

# First report of a chinchilla phenotype in Viverridae (Carnivora)

Philippe GAUBERT<sup>1</sup> and Sylvain DUFOUR<sup>2</sup>

## Abstract

We report on the first case of a chinchilla phenotype in Viverridae (Carnivora), on the basis of a skin of Hausa Genet *Genetta thierryi* originating from Réserve de Faune de Kankan, Republic of Guinea. The specimen exhibits pale rufous brown spots likely to have been caused by eumelanin degradation, and uniform pale creamy orange background coloration probably due to lower concentration of pheomelanin.

**Keywords:** coat colour, genet, *Genetta thierryi*, Republic of Guinea, West Africa

## Première mention d'un phénotype *chinchilla* chez les Viverridae

## Résumé

Nous mentionnons le premier cas de phénotype chinchilla chez les Viverridae (Carnivora), sur la base d'une peau de Genette de Villiers *Genetta thierryi* provenant de la Réserve de Faune de Kankan, en République de Guinée. Le spécimen montre des taches marron-roux pâle vraisemblablement causées par une dégradation de l'eumélanine, et une couleur de pelage orange crème pâle probablement diagnostique d'une concentration plus faible de phéomélanine.

**Mots clés:** Afrique de l'Ouest, couleur du pelage, genet, *Genetta thierryi*, République de Guinée

In mammals, the colour of hairs, skin and eyes stems from the biosynthesis of a range of melanin pigments occurring in melanocytes. Such pigments arise from a common metabolic pathway where a series of enzymes is involved in different oxidation steps catalysed by tyrosinase. Thus, mutations that affect melanin biosynthesis have a global impact on the organism, including on retinal pigments. Probably the best-known mutation of this type is albino, where the loss of the oxidative function of tyrosinase results in a white phenotype with red eyes. Melanocytes synthesise two types of melanins, namely eumelanin (brown/black) and pheomelanin (red/yellow), both requiring the action of tyrosinase (Hearing & Tsukamoto 1991, Barsh 2001, Ito & Wakamatsu 2003). Melanocytes from hair follicles may switch between eumelanin and pheomelanin (or both at the same time) synthesis, a mechanism responsible for the great coat colour polymorphism observed in natural populations of mammals (Furumura *et al.* 1996, Barsh 2001) and which is likely to be involved in adaptive cryptic colorations (Singaravelan *et al.* 2010).

The large extent of gradual variation in coat pattern and colour in carnivores (order Carnivora) is well documented, both in domestic stocks (Kaelin & Barsh 2013) and wild populations (Little 1958, Robinson 1976). The most common of the 'aberrant', genetically-determined coat colour mutants in wild carnivores is melanism (prevalence of black pigmentation), almost reaching local fixation in Jaguar *Panthera onca* (Eizirik *et al.* 2003). Other coat-colour mutants found within carnivores are albinism (absence of melanin) and erythrism (prevalence of red pigmentation) (Little 1958, Veron *et al.* 2004). Contrary to most of the gradual coat colour variation observed in 'standard' phenotypes, the adaptive nature of those three classes of phenotypic mutants is still uncertain in carnivores (Robinson 1976, Caro 2005, Hedrick 2009). Cats (Felidae) are probably the carnivores that show the widest clinal variation in coat pattern and colour (Robinson 1976), including melanic

specimens observed in 11 out of the 37 species as well as chinchilla mutants such as 'white' Lions *Panthera leo*, Tigers *P. tigris* and Ocelots *Leopardus pardalis* (Robinson 1976, McBride & Giordano 2010). Recent studies have shown the complexity of aberrant coat colour acquisition in cats, suggesting at least five independent mutational pathways encoding melanism (Eizirik *et al.* 2003, Schneider *et al.* 2012).

Civets and allies (Viverridae) exhibit a wide range of coat pattern and colour variation, also including mutants such as albino, melanistic and erythristic phenotypes (Webb 1947, Sharma 2004, Veron *et al.* 2004, Eaton *et al.* 2010, Gaubert & Mézan-Muxart 2010, Delibes *et al.* 2013). In the genets *Genetta*, the wide variation observed in coat pattern and colour has been responsible for some long-standing taxonomic confusions (Gaubert 2003, 2013, Gaubert *et al.* 2005, 2008). Aberrant phenotypes do also occur at various (but low) frequencies, including albino individuals in Common Genets *Genetta genetta* from Europe (Delibes *et al.* 2013) and melanistic specimens of Miombo Genet *G. angolensis*, Servaline Genet *G. servalina*, Rusty-spotted Genet *G. maculata* (Africa) and *G. genetta* (Europe) (Webb 1947, Gaubert & Mézan-Muxart 2010, Barrull & Mate 2012). We here add to the list of phenotypic aberrations within the Viverridae by reporting the first case of a chinchilla mutant, on the basis of a skin of Hausa Genet *G. thierryi* collected in the Réserve de Faune de Kankan, Republic of Guinea (West Africa).

Four sun-dried skins of genets, including an aberrant 'creamy-rufous' specimen, were collected from Nalankoni (10°05'N, 8°35'W), in the Réserve de Faune de Kankan, Republic of Guinea, between 26 March and 6 June 2009, during a participatory community management conservation project conducted by SYLVATROP. The Réserve de Faune de Kankan is located east in the Republic of Guinea, at the border with Côte d'Ivoire, in the Guinean savanna belt. Mean annual rainfall, temperature and relative humidity reach 1,673 mm, 26 °C and

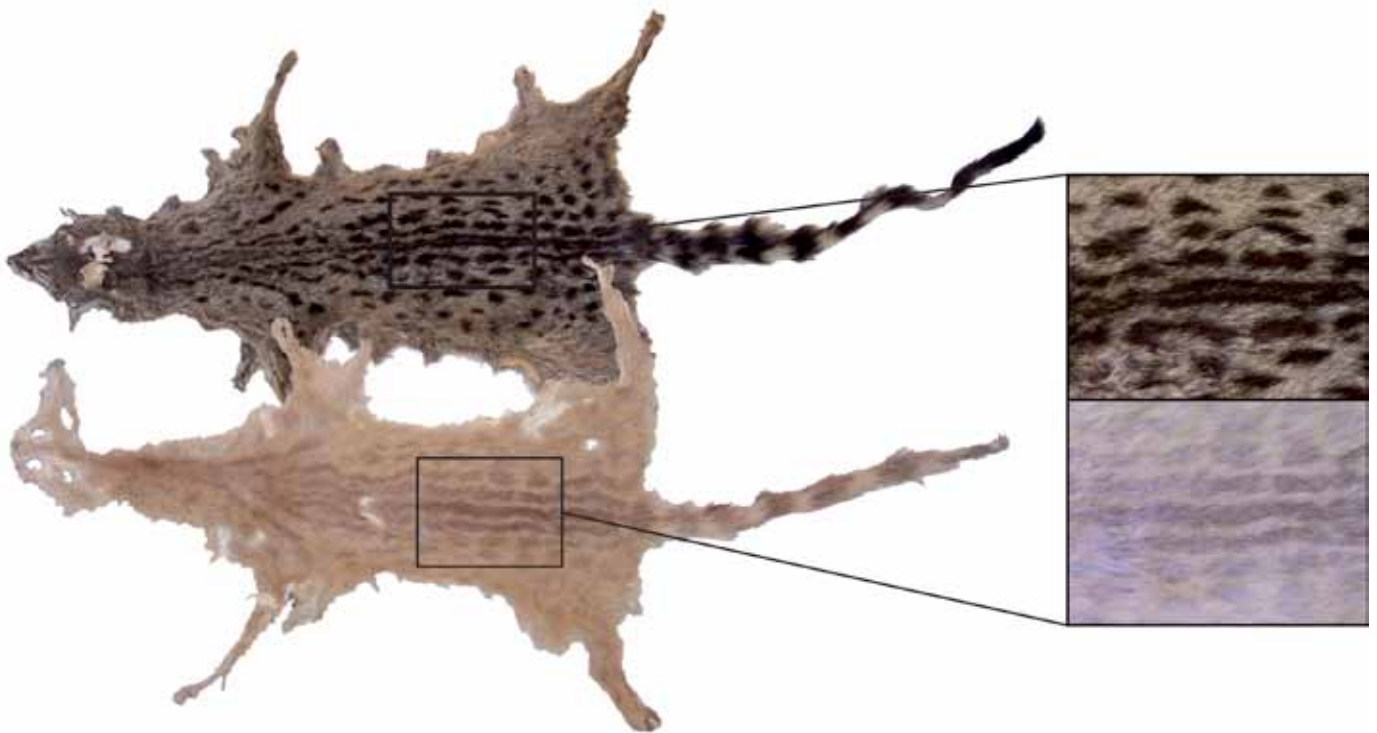
66%, respectively. The vegetation is a mix of periodically flooded grassy plain (consisting mainly of grasses on waterlogged soils), wooded savannas with scattered trees and shrubs, and forest galleries along permanent streams. The exact habitat(s) from which the genets were taken is/are unknown.

We used the computer-assisted identification key of Gaubert *et al.* (2008) and PCR-amplified short fragments of cytochrome *b* (DNA extraction protocol from Gaubert & Zenatello [2009]; specific primers from Gaubert *et al.* [2011]; data not shown) to reach species identification of the four skins collected. The specimens were deposited at the mammals and birds (Mammifères et Oiseaux) collections, Muséum National d'Histoire Naturelle, Paris (MNHN), France.

Two skins were identified as West African Large-spotted Genets *G. pardina* (MNHN 2010-1260 and -1263), while the two others were Hausa Genets *G. thierryi* (MNHN 2010-1261 and -1262), including the aberrant 'creamy-rufous' specimen (MNHN 2010-1261; Fig. 1). The latter exhibits pale rufous brown spots and uniform pale creamy orange background coloration. We could not observe any black hairs, whereas a fair proportion of white hairs occurs over the entire skin (i.e. spots and background). The other specimen of *G. thierryi* (MNHN 2010-1262) collected at the same period and coming from the same general locality has dark brown spots and brown (back) to yellow (belly) ash-grey ground coloration. Such a colour pattern is included in the phenotypic range of the species (Gaubert & Dunham 2013a). The two individuals of *G. pardina* collected from the same area exhibited standard phenotypic characters of West African Large-spotted Genets from the Guinean savannah (Gaubert 2003, Gaubert & Dunham 2013b).

Loss-of-function in genes involved in the synthesis of eumelanin can result in black coat markings turning to red. Such alterations of eumelanin synthesis have variable effects on coat colour depending on whether or not pheomelanin is synthesised (Barsh 2001). The aberrant specimen of *G. thierryi* that we describe here refers clearly to a chinchilla mutant, a phenotype already observed in carnivores such as white Lions and white Tigers. In those cats, the chinchilla mutants show a reduced amount of all the melanin pigments, with more viewable effects on the yellow (pheomelanin) than on the brown or black (eumelanin) pigmented areas (Robinson 1976). In white Lions, spots turn pale sepia brown (degradation of eumelanin) while the background coat colour becomes light fawn (low concentration of pheomelanin) (Robinson & Vos 1982). Similarly, in white Tigers pheomelanin is expressed as pale beige and eumelanin is degraded to sepia brown (Robinson & Vos 1982), in such a way that markings and spots are still visible but appear less clearly. We can also relate the chinchilla phenotype observed in the Hausa Genet to the "amber light silver mackerel tabby" Norwegian Forest Cat (a breed of domestic cat *Felis catus*) represented in Peterschmitt *et al.* (2009: 549; Fig. 2e): the individual has a light pinkish-beige colour with a toned down tabby pattern. It is unfortunate that we could not observe the genet specimen alive to reinforce our diagnosis, because chinchilla phenotypes should have bluish or whitish irides with reddish pupils (Robinson 1976).

The chinchilla phenotype is linked to the specific allele  $C^{ch}$  of the *full color* (Robinson 1976) or *albino/tyrosinase* (Lamoreux *et al.* 2001) locus *C*. The *chinchilla* allele encodes a partly functional tyrosinase, with the consequence of drastically restraining the synthesis of pheomelanin and degrading the



**Fig. 1.** Coat colour variation in Hausa Genets *Genetta thierryi* from Nalankoni, Réserve de Faune de Kankan, Republic of Guinea. Top: standard phenotype (MNHN 2010-1262). Bottom: chinchilla mutant (MNHN 2010-1261). Boxes on the right correspond to the zoomed dorsal areas of the coats.

eumelanin pigment (Robinson & Vos 1982, Ito & Wakamatsu 2011). Due to the preferential dilution of phaeomelanin observed in  $C^h$  mutants, the characteristic chinchilla phenotype has a banding pattern of eumelanin alternated with cream-coloured phaeomelanin, a pattern similar to that observed in the specimen MNHN 2010-1261. The fact that the latter does not exhibit a pure white background colour (meaning that phaeomelanin is still synthesised) does not go against its attribution to the chinchilla phenotype, since several levels of background paleness have been described among carnivore  $C^h$  mutants (Little 1958, Robinson & Vos 1982, Peterschmitt *et al.* 2009), even within the same species (Robinson 1976).

It is exceptional to find such an aberrant coat colour pattern in Hausa Genet, since it is to our knowledge the first time a chinchilla mutant is reported among the Viverridae. Given the scant data associated with the specimen collected, we cannot assess the potential adaptive or deleterious value of its aberrant phenotype. However, the individual was most likely a full-grown adult (head and body length = 51 cm; see Gaubert & Dunham 2013a), that had survived in the wild despite its pale coat colour.

The Viverridae remains a fascinating group to study coat pattern and colour variation, given its wide range of clinal variability and the existence of aberrant phenotypes such as melanistic, albino, erythristic and – as described here for the first time – chinchilla.

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**<sup>1</sup>UMR BOREA - MNHN - CNRS 7208 - IRD 207 - UPMC, Département Milieux et Peuplements Aquatiques, Muséum National d'Histoire Naturelle, 43 rue Cuvier, 75231 Paris, France.**

**Email: gaubert@mnhn.fr**

**<sup>2</sup>SYLVATROP, 26 route de Vannes, Nantes, France.**

**Email: sylvaduf@yahoo.fr**



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