Interview with Steven Austad
Evolution of Aging

KYLE JENSEN: Welcome to SAGE Crossroads, the premier online forum in issues of human aging. These podcasts feature lively discussion with the experts on the ethical, political, economic, scientific, and societal implications of aging related science. Thank you for listening.

I'm speaking now with Dr. Steven Austad. Dr. Austad is a professor in the Department of Cellular and Structural Biology at the University of Texas.

Dr. Austad, please describe some of your research that involves aging with various animal species.

STEVEN AUSTAD: Well, we're interested in finding out why some species of animal live a very short time and others live a very long time. We're looking for the clues inside the cells in what distinguishes long and short lived animals. Most recently we've been working on animal cells with DNA damage and their ability to repair it and also the ability of the cells to keep their proteins in tact, the proteins that form the structure of the cell and run all the chemical reactions. It looks like long lived animals have unique ways of repairing their DNA. It's much quicker and more complete than short lived animals. They also have ways of making their proteins look new for longer, and we're trying to understand how they do that at this point.

KYLE JENSEN: Now do you know why the species, the aging in species, is vastly different?

STEVEN AUSTAD: No, that's ultimately the question we hope to answer because if you look at the natural world the difference in aging between the shortest lived and longest lived species is vastly greater than that we can create in the laboratory. So, we feel like nature is providing us with good examples and all we need to do is figure out the key mechanisms that differ between the short and the long lived species.

KYLE JENSEN: Which theory of aging does your research lend more credibility towards?

STEVEN AUSTAD: Well, it's not clear how many theories of aging there are. I would say that a species definitely…mammals and birds is what we work on…warm blooded animals with back bones. In those it's quite clear that all kinds of damage accumulates with age and that all sorts of regulations with genes go slightly out of wack, although they do so at a much slower rate in long lived species.

KYLE JENSEN: Now, do you see a major breakthrough in unlocking the mechanisms that drive aging?

STEVEN AUSTAD: Well, ultimately I do. I think this is a problem like any other problem. Aging is not immutable. It's quite easily changed. It changes quite rapidly in evolutionary time, so I think ultimately we're going to understand what drives it and we're going to be able to alter it in humans as well as other animals.

KYLE JENSEN: Is it a field of research that's been pretty well funded?

STEVE AUSTAD: Surprisingly not. Given the fact that basically almost all of our medical costs would decrease if we could find a way to combat aging, we'd decrease heart disease and Alzheimer's disease and all the major causes of disability and death. You'd think that would be a very attractive thing for the National Institutes of Health to fund, but in fact, we get on order of 4% of the funds that go to the National Institutes of Health, so I think that we could do much better than that. There are some private foundations that fund aging research, and we're very appreciative of that, but compared to the disease foundations, aging research does not get as many resources as it probably should.

KYLE JENSEN: Lastly, the audience of SAGE Crossroads is made up of scientists, policy makers, and curious consumers. If there is one last statement that you could make to them about your research involving aging across species, what would it be?

STEVEN AUSTAD: That the natural world is likely to provide us with clues to slowing aging that we're unlikely to find in the laboratory.

KYLE JENSEN: Thank you. On behalf of SAGE Crossroads, I'm Kyle Jensen.