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Environmental Pollution and GEP-NENS – Is There an Association?

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BACKGROUND

Incidence of gastroenteropancreatic neuroendocrine neoplasms (GEP-NEMs) is increasing, but etiology of sporadic, non-familial disease remains obscure. Behavioral risk factors like smoking and alcohol consumption may be associated with GEP-NENs, yet environmental factors potentially associated with GEP-NENs remain unexamined. We regressed age-adjusted incidence rates for GEP-NENs cases in California (CA) on county-level pollution data to determine if an association between GEP-NEN incidence and environmental pollutants exists.

METHODS

GEP-NEN cases were obtained from the CA Cancer Registry for the years 2000-2012 and age-adjusted for each of 54 of 58 CA counties. Pollution scores were obtained from the Cal EnviroScreen 3.0 (CES3) database which contains census-tract level measures of exposure to several types of pollution, including air (diesel and fine particulate matter 25 ppm; ozone; residential proximity to areas of high traffic), water (drinking water quality; groundwater toxins), proximity to hazardous and solid waste sites, pesticide exposure, and an overall pollution burden score. CES3 census tract scores were averaged for each county; county-level GEP-NEN incidence rates and average pollution scores were then analyzed by linear regression. Incidence rates for gastrointestinal NENs (GI-NENs) and pancreatic NENs (P-NENs) were calculated and analyzed separately.

RESULTS

There were 8,580 GI-NENs and1,491 P-NEN cases. Median age at diagnosis was 58 for GI-NENs and 59 for P-NENs. GI-NENs were evenly distributed by sex, and a majority of cases were white race (GI-NENs 50%; P-NENs 59%). Weak or zero correlations were observed between GI- or P-NEN incidence rates and pollution. For GI-NENs the R-squared (R2) estimate from regressing incidence rate on pollution burden score was 0.06, indicating this variable explained 6% of the variation in GI-NEN county level incidence rate. For GI-NEN incidence the R2 measure for air pollution categories were the highest observed (diesel R2=0.07; traffic exposure R2=0.08). These positive correlations contrasted with results from regression of GI-NEN incidence on scores for drinking water, ozone, or groundwater which yielded R2s close to zero. Similarly, PI-NEN incidence regressed on air pollutants yielded low R2s (diesel PM R2=0.02; traffic exposure R2=0.02) but a nearly zero R2 for pollution burden score (R2=0.0004) and other categories of pollutants.

CONCLUSIONS

We observed a slight association between age-adjusted GI- or P-NEN incidence rates and county-level scores for exposure to diesel particulate matter, and exposure to air from heavy vehicular traffic. Future studies with more fine-grained measurements of air pollution exposure and GEP-NEN incidence rate may uncover a stronger association between GEP-NENs and the environment.

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