Statement of Need

By 2030 almost every fourth person will be 65 or older in Switzerland, Europe, and USA[1,2]. Since old age is the main risk factor for developing cancer, neurodegenerative, cardiovascular, and metabolic diseases, as well as other age-related pathologies (Fig. 1), the growing elderly population poses an immense social burden on our younger generations and a financial burden on our healthcare system. It has been calculated that in the US delaying the onset by two years of these diseases would save $7.1 trillion over the next fifty years[3]. The onset of these diseases varies because the rate of aging is plastic and is influenced by environmental and genetic factors[4-6]. For instance, people who live beyond 100 years (centenarians) spend most of their lives in good health with rapid decline only towards the end of life[7]. Importantly, centenarians carry polymorphisms in genes that have been discovered in model organisms, such as the nematode *Caenorhabditis elegans,* to extend healthspan (the period of life spent in good heath) and lifespan[8]. To understand the causal relationships between genes and healthy aging, *C. elegans* is indeed a pioneering system to model the aging process because of its ease for genetic manipulation, high evolutionary conservation of genes implicated in human diseases[9], and short lifespan (3 weeks). Importantly, using *C. elegans* lifespan assays as a read-out for extension of healthspan is a tractable and fast approach for discovering novel mechanisms that confer healthy aging. Several fundamental mechanisms discovered in *C. elegans* have been shown to delay age-related pathologies in higher organisms, such as mice, and these mechanisms have major implications for humans aging[6,10-21]. Hence, by using *C. elegans* to model the aging process we could rapidly identify strategies to improve human healthspan.

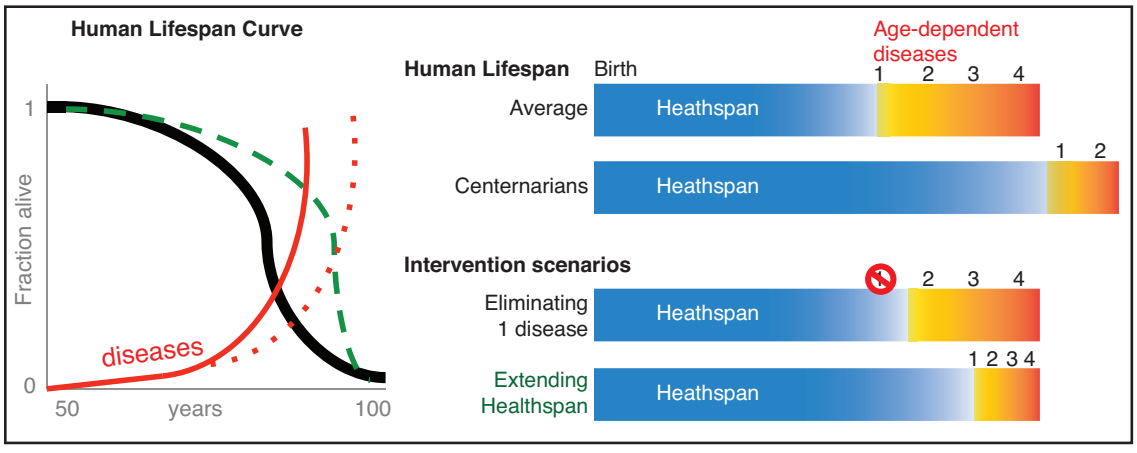

Fig.1. The goal of aging research is to extend human healthspan. Left panel shows average human lifespan (black line) and the rate of disease onset (red line). Delaying age-dependent diseases (dashed red line) would improve healthy aging (dashed green line). Right panel depicts disease free time (healthspan) and the problem of co-morbidities at the end of life for the average human population and centenarians[22]. Eliminating one disease will only slightly increase healthspan because of other age-dependent diseases. Our mission is to identify strategies that extend healthspan.

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22. Figure 1 modified from M. Collins and W. Mair.