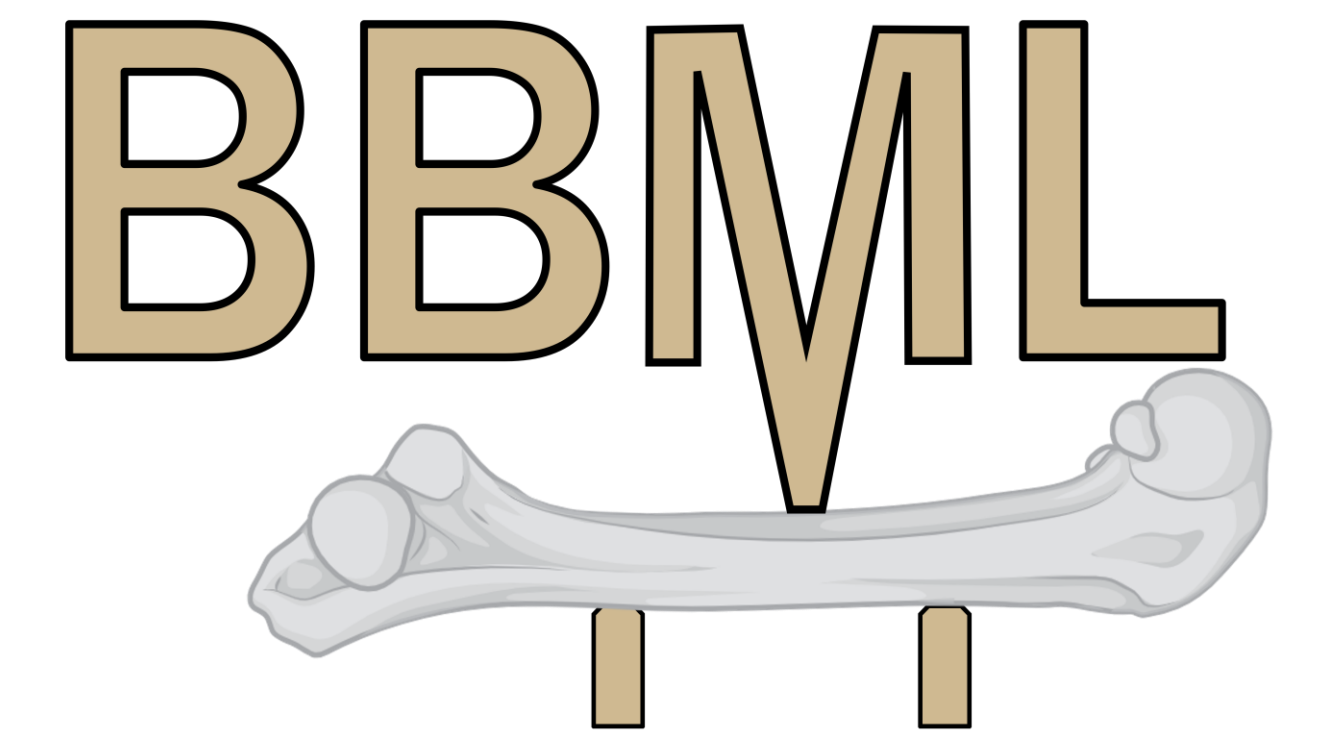


SKELETAL PHENOTYPE OF A MURINE MODEL OF AGING DIABETIC KIDNEY DISEASE

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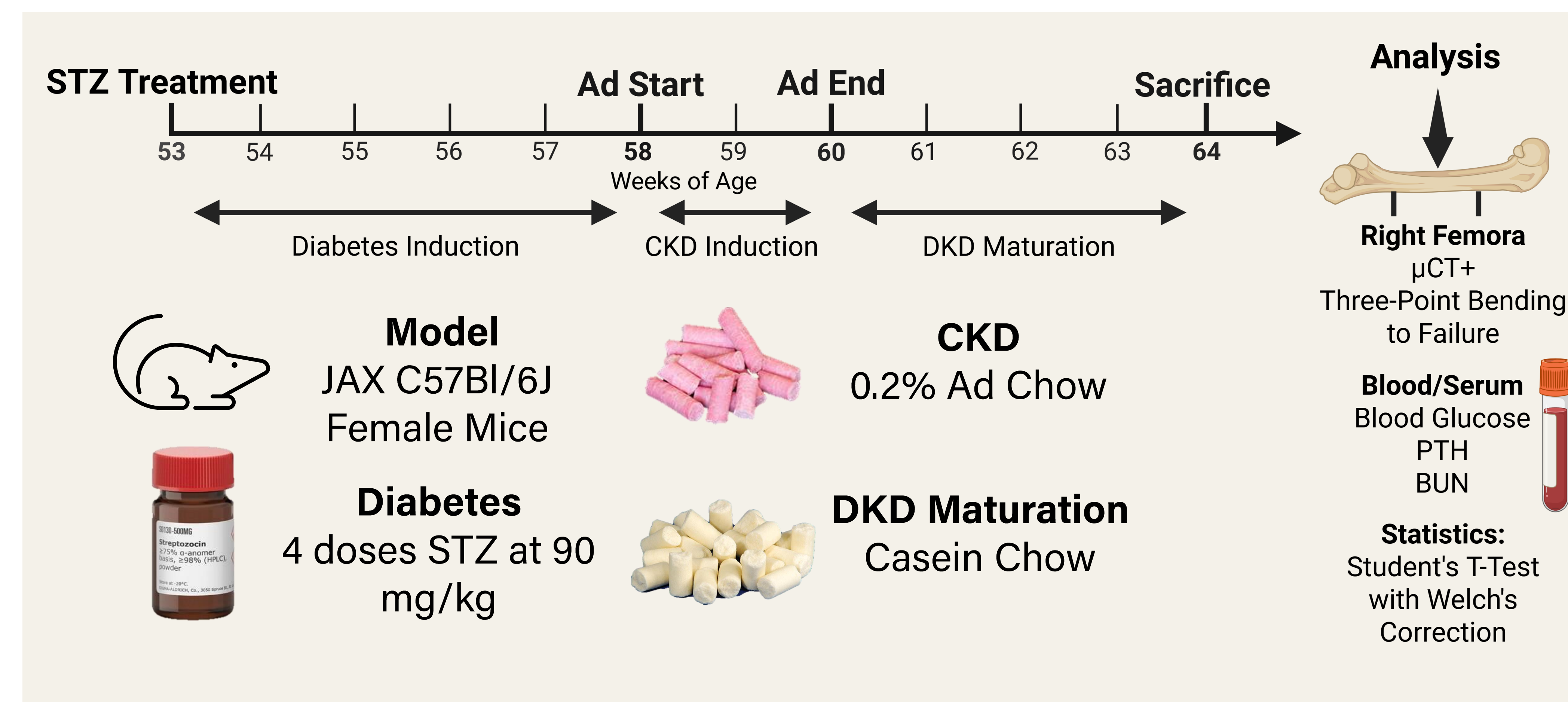
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Abstract

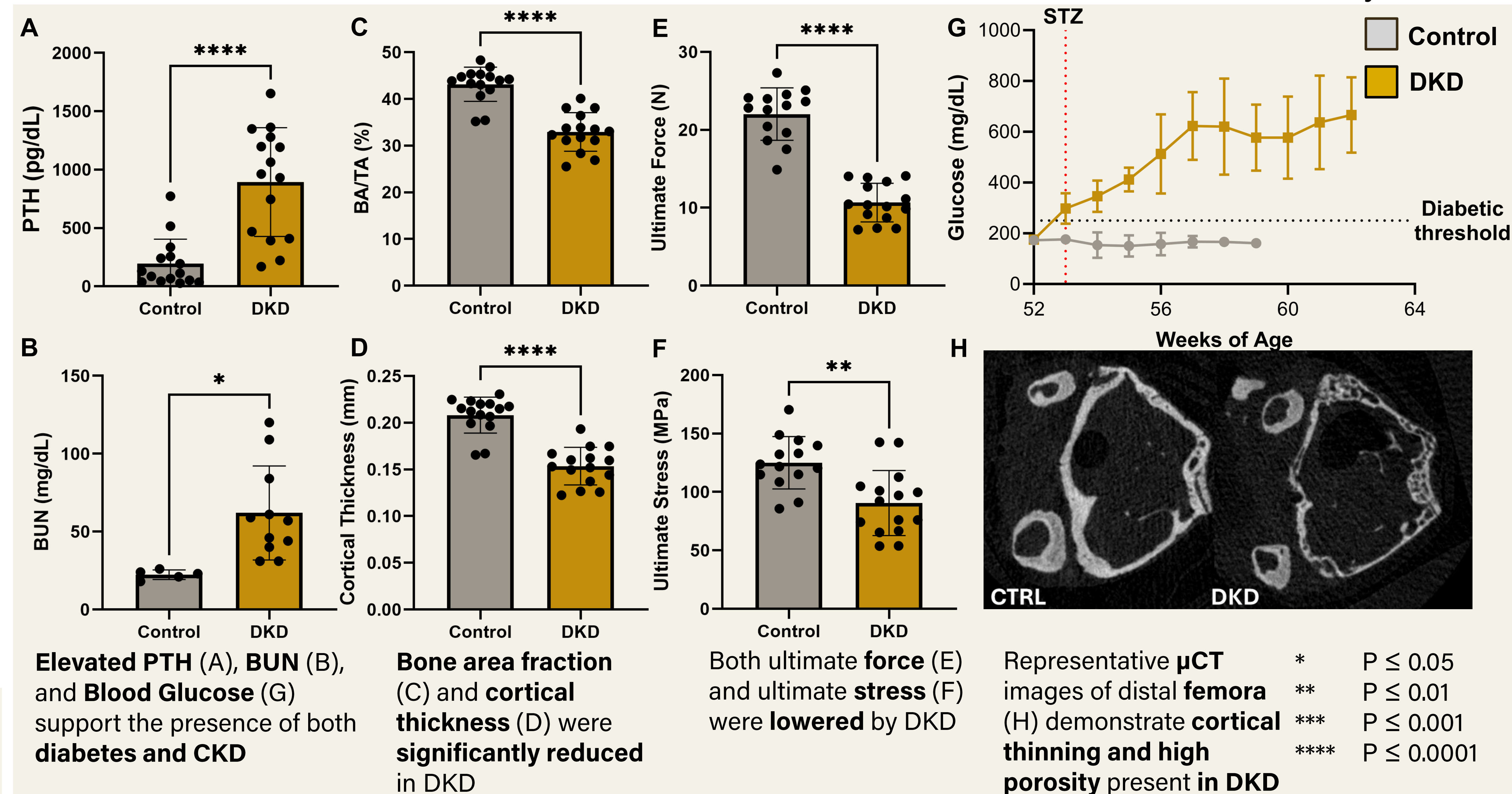
- As many as 30% of US adults over 65 are diabetic and up to 34% have chronic kidney disease (CKD): Diabetes and CKD have **high comorbidity** as diabetic kidney disease (DKD), especially in aging populations
- Mineral-bone disorder, elevated fracture risk, and **increased mortality** after injury
- Age-associated reduction in bone mass compounds with skeletal effects of DKD
- Women are more likely to suffer from age-related osteoporosis (1/3 of women over 65 versus 1/8 men)
- Aging and DKD** interact and **affect treatment** outcomes clinically
- Streptozotocin (STZ) and adenine (Ad) have been used to induce DKD in young mice
- Aging DKD models** with relevant skeletal manifestations in mice are **rare**

Hypothesis: An STZ and adenine regimen can induce a stable form of DKD (elevated PTH, BUN, and blood glucose) in aging mice to produce a severely degenerated skeletal phenotype (cortical thinning, reduced bone mass, and increased fracture risk)

Methods



Results



Discussion and Conclusions

Aging C57BL/6J Females are reliably inducible and a **suitable model of DKD**

- Females were chosen for modeling skeletal DKD because age-related is osteoporosis especially prevalent in the aging female population
- 19/20** mice became **hyperglycemic** within a month of STZ delivery (Blood Glucose > 250 mg/dL)
- Serum markers support that 2 weeks of **adenine** supplementation is **effective at inducing CKD** in aging female mice
- The degenerated **bone phenotype** and reduced mechanical strength **models** clinical presentation of **fragility in aging DKD**
- Bone quality effects currently under investigation

Special thanks to all members of the Bone Biology and Mechanics Lab

Contact

