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# Avian influenza V

## Clinical signs of avian influenza

Avian influenza infects and multiplies in a wide range of tissues including the respiratory tract, the digestive tract and the reproductive tract and the clinical signs seen reflect this. Clinical signs are also influenced by age, sex, species, immunity levels, environment and concurrent diseases.

Many infections with LPAI in wild birds produce no noticeable clinical signs, which has a significant impact on its epidemiology. In some species LPAI viruses can be immunosuppressive and cause egg drops.

In poultry clinical signs include those associated with the digestive, respiratory and reproductive tracts. On the respiratory front coughing, sneezing, râles and excessive lachrymation are commonly seen and in laying birds decreased egg production may be seen. Otherwise, signs are typical of those seen in sick birds and include huddling, depression and decreased feed and water intake.

In HPAI infections the first sign is high mortality. However, a wide range of clinical signs can be seen depending on which tissues the particular viral strain has a predilection for. These are more commonly seen in birds that survive the initial infection.

Nervous signs such as head and neck tremors, head/neck twisting and recumbency may be seen, as can severe egg drops, for example complete stopping of lay in just a few days. The longer birds survive the more likely nervous signs will appear.

Morbidity and mortality rates vary and reflect viral pathogenicity. For LPAI, mortality is often under 5%. For HPAI, morbidity and mortality are typically 60-90%, but can reach 100%, and mortality peaks within 3-5 days of the first clinical signs. Disease spreads slower in caged birds.

## Pathology

Lesions are very variable both in their severity and location.

LPAI infection often produces respiratory tract lesions such as fibrinous, catarrhal, serofibrinous or mucopurulent inflammation of the respiratory tract. The tracheal lining is often oedematous and congested and is sometimes haemorrhagic. Tracheal exudates range from serous to caseous and can cause tracheal occlusion.

Air sacculitis can be seen, as can an infra-orbital sinusitis. A bronchopneumonia can develop, especially if secondary bacterial pathogens such as Pasteurella multocida are present.

In layers an egg peritonitis can be seen, as can inflammatory debris in the oviduct. Misshapen, thin and pale eggs are often produced. Catarrhal enteritis/typhlitis (caecal inflammation) are often seen.

HPAI infection produces oedematous, haemorrhagic lesions in a variety of organs and the skin. When death is very sudden lesions can be minimal. Lesions are often influenza virus strain dependent but are most prominent in the muscles, the heart muscle and the lining of the proventriculus. Some strains produce oedematous, haemorrhagic lungs.