



## Review

## The role of humans in facilitating and sustaining coat colour variation in domestic animals

Anna Linderholm, Greger Larson\*

*Durham Evolution and Ancient DNA, Department of Archaeology, Durham University, South Road, Durham, DH1 3LE, United Kingdom*

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## ABSTRACT

Though the process of domestication results in a wide variety of novel phenotypic and behavioural traits, coat colour variation is one of the few characteristics that distinguishes all domestic animals from their wild progenitors. A number of recent reviews have discussed and synthesised the hundreds of genes known to underlie specific coat colour patterns in a wide range of domestic animals. This review expands upon those studies by asking how what is known about the causative mutations associated with variable coat colours, can be used to address three specific questions related to the appearance of non wild-type coat colours in domestic animals. Firstly, is it possible that coat colour variation resulted as a by-product of an initial selection for tameness during the early phases of domestication? Secondly, how soon after the process began did domestic animals display coat colour variation? Lastly, what evidence is there that intentional human selection, rather than drift, is primarily responsible for the wide range of modern coat colours? By considering the presence and absence of coat colour genes within the context of the different pathways animals travelled from wild to captive populations, we conclude that coat colour variability was probably not a pleiotropic effect of the selection for tameness, that coat colours most likely appeared very soon after the domestication process began, and that humans have been actively selecting for colour novelty and thus allowing for the proliferation of new mutations in coat colour genes.

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## 1. Introduction

Darwin was the first to recognize that a wide range of domestic animals share multiple phenotypic characteristics, the most obvious of which is a wide variety of coat colours [1,2]. Numerous plants also possess colour varieties that differentiate them from their wild ancestors, but in animals, the only trait other than variable coat colours that occurs ubiquitously across domestic animals are dwarf and giant varieties [3]. The commonality of variable coat

colours is interesting in light of the fact that the process of domestication likely began no more than 15,000 years ago and occurred independently in both the Old and New Worlds [4].

Domestication significantly altered the genetic, behavioural, and phenotypic characteristics of all the organisms involved, and numerous studies have revealed a great deal about the genetic basis of domestic phenotypes including, in many cases, the causative mutations themselves [5]. Since coat colour patterns often follow a Mendelian inheritance mode, they have been among the first traits to be systematically analysed at the molecular level [6] and the functional genomics of coat colour variation in domestic animals has been studied in depth [7]. Though several recent reviews have focussed on the genetic basis of coat colour variation [7–10], a

\* Corresponding author. Tel.: +44 1913341574.

E-mail address: [greger.larson@durham.ac.uk](mailto:greger.larson@durham.ac.uk) (G. Larson).

number of outstanding questions related to the role that humans played in generating the diversity of domestic animal coat colours remain unanswered.

Firstly, does the ubiquity of coat colour variation result from direct selection by humans, or are variably coat colours a consequence of selection on other behavioural traits including tameness [3]? Secondly, because most studies of domestic animal coat colour have been carried out on modern samples, is it possible that coat colour variation is just a recent phenomenon, and if not, how soon after the domestication process began did coat colour diversity begin to increase? Lastly, to what degree are humans primarily responsible for driving and maintaining coat colour variants that differentiate wild and domestic populations?

### 1.1. The genetic basis of coat colour

Animal colouration is modified through both pigmentation and nanostructure and has many functions including communication, camouflage, predatory avoidance, photoprotection, microbial resistance and thermoregulation. There are two main groups of genes affecting mammalian coat and skin colour: those that act on the pigment synthesis and those that modify the melanocytes, the pigment producing cells [8].

The process of pigment cell development plays a key role in determining coat colour and more than 300 genes have been identified that have an effect on pigmentation either directly or indirectly [11]. Most of these genes act on either the production or the regulation of two pigments, pheomelanin and eumelanin [8]. Both MC1R and ASIP act like switches, while KIT and TYR (and other associated genes) are important in regulation of melanin production (Fig. 1). A brief description of the key genes and the roles they play is described below.

The KIT gene encodes the mast cell growth factor receptor and with its ligand MGF, it plays a key role in the growth and differentiation of melanocytes, hematopoietic cells, and germ cells [12]. Since this gene plays a central role in melanogenesis and melanoblast migration and proliferation, mutations in this gene can have serious consequences. Pleiotropic effects including anaemia, sterility, premature ovarian failure, and chronic gastric ulceration have been identified in a number of different species. In particular, the negative consequences of possessing a white coat as result of a modified KIT gene are generally greater than mutations in other genes that affect coat colour [12–14].

A key enzyme regulating pigment synthesis is TYR. The TYR gene encodes the enzyme tyrosinase, which is in turn involved in the production of melanin from tyrosine. As a result, mutations in the TYR gene result in unpigmented skin, fur or feathers. Albinism caused by a deficiency of melanin pigment in the skin, hair, and eye, results from mutations in genes involved in the biosynthesis of melanin pigment and has been detected in a number of wild and domestic species [15–17]. In addition, mutations in other regions of the TYR gene can result in TYR activity becoming sensitive to temperature. This type of mutation leads to a distinct coat colour pattern where the colder parts of the body (i.e. ears, tail and legs) are coloured and the rest of the body remains white [18]. This type of pattern is prominent in both Siamese and Burmese cats. The Siamese has a phenotype referred to as “pointed” reflecting the fact that pigment is expressed at the extremities creating a mask on their faces. Burmese cats possess an allelic variant that is less temperature sensitive and they display pigment mainly on the torso rather than the extremities [18].

Most coat colour variation is the result of the ratio between the two pigments: eumelanin and pheomelanin. Both MC1R and ASIP control this ratio by acting like switches. MC1R is a G protein-coupled receptor located in the melanocyte plasma membrane (Fig. 1). The gene is highly conserved among vertebrates and has

a relatively simple genetic structure made up only of a single 1 kb exon [8]. The MC1R protein binds to a class of pituitary peptide hormones called melanocortins that regulate the synthesis of both eumelanin and pheomelanin. Darker colours are produced when eumelanin is up-regulated and light colours result when pheomelanin is increased [15].

The Agouti signal peptide (ASIP) is a paracrine signal protein that antagonizes MC1R to switch to eumelanin production [19]. Though there have been more mutations identified in MC1R, they only occur in the coding region [8], whereas mutations affecting ASIP are found in both the coding and regulatory regions [20].

## 2. Three questions

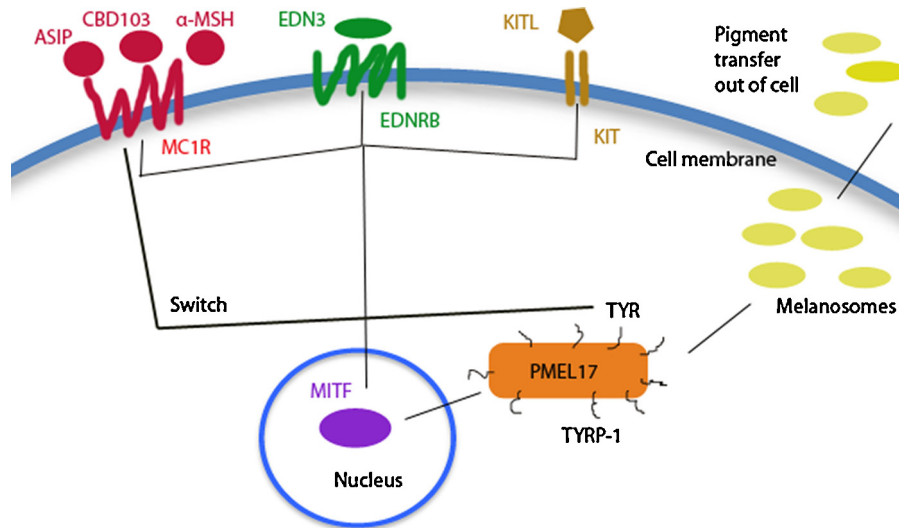
### 2.1. Tameness and domestic coat colours

Dmitry Belyaev hypothesized that the phenotypes expressed by all domestic animals resulted not from deliberate selection for individual traits, but as a consequence of selection for a behavioural characteristic. In 1959 he began testing his hypothesis by breeding silver foxes, an animal never previously domesticated. He selected against aggression and only allowed the tamest 10% of the population to breed. Belyaev expected that by selecting a specific behavioural trait he would influence the phenotype of subsequent generations, making them more domestic in appearance [21]. The experiment succeeded by producing foxes with drooping ears, upturned tails, shortened snouts and other features that commonly distinguish domestic animals from their wild ancestors.

Beyond demonstrating how readily domestic phenotypes could appear through a selection for a behavioural trait, Belyaev also provided a mechanism to explain how early animal domestication could have begun that did not require deliberate human action. For instance, wolves, initially wary of humans may have been attracted to the waste generated by human camps. Only those with a reduced fight or flight distance would have been able to take advantage of the resources available near the human niche, and it was those wolves that were likely to become more intimately involved in human settlements.

This possibility has been formalised and expanded upon by both Vigne [22] and Zeder [4]. Vigne sets out several consecutive phases of the intensification of the relationship between humans and animals that begins with anthropophily and proceeds to commensalism, control in the wild, control of captive animals, extensive breeding, intensive breeding, before ending in pets. Zeder has recently expanded this view by recognising that not all animals entered into a domestic relationship in the same way. Instead, Zeder characterised three separate domestication pathways: a commensal pathway, a prey pathway and a directed pathway [4]. The commensal pathway is similar to Vigne’s model in that the process begins with a habituation phase with no intentional human selection. Animals that were first hunted, and then later more directly managed before humans began actively controlling their breeding followed a prey pathway. The directed pathway, most commonly associated with much more recently domesticated pets (<200 years) skips the earliest phases of the other pathways and begins with the intentional capture of wild animals followed by taming in captivity.

These perspectives are important since they allow for the possibility that the genetic mechanisms and appearance of domestic coat colours may have been different amongst animals that followed separate pathways. In Belyaev’s fox-farm experiment, piebald and spotted coat colours, a trait determined by the incompletely dominant Star mutation, first appeared after only 10 generations [23]. Belyaev only selected for tameness, and given the lack of intentional human action in the early stages of the commensal pathway,



**Fig. 1.** Schematic illustration of a melanocyte depicting some of the main proteins involved in pigmentation production within the cell. Transcription of microphthalmia-associated transcription factor (MITF) is regulated through several signalling pathways controlled by melanocortin 1 receptor (MC1R), endothelin receptor type B (EDNRB), and v-kit Hardy-Zuckerman 4 feline sarcoma viral oncogene homolog (KIT). All three of these genes are in turn regulated by other extracellular proteins. MC1R is regulated by agouti signally protein (ASIP), beta defensin 103B (CBD103), and alpha melanocyte stimulating hormone ( $\alpha$ -MSH). EDNRB is regulated by endothelin 3 (EDN3), and KIT is regulated by KIT ligand (KITL). The transcription of MITF initiates the production of the melanosome, the melanin carrier, through several steps involving silver homolog (PEML17), tyrosinase (TYR) and TYRP1, tyrosinase-related protein 1. MC1R can act like a switch that determines the ratio of the production of pheomelanin (a cysteine-containing red-brown polymer of benzothiazine) and eumelanin (a brown-black polymer of dihydroxyindole carboxylic acids) and tyrosinase catalyses the first step of the biosynthetic pathway for both of these. The pigment is then transported out of the cell into to neighbouring tissues, resulting in pigmented skin, fur, and plumage. (For interpretation of the references to color in the artwork, the reader is referred to the web version of the article.)

it was conceivable that the genes controlling behaviour (at least tameness) were also involved with the first appearance of spotted coat colours. In dogs, the microphthalmia-associated transcription factor (MITF) gene has been associated with random and piebald spotting in Beagles and Newfoundlands [24] and in Boxers Bull Terriers, Dalmatians and Cavalier King Charles Spaniels [25]. Because MITF affects melanoblasts that derive from neural crest cells [26], Schmutz and Berrere [10] speculated that perhaps the Star mutation was MITF. Even if this specific correlation was not causation, it remained possible that other genes involved in a shift toward a lack of aggression also contributed to variable coat colours.

In order to test this possibility, Albert et al. [27] typed 201 genetic markers in an intercrossed population generated by mating one populations of rats selected for tameness and another selected for aggression over 60 generations. Though they identified two significant QTL for tameness and a QTL for white coat spotting, the two regions did not overlap and there was no evidence for the linkage of coat colour and behaviour. This does not mean that other phenotypic traits are not associated with selection for tameness, though no explicit link has yet been established [23].

More generally, the pattern of coat colour genes identified in a wide range of animal domesticates suggests that the same molecular mechanisms are involved regardless of when or how animals were domesticated (Table 1). Since so many of the same genes underlie common coat colour variations, and because only a few domestic animal species followed the commensal pathway in which tameness was an important prerequisite, it is unlikely that the same genes controlling behaviour alterations in domestic animals are involved with coat colour variation.

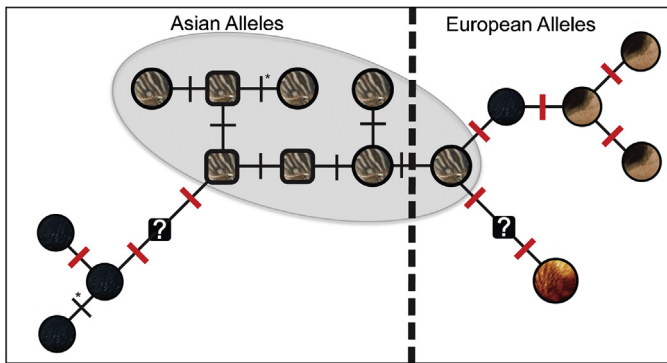
**2.2. How soon did coat colour variation appear following domestication?**

Generally speaking, wild animals possess limited coat colour variability. Given its importance in camouflage and predatory avoidance, there is a significant selection pressure against non-wild type colouring. The exceptions to uniformity most often have

**Table 1**

A list of animals by the suggested pathways [4] they entered into a domestic relationship with people, accompanied by a list of four key coat colour genes known to vary in each species. The word ‘yes’ indicates that known variants of the gene are present in the species and blank cells under each gene means that either the gene does not vary or has not been investigated in that species. The lack of a correlation between the proposed pathways and the variable coat colour genes suggest that no matter how or when animals were domesticated, similar if not identical molecular mechanisms drive coat colour variability in all domestic animals.

Pathway	Species	KIT	TYR	MC1R	ASIP	References
Commensal	Dog	Yes	Yes	Yes	Yes	[10]
	Cat	Yes	Yes	Yes	Yes	[45–47]
	Pig	Yes	Yes	Yes	Yes	[48–51]
	Rat	Yes	Yes	Yes	Yes	[52,53]
	Mice	Yes	Yes	Yes	Yes	[54–56]
	Guinea pig		Yes	Yes	Yes	[57]
	Chicken		Yes	Yes	Yes	[58–60]
	Duck	Yes	Yes	Yes	Yes	[61,62]
	Turkey			Yes		[63]
Prey	Sheep	Yes	Yes	Yes	Yes	[64–66]
	Goat	Yes	Yes	Yes	Yes	[67,68]
	Alpaca/Llama		Yes	Yes	Yes	[69,70]
	Reindeer			Yes	Yes	[71]
	Cattle	Yes	Yes	Yes	Yes	[72–74]
	Yak			Yes	Yes	[75]
	Water buffalo		Yes	Yes	Yes	[76]
Directed	Horse	Yes	Yes	Yes	Yes	[6,14,77]
	Donkey	Yes		Yes	Yes	[78]
	Camels		Yes	Yes	Yes	[79,80]
	Buffalo		Yes	Yes	Yes	[76]
	Ferret			Yes		[81]
	Hamster			Yes	Yes	[82]
	Rabbit			Yes	Yes	[83]
	Turtle		Yes			[84]
	Mink		Yes	Yes		[81,85]
	Chinchilla		Yes	Yes	Yes	[54,83]
	Gerbil		Yes	Yes	Yes	[86,87]
	Ostrich			Yes	Yes	[88]
	Emu			Yes		[89]
Goldfish			Yes	Homologue	[90]	
Zebrafish	Yes		Yes	Yes	[59,91,92]	



**Fig. 2.** A median-joining network of MC1R alleles present in Asian and European pigs. Circles represent known alleles and squares are inferred alleles that have not yet been typed in any individuals. Colours inside the shapes represent the observed and predicted coat colours and question marks mean the colour is unknown. The perpendicular black and red lines depict single synonymous and non-synonymous mutations respectively. The asterisk indicates an identical mutation at different positions on the network. The shaded region demarks all the alleles found in wild boar and the non-shaded alleles are found in domestic pigs in Europe and East Asia. Image adapted from [32].

to do with environmental variation through time and/or space. For instance, several mammalian species including hares respond to seasonal variation in solar radiation and ecological factors by becoming white during winter, before displaying darker coats in summer [28].

Skin colour in humans is also highly variable though in this case the shift in selection is not due to seasonal variation. Instead, selective pressure is thought to have created the observed inverse correlation between skin pigmentation and latitude. Lighter skin tones are generally required at higher latitudes for continued vitamin D synthesis. Given the decreased solar radiation throughout the year there is also less of a cost associated with ultraviolet rays that are more damaging closer to the equator [29,30]. Reduced MC1R function mutations are responsible for both lighter skin and red hair. A novel mutation that also reduced MC1R activity was typed in two Neanderthal individuals suggesting that varied pigmentation levels evolved independently in both modern humans and Neanderthals, perhaps as a response to living at higher latitudes [31].

A recent study of MC1R variation in wild boar sampled across the Old World demonstrated that, like many progenitors of domestic animals, there was significant purifying selection for the wild type MC1R protein [32]. Wild boar populations in Europe and East Asia possessed seven separate synonymous substitutions in the MC1R exon, suggesting that non-synonymous mutations that altered the efficiency of the MC1R protein occurred, but the individuals that possessed them did not pass the mutations onto subsequent generations. The same study also identified nine non-synonymous mutations in 31 European and 19 Asian domestic pig breeds that were associated with black, red, and black and white spotted coat colours (Fig. 2). A test of the ratio between synonymous and non-synonymous substitutions revealed a strong signature of positive selection in the domestic breeds thus suggesting that the appearance of the domestic coat colours was not the result of a relaxation of selection. Instead, humans likely preferentially selected for novel coat colours as they appeared, allowing for additional mutations to occur on top of the initial non-synonymous mutations [32].

Lastly, the accumulation of the seven synonymous mutations between East Asian and European wild boar likely took place after the split between the two populations 1.6–0.8 million years ago [33], while the nine non-synonymous mutations appeared only after pigs were domesticated independently in Western Eurasia [34] and East Asia [35] over the last ~10,000 years. The significantly

reduced time span over which the causative mutations underlying non-wild type coat colours appeared, suggests that humans actively encouraged the proliferation of novel coat colours soon after domestication began [32].

Though wild boar possess uniform camouflage coat colouring across their range, cave paintings (including the 24,700 year old images at Pech-Merle, France) suggest that pre-domestic wild horses possessed at least three coat colour variations including black, bay, and leopard spotting. A recent ancient DNA study typed the causative mutations associated with these colours and determined that each of the coats depicted in cave paintings were present in pre-domestic horse populations [36]. A separate study carried out by the same group extracted DNA from 89 samples dated from the Late Pleistocene to the Iron Age. In each of these samples they typed the variation amongst eight single nucleotide polymorphisms present in six genes (ASIP, MC1R, KIT13, KIT16, MATP and SILV9), all of which are linked to specific horse coat colours. The temporal pattern of variation elucidated in their data revealed a general dearth of variability in pre-domestic horses and a significant increase in coat colour variation coincident with the beginning of horse domestication [37].

Both the modern genetic data from domestic pigs and the ancient data from domestic horses suggest that there was virtually no lag between the origins of domestication and the onset and proliferation of coat colours actively selected against in a wild context. The evidence from Belyaev's foxes demonstrated that piebald and spotted coat colours could appear after only 10 generations [23]. This lends plausibility to the idea that novel variation could take place quickly, and that as long as humans controlled the reproduction of their domestic animals, they could encourage the proliferation of coat colour variation. This would increase the frequency of the novel phenotypes to the point that additional mutations would occur, thus leading to greater variation and further molecular distancing from the wild type allele. The fact that many of the same genes are responsible for the domestic phenotypes also suggests that the variation appears early regardless of which pathway the animal followed into domestication (Table 1).

### 2.3. Pleiotropic effects and deleterious traits

Though at least 150 genes associated with coat colour have been identified [7], the pleiotropic effects of the mutations leading to variable coat colours have mainly been investigated in humans and mice (see contribution by Reissmann and Ludwig in this issue). Genetic mutations that lead to coat colour variation and have few negative pleiotropic effects (including diseases) do not require a significant investment by humans to maintain them. In other words, as long as mutations in coat colour genes do not adversely affect the rest of the organism's ability to survive within a human niche, they require little active effort to sustain them. Given that some coat colour genes are expressed in a limited variety of tissues, the evidence suggests that mutations in many genes might not possess negative pleiotropic effects.

A recent example was demonstrated in chickens. Despite the fact that the Red junglefowl (the primary progenitor of domestic chickens) possesses grey skin (visible on the legs), the majority of domestic chickens possess yellow skin. By combining a linkage analysis and an identical-by-descent mapping approach across a wide range of breeds, Eriksson et al. [38] concluded that the gene BCDO2 was responsible for the yellow leg trait. Red junglefowl possess a version of this gene that is functional in several tissues and breaks down carotenoids, preventing their accumulation in the skin. The variant of this gene possessed by domestic chickens is functional in tissues including the liver, but is non-functional in the skin, where the build-up of carotenoids turns the skin yellow. A phylogenetic analysis demonstrated that the domestic allele

is more closely related to the gene typed in the Grey junglefowl, thus suggesting that domestic chickens acquired this gene through hybridisation with wild Grey junglefowl [38]. Because the yellow skin allele of BCDO2 functions normally in all other tissues, there are no negative consequences of possessing this colour variation and it requires very little effort on the part of humans to maintain high frequencies of the allele in domestic populations. The same is true of MC1R which has few, if any, pleiotropic effects since it is involved only in the pigmentation pathway [8].

Several other genes affecting coat colour are however linked to deleterious traits such as skin diseases, disorders of the eyes/ears and disturbed reproduction, (for an in-depth review of this see contribution by Reissmann and Ludwig in this issue). In these cases, the natural selection pressure against the deleterious traits would ordinarily reduce the frequency of the associated coat colour, thus requiring a more active effort by people to keep them in the population. For example, the greying with age trait in horses is an autosomal dominant trait. Horses that possess the underlying mutation (a duplication of STX17) responsible for this colour pattern also possess a higher incidence of melanoma [39].

In mice, as in other animals, the agouti protein affects pigmentation. This gene normally produces black hair by interfering with the ability of alpha-MSH to bind to MC1R, thus switching the melanin synthesis from eumelanin (black/brown) to pheomelanin (red/yellow). A number of dominant mutations in the non-coding region of this gene not only produce yellow coats, they also possess several pleiotropic consequences including obesity, type II diabetes and tumourgenesis [40]. Negative pleiotropic effects have also been identified in dogs. In Collies for example, a mutation in AP3 produces a diluted, grey coat colour, and causes cyclic neutropenia. In Dalmatians, polymorphisms in the MITF gene have been associated with both white spotting and deafness [10].

In pigs, up to three copies of the KIT locus were found in commercial pig breeds, and a splice mutation was found in up to two of these copies. These combinations are likely the result of unequal crossing over during meiosis. Without the aid of humans who are persistently selecting for the white colour that results from possessing the KIT variation, unequal crossing over would lead to single copy alleles lacking the splice mutation (especially since a single copy homozygous for the mutation is lethal) that will then increase in frequency since wild type pigmentation provides better camouflage and protection from UV radiation [9].

#### 2.4. Role of humans in encouraging and sustaining coat colours

The argument that humans have played a significant role in the establishment and maintenance of coat colour variation domestic animals is bolstered by the demonstration that humans have expressed a penchant for novel colours in other contexts. A recent investigation into the distribution of a Pacific island tree snail is an interesting example. The snail, *Partula hyaline*, possesses a variety of different colours and is found across the Pacific, though none of the colour variants have been transported outside of their natural range. The white variety, endemic to Tahiti, was the only variant transported by prehistoric traders between archipelagos, thus suggesting that the white snails possessed significantly greater aesthetic value for use in jewellery [41].

An innate preference for white variants appears to be a universal human trait. A recent study identified two genes that produce white grapes. Though red grape varieties possess significant genetic variation, this study also concluded that all extant white grape varieties possess the same mutations and thus a single origin, suggesting that humans were responsible for the substantial geographic proliferation of white grapes [42]. All of the wild progenitors of domesticated rice are red but the vast majority of domestic strains are white [43]. News reports often call

attention to rare white deer, white humpback and killer whales, and white tigers are popular attractions worldwide. In addition, white elephants are considered sacred in Southeast Asia and white horses have been prized for at least the last 2500 years [39].

Black animals have proved no less attractive. In domestic pigs, breeds that possess black coats are found in both Europe and East Asia. Not only are the causative MC1R mutations different between these breeds, the MC1R alleles are derived from separate wild type alleles found in wild boar from Eastern and Western Eurasia [32]. The fact that black coats were arrived upon through separate molecular mechanisms suggests that farmers independently selected for black coats across the Old World and that regardless of their historical or cultural context, humans were attracted to black pigs.

### 3. Conclusion

Given the number of genes that have been identified that can easily manipulate coat colour, and how few of them have so far been shown to possess negative pleiotropic effects, it is ironic that most of the wild progenitors of domestic animals exhibit either uniform coat colours or possess a limited number of variants. Numerous genetic mechanisms can be employed to rapidly alter coat colour even if natural selection actively suppresses the variants when they appear. The ease with which novel coat colours appear and can be selected for at little cost to the rest of the organism is taken advantage of by people who often possess a preference for the same variation that nature abhors. In this way, humans have satisfied their urge for novelty by selecting for coat colours at the expense of the survivability of domestic animals in the wild, and human manipulation of domestic animal reproduction has generated and sustained coat colours that would not have been possible in wild contexts.

A relatively small proportion of domestic animals most likely followed the commensal pathway [4]. There is not yet any evidence linking genes associated with tameness to coat colour variation [27], and many animals that followed other domestication pathways that did not require an initial, unintentional taming phase also possess coat colour variation using the same genetic mechanisms. As a result, it appears that non-wildtype coat colours appeared soon after humans began manipulating the reproduction of domestic animals and novel coat colours were not linked to selection for other traits.

The evidence from modern and ancient DNA studies of pigs and horses also suggests that coat colour changes occurred early during the domestication process and that humans actively encouraged the proliferation of novel coat colour alleles on top of which new mutations, and even more novelty became apparent. The extensive number of coat colour variants present even in those animals domesticated in the past 150 years including Syrian hamsters and Chinchillas also suggests that once in captivity, coat colour variants appear rapidly. The fact that some of the foxes in Belyaev's experiment that were not bred for tameness also expressed piebald coats suggests that coat colour variants can appear before the application of a strong behavioural selection pressure [3]. Additional ancient DNA studies of domestic animals will be able to test this hypothesis and characterise the rise in coat colour variability through time.

Lastly, it is clear that without humans, even the most basic coat colour variants would not persist in the wild for more than a generation. Though feral animals often retain other behavioural and physiological signatures of their domestication (including smaller overall brain size [44]), they display very little (if any) of the coat colour variation found in their domestic cousins. The overall impression is that the coat colour variability encouraged and maintained by humans in domestic plants and animals is the result of a selection pressure that is diametrically opposed to the one nature imparts on wild progenitors.

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