

### Ischemic Depigmentation<sup>1</sup>

Numerous observations show that, in man, dark skin or hair may lose its pigment in areas exposed to certain types of injury (e.g. by X-rays, trauma, chemical insults). Indeed, according to a recent review of the literature, several verified case reports show that sudden greying of the scalp hair can occur immediately after exposure to severe systemic stress, especially if the latter is associated with intense fear<sup>2</sup>. Here, we should like to describe a simple and reliable experimental model for the induction, by circumscribed cutaneous ischemia, of topical depigmentation in dark-haired rats.

In 10 dark-brown female rats of the ACI-42324 strain (Microbiological Associates, Walkersville, Maryland) with an average body weight of 100 g (range 90–110 g), a transient local ischemia of the skin was produced by the application of special clips. These were prepared from ordinary umbilical clamps ('Hesseltine', Ingram & Bell, Toronto) in which the simple branch (the one not bearing a hook) was covered with rubber tubing. After shaving the back with electric clippers, a skin fold was taken up between the fingers and compressed by the clip for a period of 8 h. Immediately after removal of the clip the previously ischemic skin region remained somewhat pale and, during the following hours, it developed a mild oedema, but otherwise it retained its normal appearance. During the subsequent days, however, the pigmented fur was shed and gradually replaced by completely white hair which grew much more rapidly than the unaffected brown hair in the surroundings (Figure 1).

The previously dark skin of these animals also became white in the region exposed to ischemia. However, in this strain, the dermal colour is primarily determined by the pigmentation of the hair roots and the intracutaneous portions of the hair shafts; even normally, there is no detectable melanin in the epithelial and connective-tissue cells of the skin.

Histological examination of sections (embedded in paraffin and stained with the PAS-technique or with hematoxylin-phloxine) showed that, in the affected

region, neither the root nor the shaft of the regenerating hairs contained any detectable melanin. The density of the hair was greatly diminished, the dermal fat cells virtually disappeared and the connective tissue became extremely dense. The epithelium was somewhat thickened and its normally wavy surface became straight. The defect in pigment formation showed no sign of reversibility during a 3 months observation period.

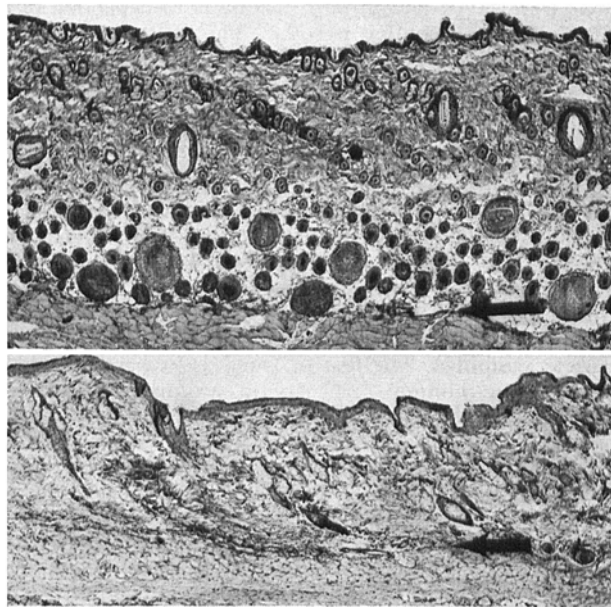


Fig. 2. Top: In the normal skin of the black rat, the medulla of each hair shaft is deeply pigmented. The epithelium is thin and wrinkled. Fat tissue is well developed especially just above the cutaneous muscle (whose outer limit is indicated by an arrow). Bottom: 1 month after application of the clip, hair density is greatly reduced and the medulla of the remaining hair (e.g. that just above the arrow which, again, indicates the outer limit of the cutaneous muscle) is totally devoid of pigment. The epithelium is thick and comparatively flat. The fat tissue has disappeared and the cutaneous muscle layer is atrophic (PAS  $\times 40$ ).

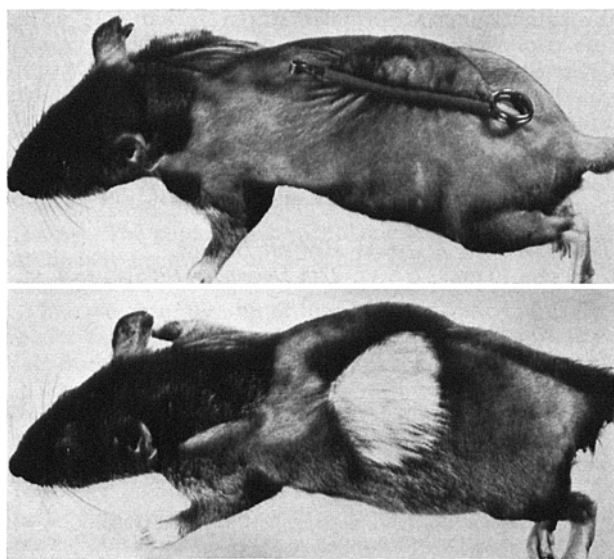


Fig. 1. Top: Clip in position on the shorn back of the rat. Bottom: 1 month after application of clip, the hair in the previously ischemic area is white and longer than in the surroundings where regeneration after shearing is irregular and slow.

**Zusammenfassung.** Durch vorübergehende Unterbrechung des Blutkreislaufes mittels einer besonderen Klemme gelingt es, in einem Hautlappen der braunen Ratte selektive Veränderungen des Fells hervorzurufen. In dem behandelten Gebiet fallen die Haare zunächst aus, werden aber dann durch besonders schnell wachsende, weisse Haare ersetzt. Diese Depigmentierung ist anscheinend auf eine permanente Schädigung der Melanin-synthese zurückzuführen.

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<sup>2</sup> R. Richter, in *Handbuch der Haut- und Geschlechtskrankheiten* (Ed. W. Jadassohn; Springer Verlag Berlin, Göttingen, Heidelberg, 1963), vol. 1, p. 282.