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Commentary: Plastic ocean and the cancer connection: 7 questions and answers

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ABSTRACT

A plethora of recent scientific reports testifies to challenges the world is facing from an ever-increasing marine plastic pollution. Toxicological concerns have been put forward, but possible links between the now ubiquitous synthetic polymers and human as well as wildlife cancers remain to be investigated. Hence, this commentary which addresses seven questions. Given numerous uncertainties on the factual impacts of plastics, we should embark on empirical studies into the validity of biologically plausible links between plastic residues and cancers and concomitantly consider ways to reduce plastics in the world within and around us.

1. Introduction

Modern life without plastics is unimaginable, but so is also the sea. Transported by currents and dispersed by the wind to the remotest parts on Earth, plastics big and small have pervaded all corners of the globe (Moore 2011; Bergmann and Klages, 2015; Seltenrich, 2015; Tibbetts, 2015). There is no disagreement on the unsightliness of beaches strewn thick with piles of plastic items. Disconcertingly, debates surrounding plastics' impact on human health may have just started (Erren et al., 2015). In this context, this commentary attempts to systematically draw attention to possible causal links between an ever increasing plastic pollution and human as well as wildlife cancers. That the increasing marine plastic pollution causes growing concern is evinced by numerous summaries of recent insights (Ryan et al., 2009; Letasiova et al., 2012; Kwon et al., 2014; Law and Thompson, 2014; Jambeck et al., 2015; Lusher et al., 2015; Tibbetts, 2015; Van Franeker and Law, 2015). With questions regarding the toxicological burden already identified (Glausiusz, 2014; Law and Thompson, 2014; Seltenrich, 2015), studies to include possible links between synthetic polymers and cancers appear to be timely and imperative. It is in this context that we pose seven questions.

Q1. What actually are "plastics"?

By definition plastics are solid materials, which are semi- or fully synthetically produced polymers composed of chemically strung-together monomers of organic molecules. Three major kinds are distinguished: (a) natural polymers like cellulose and starch derivatives, but also polyesters made by bacteria like for instance PHA (poly[3-hydroxylalconate]), a plastic resembling polypropylene; (b) carbon chain polymers like the biodegradable polyvinylalcohol (PVOH), but also the non-biodegradable polyvinylchloride (PVC), PE (polyethylene), and polypropylene (PP); (c) a polyglycolic acid derivative (PGA). Furthermore a host of so-called plasticisers need to be mentioned that are routinely added to plastics, especially to PVC and other non-biodegradable carbon chain polymers. Moreover, there is bisphenol A (BpA), serving as an antioxidant in combination with some platicisers and used as a strengthening agent in the manufacture of polycarbonate plastics as well as epoxyresins.

Q2. How long does it take to break plastics down?

The speed with which plastics are broken down depends on the specific features of the polymers and a variety of additional factors, but half-lives with the exception of biodegradable plastics, are generally long and may even last centuries (Barnes et al., 2009). However, since successful enzymatic breakdown is only possible for biodegradable plastics (Kale et al., 2007) and most

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conventional plastics and bulk plastic wastes in the environment are not biodegradable, the enzymatic breakdown does not play a significant role in the reduction of overall plastics in the environment. Moreover, breakdown not only depends on the chemical composition of the plastic, but also on its shape, size and colour. A large surface area facilitates enzymatic attack and flexibility and bendiness of the plastic help to position it optimally for enzymes to work on it. The enzymatic breakdown of a polymer depends on the specificity of the enzyme: the higher the molecular weight of the polymer, the smaller the chance that the polymer can be intracellularly digested by bacteria. Enzymes degrade plastics from the surface and oxygen availability expedites aerobic biodegradation with UV-radiation, higher temperatures (but not of a magnitude that kills microorganisms) and pH values lower and higher than 5 providing synergistic support.

Chemical reactions like hydrolysis can also play an important role and to be digestible hydrolysability is a prerequisite for a polymer's biodegradation. Double-bonds as well as oxygen and nitrogen atoms in the molecular skeleton are supportive of hydrolysis while side chains, ring structures and methyl groups as well as a high degree of crystallisation hinder it. Porosity, on the other hand, leads to the liberation of plasticisers and additives and, moreover, allows microorganisms to colonise the interior of the plastic. Surface features of the plastic determine not only which organisms and how many of them may be accommodated but also the degree to which harmful chemicals like heavy metals will be absorbed. In this way floating plastics (contamination is greatest in the uppermost layer known as the sea surface microlayer) can represent a habitat for some, and a poison trap for others. Once the absorption capacity is reached, the sinking material releases its substances to the surroundings and eventually the sediment (Michael et al., 2005).

Ultimately, no matter how long its half-life and how well shielded from UV-raditaion a plastic item is, any plastic material will be broken down into nano-sized particles “through a combination of photo-degradation, oxidation and mechanical abrasion” (Ryan et al., 2009). Although bacterial activity does play a role in the break down, it is mechanical abrasion, which is the biggest source of micro-plastics (Andrady, 2003; Hammer et al., 2012). Plastic residues and metabolites, i.e., polymer components will end up accumulating at the tip of the food pyramid and can then reach humans, including those that do not directly consume marine products since the so-called marine by-catch, turned into fishmeal and other types of animal feed, is given to poultry, pig and may be used as fertilizer.

Q3. What do “case-studies” of microplastics in food-webs convey?

A brief look into how microplastics can end up at the tip of the food chain is illuminating. Lower in the food chain are usually organisms that are either filter feeders or ingest substrates like sand and detritus, food sources therefore which are destined to contain microplastics, often referred to as nurdles. The risk of chemicals from plastics in the environment ending up in wildlife was thoroughly reviewed by Teuten et al. (2009). Data available from the Mediterranean mussel *Mytilus galloprovincialis*, a filter feeder, showed a high potential for bioaccumulation of BpA, nonylphenol and triclosan (Gatidou et al., 2010). Based on dry weight the following concentrations were already present before the exposure took place: BpA 404 ng/g, triclosan 461 ng/g, nonylphenol 158 ng/g. This means that a person weighing 75 kg consuming 1 kg mussels, will ingest ca. 0.4 mg BpA, which is less than the oral reference limit of 1.2 mg/d in a 75 kg heavy person. However, as pointed out (Erren et al., 2013) with reference to Vandenberg et al. (2007, 2009), it is currently being debated as to whether the reference value ought not to be lowered.

To unambiguously show that the concentrations of plastic additives in the tissues of animals are actually the result of microplastic ingestion or not is a problem. However, results by Browne et al. (2013) on micro-plastic ingestion by worms and observations by Van Cauwenberghe and Janssen (2014), who demonstrated the presence of micro-plastics in the tissues of the filter-feeding *Mytilus edulis* and *Crassostrea gigas* and warned of a possible threat to food safety, strongly indicated that ingested micro-plastic material could be the source of plastic-derived chemicals in the tissues. Very strong evidence that this is indeed the case comes from a recent study by Tanaka et al. (2013), who suggested that chemicals like BDE209 and BDE183 identified in the stomachs of 3 out of 12 puffins (*Puffinus tenuirostris*) caught in the wild would have had to come from plastics ingested by the birds. According to Rochman et al. (2013) the release of additives is highest in aged and brittle plastics and according to Koelmans et al. (2013) and Endo et al. (2013) facilitated in the intestine by gut fluids with “high levels of DOC and surfactants” (Koelmans et al. 2014).

According to Eyerer and Elsner (2004), all additives, whether they be stabilisers, softeners, fire resistors, co-polymers etc. are potentially more harmful than monomers and could possibly lead to health deficits. In particular additives like BpA (Welshons et al., 2006; Rubin, 2011), phthalates (Markman et al., 2007) and nonylphenol, a degradation product of the additive tris(nonylphenyl) phosphate (TNPP) (Muncke, 2009), are all suspect and could threaten the health of an organism via “endocrine disruption” (Soto et al., 1991) or “epigenetic disruption” (Bernal and Jirtle, 2010).

Q4. Could plastics or their residues contribute to cancer?

Oppenheimer et al. provided first reports of experimental links between embedded plastics and tumours in rodents more than half a century ago (Oppenheimer et al., 1953). However, whether plastic residues factually cause or contribute to the development of internal cancer is not clear.

Observations by Krishnan and colleagues in 1993 (Krishnan et al., 1993) were compatible with suggestions that oestrogenic activities of BpA, as a key component of plastics, may contribute to the development of cancer. Research into BpA, which already began some 110 years ago (Zincke, 1905; Dodds and Lawson, 1936), has been pursued at an accelerated pace in the past decade. BpA is commonly used as a model agent for endocrine disruption and perinatal exposures as this environmentally borne oestrogen mimic has been associated with mammary and prostate cancer in humans for at least ten years (Maffini et al., 2006). With regard to possible chains of cancer causation, intrauterine exposures of foetal and/or neonatal life to even low doses of BpA may contribute to cancer development via so-called ‘epigenetic programming’ (Prins et al., 2007; Vom Saal and Myers, 2008). BpA has been linked to an elevated risk of developing breast cancer (Doherty et al., 2010) and prostate carcinoma in adult humans (Soto and Sonnenschein, 2010; Tarapore et al., 2014). Overall, links between perinatal bisphenol A (BpA) exposures and mammary and prostate cancers as well as hepatic tumours in rodents (Maffini et al., 2006; Acevedo et al., 2013; Weinhouse et al., 2014) do provide plausible candidate mechanisms how plastics may contribute to the development of cancer.

With regard to occupational settings, plastics-associated styrene has been classified by the International Agency for Research on Cancer [IARC] as possibly carcinogenic [Group 2b] in 2002 (IARC, 2002) and vinyl chloride (PVC, VCM) as carcinogenic [Group 1] in 2012 (IARC, 2012). Also recently, breast cancer risks have