Novel bioindicators of urban stormwater runoff toxicity in developing fish

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Urban stormwater runoff adversely impacts the health of marine life. A more holistic approach to assessing water quality in urbanized areas should include indicators of fish health that are linked to major pollutants, like polycylic aromatic hydrocarbon (PAH) mixtures. Specific PAH families are toxic to the developing fish heart. Thus, bioindicators of PAH cardiotoxicity are needed for assessing the toxicity of runoff, and also the efficacy of green stormwater infrastructure. Both toxic and non-toxic PAHs induce the aryl hydrocarbon receptor cytochrome P4501A (AHR-CYP1A) pathway, a widely-accepted bioindicator of PAH exposure. CYP1A is thought to detoxify parent PAH compounds, producing non-toxic metabolites. My previous research used zebrafish and a simple PAH mixture of chrysene (CHR) and phenanthrene (PHN), showing that metabolic interactions between these PAHs increased the potency of mixtures. Here, I examined whether toxicity came from parent PHN or a metabolite. A liquid chromatography coupled to tandem mass spectrometry (LC-MS/MS) method was developed to quantify hydroxylated phenanthrene metabolites (OH-PHNs) in zebrafish embryos. Cardiotoxicity was measured as reduced heart-rate. Results illustrated that most OH-PHNs increased as CHR concentration increased. The most abundant metabolite was dihydroxilated phenanthrene which strongly correlated with reduced heartrate.Therefore, OH-PHNs are novel effects-linked bioindicators of urban stormwater runoff cardiotoxicity; toxicity results from metabolite, not parent PHN. Further interpretation suggests that the OH-PHN-producing enzymes act independently of AHR-CYP1A, but still belong to the AHR battery. Moreover, adapting LC-MS/MS to quantify OH-PHNs in zebrafish embryos is novel and has wide-ranging applications.