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Proteomic bio-makers of early placental changes and how they influence fetal growth restriction (FGR)

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Because smoking has complex and long-lasting effects on mother and child, a multidisciplinary approach is proposed to understand molecular mechanisms by which tobacco smoke exposure influences health. This is especially important, in light of commercial development of new "lower toxicity" products without convincing proof that they are less harmful since the drive to get more nicotine from these products requires more exposure to other toxins, especially Benzo[a]pyrene (BaP). In the current study, a discovery-based approach is proposed in which genomics, proteomics and bioinformatics will analyze protein networks through which BaP modulates placental proteome. The working assumption is that by understanding molecular pathways, through which smoke's toxic effects occur, and by identifying early biomarkers of cellular response, the effects of new products can be profiled for the development of toxicity tests, and for the development of less noxious alternatives to smoking. Due to the placenta's central position in smoke toxicity during pregnancy, we performed *in vitro* studies to detect BaP-responsive proteins using placental and control breast cell lines and actual placental tissues from smokers (of regular and/or new products), passive smokers and non-smokers. This required defining the total proteome profile using MALDI-MS coupled with subtractive proteomics. Samples from control and BaP treated groups were labeled with fluorescent tags and then processed for two-dimensional electrophoresis plus molecular imaging techniques. We found specific protein markers of response for which corresponding genes were analyzed using bioinformatics tools. Data generated was utilized to develop protein-protein interaction models that allowed tracking of BaP-induced molecular turbulence in relevant pathways, exposing BaP-responsive proteins that are central to protein folding, degradation, toxin's detoxification and cell cycle regulation. Interestingly, stress related cell responses and changes in glucose metabolism were also detected. In addition to the discovery of these BaP-responsive proteins, an avenue of investigation into new mechanisms through which BaP and other smoke toxins influence gene actions occur has been revealed. We believe that in addition to facilitating the testing effects of "low dose" smoking, this project will not only broaden our knowledge of BaP-toxicity, it will lay novel groundwork for understanding smoke-related fetal origins of adult disease (FOAD).

Biography

Ahmed Fadiel is a Molecular Biologist with extensive experience in Bioinformatics, Clinical Informatics, Genomics, and Proteomics. He has completed two Master degrees (MS, Cytogenetics; MS, Clinical Informatics) and a PhD in Molecular Genetics. He has years of research and teaching experience wherein he elucidates complex biological phenomena such as cancer initiation, progression and Fetal Growth Restriction. In addition, he explores protein folding and ligand-receptor interactions. He has also received extensive training in data analysis, mining, and processing and has gone on to develop numerous databases and whole genome analysis tools as well as participated in the development of several national and international research programs. He is a member of various societies and organization's has won an assortment of national & international awards, sat on various editorial & organizing boards and been the invited speaker at numerous conferences.

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