Lifestyle and climate factors interact to cause neuroendocrine, cardiometabolic, and immune dysfunction

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Citation:

Monsees, M., T. Jackson, D. Alewal, Mette C. Schladweiler, D. Davies, M. Hazari, A. Farraj, A. Farraj, AND U. Kodavanti. Lifestyle and climate factors interact to cause neuroendocrine, cardiometabolic, and immune dysfunction. NC Society of Toxicology Annual Meeting, Durham, NC, October 19, 2022.

Impact/Purpose:

These data demonstrate that lifestyle factors (e.g. diet) can interact with climate-induced changes in temperature and/or exposure to air pollution to induce neuroendocrine, metabolic, and immune effects, and exacerbate chronic disease outcomes.

Description:

Current climate scenarios predict more frequent and longer-lasting heatwaves alongside a rise in ambient particulate levels from widespread wildfires. These conditions are predicted to exacerbate mental health crises and chronic metabolic and immune disorders, especially in vulnerable populations. We hypothesized that high temperature housing (HT), high cholesterol diet (HCD), and weekly subchronic wildfire eucalyptus smoke exposure (WFES) would interact to disrupt neuroendocrine, cardiometabolic, and immune health of rats. Male WKY rats (4-week-old) housed at HT just above thermoneutrality (~31 °C) for 13 weeks had a substantial reduction (>30%) in body weight gain and increased lean mass compared to 22 °C (RT) housed rats. HCD increased serum cholesterol and resulted in fatty livers; conversely, HT decreased serum cholesterol. Further, HT rats had lower basal glucose, and glucose tolerance testing (GTT) revealed that HT rats had increased glucose clearance compared to RT. The GTT effects of HCD were modified by housing temperature; in RT housed rats, HCD decreased glucose clearance, but in HT rats, HCD increased glucose clearance. In all groups, rats exposed to wildfire smoke (~7 mg/m3 x 1hr/d x 1d/wk x 13 wks) showed decreased glucose clearance. Pyruvate and insulin tolerance testing revealed that HT-related changes in glucose clearance were neither due to gluconeogenesis nor insulin resistance. HT rats showed chronic indicators of systemic stress, including decreased circulating lymphocytes and bronchoalveolar lavage neutrophilic inflammation, that was exacerbated by HCD and wildfire smoke. Atherogenic vascular effects of HCD tested using an ex vivo aortic ring bath were again modified by temperature; at RT, HCD or WFES amplified vasoconstriction and minimized vasorelaxation (HCD>WFES), with a greater cumulative effect observed with the combination of HCD and WFES. At HT, the effects of HCD or WFES were mitigated. These data demonstrate that lifestyle factors (e.g. diet) can interact with climate-induced changes in temperature and/or exposure to air pollution to induce neuroendocrine,

metabolic, and immune effects, and exacerbate chronic disease outcomes. U.S. EPA Institutional Animal Care and Use Committee approved protocols prior to experiments. This abstract does not necessarily reflect US EPA policy.

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