Quantification of biological aging in young adults

Hypothesis: Biological changes will cause gradual and progressive decline in integrity of systems throughout the body with advancing age (e.g. Kirkwood & Austad 2000 Nature)

Hypothesis Test: Track changes in multiple organ systems in young, healthy adult humans.

Data from n=954 adults in the Dunedin Study who were examined at ages 26, 32, and 38 years. Analysis tested coordinated change across 18 different biomarkers of organ system integrity.

Results: Young adults who were all the same chronological age experienced different rates of biological aging. Faster agers showed deficits on tests of physical function, cognitive decline, and facial aging.
Three projects to advance translation

1. Comparison of genomic and clinical-biomarker algorithm methods to quantify biological aging (Belsky et al. R&R JGBS)

Many proposed measures. We conducted the first test of 7 different genomic and clinical biomarker methods in the same humans.

Overall, genomic measures were only weakly correlated with clinical biomarker measures. Clinical biomarker measures were more closely tied to healthspan.

2. Developing a test battery to screen patients for geroprotector trials (Belsky et al. R&R Aging Cell)

Rate of aging is normally distributed

Brief interview needed to identify fast-aging patients to ensure representation in clinical trials

15-minute interview to assess 5 risks:
- Short-lived family (no grandparents 80+)
- Grew up poor
- Adverse childhood experiences
- Low educational attainment (<high school)
- Nurse-rated personality

Cumulative risk burden identifies fast aging group

In-progress: Can measures of biological aging predict modifiable risk for AD/dementia?

New molecular assays of Duke EPESE, CALERIE, and Dunedin data

D. Belsky