Our results clearly show that *Dh* pretreatment protected *Drosophila* from gamma radiation induced lethality (Figure 1) and increased survivability of *Dh* fed *Drosophila* compared to control group. Therefore, *Dh* pretreated flies exhibit more radiation tolerance/resistance than the control flies. Our study demonstrates the radio protective potential of the edible roots of *Dh*, which has implications in cancer radiation therapy.

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A novel *GMR-Gal4* insertion produces a rough eye phenotype.

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Introduction

Much of modern research in *Drosophila* depends upon the use of the *UAS/Gal4* system (Brand and Perrimon, 1993) to express various transgenes under defined conditions. For the most part, it has been widely believed that the *Gal4* transgenes produce few negative effects in *Drosophila melanogaster*. However, *GMR-Gal4*^{12} was shown to produce an apoptosis-dependent "rough eye" phenotype (Kramer and Staveley, 2003) and the neural accumulation of the protein product of *Gal4* has been linked to neurodegeneration in *Drosophila* (Rezaval *et al.*, 2007). Apart from transcriptional interactions with the *UAS*-bearing transgenes, highly elevated levels of *Gal4* expression have been shown to lead to stress and immune responses (Liu and Lehmann, 2008). Due to the prominence of *Gal4* in *Drosophila* research, we believe that this phenomenon should be further examined.

GMR-Gal4^{12}, very commonly referred to as simply *GMR-Gal4*, was originally selected from a group of fifteen *GMR-Gal4* transgenic insertion lines (Freeman, 1996). Only two of these lines, including *GMR-Gal4*^{12}, did not display a hemizygous roughened eye phenotype at 25°C. Our group has shown, in *GMR-Gal4*^{12} homozygotes cultured at 25°C and *GMR-Gal4*^{12} hemizygotes raised at 29°C, that an apoptosis-dependent altered developmental process can produce a "rough eye" phenotype (Kramer and Staveley, 2003). To further investigate this phenomenon, we have produced a version of *GMR-Gal4* that we believe may be similar to the other original "rough eye" insertions to help evaluate the consequences of *Gal4* expression.

Materials and Methods

Drosophila media

Our standard cornmeal-yeast-molasses-agar medium is prepared with 65 g/L cornmeal, 10 g/L nutritional yeast, and 5.5 g/L agar in water, cooked by autoclave for 30 minutes (plus depressurization) then

augmented with 50 ml/L fancy grade molasses. To inhibit mold growth, 5 mL of 0.1 g/mL methyl 4-hydroxybenzoate in 95% ethanol and 2.5 mL of propionic acid are added per litre when cooled to 55 to 60°C before being poured into standard plastic shell vials and stored at 4 to 6°C.

Drosophila stocks and culture

The *GMR-Gal4*^{12} (Freeman, 1996) and *UAS-lacZ*$^{Bg4-1-2}$ (Brand and Perrimon, 1993) lines were obtained from the Bloomington Drosophila Stock Center at Indiana University. The *w; Sb PΔ2-3 e/TM6 Ubx e* line (Robertson *et al.*, 1988) was obtained from Dr. William Engels. The *w*^{1118} was obtained from Dr. H. Lipshitz.

P element transposition

The novel insertion of *GMR-Gal4* was generated by crossing *w; Sb PΔ2-3 e/TM6 Ubx e* males to *GMR-Gal4*^{12} females to produce dysgenic males which were, in turn, crossed to *w*^{1118} females at 25°C. Non-dysgenic male progeny were selected against the presence of *Sb PΔ2-3* and crossed to *w*^{1118} females. The F1 were mated and F2 were screened for the "rough eye" phenotype at 25°C. One line was isolated: *GMR-Gal4*^{H1}.

Drosophila crosses and biometric analysis

To generate critical class males, *GMR-Gal4*^{12} or *GMR-Gal4*^{H1} females were mated to either *w*^{1118} males, to produce "responder-less" progeny or *UAS-lacZ* to produce "benign responder" progeny at 25°C. These were collected, aged for three to five days at 25°C, frozen at -80°C, mounted on aluminum studs and desiccated for 24 hours or more before micrography. Scanning electron micrographs were produced by a FEI Quanta 400 Environmental SEM and analyzed using NIH ImageJ software (Abramoff *et al.*, 2004). Images were analysed from 10 individuals (n = 10) and the number of ommatidia and bristles were determined and standard error of the mean calculated.

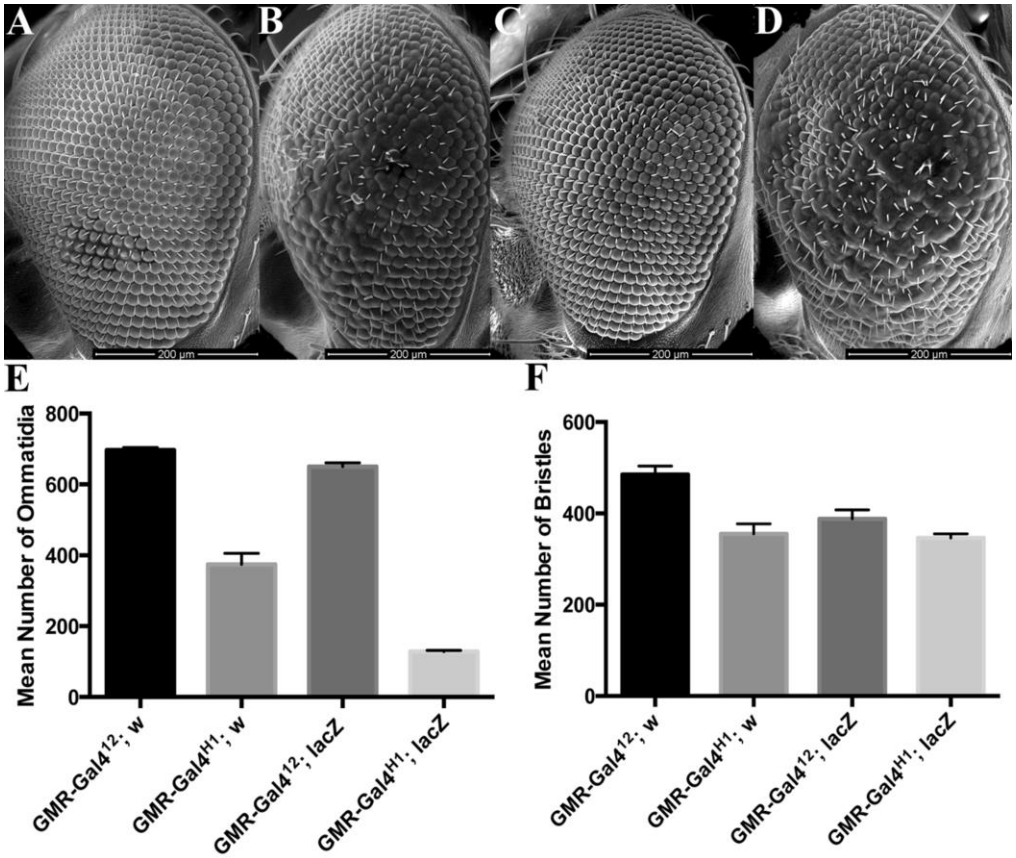

Figure 1: *GMR-Gal4*^{H1} shows a rough eye phenotype with and without *UAS-lacZ*. Scanning electron micrographs of the eyes of (A) *GMR-Gal4*^{12}/+, (B) *GMR-Gal4*^{H1}/+, (C) *GMR-Gal4*^{12}*/UAS-lacZ*, (D) *GMR-Gal4*^{H1}*/UAS-lacZ* males at 25°C. *GMR-Gal4*^{H1} shows a reduced number of (E) ommatidia and (F) interommatidial bristles when compared to the *GMR-Gal4*^{12}. Error bars indicated standard error ($p < 0.05$) and n = 10 for all four classes.

Results and Discussion

By P element transposition, a novel insertion line, *GMR-Gal4^{H1}*, has been generated. This line displays a rough eye phenotype at 25°C as a hemizygote with and without the presence of a *UAS*-controlled responder transgene (Figure 1).

Without a responding transgene, the eyes of *GMR-Gal4^{12}*/+ appear to be relatively normal, with a regular hexagonal array of ommatidia and interommatidial bristles (Figure 1A). With many fused ommatidia and reduced bristles, *GMR-Gal4^{HI}*/+ produces a "roughened" eye (Figure 1B). In the presence of the *UAS-lacZ* transgene, the eyes of *GMR-Gal4^{12}/UAS-lacZ* appear to be fairly normal (Figure 1C), while the eyes of *GMR-Gal4^{HI}/UAS-lacZ* are more severely compromised than in the absence of a responder (Figure 1D). With such an obvious developmental defect, the *GMR-Gal4^{H1}* line provides the opportunity to both suppress and enhance a phenotype that can be readily analysed through biometric means. Often utilised as control for the expression of genes of interest under any of a number of circumstances, the *UAS-lacZ* gene is usually considered to be benign in the developing eye under the control the *Gal4* transgenic drivers. Clearly, this does not seem to hold true under these conditions. Overall, the new insertion line *GMR-Gal4^{H1}* can produce striking phenotypes that seem ideal for further investigation of the toxic effects of *Gal4* expression.

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Relationship between gender difference in longevity and oxidative stress response in *Drosophila melanogaster.*

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Females have longer life expectancies than males across many species including humans, laboratory rats, and *Drosophila*. It is not clearly understood as to what genetic or environmental factors are responsible for the gender difference in longevity. According to free radical theory of aging, lifespan appears to be limited by the cumulative effects of oxidative damage from reactive oxygen species (Harman, 1959). However, there is evidence implicating the role of free radical-induced oxidative stress in aging. Previous studies in mammalian systems suggest that shorter lived males express lower levels of antioxidant defenses such as superoxide dismutase, catalase, and consequently suffers higher levels of oxidative stress than females (Ede *et al*., 2002; Tomas-Zapico *et al*., 2006).

Drosophila melanogaster offers a good model system to study the sex difference in longevity since females live significantly longer than males. Ethanol is known to induce free radical-mediated oxidative stress, and sex difference in acute ethanol responses have been reported in *Drosophila* (Das and Vasudevan, 2007; Devineni and Heberlein, 2012). However, differential susceptibility of the sexes to ethanol-induced oxidative stress in relation to their antioxidant status has not been studied in *Drosophila*. Therefore, we have investigated the differential susceptibility to ethanol induced oxidative stress in male and female *D. melanogaster* in relation to the antioxidant enzymes, superoxide dismutase, catalase.

D. melanogaster (Oregon K) flies were obtained from the *Drosophila* Stock Centre, University of Mysore, Karnataka, India. For lifespan studies, newly-eclosed male and female flies were housed separately