FB - Systems Biology

FB.01 - Different oxidative response between sexes in *Drosophila melanogaster* exposed to Biphenol S

Elize Aparecida Santos Musachio [1], Dieniffer Espinosa Janner[1], Marcia Rosula Poetini Silva[1], Luana Barreto Meichtry[1], Eliana Jardim Fernandes[1], Ketnne Hanna Poleto Pinto[1], Marina Prigol[1]
[1]Laboratory of Pharmacological and Toxicological Assessments Applied to Bioactiv, Universidade Federal do Pampa (Rio Grande do Sul, Brasil)

The intensification of regulation and prohibition of the use of Bisphenol A (BPA) in some countries, led the industry to use other types of bisphenols in the production of utensils and plastic packaging. Even without in-depth studies, Bisphenol S (BPS) is one of the most used substitutes in the production of products called “BPA-free”. Therefore, evaluating the toxicological action of BPS, and establishing whether there is a difference in the action of this chemical between the sexes, is pertinent information to be considered by scientists, legislators and society. Thus, our aim was to evaluate the effect of BPS on the oxidative stress biomarkers of *Drosophila melanogaster*, male and female, separately. Flies were separated by sex, divided into groups: Control (standard diet only), BPS 0.25 mM, BPS 0.5 mM and BPS 1mM (BPS mixed with standard diet). After 7 days of exposure, analyzes were performed on whole body samples to quantify reactive species (RS), lipid peroxidation (LPO), mitochondrial and cell viability. Female flies exposed to BPS 0.25, 0.5 and 1mM obtained increased levels of RS. In addition, they showed increased lipid peroxidation, decreased mitochondrial and cell viability at concentrations of BPS 0.5 and 1 mM, when compared to the control group. Men exposed to BPS (0.25, 0.5 and 1mM) also showed increased RS levels and decreased mitochondrial viability at BPS 0.5 and 1mM, when compared to the control group. However, male flies did not show changes in lipid peroxidation and cell viability. Changes in RS levels and decreased mitochondrial viability did not alter the cell viability of male flies. Thus, we conclude that BPS triggered different changes between genders, showing greater damage to female flies, and encouraging us to carry out future studies.
Keywords: Bisphenol S, Bisphenol A, *Drosophila melanogaster*
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FB.02 - Inference and analysis of a gene regulatory network of angiogenesis in an oxygen-induced retinopathy mouse model

Jhonatas Sirino Monteiro [1], Débora Guerra Peixe[1], Ricardo José Giordano[1], João Carlos Setubal[1]
[1]Departamento de Bioquímica, Instituto de Química, Universidade de São Paulo (São Paulo, Brasil)

Angiogenesis, the formation of new blood vessels from pre-existing ones, is an important therapeutic target. Nevertheless, not all patients benefit from current drugs available for angiogenesis-dependent diseases (i.e. cancer and retinopathies). To address this problem, we sought to achieve a better understanding of gene regulation in angiogenesis, which may help the identification of new targets for drug development. Previous work by our group has identified by RNA-seq several differentially expressed (DE) protein-coding genes in a mouse model of angiogenesis (OIR, oxygen-induced retinopathy). Here, we expanded on these studies to analyze the mRNA isoforms and regulatory RNAs. These data will then be integrated to generate a comprehensive gene network of the OIR model. Seven-day-old mice were placed in a hyperoxic environment (75% O_2) for five days and then returned to room air. This causes a sudden hypoxic condition leading to VEGFA overexpression, resulting in abnormal vascular growth and pathological angiogenesis. Retinas were dissected for RNA extraction and sequencing right after the return to room air, but also 3 and 5 days later. The RNA-Seq data were analyzed using bioinformatics pipelines. We identified 60 DE mRNA isoforms, of which 26 were inferred to be homologous to human isoforms. We also identified 99 DE miRNAs, of which 5 are new candidates. Most of the predicted targets for these miRNAs are related to angiogenesis. Among lncRNAs, we identified 218 DE, of which 57 are already described and 161 are new candidates. We predicted 5421 circRNAs, half of which are novel. The next step is to build the co-expression network using WGCNA to identify the interactions between mRNAs and these regulatory RNAs. Most of the RNAs that were identified as DE are promising candidates for future studies and validation as potential targets for drug therapies, or as diagnostics/prognosis biomarkers for angiogenesis-dependent diseases.
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