Title : Aging-Associated Changes in YEATS Family Genes Expression and the Oxidative Stress Response in the *Drosophila* Brain

Author(s) :
1. Peeraya Julia Gassenschmidt — **Student ID :** 650510260
2. **Student ID :**
3. **Student ID :**

Major : Zoology

Advisor(s) :
1. Associate Professor Dr. Manaporn Manaboon
2. Dr. Luca Lo Piccolo
3.

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ABSTRACT

Neuronal stress responses rely on precise transcriptional control, and age-related alteration in chromatin regulation are likely to contribute to increased neuronal vulnerability, potentially underlying the progressive decline in oxidative stress resilience during aging. Recent studies has identified YEATS-domain containing proteins as an emerging class of epigenetic regulators that link histone acylation and metabolic state to transcriptional control of stress-responsive gene networks. The studies have begun to define the functions of the YEATS paralogous proteins *ear* and *D12* in the *Drosophila melanogaster* nervous system. Despite sharing a conserved chromatin-binding domain, these proteins exhibit distinct neuronal roles: suppression of *D12* impairs neuronal metabolism and behavior, whereas reduction of *ear* enhances oxidative stress tolerance and extends lifespan, indicating functional divergence in neuronal stress regulation. Building on these observations, this thesis aimed to determine whether YEATS family gene expression is regulated by oxidative stress and whether aging influences this regulation. Using the wild-type *Drosophila* strain Oregon R, young (5-day-old) and aged (20-day-old) male and female flies were exposed to oral hydrogen peroxide (H_2O_2). Survival was assessed, and transcriptional changes in YEATS genes (*D12*, *ear* and *Gas41*) were analyzed in fly heads by RT-qPCR alongside the antioxidant genes *SOD1* and *CAT*. Young flies of both sexes showed high survival following H_2O_2 exposure and exhibited downregulation of *SOD1* and *CAT* without

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significant changes in YEATS gene expression. In contrast, aged flies displayed markedly increased mortality, accompanied by induction of antioxidant genes and concurrent downregulation of all three YEATS family members under oxidative stress. Together, these findings indicate that aging is associated with a coordinated shift in transcriptional stress responses, linking altered regulation of chromatin-associated genes to increased neuronal vulnerability to oxidative stress.

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