

Science for Environment Policy

Pregnancy and early life are critical stages for environmental chemical exposure

Evidence to indicate that early exposure to environmental contaminants may result in a greater risk of serious disease later on in life has been presented by researchers in a new White Paper. Measures to prevent non-contagious illnesses, such as diabetes, asthma and cancer should therefore focus on the early stages in life, including the time spent in the womb, they suggest.

All complex diseases are influenced in some way by the environment. Scientists now have a better understanding of the role of these environmental influences on disease, and while the influence of fixed genetic factors is also important, it is not as significant as previously believed.

The White Paper¹ summarises research on the impacts of contaminants in our environment on human biological development, and proposes general recommendations for policy and research. Environmental chemical exposure has been implicated in various non-communicable diseases and major public health problems, such as obesity, diabetes, hypertension, cardiovascular disease, asthma and allergy, infertility, some cancer types, osteoporosis and neurocognitive development. A particularly vulnerable life stage is development in the womb and just after birth, as the foetus or baby are exposed to environmental chemicals via the mother.

Often, the impacts of chemical exposure are not immediately noticeable and some environmental contaminants, especially endocrine disrupting chemicals (EDCs), can result in epigenetic changes that are not observable at birth, but lead to increased risk of dysfunction and disease later in life. For some tissues and organs, such as the brain, reproductive and immune system, developmental vulnerability to adverse effects from environmental chemicals continues through the neonatal period and perhaps into puberty.

EDCs are of particular concern as these disrupt the hormones that carry signals in the body. EDCs can have effects at very low doses and 900 chemicals are now suspected to be EDCs. Examples of EDCs known to affect disease susceptibility as a result of developmental exposures in animal models include some organophosphate and organochlorine pesticides, bisphenol A (from polycarbonate plastics), phthalates (a softener in plastics), nicotine (from tobacco smoking), and polybrominated diphenyl ethers (flame retardants). Often chemicals can have cumulative or interactive effects, when experienced at the same time or at different stages of life. These effects can also interact with nutritional deficiencies and imbalances. The authors pointed to several similarities between the effects of nutritional imbalances and exposure to environmental chemicals.

Because exposure to environmental contaminants tends to have a developmental impact which is often not physically observable until later in life, the researchers stress the importance of identifying and studying effective 'biomarkers' that can be measured at birth. Biomarkers are biological characteristics that can be measured to indicate the state of health, for example, anogenital distance as a biomarker of feminization. In the future it is hoped that epigenetic marks could serve as relevant biomarkers.

The researchers strongly recommend that public policy focuses on disease prevention in key developmental stages and reducing environmental chemical exposure pre-pregnancy, during pregnancy and during the first few years of life. The current data are sufficiently robust and replicable to require a policy and public health response they suggest, but further research is needed to identify the exposure pathways, i.e. exactly how specific contaminants reach and interact with humans, through the use of biomarkers, and to establish a convincing link between epigenetic alterations and later disease.



27 September 2012
Issue 299

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Source: Barouki, R.,
Gluckman, P.D.,
Grandjean, P., Hanson, M.
& Heindel, J.J. (2012)

Developmental origins of
non-communicable
disease: Implications for
research and public health.
Environmental Health.

11(42): 1-9. This study is
free to view at:

[www.ehjournal.net/content/
11/1/42](http://www.ehjournal.net/content/11/1/42)

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Theme(s): Chemicals,
Environment and health

The contents and views
included in Science for
Environment Policy are
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European Commission.

To cite this

article/service: "Science
for Environment Policy":

European Commission DG
Environment News Alert
Service, edited by
SCU, The University of the
West of England, Bristol.

1. The paper was an outcome of
the PPTOX III conference on
Environmental Stressors in the
Developmental Origins of
Disease: Evidence and
Mechanisms, attended by
leading researchers on early
exposure to environmental
contaminants and diseases.