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Dr. Connie Nugent
Department of Cell Biology & Neuroscience

Why do DNA double-strand breaks within chromosomes activate the DNA damage checkpoint and undergo repair, but the termini of linear chromosomes do not? Two key factors that allow cells to distinguish their chromosome ends from breaks are 1) the telomerase enzyme, which adds short G-rich repeats to one DNA strand of the telomere, and 2) capping proteins that bind to telomeric sequences. The capping proteins prevent activation of the DNA damage checkpoint, and afford protection from enzymes that would otherwise degrade, unwind, or inappropriately recombine the ends. How this protection is achieved is not well understood, but in proliferating cells, both telomerase and capping proteins are important for genome stability. A telomere that becomes too short can lose its capping function, triggering DNA damage checkpoints and leading to cell death or chromosome end-fusions. While the last 15 years has seen major advances in our understanding of telomere biology in model organisms and humans, the fields of DNA replication, DNA damage checkpoints, and DNA repair all intersect at telomeres, leaving much to be discovered. The Nugent lab focuses on understanding how telomere capping proteins and telomerase coordinate their activity with the DNA replication machinery.