lite and amosite are the most common amphibole fibers. The size and shape of fibers is important to the carcinogenic potential of asbestos. Fibers that are long and thin tend to be more carcinogenic than short, thick fibers. Therefore, the amphibole fibers are considered to be the most carcinogenic.25 However, in the United States both fiber types are treated the same for regulatory purposes. In humans, the association between malignant mesothelioma and asbestos exposure is well established.25–27 Malignant mesothelioma is a rare and spontaneous tumor in dogs.

Ferruginous bodies are asbestos fibers coated with ferritin and amorphous protein that can be found in the lungs of humans and dogs with malignant mesothelioma. Ferruginous bodies are considered to be markers of asbestos exposure. Although they can be found in the lungs of the general urban human population, it is the number of asbestos bodies per gram of lung tissue that separates occupational from casual environmental exposure. In a study of 18 urban dogs with mesothelioma by Glickman et al., asbestos bodies were identified in significantly higher numbers in the lungs of 3 dogs with sufficient archival tissue for analysis, when compared with lung tissue from controls.28 In humans, the association between malignant mesothelioma and asbestos exposure is well established.25 There is a strong likelihood that asbestos is a cause of this tumor in dogs. Chronic, low-grade inflammation in the lungs following asbestos exposure and fiber phagocytosis by pulmonary macrophages is cited as the mechanism of malignant transformation.25

Sunlight
One of the best examples of an environmental cancer is the development of squamous cell carcinoma following exposure to sunlight (ultraviolet or UV irradiation) in white cats, white-faced cattle, and possibly in collies and Shetland sheep dogs.29,30 White cats and white-haired areas of cats (especially the ear tips and nose) are susceptible to a chronic inflammatory dermatitis that is exacerbated by exposure to direct sunlight (Figure 4-3). These inflammatory lesions may evolve to squamous cell carcinoma. Poorly pigmented skin posterior to the planum nasale in dogs may be susceptible to a similar progression of a focal chronic inflammatory dermatitis to squamous cell carcinoma.30

The relationship between sunlight and skin cancer is well established in humans and sunlight was suspected to cause skin cancer in humans as early as 1894. There is a clear dose relationship that is expressed in experimental and epidemiological data that proves a causal relationship between accumulated UV dose and skin cancer.31–34 In humans, melanotic skin cancer risk is more related to the frequency and severity of sunburns in childhood and adolescence, while nonmelanotic skin cancer (primarily squamous cell carcinoma and basal cell carcinoma in humans, and squamous cell carcinoma in dogs and cats) is related more to cumulative long-term sun exposure. Ultraviolet B (ultraviolet light in the range of 280 to 320 nm; UV-B) is probably the portion of the ultraviolet spectrum that is involved most in nonmelanoma skin cancer in humans and animals.30,33 This assumption is based on studies with albino hairless mice that develop squamous cell carcinoma a few months after exposure to ultraviolet B.

The carcinogenic action of UV-B is believed to be associated with the formation of pyrimidine dimers, which when repaired incorrectly result in point mutations. These point mutations activate ras oncogenes in both animal and human skin cancers. Activated ras oncogenes encode mutated ras p21 that exists in the guanosine triphosphate-bound active state and, following localization to the inner side of the plasma membrane, cause cellular transformation.31 The carcinogenic action of UV-B is also attributed to immunosuppression (Figure 4-4).

About 3% of the sun’s electromagnetic output is emitted as ultraviolet light, but only a small fraction of UV-B reaches the surface of the earth. About 60% of the total solar UV-B reaches the surface of the earth between 10:00 AM and 2:00 PM. UV-C (ultraviolet light in the range of 100-280 nm) is virtually eliminated by ozone in the stratosphere and troposphere, but low levels are emitted by common fluorescent lights. UV-A (ul-
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FIG. 4-4. Presumed mechanism of immunosuppression secondary to UV light exposure. A photon of UV light can directly affect cellular DNA that leads to malignant transformation. UV-B photons may also change trans-Urocanic acid (trans-UCA) in the skin to cis-Urocanic acid (cis-UCA) that results in alteration of antigen presenting cell (APC) function. The ultimate result of excessive UV-B exposure is the stimulation of suppressor T cells that suppress the immune system and allow the promotion of tumor growth.

traviolet light in the range of 315–400 nm) is less carcinogenic than UV-B, and it is responsible for most of what we perceive as heat. UV-A is also implicated in non-neoplastic damage to the retina, lens, and cornea.

REFERENCES