Sarcoptic Mange

- Sarcoptic mange is a crusting skin disease, due to infestation by a particular species of mite.

Species Affected in Colorado
- Coyotes
- Foxes
- Less commonly affected, but reported in black bears, porcupines, and fox squirrels

What to Look For
- Hair loss, especially over the lower legs and tail (rat-tail)
- Thick crusting or scaling of the skin
- Intense itching and scratching
- Thin, debilitated animals

Cause and Transmission
Sarcoptic mange (also known as scabies) is caused by the “itch mite” *Sarcoptes scabiei*, which is a microscopic parasite that burrows tunnels in the outer layer of the skin. These tunnels are filled with mites, their eggs/feces and other irritating debris, causing intense itching and sores. The sores can progress to large areas of thick crusts and hair loss. Severely affected animals have substantial hair loss and can appear thin and weak. These animals often die due to exposure. Sarcoptic mange is highly contagious, with affected animals having thousands of immature, infective mites per square inch of infected skin surface. These immature mites are passed to a new host primarily through direct, skin-to-skin contact, allowing mange to spread more rapidly when populations of animals are densely concentrated.

Public Health Concerns
Sarcoptic mange can be transmitted between wildlife, domestic animals, and humans, although severe infections typically only occur when transmission is between animals of the same species. Direct, skin-to-skin contact is the most common method of transmission, although infections can be transmitted through contact with heavily-infested material such as bedding. Pets can be protected from infection by preventing contact with wildlife and using mite-controlling products prescribed by a veterinarian. Treatment requires prolonged therapy that is not possible for free-ranging wildlife.

Additional Information/References
*Sarcoptes scabiei* and sarcoptic mange, in *Parasitic Diseases of Wild Mammals* (Authors: Set Bornstein, Torsten Morner, and William M. Samuel)