Carcinogenic Crops

Analyzing the Effect of Aflatoxin on Global Liver Cancer Rates

Tree nuts and groundnuts, along with maize and other grains, can harbor aflatoxins, naturally occurring fungal metabolites that have been identified as risk factors for developing liver cancer. This association has most often been seen in people infected with hepatitis B virus (HBV). A new study examines the aflatoxin/HBVrelationship to offer the first quantitative risk assessment of the number of liver cancer cases worldwide caused by aflatoxin [*EHP* **118:818–824; Liu and Wu**].

Although a relatively rare malignancy in developed countries, liver cancer is a common health threat in developing regions of the world including Southeast Asia, China, and sub-Saharan Africa. These same regions have higher prevalence of HBV infection as well as higher levels of aflatoxin contamination in food due to a lack of resources to control the fungi *Aspergillus flavus* and *Aspergillus parasiticus*, which infiltrate crops and produce aflatoxin. Research has shown that individuals with chronic HBV infection and aflatoxin exposure are up to 30 times more at risk for liver cancer than uninfected individuals exposed to aflatoxin.

Aspergillus flavus

In the current study, researchers analyzed information on food consumption patterns, aflatoxin biomarker levels in serum and urine, HBV prevalence, and population size in different world regions to quantify the subsequent risk of developing liver cancer. The investigators found that consumption of maize and groundnuts was higher overall in African and Asian countries than in wealthier, more developed nations, leading to increased aflatoxin exposure. However, risk of aflatoxin-induced liver cancer could vary widely within a given nation: urban populations with more diverse diets had lower aflatoxin exposures than their rural counterparts, and there also was a lower HBV prevalence in urban populations.

The authors concluded that uncontrolled exposure to aflatoxin may cause 4.6–28.2% of all liver cancer cases globally, with China, Southeast Asia, and sub-Saharan Africa bearing the brunt of the burden. This broad range reflects the uncertainty and variability of the available data on aflatoxin exposure and HBV prevalence. One thing does seem certain, they write: if more interventions to control aflatoxin and its health risks (for instance, improved storage protocols and vaccination for HBV) were administered in regions where they are most needed, liver cancer incidence could be significantly reduced worldwide.

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Stress and the City

Measuring Effects of Chronic Stress and Air Pollution

Scientists have begun to consider an association between chronic psychosocial stress and increased susceptibility to adverse physiologic effects from exposures to air pollution. Although epidemiologic evidence tends to support that hypothesis, it has proven difficult to untangle the complex web of exposure effects, stressors, and mechanisms behind the potential differences in susceptibility. A new laboratory study documents different responses to air pollution in stressed and nonstressed rats, supporting the epidemiologic evidence that chronic stress may alter respiratory responses to air pollution [*EHP* **118:769–775; Clougherty et al.**]. The protocol described in the study may provide a template for future controlled experiments to explore associations between psychosocial and environmental factors.

The authors randomly divided 24 rats into four groups—stress plus exposure to uniform doses of concentrated ambient particulates (CAPs), nonstress plus exposure to CAPs, stress plus exposure only to filtered air (FA) from which particles were removed, and nonstress plus exposure to FA. Each animal in the two stress groups was individually put into the cage of a dominant male rat for 20 minutes at a time, twice per week; the older male behaved territorially and aggressively toward the younger test rodents, which were able to protect themselves from scratches and bites by retreating into a Plexiglas tube in the cage. On the day following each stress exposure, animals were exposed to either CAPs or FA for 5 hours. All exposures occurred at the same time each day to account for normal diurnal variation in the animals' stress hormone and activity levels.

Respiratory responses were monitored continuously during the CAPs/FA exposure periods. Both CAPs-exposed groups had a significant response to the exposure, but the stress group exhibited greater breathing frequency, shorter inhalation and exhalation times, and lower expiratory flows and tidal volumes—that is, a rapid, shallow breathing pattern—compared with the nonstress group.

The researchers also observed changes in levels of several systemic inflammatory biomarkers associated with airway disease. Stress alone or CAPs exposure alone elevated some biomarkers, but only the group exposed to both stress and CAPs showed elevated levels of C-reactive protein and increased numbers of lymphocytes and monocytes, indicating the combination of exposures may have a different effect on inflammation than either exposure alone. This finding provides evidence that chronic stress may increase susceptibility to effects of air pollution on respiratory diseases.

As far as human exposures go, the authors note that social stressors (such as poverty and violence) and environmental exposures (such as traffic-related pollution) may be spatially correlated; "thus," they write, "the most pollution-exposed communities may also be the most susceptible." Although this study was small, it points wto important new ways to characterize the combined physiologic impact of those real-world phenomena.

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