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Abstract:

Chronic kidney disease (CKD), which leads tocortical bone loss and increasedporosity,increases therisk of fracture. Animal models have confirmed that these changes compromise whole bone mechanical properties. Estimates from whole bone testing suggest that material properties are negatively affected, though tissue-level assessmentshavenot been conducted. Therefore, the goal of the present study was to examine changes in cortical bone at different length scales using a rat model with theprogressive development of CKD. At 30 weeks of age (~75% reduction in kidney function), skeletally mature male Cy/+ rats were compared to their normal littermates. Cortical bone material propertieswere assessed with reference point indentation (RPI), atomic force microscopy (AFM), Raman spectroscopy,and high performance liquid chromatography (HPLC). Bones from animals with CKD had higher (+18%) indentation distance increase and first cycle energy dissipation (+8%) as measured by RPI.AFM indentation revealed a broader distribution of elastic modulus values in CKD animals witha greater proportion of both higher and lower modulus values compared to normal controls. Yet, tissue composition, collagen morphology, and collagen cross-linking fail to account for these differences. Though the specific skeletal tissue

alterations responsible for these mechanical differences remain unclear, these results indicate that cortical bone material properties are altered in these animals and may contribute to the increased fracture risk associated with CKD.

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