Selection for a behavior, and the phenotypic traits that follow

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For over 50 years in a Russian experiment, foxes have been selected for one trait—tameness. The results have been fascinating. A population has resulted that is as tame as domestic dogs. These changes have been associated with changes in the neuroendocrine system, including lower levels of blood cortisol. Developmental changes have been seen as well. In addition to these traits seen throughout the population, other changes have appeared at a rate higher than would be expected by mutation alone. The most notable example is a white spotting pattern which often results in a star on the fox's face. The types of changes seen in the domestic fox population are remarkably similar to changes seen in many other domestic animals. This pattern, where a whole series of phenotypic changes have occurred in response to selection for one behavioral trait, may provide novel insights into rapid species diversification.

Just over 50 years ago a fascinating long-term project was initiated by Dmitry K. Belyaev. In this breeding experiment silver foxes (*Vulpes vulpes*)[1] were selected solely on their behavior towards humans. Initially the foxes were acquired from the commercial fur farming industry, which had been established about 50 years prior. This provided foxes that were a bit tamer than if they had been captured directly from the wild. The foxes were housed in cages. According to a specific protocol, the foxes were exposed to a human attempting to hand feed and touch or pet them. The foxes were evaluated based on their responses and only the tamest were bred to develop a population of domesticated foxes.

The experiment continues today at the Institute of Cytology and Genetics, Siberian Branch of the Russian Academy of Sciences. Today the domesticated population of foxes exhibit dog-like behavior such as eagerly seeking human contact, tail wagging, whining, whimpering and licking. There are also a number of phenotypic traits that have arisen which are similar to traits seen in other domestic animals. Various details of these changes are not only interesting, but have important implications for the creation model.

Genetics and behavior

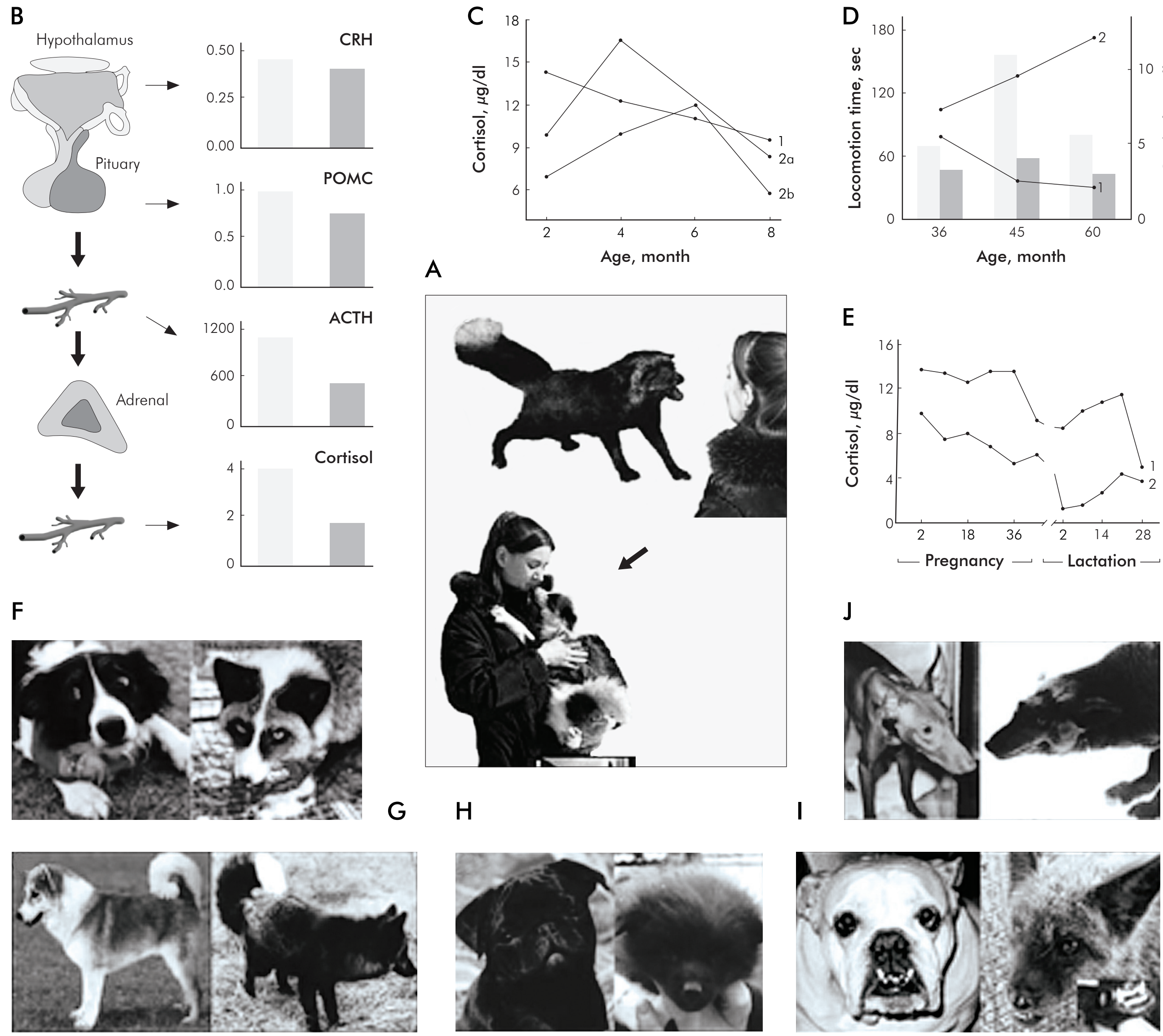
While behavior has a genetic component, it certainly isn't completely determined by genetics. One notable example is found in the practice at Thailand's Sriracha Zoo of cross-fostering tiger cubs to a sow. Not only do the tigers grow up calmer, but they are comfortable 'hanging out' with pigs.[2] Aware of this issue, efforts were made in the fox domestication study to determine the degree to which the behavior was heritable. In addition to the population of foxes bred for tameness, they maintained a separate population bred for aggressiveness. By cross-breeding, cross-fostering newborns, and even transferring embryos between foxes of different behavior, they determined that about 35 percent of the foxes' defense response was attributable to genetic factors.[3]

In an effort to keep the selection based on genetic differences, the foxes were not trained. Instead, there was limited, timed contact with humans at specific ages for the purpose of evaluating behavior. By the sixth generation some foxes appeared that not only tolerated human contact, but actively sought it. Through continued selective breeding, this behavior has become characteristic of the entire domesticated fox population.[4] Recently, two loci on fox chromosome 12 (VVU12) were identified as being associated with domesticated behavior.[5] While specific genes and their function have yet to be identified, it is interesting to note that this region corresponds to one of the major loci identified in dogs (*Canis familiaris* or *Canis lupus familiaris*), where a signature for positive selection exists in the domestication of dogs from wolves (*Canis lupus*).[6]

Neuroendocrine changes associated with behavioral changes

Cortisol is an important glucocorticoid produced by the adrenal glands. Blood levels of this hormone rise in response to stress. In the tenth generation comparisons began of blood cortisol levels between domesticated foxes and their farm-bred counterparts. At this point the domesticated foxes already had significantly lower values year round. This trend has continued with continued selection. In generation 45 both the basal and stress-induced blood cortisol levels were three to five-fold lower in the domesticated foxes than in farm-bred foxes. It was also observed that blood glucocorticoid values were lower in the blood of pregnant and lactating domestic foxes (figure 1E).

Production of cortisol is regulated by the hypothalamic-pituitary-adrenal (HPA) axis. The hypothalamus in the brain responds to various signals, including blood



A: Transformation of fox behavior resulting from selection for tameability.

Top: a fox of the farm-bred population unselected for behavior. Foxes of this population show the typical aggressive response to human.

Bottom: a fox of the experimental domesticated population. The dog-like behavior of foxes of this population is the result of many years of selection for tameability.

B–E: Activity of the HPA axis in farm-bred (light bars and/or '1') and domesticated foxes (dark bars and/or '2').

B: Hypothalamic CRF (CRFmRNA/18SmRNA) and pituitary POMC (POMCmRNA/18SmRNA) gene expression, ACTH (pg/ml) and cortisol (μg/dl) level in farm-bred and domesticated foxes.

C: Age-related changes in plasma cortisol level in farm-bred and domesticated foxes: **(1)** farm-bred foxes with aggressive response to human; foxes of the domesticated population with low **(2a)**, and high **(2b)** domestication scores.

D: Total time of locomotion, an indicator of exploratory behavior, and plasma cortisol level in farm-bred and domesticated foxes at the age of 1-2 months: locomotion is plotted on the graph; plasma cortisol level is represented as bars.

E: Plasma cortisol in silver foxes during pregnancy and lactation.

F–J: Dog-like morphological changes arisen in foxes of the domesticated population:

F: Similarity of coat depigmentation between dogs and foxes: **Left**, a border collie; **Right**, a tame fox.

G: Tail carriage, curly tail: **Left**, an Islandsk Farehund; **Right**, a tame fox.

H: Ears are floppy and face skull is widened in some pups of tame foxes: **Left**, a pug; **Right**, a tame fox pup.

I: Long jaw (elongation of the lower jaw) in the English bulldog occurs among tame foxes: **Left**, an English bulldog; **Right**, a tame fox.

J: Elongation of face skull in certain dog breeds and tame foxes: **Left,** a Pharaoh hound, **Right**, a tame fox.

Figure 1. A summary of the results of the fox domestication experiment (figure 4 from Trut *et al*.[4]).

cortisol levels, and releases corticotrophin-releasing hormone (CRH) to signal an increase in glucocorticoid production. This hormone acts on the pituitary to increase the expression of the proopiomelanocortin (POMC) gene. Depending on the cleavage of the resultant peptide, a number of different products may result including adrenocorticotropic hormone (ACTH) and β-endorphin. It is ACTH that signals the adrenals to increase production of cortisol. In foxes selected for domestication, the levels of all these (CRH, POMC, and ACTH) tended to be lower (figure 1B).

In addition to changes in the HPA axis, changes were noted in the serotonergic system in the brain. Domesticated foxes had higher levels of serotonin, a neurotransmitter, and its main metabolite (5-hydoxyindol acetic acid) in the brain than farm-bred foxes. Differences were also demonstrated in the activity of a key enzyme degrading serotonin (monoamine oxidase) as well as the primary enzyme involved in its synthesis (tryptophan hydoxylase).[4] The higher levels of serotonin are believed to inhibit aggressiveness.[7]

Developmental changes

Important developmental changes are evident in the neonatal period in domesticated foxes compared to non-domesticated foxes. It is recognized that in dogs the first weeks in life are important in developing social bonds with people. This window of time begins when pups start to sense and explore their surroundings and ends when they begin to fear unknown stimuli. The same appears to be the case in foxes, and domestication significantly widens the window (figure 1D). In the experiment, tame foxes responded to sound an average of two days earlier (14 days after birth vs 16 days) and opened their eyes about a day earlier (17–18 days vs 18–19 days) than other foxes. Similarly, they didn't show the fear response until three or more weeks after it appears in the untamed foxes (week 9 or later vs week 6).

In domestic foxes the fear response to unknown stimuli was correlated with an increase in plasma cortisol, which rises sharply at 2 to 4 months of age and then tapers off to adult levels by 8 months (figure 1C). Foxes further along in the domestication processes experienced the surge in cortisol and the fear response later.[3]

Reproductive changes

Foxes, like many wild animals, are seasonal breeders. The mating season occurs early in the year in response to increasing daylight. Fur farmers have attempted for decades to extend the breeding season, but to no avail since there is no significant variation to select from. For the most part, any variation in breeding is due to environmental factors (day length) rather than genetic ones. This is common in many mammals from middle latitudes.

It seems ironic that breeding for tameness has produced foxes which are able to mate outside the normal breeding season. In fact, a few have mated twice a year. A similar loss of seasonal breeding pattern is apparent in most domestic animals (e.g. dogs, cats, pigs, horses, cattle, etc.). The domesticated foxes also reach sexual maturity about a month earlier and have litters that average one pup more than the farm-bred foxes.[4,8]

A recurring star

A white spotting pattern, referred to as Star spotting, arose de novo numerous times in the tame population of foxes (about 124/1000). While much rarer, it also arose in the farm-bred populations (71/10,000) at a rate several orders of magnitude higher than would be expected by random mutation.[4] This trait appears to be inherited in a semi-dominant fashion. The locus has not been identified molecularly, but the allele causing the trait is designated as the *Star* (S) allele. The white patch on the head can vary in size, and when it appears as a well-defined star, it is associated with white spots on the lower jaw, breast, and belly.

There is a distinct homozygous phenotype which involves more extensive white coloration, including a blaze that extends down from between the ears and spreads out along the nose, and extensive white patches that form a collar or a belt around the animal (figure 1F). These animals have heterochromatic irises and hearing is affected, sometimes to the point of deafness. It is characteristic of homozygotes to twist their heads backwards occasionally, a behavior associated with pathology of the vestibular apparatus. Cryptorchidism is frequent, and when it occurs bilaterally, it results in sterility.[9] These pathological signs are clearly associated with the homozygous phenotype and are not likely to be the result of inbreeding depression as experimental design kept the inbreeding coefficients low (0.02–0.07).[4]

For Star spotting, there are times when it appears epigenetic factors play a role in expression of this gene. For example, when the *Star* allele (S) was inherited from the vixen (female fox), the offspring followed the pattern expected for a semi-dominant trait. Approximately half the offspring exhibited the heterozygous (Ss) Star phenotype. However, when the *Star* allele came from the male, there was a statistically significant shortage of the Star phenotype, suggesting some sort of maternal effect on inheritance.

When the offspring of a heterozygous male were examined in detail, some surprising results were observed. When the vixen was tame, there was a normal pattern of inheritance observed in the offspring. It was only when the vixen was unselected for tameness (relatively wild) that the statistically significant lack of Star offspring was observed.[9] Could the differences in the neuroendocrine system affect development in such a way that it affects this phenotype? There is some evidence suggesting this may be the case. The rate of development in pigment cell precursors (melanoblasts) was shown to be correlated to the glucocorticoid status during development.[10] Alternatively, could this be from an epigenetic silencing (e.g. via methylation) of the mutant allele in certain instances?

It was shown that the Star phenotype is the result of delayed proliferation and migration of melanoblasts in early embryonic development.[11] Thus, white areas lack melanocytes (pigment cells) due to this altered timing. It is tempting to postulate that a *KIT* mutation (either coding or regulatory) may underlie the Star phenotype. This is a common locus for mutation affecting white spotting patterns in other domestic animals, and pathologic symptoms are more common in homozygotes.[12] There are instances where *KIT* mutations are associated with hearing loss. This is associated with a lack of melanocytes in the cochlea of the inner ear. Pigmentation in the vestibular region is also affected, though it does not appear that signs of vestibular pathology, such as turning the head back seen in fox homozygotes, are a known effect of *KIT* mutations.[13] Neither does *KIT* appear to be associated with heterochromatic irises or cryptorchidism in other species.

There is another oddity in the Star inheritance pattern which initially seems to be incompatible with a *KIT* mutation. When heterozygotes (Ss) are mated, there is a significant shortage of the homozygote (SS) phenotype. Further experiments ruled out embryonic mortality and differential death of gametes as the cause. It was found that the heterozygote-to-standard phenotype was 3:1, strongly suggesting that genotypic homozygotes were expressing a heterozygous phenotype. This, along with other pedigree-derived information, provides evidence that the gene can be inactivated in a heritable fashion.[9]

It was suggested that the unusual patterns in the appearance of the Star phenotype and its inactivation might be from the activation of a silent gene followed by subsequent inactivation. This would help explain why the Star phenotype has arisen more often than expected by random mutation; the gene already is there and it is just a matter of whether or not it is expressed.[9] Initially it would seem this pattern would exclude a *KIT* mutation as causative since a functional copy of *KIT* is essential for the migration of multiple cell lines during embryogenesis. However, these foxes carry supernumerary B chromosomes, and these B chromosomes just happen to carry one intact gene, *KIT*.[14] Thus, most foxes would have more than the normal two alleles for *KIT*, since an extra allele occurs on each B chromosome. This would allow for the possibility that one or more mutant alleles could be silenced without losing *KIT* function entirely. Alternatively, gene conversion reversing the mutation is a possibility.

As engaging as it is to speculate on a potential role for B chromosomes, there are other genes involved in the same pathway as *KIT* that can influence embryonic melanoblast migration and cause white spotting phenotypes.[15] Hopefully, the underlying molecular genetics of the intriguing patterns of the Star phenotype will be elucidated in the near future.

The domestic phenotype

Many of the traits that have arisen in the tame foxes parallel changes seen in other domestic animals. Reproductive changes and the frequent appearance of coat color variations are just the beginning.[4,16] In addition, some foxes will carry their tail in a high, curled position; a trait observed in some dogs, cats, and pigs (figure 1G). Floppy ears, widened facial bones, elongation of the muzzle and of the lower jaw in particular have also been observed (figure 1H–J).

Some of the patterns observed in domestic animals are considered examples of neoteny or pedomorphism because they involve juvenile traits that are retained in adulthood. For example, floppy ears are seen in newborn fox pups, but not normally in adults except for some of the tame ones. Additionally, many of the behavioral traits in the tame foxes seem to be characteristic of younger animals. In contrast to this apparent delay in timing of phenotypic and behavioral traits, reproductive maturity is sped up.

It is a challenge to neo-Darwinism (as popularly taught) that domestication of animals from such diverse taxa would follow such a similar path. If evolution is driven by random mutations and natural selection, how could creatures have retained the same underlying structure over millions of years of evolution so that coordinated changes could be made, allowing them to favorably respond to domestication in such a similar way? Further, how could

Table 1. A summary of increased variety that often appears in domestic species (after Trut[3]).

Increased variation in size (appearance of dwarf and giant varieties)	horses (pony and draft horses), chickens (bantam breeds), dogs, pigs, cattle, sheep, goats
floppy ears	dogs, cats (Scottish fold), pigs (e.g. Landrace), sheep (e.g. Awassi), goats (e.g. Nubian)
piebald (white spotting) coloration	all
increased variation hair coat (e.g. wavy or curly hair)	sheep, poodles, donkeys, horses, pigs, goats, mice, guinea pigs
shortened tails (fewer vertebrae)	dogs, cats, sheep
reproductive changes	all

random mutation and natural selection create the proper interplay of genetic and endocrine systems to allow for domestication to occur in any animal to begin with?

A second problem is the increase in variability that was seen as a result of selection for tameness. In neo-Darwinism, random mutation and natural selection are supposed to work independently. Why did selection seem to increase variability rather than decrease it (table 1)?

Neo-Darwinism is confronted with a neo-Lamarckian view

The researchers involved in the fox domestication experiment explain this pattern by 'destabilizing selection'.[17] They define this as selection, which accelerates 'evolutionary changes' through changes in the regulatory systems that affect development. So, selection for tameness is seen to have affected the neuroendocrine system, which is known to be able to affect development (ontogeny). These changes presumably can alter genetic and heritable epigenetic information. Thus, in a sense selection becomes a sort of mutagen as it induces changes and increases variability.

In a 1998 paper by Lyudmila Trut, who heads the research on the foxes, the concept of destabilizing selection is argued to now be the *status quo*.[18] In addition to the findings in foxes, she cites research done on bacteria and *Drosophila* to support this idea. She argues that the new variation tends to be in the direction of selection. Further, she points to a study in sticklebacks suggesting that a phenotypic trait was not the result of direct selection, but a side effect of selection for a related behavior.[19] These concepts certainly appear in the literature.[20] However, use of the term 'destabilizing selection' for this seems to be largely confined to the Russian literature.[21]

The main point here is that actual evolutionary researchers are not confining their thinking to the neo-Darwinian mechanisms most commonly promoted to the public. Many recognize 'pathways' by which significant directional changes can take place. Obviously, the vague term evolution is applied to these changes and the researchers likely remain committed to the evolutionary model for origins. However, much of the data is from within created kinds. Such observationally based research has always proved tremendously valuable to creationists.

Creationist implications

Creationists have recognized that the environment can sometimes induce changes. For example, adaptive mutations[22] in bacteria have been observed in response to environmental challenges.[23] I have proposed that similar adaptive changes can explain some examples of pesticide resistance in insects.[24] I have referred to these types of mutations as providential because they cannot be logically attributed to just 'copying errors' in a system that arose by random processes.[25] Instead, they can be interpreted as evidence of a God who cares for His creation even in its current fallen state.

Additionally, creationists are certainly aware that mutations can be pleiotropic, due to the fact that genes and their products are part of highly complex interrelated networks. What I have not noticed is much discussion of the concept that God designed many of these networks so that, in response to environmental clues, a *series* of potentially adaptive changes could appear. That would be some impressive engineering!

If this is so, it may explain certain patterns we see in nature. For example, weasels, foxes, hares, and lemmings are presumably all from different created kinds. Each has species adapted to the Arctic climate which exhibit a seasonal white hair coat. Did this arise merely through selection of previously existing alleles? Unlikely. Were the various cold adaptive features produced through random mutation followed by natural selection? Again, very unlikely. However, if environmental stress can induce an increase in variability which is potentially adaptive, then we have an excellent explanation for how these animals became so well adapted to their environments relatively soon after the Flood and for why so many of the changes are similar.

These results also have potential implications for proposed scenarios about how natural selection operates. Notice that reproductive changes did not come about with selection for reproduction, but instead selection for behavior. This means natural selection, as it is currently understood, may have little to do with the types of changes we observe. What if some animals exhibit the behavior that they will tolerate a new niche? What if this behavior plus environmental stresses trigger a series of physiological changes that end up translating into genetic changes? This would suggest that animals were designed with the ability to adapt to new environments genetically, consistent with God's intention that they fill the earth so it will be inhabited (Genesis 1:22; 8:17; Isaiah 45:18). This pattern would fit incredibly well in the creation model. Adaptation is expected in the creation model and a testimony to the awesome Designer and Sustainer of Life!

Caveats and conclusions

While there is plenty of evidence that something interesting is going on that is inconsistent with the popular version of neo-Darwinism, there are still numerous details that are unknown. For example, the molecular basis of the Star phenotype has yet to be elucidated. Was it the result of mutation, perhaps via some variation-inducing genetic elements?[26] If so, it is more evidence that some evolutionary ideas about mutation rates need to be reassessed.[27] In what way might neuroendocrine or resulting developmental changes affect the DNA sequence or epigenetic status of genes? Are there any maternal chemical signals that could facilitate epigenetic or genetic changes in the developing embryo?

The answers to some of these questions may be buried in the current scientific literature. Others are likely to appear in the coming years. One thing is clear, the neo-Darwinian mechanisms of random mutation and natural selection do not adequately account for the diversity that currently exists within created kinds. Instead, current research findings continue to point to an awesome Creator who cares for His creatures.

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