

## Pigmentation Disorders

*Albinism* is an inherited condition in which melanocytes, due to a lack of tyrosinase, do not synthesize melanin. Albinism is characterized by a lack of melanin in skin, hair, and feathers, and in the iris of the eye. Albino animals are much used as laboratory animals. They are easily distinguished by their white fur, and by their red eye color, which is due to reflection of light from the blood circulating in the choroid tissue in the rear of the eye. In normally pigmented animals, melanin in the deep part of the iris and in the choroid absorbs the light before it reaches the blood capillaries.

Normal pigmentation of skin and hairs occurs by microtubular transfer of melanin-containing vesicles, melanosomes, into thin extensions of the melanocytes, followed by transfer of melanin-containing melanosomes to keratinocytes (Fig. 17.3). In individuals suffering from the inborn Chediak-Higashi syndrome, there is abnormal fusion and trafficking of melanosomes, resulting in huge organelles that cluster near the nucleus of the melanocytes. This results in abnormally light pigmentation of skin and

hairs. The defects in vesicle formation, fusion, and trafficking are also present in, and affect the functions of other cell types, particularly cells of the immune system and blood platelets (p. 422). The Chediak-Higashi syndrome has been observed in Persian cats, Aleutian mink (a special genotype with light-colored fur), in mutants of blue foxes and silver foxes, in Hereford cattle, and in humans.

Certain diseases in animals and humans are characterized by a patchy or general increase in pigment formation in the skin. Such *hyperpigmentation* can be caused by abnormally increased secretion of the pituitary hormones MSH and ACTH. The receptors for MSH and ACTH are rather similar (p. 266). Addison's disease (p. 279) is caused by increased secretion of ACTH, and hyperpigmentation is commonly associated with the disease. The high concentrations of ACTH stimulate MSH receptors in the melanocytes, thereby increasing the formation of black eumelanin in the skin. Hyperpigmentation also occurs in other endocrine diseases in pet animals.

and the same papilla can also produce feathers with strikingly different patterns and colors during different seasons (Fig. 17.8c), such as black or brown-striped in the spring, and white in the autumn. In addition to coloration, the melanin pigments also give a lustrous shine to the feather coat as light is reflected off the barbs.

Factors other than melanin also contribute to the color of feathers. Blue and shiny colors are not derived from pigments, but are due to re-

flection, refraction, and diffraction of light that strikes minute particles (diameter  $< 0.6 \mu\text{m}$ ) or air bubbles that are incorporated into the feathers. Coloration that is not based on pigments is called structured coloration.

In some birds, the precursors of vitamin A, the carotenoids, contribute to the coloration of the skin and feathers, such as the characteristic bright yellow color in canaries and the red color in ibis. The same carotenoids are responsible for

A particular papilla can produce feathers with varying colors during different seasons

Some birds use precursors of vitamin A for coloring

**Figure 17.8** Structure of feathers. **a** Flight feather from the wing. **b** Microscopic structure of barbs and barbules forming a coherent surface (the vane) of a flight feather. The hooklets, which are present only on the barbules on one side of the barb, hook onto the smooth barbule from the adjacent barb. **c** Feathers that develop from the same feather follicle can have different pigmentation, depending on the season of the year. The winter feathers on the breast of the willow grouse are white (no pigment), whereas the summer feathers exhibit a series of bands with different pigmentation.

