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Revisiting the Case for 'Feral' Humans Under the Light of the Human Self-Domestication Hypothesis: Focusing on Language

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Supplementary Materials: Data [see Index of Supplementary Materials]

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 Fukase et al., 2015; Herrmann et al., 2011; Langley et al., 2019; Leach, 2003; Márquez et al., 2014; Plavcan, 2012; Shea, 1989; Stringer, 2016; Thomas & Kirby, 2018; Zollikofer & Ponce de León, 2010, 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., 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; Nikolsky & Benítez-Burraco, 2022; Pisor & Surbeck, 2019; Spikins et al., 2021). 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 the 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, 2017) paved the way towards claims that HSD might have played a role in the emergence of modern languages (see Thomas & Kirby, 2018 and Benítez-Burraco & 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 & 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; Benítez-Burraco & Progovac, 2020; Progovac & Benítez-Burraco, 2019, 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, 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 how, 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 (Cieri et al., 2014; Leach, 2003). The presentation of HSD features in present-day human populations can also be quite variable (Gleeson & Kushnick, 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 to compare 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 & 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 us 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 than 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 mostly externally caused, 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, set 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' (Butler, 2003; Kapoor, 1973). 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 1950s. 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 (Bourguignon et al., 2012; Eigsti et al., 2007; Tager-Flusberg, 2006; Tager-Flusberg et al. 2005), 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 & 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 (Bhandari et al., 2020; Bölte et al., 2019; Manoli & State, 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 (Corval et al., 2020; Spangler et al., 2009). 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. To do 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 Sánchez-Villagra et al., 2016; Wilkins et al., 2014), but also on publications describ-

ing the HSD hypothesis (Cieri et al., 2014; Hare, 2017; Thomas & 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.

Table 1

Summary Table of the Presentation of Features of Domestication/HSD in the Four Conditions Under Scrutiny in the Paper

Feature 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

Feature of domestication/HSD	Feralized animals	ASD	‘Feral’ children	RAD
Enhanced playing behavior	?	X	?	?
Reduced aggressive behavior	X	X	X	X
Neuroendocrine system				
Decreased levels of glucocorticoids	?	X	?	?
Decreased levels of basal adrenocorticotropic 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	?	?

Note. X = absent feature; ✓ = attested feature; ? = not enough data available.

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Table 2

A Detailed Characterization 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 (Birks & Kitchener, 1999; Kruska, 2005; Kruska & Röhrs, 1974; Röhrs & Ebinger, 1999); 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)	*Not enough data available*	*Not enough data available*
Relative decrease of specific brain areas (particularly, the hippocampus, the thalamus, and the striatum)	Larger hippocampus found in feral pigs (Kruska & Röhrs, 1974)	Larger relative hippocampal volumes in children; atypical coupling between hippocampal volumes and brain size (Reinhardt et al. 2020)	*Not enough data available*	Decreased size of hippocampus (Corbin, 2007; Teicher, 2002); 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, 2005, 2014; Kruska & Röhrs 1974); no changes in feral cats, dogs, pigs, goats, and donkeys (Röhrs & Ebinger, 1999; Smith et al., 2018)	Larger amygdala than TD peers (Mosconi et al., 2009; Murphy et al., 2012); atypical development during growth (Courchesne et al., 2011; Schumann et al., 2004)	*Not enough data available*	Decreased size of the amygdala (Corbin, 2007; Teicher, 2002)
Ear size and shape				
Reduced ear size/changes in ear shape	Dingoes exhibit wolf-like prominent ears, contrary to most	Abnormal ear shape (Manouilenko et al., 2014)	*Not enough data available*	*Not enough data available*

Features of domestication/HSD	Feralized animals	ASD	'Feral' children	RAD
	dog breeds (Schweizer et al., 2017; Smith et al., 2019)			
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 (Ballard & Wilson, 2019; Kruska & Röhrs,1974; Smith et al., 2018)	Flatter nose bridge documented in patients with ASD (Aldridge et al., 2011)	*Not enough data available*	*Not enough data available*
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 & Litchfield, 2010)	Number of tooth irregularities documented in ASD, including missing teeth and diastemas (Luppanapornlarp et al., 2010)	*Not enough data available*	*Not enough data available*
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 (Cuyvers et al., 2020; Hinshaw-Fuselier et al., 1999)
Enhanced attentiveness and sensitivity to eye or facial movements or gestures	Eye contact is avoided (Johnston et al., 2017; Park et al., 2016)	Eye contact is avoided (Johnston et al., 2017; Park et al., 2016)	Eye contact is avoided (Butler, 2003)	Eye contact is avoided (Ellis et al., 2020; Miellet et al., 2014)
Increased sociability	Avoidance of contact with humans (Johnston et al., 2017; Owens et al., 2017; Rose et al., 1985)	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);

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Features of domestication/HSD	Feralized animals	ASD	'Feral' children	RAD
				minimal response to social and emotional stimuli (Ellis et al., 2020; Miellet et al., 2014)
Enhanced playing behavior	*Not enough data available*	Children with ASD lack 'pretend play' behavior (Bettelheim, 1959; Cuyvers et al., 2020; Ellis et al., 2020; Naber et al., 2008)	*Not enough data available*	*Not enough data available*
Reduced aggressive behavior	Increased levels of aggression (Johnston et al., 2017; Owens et al., 2017; Rose et al., 1985)	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	*Not enough data available*	Higher levels of glucocorticoids (Hamza et al., 2010; Hollocks et al., 2014; Spratt et al., 2012)	*Not enough data available*	*Not enough data available*
Decreased levels of basal adrenocorticotropic hormone in plasma	*Not enough data available*	Higher levels of adrenocorticotropic hormone (Curin et al., 2003; Hamza et al., 2010; Hollocks et al., 2014; Spratt et al., 2012)	*Not enough data available*	Higher levels of adrenocorticotropic hormone correlated to parental neglect/abuse (Kemph & 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 & Corbett, 2014), slower in situations of social threat (Jacobson, 2014; Spratt et al., 2012); higher serum cortisol responses in children with ASD (Spratt et al., 2012); disrupted patterns of cortisol levels in children (Tomarken et al., 2015)	*Not enough data available*	HPA axis hyper reactivity reported in response to stress (Kemph & Voeller, 2008); lower levels of cortisol documented (Kočovská et al., 2013)

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Features of domestication/HSD	Feralized animals	ASD	'Feral' children	RAD
Increased basal levels of oxytocin (and more marked release patterns).	*Not enough data available*	Lower blood oxytocin levels reported (John & Jaeggi, 2021)	*Not enough data available*	Lower salivary oxytocin/atypical secretion reported (Fries et al., 2005; Suzuki et al., 2020)
Increased basal levels of vasopressin (and more marked release patterns).	*Not enough data available*	Decreased vasopressin levels in children (Oztan et al., 2018; Parker et al., 2018)	*Not enough data available*	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 (Anderson et al., 2009; Derelle et al., 2013; Feulner et al., 2013; Gering et al., 2019; Linderholm et al., 2016; Roulin, 2004)	Various hypomelanotic diseases present autistic symptoms (Akefeldt & Gillberg, 1991; Gómez-Lado et al., 2004; von Aster et al., 1997)	*Not enough data available*	*Not enough data available*
Higher levels of vitamin D in blood	*Not enough data available*	Lower serum levels of vitamin D reported in children with ASD (Cannell & Grant, 2013)	*Not enough data available*	*Not enough data available*
Vital cycle				
General undergrowth	Feral canids and pigs reported to be larger than their closest domesticated relatives (Gering et al., 2019; Kruska & Röhrs, 1974; Smith et al., 2018)	General overgrowth reported during childhood (Campbell et al., 2014; Chawarska et al., 2011)	*Not enough data available*	General undergrowth reported (Dobrova-Krol et al., 2008; Stinehart et al., 2012)
Neoteny	*Not enough data available*	Neotenous features uncommon due to increased body size, higher levels	*Not enough data available*	*Not enough data available*

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Features of domestication/HSD	Feralized animals	ASD	'Feral' children	RAD
		of androgens, and earlier puberty than TD subjects (Campbell et al., 2014; Chawarska et al., 2011; El-Baz et al., 2014)		
Reproductive cycle				
Reduced levels of androgens (with a focus on testosterone)	*Not enough data available*	Higher levels of androgens reported (El-Baz et al., 2014; Hauth et al., 2014)	*Not enough data available*	Higher levels of androgens linked to higher stress in early life (Hauth et al., 2014), 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)	*Not enough data available*	*Not enough data available*

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Additionally, we compared the genetic basis of these conditions against the background of the genetics of domestication. Domestication has been hypothesized to result from changes in various genes involved in the development and function of different body parts. Wilkins and colleagues (2014) have highlighted a set of core candidate genes for domestication in mammals, whereas Niego and Benítez-Burraco (2019) have compiled an expanded list of candidates based on several recent analyses of the signatures of domestication in selected domesticates (see Supplementary Materials File 1; column A). That said, as noted by Jensen (2006) or Wilkins and colleagues (2014), and discussed as well in a recent paper by Anastasiadi and colleagues (2022), domestication can be expected to also result in part from pre-existing genetic variation, and even from epigenetic changes. The genetic scenario for feralization is pretty similar, but with the additional circumstance that some genetic differences between feralized and domesticated animals can be due to interbreeding with wild populations, and not to feralization per se (see Zhang et al., 2020 for discussion). Niego and Benítez-Burraco (2021) have also compiled an enlarged list of candidates for feralization relying on available data from a variety of studies (see Supplementary Materials File 1; column B). An important conclusion of Zhang and colleagues' study is that the overlap between the genetic signatures of domestication and feralization is not as complete and one would expect, suggesting that the (partial) reversion of the traits of domestication as found in feralized animals is achieved through modifications in genes not directly involved in domestication; at the same time, some common candidates can be found between these two conditions, which can be expected to account for the reversion of specific traits of domestication observed in feralized animals. Genetic studies on 'feral' children have not been done for obvious reasons. Regarding the genetic basis of RAD, there is evidence that it is genetically influenced (Minnis et al., 2007). Several studies point to the involvement of genes related to dopaminergic and serotonergic activity in abnormal changes in attachment behavior by children (Bakermans-Kranenburg & van Ijzendoorn, 2007; Caspers et al., 2009; Lakatos et al., 2000; Luijk et al., 2011; Spangler et al., 2009; Wazana et al., 2015). Animal models suggest that changes in receptors for oxytocin and endogenous opioid could also result in RAD (Winslow & 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 genetic 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).

Table 3

Structural and Functional Features of Common Candidates for ASD, RAD, and Feralization

Gene	Protein name	Protein function	Relation to feralization	Relation to ASD	Relation to RAD
TRHDE	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 & Sevarino, 1999)	Linked to enhanced dopaminergic and/or reduced serotonergic activity in children; linked to hypothalamic dysfunction (Hashimoto et al., 1991)	*Not enough data available*
OXTR	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)	*Not enough data available*
MAOA	Monoamine Oxidase A	Encodes monoamine oxidase A, involved in degradation of amine neurotransmitters	*Not enough data available*	Alleles associated with increased severity of ASD (Cohen et al., 2003; Cohen et al., 2011; Yoo et al., 2009); alleles correlated with IQ levels, adaptive skills, and language skills (Cohen et al.,	Some polymorphisms moderate detrimental effects of childhood abuse (Caspi et al., 2002); high levels of *MAOA* correlate to less social problems, including disrupted attachment (Caspi et

Gene	Protein name	Protein function	Relation to feralization	Relation to ASD	Relation to RAD
				2003; Cohen et al., 2011)	al., 2002); selected epigenetic mutations correlate to high rates of aggression (Márquez et al., 2013)
5HTT	Serotonin transporter	Transports the neurotransmitter serotonin from synapses to presynaptic neurons	Polymorphisms associated to fear response in hens (Krause et al., 2019) and defensive behaviors in animals (Blanchard et al., 1998); enhancement of serotonin activity inhibits predatory aggression in several species (Nikulina, 1991)	Lower 5-HTT availability in various brain regions correlated to increased levels of serotonin in individuals with ASD (Andersson et al., 2021; Coutinho et al., 2004; Veenstra-VanderWeele et al., 2012)	Significant correlation between one allele of 5HTT and maltreatment, stress, and adversity in childhood resulting in violent/aggressive behavior later in life (Reif et al., 2007)
TH	Tyrosine Hydroxylase	Involved in conversion of tyrosine to dopamine; implicated in the physiology of adrenergic neurons.	Affects behavior and temperament in cattle (Lourenco-Jaramillo et al., 2012)	*Not enough data available*	*Not enough data available*
DRD4	Dopamine Receptor D4	Inhibits adenylyl cyclase.	Variants in *DRD4* correlate with levels of aggression linked to domestication in chickens (Komiyama et al., 2014)	An allele of the gene may increase risk for clinically elevated ASD symptoms in children and adolescents with ADHD (Reiersen & Todorov, 2011)	Polymorphisms linked to disordered attachment such as that found in RAD (Bakermans-Kranenburg & van Ijzendoorn, 2007; Spangler et al., 2009)

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 Supplementary Materials 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.

Table 4

Structural and Functional Features of Common Candidates for ASD and Feralization

Gene	Protein name	Protein function	Relation to feralization	Relation to ASD
CACNA1A	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 *CACNA1C* also correlate to lower performance in semantic tasks in people with schizophrenia (Krug et al. 2010)
FABP4	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 *Fabp4* show ASD-like features, including abnormal behavior and abnormal morphology of pyramidal neurons (Maekawa et al., 2020)
KIT	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, migration and function, among other things (Naumann et al., 2021)	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 (Kilsby et al., 2013; Rothschild et al., 2003)
MBD4	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	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)

Gene	Protein name	Protein function	Relation to feralization	Relation to ASD
		DNA repair (Bellacosa et al., 1999)		
MFRP	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)
MIB1	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)
MITF	Melanocyte Inducing Transcription Factor	Encodes a melanocyte inducing transcription factor, responsible for pigment cell specific transcription of the melanogenesis enzyme genes. Mutations of *MITF* result in auditory-pigmentary syndromes, such as Waardenburg syndrome type 2 and Tietz syndrome (Goding & 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 & Carracedo, 2021)
NCOR1	Nuclear Receptor Corepressor 1	Involved in chromatin condensation and specifically regulates transcription repression of thyroid-hormone and retinoic acid receptors (Zhou et al., 2019).	It is found among Zhang et al. (2020)'s candidates for feralization	Candidate for Mitochondrial Complex III Deficiency, Nuclear Type 2, a condition 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 (Lyst et al., 2013). Pathogenic variants of *NCOR1* have been found in people on the ASD spectrum (Iossifov et al., 2012; Wang et al., 2016)
NF1	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)

Gene	Protein name	Protein function	Relation to feralization	Relation to ASD
NUP155	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)
PACS1	Phosphofurin Acidic Cluster Sorting Protein	Encodes a protein involved in the localization of trans-Golgi network (TGN) membrane proteins (Hinners 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)
PRKCA	Protein Kinase C Alpha	Encodes a protein kinase involved in cell proliferation, differentiation, migration and adhesion (Haughian & 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)
PSMD12	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 (Küry et al., 2017)
ROBO2	Roundabout Guidance Receptor 2	Encodes a protein involved in axonogenesis and which plays a key role in many aspects of neurodevelopment and cognition (Brose et al., 1999; Kidd et al., 1998)	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 (Prasad et al., 2012; Suda et al., 2011), 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)
SLC25A27	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)
SOX6	SRY-Box Transcription Factor 6	Encodes a transcription factor involved in the regulation of many body functions (Cantù et al., 2011)	Affects both feralization and domestication due to its association with the Wnt signalling pathway, which plays a key role in	Mutations have been associated to a complex condition encompassing neurodevelopmental problems and behavioral abnormalities, including

Gene	Protein name	Protein function	Relation to feralization	Relation to ASD
			initial neural crest cell differentiation (Wang et al., 2018)	ADHD and/or ASD features (Tolchin et al., 2020)
TTN	Connectin	Encodes connectin, a protein important for muscle elasticity (Chauveau et al., 2014)	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, 2012)

4 Human 'Feralization' and the Evolution of Language

In the previous section, we have shown 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 that 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 for 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 replacing physical confrontation with verbal confrontation (see Benítez-Burraco & Progovac, 2020; Progovac & Benítez-Burraco, 2019 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., 2021a for details). In this section, we compare 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 aims 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 for the view that changes impacting on our socialization make language/communication simpler and less efficient; and ultimately, iv) provide additional support for the HSD hypothesis of language evolution.

4.1 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 (Fromkin et al., 1974; Kapoor, 1973; 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 et al., 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. Lindgren et al., 2009; Wolk & Edwards, 1993; 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 & Tager-Flusberg, 2001; or McCann et al., 2007 for an opposing 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 Tager-Flusberg, 2006 for discussion), including difficulties with binding, relative clauses, wh-questions, raising and passives (Perovic & Janke, 2013; see Benítez-Burraco & Murphy, 2016 for review). Regarding RAD, a study by Raaska and colleagues (2012) 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 & 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 (Curtiss et al., 1974; Fromkin 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; Happé, 1993; Kaland et al., 2002; Kissine, 2012; Lord & Paul, 1997; Surian et al., 1996; Tager-Flusberg, 1992, 1993, 2000). 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 (Bennett et al., 2009; Green & Goldwyn, 2002). 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 et al. (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 require 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., 2012 for discussion). In the case of ASD, pragmatic deficits, specifically, have been also attributed to deficient socialization patterns (Bailey et al., 1996), resulting from an internal deficit. This is specifically the case with mind-reading, which impedes the detection and recognition of 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 Baron-Cohen, 2000; Happé, 1993, 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; Colich et al., 2012; Schultz et al., 2003; Wang et al., 2006; Wang et al., 2007). 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; Gupta et al, 2016; Heim et al., 2013).

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 (see Rolls, 2015 for review). It has been argued that the hippocampus supports 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 & Ritchey, 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 & 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 & Benítez-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 to find additional evidence that these abnormal language profiles might result from an attenuation of HSD features as levels of reactive aggression increase.

4.2 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, 2017; Tyack, 2020; Vernes, 2017). 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 (Sánchez-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 & Nitzschner, 2013; Range & Virányi, 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., 2021b 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., 2015; Shilton et al., 2020; vonHoldt et al., 2017) 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 (2021b) 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 (Shipman, 2021; Smith & Litchfield, 2010). 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 & 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 & 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 (Bräuer et al., 2006; Hare & Tomasello, 1999; 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. Paul et al., 2008; Volkmar et al., 2005; Wetherby et al., 2007) 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, characterizations 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 (Bradshaw & Cameron-Beaumont, 2000; Brown, 1993). 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 with humans, while domestic cats are more vocal in social situations, and less likely to use agonistic vocalizations. These findings parallel those of Nicastro (2004), who compared domestic cat vocalizations with those of wild cats, finding that in general domestic cats produce calls of a shorter duration and a higher pitch frequency than wild cats. Domestic cats are also known to produce vocalizations typical of kittens, such as purring and chirping, which is not the case in feral cats (Bradshaw & Cameron-Beaumont, 2000), which is in line with the neotenic features typically associated to domestication. Regarding dogs and dingoes, Smith and Litchfield (2010) found in their research that, similarly to wolves, dingoes are found to howl and whimper more than domestic dogs, and bark less. Moreover, their barks tend to be shorter than their domestic counterparts, and used almost exclusively for giving warnings. In contrast, domestic dogs’ barks are more versatile, and used in a variety of social situations (Corbett, 2004). Studies on domestic and feral pigs show that they have a wide and varied repertoire that differs in some respects—for example, the ‘trumpeting’ sound produced by non-domesticates has not been recorded in domestic pigs (Garcia et al., 2016; Tallet et al., 2013). Evidence on pigs is difficult to interpret, however, given the blurred lines that exist between ‘feral’ pigs and ‘wild’ pigs; since a great deal of admixture takes place between the types of swine, it is hard to attribute changes to feralization (Garcia et al., 2016). Although vocal communication of mammals is more relevant to our research because of their phylogenetic proximity, patterns from bird species can also contribute to our knowledge of changes conferred through feralization, particularly because of the abundant research on the notable effects of domestication on birdsongs. Gering and colleagues (2015) found that feral chickens on Kauai have more extreme calls, and exhibit much more variation in their 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 & 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).

5 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 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 & Kushnick, 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.

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

Data Availability: For this article, data for the comparison of the genetic basis of Domestication-Feralization are freely available (Niego & Benítez-Burraco, 2022).

Supplementary Materials

The Supplementary Materials contain data for the comparison of the genetic basis of Domestication-Feralization (for access see Index of Supplementary Materials below).

Index of Supplementary Materials

Niego, A., & Benítez-Burraco, A. (2022). *Supplementary materials to "Revisiting the case for 'feral' humans under the light of the human self-domestication hypothesis: Focusing on language"* [Data]. PsychOpen GOLD. https://doi.org/10.23668/psycharchives.8216

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