Chicken Tail Vertebral Fusion Sheds Light on a Human Backbone Disease

Kevin Surya *, Honors College, Montana State University, Bozeman
Dana J. Rashid, Cell Biology and Neuroscience, Montana State University, Bozeman
Susan C. Chapman, Biological Sciences, Clemson University, Clemson

*Indicates Presenter

Ankylosing spondylitis (AS) is an inflammatory disease that causes vertebral fusion, affecting approximately 0.6% of the US population. Genome-wide association studies have shown that over 90% of AS patients harbor the HLA-B27 allele, which is associated with the histocompatibility complex. Still, underlying cellular mechanisms of AS are little understood, and a trigger has not been identified. Since AS mechanisms are difficult to study in humans, we require a suitable animal model. The chicken naturally fuses four of its last tail vertebrae during post-hatching growth, forming a compound bone called the pygostyle. As part of this fusion process, cartilaginous intervertebral discs remodel to become bone, and the nuclei pulposi at the centers of the discs completely degrade. How do chicken discs mineralize and does this phenomenon follow a similar path as human AS? We have observed several analogous features between chicken and AS vertebral fusion, including tissue bands bridging fusing vertebrae and fat deposits at the base of fusing vertebrae. Also, we observe an infiltration of neutrophil-like cells, suggesting that chicken vertebral fusion, like in AS, involves the immune system. Histology stains indicate that mineralization is present, but osteoid and osteoclasts are absent; fusion does not occur by endochondral ossification. We hypothesize this phenomenon represents the unusual transchondral ossification, where chondrocytes transform into osteoblasts. Currently, we're testing for cell death using TUNEL assays. Cell deaths would suggest that chondrocytes are dying not transforming. A possible trigger scenario involves transdifferentiation of disc chondrocytes into osteoblasts. Dying notochordal cells may cause the release of Complement 3 which facilitates transchondral ossification by recruiting neutrophil cells and promotes transdifferentiation of chondrocytes to osteoblasts. Finally, this study provides insights into AS mechanisms and elevates the chicken as an animal model.