



Novel approaches to improving the chemical safety of the meat chain towards toxicants



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ABSTRACT

In addition to microbiological issues, meat chemical safety is a growing concern for the public authorities, chain stakeholders and consumers. Meat may be contaminated by various chemical toxicants originating from the environment, treatments of agricultural production or food processing. Generally found at trace levels in meat, these toxicants may harm human health during chronic exposure. This paper overviews the key issues to be considered to ensure better control of their occurrence in meat and assessment of the related health risk. We first describe potential contaminants of meat products. Strategies to move towards a more efficient and systematic control of meat chemical safety are then presented in a second part, with a focus on emerging approaches based on toxicogenomics. The third part presents mitigation strategies to limit the impact of process-induced toxicants in meat. Finally, the last part introduces methodological advances to refine chemical risk assessment related to the occurrence of toxicants in meat by quantifying the influence of digestion on the fraction of food contaminants that may be assimilated by the human body.

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1. Introduction

An aging population and constant progress in medical diagnosis contribute to the increasing prevalence of age-related pathologies such as cancer and neurodegenerative diseases. Several recent scientific studies suggest that diet, and especially proteinaceous food, plays a major role in their occurrence by exposing the consumer to various toxic contaminants present in food products. In meat, these chemical risks are mainly related to two different kinds of food toxicants, namely micropollutants and process-induced toxicants. Micropollutants are generated by human activity, or may come from veterinary or plant treatments. They are then trapped in living tissues during the production of animal raw materials and may eventually be transferred to food products. Process-induced toxicants are formed during food processing such as heating or smoking.

Although they are generally found at trace levels in meat, these micropollutants and process-induced toxicants may endanger human health through repeated exposure during a consumer's lifetime. To address this chemical risk, we therefore need to develop novel and efficient approaches to monitor their presence, limit their occurrence in food and refine risk assessment for consumers' health. After a brief overview of the main contaminants monitored in meat products by health authorities, the present paper addresses these three issues.

2. Toxic contaminants in meat products

2.1. Micropollutants

Micropollutants, whether their origin is the environment or veterinary/phytosanitary practice, can contaminate meat products. The risks arising from their presence in the diet are regularly evaluated by large-scale exposure studies conducted by health authorities (ANSES, 2011a).

Micropollutants belong to several different families (Fig. 1).

Mycotoxins are metabolites produced by molds. They are carcinogenic, immunosuppressive, hepatotoxic or neurotoxic in both animals and humans. Human exposure to mycotoxins occurs directly through intake of contaminated agricultural products (cereals, corn, fruits, etc.) or indirectly through the consumption of animal-derived products contaminated by the feed given to the animals or during the maturation process of meat (Capriotti et al., 2012; FDA, 2008; Marroquín-Cardona, Johnson, Phillips, & Hayes, 2014). In 1985, the Food and Agriculture Organization of the United Nations estimated at 25% the proportion of cereal crops contaminated by mycotoxins in the world, thus reducing the global food supply. This contamination also raises the question of livestock contamination *via* diet or during maturation, and subsequent human contamination *via* the consumption of animal-derived foods. The carry-over of several mycotoxins into animal-derived products is closely monitored and seems greatest for blood, liver and kidney, and less problematic for muscle, milk and eggs (Kan & Meijer, 2007). Specific maximum limits for mycotoxins in food are given in several regulations (European Commission, 2006; FDA, 2011).

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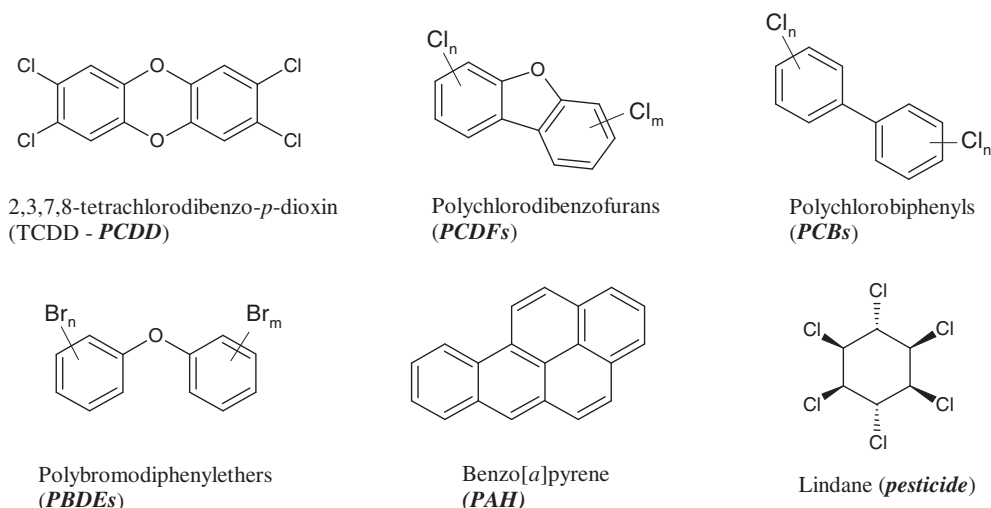


Fig. 1. Examples of environmental micropollutants found in proteinaceous products.

Heavy metals such as cadmium, lead, arsenic, and mercury are environmental contaminants likely to cause health problems when present in the diet. Heavy metals enter the human body mainly through ingestion of water and food and much attention has been recently focused on the concentrations of heavy metals in fish and other foods in order to check for those hazardous to human health (Demirezen & Uruç, 2006). Today their amounts in the diet are tightly controlled by strict standards (European Commission, 2006). Cadmium, lead and mercury are monitored in livestock, but it is relatively easy to reduce their amount in feed by avoiding the use of certain contaminated feedstuffs. Muscle is not likely to show high levels of heavy metals when animals are exposed via diet, whereas liver and kidney often show a clear dose–response related increase in heavy metal concentration after dietary exposure (Kan & Meijer, 2007). However, their levels in animal products are often tested on a survey basis, and contamination of meat products with high levels of heavy metals seldom occurs, if ever (Kan & Meijer, 2007).

Another family of environmental micropollutants that is closely monitored by health authorities are the dioxins and dioxin-like products (AFSSA, 2005; World Health Organization (WHO), 2002), mainly produced by industrial processes although they can have natural origins (e.g. forest fires). These dioxins are undesirable by-products of many manufacturing processes, and are persistent organic pollutants, being very resistant to chemical and biological degradation processes (Larsen, 2006). They fall into three groups: PCDDs (polychlorodibenzo-*para*-dioxins), PCDFs (polychlorodibenzofurans) and PCBs (polychlorobiphenyls). Only ten PCDDs and seven PCDFs are toxic. The most problematic one is TCDD, or Seveso dioxin, which is ten times more toxic than the other dioxins and dioxin-like compounds, and the only one considered as carcinogenic. Certain dioxin-like PCBs have similar toxic properties, 12 of them inducing the same cascades of cellular events as dioxins (Lauby-Secretan et al., 2013). These dioxins and dioxin-like compounds come from chemical reactions produced during combustion in the presence of oxygen and organochloride products. Although human exposure to dioxins from the environment has declined significantly, the monitoring of these toxicants required by health authorities is justified because food is currently the primary source of human exposure to dioxins (Charnley & Doull, 2005). Biomagnification occurs through the food chain, and high tissue concentrations can often occur in top predator species (Van den Berg et al., 2006), in particular in fatty tissues of animals (Larsen, 2006).

Brominated flame retardants (BFRs) are mixtures of chemical compounds added to a broad variety of products to make them less flammable. They are widely used for plastics, textiles, electric and electronic

equipment and also for materials used to build livestock buildings. They belong to five classes: polybromodiphenylethers (PBDEs), hexabromocyclododecane (HBCD), tetrabromobisphenol A (TBBPA) and other phenols, polybromobiphenyls (PBBs) and the other brominated flame retardants. Their use in European Union is forbidden or restricted, but concerns remain worldwide about their risks for public health because of their persistence in the environment (AFSSA, 2005; ANSES, 2011a; US EPA, 2010). Materials treated with BFRs and used for livestock buildings will release BFRs that contaminate animals' tissues and can eventually be transferred to meat products. As an example, HBCD, a suspected potent endocrine disruptor, was withdrawn from the European market in 2011 and is now closely monitored by ANSES (2012) in animal-derived food products, particularly meat products.

Polycyclic aromatic hydrocarbons (PAHs) can be either of environmental origin or generated during the processing of foods (this origin will be discussed in Section 2.2.) and are also closely monitored by sanitary agencies (Schroeder, 2010). Concerning the environmental origin, the primary natural sources of airborne PAHs are forest fires and volcanoes. Their anthropogenic sources include residential burning of wood, oil, gas and charcoal, together with industrial power generation, incineration, and the production of several metals. These sources account for some 80% of total annual PAH emissions, to which must be added emission from the vehicle exhausts of gasoline and diesel-powered engines (EFSA, 2008). The main source of human exposure, for non-smoking individuals, is food consumption, as these PAHs build up in plants, thereby contaminating crops and feeds for livestock. Among PAHs, benzo[*a*]pyrene (BaP) is the most toxic due to its ability to form DNA adducts that can induce mutagenic and carcinogenic effects (IARC, 2010a). It was therefore long used as the sole marker of PAH presence (Srogi, 2007). The European Union established maximum levels of BaP for different food categories (European Commission, 2011) with a maximum level of BaP set at 2 µg/kg for smoked meat. However, it was recently shown that this was insufficient, and that it is necessary also to consider the sum of benzo[*a*]pyrene, benz[*a*]anthracene, benzo[*b*]fluoranthene and chrysene (European Commission, 2011).

Antibiotics given to animals are strictly regulated (FDA, 2005), and their absence in meat products is monitored at different steps in the production chain. It is noteworthy that in the case of feed for livestock, antibiotics are considered as additives, although stricter rules could be applied to them, such as those for antibiotics in humans. However, the only antibiotics allowed as feed additives are coccidiostats and histomonostats (European Commission, 2003) because other antibiotics, especially if also used in human medicine, could induce antimicrobial resistance in humans (Chattopadhyay, 2014). Even so, the use of

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