Scoliosis

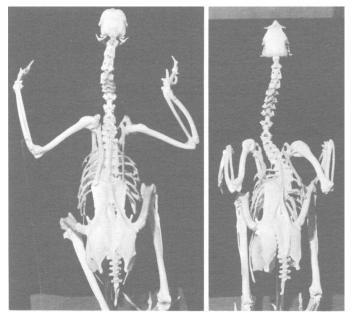
Idiopathic scoliosis (crooked spine) can be a crippling disease in humans. A common form primarily affects adolescent girls. There is clear evidence that idiopathic scoliosis is inherited. However, in humans, the pattern of inheritance is not clear.

Several years ago, Dr. L. W. Taylor of this department (Berkeley campus) developed an inbred strain of chickens in which up to 50 percent of the birds developed scoliosis. Subsequent research revealed a number of striking similarities between scoliosis in these birds and man. A more extensive study, with support from an Orthopaedic Research and Education grant and an NIH grant, is underway.

Cooperating with the UCD Departments of Orthopaedics, Animal Science and Nutrition, we have found that, in the scoliotic strain, the first spinal curves are detected after birds reach four weeks of age, usually between the fifth and sixth weeks. As these birds mature, progressively more severe curves develop until spontaneous fusion of the thoracic vertebrae occurs. The incidence of curved spines in sexually mature adults is now about 55 percent and is more frequent in males than in females. Thus, in chickens as well as in humans, the homogametic sex is more prone to scoliosis.

Our work also shows that abnormalities of growth and development of the spine are not the primary cause of scoliosis, nor is simple muscle imbalance involved.

-U. K. Abbott

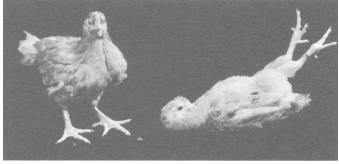


Normal spine (left) and scoliotic spine (right) in chicken skeletons.

Muscular dystrophy

Chickens with muscular dystrophy have become a useful tool in medical and meat research since the first mutant dystrophic chickens were brought to the attention of and diagnosed by UC avian scientists almost 30 years ago.

We now raise dystrophic chickens for worldwide scientific use and are using them in our laboratories to study how muscles grow and how nerves influence muscle development.



Chickens with muscular dystrophy are used in medical research.

This research has revealed some of the events that bring about muscular dystrophy and suggests ways muscular dystrophy might be alleviated.

We show that an enzyme, acetylcholinesterase (AChE) was defective in its regulation in dystrophic chicken muscle and that the levels and properties of this molecule were regulated in part by nerves. AChE is found at the junction of nerves and muscle fibers and plays an important role in regulating how muscles contract. To find out whether the defect was caused by nerves or by muscle, Dr. G. Wendell Yee and Dr. Thomas K. Linkhart, when they were graduate students, operated on young embryos 31/2 days old and transplanted the tiny wing buds from embryos of one strain to another. Nerves from the host embryo joined to limbs from the donor. When they studied the chick resulting from these operations, they found that all the properties of dystrophy they studied, including AChE properties, accompanied the wing bud and not the host. They concluded that the nerves of dystrophic birds were normal and that after 31/2 days of incubation, dystrophy was brought about by defects in the muscles themselves.

One of the symptoms of dystrophy that accompanied the limbs in the transplants was a hypersensitivity of the muscles to stimulation — a condition known as myotonia. Dr. Richard Entrikin has used this property as a starting point for chemotherapy studies. We found that diphenylhydantoin (DPH) corrected the myotonia and alleviated many of the functional properties of dystrophy for the 30 days of the experiment. In particular, dystrophic chickens were able to turn over and get up when laid on their backs, and levels of AChE in their muscles went down. We are now studying several other drugs with the help of the Muscular Dystrophy Association.

-B. W. Wilson

Newcastle disease research

Among diseases of poultry, Newcastle disease is the greatest threat to the chicken and turkey industry in our country. The virus that causes the disease is unique in that it may appear as a mild asymptomatic infection or as an exotic, highly lethal, rapidly spreading malady referred to as Velogenic Viscerotropic Newcastle Disease (VVND).

The existence of the disease in this country was first recognized and identified by scientists of the University of California in 1940 and an inactivated vaccine for its control developed by 1945. Later, an improved live virus vaccine was developed which gave an excellent protection against overt disease (including a drop in egg production). It was prepared in tissues other than the avian egg which eliminated introduction of transovarian avian pathogens by vaccination; it