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The Parasite-Mediated Domestication Hypothesis

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ABSTRACT

Based on the premise that parasites indirectly influence literally all of the main processes that otherwise underlie the domestication syndrome, it is hypothesised here that endoparasites (helminths and protozoa) have played an important mediating role in the process of (proto)domestication. The hypothesis predicts that the frequency of domestication syndrome traits such as tameness, depigmentation, mottling, piebaldism, floppy ears, short and curled tail, reduced size of the adrenal gland, etc. in the (wild) population increases i) with decreasing genetic resistance to parasites and/or ii) with increasing parasite load. According to the parasite-mediated domestication hypothesis, the features of the domestication syndrome could be genetically linked to genes related to resistance/tolerance to parasites, the role of miRNA in the process of epigenetic inheritance or the transgenerational inheritance of stress pathology.

Keywords: domestication syndrome, endoparasites, genetic resistance, neural crest cells, endocrine system, miRNA

BACKGROUND

There are two competing hypotheses about the path of initial domestication of animals, with the wolf (*Canis lupus*) being the first animal to undergo this process. The first and predominant hypothesis is the commensal scavenger or self-domestication hypothesis, and the second is the pet keeping or cross-species adoption hypothesis (Serpell, 2021; Mech and Janssens, 2022). The first states that domestication was initiated by individuals of the wild population approaching human settlements where the remains of human prey were found, while the second states that Palaeolithic humans took wolf pups from dens at an early age and raised them.

Either way, to be selected for further breeding by humans, the animal had to have an attenuated stress response, as individuals with low levels of prosociality were shunned or killed (Losey, 2022). In other words, the wild individuals that entered the (self-)domestication process had to express at least some degree of tameness by nature, which is thus a key trait of the domestication syndrome.

The domestication syndrome is defined as a set of phenotypic traits common to different species of domesticated animals, regardless of the path of domestication and the further methodical selection to which they have been subjected. The phenomenon was first described by Darwin (1868) but not explained until more than a century later by Belyaev (1979), whose silver fox domestication experiment showed that selection for tameability destabilises the regulatory systems controlling morphological and behavioural development, resulting in changes otherwise characteristic of the domestication syndrome, i.e. tameness, floppy ears, upturned tail, depigmentation, etc.

Belyaev (1979) proposed that the traits of the domestication syndrome are genetically linked to genes associated with tameness. He proposed that selection for tame behaviour, i.e. an impaired stress response, leads to significant

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changes in the entire 'hypothalamic-pituitary-adrenal' system (HPA axis) and thus to glucocorticoid secretion - the domestication syndrome is thus based on the close relationship between the nervous and endocrine systems and their effects on ontogenetic development. Later, Crockford (2002) proposed that genetically controlled changes in the rhythm of thyroid hormone secretion are crucial for the initiation and/or implementation of heterochronic changes and thus play an important role in the domestication syndrome.

Indeed, such changes in the endocrine system alter embryonic and postnatal growth, maturation, stress response, brain development, hair/skin production and pigmentation, adrenal gland function/size, and gonadal development and function, which is crucial in the context of the domestication [syndrome] (Dobney and Larson, 2006; Lord et al., 2020).

Finally, Wilkins et al. (2014) deepened the understanding of the mechanism underlying the domestication syndrome by proposing that the main phenotypic components of the syndrome are neural crest cell (NCC) derivatives. Accordingly, the multiple phenotypic changes that characterise the domestication syndrome reflect a developmental reduction in NCC input for the affected phenotypic traits. They further suggested that the deficits of NCCs during embryonic development could be due to the lower number of NCCs initially formed, the lower migratory capacity of NCCs and consequently the lower number of NCCs at the final sites or the lower proliferation of these cells at these sites.

HYPOTHESIS ON THE POSSIBLE ROLE OF ENDOPARASITES IN THE PROCESS OF DOMESTICATION

Since the domestication of animals, there appears to have been a large increase in the number of parasites common to humans and domesticated animals (McNeill, 1976; Morand et al., 2014). This relationship seems to be an expected consequence of the advent of domestication, a suitable condition for intraspecific transmission of parasites. However, another aspect should be highlighted here, namely the causality of this relationship, whereby the evolutionary adaptation of parasites to maximise their dispersal may have played an important role in making this relationship possible in the first place. Therefore, they might have played an important role in (proto-)domestication, particularly in self-domesticated mammals, i.e. at a very early stage of the domestication process initiated by the animals themselves.

Behavioural changes are usually mediated by many, often interacting, endocrine and neuromodulatory mechanisms (Kaushik et al., 2012; Del Giudice, 2019). Although not all behavioural changes are adaptive, parasites can influence these mechanisms and alter host behaviour in ways that increase their likelihood of transmission and give them a specific adaptive advantage (Poulin, 2010; Poulin, 2013; Del Giudice, 2019; Hughes and Libersad, 2019). In particular, parasites can influence fear responses and anxiety, i.e. reduce them in order to increase inter- and intraspecific contacts. In the early stages of domestication, such tame behaviours aimed at reducing animals' fear of humans were necessary for life in an anthropogenic environment (Herbeck et al., 2022).

The fear response is controlled by the HPA axis, in particular by the secretion of glucocorticoids (GC), which is also deviated by parasite infestations. Although GC levels increase on average in affected individuals, their secretion varies greatly depending on the phase of parasite infestation (O'Dwyer et al., 2020). The temporal activation of the HPA axis is also highly dependent on the severity and duration of the stressor(s) and the extent to which the organism can anticipate or cope with the challenge. However, stress responses are usually inhibited by negative feedback mechanisms, with GC reducing drive/instinct (processed in the brainstem) and promoting trans-synaptic inhibition by limbic structures, e.g. the hypothalamus (Herman et al., 2016). However, if either stimulus is intense enough, the facilitation will still override the feedback inhibition (Gann et al., 1977). In contrast, under certain conditions (e.g. chronic drive), GC can also induce positive feedback in some brain structures and increase the reactivity of the HPA axis (Herman et al., 2016).

Elevated GC indeed have different effects, which also depend on the particular time window in which the elevations occur. For example, prenatal stress of various kinds, including parasite-induced stress, can cause postnatal changes in HPA activity, including a reduction in adrenal weight and thus an inappropriate stress response (Welberg and Seckl, 2001; O'Dwyer et al., 2020).

Parasites also affect the function of the thyroid gland, a gland that secretes thyroid hormones in a distinctly pulsatile manner and is crucially involved in the regulation of developmental processes. Indeed, a certain level of its hormones is required to initiate migration, differentiation and maturation of early embryonic cells, growth, central nervous system development, hair growth, adrenal gland function, pigment (eumelanin) production, etc. Given the importance of the thyroid gland in the timing of developmental processes, it has been suggested that disruption of thyroid function leads to the heterochronic changes that are otherwise characteristic of domesticated animals (see Crockford, 2004). And parasites may also play a role in this aspect of domestication, as endoparasites, especially helminths, have been shown to cause various thyroid disorders, including thyroid nodules, hypo- and hyperthyroidism (Raizada, 2021).

Further, parasite infestation confined to the maternal intestine has been shown to positively influence postnatal brain development in the context of long-term potentiation

related to learning and cognition (Haque et al., 2019). Accordingly, there is evidence that domestication, artificial selection and breeding lead to an improvement in spatial memory in human-made experimental settings, which has been explained as an adaptation to the anthropogenic environment that enables more efficient task solving (Lewejohann et al., 2010). However, this could also be partly due to the higher threshold for acute stressors in domestic animals, i.e. less anxiety and fear, as mentioned earlier, because acute stress can impair cognition, learning and (spatial) memory (Wolf 2003; Sandi et al., 2005).

Furthermore, parasites cause changes in the host profile of miRNA, small non-coding RNAs that can control gene expression (Paul et al., 2020), not only through parasite infestation itself, but also through the secretion of vesicles containing miRNAs into host cells (Buck et al., 2014). miRNAs are significantly involved in many prenatal and postnatal developmental and physiological processes (Floris et al., 2016). In the context of the domestication syndrome, it is particularly important to note that miRNAs are also substantially involved in the formation of the neural crest during embryonic development; either in the induction, specification, delamination, migration or differentiation of NCCs (Weiner, 2018). In addition, there are some miRNA families involved in parasitic diseases caused by endoparasites as well as in NCCs differentiation (Table 1). Since small non-coding RNAs, including miRNAs, are known to enter the foetus via the placenta to regulate foetal development (Li et al., 2015), it is reasonable to assume that parasites indirectly influence embryonic development along the 'miRNA-transplacental transport-NCCs' axis and may also mediate the phenotypic changes that characterise the domestication syndrome.

In addition to the NCCs, miRNAs are involved in the activity of the endocrine system and regulate hormone production, activity and responsiveness of target cells. They can directly act on target genes encoding hormones or enzymes involved in hormone production or metabolism, thus affecting hormone concentrations (Peng and Wang, 2018; Peng and Li, 2022). For example, miRNA-21, miRNA-let-7, miRNA-16 and miRNA-24 are among the miRNAs involved in various parasitic diseases (see Table 1 for references), as well as possible neuroendocrine cell function (Park, 2017).

Finally, parasites have been shown to cause disruptive selection (favouring otherwise extreme phenotypes) in animals and thus can rapidly increase genetic variance within a host population (Duffy et al., 2008; Blanchet et al., 2009). High phenotypic variability (i.e. a high frequency of extreme phenotypes) is otherwise observed in populations of domestic animals in which breeds of the same species differ from each other to the same extent as species of the same genus in the natural state (Darwin, 1868). Barker (2001) reported that much of the genetic variance of a domesticated species is due to differences between breeds, while Groeneveld (2010) stated that the uniqueness of a breed is not evident from molecular data, which on the contrary show that most of the genetic diversity exists within a breed and not between breeds.

The genetic background for the possible role of parasites in the process of domestication remains to be explored, but few starting points are suggested here. First, since resistance and tolerance to parasites are genetically determined and heritability is relatively high (Steer and Wakelin, 1998; Mazé-Guilmo, 2014), the characteristics of the domestication syndrome could be genetically linked to genes related to resistance/tolerance to parasites, with the (proto-)domestication selection favouring less genetically resistant individuals. Second, given the influence of parasites on the host profile of miRNA, the role of miRNA in the process of epigenetic inheritance (Sharma, 2014; van Otterdijk and Michels, 2016) should also be considered. Finally, manipulating the foetal

Table 1: Some miRNAs involved in various parasitic diseases as well as in neural crest (NC) development

miRNAs	Influence during NC development	References
miRNA-140	bone development, chondrogenesis	Buck et al., 2014 Weiner, 2018 Paul et al., 2020 Antonaci and Wheeler, 2022 He and Pan, 2022
miRNA-27	chondrogenesis	
miRNA-124	chromaffin cells	
miRNA-let-7a	craniofacial development	
miRNA-17~92	NC induction/specification, craniofacial development, chondrogenesis	
miRNA-24	NC induction/specification	
miRNA-21	Schwann cells	
miRNA-let-7	epithelial to mesenchymal transition/migration, chondrogenesis	
miRNA-1	craniofacial and hart development, pigment cells	
miRNA-20a	NC induction/specification	

environment by exposing the mother to adverse conditions during pregnancy - in our case parasites - can alter the response to stress not only in the offspring but over several generations (Matthews and Phillips, 2012). Therefore, in the context of changes in the HPA system (stress response) associated with domestication, transgenerational programming of HPA function and behaviour, i.e. inheritance of parasite-induced stress pathology, should not be neglected.

Based on the premise that parasites indirectly influence literally all the main processes that otherwise underlie the domestication syndrome, it is hypothesised that parasites (specifically endoparasites: helminths and protozoa) played an important mediating role in the process of domestication. However, it is important to stress at this point that a heavy, persistent parasite load would be detrimental to the health of the animals and could result in the parasite load being too high for the animals to survive even under ideal living conditions. It is therefore difficult to imagine how this situation could be maintained over hundreds or thousands of generations. Moreover, wild animals can be infected with various parasites that only make them sick, but not domesticated or even more accessible to humans.

The hypothesis presented here assumes that a 'parasite effect' is primarily involved in the emergence of the domesticated state (proto-domestication) and not necessarily in its continuous maintenance. According to the parasite-mediated domestication hypothesis, it is predicted that the frequency of domestication syndrome traits, such as tameness, depigmentation and mottling, floppy ears, short and curled tail, reduced adrenal gland size etc. in the (wild) population increases i) with decreasing genetic resistance to parasites, i.e. the frequency of parasite resistance alleles in the population and/or ii) with increasing parasite load. Therefore, it would also be expected that domestic animals are genetically less resistant to the parasites. In this regard, there have already been some comparative studies examining the parasite load in wild and domestic animals, with slightly contradictory results. In pigs, for example, Ineson (1954) showed differences in parasite load depending on the parasite species, with domestic pigs having a slightly higher parasite load than wild pigs. In contrast, Alwin et al. (2015) found that parasite loads were higher in wild boars than in semi-free-range domestic pigs and on-farm domestic pigs, with the latter having the lowest parasite load. The studies on wild and domestic canines cannot provide a clear answer either (see Eguía-Aguilara et al., 2005; Mitrápková et al., 2015; Čabanová et al., 2016; Čabanová et al., 2017). However, in such studies comparing wild and domestic counterparts, the living conditions and the general possibility of the animals to be exposed to the parasites are most likely determining factors for the parasites incidence, so that the parasite load cannot simply be considered as an indicator of the degree of genetic resistance to parasites.

To test the hypothesis proposed here, genetic resistance should be tested primarily in wild individuals showing signs of the domestication syndrome or secondarily in domestic animals or domestic-wild hybrids and compared with their fully wild counterparts. However, this requires that the genetic markers for resistance to parasites are known. The parasite load should be investigated either in the wild population consisting of individuals showing signs of the domestication syndrome (tameness, specific morphology and colouration, etc.) or in the population of domestic animals, domestic-wild hybrids and wild animals of the same species sharing the same environment.

It can then be predicted that the wild individuals showing signs of the domestication syndrome, as well as the domestic animals and domestic-wild hybrids, are genetically less resistant to parasites and/or more infested with parasites than the fully wild individuals.

Last but not least, special caution must be taken with domesticated animals that may have been artificially selected for parasite resistance, because in this case the results are inevitably misleading and the conclusions biased.

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Hipoteza o udomačitvi preko parazitov

IZVLEČEK

Izhajajoč iz predpostavke, da paraziti posredno vplivajo pravzaprav na vse glavne procese, ki sodelujejo pri pojavu sindroma udomačevanja, domnevamo, da so imeli endoparaziti (helminti in protozoji) pomembno posredniško vlogo v procesu (proto) domestikacije. Hipoteza predvideva, da se pogostnost lastnosti sindroma udomačitve, kot so krotkost, depigmentacija, lisavost, pegavost, povešena ušesa, kratek in zavit rep, zmanjšana velikost nadledvične žleze itd., v (divji) populaciji povečuje i) z manjšo genetsko odpornostjo proti parazitom in/ali ii) z naraščajočo obremenitvijo s paraziti. V skladu s hipotezo o udomačitvi preko parazitov, bi lahko bile značilnosti sindroma udomačitve genetsko povezane z geni, povezanimi z odpornostjo/toleranco na parazite, vlogo miRNA v procesu epigenetskega dedovanja ali transgeneracijskega dedovanja stresne patologije.

Ključne besede: sindrom udomačitve, endoparaziti, genetska odpornost, celice nevralnega grebena, endokrini sistem, miRNA