self-domestication hypothesis: focusing on language

Running title: ‘Feral’ humans, self-domestication, and language

Authors: Amy Niego¹ and Antonio Benítez-Burraco²

1. PhD Program, Faculty of Philology, University of Seville, Seville, Spain

2. Department of Spanish, Linguistics, and Theory of Literature (Linguistics), Faculty of Philology, University of Seville, Seville, Spain

Corresponding author: Antonio Benítez Burraco

Antonio Benítez Burraco

orcid.org/0000-0003-4574-5666

Email: abenitez8@us.es

Telephone: +34 955 420 225

Correspondence:

Dr. Antonio Benítez Burraco

Área de Lingüística. Departamento de Lengua Española, Lingüística y Teoría de la Literatura.

Facultad de Filología. Universidad de Sevilla. C/ Palos de la Frontera s/n. 41004-Sevilla

(España/Spain)

21 Acknowledgments

22 This research was funded by the Spanish Ministry of Science and Innovation (grant PID2020-
23 114516GB-I00 [AEI/FEDER, UE] to ABB).

24

25 Statement of Ethics

26 The research conducted for the paper relied on previously published data by others and available
27 datasets, hence no ethics approval was required.

28

29 Disclosure Statement

30 The authors have no conflicts of interest to declare.

31

32 Author Contributions

33 ABB and AN conceived the paper, reviewed the literature, performed the data analyses and wrote
34 and approved the final manuscript.

35

36 Data availability

37 The data that supports the findings of this study are available in the supplementary material of this
38 article.

39

40 Geolocation information

41 Seville, Spain, Europe.

42

Abstract

Contemporary descriptions of ‘feral’ children generally preclude any insightful inference about the language deficits exhibited by these children, as well as the ultimate causes of their problems with language. However, they have been regularly used to support the view that language acquisition requires a proper social environment in order to occur. In this paper, we revisit the case for ‘feral’ children with the viewpoint that human evolution entailed a process of self-domestication that parallels what we find in domesticated animals. Because feralization commonly occurs in nature and because it entails a partial reversion of features of domestication, this self-domestication approach to the evolution of language reassesses the case for ‘feral’ children, particularly when compared with present-day conditions involving abnormal patterns of socialization, whether they are genetically-triggered as in autism spectrum disorder, or environmentally-triggered, as in reactive attachment disorder.

Keywords: feral children, language acquisition, language evolution, self-domestication, ASD, RAD

1. Introduction

A recent view of human evolution argues that our species went through a process similar to that experienced by domesticated mammals, which would account for many human-specific features in the physical, behavioral, and cognitive domains. This is the Human Self-Domestication (HSD) hypothesis of human evolution (Hare, 2017). Testing this hypothesis is problematic. Whereas in other instances of alleged self-domestication, such as bonobos (Hare et al., 2012), the non-domesticated counterpart is still extant (i.e. chimpanzees) and can be used for direct comparisons, the only available evidence in the case of humans is indirect, as the closest species to us became extinct. Accordingly, the HSD hypothesis builds on findings in present-day humans of traits commonly found in domesticates, as compared to other hominins (principally, Neanderthals) and archaic modern humans, but also to primate relatives. These features include physical traits, that are easier to retrieve from the fossil register (e.g. reduced cranial robusticity, reduced brain size, reduced tooth size, juvenile cranial shape retained in adulthood, smaller teeth, reduced brow ridge, smaller jaw and nasal bone projection, reduced sexual dimorphism), but also behavioral traits, which can be inferred from the fossil and archaeological registers (e.g. reduced reactive aggression, increased prosocial behavior, prolonged play behavior) (see Shea 1989, Leach 2003, Zollikofer and Ponce de León, 2010; Herrmann et al. 2011; Plavcan 2012; Márquez et al., 2014; Fukase et al., 2015; Stringer, 2016; Thomas and Kirby, 2018, Langley et al., 2019 among others for discussion). There is also some incidental genetic evidence to support the HSD hypothesis. Studies have found that the human genome contains a host of pseudogenes, something that is commonly associated with domestication (Deacon, 2009). More importantly, candidate genes for domestication in mammals are overrepresented in regions that show signals of positive selection

in modern humans compared to Neanderthals (Theofanopoulou et al., 2017), with selection on specific genes tracing back as late as 6,000 years ago (Benítez-Burraco et al., in 2021b).

The “self-“ in *self-domestication* refers to the fact that, contrary to animal domestication, which results from an active selection by humans of more tamed individuals, the alleged domestication of humans can be instead explained by the agency of diverse external factors, like the advent of co-parenting, changes in our foraging ecology, and/or the deterioration of the human environment during the Last Glaciation (Hare et al., 2012; Pisor and Surbeck, 2019; Spikins et al., 2021; Nikolsky and Benítez-Burraco, 2022). These factors seemingly favored more prosocial and cooperative behaviors. But because the physiological management of aggression ultimately involves diverse hormones impacting on different parts of the body (particularly, the hypothalamic-pituitary-adrenal axis), this process seemingly resulted in extended changes in our body, behavior, and perhaps cognition too. This co-occurrence of modified traits after the initial selection for tameness is what we indeed observe in most domesticated animals, termed as a “domestication syndrome” (Wilkins et al., 2014). According to the proponents of the HSD hypothesis, the behavioral changes brought about by our domestication would have facilitated the emergence of many of our species-specific distinctive features, including our enhanced social cognition, increased cooperation, extended social networks, and ultimately, our sophisticated culture and advanced technology (see Hare, 2017; Hare and Woods, 2020 for details).

In the recent years, the evolution of language under the effect of HSD has been object of particular interest. This is certainly explained by the fact that language is a hallmark of the human condition, but also because language acquisition demands both cognitive and behavioral pre-requisites that

are not all found in other species, and because it certainly results from a complex interaction between our biological endowment and the social environment. The finding that in some avian species domestication increases song complexity (see Okanoya et al., 2017) paved the way towards claims that HSD might have played a role in the emergence of modern languages (see Thomas and Kirby, 2018 and Benítez-Burraco and Progovac, 2020 for general discussions). A common view is that HSD favored the creation of the cultural niche that enables the sophistication of language via a cultural mechanism (Thomas and Kirby, 2018), mostly through its effects on selected aspects of our behavior (e.g. increased contacts between individuals, enhanced language learning and teaching, or increased language play; Progovac and Benítez-Burraco, 2019; Benítez-Burraco and Progovac, 2020 for details), but perhaps also of our body (e.g. the development of a white sclera that facilitates eye tracking and gazing during face-to-face interactions; Tomasello et al., 2007) and our cognition (e.g. improved episodic memory; Benítez-Burraco et al., 2021). Ultimately, these positive-for-language effects of HSD are thought to boil down to changes in the management of aggression (mostly reactive aggression), because, as noted, selection for tameness results in multiple changes in domesticates. Unfortunately, we lack any linguistic records from the remote past. To test and eventually prove this hypothesis about language evolution, we need to rely on indirect evidence, specifically, inferred changes in our communal living, behavior, social cognition, cooperation patterns, technology and know-hows, social networks, culture, and the like.

Interestingly, the finding that, as noted, candidate genes for domestication have been under selection in humans until very recently, suggests that HSD might be an ongoing process. In truth, features associated with HSD do not appear suddenly in the fossil register, but gradually, with signs of domestication reaching their peak quite late, seemingly at the end of the Upper Paleolithic

(Leach 2003; Cieri et al., 2014). The presentation of HSD features in present-day human populations can also be quite variable (Gleeson and Kushnik, 2018). Overall, this evidence suggests that HSD might be not a totally fixed phenotype in humans, but rather one where features of domestication are increased or attenuated in response to environmental conditions, mostly social features. This suggests that under some specific conditions HSD may revert to a less-domesticated condition. Because we have no access to living, archaic, less self-domesticated humans, a possible way to understand how HSD took place and how it impacted on our cognition and behavior is comparing such less-domesticated phenotypes with fully domesticated phenotypes, as hypothesized for present-day human beings. In fact, animal domestication seems to be a reversible condition too. Feralization refers to the process by which once-domesticated animals change and return to a wild-like state as a result of being reintroduced to wild-type environments without human contact (Daniels and Bekoff, 1989). As noted earlier and as we discuss below in detail, cases of ‘feral’ children have been described in the literature. Comparing these ‘feral’ children with ‘neurotypical’ children could thus help understand the effects of HSD on our cognition and behavior and ultimately, on our language abilities. Some caution is in order. For one, as we also show below, the term ‘feral’ child is not uncontroversial and posits its own problems. Moreover, feralization cannot be regarded as an exact mirror condition to domestication. As discussed in detail in Niego and Benítez-Burraco (2021), not all features of domestication reverse in feralized animals and to a great extent feralization is achieved through alternative genetic routes to domestication. Still, certain distinctive traits of domestication, particularly those that are more relevant to behaviors important for language acquisition (e.g. reduced reactive aggression, prosocial behavior), are found to be reversible. Likewise, a subset of the genes involved in feralization are also candidates for domestication. Overall, we can confidently expect feralization

to help us to understand domestication (and vice versa, of course), particularly if one focuses on the features and the biological mechanisms that are shared by these two conditions, that, as noted, are mostly related to response to the social environment.

In this paper we explore the possibility that the examination of cases of ‘feral’ children can shed some light, as noted, on the process of HSD and its effects on the complexification of language under HSD forces. We first review the available literature about ‘feral’ children, with a focus on their language (dis)abilities. As we will show, evidence is scarce and controversial, particularly because some of these children likely suffered from underlying cognitive and/or behavioral problems. This is why in a subsequent section of the paper we compare, vis a vis HSD features, this ‘feral’ phenotype with two conditions entailing increased reactive aggression and decreased prosocial behavior, namely, autism spectrum disorder (ASD) and reactive attachment disorder (RAD). Apart from the fact that many ‘feral’ children might suffer specifically from these exact conditions, a more important reason is that in both ASD and RAD one finds abnormally high levels of reactive aggression (internally triggered mostly, in the case of ASD; and externally caused mostly, in the case of RAD). Likewise, whereas domestication mostly results from selection for tameness, feralization essentially entails the reactivation of mechanisms triggering reactive aggression. At least in the case of ASD, there is evidence that this condition can be construed as a phenotype with attenuated features of HSD (Benítez-Burraco et al., 2016). Additionally, in the paper we delve into the genetic underpinnings of feralization, ASD and RAD, against the genetic background of HSD, to identify candidate genes of particular relevance for explaining the observed similarities and differences at the phenotypic level. Common candidates for these conditions are expected to be involved in the features of HSD that are more sensitive to the social environment.

In a subsequent section of the paper, we put the focus on the communication and language (dis)abilities of ‘feral’ children, and we compare them with children on the ASD spectrum and children with RAD, also vis-à-vis the hypothesized changes in human language/communication under the effects of HSD, but also the changes in communication patterns observed in feralized animals. We conclude with a discussion centered around the relevance of this line of inquiry for the study of language evolution and with suggestions for future research.

To finish, we wish to make it clear that throughout the paper, labels like ‘domesticated’ or ‘feral’ as applied to humans are used in the technical sense they have within this particular evolutionary framework. By no means are they intended to qualify humans as less or more similar to animals. They are used, respectively, as a shorthand for the physical, behavioral, and cognitive effects of a potentiation or an attenuation of the physiological mechanisms involved in the management of reactive aggression.

2. ‘Feral’ children

For the reasons mentioned in the previous section, in order to achieve a better understanding of the effects of HSD on the emergence of modern languages, it is of interest to look into descriptions of ‘feral’ humans (all of them children), and what has been documented about their physical and behavioral features, and particularly, about their language. The term *feral child* refers to a child who has been deprived of human contact, either intentionally or through accidental circumstances, and whose development has suffered as a result. As a consequence, ‘feral’ children do not develop many of the typical human skills, particularly those necessary to become fully integrated into

society, specifically, language; one reason for this is the existence of a critical period during childhood for acquiring most of these traits (Stendler, 1952; Kapoor, 1973).

A great deal of historical descriptions of ‘feral’ children are of those said to have been raised by animals. For hundreds of years, society has had a fascination with these cases, from the so-called ‘wolf boy’ to ‘savage girls’ and even one ‘gazelle boy’ (Kapoor, 1973; Butler, 2003). Over fifty cases of such ‘feral’ children have been documented from all over the world, most from centuries past, although some surfaced as late as the 1950’s. Most descriptions are quite similar: after their rescue from the wild, the children initially exhibit ‘animal behaviors’ such as growling like wolves, running on all fours, or smelling food before eating it, but all of them exhibit, specifically, severe deficits in language skills (Kapoor, 1973; Steeves, 2011). A typical case is that of Kamala and Amala, known as the ‘wolf children of Midnapore’. These two girls, aged 8 and 1 ½, were found in the company of wolves on the outskirts of a small village in India, and it was assumed from their condition that they had spent most of their young lives being raised by wolves (Butler, 2003). After being taken to an orphanage, observers reported that the girls acted like wolves; they chewed on bones, walked on all fours, howled, stayed awake all night, ate raw meat, and snarled or growled at other children who approached (Gesell, 1942). Although Kamala lived for 9 years at an orphanage after her removal from the wild, she was only able to develop a vocabulary of 45 words and understand basic verbal instructions (Gesell, 1942). Some speculate that Kamala never actually learned to speak, but instead learned to signal, much like an animal would (Butler, 2003). Similar descriptions exist for many other ‘feral’ children; in fact, of the nearly 50 documented cases of ‘feral’ children, the most successful ‘recovery’ of speech was Kamala; many of the other children remained mute, or never progressed past a drastically limited vocabulary (McNeil et al.,

1984). They were never able to fully integrate into society either, exhibiting high aggression levels, severe deficits in social cognition, and behavior that ultimately isolated them socially (Bettelheim, 1959).

Although many of these cases of ‘feral’ children involve them growing up alone or being raised by wild animals, the term ‘feral’ also applies to children who have suffered abuse or extreme neglect that interferes with normal social and cognitive development. One such case, perhaps the most famous one, is that of Genie, a girl who was locked in a room for most of her young life. When she was found at the age of 13, Genie was unable to speak and exhibited many ‘feral’ characteristics: she was highly antisocial, displayed high levels of reactive aggression, and avoided touch and eye contact (Curtiss et al., 1974). Sadly, Genie was never able to fully integrate into society, and never learned to speak past a rudimentary stage, prompting many linguists to claim her story as evidence for a distinct critical window for language (e.g. Fromkin et al., 1974). A similar case is that of Victor of Aveyron, a child in 16th century France who was born to alcoholic parents and severely neglected for most of his childhood (Lane, 1976). Known as ‘the Savage of Aveyron’, Victor displayed some typical behavior of ‘feral’ children in that he didn’t speak, and only learned rudimentary language, ate raw meat, had no social skills, and avoided human contact, seemingly more comfortable with living alone in the wild (Lane, 1976). It should be noted that in the case of both Genie and Victor of Aveyron, some researchers who studied the cases indicated that the cognitive and social deficits were the result of undiagnosed conditions: intellectual disability in the case of Genie (Rymer, 1994) and ASD in the case of Victor (Bettelheim, 1959). One more modern case study supports this idea; Swain and colleagues (2005) reviewed a case of an adolescent boy described as having ‘feral child syndrome’; they explained that his symptoms

most likely stemmed from a condition called ‘reactive attachment disorder’(RAD), which will be discussed in more detail in a subsequent section. This child’s symptoms were typical of those described in ‘feral’ children: ‘wolf-like’ aggressive behaviors, walking on all fours, and grunting instead of speaking. The doctors who eventually diagnosed the boy pointed to his traumatic early life events and ‘pathological caregiving’--that is, a mother who was mentally unstable and unable to form a typical attachment to the son. It is easy to see that, had the child been diagnosed a century earlier, the diagnosis would most likely have been ‘feral’.

The previous case studies point to the crux of the problem in cases of ‘feral’ children: it is frequently difficult to discern whether ‘feral’ traits in children came as a result of isolation from humans per se, from the trauma involved in these cases (as in most cases the isolation was also accompanied by neglect or abuse from caregivers), or from some underlying condition favoring isolation, such as ASD, intellectual disability, RAD, or any number of other developmental disabilities. To further complicate matters, many of these cases were documented before the fields of psychology or clinical linguistics were prepared to identify symptoms as cognitive, psychological or language impairments. Still, as early as 1959, Bettelheim wrote that ‘{Feralization} seems to be the result of some persons’-usually their parents’-inhumanity and not the result, as was assumed, of animals’-particularly, wolves'-humanity.’ He also notoriously noted that, in his opinion, many cases of so-called ‘feral’ children were most likely undiagnosed cases of severely autistic or cognitively impaired children. The parallels between past descriptions of ‘feral’ children and modern descriptions of children with ASD are remarkable, e.g. children tearing up food with their hands and/or teeth, growling, little to no use of language, avoidance of human contact, irritability, aggression, social anxiety, and a host of others. Because, as noted in the

introduction, ASD has been hypothesized to entail attenuated features of HSD, and because individuals with ASD exhibit notorious language and communicative deficits (Tager-Flusberg et al. 2005, Tager-Flusberg 2006, Eigsti et al. 2007, Bourguignon et al. 2012), in the next section we will consider ASD together with cases of ‘feral humans’ in our discussion of a putative reversion of the HSD phenotype and its effects on language. For the reasons exposed above, we will also consider RAD, which is admittedly a less known and less diagnosed disorder.

Finally, it is important to note that in most cases ‘feral’ children experienced extreme isolation that does not properly parallel what we find in feralized animals, who still maintain social contact with conspecifics, and what we can infer for archaic humans (exhibiting attenuated features of HSD), who maintained some sort of social networks, albeit less complex than those of present-day humans (e.g. Sikora et al., 2017). This circumstance is worth taking into account when comparing the ‘feral’ phenotype of these children with the traits observed in feralized animals, and particularly, when hypothesizing about the presentation of the HSD phenotype in the past from what we can observe in these children.

3. ‘Feral’ children, ASD, RAD and HSD vis-a-vis

In this section we will explore the parallels and differences between ‘feral’ children and children with ASD or RAD, through the framework of the HSD hypothesis and with the process of animal feralization in mind. As noted in the introduction, HSD is hypothesized to have entailed some genetic selection in several genes that contributed to fixing physical and behavioral (and perhaps cognitive) features, resulting in a new social environment that favored the cultural evolution of languages, among other features. Feralization is also purported to entail some genetic changes that

contribute to the readjustment to the wild environment (with only some overlap of the involved genes with genes involved in domestication) (see Niego and Benítez-Burraco, 2021, for a detailed discussion). ‘Feral’ children are not expected to have suffered any kind of genetic change that accounts for their condition, which is therefore only due to the extreme environment in which they grew up. This ‘feral’ phenotype is thus expected to result mostly from physiological and developmental changes. Nonetheless, the famous farm-fox experiment, involving selection for tameness in wild foxes, suggests that domestication can be achieved relatively quickly (Trut et al., 2009), and the same has been shown in feralization: in some feral pigs, feral traits such as hairiness, aggression, and tusk length were seen to revert to a wilder phenotype in as little as a generation (Bach, 2007). These changes can be fixed (epi)genetically quite quickly too (see Anastasiadi et al., 2022 for discussion). Finally, ASD is known to result from both genetic and environmental causes (Bölte et al., 2019; Bhandari et al., 2020; Manoli and States, 2021), but in general, children with ASD are reared in a normal environment (although some adaptations to their condition are expected). By contrast, RAD is generally diagnosed in children who experience extreme social adversity in early childhood, although it has been claimed to have some genetic basis too (Spangler et al., 2009; Corval et al., 2020). To put this differently, we can regard i) neurotypical individuals as those who have HSD cognition/behavior and have been raised in a HSD-resulting environment; ii) ‘feral’ children and children with RAD as those having HSD cognition/behavior, but raised in a non-HSD-conducive environment; iii) children with ASD as individuals with non-fully HSD cognition/behavior, but who have been raised in a HSD-conducive environment; and iv) feralized animals, as the closest proxy to truly ‘feral humans’ (or archaic humans), that is, humans having non-HSD cognition/behavior who were raised, as a consequence, in a non-HSD environment. Needless to say, the former characterization entails some undesirable simplification, as one should

expect that both biological and environmental factors contribute to these four conditions, although at the same time, the respective contributions of internal and external factors can be also expected to differ from one condition to another.

With all these qualifications in mind, we first compared these four phenotypes (‘feral’ children, children with ASD, children with RAD, and feralized animals) looking for the presentation of features of the domesticated/HSD phenotype. For doing this, we first compiled a comprehensive set of 29 features found in domesticated animals (and allegedly, in neurotypical modern humans too, according to the HSD hypothesis). We relied on the existing literature on domestication, specifically, the domestication syndrome in animals (see Wilkins et al., 2014; Sánchez-Villagra et al., 2016), but also on publications describing the HSD hypothesis (Cieri et al., 2014; Hare, 2017; Thomas and Kirby, 2018, among others). We then conducted a comprehensive literature search of the PubMed database (<https://pubmed.ncbi.nlm.nih.gov/>) to gain knowledge about the presentation of these 29 domestication-related traits in the four phenotypes under scrutiny. The search strategy included ‘ASD’, ‘feral animals’, ‘RAD’ or ‘feral children’ and keywords likely to capture publications reporting on the salient traits. In spite of some limitations in the case of ‘feral’ children, for which data are scarce, we found that most features of domestication/HSD are absent or attenuated in children with ASD, with RAD, or in ‘feral’ children (see Table 1 for a summary of our findings and Table 2 for a more detailed report). Whereas conflicting results are observed in the case of the brain, this is particularly true of the physiological mechanisms underlying the aggression response and particularly, social behavior. In the next section, we focus on communicative abilities, for which we also find notable parallels also in line with the predictions of the HSD hypothesis.

Features of domestication/HSD	Feralized animals	ASD	'Feral' children	RAD
Head/Brain				
Reduced head circumference, reduced total brain volumes	Mixed evidence	X	?	?
Relative decrease of specific brain areas (particularly, the hippocampus, the thalamus, and the striatum)	X	X	?	✓
Relative increase of specific brain areas (particularly, the amygdala)	Mixed evidence	✓	?	X
Ear size and shape				
Reduced ear size/changes in ear shape	X	✓	?	?
Orofacial region				
Shorter nose	Mixed evidence	✓	?	?
Dentition				
Reduced tooth size/length	Mixed evidence	✓	?	?
Behaviour				
Reduced anxious response to non-social and social aspects of life	X	X	X	X
Enhanced attentiveness and sensitivity to eye or facial movements or gestures	X	X	X	X
Increased sociability	X	X	X	X
Enhanced playing behavior	?	X	?	?
Reduced aggressive behavior	X	X	X	X
Neuroendocrine system				
Decreased levels of glucocorticoids	?	X	?	?
Decreased levels of basal adrenocorticotrophic hormone in plasma	?	X	?	X
Decreased stress response of the HPA axis	X	X	?	X
Increased basal levels of oxytocin (and more marked release patterns)	?	X	?	X
Increased basal levels of vasopressin (and more marked release patterns)	?	X	?	X
Skin, related features				
Hypopigmentation (skin, eyes)	Mixed evidence	✓	?	?
Higher levels of vitamin D in blood	?	X	?	?
Vital cycle				

General undergrowth	X	X	?	X
Neoteny	?	X	?	?
Reproductive cycle				
Reduced levels of androgens (with a focus on testosterone)	?	X	?	?
Earlier age of menarche	X	X	?	?

Table 1. Summary table of the presentation of features of domestication/HSD in the four conditions under scrutiny in the paper.

Features of domestication/HSD	Feralized animals	ASD	‘Feral’ children	RAD
Head/Brain				
Reduced head circumference, reduced total brain volumes	Smaller head circumference compared to wild counterparts (Kruska and Röhrs, 1974; Birks and Kitchener, 1999; Röhrs and Ebinger, 1999; Kruska, 2005); dingoes show a larger cranium than dogs (Smith et al., 2018).	Larger head circumference and/or brain size than TD subjects (Aldridge et al., 2011, Sacco et al., 2015).	<i>Not enough data available</i>	<i>Not enough data available</i>
Relative decrease of specific brain areas (particularly, the hippocampus, the thalamus, and the striatum)	Larger hippocampus found in feral pigs (Kruska and Rohrs, 1974).	Larger relative hippocampal volumes in children; atypical coupling between hippocampal volumes and brain size (Reinhardt et al. 2020)	<i>Not enough data available</i>	Decreased size of hippocampus (Teicher, 2002; Corbin, 2007); significant decrease of activity in the striatum (Takiguchi et al., 2015); greater fiber density, axonal diameter, and myelination in selected thalamic pathways (Makita et al., 2020); larger volume in right

				thalamus (Jung et al, 2020).
Relative increase of specific brain areas (particularly, the amygdala)	Higher density of the amygdala in some species (Kruska and Rohrs 1974; Kruska, 2005, 2014); no changes in feral cats, dogs, pigs, goats, and donkeys (Röhrs and Ebinger, 1999; Smith et al., 2018).	Larger amygdala than TD peers (Mosconi et al., 2009; Murphy et al., 2012); atypical development during growth (Schumann 2004; Courchesne et al., 2011).	<i>Not enough data available</i>	Decreased size of the amygdala (Teicher, 2002; Corbin, 2007)
Ear size and shape				
Reduced ear size/changes in ear shape	Dingoes exhibit wolf-like prominent ears, contrary to most dog breeds (Schweizer et al., 2017; Smith et al., 2019).	Abnormal ear shape (Manouilenko et al., 2014).	<i>Not enough data available</i>	<i>Not enough data available</i>
Orofacial region				
Shorter nose	Some breeds of pigs retain shorter snouts after feralization (Physical Characteristics of Feral Hogs, 2019), others revert to a longer, wild type snout (Kruska and Röhrs, 1974; Smith et al., 2018; Ballard and Wilson, 2019).	Flatter nose bridge documented in patients with ASD (Aldridge et al., 2011).	<i>Not enough data available</i>	<i>Not enough data available</i>
Dentition				
Reduced tooth size/length	Smaller teeth documented in feral pigs, but maintain 'wild-type' morphology (Evin et al., 2015); dingoes show larger carnassial teeth and longer canine teeth (Smith and Litchfield, 2010)	Number of tooth irregularities documented in ASD, including missing teeth and diastemas (Luppanapornlarp et al., 2010).	<i>Not enough data available</i>	<i>Not enough data available</i>
Behaviour				

Reduced anxious response to non-social and social aspects of life	Increased 'shoaling' behavior in feral fish suggestive of higher anxiety levels (Swaney et al., 2015).	Increased anxiety (Park et al., 2016).	Increased social/non-social anxiety (Bettelheim, 1959; Butler, 2003)	Unexplained fearful reactions during non-threatening interactions with caregivers (Ellis et al., 2020), general increased anxiety reported (Hinshaw, 1999; Cuyvers, 2020).
Enhanced attentiveness and sensitivity to eye or facial movements or gestures	Eye contact is avoided (Park et al., 2016; Johnston et al., 2017).	Eye contact is avoided (Park et al., 2016; Johnston et al., 2017).	Eye contact is avoided (Butler, 2003).	Eye contact is avoided (Miellet et al., 2014; Ellis et al., 2020).
Increased sociability	Avoidance of contact with humans (Rose et al., 1985; Johnston et al., 2017; Owens et al., 2017)	Reduced sociability commonly (Park et al., 2016).	Avoidance of human contact (Butler, 2003)	Muted positive response to socially stimulating situations, general social dysfunction (Ellis et al., 2020); minimal response to social and emotional stimuli (Miellet et al., 2014; Ellis et al., 2020).
Enhanced playing behavior	<i>Not enough data available</i>	Children with ASD lack 'pretend play' behavior (Bettelheim, 1959; Naber et al., 2008; Cuyvers et al., 2020; Ellis et al., 2020).	<i>Not enough data available</i>	<i>Not enough data available</i>
Reduced aggressive behavior	Increased levels of aggression (Rose et al., 1985; Johnston et al., 2017; Owens et al., 2017)	Reactive aggression/irritability commonly documented (Farmer et al., 2015, Mikita et al., 2015).	High prevalence of increased aggression (Butler, 2003).	Episodes of aggression and/or irritability widely documented (Ellis et al., 2020).
Neuroendocrine system				
Decreased levels of glucocorticoids	<i>Not enough data available</i>	Higher levels of glucocorticoids (Hamza et al., 2010; Spratt et al., 2012; Hollocks et al., 2014).	<i>Not enough data available</i>	<i>Not enough data available</i>
Decreased levels of basal	<i>Not enough data available</i>	Higher levels of adrenocorticotrophic	<i>Not enough data available</i>	Higher levels of adrenocorticotrophic

adrenocorticotrophic hormone in plasma		hormone (Curin et al., 2003; Hamza et al., 2010; Spratt et al., 2012; Hollocks et al., 2014)		hormone correlated to parental neglect/abuse (Kemph and Voeller, 2008).
Decreased stress response of the HPA axis	Heightened defense responses to predators linked to changes in the HPA axis (Swaney et al., 2015).	HPA axis is hyper-responsive in benign social environments (Taylor and Corbett, 2014), slower in situations of social threat (Spratt et al., 2012; Jacobson, 2014); higher serum cortisol responses in children with ASD (Spratt et al., 2012); disrupted patterns of cortisol levels in children (Tomarken et al., 2015).	<i>Not enough data available</i>	HPA axis hyper reactivity reported in response to stress (Kemph and Voeller, 2008); lower levels of cortisol documented (Kočovská et al., 2013).
Increased basal levels of oxytocin (and more marked release patterns)	<i>Not enough data available</i>	Lower blood oxytocin levels reported (John and Jaeggi, 2021)	<i>Not enough data available</i>	Lower salivary oxytocin/atypical secretion reported (Fries et al., 2005; Suzuki et al., 2020).
Increased basal levels of vasopressin (and more marked release patterns)	<i>Not enough data available</i>	Decreased vasopressin levels in children (Oztan et al., 2018; Parker et al., 2018)	<i>Not enough data available</i>	Abnormal binding of vasopressin receptor (Carter, 2003; Carter et al., 2008; Heinrichs et al., 2009); significantly lower basal levels of plasma vasopressin (Fries et al., 2005).
Skin, related features				
Hypopigmentation (skin, eyes)	Great deal of variation in pigmentation is present, certain colorations are conserved when advantageous in the feral environment (Roulin, 2004; Anderson et al., 2009; Derelle et al., 2013; Feulner et al., 2013; Linderholm et	Various hypomelanotic diseases present autistic symptoms (Akefeldt and Gillberg, 1991; von Aster et al., 1997; Gómez-Lado et al., 2004)	<i>Not enough data available</i>	<i>Not enough data available</i>

	al., 2016; Gering et al., 2019).			
Higher levels of vitamin D in blood	<i>Not enough data available</i>	Lower serum levels of vitamin D reported in children with ASD (Cannell and Grant, 2013).	<i>Not enough data available</i>	<i>Not enough data available</i>
Vital cycle				
General undergrowth	Feral canids and pigs reported to be larger than their closest domesticated relatives (Kruska and Röhrs, 1974; Smith et al., 2018; Gering et al., 2019)	General overgrowth reported during childhood (Chawarska et al., 2011; Campbell et al., 2014).	<i>Not enough data available</i>	General undergrowth reported (Dobrova-Krol et al., 2008; Stinehart et al., 2012)
Neoteny	<i>Not enough data available</i>	Neotenus features uncommon due to increased body size, higher levels of androgens, and earlier puberty than TD subjects (Chawarska et al., 2011; Campbell et al., 2014; El-Baz et al., 2014)	<i>Not enough data available</i>	<i>Not enough data available</i>
Reproductive cycle				
Reduced levels of androgens (with a focus on testosterone)	<i>Not enough data available</i>	Higher levels of androgens reported (El-Baz et al., 2014; Hauth et al., 2000).	<i>Not enough data available</i>	Higher levels of androgens linked to higher stress in early life (Hauth et al., 2000), and to disrupted HPA axis function (Goozen et al., 2000).
Earlier age of menarche	Delayed onset of sexual maturation (Abbott et al., 1997; Lord et al., 2013).	General delay in the age of menarche (Knickmeyer et al., 2006)	<i>Not enough data available</i>	<i>Not enough data available</i>

Table 2. A detailed characterization of the presentation of features of domestication/HSD in the four conditions under scrutiny in the paper.

345 Additionally, we compared the genetic basis of these conditions against the background of the
346 genetics of domestication. Domestication has been hypothesized to result from changes in several
347 genes involved in the development and function of different body parts. Wilkins and colleagues
348 (2014) have highlighted a set of core candidate genes for domestication in mammals, whereas
349 Niego and Benítez-Burraco (2019) have compiled an expanded list of candidates based on several
350 recent analyses of the signatures of domestication in selected domesticates (see Supplemental file
351 1; column A). That said, as noted by Jensen (2006) or Wilkins and colleagues (2014), and discussed
352 as well in a recent paper by Anastasiadi and colleagues (2022), domestication can be expected to
353 also result in part from pre-existing genetic variation, and even from epigenetic changes. The
354 genetic scenario for feralization is pretty similar, but with the additional circumstance that some
355 genetic differences between feralized and domesticated animals can be due to interbreeding with
356 wild populations, and not to feralization per se (see Zhang et al., 2020 for discussion). Niego and
357 Benítez-Burraco (2021) have also compiled an enlarged list of candidates for feralization relying
358 on available data from a variety of studies (see Supplemental file 1; column B). An important
359 conclusion of Zhang and colleagues' study is that the overlap between the genetic signatures of
360 domestication and feralization is not as complete and one could expect, this suggesting that the
361 (partial) reversion of the traits of domestication as found in feralized animals is achieved through
362 modifications in genes not directly involved in domestication; at the same time, some common
363 candidates can be found between these two conditions, which can be expected to account for the
364 reversion of specific traits of domestication observed in feralized animals. Genetic studies on
365 'feral' children have not been done for obvious reasons. Regarding the genetic basis of RAD, there
366 is evidence that it is genetically influenced (Minnis et al., 2007). Several studies point to the
367 involvement of genes related to dopaminergic and serotonergic activity in abnormal changes in

attachment behavior by children (Lakatos et al., 2000; Bakermans-Kranenburg and van Ijzendoorn, 2007; Caspers et al., 2009; Spangler et al., 2009; Luijk et al., 2011; Wazana et al., 2015). Animal models suggest that changes in receptors for oxytocin and endogenous opioid could also result in RAD (Winslow and Insel, 2002; Moles et al., 2004). However, our knowledge about the genes that might predispose RAD is more limited than in the case of ASD, domestication, or even feralization. In spite of these limitations, from the comparison of what we know about the genetics basis of ASD, feralization, and RAD, we can gain some insights about the genetics of human domestication/feralization. Table 3 summarizes our findings. The abnormal expression of these genes in response to internal factors (e.g. mutations) or external factors (e.g. adverse rearing conditions) are expected to account, at least in part, for the shared features between these three conditions in the domains of social cognition and behavior, and ultimately, communication (dis)abilities, to which we dedicate the last section of the paper. These common candidates also emerge as promising candidates for explaining, at least in part, the changes in social cognition, behavior, and language which occurred during our evolution under the forces of HSD. One reason is the deep link that exists between evolution and abnormal development. Hence, as noted by e.g. Pattabiraman et al. (2020) with regard to cognitive diseases, our species-specific distinctive cognitive abilities resulted to a great extent from changes in the organization and function of preexisting neural devices (in response to specific mutations, but also to selective pressures favoring a more prosocial behavior, as claimed by the HSD hypothesis), but these changes also account for our predisposition to suffer from neurodegenerative diseases, because of the reduced resilience and resistance to damage of recently evolved neuronal networks (see Toro et al. 2010) for ASD).

GENE	PROTEIN NAME	PROTEIN FUNCTION	RELATION TO FERALIZATION	RELATION TO ASD	RELATION TO RAD
<i>TRHDE</i>	Thyrotropin Releasing Hormone Degrading Enzyme	Encodes a member of the peptidase M1 family, which inactivates the neuropeptide thyrotropin releasing hormone	A candidate for stress-response differences observed between feralized animals and their domesticated counterparts (Nillni et al., 1999).	Linked to enhanced dopaminergic and/or reduced serotonergic activity in children; linked to hypothalamic dysfunction (Hashimoto et al., 1991)	<i>Not enough data available</i>
<i>OXTR</i>	Oxytocin Receptor	Plays a key role in socialization and regulation of the HPA axis' response to stress	Candidate for feralization (Pan et al., 2018).	Hypermethylated in subjects on the spectrum; hypermethylation correlates to severity of social cognitive deficits (Andari et al., 2020; Maud et al., 2018), as well as to reduced attention to social information (Puglia et al., 2018)	<i>Not enough data available</i>
<i>MAOA</i>	Monoamine Oxidase A	Encodes monoamine oxidase A, involved in degradation of amine neurotransmitters	<i>Not enough data available</i>	Alleles associated with increased severity of ASD (Cohen et al., 2003; Yoo et al., 2009; Cohen et al., 2011); alleles correlated with IQ levels, adaptive skills, and language skills (Cohen et al., 2003; Cohen et al., 2011).	Some polymorphisms moderate detrimental effects of childhood abuse (Caspi et al., 2002); high levels of <i>MAOA</i> correlate to less social problems, including disrupted attachment (Caspi et al., 2002); selected epigenetic mutations correlate to high rates of aggression (Marquez et al., 2013)
<i>5HTT</i>	Serotonin transporter	Transports the neurotransmitter serotonin from synapses to	Polymorphisms associated to fear response in hens (Krause et al., 2019) and defensive	Lower 5-HTT availability in various brain regions correlated to increased levels of	Significant correlation between one allele of 5HTT and maltreatment, stress, and adversity

		presynaptic neurons	behaviors in animals (Blanchard et al., 1998); enhancement of serotonin activity inhibits predatory aggression in several species (Nikulina, 1991)	serotonin in individuals with ASD (Coutinho et al., 2004; Veenstra-VanderWeele et al. 2012; Andersson et al., 2021).	in childhood resulting in violent/aggressive behavior later in life (Reif et al., 2007).
<i>TH</i>	Tyrosine Hydroxylase	Involved in conversion of tyrosine to dopamine; implicated in the physiology of adrenergic neurons.	Affects behavior and temperament in cattle (Lourencou-Jaramillo et al., 2012).	<i>Not enough data available</i>	<i>Not enough data available</i>
<i>DRD4</i>	Dopamine Receptor D4	Inhibits adenylyl cyclase.	Variants in <i>DRD4</i> correlate with levels of aggression linked to domestication in chickens (Komiya et al., 2014).	An allele of the gene may increase risk for clinically elevated ASD symptoms in children and adolescents with ADHD (Reiersen and Todorov, 2011).	Polymorphisms linked to disordered attachment such as that found in RAD (Bakermans-Kranenburg and van Ijzendoorn, 2007; Spangler et al., 2009).

Table 3. Structural and functional features of common candidates for ASD, RAD, and feralization.

Finally, because evidence about the genetics of ASD is more abundant than in the case of RAD, we examined in more detail the overlap that exists, specifically, between the genetics of feralization and ASD, with the aim of uncovering additional candidates that can account for the changes in human socialization and communication patterns under the effects of environmental changes, in line with the HSD hypothesis. Among the candidate genes for feralization (see supplemental file 1; column B for an updated list) one finds many candidates or risk factors for ASD. Our findings are summarized in Table 4. Although the genes discussed in this section are by no means a comprehensive list of the genes implicated in feralization, RAD, and/or ASD, they emerge, as noted, as promising candidates for the changes in human behavior under (self-)domestication forces. Further research is undoubtedly needed in order to draw conclusions about

the genetic basis of ‘feralization’ in humans, but the fact that many of these genes are related to neurotransmitter function points in a useful direction.

GENE	PROTEIN NAME	PROTEIN FUNCTION	RELATION TO FERALIZATION	RELATION TO ASD
<i>CACNA1A</i>	Calcium Voltage-Gated Channel Subunit Alpha1 A	Encodes the alpha 1C subunit of the Cav1.2 voltage-dependent L-type calcium channel, which contributes to the production of β and γ waves during wakefulness and REM sleep (Kumar et al. 2015).	Candidate for feralization (Pan et al., 2018)	Linked to a complex condition entailing intellectual disability, executive dysfunction, Attention-Deficit/Hyperactivity Disorder (ADHD) and/or ASD, co-occurring with childhood-onset epilepsy (Damaj et al. 2015); risk alleles of <i>CACNA1C</i> also correlate to lower performance in semantic tasks in people with schizophrenia (Krug et al. 2010).
<i>FABP4</i>	Fatty Acid binding protein 4	Encodes a fatty acid binding protein involved in fatty acid uptake, transport, and metabolism (Furuhashi, 2019).	Candidate for feralization (Pan et al., 2018)	FABP4 levels are significantly lower in children on the ASD spectrum (Maekawa et al., 2020). Mice bearing defective copies of <i>Fabp4</i> show ASD-like features, including abnormal behavior and abnormal morphology of pyramidal neurons (Maekawa et al., 2020).
<i>KIT</i>	KIT Proto-Oncogene, Receptor Tyrosine Kinase	The protein encoded by KIT influences the proliferation, differentiation, migration and apoptosis of various cell types, playing a key role in hematopoiesis, stem cell maintenance, gametogenesis, melanogenesis, and in mast cell development,	A strong candidate for domestication due to its connection with the neural crest (Wilkins et al., 2014), and also linked to feralization (Pan et al., 2018)	Involved in symptoms of ASD, possibly due to its connection with the neural crest (Rothschild et al., 2003; Kilsby et al., 2013)

		migration and function, among other things (Naumann et al., 2021).		
<i>MBD4</i>	Methyl-CpG Binding Domain 4, DNA Glycosylase	Encodes a protein that binds to methylated DNA and that plays a role in the epigenetic regulation of gene expression and DNA repair (Bellacosa et al., 1999).	Candidate for feralization (Pan et al., 2018)	There is evidence of an association of the gene to some cohorts of people with ASD (Cukier et al., 2010).
<i>MFRP</i>	Membrane Frizzled-Related Protein	Encodes a protein associated to certain retinal conditions (Katoh, 2001)	Candidate for feralization (Pan et al., 2018).	Strongly enriched for variants likely to affect ASD risk (Sanders et al., 2015)
<i>MIB1</i>	MIB E3 Ubiquitin Protein Ligase 1	Encodes an ubiquitin-protein ligase that regulates apoptosis (Itoh et al., 2003).	Candidate for feralization (not domestication) (Zhang et al., 2020)	Strongly enriched for variants likely to affect ASD risk (De Rubeis et al., 2014).
<i>MITF</i>	Melanocyte Inducing Transcription Factor	Encodes a melanocyte inducing transcription factor, responsible for pigment cell specific transcription of the melanogenesis enzyme genes. Mutations of <i>MITF</i> result in auditory-pigmentary syndromes, such as Waardenburg syndrome type 2 and Tietz syndrome (Goding and Arnheiter, 2019).	Found among candidates for feralization in sheep (Pan et al., 2018), and it is one core candidate as well for domestication processes (Wilkins et al., 2014).	Associated to gastrointestinal problems commonly found in people with ASD (Rodríguez-Fontenla and Carracedo, 2021).
<i>NCOR1</i>	Nuclear Receptor Corepressor 1	Involved in chromatin condensation and	It is found among Zhang et al. (2020)'s candidates	Candidate for Mitochondrial Complex III Deficiency, Nuclear Type 2, a condition

		specifically regulates transcription repression of thyroid-hormone and retinoic acid receptors (Zhou et al., 2019).	for feralization	which features motor problems, including apraxia and dysarthria, as well as cognitive impairment (Ghezzi et al., 2011); it is also a candidate for Rett Syndrome, which is characterized by loss of acquired motor and language skills, stereotypic movements, and ASD-like features (Sharma, 2009; Lyst et al., 2013). Pathogenic variants of <i>NCOR1</i> have been found in people on the ASD spectrum (Iossifov et al., 2012; Wang et al., 2016).
<i>NF1</i>	Neurofibromin	Encodes a GTPase involved in the negative regulation of cell proliferation (Sabbagh et al., 2013)	Related to altitude adaptation and response to hypoxia by feralized sheep (Pan et al., 2018)	Occasionally associated with ASD (Marui et al., 2004; Sanders et al., 2012).
<i>NUP155</i>	Nucleoporin	Encodes a protein related to the transportation of macromolecules across the nuclear envelope, which has been related to cardiac physiology (Zhang et al., 1999).	Associated with dog feralization, but not domestication (Zhang et al., 2020)	Protein-truncating variants in this gene have been found in people with ASD (Satterstrom et al., 2020).
<i>PACSI</i>	Phosphofurin Acidic Cluster Sorting Protein	Encodes a protein involved in the localization of trans-Golgi network (TGN) membrane proteins (Hinnens et al., 2003)	It is found among Pan et al. (2018)'s candidates for feralization	Related to a syndromic form of intellectual disability also encompassing ASD features (Schuurs-Hoeijmakers et al., 2016).
<i>PRKCA</i>	Protein Kinase C Alpha	Encodes a protein kinase involved in cell proliferation, differentiation, migration and adhesion (Haughian and Bradford, 2009).	It is found among Pan et al. (2018)'s candidates for feralization	Variants of the gene have been identified in three ASD probands (De Rubeis et al., 2014; Iossifov et al., 2014).

<i>PSMD12</i>	Proteasome 26S Subunit, Non-ATPase 12	Encodes a component of a proteasome involved peptide cleavage (Saito et al., 1997).	It is found among Pan et al. (2018)'s candidates for feralization	Deletions and loss-of-function point mutations in this gene have been identified in people with a syndromic neurodevelopmental disorder characterized by intellectual disability and ASD features (Kury et al., 2017).
<i>ROBO2</i>	Roundabout Guidance Receptor 2	Encodes a protein involved in axonogenesis and which plays a key role in many aspects of neurodevelopment and cognition (Kidd et al., 1998; Brose et al., 1999)	A candidate for feralization in animals, it has also been related to the olfactory system and food seeking (Bates et al., 2011; Zhang et al. 2020).	Candidate for ASD (Suda et al., 2011; Prasad et al., 2012), as well as dyslexia (Fisher et al., 2002), speech-sound disorder (Stein et al., 2004), and expressive vocabulary growth in the normal population (St Pourcain et al. 2014)
<i>SLC25A27</i>	Solute Carrier Family 25 Member 27	Encodes a component of an ion transporter across the mitochondrial membrane (Anitha et al., 2012).	Associated to feralization, but not domestication (Zhang et al., 2020).	Downregulated in various brain regions of subjects on the ASD spectrum (Anitha et al., 2012).
<i>SOX6</i>	SRY-Box Transcription Factor 6	Encodes a transcription factor involved in the regulation of many body functions (Cantu et al., 2011).	Affects both feralization and domestication due to its association with the Wnt signalling pathway, which plays a key role in initial neural crest cell differentiation (Wang et al., 2018).	Mutations have been associated to a complex condition encompassing neurodevelopmental problems and behavioral abnormalities, including ADHD and/or ASD features (Tolchin et al., 2020).
<i>TTN</i>	Connectin	Encodes connectin, a protein important for muscle elasticity (Saladin, 2015)	It is found among Pan et al. (2018)'s candidates for feralization	Rare mutations in this gene have been identified in people with ASD (O'Roak et al., 2011; O'Roak et al., 2012).

Table 4. Structural and functional features of common candidates for ASD and feralization.

4. Human ‘feralization’ and the evolution of language

In the previous section, we have showed that changes in our biological endowment (as found in ASD) or in the external environment (as found in RAD) can impact negatively on our socialization and result in ‘feral’ phenotypes in our species than resemble the features exhibited by truly feral animals compared with their wild conspecifics. Phenotypes like ASD and RAD, as well as those exhibited by ‘feral’ children, can be thus construed as hypodomesticated human phenotypes. This is seemingly explained, at least partially, by the impact of such internal and external factors on the physiological mechanisms controlling aggression, which result in turn in cognitive and behavioral changes that impede the normal acquisition of social abilities. This impact is mediated by genes involved in domestication (and presumably, in HSD too). This circumstance provides additional support the long tradition of using of these ‘feral’ phenotypes in humans as confident proxies for previous stages in the evolution of human language(s), but mostly, we wish to add, under the umbrella of the HSD hypothesis.

As we advanced in the introduction, the HSD hypothesis of language evolution has recently emerged as a promising view of how the features exhibited by present-day human languages might have appeared during our history. In brief, the behavioral (but also cognitive and physical) changes brought about by HSD would have favored the creation of the niche that enables the complexification of language through a cultural mechanism; in turn these increasingly sophisticated forms of language would have favored HSD, as they contributed to replace physical confrontation with verbal confrontation (see Progovac and Benítez-Burraco, 2019; Benítez-Burraco and Progovac, 2020 for details). HSD can be expected to have favored the emergence of modern uses of language (pragmatics) as well, via this feedback loop between the control of

aggression and language sophistication. Accordingly, the reduction in reactive aggression brought about by HSD would have enabled the full exploitation of our cognitive and interactional potential as it applied to linguistic exchanges, and ultimately, favored the evolution of a specific form of communication governed by persuasive reciprocity (see Benítez-Burraco et al., 2021 for details). In this section, we review the language deficits exhibited by ‘feral’ children with the language problems observed in children with ASD or RAD, vis-a-vis the changes in communication patterns observed in feralized animals compared to their domesticated conspecifics. Such a broad characterization of the linguistic and interactional profiles of people with these three conditions is aimed to i) check whether the parallels observed in the physical, cognitive, and behavioral domains can be also observed in the domain of language/communication; ii) discuss whether these putative shared features parallel the reversion of communication abilities observed in feralized animals; iii) provide additional support to the view that changes impacting on our socialization make language/communication simpler and less efficient; and ultimately, iv) provide additional support to the HSD hypothesis of language evolution.

Communication deficits in ASD, RAD, and ‘feral’ children

With regards to ‘feral’ children, as mentioned in section 2, the most common trait observed in historical descriptions of these children is their lack of language skills (Kapoor, 1973; Fromkin et al., 1974; McNeil et al., 1984; Vyshedskiy et al., 2017). As also observed previously, very few ‘feral’ children have recovered language skills, and although some progress was noted in certain cases (see Rymer, 1994), most of them ended up with serious deficits, including drastically reduced vocabularies, and little to no syntactic ability (Bettelheim, 1959; Kapoor, 1973; McNeil, 1984, Vyshedskiy et al, 2017). Because many descriptions of ‘feral’ children were written when the

fields of clinical linguistics and cognitive sciences were in their infancy, solid evidence is scarce. Much of the data referred to in this subsection was gleaned from the in- depth linguistic analysis of Genie, briefly discussed in section 2 above, which is to date the most detailed account of language in ‘feral’ children that exists (Fromkin et al., 1974). Additional data on language development in ‘feral’ children has been collected from descriptions children raised in physical isolation because of neglect, adverse circumstances, or abuse. Overall, parallels with ASD and RAD are notable, as we show below.

In the domain of phonology, some authors (e.g. Wolk and Edwards, 1993; Lindgren et al., 2009; Wolk et al., 2016) support the view that the language of people on the ASD spectrum exhibits phonetic and phonological deficits, including consonant deletions, cluster reductions, changes in voicing, and syllable coalescence (but see Bartolucci et al., 1976; Kjelgaard and Tager-Flusberg, 2001; or McCann et al., 2007 for an opposite view). Many of these features correlate to findings on ‘feral’ children (Curtiss et al., 1974; Vyshedskiy et al., 2017). Problems with prosody have also been documented in both groups, particularly with regards to paralinguistic uses of prosody.

Regarding the domain of syntax, the general consensus from studies of ‘feral’ children is that they experience marked difficulties in grasping many types of syntactic constructions, like passives, wh-questions, or movement transformations, but also other simpler aspects like constructions involving spatial prepositions (Curtiss et al., 1974; Rymer, 1994; Vyshedskiy et al., 2017). Research on ASD shows related problems with structural aspects of language (see Tager-Flusberg et al., 2005 and 2006 for discussion), including difficulties with binding, relative clauses, wh-questions, raising and passives (Perovic and Janke, 2013; Wada, 2015; see Benítez-Burraco and

Murphy, 2016 for review). Regarding RAD, a study by Raaska and colleagues (2013) showed that children with this condition also exhibit marked difficulties with language compared to their typically-developing (TD) peers-- most notably in expressive language skills, communication, and comprehension (see also Zangl and Mills, 2007).

As one would imagine, pragmatics is an area that poses a great deal of difficulty for ‘feral’ children, people on the ASD spectrum, and people with RAD. To begin with, people with ASD and ‘feral’ children have shown difficulty inferring meaning from changes in voice inflection, as well as producing appropriate pitch/prosodic inflection with their utterances or to match their interlocutor (Fromkin, et al., 1974; Curtiss et al., 1974; Fusaroli et al., 2017; Vyshedskiy et al., 2017). Other pragmatic deficits commonly found in people on the ASD spectrum include problems with understanding figurative use of language, including metaphor, irony, jokes, and sense of humor (see Baron-Cohen 1988, 1997; Kissine, 2012). Additionally, individuals with ASD show impairments in conversational skills. For instance, individuals on the spectrum generally exhibit difficulty taking turns in conversation, detecting faux pas such as interruptions, or constructing a coherent narrative discourse (Baron-Cohen 2000, 1999; Kaland et al., 2002; Kisine, 2012; Lord and Paul, 1997; Surian et al., 1996; Tager-Flusberg 1992, 1993, 2000; Happé 1993). Similar results have been found for individuals with RAD and for ‘feral’ children. Children with RAD show significant deficits when asked to interpret social cues, or to understand social hierarchies, on par with difficulties reported in ASD (Green and Goldwyn, 2002; Bennett et al., 2009). According to Sadiq and colleagues’ (2012) study, appropriate use of expressive language also proves to be a difficulty for those with RAD (Smyke et al., 2002), which is in line with their failure to develop relevant social relationships (Rutter et al., 2009). In fact, Sadiq and colleagues found that children

with RAD had more difficulty with pragmatics than their counterparts with ASD, demonstrating lower scores in areas such as use of context, social relationships, and rapport. Vyshedskiy (2017) performed an analysis of language structure and function on linguistically deprived individuals, including so-called ‘feral’ children, and found significant deficits in the category of mental synthesis, which is a prerequisite for pragmatic functions. Further research on children who have been subject to neglect by parents or caregivers corroborates the idea that pragmatic language is one of the most dramatically affected aspects of language in these cases. For example, Di Sante and colleagues (2019) found that nearly half (44.4%) of neglected children in their study presented pragmatic problems in their language, from simple tasks such as asking for help to more complex ones like adapting the conversation towards one’s speaking partner. In a more recent study, Di Sante and colleagues (2020) found a significant link between parental neglect and children’s pragmatic abilities in areas like responsiveness, conversation support, and affect. In his work about the famous ‘feral’ child Victor of Aveyron, Lebrun (1980) pointed out that many so called ‘feral’ children, as well as those who experience speech deprivation due to neglect or other factors, have noted difficulty with pragmatic aspects of language such as turn taking, interpersonal turn taking, and understanding inference.

Since humans necessitate social interaction during formative years for language to develop, it has been suggested that the communicative deficits displayed by ‘feral’ children are the result of this missed window of opportunity to interact with their conspecifics (Curtiss et al., 1974; Rymer, 1994), although, as noted above, the high incidence of abuse and neglect as well as the high probability of undiagnosed conditions makes it difficult to exclude other possible causes. With regards to RAD, delays in language acquisition commonly observed in this condition have been

hypothesized to mostly stem from the deprived social environment that most children with RAD experience early in life, similarly to what has been suggested for ‘feral’ children. However, poor language performance in RAD could also result from some neurodevelopmental deficits such as impulsiveness and poor executive function, both associated with this condition (see Gleason et al., 2011; Raaska et al., 2013 for discussion). In the case of ASD, pragmatic deficits, specifically, have been also attributed to deficient socialization patterns (Bailey et al., 1996), but resulting from an internal deficit, specifically with mindreading, which impedes detecting and recognizing others’ mental states based on external cues, such as people’s facial expressions. Typically, individuals on the ASD spectrum show difficulties with the classic ‘false belief’ task, where the participant is required to imagine the mental state of another (see Happé, 1993; Baron-Cohen 2000, for overview). A well-known hypothesis supports the view that these problems stem from a deficit in the Theory of Mind system (ToM) (Happé, 1995). ToM grants us the ability to mentalize and carry out tasks such as inferring meaning, imagining mental states of others, and interpreting communicative intentions of our interlocutors. The brain regions supporting ToM (the bilateral temporal-parietal junction, the posterior superior temporal sulcus, and the medial prefrontal cortex) show atypical activation patterns in people with ASD (Castelli et al., 2002; Schultz et al., 2003; Wang et al., 2006; Wang et al., 2007; Colich et al., 2012). Similar findings have been observed for children with RAD (and potentially, for ‘feral’ children). For example, research by Teicher and Samson (2016) found that childhood neglect (a common element in both conditions) results in atypical size, density, and function of the prefrontal cortex (see also Baker et al., 2013; Heim et al., 2013; Gupta et al, 2015).

Our contention here is that structural and functional problems with language exhibited by ‘feral’ children, children with RAD, and children on the ASD spectrum might result, at least partially, from the abnormal presentation of HSD features. On the physiological side, the hyper-responsiveness of the HPA axis in response to stress certainly impedes normal socialization and accordingly, the acquisition of language through the interaction with their peers and caregivers. Also, the brain changes found in these groups, particularly in the amygdala, the hippocampus, and the thalamus, could also account for the observed language deficits. The amygdala and the hippocampus are components of the limbic system, a group of brain structures involved in the regulation of emotion, but also in functions like motivation and memory (Rolls, 2015 for review). The hippocampus has been argued to support our notable episodic memory that allows us to mentally travel both forward and backward in time. According to Corballis (2018, 2019) this Mental Time Travel (MTT) ability also supports some core features of human language, including recursion and displacement. In ASD, difficulties in MTT have been linked to hippocampal abnormalities (Cooper et al 2019), and studies on childhood neglect have also shown that lack of a caregiver affects the structure of the hippocampus by slowing down or altering synaptic development (Andersen and Teicher, 2004; Pickering et al., 2006). Regarding the thalamus, changes in the thalamus have been hypothesized to contribute to the emergence of our species-specific ability for learning and using languages (Boeckx and Benitez-Burraco, 2014).

Given the similar profiles of people with ASD, children with RAD, and ‘feral’ children in the domain of language structure and use, we end this section with an examination of the effects of feralization in the communicative behavior of domesticated animals. Our aim is finding additional

evidence that this abnormal language profiles might result from an attenuation of HSD features as levels of reactive aggression increase.

Communication in feralized animals compared to wild and domestic animals

Animals are routinely used as a model for language evolution, because of the strong continuity found between human language and animal cognition/communication/socialization (as language fulfills so many different functions, many domains need to be explored) (Okanoya et al., 2017; Vernes et al., 2017; Tyack, 2020). This is also true for the putative effects of HSD on our language abilities: as noted in the Introduction, animal communication becomes more complex because of the relaxation of selective pressure brought about by the domestic environment. Accordingly, we find it worth examining the effects of feralization on animal communication: this should enable us to better understand how language deficits in conditions like ASD or RAD result from the attenuation of HSD features.

Naturally, dogs provide an interesting testing ground. The reason is that, like modern humans, they exhibit almost the whole suite of features associated with domestication (Sanchez-Villagra et al., 2016), seemingly because they were domesticated almost entirely based on selection against aggression and not on other traits of interest. In dogs, domestication resulted in, or reinforced, cognitive abilities which support the acquisition and use of sophisticated communication devices, such as joint attention (Nagasawa et al., 2015), gaze following (Kaminski and Nitzschner, 2013; Range and Viranyi, 2013), over-imitation (Huber et al., 2020), or the ability to solve problems by relying on social cues (Hernádi et al., 2012; Udell, 2015). On the genetic level, research shows that humans and dogs share common genetic determinants that are associated with key

physiological processes involved in domestication (see Benítez-Burraco et al., 2020 for review). Specifically, signals of selection of specific changes in cis-regulatory regions of the oxytocin receptor gene, *OXTR*, have been found both in dogs (Oliva et al., 2016; vonHoldt et al., 2017; Shilton et al., 2020) and humans (Schaschl et al., 2015). In both species, the genetic variation of *OXTR* is further associated with differences in social behavior (Eales, 1989; Pfenning et al., 2014; Shilton et al., 2020). More generally, the genes that have been positively selected in humans compared to Neanderthals are enriched in candidates for mammal domestication, particularly dog domestication (Theofanopoulou et al., 2017). Benítez-Burraco and colleagues (2020) have hypothesized a positive feedback loop between dog domestication and HSD, through the reduction of reactive aggression, contributing, albeit tangentially, to aspects of language evolution. Interestingly, dingoes are dogs that went feral around 5000 years ago and have since remained isolated from other dogs (Smith and Litchfield, 2010; Shipman, 2021). From the evidence gathered, it seems that dingoes fall somewhere between domesticated and wild canids in both phenotypic terms and in terms of their ability to communicate with humans (Smith and Litchfield, 2010). Thus, dingoes are able to read human cues, such as pointing, tapping, and gazing, to reach an object---usually more successfully than wolves---displaying some skills that are more on par with domestic dogs (Smith and Litchfield, 2010). However, when it comes to more nuanced tasks such as following only the experimenter's gaze to find food or following a signal from an incorrect location, it seems that dingoes underperform relative to domesticated dogs (Hare and Tomasello; 1999; Bräuer et al. 2006; Udell et al. 2008; Virányi et al. 2008). Additionally, Smith and Litchfield (2010) found that dingoes need to be familiar with an experimenter before being able to carry out the tasks in the experiment. This contrasts with domestic dogs, who can follow cues from any human subject. Parallels can be seen with ASD and 'feral' children. For instance, Watkins and

colleagues (2013) found that children with ASD performed significantly better on standardized tests when they were familiar with the examiner, as opposed to when the examiner was a stranger (Watkins et al., 2013). When looking at aspects like joint attention, and/or gaze following, various studies on ASD (e.g. Volkmar and Chawarska, 2005; Wetherby et al., 2007; Paul et al., 2008) show that people on the spectrum exhibit abnormal gaze patterns, and lower frequencies of joint attention, similar to the results found in feral canids. Additionally, characterization of ‘feral’ children describe avoidance of eye contact as a common trait, especially during initial exposure (Butler, 2003). These results indicate that domestication does seem to mark a difference in canids in terms of certain precursors of language (and particularly, language use), like joint attention, or the use of social cues to solve problems, and that these components are particularly impaired in conditions like ASD or ‘feral’ phenotypes in humans.

Another area of interest is the changes in vocal communication brought about by feralization, that can be gleaned from studies on feral cats and dogs as well. Yeon and colleagues (2011) found that feral cats generally use antagonistic vocalizations such as the growl and hiss much more than communicative ones (i.e. meow), but they do meow in response to a variety of interactions--both human and non-human. In contrast, domestic cats reserve the meow exclusively for the approach of a human, indicating a more communicative intent for the vocalization (Yeon et al., 2011). These findings concur with other research, for example, in domestic cats, the meow is heard much more frequently during cat-human interaction and much more rarely during cat-cat interactions (Brown, 1993; Bradshaw and Cameron Beaumont, 2000). Yeon and colleagues (2011) also found that feral cats generally produce longer vocalizations at a lower frequency (pitch) when in contact with humans, and tend to use agonistic vocalizations such as growling and hissing during encounters

638 with humans, while domestic cats are more vocal in social situations, and less likely to use
639 agonistic vocalizations. These findings parallel those of Nicastro (2004), who compared domestic
640 cat vocalizations with those of wild cats, finding that in general domestic cats produce calls of a
641 shorter duration and a higher pitch frequency than wild cats. Domestic cats are also known to
642 produce vocalizations typical of kittens, such as purring and chirping, which is not the case in feral
643 cats (Bradshaw and Cameron-Beaumont, 2000), which is in line with the neotenic features
644 typically associated to domestication. Regarding dogs and dingoes, Smith and Litchfield (2010)
645 found in their research that, similarly to wolves, dingoes are found to howl and whimper more than
646 domestic dogs, and bark less. Moreover, their barks tend to be shorter than their domestic
647 counterparts, and used almost exclusively for giving warnings. In contrast, domestic dogs' bark is
648 more versatile, and used in a variety of social situations (Corbett, 2004). Studies on domestic and
649 feral pigs show that they have a wide and varied repertoire that differs in some respects--for
650 example, the 'trumpeting' sound produced by non-domesticates has not been recorded in domestic
651 pigs (Tallet et al., 2013; Garcia et al., 2016). Evidence on pigs is difficult to interpret, however,
652 given the blurred lines that exist between 'feral' pigs and 'wild' pigs; since a great deal of
653 admixture takes place between the types of swine, it is hard to attribute changes to feralization
654 (Garcia et al., 2016). Although the vocal communication of mammals is more relevant to our
655 research because of their phylogenetic proximity, patterns from bird species can also contribute to
656 our knowledge of changes conferred through feralization, particularly because of the abundant
657 research on the notable effects of domestication on birdsongs. Gering and colleagues (2015) found
658 that feral chickens on Kauai have more extreme calls, and exhibit much more variation in their
659 song, than either wild jungle fowl or domestic chickens that share their habitat. More research is

necessary to see whether this change in vocalization complexity results from genetic admixture, environmental changes, or from a host of other epigenetic factors (Gering et al., 2015).

The vocal behavior of people with ASD seems to mirror some of the characteristics found in feral animals in the sense that it doesn't follow the normal patterns of TD peers. Vocalizations made by individuals with ASD are often described as 'different' (Ricks and Wing, 1976). Wallace and colleagues (2008) found that toddlers with ASD tend to produce a larger range of syllables with atypical pronunciation than their TD peers. Schoen and colleagues (2011) found that toddlers with ASD produce more atypical vocalizations than language-matched peers, with high pitched squeals being much more common in the ASD group. Kent and Murray (1982) also found that children on the ASD spectrum produce much more irregular and complex pitch patterns within the same breath than typically developing counterparts. Problems with prosody and pitch shifting are also reported by Russo and colleagues (2008), who speculated that problems with the auditory-motor pathway are to blame. Descriptions of 'feral' children can be seen as also support for this view, since similar characteristics (e.g. inappropriate control of pitch and intonation) are often observed in historical descriptions (Lebrun, 1980).

Conclusion

From our review of the literature on the physical, behavioral, and cognitive features exhibited by 'feral' children, people with ASD, and children with RAD, it can be concluded that many parallels exist between these conditions, and that differences with neurotypical subjects can be construed, to a certain extent, as a reversion of the changes brought about by HSD. This is also true, specifically, of the communication/language abilities, with people with these conditions exhibiting certain

specific communicative deficits relative to TD counterparts, which may to some extent parallel differences seen in feralized animals compared to domesticated variants.

Overall, these differences can be interpreted as a ‘hypo-domesticated’ phenotype, resulting from some genetic differences, in the case of ASD (see Benítez-Burraco et al., 2016 for a more detailed discussion), or from some environmental effects, specifically, abnormal patterns of socialization, in the case of ‘feral’ children and people with RAD. Our findings qualify these present-day phenotypes as useful proxies for studying the effects of HSD on our language abilities in the past, particularly because comparative evidence had already suggested that the HSD phenotype is not totally fixed in present-day human populations and, accordingly, can present in a quite variable fashion (Gleeson and Kushnik, 2018). In our opinion, the specific details of our research offer fertile ground for future hypothesis generation and experimental testing. This includes, first and foremost, genetic studies aimed at enlarging the set of common genetic determinants for ASD and RAD, as well as feralization. Specifically, it would be very interesting to know whether these common genes have been subject to recent selection in human populations. Likewise, neuroimaging research on feralized animals and on people with ASD or RAD during communication/language processing tasks should help identify the brain areas with a role in language processing that are more sensitive to HSD processes, and also, to provide additional support to the view that altered socialization patterns result in abnormal processing patterns of social cues at the brain level with a negative impact on language acquisition and use. Finally, because core aspects of human languages seem to result from cultural transmission, and because the two main conditions examined in the paper, namely ASD and RAD, feature abnormal socialization patterns, it would be interesting to conduct experiments with artificial languages

aimed at knowing how language is learnt and transmitted in atypical populations, and how this abnormal transmission impacts language features.

Besides its relevance for improving current theories about the evolution of language in the species, the research outlined in this paper is expected to have some clinical applications too. This HSD approach to clinical conditions like ASD or RAD is in line with systems biology approaches to complex conditions, arguing for a holistic approach to diseases, which examines all the involved factors (from genes to the environment), and their complex interaction during development. (see Benítez-Burraco, 2020 for discussion). From the considerations above about how our body, cognition, and behavior can be modified in response to environmental changes, we can expect to design better learning stimuli and more suitable learning environments for children suffering from these conditions.

References

Abbott, D.H., Dumesic, D.A., Eisner, J.R., Kemnitz, J.W., Goy, R. (1997) The prenatally androgenized female rhesus monkey as a model for PCOS. In: Azziz R, Nestler JE, Dewailly D, editors. *Androgen Excess Disorders in Women*. Philadelphia: Lippincott–Raven. 369–382.

Akefeldt, A., & Gillberg, C. (1991). Hypomelanoses of Ito in three cases with autism and autistic-like conditions. *Developmental medicine and child neurology*, 33(8), 737–743.

Aldridge, K., George, I. D., Cole, K. K., Austin, J. R., Takahashi, T. N., Duan, Y., & Miles, J. H. (2011). Facial phenotypes in subgroups of prepubertal boys with autism spectrum disorders are correlated with clinical phenotypes. *Molecular autism*, 2(1), 15.

Anastasiadi, D., Piferrer, F., Wellenreuther, M., & Benítez Burraco, A. (2022). Fish as Model Systems to Study Epigenetic Drivers in Human Self-Domestication and Neurodevelopmental Cognitive Disorders. *Genes*, 13(6), 987. MDPI AG. Retrieved from <http://dx.doi.org/10.3390/genes13060987>

Andari, E., Nishitani, S., Kaundinya, G., Caceres, G. A., Morrier, M. J., Ousley, O., Smith, A. K., Cubells, J. F., & Young, L. J. (2020). Epigenetic modification of the oxytocin receptor gene: implications for autism symptom severity and brain functional connectivity. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*, 45(7), 1150–1158.

Andersen, S. L., & Teicher, M. H. (2004). Delayed effects of early stress on hippocampal development. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*, 29(11), 1988–1993.

Anderson, T. M., vonHoldt, B. M., Candille, S. I., Musiani, M., Greco, C., Stahler, D. R., Smith, D. W., Padhukasahasram, B., Randi, E., Leonard, J. A., Bustamante, C. D., Ostrander, E. A., Tang, H., Wayne, R. K., & Barsh, G. S. (2009). Molecular and evolutionary history of melanism in North American gray wolves. *Science (New York, N.Y.)*, 323(5919), 1339–1343.

751

752 Andersson, M., Tangen, Ä., Farde, L., Bölte, S., Halldin, C., Borg, J., & Lundberg, J. (2021).
 753 Serotonin transporter availability in adults with autism-a positron emission tomography
 754 study. *Molecular psychiatry*, 26(5), 1647–1658.

755

756 Anitha, A., Nakamura, K., Thanseem, I., Yamada, K., Iwayama, Y., Toyota, T., Matsuzaki, H.,
 757 Miyachi, T., Yamada, S., Tsujii, M., Tsuchiya, K. J., Matsumoto, K., Iwata, Y., Suzuki, K.,
 758 Ichikawa, H., Sugiyama, T., Yoshikawa, T., & Mori, N. (2012). Brain region-specific altered
 759 expression and association of mitochondria-related genes in autism. *Molecular autism*, 3(1), 12.

760

761 Bach, M. (November 28, 2007) Domestic pigs quickly revert to wild roots. Retrieved from
 762 https://www.mlive.com/flintjournal/outdoors/2007/11/domestic_pigs_quickly_revert_t.html

763

764 Bailey, A., Phillips, W., & Rutter, M. (1996). Autism: towards an integration of clinical, genetic,
 765 neuropsychological, and neurobiological perspectives. *Journal of child psychology and*
 766 *psychiatry, and allied disciplines*, 37(1), 89–126.

767

768 Baker, L. M., Williams, L. M., Korgaonkar, M. S., Cohen, R. A., Heaps, J. M., & Paul, R. H.
 769 (2013). Impact of early vs. late childhood early life stress on brain morphometrics. *Brain imaging*
 770 *and behavior*, 7(2), 196–203.

771

772 Bakermans-Kranenburg, M. J., & Van Ijzendoorn, M. H. (2004). No association of the dopamine
 773 D4 receptor (DRD4) and -521 C/T promoter polymorphisms with infant attachment
 774 disorganization. *Attachment & human development*, 6(3), 211–222.

775

776 Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2007). Research Review: genetic
 777 vulnerability or differential susceptibility in child development: the case of attachment. *Journal of*
 778 *child psychology and psychiatry, and allied disciplines*, 48(12), 1160–1173.

779

780 Ballard, J., & Wilson, L. (2019). The Australian dingo: untamed or feral?. *Frontiers in zoology*, 16,
 781 2.

782

783 Baron-Cohen S. (1988). Social and pragmatic deficits in autism: cognitive or affective?. *Journal*
 784 *of autism and developmental disorders*, 18(3), 379–402.

785

786 Baron-Cohen S. (1997). Hey! It was just a joke! Understanding propositions and propositional
 787 attitudes by normally developing children and children with autism. *The Israel journal of*
 788 *psychiatry and related sciences*, 34(3), 174–178.

789

790 Baron-Cohen, S., O'Riordan, M., Stone, V., Jones, R., & Plaisted, K. (1999). Recognition of faux
 791 pas by normally developing children and children with Asperger syndrome or high-functioning
 792 autism. *Journal of autism and developmental disorders*, 29(5), 407–418.

793

794 Baron-Cohen, S. (2000). Theory of mind and autism: A fifteen year review. In S. Baron-Cohen,
795 H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding other minds: Perspectives from*
796 *developmental cognitive neuroscience* (pp. 3–20). Oxford University Press.

797

798 Bartolucci, G., Pierce, S., Streiner, D., & Eppel, P. T. (1976). Phonological investigation of verbal
799 autistic and mentally retarded subjects. *Journal of autism and childhood schizophrenia*, 6(4), 303–
800 316.

801

802 Bates, T. C., Luciano, M., Medland, S. E., Montgomery, G. W., Wright, M. J., & Martin, N. G.
803 (2011). Genetic variance in a component of the language acquisition device: ROBO1
804 polymorphisms associated with phonological buffer deficits. *Behavior genetics*, 41(1), 50–57.

805

806 Bellacosa, A., Cicchillitti, L., Schepis, F., Riccio, A., Yeung, A. T., Matsumoto, Y., Golemis, E.
807 A., Genuardi, M., & Neri, G. (1999). MED1, a novel human methyl-CpG-binding endonuclease,
808 interacts with DNA mismatch repair protein MLH1. *Proceedings of the National Academy of*
809 *Sciences of the United States of America*, 96(7), 3969–3974.

810

811 Benítez-Burraco, A., Ferretti, F., & Progovac, L. (2021a). Human Self-Domestication and the
812 Evolution of Pragmatics. *Cognitive science*, 45(6), e12987.

813

814 Benítez-Burraco, A., Lattanzi, W., & Murphy, E. (2016). Language Impairments in ASD Resulting
815 from a Failed Domestication of the Human Brain. *Frontiers in neuroscience*, 10, 373.

816

817 Benítez-Burraco, A., & Murphy, E. (2016). The Oscillopathic Nature of Language Deficits in
818 Autism: From Genes to Language Evolution. *Frontiers in human neuroscience*, 10, 120.
819

820 Benítez-Burraco, A., Pörtl, .D, Jung, C.A.. (2021b). Did dog domestication contribute to language
821 evolution? *Frontiers in Psychology*, 12, 695116. .
822

823 Benítez-Burraco, A., Progovac, L. (2020) A four-stage model for language evolution under the
824 effects of human self-domestication. *Language and Communication*, 73, 1–17.
825

826 Benítez-Burraco, A., Theofanopoulou, C., Boeckx, C. (2016) Globularization and domestication.
827 *Topoi*, 37, 265–278.
828

829 Benítez-Burraco A. (2017). Figurative Language, Language Disorders, and Language(s)
830 Evolution. *Frontiers in psychology*, 8, 1713.
831

832 Benítez-Burraco, A. (2020). The golden mean: A systems biology approach to language disorders.
833 *Pragmalingüística* (2), 30-44.
834

835 Bennett, J., Espie, C., Duncan, B., & Minnis, H. (2009). A qualitative exploration of children's
836 understanding of indiscriminate friendliness. *Clinical child psychology and psychiatry*, 14(4),
837 595–618.
838

839 Bettelheim, B. (1959) Feral Children and Autistic Children. *American Journal of Sociology*. 64,
840 5, 455-467.

841

842 Bhandari, R., Paliwal, J. K., & Kuhad, A. (2020). Neuropsychopathology of Autism Spectrum
843 Disorder: Complex Interplay of Genetic, Epigenetic, and Environmental Factors. *Advances in*
844 *neurobiology*, 24, 97–141.

845

846 Birks, J.D.S., Kitchener, A.C., eds. (1999) Ecology of the Polecat in Lowland England. In: J.D.S.
847 Birks and A.C. Kitchener, eds. The distribution and status of the polecat *Mustela putorius* in Britain
848 in the 1990s. The Vincent Wildlife Trust, London, 111–130.

849

850 Blanchard, D. C., Griebel, G., Rodgers, R. J., & Blanchard, R. J. (1998). Benzodiazepine and
851 serotonergic modulation of antipredator and conspecific defense. *Neuroscience and biobehavioral*
852 *reviews*, 22(5), 597–612.

853

854 Boeckx, C., & Benítez-Burraco, A. (2014). The shape of the human language-ready
855 brain. *Frontiers in psychology*, 5, 282.

856

857 Bölte, S., Mahdi, S., de Vries, P. J., Granlund, M., Robison, J. E., Shulman, C., Swedo, S., Tonge,
858 B., Wong, V., Zwaigenbaum, L., Segerer, W., & Selb, M. (2019). The Gestalt of functioning in
859 autism spectrum disorder: Results of the international conference to develop final consensus
860 International Classification of Functioning, Disability and Health core sets. *Autism : the*
861 *international journal of research and practice*, 23(2), 449–467.

862

863 Bourguignon, N., Nadig, A., Valois, D. (2012) The biolinguistics of autism: emergent
864 perspectives. *Biolinguistics*, 6, 124–165.

865

866 Bradshaw, J.W.S., Cameron-Beaumont, C.L. (2000) The signalling repertoire of the domestic cat
867 and its undomesticated relatives. In: *The Domestic Cat: The Biology of Its Behaviour* (Turner, D.
868 C. & Bateson, P. P. G., eds) Cambridge University Press, New York, NY, 68—93.

869

870 Bräuer, J., Kaminski, J., Riedel, J., Call, J., & Tomasello, M. (2006). Making inferences about the
871 location of hidden food: social dog, causal ape. *Journal of comparative psychology* (Washington,
872 D.C. : 1983), 120(1), 38–47.

873

874 Brose, K., Bland, K. S., Wang, K. H., Arnott, D., Henzel, W., Goodman, C. S., Tessier-Lavigne,
875 M., & Kidd, T. (1999). Slit proteins bind Robo receptors and have an evolutionarily conserved
876 role in repulsive axon guidance. *Cell*, 96(6), 795–806.

877

878 Brown, S.L. (1993) The social behaviour of neutered domestic cats. Unpublished doctoral
879 dissertation. University of Southampton, United Kingdom.

880

881 Burnside, K., Wright, K., & Poulin-Dubois, D. (2017). Social motivation and implicit theory of
882 mind in children with autism spectrum disorder. *Autism research : official journal of the*
883 *International Society for Autism Research*, 10(11), 1834–1844.

884

885 Butler, T. (2003) Language and consciousness: what can we learn about feral children? Masters
 886 thesis, Memorial University of Newfoundland.

887

888 Campbell, D. J., Chang, J., & Chawarska, K. (2014). Early generalized overgrowth in autism
 889 spectrum disorder: prevalence rates, gender effects, and clinical outcomes. *Journal of the*
 890 *American Academy of Child and Adolescent Psychiatry*, 53(10), 1063–73.e5.

891

892 Cannell, J. J., & Grant, W. B. (2013). What is the role of vitamin D in autism?. *Dermato-*
 893 *endocrinology*, 5(1), 199–204.

894

895 Cantù, C., Ierardi, R., Alborelli, I., Fugazza, C., Cassinelli, L., Piconese, S., Bosè, F., Ottolenghi,
 896 S., Ferrari, G., & Ronchi, A. (2011). Sox6 enhances erythroid differentiation in human erythroid
 897 progenitors. *Blood*, 117(13), 3669–3679.

898

899

900 Carter, C. S., Grippo, A. J., Pournajafi-Nazarloo, H., Ruscio, M. G., & Porges, S. W. (2008).
 901 Oxytocin, vasopressin and sociality. *Progress in brain research*, 170, 331–336.

902

903 Carter, C.S. (2003) Developmental consequences of oxytocin. *Physiology of Behavior*, 79 (3),
 904 383-97.

905

Caspers, K. M., Paradiso, S., Yucuis, R., Troutman, B., Arndt, S., & Philibert, R. (2009). Association between the serotonin transporter promoter polymorphism (5-HTTLPR) and adult unresolved attachment. *Developmental psychology*, 45(1), 64–76.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., Taylor, A., & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science (New York, N.Y.)*, 297(5582), 851–854.

Castelli, F., Frith, C., Happé, F., & Frith, U. (2002). Autism, Asperger syndrome and brain mechanisms for the attribution of mental states to animated shapes. *Brain : a journal of neurology*, 125(Pt 8), 1839–1849.

Chawarska, K., Campbell, D., Chen, L., Shic, F., Klin, A., & Chang, J. (2011). Early generalized overgrowth in boys with autism. *Archives of general psychiatry*, 68(10), 1021–1031.

Cieri, R., Churchill, S., Franciscus, R., Tan, J., Hare, B. (2014) Craniofacial Feminization, Social Tolerance, and the Origins of Behavioral Modernity. *Current Anthropology*, 55, 419-443.

Cohen, I. L., Liu, X., Lewis, M. E., Chudley, A., Forster-Gibson, C., Gonzalez, M., Jenkins, E. C., Brown, W. T., & Holden, J. J. (2011). Autism severity is associated with child and maternal MAOA genotypes. *Clinical genetics*, 79(4), 355–362.

Cohen, I. L., Liu, X., Schutz, C., White, B. N., Jenkins, E. C., Brown, W. T., & Holden, J. J. (2003). Association of autism severity with a monoamine oxidase A functional polymorphism. *Clinical genetics*, 64(3), 190–197.

Colich, N. L., Wang, A. T., Rudie, J. D., Hernandez, L. M., Bookheimer, S. Y., & Dapretto, M. (2012). Atypical Neural Processing of Ironic and Sincere Remarks in Children and Adolescents with Autism Spectrum Disorders. *Metaphor and symbol*, 27(1), 70–92.

Cooper, R. A., & Ritchey, M. (2019). Cortico-hippocampal network connections support the multidimensional quality of episodic memory. *eLife*, 8, e45591.

Corballis M. C. (2019). Language, Memory, and Mental Time Travel: An Evolutionary Perspective. *Frontiers in human neuroscience*, 13, 217.

Corballis M. C. (2018). Space, time, and language. *Cognitive processing*, 19(Suppl 1), 89–92.

Corbett, L. (2004) Dingo. In Claudio Sillero-Zubiri; Michael Hoffmann; David W. Macdonald (eds.) Canids: Foxes, Wolves, Jackals and Dogs. International Union for Conservation of Nature and Natural Resources.

Corbin, J. (2007) Reactive Attachment Disorder: A Biopsychosocial Disturbance of Attachment. *Child and Adolescent Social Work Journal*, 24, 539-552.

951 Corval, R., Mesquita, A., & Soares, I. (2020). Links Between Reactive Attachment Disorder,
 952 Caregiving and Temperament: A Differential Susceptibility Perspective. *The Journal of genetic*
 953 *psychology*, 181(5), 405–412.

954

955 Courchesne, E., Campbell, K., & Solso, S. (2011). Brain growth across the life span in autism:
 956 age-specific changes in anatomical pathology. *Brain research*, 1380, 138–145.

957

958 Coutinho, A. M., Oliveira, G., Morgadinho, T., Fesel, C., Macedo, T. R., Bento, C., Marques, C.,
 959 Ataíde, A., Miguel, T., Borges, L., & Vicente, A. M. (2004). Variants of the serotonin transporter
 960 gene (SLC6A4) significantly contribute to hyperserotonemia in autism. *Molecular*
 961 *psychiatry*, 9(3), 264–271.

962

963 Cukier, H. N., Rabionet, R., Konidari, I., Rayner-Evans, M. Y., Baltos, M. L., Wright, H. H.,
 964 Abramson, R. K., Martin, E. R., Cuccaro, M. L., Pericak-Vance, M. A., & Gilbert, J. R. (2010).
 965 Novel variants identified in methyl-CpG-binding domain genes in autistic
 966 individuals. *Neurogenetics*, 11(3), 291–303.

967

968 Curin, J. M., Terzić, J., Petković, Z. B., Zekan, L., Terzić, I. M., & Susnjara, I. M. (2003). Lower
 969 cortisol and higher ACTH levels in individuals with autism. *Journal of autism and developmental*
 970 *disorders*, 33(4), 443–448.

971

972 Curtiss, S., Fromkin, V., Krashen, S., Rigler, M., Rigler, D. (1974) The linguistic development of
 973 Genie. *Language*, 50, 528-554.

974

975 Cuyvers, B., Vervoort, E., & Bosmans, G. (2020). Reactive attachment disorder symptoms and
 976 prosocial behavior in middle childhood: the role of Secure Base Script knowledge. *BMC*
 977 *psychiatry*, 20(1), 524.

978

979 Damaj, L., Lupien-Meilleur, A., Lortie, A., Riou, É., Ospina, L. H., Gagnon, L., Vanasse, C., &
 980 Rossignol, E. (2015). CACNA1A haploinsufficiency causes cognitive impairment, autism and
 981 epileptic encephalopathy with mild cerebellar symptoms. *European journal of human genetics* :
 982 *EJHG*, 23(11), 1505–1512.

983

984 Daniels, T. J., & Bekoff, M. (1989). Feralization: The making of wild domestic
 985 animals. *Behavioural processes*, 19(1-3), 79–94.

986

987 De Rubeis, S., He, X., Goldberg, A. P., Poultney, C. S., Samocha, K., Cicek, A. E., Kou, Y., Liu,
 988 L., Fromer, M., Walker, S., Singh, T., Klei, L., Kosmicki, J., Shih-Chen, F., Aleksic, B., Biscaldi,
 989 M., Bolton, P. F., Brownfeld, J. M., Cai, J., Campbell, N. G., ... Buxbaum, J. D. (2014). Synaptic,
 990 transcriptional and chromatin genes disrupted in autism. *Nature*, 515(7526), 209–215.

991

992 Deacon, T.W. (2009) Relaxed selection and the role of epigenesis in the evolution of language. In
 993 Oxford Handbook of Developmental Behavioral Neuroscience, eds Blumberg, M., Freeman, J.,
 994 Robinson, S. (New York, NY: Oxford University Press).

995

Derelle, R., Kondrashov, F. A., Arkhipov, V. Y., Corbel, H., Frantz, A., Gasparini, J., Jacquin, L., Jacob, G., Thibault, S., & Baudry, E. (2013). Color differences among feral pigeons (*Columba livia*) are not attributable to sequence variation in the coding region of the melanocortin-1 receptor gene (MC1R). *BMC research notes*, 6, 310.

Di Sante, M., Sylvestre, A., Bouchard, C., & Leblond, J. (2019). The Pragmatic Language Skills of Severely Neglected 42-Month-Old Children: Results of the ELLAN Study. *Child maltreatment*, 24(3), 244–253.

Di Sante, M., Sylvestre, A., Bouchard, C., & Leblond, J. (2020). Parental behaviors associated with the level of pragmatic language ability among 42-month-old neglected children. *Child abuse & neglect*, 104, 104482.

Dobrova-Krol, N. A., van Ijzendoorn, M. H., Bakermans-Kranenburg, M. J., Cyr, C., & Juffer, F. (2008). Physical growth delays and stress dysregulation in stunted and non-stunted Ukrainian institution-reared children. *Infant Behavior and Development*, 31, 539–553.

Eales, L.A. (1989) The influences of visual and vocal interaction on song learning in zebra finches. *Animal Behavior*, 37, 507–520.

Eigsti, I. M., Bennetto, L., & Dadlani, M. B. (2007). Beyond pragmatics: morphosyntactic development in autism. *Journal of autism and developmental disorders*, 37(6), 1007–1023.

1019 El-Baz, F., Hamza, R. T., Ayad, M. S., & Mahmoud, N. H. (2014). Hyperandrogenemia in male
1020 autistic children and adolescents: relation to disease severity. *International journal of adolescent*
1021 *medicine and health*, 26(1), 79–84.

1022

1023 Ellis, E. E., Yilanli, M., & Saadabadi, A. (2021). Reactive Attachment Disorder. In *StatPearls*.
1024 StatPearls Publishing.

1025

1026 Evin, A., Dobney, K., Schafberg, R., Owen, J., Vidarsdottir, U. S., Larson, G., & Cucchi, T. (2015).
1027 Phenotype and animal domestication: A study of dental variation between domestic, wild, captive,
1028 hybrid and insular *Sus scrofa*. *BMC evolutionary biology*, 15(1), 6.

1029

1030 Farmer, C., Butter, E., Mazurek, M. O., Cowan, C., Lainhart, J., Cook, E. H., DeWitt, M. B., &
1031 Aman, M. (2015). Aggression in children with autism spectrum disorders and a clinic-referred
1032 comparison group. *Autism : the international journal of research and practice*, 19(3), 281–291.

1033

1034 Feulner, P., Gratten, J., Kijas, J. W., Visscher, P. M., Pemberton, J. M., & Slate, J. (2013).
1035 Introgression and the fate of domesticated genes in a wild mammal population. *Molecular*
1036 *ecology*, 22(16), 4210–4221.

1037

1038 Fisher, S. E., Francks, C., Marlow, A. J., MacPhie, I. L., Newbury, D. F., Cardon, L. R., Ishikawa-
1039 Brush, Y., Richardson, A. J., Talcott, J. B., Gayán, J., Olson, R. K., Pennington, B. F., Smith, S.
1040 D., DeFries, J. C., Stein, J. F., & Monaco, A. P. (2002). Independent genome-wide scans identify
1041 a chromosome 18 quantitative-trait locus influencing dyslexia. *Nature genetics*, 30(1), 86–91.

1042

1043 Fries, A. B., Ziegler, T. E., Kurian, J. R., Jacoris, S., & Pollak, S. D. (2005). Early experience in
 1044 humans is associated with changes in neuropeptides critical for regulating social
 1045 behavior. *Proceedings of the National Academy of Sciences of the United States of*
 1046 *America*, 102(47), 17237–17240.

1047

1048 Fromkin, V., Krashen, S., Curtiss, S., Rigler, D., Rigler, M. (1974) The Development of Language
 1049 in Genie: a Case of Language Acquisition beyond the ‘Critical Period’. *Brain and Language*, 1,
 1050 81-107.

1051

1052 Fujisawa, T. X., Shimada, K., Takiguchi, S., Mizushima, S., Kosaka, H., Teicher, M. H., &
 1053 Tomoda, A. (2018). Type and timing of childhood maltreatment and reduced visual cortex volume
 1054 in children and adolescents with reactive attachment disorder. *NeuroImage. Clinical*, 20, 216–221.

1055

1056 Fukase, H., Kondo, O., & Ishida, H. (2015). Size and placement of developing anterior teeth in
 1057 immature Neanderthal mandibles from Dederiyeh Cave, Syria: implications for emergence of the
 1058 modern human chin. *American journal of physical anthropology*, 156(3), 482–488.

1059

1060 Furuhashi M. (2019). Fatty Acid-Binding Protein 4 in Cardiovascular and Metabolic
 1061 Diseases. *Journal of atherosclerosis and thrombosis*, 26(3), 216–232.

1062

1063 Fusaroli, R., Lambrechts, A., Bang, D., Bowler, D. M., & Gaigg, S. B. (2017). "Is voice a marker
 1064 for Autism spectrum disorder? A systematic review and meta-analysis". *Autism research : official
 1065 journal of the International Society for Autism Research*, 10(3), 384–407.

1066

1067 Garcia, M., Gingras, B., Bowling, D. L., Herbst, C. T., Boeckle, M., Locatelli, Y., & Fitch, W. T.
 1068 (2016). Structural Classification of Wild Boar (*Sus scrofa*) Vocalizations. *Ethology : formerly
 1069 Zeitschrift fur Tierpsychologie*, 122(4), 329–342.

1070

1071 Gering, E., Incorvaia, D., Henriksen, R., Conner, J., Getty, T., & Wright, D. (2019). Getting Back
 1072 to Nature: Feralization in Animals and Plants. *Trends in ecology & evolution*, 34(12), 1137–1151.

1073

1074 Gering, E., Johnsson, M., Willis, P., Getty, T., & Wright, D. (2015). Mixed ancestry and admixture
 1075 in Kauai's feral chickens: invasion of domestic genes into ancient Red Junglefowl
 1076 reservoirs. *Molecular ecology*, 24(9), 2112–2124.

1077

1078 Gesell, A. (1942) Wolf Child and Human Child. London, The Scientific Book Club.

1079

1080 Ghezzi, D., Antognazza, M. R., Dal Maschio, M., Lanzarini, E., Benfenati, F., & Lanzani, G.
 1081 (2011). A hybrid bioorganic interface for neuronal photoactivation. *Nature communications*, 2,
 1082 166.

1083

1084 Gleason, M. M., Fox, N. A., Drury, S., Smyke, A., Egger, H. L., Nelson, C. A., 3rd, Gregas, M.
 1085 C., & Zeanah, C. H. (2011). Validity of evidence-derived criteria for reactive attachment disorder:

1086 indiscriminately social/disinhibited and emotionally withdrawn/inhibited types. *Journal of the*
 1087 *American Academy of Child and Adolescent Psychiatry*, 50(3), 216–231.e3.

1088

1089 Gleeson, B. T., & Kushnick, G. (2018). Female status, food security, and stature sexual
 1090 dimorphism: Testing mate choice as a mechanism in human self-domestication. *American journal*
 1091 *of physical anthropology*, 167(3), 458–469.

1092

1093 Goding, C. R., & Arnheiter, H. (2019). MITF-the first 25 years. *Genes & development*, 33(15-16),
 1094 983–1007.

1095

1096 Gómez-Lado, C., Eirís-Puñal, J., Blanco-Barca, O., del Río-Latorre, E., Fernández-Redondo, V.,
 1097 & Castro-Gago, M. (2004). Hipomelanosis de Ito. Un síndrome neurocutáneo heterogéneo y
 1098 posiblemente infradiagnosticado [Hypomelanosis of Ito. A possibly under-diagnosed
 1099 heterogeneous neurocutaneous syndrome]. *Revista de neurologia*, 38(3), 223–228.

1100

1101 Goozen, S. H., van den Ban, E., Matthys, W., Cohen-Kettenis, P. T., Thijssen, J. H., & van
 1102 Engeland, H. (2000). Increased adrenal androgen functioning in children with oppositional defiant
 1103 disorder: a comparison with psychiatric and normal controls. *Journal of the American Academy of*
 1104 *Child and Adolescent Psychiatry*, 39(11), 1446–1451.

1105

1106 Green, J., & Goldwyn, R. (2002). Annotation: attachment disorganisation and psychopathology:
 1107 new findings in attachment research and their potential implications for developmental

1108 psychopathology in childhood. *Journal of child psychology and psychiatry, and allied*
1109 *disciplines*, 43(7), 835–846.

1110

1111 Gupta, A., Labus, J., Kilpatrick, L. A., Bonyadi, M., Ashe-McNalley, C., Heendeniya, N., Bradesi,
1112 S., Chang, L., & Mayer, E. A. (2016). Interactions of early adversity with stress-related gene
1113 polymorphisms impact regional brain structure in females. *Brain structure & function*, 221(3),
1114 1667–1679.

1115

1116 Hamza, R. T., Hewedi, D. H., & Ismail, M. A. (2010). Basal and adrenocorticotrophic hormone
1117 stimulated plasma cortisol levels among Egyptian autistic children: relation to disease
1118 severity. *Italian journal of pediatrics*, 36, 71.

1119

1120 Happé F. G. (1995). The role of age and verbal ability in the theory of mind task performance of
1121 subjects with autism. *Child development*, 66(3), 843–855.

1122

1123 Happé F. G. (1993). Communicative competence and theory of mind in autism: a test of relevance
1124 theory. *Cognition*, 48(2), 101–119.

1125

1126 Hare, B., Tomasello, M. (1999) Domestic dogs (*Canis familiaris*) use human and conspecific social
1127 cues to locate hidden food. *Journal of Comprehensive Psychology*, 113, 173–177.

1128

1129 Hare, B., Wobber, V., Wrangham, R.W. (2012) The self-domestication hypothesis: bonobos
1130 evolved due to selection against male aggression. *Animal Behavior*, 83, 573–585.

1131

1132 Hare B. (2017). Survival of the Friendliest: Homo sapiens Evolved via Selection for
 1133 Prosociality. *Annual review of psychology*, 68, 155–186.

1134

1135 Hare, B., & Woods, V. (2020). *Survival of the friendliest: Understanding our origins and*
 1136 *rediscovering our common humanity*. New York: Penguin Random House.

1137 Hashimoto, T., Aihara, R., Tayama, M., Miyazaki, M., Shirakawa, Y., & Kuroda, Y. (1991).
 1138 Reduced thyroid-stimulating hormone response to thyrotropin-releasing hormone in autistic
 1139 boys. *Developmental medicine and child neurology*, 33(4), 313–319.

1140

1141 Haughian, J. M., & Bradford, A. P. (2009). Protein kinase C alpha (PKCalpha) regulates growth
 1142 and invasion of endometrial cancer cells. *Journal of cellular physiology*, 220(1), 112–118.

1143

1144 Heim, C. M., Mayberg, H. S., Mletzko, T., Nemeroff, C. B., & Pruessner, J. C. (2013). Decreased
 1145 cortical representation of genital somatosensory field after childhood sexual abuse. *The American*
 1146 *journal of psychiatry*, 170(6), 616–623.

1147

1148 Heinrichs, M., von Dawans, B., & Domes, G. (2009). Oxytocin, vasopressin, and human social
 1149 behavior. *Frontiers in neuroendocrinology*, 30(4), 548–557.

1150

1151 Hernádi, A., Kis, A., Turcsán, B., & Topál, J. (2012). Man's underground best friend: domestic
 1152 ferrets, unlike the wild forms, show evidence of dog-like social-cognitive skills. *PloS one*, 7(8),
 1153 e43267.

1154

1155 Herrmann, E., Hare, B., Cissewski, J., & Tomasello, M. (2011). A comparison of temperament in
 1156 nonhuman apes and human infants. *Developmental science*, 14(6), 1393–1405.

1157

1158 Hinners, I., Wendler, F., Fei, H., Thomas, L., Thomas, G., & Tooze, S. A. (2003). AP-1 recruitment
 1159 to VAMP4 is modulated by phosphorylation-dependent binding of PACS-1. *EMBO reports*, 4(12),
 1160 1182–1189.

1161

1162 Heller, S. S., Boris, N. W., Fuselier, S. H., Page, T., Koren-Karie, N., & Miron, D. (2006). Reactive
 1163 attachment disorder in maltreated twins follow-up: from 18 months to 8 years. *Attachment &*
 1164 *human development*, 8(1), 63–86.

1165

1166 Hollocks, M. J., Howlin, P., Papadopoulos, A. S., Khondoker, M., & Simonoff, E. (2014).
 1167 Differences in HPA-axis and heart rate responsiveness to psychosocial stress in children with
 1168 autism spectrum disorders with and without co-morbid anxiety. *Psychoneuroendocrinology*, 46,
 1169 32–45.

1170

1171 Hong, M., Moon, D. S., Chang, H., Lee, S. Y., Cho, S. W., Lee, K. S., Park, J. A., Lee, S. M., &
 1172 Bahn, G. H. (2018). Incidence and Comorbidity of Reactive Attachment Disorder: Based on
 1173 National Health Insurance Claims Data, 2010-2012 in Korea. *Psychiatry investigation*, 15(2),
 1174 118–123.

1175

1176 Huber, L., Salobir, K., Mundry, R., Cimorelli, G. Selective overimitation in dogs. (2020) Learning
 1177 Behavior, 48, 113-123.
 1178
 1179 Iossifov, I., O'Roak, B. J., Sanders, S. J., Ronemus, M., Krumm, N., Levy, D., Stessman, H. A.,
 1180 Witherspoon, K. T., Vives, L., Patterson, K. E., Smith, J. D., Paepker, B., Nickerson, D. A., Dea,
 1181 J., Dong, S., Gonzalez, L. E., Mandell, J. D., Mane, S. M., Murtha, M. T., Sullivan, C. A., ...
 1182 Wigler, M. (2014). The contribution of de novo coding mutations to autism spectrum
 1183 disorder. *Nature*, 515(7526), 216–221.
 1184
 1185 Iossifov, I., Ronemus, M., Levy, D., Wang, Z., Hakker, I., Rosenbaum, J., Yamrom, B., Lee, Y.
 1186 H., Narzisi, G., Leotta, A., Kendall, J., Grabowska, E., Ma, B., Marks, S., Rodgers, L., Stepansky,
 1187 A., Troge, J., Andrews, P., Bekritsky, M., Pradhan, K., ... Wigler, M. (2012). De novo gene
 1188 disruptions in children on the autistic spectrum. *Neuron*, 74(2), 285–299.
 1189
 1190 Itoh, M., Kim, C. H., Palardy, G., Oda, T., Jiang, Y. J., Maust, D., Yeo, S. Y., Lorick, K., Wright,
 1191 G. J., Ariza-McNaughton, L., Weissman, A. M., Lewis, J., Chandrasekharappa, S. C., & Chitnis,
 1192 A. B. (2003). Mind bomb is a ubiquitin ligase that is essential for efficient activation of Notch
 1193 signaling by Delta. *Developmental cell*, 4(1), 67–82.
 1194
 1195 Jacobson L. (2014). Hypothalamic-pituitary-adrenocortical axis: neuropsychiatric
 1196 aspects. *Comprehensive Physiology*, 4(2), 715–738.
 1197

1198 John, S., & Jaeggi, A. V. (2021). Oxytocin levels tend to be lower in autistic children: A meta-
 1199 analysis of 31 studies. *Autism : the international journal of research and practice*, 25(8), 2152–
 1200 2161.

1201

1202 Johnston, A.M., Turrin, C., Watson, L., Arre, A.M., Santos, L.R. (2017) Uncovering the origins
 1203 of dog-human eye contact: dingoes establish eye contact more than wolves, but less than dogs.
 1204 *Animal Behavior*, 133, 123–9.

1205

1206 Jung, M., Takiguchi, S., Hamamura, S., Mizuno, Y., Kosaka, H., & Tomoda, A. (2020). Thalamic
 1207 Volume Is Related to Increased Anterior Thalamic Radiations in Children with Reactive
 1208 Attachment Disorder. *Cerebral cortex (New York, N.Y. : 1991)*, 30(7), 4238–4245.

1209

1210 Kaland, N., Møller-Nielsen, A., Callesen, K., Mortensen, E. L., Gottlieb, D., & Smith, L. (2002).
 1211 A new 'advanced' test of theory of mind: evidence from children and adolescents with Asperger
 1212 syndrome. *Journal of child psychology and psychiatry, and allied disciplines*, 43(4), 517–528.

1213

1214 Kaminski, J., Nitzschner, M. (2013) Do dogs get the point? A review of dog-human
 1215 communication ability. *Learning and Motivation*, 44, 294–302.

1216

1217 Kapoor, S. (1973) Socialization and Feral Children. *Revista Internacional de Sociología*, 31,195.

1218

1219 Katoh M. (2001). Molecular cloning and characterization of MFRP, a novel gene encoding a
 1220 membrane-type Frizzled-related protein. *Biochemical and biophysical research*
 1221 *communications*, 282(1), 116–123.
 1222
 1223 Kempf, J., Voeller, K. (2008) Reactive attachment disorder in adolescence. *Adolescent Psychiatry*
 1224 (United States), 30, 159-178.
 1225
 1226 Kent, R. D., & Murray, A. D. (1982). Acoustic features of infant vocalic utterances at 3, 6, and 9
 1227 months. *The Journal of the Acoustical Society of America*, 72(2), 353–365.
 1228
 1229 Kidd, T., Brose, K., Mitchell, K. J., Fetter, R. D., Tessier-Lavigne, M., Goodman, C. S., & Tear,
 1230 G. (1998). Roundabout controls axon crossing of the CNS midline and defines a novel subfamily
 1231 of evolutionarily conserved guidance receptors. *Cell*, 92(2), 205–215.
 1232
 1233 Kilsby, A. J., Cruwys, M., Kukendrajah, C., Russell-Eggitt, I., Raglan, E., Rajput, K., Lohse, P.,
 1234 & Brady, A. F. (2013). Homozygosity for piebaldism with a proven KIT mutation resulting in
 1235 depigmentation of the skin and hair, deafness, developmental delay and autism spectrum
 1236 disorder. *Clinical dysmorphology*, 22(2), 64–67.
 1237
 1238 Kissine, M. (2012) Pragmatics, cognitive flexibility and autism spectrum disorders. *Mind and*
 1239 *Language*, 27, 1–28.
 1240

1241 Kjelgaard, M. M., & Tager-Flusberg, H. (2001). An Investigation of Language Impairment in
 1242 Autism: Implications for Genetic Subgroups. *Language and cognitive processes*, 16(2-3), 287–
 1243 308.
 1244
 1245 Knickmeyer, R. C., Wheelwright, S., Hoekstra, R., & Baron-Cohen, S. (2006). Age of menarche
 1246 in females with autism spectrum conditions. *Developmental medicine and child neurology*, 48(12),
 1247 1007–1008.
 1248
 1249 Kočovská, E., Wilson, P., Young, D., Wallace, A. M., Gorski, C., Follan, M., Smillie, M.,
 1250 Puckering, C., Barnes, J., Gillberg, C., & Minnis, H. (2013). Cortisol secretion in children with
 1251 symptoms of reactive attachment disorder. *Psychiatry research*, 209(1), 74–77.
 1252
 1253 Komiyama, T., Iwama, H., Osada, N., Nakamura, Y., Kobayashi, H., Tateno, Y., & Gojobori, T.
 1254 (2014). Dopamine receptor genes and evolutionary differentiation in the domestication of fighting
 1255 cocks and long-crowing chickens. *PloS one*, 9(7), e101778.
 1256
 1257 Krause, E. T., Kjaer, J. B., Dudde, A., Schrader, L., & Phi-van, L. (2019). Fear but not social
 1258 behaviour is affected by a polymorphism in the 5'-flanking region of the serotonin transporter (5-
 1259 HTT) gene in adult hens. *Behavioural brain research*, 361, 50–53.
 1260
 1261 Krug, A., Nieratschker, V., Markov, V., Krach, S., Jansen, A., Zerres, K., Eggermann, T., Stöcker,
 1262 T., Shah, N. J., Treutlein, J., Mühleisen, T. W., & Kircher, T. (2010). Effect of CACNA1C

rs1006737 on neural correlates of verbal fluency in healthy individuals. *NeuroImage*, 49(2), 1831–1836.

Kruska D. C. (2005). On the evolutionary significance of encephalization in some eutherian mammals: effects of adaptive radiation, domestication, and feralization. *Brain, behavior and evolution*, 65(2), 73–108.

Kruska, D.C.T. (2014) Comparative quantitative investigations on brains of wild cavies (*Cavia aperea*) and guinea pigs (*Cavia aperea* f. *porcellus*) A contribution to size changes of CNS structures due to domestication. *Mammalian Biology—Zeitschrift für Säugetierkunde*, 79, 230–9.

Kruska, D., & Röhrs, M. (1974). Comparative--quantitative investigations on brains of feral pigs from the Galapagos Islands and of European domestic pigs. *Zeitschrift für Anatomie und Entwicklungsgeschichte*, 144(1), 61–73.

Kumar, D., Koyanagi, I., Carrier-Ruiz, A., Vergara, P., Srinivasan, S., Sugaya, Y., Kasuya, M., Yu, T. S., Vogt, K. E., Muratani, M., Ohnishi, T., Singh, S., Teixeira, C. M., Chérasse, Y., Naoi, T., Wang, S. H., Nondhalee, P., Osman, B., Kaneko, N., Sawamoto, K., ... Sakaguchi, M. (2020). Sparse Activity of Hippocampal Adult-Born Neurons during REM Sleep Is Necessary for Memory Consolidation. *Neuron*, 107(3), 552–565.e10.

Küry, S., Besnard, T., Ebstein, F., Khan, T. N., Gambin, T., Douglas, J., Bacino, C. A., Craigen, W. J., Sanders, S. J., Lehmann, A., Latypova, X., Khan, K., Pacault, M., Sacharow, S., Glaser, K.,

1286 Bieth, E., Perrin-Sabourin, L., Jacquemont, M. L., Cho, M. T., Roeder, E., ... Isidor, B. (2017).
1287 De Novo Disruption of the Proteasome Regulatory Subunit PSMD12 Causes a Syndromic
1288 Neurodevelopmental Disorder. *American journal of human genetics*, 100(2), 352–363.
1289

1290 Lakatos, K., Toth, I., Nemoda, Z., Ney, K., Sasvari-Szekely, M., & Gervai, J. (2000). Dopamine
1291 D4 receptor (DRD4) gene polymorphism is associated with attachment disorganization in
1292 infants. *Molecular psychiatry*, 5(6), 633–637.
1293

1294 Lane, H. (1976) The Wild Boy of Aveyron. Cambridge, Harvard University Press.
1295

1296 Langley, S. A., Miga, K. H., Karpen, G. H., & Langley, C. H. (2019). Haplotypes spanning
1297 centromeric regions reveal persistence of large blocks of archaic DNA. *eLife*, 8, e42989.
1298

1299 Leach, H.M. (2003) Human domestication reconsidered. *Current Anthropology*, 44, 349–368.
1300

1301 Linderholm, A., Spencer, D., Battista, V., Frantz, L., Barnett, R., Fleischer, R. C., James, H. F.,
1302 Duffy, D., Sparks, J. P., Clements, D. R., Andersson, L., Dobney, K., Leonard, J. A., & Larson,
1303 G. (2016). A novel *MC1R* allele for black coat colour reveals the Polynesian ancestry and
1304 hybridization patterns of Hawaiian feral pigs. *Royal Society open science*, 3(9), 160304.
1305

1306 Lindgren, K. A., Folstein, S. E., Tomblin, J. B., & Tager-Flusberg, H. (2009). Language and
1307 reading abilities of children with autism spectrum disorders and specific language impairment and

1308 their first-degree relatives. *Autism research : official journal of the International Society for*
 1309 *Autism Research*, 2(1), 22–38.
 1310
 1311 Lord, K., Feinstein, M., Smith, B., & Coppinger, R. (2013). Variation in reproductive traits of
 1312 members of the genus *Canis* with special attention to the domestic dog (*Canis*
 1313 *familiaris*). *Behavioural processes*, 92, 131–142.
 1314
 1315 Lord, C., Paul, R. (1997) Language and communication in autism. In D. J. Cohen and F. R.
 1316 Volkmar (eds), *Handbook of Autism and Pervasive Developmental Disorders*, 2nd edn. New York,
 1317 Wiley, 195–225.
 1318
 1319 Lourenco-Jaramillo, D. L., Sifuentes-Rincón, A. M., Parra-Bracamonte, G. M., de la Rosa-Reyna,
 1320 X. F., Segura-Cabrera, A., & Arellano-Vera, W. (2012). Genetic diversity of tyrosine hydroxylase
 1321 (TH) and dopamine β -hydroxylase (DBH) genes in cattle breeds. *Genetics and molecular*
 1322 *biology*, 35(2), 435–440.
 1323
 1324 Luijk, M. P., Roisman, G. I., Haltigan, J. D., Tiemeier, H., Booth-Laforce, C., van Ijzendoorn, M.
 1325 H., Belsky, J., Uitterlinden, A. G., Jaddoe, V. W., Hofman, A., Verhulst, F. C., Tharner, A., &
 1326 Bakermans-Kranenburg, M. J. (2011). Dopaminergic, serotonergic, and oxytonergic candidate
 1327 genes associated with infant attachment security and disorganization? In search of main and
 1328 interaction effects. *Journal of child psychology and psychiatry, and allied disciplines*, 52(12),
 1329 1295–1307.
 1330

1331 Luppapornlarp, S., Leelataweewud, P., Putongkam, P., & Ketanont, S. (2010). Periodontal
 1332 status and orthodontic treatment need of autistic children. *World journal of orthodontics*, 11(3),
 1333 256–261.
 1334
 1335 Lyst, M. J., Ekiert, R., Ebert, D. H., Merusi, C., Nowak, J., Selfridge, J., Guy, J., Kastan, N. R.,
 1336 Robinson, N. D., de Lima Alves, F., Rappsilber, J., Greenberg, M. E., & Bird, A. (2013). Rett
 1337 syndrome mutations abolish the interaction of MeCP2 with the NCoR/SMRT co-repressor. *Nature*
 1338 *neuroscience*, 16(7), 898–902.
 1339
 1340 Lyst, M. J., Ekiert, R., Ebert, D. H., Merusi, C., Nowak, J., Selfridge, J., Guy, J., Kastan, N. R.,
 1341 Robinson, N. D., de Lima Alves, F., Rappsilber, J., Greenberg, M. E., & Bird, A. (2013). Rett
 1342 syndrome mutations abolish the interaction of MeCP2 with the NCoR/SMRT co-repressor. *Nature*
 1343 *neuroscience*, 16(7), 898–902.
 1344
 1345 Maekawa M, Ohnishi T, Toyoshima M, Shimamoto-Mitsuyama C, Hamazaki K, Balan S, Wada
 1346 Y, Esaki K, Takagai S, Tsuchiya KJ, Nakamura K, Iwata Y, Nara T, Iwayama Y, Toyota T, Nozaki
 1347 Y, Ohba H, Watanabe A, Hisano Y, Matsuoka S, Tsujii M, Mori N, Maekawa, M., Ohnishi, T.,
 1348 Toyoshima, M., Shimamoto-Mitsuyama, C., Hamazaki, K., Balan, S., Wada, Y., Esaki, K.,
 1349 Takagai, S., Tsuchiya, K. J., Nakamura, K., Iwata, Y., Nara, T., Iwayama, Y., Toyota, T., Nozaki,
 1350 Y., Ohba, H., Watanabe, A., Hisano, Y., Matsuoka, S., ... Yoshikawa, T. (2020). A potential role
 1351 of fatty acid binding protein 4 in the pathophysiology of autism spectrum disorder. *Brain*
 1352 *communications*, 2(2), fcaa145.
 1353

1354 Makita, K., Takiguchi, S., Naruse, H., Shimada, K., Morioka, S., Fujisawa, T. X., Shimoji, K., &
1355 Tomoda, A. (2020). White matter changes in children and adolescents with reactive attachment
1356 disorder: A diffusion tensor imaging study. *Psychiatry research. Neuroimaging*, 303, 111129.
1357

1358 Manoli, D. S., & State, M. W. (2021). Autism Spectrum Disorder Genetics and the Search for
1359 Pathological Mechanisms. *The American journal of psychiatry*, 178(1), 30–38.
1360

1361 Manouilenko, I., Eriksson, J. M., Humble, M. B., & Bejerot, S. (2014). Minor physical anomalies
1362 in adults with autism spectrum disorder and healthy controls. *Autism research and*
1363 *treatment*, 2014, 743482.
1364

1365 Margoliash D. (2002). Evaluating theories of bird song learning: implications for future
1366 directions. *Journal of comparative physiology. A, Neuroethology, sensory, neural, and behavioral*
1367 *physiology*, 188(11-12), 851–866.
1368

1369 Marler, P., & Sherman, V. (1983). Song structure without auditory feedback: emendations of the
1370 auditory template hypothesis. *The Journal of neuroscience : the official journal of the Society for*
1371 *Neuroscience*, 3(3), 517–531.
1372

1373 Márquez, C., Poirier, G. L., Cordero, M. I., Larsen, M. H., Groner, A., Marquis, J., Magistretti, P.
1374 J., Trono, D., & Sandi, C. (2013). Peripuberty stress leads to abnormal aggression, altered
1375 amygdala and orbitofrontal reactivity and increased prefrontal MAOA gene
1376 expression. *Translational psychiatry*, 3(1), e216.

1377

1378 Márquez, S., Pagano, A. S., Delson, E., Lawson, W., & Laitman, J. T. (2014). The nasal complex
 1379 of Neanderthals: an entry portal to their place in human ancestry. *Anatomical record (Hoboken,*
 1380 *N.J. : 2007)*, 297(11), 2121–2137.

1381

1382 Marui, T., Hashimoto, O., Nanba, E., Kato, C., Tochigi, M., Umekage, T., Ishijima, M., Kohda,
 1383 K., Kato, N., & Sasaki, T. (2004). Association between the neurofibromatosis-1 (NF1) locus and
 1384 autism in the Japanese population. *American journal of medical genetics. Part B, Neuropsychiatric*
 1385 *genetics : the official publication of the International Society of Psychiatric Genetics*, 131B(1),
 1386 43–47.

1387

1388 Maud, C., Ryan, J., McIntosh, J. E., & Olsson, C. A. (2018). The role of oxytocin receptor gene
 1389 (OXTR) DNA methylation (DNAm) in human social and emotional functioning: a systematic
 1390 narrative review. *BMC psychiatry*, 18(1), 154.

1391

1392 McCann, J., Peppé, S., Gibbon, F. E., O'Hare, A., & Rutherford, M. (2007). Prosody and its
 1393 relationship to language in school-aged children with high-functioning autism. *International*
 1394 *journal of language & communication disorders*, 42(6), 682–702.

1395

1396 McNeil, M., Polloway, E., Smith, D. (1984) Feral and isolated children: historical review and
 1397 analysis. *Education and training of the mentally retarded*, 19, 70-79.

1398

1399 Miellet, S., Caldara, R., Gillberg, C., Raju, M., & Minnis, H. (2014). Disinhibited reactive
1400 attachment disorder symptoms impair social judgements from faces. *Psychiatry research*, 215(3),
1401 747–752.

1402

1403 Mikita, N., Hollocks, M. J., Papadopoulos, A. S., Aslani, A., Harrison, S., Leibenluft, E., Simonoff,
1404 E., & Stringaris, A. (2015). Irritability in boys with autism spectrum disorders: an investigation of
1405 physiological reactivity. *Journal of child psychology and psychiatry, and allied*
1406 *disciplines*, 56(10), 1118–1126.

1407

1408 Minnis, H., Reekie, J., Young, D., O'Connor, T., Ronald, A., Gray, A., & Plomin, R. (2007).
1409 Genetic, environmental and gender influences on attachment disorder behaviours. *The British*
1410 *journal of psychiatry : the journal of mental science*, 190, 490–495.

1411

1412 Moles, A., Kieffer, B. L., & D'Amato, F. R. (2004). Deficit in attachment behavior in mice lacking
1413 the mu-opioid receptor gene. *Science (New York, N.Y.)*, 304(5679), 1983–1986.

1414

1415 Mosconi, M. W., Cody-Hazlett, H., Poe, M. D., Gerig, G., Gimpel-Smith, R., & Piven, J. (2009).
1416 Longitudinal study of amygdala volume and joint attention in 2- to 4-year-old children with
1417 autism. *Archives of general psychiatry*, 66(5), 509–516.

1418

1419 Murphy, C. M., Deeley, Q., Daly, E. M., Ecker, C., O'Brien, F. M., Hallahan, B., Loth, E., Toal,
1420 F., Reed, S., Hales, S., Robertson, D. M., Craig, M. C., Mullins, D., Barker, G. J., Lavender, T.,
1421 Johnston, P., Murphy, K. C., & Murphy, D. G. (2012). Anatomy and aging of the amygdala and

1422 hippocampus in autism spectrum disorder: an in vivo magnetic resonance imaging study of
 1423 Asperger syndrome. *Autism research : official journal of the International Society for Autism*
 1424 *Research*, 5(1), 3–12.

1425

1426 Naber, F. B., Bakermans-Kranenburg, M. J., van Ijzendoorn, M. H., Swinkels, S. H., Buitelaar, J.
 1427 K., Dietz, C., van Daalen, E., & van Engeland, H. (2008). Play behavior and attachment in toddlers
 1428 with autism. *Journal of autism and developmental disorders*, 38(5), 857–866.

1429

1430 Nagasawa, M., Mitsui, S., En, S., Ohtani, N., Ohta, M., Sakuma, Y., Onaka, T., Mogi, K., &
 1431 Kikusui, T. (2015). Social evolution. Oxytocin-gaze positive loop and the coevolution of human-
 1432 dog bonds. *Science (New York, N.Y.)*, 348(6232), 333–336.

1433

1434 Naumann, N., Lübke, J., Shomali, W., Reiter, L., Horny, H. P., Jawhar, M., Dangelo, V., Fabarius,
 1435 A., Metzgeroth, G., Kreil, S., Sotlar, K., Oni, C., Harrison, C., Hofmann, W. K., Cross, N., Valent,
 1436 P., Radia, D., Gotlib, J., Reiter, A., & Schwaab, J. (2021). Clinical and histopathological features
 1437 of myeloid neoplasms with concurrent Janus kinase 2 (JAK2) V617F and KIT proto-oncogene,
 1438 receptor tyrosine kinase (KIT) D816V mutations. *British journal of haematology*,
 1439 10.1111/bjh.17567. Advance online publication.

1440

1441 Nicastro N. (2004). Perceptual and acoustic evidence for species-level differences in meow
 1442 vocalizations by domestic cats (*Felis catus*) and African wild cats (*Felis silvestris lybica*). *Journal*
 1443 *of comparative psychology (Washington, D.C. : 1983)*, 118(3), 287–296.

1444

1445 Niego, A., & Benítez-Burraco, A. (2019). Williams Syndrome, Human Self-Domestication, and
 1446 Language Evolution. *Frontiers in psychology*, 10, 521.

1447

1448 Niego, A., Benitez-Burraco, A. (2021). Are feralization and domestication truly mirror processes?
 1449 *Ethology, Ecology, and Evolution*, 2021

1450

1451 Nikolsky, A., & Benítez-Burraco, A. (2022, January 9). Human aggression and music evolution:
 1452 a model. <https://doi.org/10.31234/osf.io/a8up7>

1453

1454 Nikulina E. M. (1991). Neural control of predatory aggression in wild and domesticated
 1455 animals. *Neuroscience and biobehavioral reviews*, 15(4), 545–547.

1456

1457 Nillni, E. A., & Sevarino, K. A. (1999). The biology of pro-thyrotropin-releasing hormone-derived
 1458 peptides. *Endocrine reviews*, 20(5), 599–648.

1459

1460

1461 Okanoya K. (2017). Sexual communication and domestication may give rise to the signal
 1462 complexity necessary for the emergence of language: An indication from songbird
 1463 studies. *Psychonomic bulletin & review*, 24(1), 106–110.

1464

1465 Oliva, J. L., Rault, J. L., Appleton, B., & Lill, A. (2015). Oxytocin enhances the appropriate use
 1466 of human social cues by the domestic dog (*Canis familiaris*) in an object choice task. *Animal*
 1467 *cognition*, 18(3), 767–775.

1468

1469 O’Roak, B. J., Deriziotis, P., Lee, C., Vives, L., Schwartz, J. J., Girirajan, S., Karakoc, E.,
 1470 Mackenzie, A. P., Ng, S. B., Baker, C., Rieder, M. J., Nickerson, D. A., Bernier, R., Fisher, S. E.,
 1471 Shendure, J., & Eichler, E. E. (2011). Exome sequencing in sporadic autism spectrum disorders
 1472 identifies severe de novo mutations. *Nature genetics*, 43(6), 585–589.

1473

1474 O’Roak, B. J., Vives, L., Girirajan, S., Karakoc, E., Krumm, N., Coe, B. P., Levy, R., Ko, A., Lee,
 1475 C., Smith, J. D., Turner, E. H., Stanaway, I. B., Vernot, B., Malig, M., Baker, C., Reilly, B., Akey,
 1476 J. M., Borenstein, E., Rieder, M. J., Nickerson, D. A., ... Eichler, E. E. (2012). Sporadic autism
 1477 exomes reveal a highly interconnected protein network of de novo mutations. *Nature*, 485(7397),
 1478 246–250.

1479

1480 Owens, J. L., Olsen, M., Fontaine, A., Kloth, C., Kershbaum, A., & Waller, S. (2017). Visual
 1481 classification of feral cat *Felis silvestris catus* vocalizations. *Current zoology*, 63(3), 331–339.

1482

1483 Oztan, O., Garner, J. P., Partap, S., Sherr, E. H., Hardan, A. Y., Farmer, C., Thurm, A., Swedo, S.
 1484 E., & Parker, K. J. (2018). Cerebrospinal fluid vasopressin and symptom severity in children with
 1485 autism. *Annals of neurology*, 84(4), 611–615.

1486

1487 Pan, Z., Li, S., Liu, Q., Wang, Z., Zhou, Z., Di, R., Miao, B., Hu, W., Wang, X., Hu, X., Xu, Z.,
 1488 Wei, D., He, X., Yuan, L., Guo, X., Liang, B., Wang, R., Li, X., Cao, X., Dong, X., ... Li, Y.
 1489 (2018). Whole-genome sequences of 89 Chinese sheep suggest role of RXFP2 in the development
 1490 of unique horn phenotype as response to semi-feralization. *GigaScience*, 7(4), giy019.

1491

1492 Park, H. R., Lee, J. M., Moon, H. E., Lee, D. S., Kim, B. N., Kim, J., Kim, D. G., & Paek, S. H.

1493 (2016). A Short Review on the Current Understanding of Autism Spectrum

1494 Disorders. *Experimental neurobiology*, 25(1), 1–13.

1495

1496 Parker, K. J., Garner, J. P., Oztan, O., Tarara, E. R., Li, J., Sclafani, V., Del Rosso, L. A., Chun,

1497 K., Berquist, S. W., Chez, M. G., Partap, S., Hardan, A. Y., Sherr, E. H., & Capitanio, J. P. (2018).

1498 Arginine vasopressin in cerebrospinal fluid is a marker of sociality in nonhuman primates. *Science*

1499 *translational medicine*, 10(439), eaam9100.

1500

1501 Pattabiraman, K., Muchnik, S.K., & Sestan, N. (2020) The evolution of the human brain and

1502 disease susceptibility. *Current Opinion in Genetics and Development*, 65, 91-97.

1503

1504 Paul, R., Bianchi, N., Augustyn, A., Klin, A., & Volkmar, F. (2008). Production of Syllable Stress

1505 in Speakers with Autism Spectrum Disorders. *Research in autism spectrum disorders*, 2(1), 110–

1506 124.

1507

1508 Per, Jensen P. (2006) Domestication—From behaviour to genes and back again. *Applied Animal*

1509 *Behaviour Science*, 97, 3-15.

1510

1511 Perovic, A., Janke, V. (2013) Issues in the acquisition of binding and control in high-functioning

1512 children with autism. *UCL Working Papers in Linguistics*, 25, 131-143.

1513

1514 Pfenning, A. R., Hara, E., Whitney, O., Rivas, M. V., Wang, R., Roulhac, P. L., Howard, J. T.,
 1515 Wirthlin, M., Lovell, P. V., Ganapathy, G., Mouncastle, J., Moseley, M. A., Thompson, J. W.,
 1516 Soderblom, E. J., Iriki, A., Kato, M., Gilbert, M. T., Zhang, G., Bakken, T., Bongaarts, A., ...
 1517 Jarvis, E. D. (2014). Convergent transcriptional specializations in the brains of humans and song-
 1518 learning birds. *Science (New York, N.Y.)*, 346(6215), 1256846.
 1519
 1520 Physical Characteristics of Feral Hogs. (2019, August 28) Feral Hogs.
 1521 <https://feralhogs.extension.org/physical-characteristics-of-feral-hogs/>
 1522
 1523 Pickering, C., Gustafsson, L., Cebere, A., Nylander, I., & Liljequist, S. (2006). Repeated maternal
 1524 separation of male Wistar rats alters glutamate receptor expression in the hippocampus but not the
 1525 prefrontal cortex. *Brain research*, 1099(1), 101–108.
 1526
 1527 Pisor, A.C., & Surbeck, M. (2019). The evolution of intergroup tolerance in nonhuman primates
 1528 and humans. *Evolutionary Anthropology: Issues, News, and Reviews*, 28, 210–23.
 1529
 1530 Plavcan J. M. (2012). Sexual size dimorphism, canine dimorphism, and male-male competition in
 1531 primates: where do humans fit in?. *Human nature (Hawthorne, N.Y.)*, 23(1), 45–67.
 1532
 1533 Prasad, A., Merico, D., Thiruvahindrapuram, B., Wei, J., Lionel, A. C., Sato, D., Rickaby, J., Lu,
 1534 C., Szatmari, P., Roberts, W., Fernandez, B. A., Marshall, C. R., Hatchwell, E., Eis, P. S., &
 1535 Scherer, S. W. (2012). A discovery resource of rare copy number variations in individuals with
 1536 autism spectrum disorder. *G3 (Bethesda, Md.)*, 2(12), 1665–1685.

1537

1538 Prather, J. F., Peters, S., Nowicki, S., & Mooney, R. (2008). Precise auditory-vocal mirroring in
 1539 neurons for learned vocal communication. *Nature*, *451*(7176), 305–310.

1540

1541 Progovac, L., & Benítez-Burraco, A. (2019). From Physical Aggression to Verbal Behavior:
 1542 Language Evolution and Self-Domestication Feedback Loop. *Frontiers in psychology*, *10*, 2807.

1543

1544 Puglia, M. H., Connelly, J. J., & Morris, J. P. (2018). Epigenetic regulation of the oxytocin receptor
 1545 is associated with neural response during selective social attention. *Translational psychiatry*, *8*(1),
 1546 116.

1547

1548 Raaska, H., Lapinleimu, H., Sinkkonen, J., Salmivalli, C., Matomäki, J., Mäkipää, S., & Elovainio,
 1549 M. (2012). Experiences of school bullying among internationally adopted children: results from
 1550 the Finnish Adoption (FINADO) Study. *Child psychiatry and human development*, *43*(4), 592–
 1551 611.

1552

1553 Range, F., & Virányi, Z. (2013). Social learning from humans or conspecifics: differences and
 1554 similarities between wolves and dogs. *Frontiers in psychology*, *4*, 868.

1555

1556 Reif, A., Rösler, M., Freitag, C. M., Schneider, M., Eujen, A., Kissling, C., Wenzler, D., Jacob, C.
 1557 P., Retz-Junginger, P., Thome, J., Lesch, K. P., & Retz, W. (2007). Nature and nurture predispose
 1558 to violent behavior: serotonergic genes and adverse childhood

1559 environment. *Neuropsychopharmacology : official publication of the American College of*
 1560 *Neuropsychopharmacology*, 32(11), 2375–2383.
 1561
 1562 Reinhardt, V. P., Iosif, A. M., Libero, L., Heath, B., Rogers, S. J., Ferrer, E., Nordahl, C., Ghetti,
 1563 S., Amaral, D., & Solomon, M. (2020). Understanding Hippocampal Development in Young
 1564 Children With Autism Spectrum Disorder. *Journal of the American Academy of Child and*
 1565 *Adolescent Psychiatry*, 59(9), 1069–1079.
 1566
 1567 Ricks, D.M., Wing, L. (1976) Language, communication and use of symbols. In: Wing L, editor.
 1568 Early childhood autism. Oxford, Pergamon, 93–134.
 1569
 1570 Rodriguez-Fontenla, C., & Carracedo, A. (2021). UTMOST, a single and cross-tissue TWAS
 1571 (Transcriptome Wide Association Study), reveals new ASD (Autism Spectrum Disorder)
 1572 associated genes. *Translational psychiatry*, 11(1), 256.
 1573
 1574 Röhrs, M., & Ebinger, P. (1999). Verwildert ist nicht gleich wild: Die Hirngewichte verwilderter
 1575 Haussäugetiere [Wild is not really wild: brain weight of wild domestic mammals]. *Berliner und*
 1576 *Munchener tierärztliche Wochenschrift*, 112(6-7), 234–238.
 1577
 1578 Rolls E. T. (2015). Limbic systems for emotion and for memory, but no single limbic
 1579 system. *Cortex; a journal devoted to the study of the nervous system and behavior*, 62, 119–157.
 1580

1581 Rose, K.M., Wodzicka-Tomaszewska, M., Cumming, R.B. (1985) Agonistic behaviour, responses
 1582 to a novel object and some aspects of maintenance behaviour in feral-strain and domestic chickens.
 1583 *Applied Animal Behavioral Science*, 13, 283-294.

1584

1585 Rothschild, G., Sottas, C. M., Kissel, H., Agosti, V., Manova, K., Hardy, M. P., & Besmer, P.
 1586 (2003). A role for kit receptor signaling in Leydig cell steroidogenesis. *Biology of*
 1587 *reproduction*, 69(3), 925–932.

1588

1589 Roulin A. (2004). The evolution, maintenance and adaptive function of genetic colour
 1590 polymorphism in birds. *Biological reviews of the Cambridge Philosophical Society*, 79(4), 815–
 1591 848.

1592

1593 Russo, N., Larson, C., & Kraus, N. (2008). Audio-vocal system regulation in children with autism
 1594 spectrum disorders. *Experimental brain research*, 188(1), 111–124.

1595

1596 Rutter, M., Kreppner, J., & Sonuga-Barke, E. (2009). Emanuel Miller Lecture: Attachment
 1597 insecurity, disinhibited attachment, and attachment disorders: where do research findings leave the
 1598 concepts?. *Journal of child psychology and psychiatry, and allied disciplines*, 50(5), 529–543.

1599

1600 Rymer, R. (1994) *Genie: A Scientific Tragedy* (2 ed.). New York, NY: Harper Perennial.

1601

1602 Sabbagh, A., Pasmant, E., Imbard, A., Luscan, A., Soares, M., Blanché, H., Laurendeau, I., Ferkal,
 1603 S., Vidaud, M., Pinson, S., Bellanné-Chantelot, C., Vidaud, D., Parfait, B., & Wolkenstein, P.

1604 (2013). NF1 molecular characterization and neurofibromatosis type I genotype-phenotype
1605 correlation: the French experience. *Human mutation*, 34(11), 1510–1518.

1606

1607 Sacco, R., Gabriele, S., & Persico, A. M. (2015). Head circumference and brain size in autism
1608 spectrum disorder: A systematic review and meta-analysis. *Psychiatry research*, 234(2), 239–251.

1609

1610 Sadiq, F. A., Slator, L., Skuse, D., Law, J., Gillberg, C., & Minnis, H. (2012). Social use of
1611 language in children with reactive attachment disorder and autism spectrum disorders. *European*
1612 *child & adolescent psychiatry*, 21(5), 267–276.

1613

1614 Saito, A., Watanabe, T. K., Shimada, Y., Fujiwara, T., Slaughter, C. A., DeMartino, G. N.,
1615 Tanahashi, N., & Tanaka, K. (1997). cDNA cloning and functional analysis of p44.5 and p55, two
1616 regulatory subunits of the 26S proteasome. *Gene*, 203(2), 241–250.

1617

1618 Saladin, K.S., Gan, C.A., Cushman, H.N. (2015) *Anatomy & Physiology: the Unity of Form and*
1619 *Function*, 8th edn. New York, NY, McGraw-Hill Education.

1620

1621 Sánchez-Villagra, M. R., Geiger, M., & Schneider, R. A. (2016). The taming of the neural crest: a
1622 developmental perspective on the origins of morphological covariation in domesticated
1623 mammals. *Royal Society open science*, 3(6), 160107.

1624

1625 Sanders, S. J., He, X., Willsey, A. J., Ercan-Sencicek, A. G., Samocha, K. E., Cicek, A. E., Murtha,
1626 M. T., Bal, V. H., Bishop, S. L., Dong, S., Goldberg, A. P., Jinlu, C., Keaney, J. F., 3rd, Klei, L.,

1627 Mandell, J. D., Moreno-De-Luca, D., Poultney, C. S., Robinson, E. B., Smith, L., Solli-Nowlan,
 1628 T., ... State, M. W. (2015). Insights into Autism Spectrum Disorder Genomic Architecture and
 1629 Biology from 71 Risk Loci. *Neuron*, 87(6), 1215–1233.
 1630
 1631 Sanders, S. J., Murtha, M. T., Gupta, A. R., Murdoch, J. D., Raubeson, M. J., Willsey, A. J., Ercan-
 1632 Sencicek, A. G., DiLullo, N. M., Parikshak, N. N., Stein, J. L., Walker, M. F., Ober, G. T., Teran,
 1633 N. A., Song, Y., El-Fishawy, P., Murtha, R. C., Choi, M., Overton, J. D., Bjornson, R. D., Carriero,
 1634 N. J., ... State, M. W. (2012). De novo mutations revealed by whole-exome sequencing are
 1635 strongly associated with autism. *Nature*, 485(7397), 237–241.
 1636
 1637 Satterstrom, F. K., Kosmicki, J. A., Wang, J., Breen, M. S., De Rubeis, S., An, J. Y., Peng, M.,
 1638 Collins, R., Grove, J., Klei, L., Stevens, C., Reichert, J., Mulhern, M. S., Artomov, M., Gerges, S.,
 1639 Sheppard, B., Xu, X., Bhaduri, A., Norman, U., Brand, H., ... Buxbaum, J. D. (2020). Large-Scale
 1640 Exome Sequencing Study Implicates Both Developmental and Functional Changes in the
 1641 Neurobiology of Autism. *Cell*, 180(3), 568–584.e23.
 1642
 1643 Schaschl, H., Huber, S., Schaefer, K., Windhager, S., Wallner, B., & Fieder, M. (2015). Signatures
 1644 of positive selection in the cis-regulatory sequences of the human oxytocin receptor (OXTR) and
 1645 arginine vasopressin receptor 1a (AVPR1A) genes. *BMC evolutionary biology*, 15, 85.
 1646
 1647 Schoen, E., Paul, R., & Chawarska, K. (2011). Phonology and vocal behavior in toddlers with
 1648 autism spectrum disorders. *Autism research : official journal of the International Society for*
 1649 *Autism Research*, 4(3), 177–188.

1650

1651 Schultz, R. T., Grelotti, D. J., Klin, A., Kleinman, J., Van der Gaag, C., Marois, R., & Skudlarski,
 1652 P. (2003). The role of the fusiform face area in social cognition: implications for the pathobiology
 1653 of autism. *Philosophical transactions of the Royal Society of London. Series B, Biological*
 1654 *sciences*, 358(1430), 415–427.

1655

1656 Schumann, C. M., Hamstra, J., Goodlin-Jones, B. L., Lotspeich, L. J., Kwon, H., Buonocore, M.
 1657 H., Lammers, C. R., Reiss, A. L., & Amaral, D. G. (2004). The amygdala is enlarged in children
 1658 but not adolescents with autism; the hippocampus is enlarged at all ages. *The Journal of*
 1659 *neuroscience : the official journal of the Society for Neuroscience*, 24(28), 6392–6401.

1660

1661 Schuurs-Hoeijmakers, J. H., Landsverk, M. L., Foulds, N., Kukolich, M. K., Gavrilova, R. H.,
 1662 Greville-Heygate, S., Hanson-Kahn, A., Bernstein, J. A., Glass, J., Chitayat, D., Burrow, T. A.,
 1663 Husami, A., Collins, K., Wusik, K., van der Aa, N., Kooy, F., Brown, K. T., Gadzicki, D., Kini,
 1664 U., Alvarez, S., ... DDD study (2016). Clinical delineation of the PACS1-related syndrome--
 1665 Report on 19 patients. *American journal of medical genetics. Part A*, 170(3), 670–675.

1666

1667 Schweizer, A. V., Lebrun, R., Wilson, L., Costeur, L., Schmelzle, T., & Sánchez-Villagra, M. R.
 1668 (2017). Size Variation under Domestication: Conservatism in the inner ear shape of wolves, dogs
 1669 and dingoes. *Scientific reports*, 7(1), 13330.

1670

1671 Sharma, N. (2009) Rett Syndrome. *Encyclopedia of Neuroscience*.

1672

1673 Shea, B. (1989) Heterochrony in human evolution: the case for neoteny reconsidered. American
1674 Journal of Physical Anthropology, 32, 69–102.

1675

1676 Shilton, D., Breski, M., Dor, D., & Jablonka, E. (2020). Human Social Evolution: Self-
1677 Domestication or Self-Control?. *Frontiers in psychology*, 11, 134.

1678

1679 Shipman P. (2021). What the dingo says about dog domestication. *Anatomical record (Hoboken,*
1680 *N.J. : 2007)*, 304(1), 19–30.

1681

1682 Sikora, M., Seguin-Orlando, A., Sousa, V. C., Albrechtsen, A., Korneliussen, T., Ko, A.,
1683 Rasmussen, S., Dupanloup, I., Nigst, P. R., Bosch, M. D., Renaud, G., Allentoft, M. E., Margaryan,
1684 A., Vasilyev, S. V., Veselovskaya, E. V., Borutskaya, S. B., Deviese, T., Comeskey, D., Higham,
1685 T., Manica, A., ... Willerslev, E. (2017). Ancient genomes show social and reproductive behavior
1686 of early Upper Paleolithic foragers. *Science (New York, N.Y.)*, 358(6363), 659–662.

1687

1688 Spikins, P., French, J. C., John-Wood, S., & Dytham, C. (2021). Theoretical and methodological
1689 approaches to ecological changes, social behaviour and human intergroup tolerance 300,000 to
1690 30,000 BP. *The Journal of Archaeological Method and Theory*, 28, 53–75.

1691

1692 Smith, B. P., Cairns, K. M., Adams, J. W., Newsome, T. M., Fillios, M., Déaux, E. C., Parr, W.,
1693 Letnic, M., VAN Eeden, L. M., Appleby, R. G., Bradshaw, C., Savolainen, P., Ritchie, E. G.,
1694 Nimmo, D. G., Archer-Lean, C., Greenville, A. C., Dickman, C. R., Watson, L., Moseby, K. E.,

1695 Doherty, T. S., ... Crowther, M. S. (2019). Taxonomic status of the Australian dingo: the case for
 1696 *Canis dingo* Meyer, 1793. *Zootaxa*, 4564(1), zootaxa.4564.1.6.
 1697
 1698 Smith, B. P., & Litchfield, C. A. (2010). Dingoes (*Canis dingo*) can use human social cues to locate
 1699 hidden food. *Animal cognition*, 13(2), 367–376.
 1700
 1701 Smith, B.P., Lucas, T.A., Norris, R.M., Henneberg, M. (2018) Brain size/body weight in the dingo
 1702 (*Canis dingo*): comparisons with domestic and wild canids. *Australian Journal of Zoology*. 65(5):
 1703 292-301.
 1704
 1705 Smyke, A. T., Dumitrescu, A., & Zeanah, C. H. (2002). Attachment disturbances in young
 1706 children. I: The continuum of caretaking casualty. *Journal of the American Academy of Child and*
 1707 *Adolescent Psychiatry*, 41(8), 972–982.
 1708
 1709 Somel, M., Franz, H., Yan, Z., Lorenc, A., Guo, S., Giger, T., Kelso, J., Nickel, B., Dannemann,
 1710 M., Bahn, S., Webster, M. J., Weickert, C. S., Lachmann, M., Pääbo, S., & Khaitovich, P. (2009).
 1711 Transcriptional neoteny in the human brain. *Proceedings of the National Academy of Sciences of*
 1712 *the United States of America*, 106(14), 5743–5748.
 1713
 1714 Spangler G, Johann M, Ronai Z, Zimmermann P. Genetic and environmental influence on
 1715 attachment disorganization. *J Child Psychol Psychiatry*. 2009 Aug;50(8):952-61.
 1716

1717 Spratt, E. G., Nicholas, J. S., Brady, K. T., Carpenter, L. A., Hatcher, C. R., Meekins, K. A.,
 1718 Furlanetto, R. W., & Charles, J. M. (2012). Enhanced cortisol response to stress in children in
 1719 autism. *Journal of autism and developmental disorders*, 42(1), 75–81.
 1720
 1721 St Pourcain, B., Cents, R. A., Whitehouse, A. J., Haworth, C. M., Davis, O. S., O'Reilly, P. F.,
 1722 Roulstone, S., Wren, Y., Ang, Q. W., Velders, F. P., Evans, D. M., Kemp, J. P., Warrington, N.
 1723 M., Miller, L., Timpson, N. J., Ring, S. M., Verhulst, F. C., Hofman, A., Rivadeneira, F., Meaburn,
 1724 E. L., ... Davey Smith, G. (2014). Common variation near ROBO2 is associated with expressive
 1725 vocabulary in infancy. *Nature communications*, 5, 4831.
 1726
 1727 Steeves, Peter H. (2011) Humans and animals at the divide: the case of feral children. *Between the*
 1728 *species*, 13, 3-7.
 1729
 1730 Stein, C. M., Schick, J. H., Gerry Taylor, H., Shriberg, L. D., Millard, C., Kundtz-Kluge, A., Russo,
 1731 K., Minich, N., Hansen, A., Freebairn, L. A., Elston, R. C., Lewis, B. A., & Iyengar, S. K. (2004).
 1732 Pleiotropic effects of a chromosome 3 locus on speech-sound disorder and reading. *American*
 1733 *journal of human genetics*, 74(2), 283–297.
 1734
 1735 Stendler, C. (1952) Critical Periods in Socialization and Overdependency. *Child Development*, 23,
 1736 3-12.
 1737

1738 Stinehart, M.A., Scott, D.A., Barfield, H.G. (2012). Reactive Attachment Disorder in adopted and
 1739 foster care children: implications for mental health professionals. *The Family Journal*, 20(4), 355-
 1740 360.

1741

1742 Stringer C. (2016). The origin and evolution of Homo sapiens. *Philosophical transactions of the*
 1743 *Royal Society of London. Series B, Biological sciences*, 371(1698), 20150237.

1744

1745 Suda, S., Iwata, K., Shimmura, C., Kamenno, Y., Anitha, A., Thanseem, I., Nakamura, K.,
 1746 Matsuzaki, H., Tsuchiya, K. J., Sugihara, G., Iwata, Y., Suzuki, K., Koizumi, K., Higashida, H.,
 1747 Takei, N., & Mori, N. (2011). Decreased expression of axon-guidance receptors in the anterior
 1748 cingulate cortex in autism. *Molecular autism*, 2(1), 14.

1749

1750 Surian, L., Baron-Cohen, S., & Van der Lely, H. (1996). Are children with autism deaf to gricean
 1751 maxims?. *Cognitive neuropsychiatry*, 1(1), 55–72.

1752

1753 Suzuki, S., Fujisawa, T. X., Sakakibara, N., Fujioka, T., Takiguchi, S., & Tomoda, A. (2020).
 1754 Development of Social Attention and Oxytocin Levels in Maltreated Children. *Scientific*
 1755 *reports*, 10(1), 7407.

1756

1757 Swain, J. E., Leckman, J. F., & Volkmar, F. R. (2005). The wolf boy: reactive attachment disorder
 1758 in an adolescent boy. *Psychiatry (Edgmont (Pa. : Township))*, 2(11), 55–61.

1759

1760 Swaney, W. T., Cabrera-Álvarez, M. J., & Reader, S. M. (2015). Behavioural responses of feral
 1761 and domestic guppies (*Poecilia reticulata*) to predators and their cues. *Behavioural processes*, 118,
 1762 42–46.

1763

1764 Syal, S., & Finlay, B. L. (2011). Thinking outside the cortex: social motivation in the evolution
 1765 and development of language. *Developmental science*, 14(2), 417–430.

1766

1767 Losh, M., & Gordon, P. C. (2014). Quantifying narrative ability in autism spectrum disorder: a
 1768 computational linguistic analysis of narrative coherence. *Journal of autism and developmental*
 1769 *disorders*, 44(12), 3016–3025.

1770

1771 Tager-Flusberg, H. (2006) Defining language phenotypes in autism. *Clinical Neuroscience*
 1772 *Research*, 6, 219–224.

1773

1774 Tager-Flusberg H. (1992). Autistic children's talk about psychological states: deficits in the early
 1775 acquisition of a theory of mind. *Child development*, 63(1), 161–172.

1776

1777 Tager-Flusberg H. (1993) What language reveals about the understanding of minds in children
 1778 with autism. In S. Baron-Cohen, H. Tager-Flusberg and D. J. Cohen (eds), *Understanding other*
 1779 *Minds. Perspectives from Autism*, Oxford, Oxford University Press, 138–57.

1780

1781 Tager-Flusberg, H. 2000. Language and the understanding minds: connections in autism. In S.
1782 Baron-Cohen, H. Tager-Flusberg and D. J. Cohen (eds), *Understanding Other Minds. Perspectives*
1783 *from Developmental Cognitive Science*, 2nd edn. Oxford: Oxford University Press, 124–149.

1784

1785 Takiguchi, S., Fujisawa, T. X., Mizushima, S., Saito, D. N., Okamoto, Y., Shimada, K., Koizumi,
1786 M., Kumazaki, H., Jung, M., Kosaka, H., Hiratani, M., Ohshima, Y., Teicher, M. H., & Tomoda,
1787 A. (2015). Ventral striatum dysfunction in children and adolescents with reactive attachment
1788 disorder: functional MRI study. *BJPsych open*, 1(2), 121–128.

1789

1790 Tallet, C., Linhart, P., Policht, R., Hammerschmidt, K., Šimeček, P., Kratinova, P., & Špinka, M.
1791 (2013). Encoding of situations in the vocal repertoire of piglets (*Sus scrofa*): a comparison of
1792 discrete and graded classifications. *PloS one*, 8(8), e71841.

1793

1794 aylor, J. L., & Corbett, B. A. (2014). A review of rhythm and responsiveness of cortisol in
1795 individuals with autism spectrum disorders. *Psychoneuroendocrinology*, 49, 207–228.

1796

1797 Teicher, M. H., & Samson, J. A. (2016). Annual Research Review: Enduring neurobiological
1798 effects of childhood abuse and neglect. *Journal of child psychology and psychiatry, and allied*
1799 *disciplines*, 57(3), 241–266.

1800

1801 Theofanopoulou, C., Gastaldon, S., O'Rourke, T., Samuels, B. D., Martins, P. T., Delogu, F.,
1802 Alamri, S., & Boeckx, C. (2017). Self-domestication in *Homo sapiens*: Insights from comparative
1803 genomics. *PloS one*, 12(10), e0185306.

1804

1805 Thomas, J., Kirby S. (2018) Self domestication and the evolution of language. *Biological*

1806 *Philosophy*, 33, 9.

1807

1808 Reiersen, A. M., & Todorov, A. A. (2011). Association between DRD4 genotype and Autistic

1809 Symptoms in DSM-IV ADHD. *Journal of the Canadian Academy of Child and Adolescent*

1810 *Psychiatry* 20(1), 15–21.

1811

1812 Tolchin, D., Yeager, J. P., Prasad, P., Dorrani, N., Russi, A. S., Martinez-Agosto, J. A., Haseeb,

1813 A., Angelozzi, M., Santen, G., Ruivenkamp, C., Mercimek-Andrews, S., Depienne, C., Kuechler,

1814 A., Mikat, B., Ludecke, H. J., Bilan, F., Le Guyader, G., Gilbert-Dussardier, B., Keren, B., Heide,

1815 S., ... Le Caignec, C. (2020). De Novo SOX6 Variants Cause a Neurodevelopmental Syndrome

1816 Associated with ADHD, Craniosynostosis, and Osteochondromas. *American journal of human*

1817 *genetics*, 106(6), 830–845.

1818

1819 Tomarken, A. J., Han, G. T., & Corbett, B. A. (2015). Temporal patterns, heterogeneity, and

1820 stability of diurnal cortisol rhythms in children with autism spectrum

1821 disorder. *Psychoneuroendocrinology*, 62, 217–226.

1822

1823 Tomasello, M., Hare, B., Lehmann, H., & Call, J. (2007). Reliance on head versus eyes in the gaze

1824 following of great apes and human infants: the cooperative eye hypothesis. *Journal of human*

1825 *evolution*, 52(3), 314–320.

1826

1827 Toro, R., Konyukh, M., Delorme, R., Leblond, C., Chaste, P., Fauchereau, F., Coleman, M.,
1828 Leboyer, M., Gillberg, C., & Bourgeron, T. (2010). Key role for gene dosage and synaptic
1829 homeostasis in autism spectrum disorders. *Trends in Genetics*, 26(8), 363-372.

1830

1831 Trut, L., Oskina, I., & Kharlamova, A. (2009). Animal evolution during domestication: the
1832 domesticated fox as a model. *BioEssays : news and reviews in molecular, cellular and*
1833 *developmental biology*, 31(3), 349–360.

1834

1835 Tyack P. L. (2020). A taxonomy for vocal learning. *Philosophical transactions of the Royal*
1836 *Society of London. Series B, Biological sciences*, 375(1789), 20180406.

1837

1838 Udell, M., Dorey, N., Wynne, C. (2008) Wolves outperform dogs in following human social cues.
1839 *Animal Behavior*, 76, 1767–1773.

1840

1841 Udell M. A. (2015). When dogs look back: inhibition of independent problem-solving behaviour
1842 in domestic dogs (*Canis lupus familiaris*) compared with wolves (*Canis lupus*). *Biology*
1843 *letters*, 11(9), 20150489.

1844

1845 Veenstra-VanderWeele, J., Muller, C. L., Iwamoto, H., Sauer, J. E., Owens, W. A., Shah, C. R.,
1846 Cohen, J., Mannangatti, P., Jessen, T., Thompson, B. J., Ye, R., Kerr, T. M., Carneiro, A. M.,
1847 Crawley, J. N., Sanders-Bush, E., McMahon, D. G., Ramamoorthy, S., Daws, L. C., Sutcliffe, J.
1848 S., & Blakely, R. D. (2012). Autism gene variant causes hyperserotonemia, serotonin receptor

1849 hypersensitivity, social impairment and repetitive behavior. *Proceedings of the National Academy*
1850 *of Sciences of the United States of America*, 109(14), 5469–5474.

1851

1852 Vernes S. C. (2017). What bats have to say about speech and language. *Psychonomic bulletin &*
1853 *review*, 24(1), 111–117.

1854

1855 Virányi, Z., Gácsi, M., Kubinyi, E., Topál, J., Belényi, B., Ujfalussy, D., & Miklósi, A. (2008).
1856 Comprehension of human pointing gestures in young human-reared wolves (*Canis lupus*) and dogs
1857 (*Canis familiaris*). *Animal cognition*, 11(3), 373–387.

1858

1859 Volkmar, F., Chawarska, K., & Klin, A. (2005). Autism in infancy and early childhood. *Annual*
1860 *review of psychology*, 56, 315–336.

1861

1862 Volman, S. F., & Khanna, H. (1995). Convergence of untutored song in group-reared zebra finches
1863 (*Taeniopygia guttata*). *Journal of comparative psychology (Washington, D.C. : 1983)*, 109(3),
1864 211–221.

1865

1866 von Aster, M., Zachmann, M., Brandeis, D., Wohlrab, G., Richner, M., & Steinhausen, H. C.
1867 (1997). Psychiatric, neuropsychiatric, and neuropsychological symptoms in a case of hypomelanosis
1868 of Ito. *European child & adolescent psychiatry*, 6(4), 227–233.

1869

1870 vonHoldt, B. M., Shuldiner, E., Koch, I. J., Kartzinel, R. Y., Hogan, A., Brubaker, L., Wanser, S.,
1871 Stahler, D., Wynne, C., Ostrander, E. A., Sinsheimer, J. S., & Udell, M. (2017). Structural variants

1872 in genes associated with human Williams-Beuren syndrome underlie stereotypical
1873 hypersociability in domestic dogs. *Science advances*, 3(7), e1700398.

1874

1875 Vyshedskiy, A., Mahapatra, S., Dunn, R. (2017) Linguistically deprived children: meta-analysis
1876 of published research underlines the importance of early syntactic language use for normal brain
1877 development. *Research Ideas and Outcomes*, 3:e20696.

1878

1879 Wada, M., Suzuki, M., Takaki, A., Miyao, M., Spence, C., & Kansaku, K. (2014). Spatio-temporal
1880 processing of tactile stimuli in autistic children. *Scientific reports*, 4, 5985.

1881

1882 Wallace, M., Cleary, J., Buder, E., Oller, D., Sheinkopf, S., Mundy, P. (2008) An acoustic
1883 inspection of vocalizations in young children with ASD. Poster presented at the International
1884 Meeting for Autism Research; London.

1885

1886 Wang, A. T., Lee, S. S., Sigman, M., & Dapretto, M. (2006). Neural basis of irony comprehension
1887 in children with autism: the role of prosody and context. *Brain : a journal of neurology*, 129(Pt 4),
1888 932–943.

1889

1890 Wang, A. T., Lee, S. S., Sigman, M., & Dapretto, M. (2007). Reading affect in the face and voice:
1891 neural correlates of interpreting communicative intent in children and adolescents with autism
1892 spectrum disorders. *Archives of general psychiatry*, 64(6), 698–708.

1893

1894 Wang, T., Guo, H., Xiong, B., Stessman, H. A., Wu, H., Coe, B. P., Turner, T. N., Liu, Y., Zhao,
1895 W., Hoekzema, K., Vives, L., Xia, L., Tang, M., Ou, J., Chen, B., Shen, Y., Xun, G., Long, M.,
1896 Lin, J., Kronenberg, Z. N., ... Eichler, E. E. (2016). De novo genic mutations among a Chinese
1897 autism spectrum disorder cohort. *Nature communications*, 7, 13316.

1898

1899 Wang, Z., Li, J., Li, K., & Xu, J. (2018). SOX6 is downregulated in osteosarcoma and suppresses
1900 the migration, invasion and epithelial-mesenchymal transition via TWIST1 regulation. *Molecular*
1901 *medicine reports*, 17(5), 6803–6811.

1902

1903 Watkins, M., Szarko, J., Brown, A. (2013) Examiner familiarity effects for children with autism
1904 spectrum disorders. *Journal of Applied School of Psychology*, 29, 37-51.

1905

1906 Wazana, A., Moss, E., Jolicoeur-Martineau, A., Graffi, J., Tsabari, G., Lecompte, V., Pascuzzo,
1907 K., Babineau, V., Gordon-Green, C., Mileva, V., Atkinson, L., Minde, K., Bouvette-Turcot, A. A.,
1908 Sassi, R., St-André, M., Carrey, N., Matthews, S., Sokolowski, M., Lydon, J., Gaudreau, H., ...
1909 Meaney, M. J. (2015). The interplay of birth weight, dopamine receptor D4 gene (DRD4), and
1910 early maternal care in the prediction of disorganized attachment at 36 months of age. *Development*
1911 *and psychopathology*, 27(4 Pt 1), 1145–1161.

1912

1913 Wetherby, A. M., Watt, N., Morgan, L., & Shumway, S. (2007). Social communication profiles of
1914 children with autism spectrum disorders late in the second year of life. *Journal of autism and*
1915 *developmental disorders*, 37(5), 960–975.

1916

1917 Wilkins, A. S., Wrangham, R. W., & Fitch, W. T. (2014). The "domestication syndrome" in
 1918 mammals: a unified explanation based on neural crest cell behavior and genetics. *Genetics*, 197(3),
 1919 795–808.
 1920
 1921 Winslow, J. T., & Insel, T. R. (2002). The social deficits of the oxytocin knockout
 1922 mouse. *Neuropeptides*, 36(2-3), 221–229.
 1923
 1924 Wolk, L., Edwards, M., Brennan, C. (2016) Phonological difficulties in children with autism: An
 1925 overview. *Speech, Language and Hearing*, 19, 160204020132009.
 1926
 1927 Wolk, L., & Edwards, M. L. (1993). The emerging phonological system of an autistic
 1928 child. *Journal of communication disorders*, 26(3), 161–177.
 1929
 1930 Yeon, S. C., Kim, Y. K., Park, S. J., Lee, S. S., Lee, S. Y., Suh, E. H., Houpt, K. A., Chang, H. H.,
 1931 Lee, H. C., Yang, B. G., & Lee, H. J. (2011). Differences between vocalization evoked by social
 1932 stimuli in feral cats and house cats. *Behavioural processes*, 87(2), 183–189.
 1933
 1934 Yoo, H. J., Lee, S. K., Park, M., Cho, I. H., Hyun, S. H., Lee, J. C., Yang, S. Y., & Kim, S. A.
 1935 (2009). Family- and population-based association studies of monoamine oxidase A and autism
 1936 spectrum disorders in Korean. *Neuroscience research*, 63(3), 172–176.
 1937
 1938 Yvan, L. (1980) Victor of Aveyron: A reappraisal in light of more recent cases of feral speech.
 1939 *Language Science*, 2, 32-43.

1940

1941 Zangl, R., Mills, D.L. (2007) Increased Brain Activity to Infant-Directed Speech in 6- and 13-

1942 Month-Old Infants. *Infancy*, 11, 31-62.

1943

1944 Zhang, S. J., Wang, G. D., Ma, P., Zhang, L. L., Yin, T. T., Liu, Y. H., Otecko, N. O., Wang, M.,

1945 Ma, Y. P., Wang, L., Mao, B., Savolainen, P., & Zhang, Y. P. (2020). Genomic regions under

1946 selection in the feralization of the dingoes. *Nature communications*, 11(1), 671.

1947

1948 Zhang, X., Yang, H., Corydon, M. J., Zhang, X., Pedersen, S., Korenberg, J. R., Chen, X. N.,

1949 Laporte, J., Gregersen, N., Niebuhr, E., Liu, G., & Bolund, L. (1999). Localization of a human

1950 nucleoporin 155 gene (NUP155) to the 5p13 region and cloning of its cDNA. *Genomics*, 57(1),

1951 144–151.

1952

1953 Zhou, W., He, Y., Rehman, A. U., Kong, Y., Hong, S., Ding, G., Yalamanchili, H. K., Wan, Y.

1954 W., Paul, B., Wang, C., Gong, Y., Zhou, W., Liu, H., Dean, J., Scalais, E., O'Driscoll, M., Morton,

1955 J., DDD study, Hou, X., Wu, Q., ... Sun, Z. (2019). Loss of function of NCOR1 and NCOR2

1956 impairs memory through a novel GABAergic hypothalamus-CA3 projection. *Nature*

1957 *neuroscience*, 22(2), 205–217.

1958

1959 Zollikofer, C. P., & Ponce de León, M. S. (2010). The evolution of hominin ontogenies. *Seminars*

1960 *in cell & developmental biology*, 21(4), 441–452.

1961

1962 Zufferey, S. (2015) *Acquiring pragmatics. Social and cognitive perspectives*. Routledge: London
1963 and New York.
1964