

## GENETIC BASIS OF COAT COLOUR INHERITANCE IN FERRETS (*MUSTELA PUTORIUS FURO*): PEDIGREE ANALYSIS

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### ABSTRACT

Apart from fur farming, the popularity of a ferret also as a domestic animal has been increasing since the 1980s. Years of breeding work, initially on farms and then continued by amateur breeders, allowed for obtaining multiple colour variations. A uniform classification of ferret coat colour types has not been developed so far, thus it may vary depending on a country or a breeding society. This study aims to determine the genotype responsible for coat colour in ferrets and the way in which it is inherited, based on the corresponding knowledge for various mammalian species and a pedigree analysis of 201 pups from two ferretries (one Polish and one Italian) from 2009–2017. Altogether, the pedigrees contained 354 specimens (178 females and 176 males). The analysis used the Polish ferretty as the main data source, and the results were checked using the Italian ferretty. The following genes were assumed to be responsible for coat colour in ferrets: *ASIP* for agouti, *TYRP1* for brown, *MC1R* for extension, *TYR* for albino, *MYO5A* for dilution, *KIT* for dominant white, *Rn* for roan, *STX17* for progressive greying, and *EDNRB* for piebald spotting. Presented are simplified genotypes and the rules of inheritance of individual colours, taking into account basic colour (including colour uniformly white), a colour concentration pattern, and white markings. The results for white markings were ambiguous, so only hypothetical suggestions of genotypes were given.

**Key words:** *Mustelidae*; fur colour; coat pattern; genotype

### INTRODUCTION

Animal coat colour depends on the presence and content of melanin in the skin and hair, where two melanin types can occur – alone or together – namely eumelanin and pheomelanin [Hoekstra 2006, Cieslak et al. 2011, Ata and Majewski 2013, Rzepka et al. 2016]. Melanin is formed from tyrosine and cysteine in the catalysis process in melanosomes, tyrosinase being the main enzyme involved in this process: its activity is essential in the melanogenesis process. Equally important are two other proteins: Tyrosinase-related protein 1 (*TRP1*, *TYRP1*) and Tyrosinase-related protein 2 (*TRP2*, *TYRP2*, *DCT*) [Otręba et al. 2012, Ata and Majewski 2013, Rzepka et al. 2016]. Melanocytes can synthesise both types of melanin, but not at the same time. The production of eumelanin by melanocytes depends on melanotropin (the hormone  $\alpha$ MSH), secreted by the pituitary gland.  $\alpha$ MSH is bound by surface melanocyte receptors, leading to the activation of adenylyl cyclase, an enzyme stimulating melanocy-

tes to produce eumelanin. In the absence of the  $\alpha$ MSH-dependent signal and the surface receptors, melanocytes produce pheomelanin [Kuzniewicz and Filistowicz 1999, Hoekstra 2006, Rzepka et al. 2016]. The basic colour of an animal coat is determined by the ratio of eumelanin to pheomelanin, the ratio being controlled mainly by the agouti signalling protein *ASIP* and the melanocortin 1 receptor *MC1R* gene [Hoekstra 2006, Cieslak et al. 2011].

Many genes play important roles at various stages of melanogenesis. Some loci affect the differentiation and migration of neural crest cells, and others affect melanocytes' morphology or ability to locate melanosomes in the hair and skin. Another group of loci directly affects enzyme activity and protein compounds responsible for the melanogenesis process. Certain genes have alleles that affect the binding of  $\alpha$ MSH to melanocytes. All the above-mentioned loci affect the final phenotype of an individual [Ruvinsky and Sampson 2001, Hoekstra 2006].

Coat colours are classified primarily based on the phenotype, a problematic approach for several reasons.

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Different alleles of one gene may result in different phenotypes, and similar phenotypes may be caused by changes in the alleles of different genes. For example, white coat can be due to the action of several genes, namely, *KIT*, *EDNRB* (both causing leucism), *TYR* (albino locus), and *STX17* (progressive greying) [Cieslak et al. 2011]. The analysis of the biochemical process of creating coat colour should help to determine genes responsible for this process.

This study aimed to determine (i) the genotype responsible for coat colour in ferrets (*M. putorius furo*) and (ii) the way in which coat colour – including basic colour, a colour concentration pattern, and white markings – is inherited. With these aims, a pedigree analysis of ferret pups born in two small ferretries was conducted. The research also aimed to draw attention to the importance of correct classification of colour coat, one that would have the genetic basis, not only the phenotypic one.

## MATERIAL AND METHODS

The research included the pedigrees of 201 ferrets born in 2009–2017 in two small ferretries, Ferretta Passion (Poland) and Ferret Vendetta (Italy), covering altogether 354 specimens (178 females and 176 males in the pre-reproductive and reproductive age) for whom it was possible to determine basic coat colour, a concentration pattern, and white markings. The ferrets from both ferretries were related, there were two pairs of full siblings (one of each pair was in either of the ferretries). Additionally 22 ferrets appeared in the pedigrees from both ferretries. Breeding lines and repeated matings both ferretries carried out allowed for accurate analysis and increased the reproducibility of the research. The classification of colour variations used was based mainly on the [Associazione Italiana Furetti \[2017\]](#) (AIF) and the [American Ferret Association \[2017\]](#) (AFA).

The pedigrees originated from private breeders' resources and the international Internet database "Feritage – ferret database system" [Feritage 2017], a ferret pedigree database supervised by Marit Nybakken. Unfortunately, the database still uses some obsolete names and lacks some of the colours recognised by AFA and AIF and used by breeders. Therefore, the database was adjusted based on photo documentation and consultations with breeders.

Ferretta Passion (Poland) was used for modelling the genotypes, while Ferret Vendetta (Italy) for testing the genotypes modelled that way. To determine the genotype for individual basic colours, patterns, white markings, and uniformly white coats, nine genes were used that form the basis for shaping different types of coat colour: *ASIP* for agouti (*A*) [Ruvinsky and Sampson 2001, Kerns et al. 2003, Hoekstra 2006, Fontanesi et al. 2010, Cieslak et al. 2011], *TYRP1* for brown (*b*) [Nes et al. 1988,

Bednarz and Frindt 1991, Kuźniewicz and Filistowicz 1999, Ruvinsky and Sampson 2001, Kerns et al. 2003, Schmidt-Küntzel et al. 2005, Cieslak et al. 2011], *MC1R* for extension (*e*) [Ruvinsky and Sampson 2001, Eizirik et al. 2003, Hoekstra 2006, Fontanesi et al. 2010, Cieslak et al. 2011, Fontanesi and Russo 2013], *TYR* for albino (*c*) [Ruvinsky and Sampson 2001, Lyons et al. 2005, Błaszczyk et al. 2007, Benkel et al. 2009, Cieslak et al. 2011], *MYO5A* for dilution (*d*) [Ruvinsky and Sampson 2001, Webb and Cullen 2010, Cieslak et al. 2011], *KIT* for dominant white (*W*) [Cooper et al. 2005, Webb and Cullen 2010, Cieslak et al. 2011, Strain 2011, Piazza et al. 2014], *Roan* for roan (*Rn*) [Cieslak et al. 2011, Wilisowska 2014a], *STX17* for progressive greying (*G*) [Cieslak et al. 2011, Wilisowska 2014a], and *EDNRB* for piebald spotting (*S*) [Bennet and Lamoreux 2003, Cooper et al. 2005, Webb and Cullen 2010, Cieslak et al. 2011, Strain 2011, Piazza et al. 2014, Wilisowska 2014b].

First, genotypes were established based on the assumptions presented in scientific literature on coat colour inheritance. Then, these results were adjusted so as to obtain correct genotypes that would agree with the colours obtained in the subsequent generations. The analysis included litters born in 2009–2017, older ones analysed first. The research used birth certificates going back three generations.

Based on the pedigrees of the Ferretta Passion ferrets, genotypes were established for six basic coat colours (black, black-sable, chocolate, champagne, and cinnamon) in each of the five colour patterns (self, solid, standard, point/Siamese, and roan); for white, DEW (Dark Eyed White), and albino coats; and for white markings (striped, panda, badger/blaze, milk mouth, and mitt). The compositions received were then tested for their correctness based on the pedigrees from the Ferret Vendetta ferrety.

Because 26 individuals occurred in the pedigrees from both ferretries, some pedigrees of the Polish (base) ferrety were adjusted based on the results from the Italian (test) ferrety.

## RESULTS AND DISCUSSION

Tables 1 and 2 present the final representations of the proposed genotypes responsible for various coat colours in ferrets. The concept of the *ASIP* gene was mainly based on coat colour inheritance in dogs. Here, the  $A^S$  allele was proposed as the one causing the solid pattern, but it was placed lower in the dominance series than was the *A* allele (of wild type). Proposed as causing the self-colour pattern, the  $A^a$  allele was classified as recessive in relation to the other two alleles. Since ferrets with the self pattern do not have to have a uniform colour (they can have white spots around their ears and on the muzzle), the self pattern was not assigned to the non-agouti *a* allele. Individuals

**Table 1.** A simplified form of the ferret coat genotype for the six basic colours in each of the five colour concentration patterns.

**Tabela 1.** Forma uproszczona genotypu umaszczeń dla sześciu barw podstawowych w każdej z pięciu koncentracji barwy

Coat colour/ Concentration pattern Barwa/ Koncentracja koloru	Sable	Black-sable	Black	Chocolate	Champagne	Cinnamon
Standard	A <sub>-</sub> B <sub>-</sub> E <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sub>-</sub> B <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sub>-</sub> B <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sub>-</sub> b <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>s</sup> <sub>-</sub> b <sup>c</sup> <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sub>-</sub> B <sub>-</sub> ee C <sub>-</sub> D <sub>-</sub>
Solid	A <sup>s</sup> <sub>-</sub> B <sub>-</sub> E <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>s</sup> <sub>-</sub> B <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>s</sup> <sub>-</sub> B <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>s</sup> <sub>-</sub> b <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>s</sup> <sub>-</sub> b <sup>c</sup> <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>s</sup> <sub>-</sub> B <sub>-</sub> ee C <sub>-</sub> D <sub>-</sub>
Self	A <sup>a</sup> <sub>-</sub> B <sub>-</sub> E <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>a</sup> <sub>-</sub> B <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>a</sup> <sub>-</sub> B <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>a</sup> <sub>-</sub> b <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>a</sup> <sub>-</sub> b <sup>c</sup> <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub>	A <sup>a</sup> <sub>-</sub> B <sub>-</sub> ee C <sub>-</sub> D <sub>-</sub>
Point (Siamese)	A <sub>-</sub> B <sub>-</sub> E <sub>-</sub> c <sup>s</sup> <sub>-</sub> D <sub>-</sub>	A <sub>-</sub> B <sub>-</sub> E <sup>D</sup> <sub>-</sub> c <sup>s</sup> <sub>-</sub> D <sub>-</sub>	A <sub>-</sub> B <sub>-</sub> E <sup>D</sup> <sub>-</sub> c <sup>s</sup> <sub>-</sub> D <sub>-</sub>	A <sub>-</sub> b <sub>-</sub> E <sup>D</sup> <sub>-</sub> c <sup>s</sup> <sub>-</sub> D <sub>-</sub>	A <sub>-</sub> b <sup>c</sup> <sub>-</sub> E <sup>D</sup> <sub>-</sub> c <sup>s</sup> <sub>-</sub> D <sub>-</sub>	A <sub>-</sub> B <sub>-</sub> ee c <sup>s</sup> <sub>-</sub> D <sub>-</sub>
Roan (Silver)	A <sub>-</sub> B <sub>-</sub> E <sub>-</sub> C <sub>-</sub> D <sub>-</sub> Rn <sub>-</sub> __Ss	A <sub>-</sub> B <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub> Rn <sub>-</sub> __Ss	A <sub>-</sub> B <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub> Rn <sub>-</sub> __Ss	A <sub>-</sub> b <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub> Rn <sub>-</sub> __Ss	A <sub>-</sub> b <sup>c</sup> <sub>-</sub> E <sup>D</sup> <sub>-</sub> C <sub>-</sub> D <sub>-</sub> Rn <sub>-</sub> __Ss	A <sub>-</sub> B <sub>-</sub> eeC <sub>-</sub> D <sub>-</sub> Rn <sub>-</sub> __Ss

Underscore “\_” means it irrelevant whether the allele is of the same or lower dominance in the series; for the roan pattern, there are two additional places in case of the appearance of the progressive greying gene (G).

Symbol „\_” oznacza dowolność występowania alleli takich samych lub będących niżej w szeregu dominacji, natomiast w przypadku koncentracji roan dodatkowe dwa miejsca przeznaczone są na możliwość pojawienia się alleli genu progresywnego siwienia (G).

**Table 2.** The allelic series for the genes from Table 1

**Tabela 2.** Szereg alleliczny dla poszczególnych genów z Tabeli 1

Gene Gen	Agouti *	Brown *	Extension *	Albino *	Dilution *	Dominant White */†	Roan **	Progressive Greying **	Piebald spotting **
Alleles	A	B	E <sup>D</sup>	C	D	W	Rn	G	S
Allele	A <sup>s</sup>	b	E	c <sup>s</sup>	d	w	m	g	s
	A <sup>a</sup>	b <sup>c</sup>	e	c					s <sup>m</sup>
	a‡								s <sup>b</sup>
									s <sup>p</sup>
									s <sup>s</sup>

At the top are the most dominant genes, at the bottom recessive ones; \* – complete dominance, \*\* – incomplete dominance, † – lethality in the dominant homozygous system, ‡ – lethality or semilethality in the recessive homozygous system – a hypothetical allele.

Na górze znajdują się geny najbardziej dominujące, natomiast na dole recesywne; \* – pełna dominacja, \*\* – niepełna dominacja, † – letalność w układzie homozygotycznym dominującym, ‡ – letalność/semiletalność lub w układzie homozygoty recesywnej – allel hipotetyczny.

with a uniform coat colour pattern (of both undercoat and topcoat hair) are most likely very rare, if not semilethal or even lethal. Due to the hypothetical existence of the a allele in ferrets, this pattern was omitted in the genotype.

The concept of a double recessive homozygote at the Brown (*TYRP 1*) and Dilution (*MYO5A*) loci had been taken up, but later on it was rejected in favour of expanding the allelic series at the Brown locus with the *b<sup>c</sup>* allele. Thus, since chocolate coat colour is determined in both *bb* and *bb<sup>c</sup>* systems, ferrets with a chocolate coat can have any second recessive allele. During the pedigree analysis, the designation “*b-*” was introduced into the genotype of individuals that carried one of the two recessive alleles of the Brown locus, because in most cases it was impossible to determine which version of the allele the offspring could inherit. The *B* allele was assigned as determining the appearance of a black pigment.

The *E<sup>D</sup>* allele (dominant black), determining the eumelanistic phenotype, was proposed to be responsible for black, black-sable, chocolate, and champagne colours in ferrets – because it was assumed that these colours are based on eumelanin. In addition, chocolate and champagne ferrets always had at least one parent who carried the *E<sup>D</sup>* gene. The colour cinnamon – as typically pheomelanistic and rarer than the other five – was assumed to be determined by the homozygous system of the recessive *e* allele, and the colour sable to be determined by the *E* allele.

At this stage of the research, the colours black and black-sable turned out to be genotypically indistinguishable. A possible reason was a frequent incorrect colour determination and the resulting assignment of the colour black to black-sable ferrets – a most likely reason being that the former is more desirable in domestic

breeding than is the latter. Ferrets of the self pattern are considered more attractive than those of the solid or standard patterns, so it is possible that the determination of the self and solid patterns inheritance was similarly problematic, with the solid pattern being incorrectly determined as self.

The suggested alleles for the Albino locus was *C*, determining the appearance of the pigment; *c<sup>s</sup>*, responsible for the Siamese pattern, in ferrets also called the point pattern; and *c*, determining the albino coat colour. The Himalayan coat colour, also known as Siamese, is common in the genetics of animal coat colours, occurring, among others, in mice, rats, guinea pigs (*c<sup>h</sup>*), cats, and American mink (*c<sup>s</sup>*). It is this commonness that suggested their use in the ferret genotype.

The Dilution gene (*MYO5A*) was proposed as one of the genes responsible for the colour champagne. The initial assumptions were considered incomplete, due to the unclear contribution of the Dilution gene, considered affecting both eumelanin and pheomelanin. In rare variations, recessive alleles can accumulate – but champagne ferrets are not rare, so they are debatable as a double recessive (*bbdd*) variation. Therefore, the concept of inheritance of this colour was modified. Despite the final exclusion of *MYO5A* (as a gene partly determining the colour champagne), it was not eliminated from the genotype – since it might affect coat colour in ferrets. Animal species in which colour dilution was noted were characterised by the light colour of the coat. Many variations of coat colour in ferrets are characterised by the presence of several shades of one colour, such as sable, chocolate, or champagne. This effect may be caused by colour lightening, but there are no premises to determine whether such lightening results from a single or a double recessive allele. In addition, the Dilution locus in dogs and cats is responsible for colours such as blue and cream, which have not been described in ferrets until now.

The Roan gene was chosen as the gene responsible for the roan pattern. Due to the large number of ferrets with this pattern, the theory adopted from the coat colour inheritance in horses that suggests lethality in the homozygous dominant *RnRn* system was rejected. It is also possible that homozygotes dominating in the Roan locus (*RnRn*) have more white hair in the coat than have heterozygotes (*Rnrn*), and that homozygous dominant in the *STX17* gene (*GG*) – which has been proposed as a gene responsible for progressive greying – grey faster than do heterozygotes (*Gg*). Such an arrangement would explain the large phenotypic divergence of the roan pattern and the differences in the rate and the method of greying (uniform over the whole body surface or partial ones, e.g., from the rump).

Presented below are simplified representations of the genotypes for the uniformly white coat colour and for white markings. The Dominant White gene (*KIT*) was chosen as a locus determining the uniformly white coat of Dark Eyed White (DEW) ferrets. That there are only a few uniformly white ferrets (excluding albinos) confirms the assumption that this colour may be determined by the Dominant White gene. This gene in the dominant homozygous system is lethal in species such as mice, horses, and foxes. The genes given in parentheses (in the representations below) were chosen as most likely to be linked to the genes responsible for the type of coat described. In the case of the colour Dark Eyed White (DEW), the Progressive Greying and Piebald spotting (recessive homozygote) were considered the linked genes. In addition, after the analyses, it was found that the *EDNRB* gene was most likely linked to the roan pattern and to the Progressive greying locus. All ferrets with the roan pattern had mitts, a type of white markings. In addition, many ferrets with the roan concentration pattern showed premature depigmentation of the coat:

- Albinos: \_\_\_\_\_*cc*\_\_\_\_\_
- DEW : \_\_\_\_\_*C*\_\_\_\_\_ *Ww* (*G\_ Ss\_/s\_s\_*)
- White markings:
  - mitt (arlekin): \_\_\_\_\_ *C*\_\_\_\_\_ (*G*) *Ss/s\_*
  - milk (milkmouth): \_\_\_\_\_ *C*\_\_\_\_\_ (*G\_*) *Ss<sup>m</sup>/s<sup>m</sup>\_*
  - blaze: \_\_\_\_\_ *C*\_\_\_\_\_ (*G\_*) *Ss<sup>b</sup>/s<sup>b</sup>\_*
  - panda: \_\_\_\_\_ *C*\_\_\_\_\_ (*G\_*) *Ss<sup>p</sup>/s<sup>p</sup>\_*
  - striped: \_\_\_\_\_ *C*\_\_\_\_\_ (*G\_*) *Ss<sup>s</sup>/s<sup>s</sup>\_*

The above allelic series for the Piebald spotting gene is theoretical, because in the practice of ferret breeding, individuals with white markings on their heads are not combined into breeding pairs. More frequent than other white markings, mitts can be assumed a dominant type of white markings. The allelic series for the spotting gene in ferrets was modelled on the Piebald spotting locus in dogs, characterised by an increase in the area of white spots on the body along with the greater recessiveness of the allele. In addition, as in ferrets, the piebald pattern in the head area and extremely large white spots in dogs are associated with congenital deafness. During the research, the assumption was tested that white markings are determined by the double recessive allele (*ss*) and the possibility of incomplete dominance (*Ss*). Both theories, however, showed similar numbers of errors (i.e., inconsistencies in inheritance) – the difference of two animals – which is why it was impossible to state which theory is correct.

The knowledge of the genetics of coat colour in ferrets is scarce, most of it being rudimentary information about hair colour inheritance, focused on the Albino and Brown loci. Back in 1993–1994, Fara Shimbo tried to systematise the genotypes of some coat colour variations as well as the knowledge of the inheritance of coat colour

in ferrets; Lewington [2007] described the effects of her work.

Shimbo's assumptions of the genetics of coat colour in ferrets, however, differed from those this research based upon. The first difference is the thesis about the gene determining the champagne coat. Shimbo assumed that this colour was determined by the  $c^p$  allele, which is recessive to the  $C$  allele (full expression of pigment) and dominant to the  $cc$  allele (albinism). According to Shimbo, the colour champagne is determined in ferrets in a similar way as the colour Palomino (the colour isabelline) in horses, that is, by the single  $C^{cr}$  allele. As described in the results section, in ferrets the colour champagne is most probably determined by the mutation in the Brown locus [Nes et al. 1988, Bednarz and Frindt 1991]. Against Shimbo's theory, in this research the colour champagne was present in the point (Siamese) concentration pattern, determined by the  $c^s$  allele [Lyons et al. 2005, Schmidt-Küntzel et al. 2005, Benkel et al. 2009]. Even though the theory assumes that the point pattern is determined by the  $i^p$  allele in locus  $I$ , which would be responsible for inhibiting or expressing the genes associated with colour, it provides neither the full form of the gene's abbreviation nor references to other species – for which reason it was impossible to identify the gene and learn the full spectrum of its activity.

Shimbo assumes only two coat colour variations in ferrets, namely standard and self. She claims that black self ferrets are a hybrid of ferrets and mink, but she does not explain whether she meant American mink (which does not interbreed with individuals from the *Mustela* family [Nes et al. 1988]) or European mink. Nonetheless, she underlines the fact that ferrets in the self pattern should have the same colour of topcoat and undercoat. According to Shimbo, the standard pattern is completely dominant, the same assumption as the one made in this research. The self pattern, though, is determined by the recessive  $a$  allele, the inheritance of which she describes as unclear. This research made similar assumptions about the inheritance of piebald spotting as those Shimbo made, describing them as analogous to “Irish spotting”, in ferrets referred to as “mitt”.

The theory of progressive greying presented in this paper disagrees with Shimbo's assumption that this trait is recessive, an assumption she made even though in dogs and horses (the two species with progressive greying she mentions) this trait is determined by the dominant allele. What is more, Shimbo uses the terms “progressive greying” and “roan” interchangeably. Another discrepancy occurs in the hypothesis regarding the roan pattern, for which Shimbo uses the term “silvermitt”. She classifies it as recessive, unlike in sabino horses, an example Shimbo actually used to form her theory. Horse nomenclature describes sabino as “false roan”, because only some sabino

horses have a mixture of coloured and white hairs. Horses with the sabino pattern have white markings on the heads, legs, and often the bellies, which move up the body, that is, from the limbs and the abdomen. The co-occurrence of the sabino and roan patterns can be due to the close location of the corresponding genes (*Sb1* responsible for sabino and *Rn* for roan) [Wilisowska 2014c], suggesting that Shimbo incorrectly determined the gene responsible for sabino.

## CONCLUSIONS

Correct determination of coat colour in ferrets will allow for more accurate verification of genes participating in shaping coat colour. Such knowledge would enable one to determine coat colour at both phenotypic and genotypic levels.

A ferretry using appropriate selection and keeping scrupulous breeding documentation has greater chances to breed more coloured variations, but also to eliminate diseases, many of which have a genetic basis, such as congenital deafness.

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## DZIEDZICZENIE BARWY OKRYWY WŁOSOWEJ U FRETEK (*MUSTELA PUTORIUS FURO*) NA PODSTAWIE ANALIZY RODOWODÓW

### STRESZCZENIE

Celem badań była próba ustalenia schematu dziedziczenia umaszczenia u fretki (*M. putorius furo*), czyli barwy podstawowej, koncentracji barwy oraz białych znaczeń lub ich braku na podstawie analizy rodowodów. Analizie poddano rodowody 201 szceniąt z dwóch hodowli (polskiej i włoskiej) z lat 2009–2017, które łącznie zawierały 348 osobników w rodowodach. Dla analizowanej populacji stwierdzono dominację umaszczeń o barwie podstawowej w kolorach czarnych i ciemnobrązowych nad brązami i beżami, a także tych posiadających miedziane refleksy. Podobny szereg dominacji odnotowano także w innych badaniach u tchórzy hodowlanych i nerek. Koncentracja barwy w typie standard dominowała nad pozostałymi czterema, natomiast najbardziej recesywna okazała się koncentracja w typie point. Również u większości gatunków ssaków koncentracja w „typie dzikim” dominuje nad wzorami jednolitymi oraz tymi odbiegającymi od umaszczenia występującego u form nieudomowionych, czyli w przypadku fretek tchórze europejskiego. Typ point (siamese) jest zbliżony fenotypowo do mutacji himalajskich, występujących m.in. u kotów, myszy, szczurów i królików, które także wykazywały charakter recesywny w stosunku do typu dzikiego. Nie można było jednoznacznie wykazać, czy brak białych znaczeń dominuje nad ich obecnością, ze względu na niewielkie różnice w liczbie urodzonych zwierząt w odniesieniu do posiadania znaczeń lub ich braku. Spośród białych znaczeń najbardziej dominujący charakter wykazał wzór mitt. Dla pozostałych wzorów nie można było ustalić szeregu dominacji ze względu na małą liczbę osobników. Otrzymane wyniki odnoszą się do określonej populacji zwierząt i są oparte jedynie na klasyfikacji fenotypowej. Dlatego też w celu dokładniejszych ustaleń dotyczących dziedziczenia umaszczeń w dalszych badaniach powinien zostać ujęty także aspekt genetyczny.

**Słowa kluczowe:** *Mustelidae*, barwa okrywy włosowej, wzór umaszczenia, genotyp

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