### **ORAL PRESENTATION**





# A *Drosophila* model linking diet-induced metabolic disease and cancer

Susumu Hirabayashi<sup>1\*</sup>, Thomas Baranski<sup>2</sup>, Ross Cagan<sup>1</sup>

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#### Background

Epidemiological studies have provided strong evidence for the association between cancer and metabolic diseases including obesity and diabetes, but the underlying mechanism remains poorly understood.

#### Methods

Feeding *Drosophila* a diet high in sucrose was previously demonstrated to direct important aspects of type 2 diabetes including insulin-resistance, hyperglycemia, increased insulin levels, accumulation of fat, and heart dysfunction [1,2]. We used this model to explore the effects of high dietary sucrose on tumor progression of Ras/Src co-activated *Drosophila* tumor model.

#### Results

We demonstrate that high dietary sucrose, but not high dietary fat transforms Ras/Src-activated cells from localized growths to aggressive tumors with emergent metastases. Surprisingly, while most tissues displayed aspects of metabolic dysfunction including insulin resistance, Ras/Src-activated tumors retained insulin pathway sensitivity and exhibited an increased ability to import glucose. We provide evidence that this reflects increased insulin signaling, which in turn acts through Wingless/ Wnt signaling to promote diet-mediated malignant phenotypes within Ras/Src-activated tumors [3]. These fly models should provide useful paradigms to study the link between metabolic dysfunction and tumorigenesis in the context of a whole animal.

#### Authors' details

<sup>1</sup>Department of Developmental and Regenerative Biology, Icahn School of Medicine at Mount Sinai, New York, NY 10029, USA. <sup>2</sup>Department of

<sup>1</sup>Department of Developmental and Regenerative Biology, Icahn School of Medicine at Mount Sinai, New York, NY 10029, USA Full list of author information is available at the end of the article Medicine, Washington University School of Medicine, St. Louis, MO 63110, USA.

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