Cancer & Metabolism

ORAL PRESENTATION Open Access

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

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From Metabolism, Diet and Disease 2014: Cancer and metabolism
Washington DC, USA. 28-30 May 2014

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.

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Published: 28 May 2014

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doi:10.1186/2049-3002-2-S1-O19
Cite this article as: Hirabayashi *et al.*: A *Drosophila* model linking diet-induced metabolic disease and cancer. *Cancer & Metabolism* 2014 **2**(Suppl 1):O19.

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