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The molecular epidemiology of aflatoxin driven stunting

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Stunting is a significant public health burden in many global regions, yet remains poorly explained by either dietary insufficiency or poor hygiene; and some are suggesting another major player must be contributing to the burden of disease. Aflatoxins are a family of well establish highly toxic metabolites from Aspergillus species that contaminate dietary staples predominantly in tropical world regions. Despite them being identified as potent liver carcinogens more than 20 years ago, approximately 0.5 billion individuals are still at significant risk of exposure. Animal data clearly indicate that aflatoxin B1 (AFB1), the most frequently occurring and toxic form additionally causes poor early life growth. In this presentation I will critique the epidemiological data that provides compelling evidence for a role of dietary AFB1 exposure and early life stunting. Cross-sectional and longitudinal studies using well established exposure biomarkers for aflatoxin reveal that both in utero and early life exposures are important. Early introduction of complimentary foods increases the risk of high levels of aflatoxin exposure in infants, further pushing educational need in breast feeding practices, while associations between early aflatoxin exposure and stunting seem clear, good mechanistic data of this affect are mostly absent or occasionally poorly developed. The mechanisms are likely complex perhaps involving direct gut toxicity, immune suppression and/or liver toxicity. Other fungal metabolites may contribute, and complex mixtures of exposure are common; but in reality stunting is likely a consequence of combination of diet, hygiene and aflatoxin. I will outline some of the larger initiatives that are being developed to investigate this mixture of contributing factors. It is important to understand their relative contribution because it is unlikely that any of the three contributors will be completely eradicated in the foreseeable future; however useful and sustainable interventions may be successful by partial improvement of each.

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Genotoxicity of gamma rays in liver, lungs and lymphocytes of albino mice

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Radiation is a process that allows movement of energy from one place to other. In the last many years use of different types of ionizing radiations in diagnosis and treatment of various diseases has been increased. These radiations contain enormous amount of energy. When they strike on any living organism they penetrate into that organism crossing cell membrane and ultimately they reach the DNA causing damage to the DNA. Contact to ionizing radiation can cause damage to living tissues, which can result in mutation and some mutations may lead to cancer. A study was designed to detect and compare the impact of Gamma Rays on the liver, lungs and peripheral lymphocytes of the albino mice. Single cell gel electrophoresis assay commonly known as comet assay was used to detect the damage to the DNA. Albino mice were used in the study and were randomly divided into four groups and named as G1, G2, G3 and C. Each group contains 10 animals. G_1, G_2 and G_3 were exposed to 1cGy, 3cGy and 5cGy of Gamma rays respectively for 1min. Group C was not exposed with any radiations and it served as negative control group. Immediately after exposure to gamma rays peripheral blood was isolated, mice were dissected; organs (liver and lungs) were minced and were subjected to comet assay. Significant damage ($P<0.001$) was observed in all three doses in liver, lungs and lymphocytes when compared with control. G1 and G2 were not significantly different from each other; however there was significantly higher DNA damage in G3 as compared to G1 and G2. Results of the present study indicate that even low doses of Gamma rays for short period are capable of causing DNA damage in blood and different organs of the mice. Damage further increases with increase in intensity.

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