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Robert Haltiwanger is a past-President of the Society for Glycobiology and is currently Editor-in-Chief of the Society's journal, *Glycobiology*. He and his colleagues work on unusual *O*-linked carbohydrate modifications found on small cysteine-rich protein modules: epidermal growth factor-like (EGF) repeats and thrombospondin type 1 repeats (TSR). They were the first to report the existence of *O*-fucose and *O*-glucose glycans on the EGF repeats of the Notch receptor and have played key roles in identifying the enzymes responsible for the addition of these glycans including Fringe, protein *O*-fucosyltransferase 1, and Rumi. These glycans play essential roles in Notch function and are known modulators of Notch activity in numerous biological contexts. Dysregulation of Notch signaling results in a number of human disorders including several types of cancer. The Haltiwanger lab is particularly interested in determining molecular mechanisms by which these glycans affect the function of Notch. The Haltiwanger laboratory has collaborated extensively with laboratories studying the role of Notch in developmental systems including flies (Kenneth Irvine, Rutgers; Hugo Bellen, Baylor) and mice (Pamela Stanley, Einstein).

In addition to studying *O*-glycosylation of EGF repeats, the Haltiwanger laboratory also examines *O*-fucosylation of thrombospondin type 1 repeats. This modification is mediated by a distinct enzyme, protein *O*-fucosyltransferase 2, and recent work from the Haltiwanger lab has demonstrated that elimination of the gene encoding this enzyme in mice results in early embryonic lethality. The embryos display defects consistent with enhanced epithelial to mesenchymal transition and altered angiogenesis. Current work is focusing on identifying the targets of protein *O*-fucosyltransferase 2 and the molecular mechanism of these phenotypes.