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Adverse effects of MeHg on brain development and function have been documented in zebrafish embryos (Lemos et al., 2004) and larval zebrafish (Monteiro et al., 2004) exposed in the water to as low as 0.01  $\mu\text{M}$  MeHg in the early development stages. In this study, we hypothesized that the toxic effects of MeHg, at low concentrations, are a result of the reduced production of toxic intermediates during the MeHg methylation cycle. These intermediates have been observed in fish and other vertebrates at concentrations that are lower than those present in the environment (Rudel et al., 1998; Pohl et al., 2003). Because selenium acts as a cofactor for glutathione peroxidase and thioredoxin reductase, the concentration of selenium in the diet can be used as an indicator of the rate of MeHg methylation in fish. We used a two-generation feeding assay to assess the ability of this animal model to metabolize and methylate MeHg in an environmentally relevant manner and to test the effects of reduced dietary selenium on this process. Exposure to 40  $\mu\text{g}$  Se/g diet in the first generation (F1) significantly increased the body weight of juveniles exposed to MeHg in F1 to an extent that was comparable to the effect of Se deficiency. The most important observation of the present study was that the effects of low-level MeHg exposure were detected only at later stages in the lives of

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the mice. Except for the central-area occupancy in OPF in MT-null females, no statistically significant effects of MeHg were observed in any of the three behavioral tests at 12 weeks of age. In contrast, significant effects were observed in all three tests at 52 weeks of age. The brain Hg concentration of the exposed groups had leveled off and was not distinguishable from the non-exposed group at 13 weeks of age, immediately after the first phase of the behavioral testing. Therefore, in the present study, there was a latency period in which the dose and effects could not be detected, although effects were observed 9 months later. Another notable observation was that the emergent manifestation of toxicity was also recognized in the suppression of body weight (except for wild-type females), which only became apparent on or after 28 weeks of age.

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Accompanying this paper is an annotated bibliography (Callan, Callan and Hall, forthcoming; Callan et al., forthcoming ) to provide some contextual and historical references for the issues discussed here. The annotated bibliography provides links to the objects of study presented in the paper as well as papers that provide further background for the analysis. The bibliography focuses on recent work on product hopping and hoppers, as well as to the broader theoretical and historical literature on the topic. The scholarly literature on product hopping has largely moved beyond the purview of antitrust law, in part because of the need to engage with the larger scholarly literature, but also because legal analysis is often divorced from empirical analyses and history. The annotated bibliography synthesises the scholarship in these areas, and provides as much as possible about the actors and stories involved in product hopping, the social and cultural contexts in which these events took place, as well as how the consequences of product hopping are informed by the particularities of these events. This paper has primarily focussed on the counterfactual analysis of product hopping claims. However, a key reason for doing so is that a market wide ban, rather than a case by case analysis of product hopping, could fundamentally disrupt the classical theatre market. It is possible that such a ban could allow for products to be sold and performances to be staged based on the quality of the individual products and performances, rather than the level of interchangeability of the products and performances. Thus, whilst a targeted assessment of the role of product hopping within contemporary classical theatre would be illuminating, this analysis should be balanced by the possibility that such a ban could have unintended negative consequences. 5ec8ef588b

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