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My 101

Osteoarthritis (OA) is the most common form of arthritis and affects more than half of people over the age of 65 in the US. It is the most common cause of disability in our country, and the only definitive treatment for knee OA, joint replacement, is the #1 expense for Medicare each year. Despite all this, we have no medications that can slow or stop OA progression, nor do we have biomarkers to predict OA development or progression.

Unlike some other forms of arthritis, relatively few specific genetic associations have been found in OA. It seems that non-genetic, environmental factors play the biggest role in development and progression, including aging, obesity, and previous joint trauma. My laboratory is dedicated to figuring out how these non-genetic factors affect the way that cells within joints and in the immune system through epigenetic modifications. Epigenetics, meaning literally “above the gene”, are mechanisms that turn genes on and off without affecting the actual genetic code, usually by adding bits of chemical signals to DNA and other DNA-associated factors. We hope to unravel the ways in which each particular OA risk factor affects the epigenetic state of genes in multiple types of cells throughout the body in an effort to help combat this disease. We are also focusing on interventions that might “fix” these epigenetic aberrations, including through modifying the microbiome, in hopes that we might be able to prevent OA or limit its severity.

My laboratory is also interested in the microbiome in OA. We were the first to identify a microbiome within human and mouse cartilage, which changes as patients develop OA. Furthermore, we have shown that microbiome transplantation in mice can significantly improve cartilage healing, something we are following up as a potential new OA therapy.