Increase of wildlife cancer: an echo of plastic pollution?

Thomas Erren, Dominique Zeuß, Frank Steffany and Benno Meyer-Rochow

The Science and Society article Wildlife cancer: a conservation perspective (*Nature Rev. Cancer* **9**, 517–526 (2009))¹ sparked our interest. Its authors McAloose and Newton noted that “in the marine environment, increases in spontaneous benign and malignant tumours have been identified over the past two decades”. With regard to the causes of wildlife cancers, the authors acknowledge that besides mechanisms such as allograft transmission and viral oncogenesis, environmental contributors to cancer in animals should be identified as a priority.

Although a range of industrial and agricultural pollutants with effects on the environment has been identified, these defined contaminants are usually confined to some locality and are therefore unlikely to be the cause of the suggested global increase of wildlife cancers, particularly in oceans. This is why we wish to attract attention to a global, rather than local, candidate that possibly contributes to the global increase in cancers: plastics (or synthetic monomers²), in all their variety.

Not only has the amount of plastic debris been rapidly increasing in the past few decades, but the debris also poses identified health threats². That plastic materials potentially cause or at least contribute to cancers in many species, including humans, is exemplified by experiments with bisphenol A (BPA). Suggestions that the oestrogenic activities of this component of plastics may contribute to cancer came from Krishnan and colleagues in the early 1990s³. Since then, research into BPA, which began

some 100 years ago^{4,5}, has been pursued at an accelerated pace. Importantly, perinatal exposure to the environmentally borne oestrogen mimic BPA (used as a model agent for endocrine disruption) has been linked with mammary and prostate cancer in humans⁶. At this stage, we do not know whether BPA, or other xeno-oestrogens that are released from plastics⁷, can cause cancers, including those affecting humans. However, the ubiquitous plastic debris seems to be capable of representing a critical burden. It is noteworthy that in rodent experiments even very low doses of BPA experienced in fetal and/or neonatal life can lead to cancer development through so-called ‘epigenetic programming’ (REFS 8,9).

In this context, marine species can have two roles: first, in line with McAloose and Newton, as sentinel and sensitive indicators of an environmental threat to many species, including humans; and second, as possible vectors of plastic components. Indeed, the plastics are, with rare exceptions, not biodegradable, but they can bio-accumulate through the food chain to ultimately reach humans through ingestion.

In conclusion, we should ask ourselves whether humans are aware of, and indeed prepared for, the consequences of an ‘echo’ of the plastic age. Indeed, like an echo, the effects of plastics and the consequences of global plastic pollution may reach us some time after our actions — that is, producing huge amounts of plastics at economically reasonable, but evolutionarily detrimental, costs. The time it takes before the echo reaches us is

likely to be determined by the time it takes to degrade the plastic materials (we do not know the half-life of most plastics, which could be centuries). In the meantime, compounds we know little of enter the marine and terrestrial food web, ultimately reaching the human body. Perhaps the global increase in wildlife cancers is a ‘wake-up call’ at the right time.

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