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## Late insights into early origins of disease

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Burgeoning research into the developmental programming of organ functions has opened a new perspective of environmental health science and its policy implications. At the beginning of my 25-year tenure in Odense, I studied lead poisoning and the role of predisposition in shaping the outcome of developmental neurotoxicity. I realized that some effects might not be clinically visible in the short term, but could cause a decrease in reserve capacity that would be silent only until unmasked. I was concerned that other environmental pollutants might cause similar subclinical effects and therefore, in 1985, initiated studies of methylmercury with Pal Weihe. Our first paper on 7-year-old Faroese children with prenatal methylmercury exposure was rejected by major journals but finally appeared in 1997. The following year, the U.S. White House invited 30 international experts for a workshop to identify the weaknesses related to the evidence on methylmercury toxicity. However, as we began to study the various types of uncertainties, we found that they mainly caused an underestimation of methylmercury toxicity. This insight then inspired additional studies of other neurotoxicant exposures. Epidemiological research into delayed consequences of developmental exposures is fraught with difficulties that tend to blur the picture, and our innate scepticism against new research findings can then inadvertently lead to propagation of toxic exposures that can cause severe adverse effects on the next generation. This new research angle therefore calls for a critical re-appraisal of research paradigms in environmental health and our application of research data for prevention purposes.