are heat stable and are not destroyed by domestic or commercial cooking. Affected fish typically have a sharply metallic or peppery taste; however, they may be normal in appearance, color, and flavor. Not all persons who eat a contaminated fish necessarily become ill, perhaps because of uneven distribution of decay within the fish.

Symptoms develop within 15–90 min of ingestion. Most cases are mild, with tingling of lips and mouth, mild abdominal discomfort, and nausea. The more severe and commonly described presentation includes flushing (sharply demarcated; exacerbated by ultraviolet exposure; particularly pronounced on the face, neck, and upper trunk), a sensation of warmth without elevated core temperature, conjunctival hyperemia, pruritus, urticaria, and angioneurotic edema. This syndrome may progress to bronchospasm, nausea, vomiting, diarrhea, epigastric pain, abdominal cramps, dysphagia, headache, thirst, pharyngitis, gingival burning, palpitations, tachycardia, dizziness, and hypotension. Without treatment, the symptoms generally resolve within 8–12 h. Because of blockade of gastrointestinal tract histaminase, the reaction may be more severe in a person who is concurrently ingesting isoniazid.

TREATMENT SCOMBROID POISONING

Therapy is directed at reversing the histamine effect with antihistamines, either H-1 or H-2. If bronchospasm is severe, an inhaled bronchodilator—or in rare, extremely severe circumstances, injected epinephrine—may be used. Glucocorticoids are of no proven benefit. Protracted nausea and vomiting, which may empty the stomach of toxin, may be controlled with a specific antiemetic, such as ondansetron or prochlorperazine. The persistent headache of scombroid poisoning may respond to cimetidine or a similar antihistamine if standard analgesics are not effective.

475 Ectoparasite Infestations and Arthropod Injuries

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Ectoparasites include arthropods and creatures from other phyla that infest the skin or hair of animals; the host animals provide them with sustenance and shelter. The ectoparasites may penetrate within or beneath the surface of the host or may attach by mouthparts and specialized claws. These organisms may inflict direct mechanical injury, consume blood or nutrients, induce hypersensitivity reactions, inoculate toxins, transmit pathogens, and incite fear or disgust. Humans are the sole or obligate hosts for many kinds of ectoparasites and serve as facultative or paratenic (accidental) hosts for many others.

Arthropods that are ectoparasitic or otherwise cause injury include insects (such as lice, fleas, bedbugs, wasps, ants, bees, and flies), arachnids (spiders, scorpions, mites, and ticks), millipedes, and centipedes. Certain nematodes (helminths), such as the hookworms (Chap. 256), are ectoparasitic in that they penetrate and migrate through the skin. Infrequently encountered ectoparasites in other phyla include the pentastomes (tongue worms) and leeches.

Arthropods may also cause injury when they attempt to take a blood meal or as they defend themselves by biting, stinging, or exuding venoms. Various arachnids (spiders and scorpions), insects (bees, hornets, wasps, ants, flies, true bugs, caterpillars, and beetles), millipedes, and centipedes produce ill effects during these behaviors. Similarly, certain ectoparasites (e.g., ticks, biting mites, and fleas) that typically infest nonhuman animals can be medically significant. In the United States, lesions caused by arthropod bites and stings are so diverse and variable that it is rarely possible to identify the precise causative organism without a bona fide specimen and taxonomic expertise.

SCABIES

The human itch mite, *Sarcoptes scabiei* var. *hominis,* is a common cause of itching dermatosis, infesting ~300 million persons worldwide at any one time. Gravid female mites (~0.3 mm in length) burrow superficially within the stratum corneum, depositing three or fewer eggs per day. Six-legged larvae mature to eight-legged nymphs and then to adults. Gravid adult females emerge to the surface of the skin about 8 days later and then (re)invade the skin of the same or another host. Newly fertilized female mites are transferred from person to person mainly by direct skin-to-skin contact; transfer is facilitated by crowding, poor hygiene, and sex with multiple partners. Generally, these mites die within a day or so in the absence of host contact. Transmission via sharing of contaminated bedding or clothing occurs far less frequently than is often thought. In the United States, scabies may account for up to 5% of visits to dermatologists. Outbreaks occur in preschools, hospitals, nursing homes, and other residential institutions.

The itching and rash associated with scabies derive from a sensitization reaction to the mites and their secretions/excretions. A person's initial infestation remains asymptomatic for up to 6 weeks before the onset of intense pruritus, but a reinfestation produces a hypersensitivity reaction without delay. Burrows become surrounded by inflammatory infiltrates composed of eosinophils, lymphocytes, and histiocytes, and a generalized hypersensitivity rash later develops in remote sites. Immunity and associated scratching limit most infestations to <15 mites per person. Hyperinfestation with thousands of mites, a condition known as *crusted scabies* (formerly termed *Norwegian scabies*), may result from glucocorticoid use, immunodeficiency, and neurologic or psychiatric illnesses that limit the itch and/or the scratch response.

Pruritus typically intensifies at night and after hot showers. Classic burrows are often difficult to find because they are few in number and may be obscured by excoriations. Burrows appear as dark wavy lines in the upper epidermis and are 3–15 mm long. Scabetic lesions are most common on the volar wrists and along the digital web spaces. In males, the penis and scrotum become involved. Small papules and vesicles, often accompanied by eczematous plaques, pustules, or nodules, appear symmetrically at those sites; within intertriginous areas; around the navel and belt line; in the axillae; and on the buttocks and upper thighs. Except in infants, the face, scalp, neck, palms, and soles are usually spared. Crusted scabies often resembles psoriasis: both are characterized by widespread thick keratotic crusts, scaly plaques, and dystrophic nails. Characteristic burrows are not seen in crusted scabies, and patients usually do not itch, although their infestations are highly contagious and have been responsible for outbreaks of classic scabies in hospitals.

Scabies should be considered in patients with pruritus and symmetric superficial, excoriated, papulovesicular skin lesions in characteristic locations, particularly if there is a history of household contact with an infested person. Burrows should be sought and unroofed with a sterile needle or scalpel blade, and the scrapings should be examined microscopically for mites, eggs, and fecal pellets. Examination of skin biopsies (including superficial cyanoacrylate biopsy) or scrapings, dermatoscopic imaging of papulovesicular lesions, and microscopic inspection of clear cellophane tape lifted from lesions also may be diagnostic. In the absence of identifiable mites or eggs, the diagnosis is based on a history of pruritus, a clinical examination, and an epidemiologic link. Diverse kinds of dermatitis from other causes frequently are misdiagnosed as scabies, particularly in presumed "outbreak" situations. Scabies mites of other animals may cause transient irritation, but they do not reside or reproduce in human hosts.

TREATMENT SCABIES

Permethrin cream (5%) is less toxic than 1% lindane preparations and is effective against lindane-tolerant infestations. Scabicides are applied thinly but thoroughly behind the ears and from the neck down after bathing—with careful application to interdigital spaces and the umbilicus and under the fingernails—and are removed