

AMERICAN COLLEGE OF RHEUMATOLOGY RESEARCH
AND EDUCATION FOUNDATION-ASP
JUNIOR CAREER DEVELOPMENT AWARD IN GERIATRIC MEDICINE



AWARD RECIPIENT

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PROJECT

"BONE ATTRITION IN OSTEOARTHRITIS:
INTERPLAY OF CARTILAGE LOSS, MALALIGNMENT, AND BONE DENSITY"

MENTORSHIP TEAM

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Osteoarthritis (OA), the most common form of arthritis, increases in prevalence with age, and is the leading cause of disability in US elders. In particular, OA of the knee is a significant cause of morbidity. To date, there are no proven therapies for the management of OA. Given the aging of our population, this constitutes a major public health concern. Furthermore, the pain and disability due to OA can lead to loss of independence and diminishment in quality of life for older adults. Therefore, from a patient perspective and a societal perspective, understanding the determinants of OA and therapies for OA are of great importance to a geriatric population.

Subchondral bone is thought to play an important role in both the pain and progression of OA. One example of subchondral alteration seen in OA is bone attrition, which is a depression of the subchondral bony surface unrelated to gross fracture. The altered properties of subchondral bone likely reflect both mechanical and systemic factors. Knee malalignment alters the distribution of load transmitted across a knee, which likely contributes to bone remodeling, with bone attrition as a possible consequence. Additionally, the presence of subchondral bone attrition, with subsequent alteration in the shape of the joint surface, may itself contribute to malalignment, stimulating further abnormal bone remodeling through the altered focal contact stresses, leading to further bone attrition. Systemic factors affecting the ability of bone to remodel or the quality of bone may also play an important role. The association of OA with bone mineral density (BMD) is complex and not

well-understood. It is possible that poor bone quality, as reflected by low BMD, may contribute to subchondral bone attrition. Further, it is possible that the effects of abnormal bone remodeling and malalignment interact to produce subchondral bone attrition and contribute to the progression of OA.

We propose studying these potential relationships in a large longitudinal cohort study. Specifically, we will study the effects of abnormal bone quality as measured by BMD and malalignment on subchondral bone attrition in OA. Studies will also be performed on how such bone attrition may contribute to the progression of OA as measured by cartilage loss. Should subchondral bone attrition be associated with cartilage loss, and mechanical or systemic factors be found to contribute to bone attrition, this would provide rational targets for therapeutic intervention in the management of OA.

The support of the American College of Rheumatology Research and Education Foundation-ASP Junior Career Development Award in Geriatric Medicine, together with the mentorship of David T. Felson, MD, Douglas P. Kiel, MD, and Sarah L. Booth, PhD, will allow me to continue my training toward becoming an independent investigator with a focus on rheumatologic disorders, particularly OA, of older adults. In addition to completing my doctoral degree in epidemiology, the award will allow me to use the knowledge gained from working on this project to help develop a future ROI application.