

those with sufficient intake of all 3 essential nutrients (5%, 95% CI: -7, 19%). Conclusions: Our preliminary findings suggest that women with deficient calcium, zinc, and/or iron intake have increased absorption of cadmium from the diet.

Evaluation of the ERA5-based UTCI on mortality data in Europe

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TPS 664: Climate change: temperature effects 2, Exhibition Hall, Ground floor, August 27, 2019, 3:00 PM - 4:30 PM

The study was conducted on behalf of the MCC Collaborative Research Network (<http://mccstudy.lshtm.ac.uk/>).

Background: ERA5 is a novel climate reanalysis product from the ECMWF (European Centre for Medium-Range Weather Forecasts). It provides estimates of surface and atmospheric parameters at much higher resolution (31 x 31 km) than any previous climate reanalyses.

Methods: From ERA5 parameters the Universal Thermal Climate Index (UTCI) can be computed as a gridded parameter at the ERA5 resolution for the whole European continent. Using daily mortality data from European members of the MCC Collaborative Research Network, we will explore the potential of the ERA5-based UTCI as a health-related tool by evaluating UTCI-mortality relationships in 20 cities across 10 European countries. Distributed Lag Nonlinear Models (DLNM) will be used to analyse exposure-response relationships between mortality and UTCI in selected cities calculated from (i) the ERA5 reanalysis and (ii) station-based data.

Results: Preliminary results suggest that both ERA5- and station-based UTCI explain mortality in European cities in a comparable way.

Conclusions: The comparison of the exposure-response relationships between the ERA5- and station-based UTCI is an important step towards the development of a pan-European health-hazard warning system that would be able to assess thermal conditions in locations where high-quality station data are not available.

Persistent organic pollutants and the association with maternal and child thyroid hormone levels

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TPS 634: Health effects of pops, voc and other chemicals, Johan Friso Foyer, Floor 1, August 27, 2019, 3:00 PM - 4:30 PM

Background/Aim: Thyroid hormones regulate a wide array of biological functions in the human body. Exposure to persistent organic pollutants (POPs) has been linked to metabolic disturbances in epidemiological and animal studies, but their effects on thyroid hormone homeostasis remain controversial. We evaluated the association between maternal POPs exposure and thyroid function in 850 mother-child pairs from the RHEA pregnancy cohort in Crete, Greece.

Methods: Concentrations of several PCBs, dichlorodiphenyldichloroethene (DDE), and hexachlorobenzene (HCB) were determined in first trimester maternal serum. Thyroid hormones (thyroid stimulating hormone (TSH), free T(4), and free T(3)) were measured in serum samples

collected from mothers (1st trimester) and their children at 4 years of age. Maternal hypothyroxinemia was defined as TSH within the normal range and fT4 below the 10th percentile. Adjusted associations were obtained via multivariable regression analyses.

Results: Elevated prenatal DDE levels (third vs. first tertile) were associated with 14.3% (95% CI: -27.2, 0.9) lower maternal TSH levels. Women in the medium and high tertiles of PCBs, had 18.1% (95% CI: -30.6, -3.4) and 16.61% (95% CI: -31.6, 1.7) lower TSH levels respectively, compared to women in the lowest tertile. Elevated odds of hypothyroxinemia were observed for women in the highest tertile of PCBs exposure (OR=2.5, 95% CI: 1.0, 6.3). Girls born to mothers within the high HCB tertile of exposure had -7.9 ng/dL (95% CI: -15.2, 0.03, p-interaction=0.027) lower mean concentrations of FT4 compared to girls born to mothers in the lowest tertile. We did not observe other associations between maternal POP concentrations and child thyroid hormones.

Conclusions: The present results suggest that low-level exposure to POPs can alter maternal and offspring thyroid homeostasis. Considering the importance of thyroid hormones during gestation and early life stages, further work is needed to examine the underlying mechanisms.

Association between prenatal exposure to air pollution and inflammatory markers in children

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TPS 911: Air pollution, epigenetics, biomarkers, Exhibition Hall, Ground floor, August 26, 2019, 3:00 PM - 4:30 PM

Background/Aim: Early-life exposure to ambient air pollution has been associated with adverse health effects in children but little is known about its potential effects on cytokine levels. We evaluated the association of pregnancy exposure to particulate matter with child inflammatory biomarkers in 500 mother-child pairs from the RHEA pregnancy cohort in Crete, Greece.

Methods: Mean concentrations of particulate matter with an aerodynamic diameter of less than 2.5 µm (PM2.5) and less than 10 µm (PM10) during pregnancy were estimated at maternal home addresses with temporally adjusted land-use regression models. Levels of several inflammatory biomarkers were determined in child serum at 4 years of age via immunoassay. Exposure-outcome associations were assessed using log-binomial or Poisson regression with robust variances, in cases of non-convergence. Potential effect modification from maternal and offspring characteristics was examined by introducing interaction terms in multivariate models.

Results: A 5 µg/m³ increase in concentration of PM2.5 and PM10 during pregnancy was associated with an increased risk of high levels (in the 5th quantile) of child interleukin-6 (IL-6) levels at 4 years (RR=2.68, 95%CI: 1.38, 5.20 and 1.28, 95%CI: 1.04, 1.57, respectively). Effects of prenatal exposure to both PM2.5 and PM10 remained significant only for non-smoking mothers in stratified analysis by maternal smoking status (p-interaction: 0.037 and 0.071, respectively). Child overweight/obesity (p-interaction: 0.013 and 0.001) and asthma status (p-interaction: 0.007 and 0.001) more than doubled the effects of maternal exposure to PM2.5 and PM10 on IL-6 at 4 years. Similar effects were found with other inflammatory biomarkers under study.

Conclusions: Our results indicate alterations in systemic inflammatory markers in 4-y-old children in relation to prenatal exposure to traffic-related air pollution. Further work is needed to examine