NUMMER 46
DECEMBER 2025

TCDD
TOXICOLOGIE

SPECIAL THEME

Food Safety

- HO HO HO! SUGAR OR SWEETENER? SANTA OR KRAMPUS?
- PERSONALIZED PREVENTION MEETS FOOD SAFETY: INSIGHTS FROM NUTRIGENOMICS
- LET'S GET HEAVY METAL POISONING FOR CHRISTMAS
- ALL I WANT FOR CHRISTMAS IS... ULTRA-PROCESSED FOOD?
- METHANOL POISONING FROM ALCOHOLIC DRINKS: A GLOBAL CRISIS
- SANTA'S NAUGHTY-OR-NICE FOOD CHAT ACROSS THE ATLANTIC

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Sluitingsdata kopij 2026
February 13, 2026,
April 10, 2026,
September 11, 2026,
November 13, 2026.

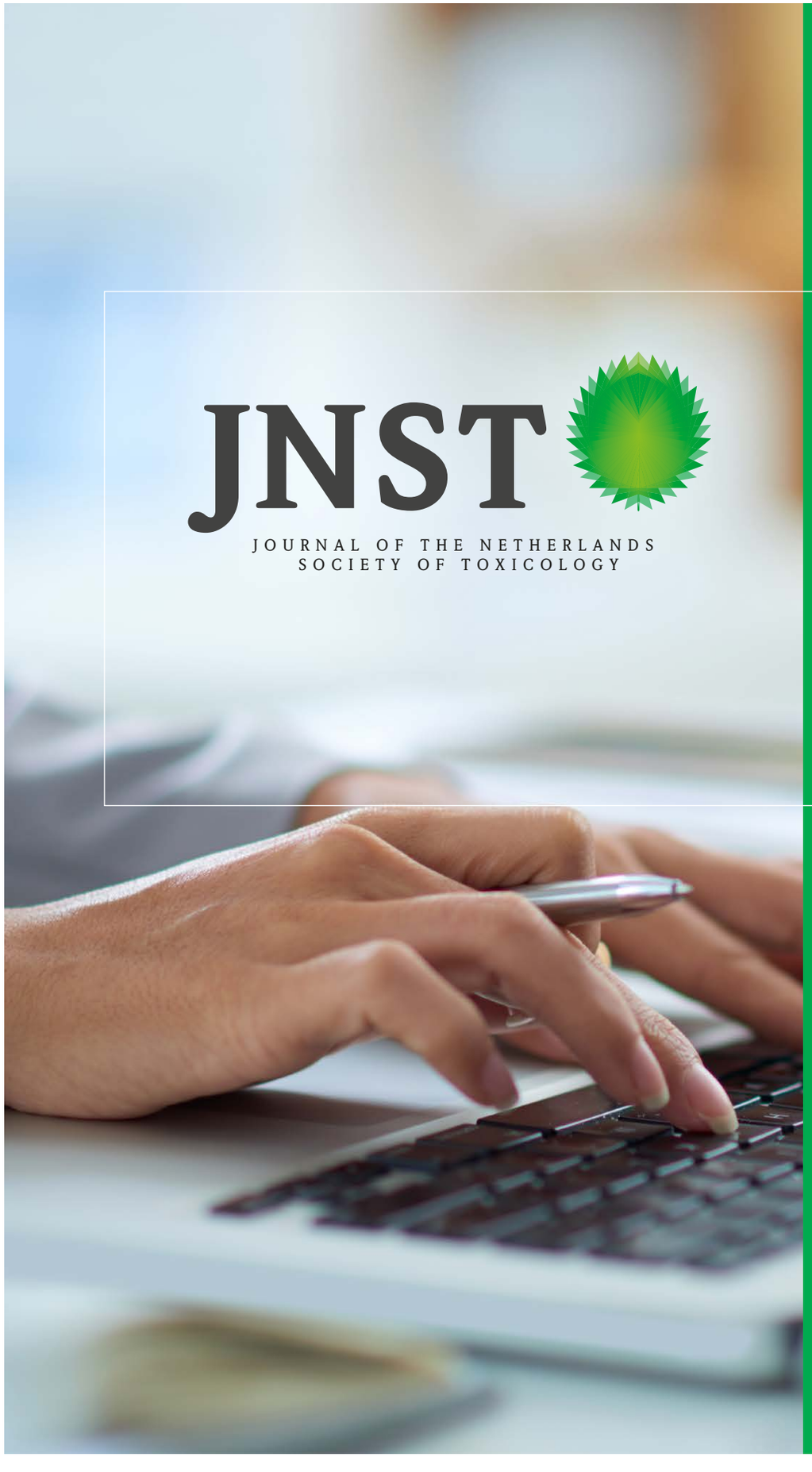
Kopijbus
redactie@toxicologie.nl

Website NVT
http://www.toxicologie.nl

Vormgeving
Marleen Mulder
Green Bean Design
www.greenbeandesign.nl

TCDD
TOXICOLOGIE

Inhoud



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TERUG NAAR INHOUD

Editorial

With the year coming to an end, this is the last TCDD of 2025. But for me, it is the first! My name is Puck and I recently joined the TCDD team. I am a second-year PhD student, working on regulatory acceptability of New Approach Methodologies, specifically organoids. My project is a collaboration between the Dutch Medicines Evaluation Board, Utrecht University and the Princess Máxima Center. I hope my research will contribute to bridging the gap between the development of these models and application of these models for pharmaceutical regulatory testing.

This issue of TCDD, however, is about something completely different. With the holidays coming up, this issue is all about “Food Safety”. We will delve into sugar and artificial sweeteners, ultra-processed foods, nutrigenomics, a comparison between EU and US Christmas treats, and much more. On top of that, this issue features the farewell lecture of prof. Frederik-Jan van Schooten upon his retirement from Maastricht University, a PhD student presenting his work in the Toxafette, and several travel reports.

We hope that we do not scare you too much for your Christmas dinner. After all, the holidays are also a time of Christmas cookies, roast turkey, champagne and oliebollen. On behalf of the editorial team, I wish you very happy holidays and a delicious Christmas dinner!

Sincerely,

Puck Roos

Photo by Jess Bailey on Unsplash

News from the Board

The preparations for the 47th NVT Annual Meeting are underway. Please save **23 - 24 June 2026** for our next Annual Meeting at De Eenhoorn in Amersfoort. To reduce pressure on the programme, next year's Business Meeting will be held online prior to the Annual Meeting.

The working group advancing the NVT's communication strategy and developing **a new website** is making good progress. We are now entering the design and technical phase with a web developer and are looking for 1–2 junior toxicologists who would like to help shape the design of the new site. As the NVT depends heavily on volunteers, we warmly invite members to contribute - especially since similar calls in previous TCDD issues have unfortunately received almost no responses.

Vacancies – urgent call for a Treasurer
Several Board and section positions remain open. Despite highlighting these vacancies in the previous TCDD, we have not received *any* applications. The Board urgently seeks a **Treasurer** who can work alongside the current treasurer for one year. Without this role filled, the continuity of our activities will be under pressure. We strongly encourage interested members to contact us.

Internationally, we are excited that the **11th German Pharm-Tox Summit (GPTS 2026)** will be organised jointly with the NVT and will take place on **17–20 March 2026** in Düsseldorf.

We are also proud to share that **Flemming Cassee (RIVM)** was elected **Secretary-General of the IUTOX Executive Committee** during IUTOX's 17th International Congress of Toxicology in Beijing. Professor Cassee's extensive experience, significant scientific contributions, and proven leadership make him exceptionally well equipped for this position. Congratulations, Flemming!

Finally, we warmly encourage submissions to the *Journal of the Netherlands Society of Toxicology (JNST)*.

The Board wishes all members a wonderful holiday season and an inspiring start to 2026.

Prof. Dr. F.J. van Schooten

SECTIONS
PHARMACEUTICAL
TOXICOLOGY AND
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Extractables and Leachables: a concern for our health?

On April 15th 2025, the sections Pharmaceutical Toxicology and Risk Assessment jointly organised a scientific spring meeting with the theme: 'Extractables and Leachables: a concern for our health?'. The PDFs of the presentations are available at the Pharmaceutical toxicology section page of https://toxicologie.nl/

Ho Ho Ho!
Sugar or Sweetener? Santa or Krampus?

By *Marcha Verheijen*

It’s that time of year again—the tree is sparkling, the lights are twinkling, and every flat surface seems to be covered with cookies, chocolate truffles, and mysterious tins that *probably* contain biscuits (but may also contain sewing supplies). As you glide through the room—perhaps in a sugar-induced haze—you might wonder: *Is sweetness this season a gift from Santa… or a trick from Krampus?* Let’s unwrap the science behind sugar and sweeteners.

A Spoonful of Sugar (or Three… or Twelve)

When we talk about *sugar*, we often picture the classic white stuff we add into batter or stir into holiday coffee. But sugar is actually an umbrella term for many simple carbohydrates. Sucrose—table sugar—is just one of several naturally occurring sugars, along with fructose, glucose, galactose, maltose, and lactose [1].

Sugar itself isn’t inherently naughty. Our bodies run on carbohydrates, and removing natural sources—like fruit, grains, and dairy—would make even Santa grumpy. But added sugar? That’s another story. Added sugars sneak into processed foods, contributing calories without nutrients and increasing risks such as high blood sugar, insulin resistance, metabolic syndrome, dental cavities, obesity, and type 2 diabetes [1]. The World Health Organization (WHO) recommends that no more than 10% of daily energy intake comes from free sugars, and preferably less than 5% [2]. Which, depending on how many gingerbread men you already ate today, may be cause for some light seasonal self-reflection.

A Sweet Solution? The Rise (and Controversy) of Artificial Sweeteners

To reduce sugar intake without sacrificing taste, artificial sweeteners (AS) entered the scene—hailed initially as the holly-jolly heroes of dietary management. These compounds provide intense sweetness without the caloric load of sugar [2]. But according to WHO’s 2023 guidance, using non-sugar sweeteners (NSS) for weight control may not offer long-term benefits—and might even come with risks [3,4]. The systematic review informing the guideline found no sustained reduction in body fat in adults or children using NSS, and suggested potential undesirable long-term effects such as increased risk of type 2 diabetes, cardiovascular disease, and even overall mortality [4]. IMPORTANT: The WHO emphasizes that

these recommendations are **not** based on toxicological evaluations and do not replace safety guidelines for specific sweeteners [5]. So… not quite Santa. But maybe not Krampus either.

Meet the Sweeteners: The Holiday Party

Before we unwrap the science of sweetness, let's take a moment to look at the main "families" gathered around the holiday table. From artificial to natural, each group brings its own flavor to the festivities.

1. Natural Sweeteners

Honey, maple syrup, coconut sugar, palm sugar… These have been around far longer than Santa's workshop. They contain beneficial compounds and typically have lower glycemic indices than sucrose, but they are still sugars—and too much can send your metabolism sliding like a runaway sleigh [6].

2. Artificial Sweeteners (Non-Nutritive Sweeteners, NNS)

These include acesulfame K, aspartame, sucralose, saccharin, neotame, and advantame. They have a glycemic index of zero and don't cause tooth decay [6]. The FDA regulates them as food additives and deems them safe when consumed below the acceptable daily intake [1]. Yet, the science is still evolving. Some animal studies raised concerns, but human studies generally support their safety under regulated conditions—though ongoing debates keep toxicologists on their toes like overcaffeinated elves.

3. Sugar Alcohols (Polyols)

Found in gum, candies, and many "sugar-free" products, sugar alcohols such as erythritol, sorbitol, and xylitol provide sweetness and texture. They have lower glycemic impact, don't feed oral bacteria, and therefore don't contribute to tooth decay [6]. But they have a mischievous side—causing bloating, gas, or diarrhea in some people [1]. Think of them as the festive elves of the holiday treat tray—usually delightful, but occasionally stirring up a little too much cheer in your gut.

4. Novel Sweeteners

These plant-derived sweeteners—like monk fruit, stevia, tagatose, and allulose—offer sweetness with few calories and minimal glycemic impact [1]. The FDA regards them as generally safe, and they tend to be less processed than traditional artificial sweeteners [6]. Think of them as the new kids at the Christmas party—still earning their stockings on the mantle.

Sweeteners and Weight: Not All That Glitters Is Gold (or Sugar Crystals)

Even though artificial sweeteners were designed to reduce calorie intake, studies suggest that they may not guarantee jollier waistlines. Some AS appear to promote weight gain and fat accumulation independently of caloric intake, perhaps through changes in energy efficiency [2]. Saccharin and aspartame, for example, have been associated in some studies with increased adiposity despite their zero-calorie status. Metabolically, AS may impair glucose regulation. High consumers have shown a significantly greater risk of developing type 2 diabetes (HR 1.69) compared to non-consumers. AS can also influence hunger hormones like ghrelin and leptin, though evidence is mixed. But the take-home message? Swapping sugar for sweeteners isn't necessarily a sleigh ride to effortless weight loss.

Cardiovascular & Cancer Considerations: What Does the Science Say?

Research indicates a potentially harmful association between nonnutritive sweeteners and cardiovascular disease risk [2], though causality remains uncertain. The relationship between AS and cancer is even more debated. Some studies have found associations—for instance, a 30% increased risk of non-Hodgkin lymphoma among moderate aspartame consumers. Yet other human studies found no overall increase in cancer risk. Overall, the evidence is inconsistent, and no definitive link has been established. In short: The jury is still out, enjoying its holiday break.

Neurological Notes: Sugar, Sweeteners, and the Brain

Excessive sugar consumption has been implicated in impaired cognitive function, worse memory, and an increased risk of dementia. Prenatal exposure to high sugar intake has been linked to attention and impulsivity issues in offspring, resembling ADHD-like symptoms, and poorer cognitive development in children [2].

But sweeteners have their own spotlight moment—especially erythritol. A 2024 study reported that erythritol may damage blood-brain barrier (BBB) cells, inducing oxidative stress, reducing nitric oxide, increasing endothelin-1, and impairing the natural "clot-busting" system (tPA) [7]. These changes could theoretically increase the risk of ischemic stroke. Regulatory agencies still deem erythritol safe, but this research has raised new questions. With the glow of the holidays around us, it's a perfect moment to savor sweetness with care, keeping our brains as bright as the lights on the tree.

So… Santa or Krampus?

Well… neither. Both sugar and sweeteners sit somewhere in the grey area—right between a candy cane and a lump of coal. Sugar isn't toxic, and sweeteners aren't magical solutions. Lifestyle factors—dietary patterns, physical activity, and overall metabolic health—shape outcomes far more than any single ingredient [6]. Removing natural sugars entirely is unhealthy [1]. Dietitians recommend reducing both added sugars and artificial sweeteners, but not eliminating natural carbohydrates [1]. As WHO's Francesco Branca puts it: "People should reduce the sweetness of the diet altogether… to improve their health."

[4] Easier said than done when the office table is groaning under fruitcake. So, as with everything in toxicology, “the dose makes the poison”—but emerging findings remind us to stay vigilant.

So here’s my holiday wisdom: **A Merry, Moderate Christmas**

You don’t need to banish sugar like Krampus, nor embrace artificial sweeteners as your personal Christmas miracle. The key is keeping your balance, much like a gift tower that *just* avoids tipping. Personally, I don’t want to live in a world without Christmas cookies. But I *can* have one instead of three—and perhaps drink tea with honey instead of soda. Ultimately, savor the season without overstuffing it. With some sweetness, a little restraint, and plenty of joy, you can enjoy every bite and still greet the New Year feeling bright.

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Personalized prevention meets food safety: **Insights from nutrigenomics**

Contribution by dr. Simone van Breda and dr. Susan Steinbusch-Coort, part of the Nutrigenomics theme of the department of Translational Genomics (formerly Toxicogenomics; and merged with the BigCat department in 2024) at Maastricht University.

By *dr. Simone van Breda*

By *dr. Susan Steinbusch-Coort*

Food safety is traditionally viewed through the lens of population-wide regulations, contamination monitoring and risk assessment. However, emerging research shows that individuals can differ substantially in how their bodies respond to dietary exposures and food-borne toxicants. At the intersection of nutrition, toxicology, and omics technologies, personalized prevention is rapidly becoming a promising approach to protect health more effectively and sustainably. Within the Nutrigenomics theme of the Translational Genomics department at Maastricht University, the work exemplifies how molecular insights can help us move towards individually optimized dietary strategies to reduce disease risk and to improve health outcome.

One of the challenges in food safety is explaining why two people exposed to the same dietary compound can have completely different biological responses. These differences may originate from genetic variation, but also from epigenetic programming, metabolic state, lifestyle, or previous environmental exposures. Our research aims to unravel these layers of biological diversity. By understanding who is at risk, why they are at risk, and how their biology responds, we are able to design prevention strategies.

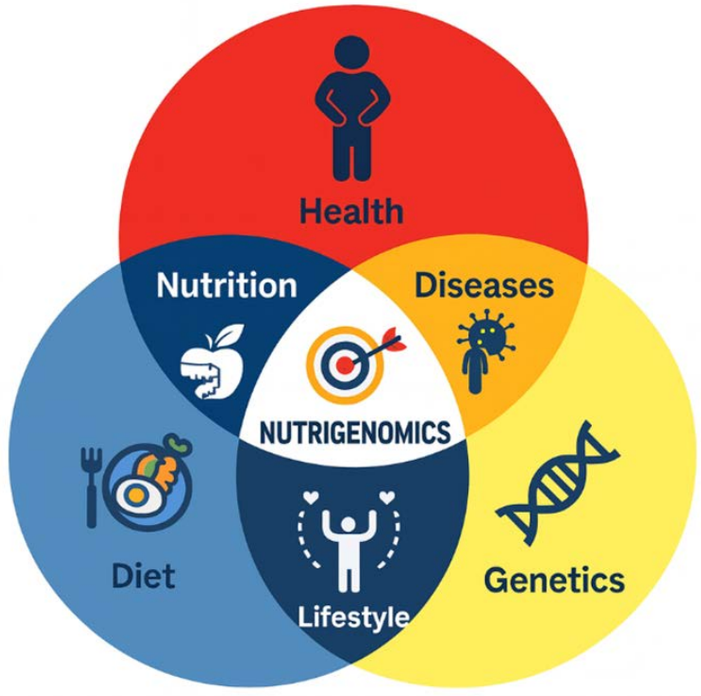
An example of a study that combines these different viewpoints is the blueberry-apple juice human dietary intervention study which is a risk–benefit assessment of the flavonoid quercetin within a realistic fruit matrix. After four weeks of daily intake, we observed a clear beneficial signal: circulating antioxidant-related measures increased and participants showed protection against oxidative DNA damage induced *ex vivo*. At the same time, we also saw an increase in *ex vivo*-induced BPDE–DNA adduct formation, illustrating that an intervention popularly framed as “protective” can also shift pathways relevant to genotoxic risk. Importantly, these outcomes were not uniform across individuals. By integrating polymorphisms in genes involved in quercetin and xenobiotic metabolism, oxidative stress handling, and DNA repair with phenotypic markers and whole-genome expression profiles, we could identify subgroups that benefited more, those that benefitted less, and those in whom the balance could tilt in an unfavorable direction. The transcriptome data helped interpret this complexity mechanistically, thereby translating molecular responses into more person-centered risk/benefit thinking (1-3).

A second line of work, the PHYTOME project, addresses food safety in a more direct “processing and formulation” sense: how to reduce exposure to nitrite-related, potentially harmful *N*-nitroso compounds from processed red meat while maintaining product quality and safety functions that nitrite normally provides. By partly reducing nitrite and adding phytochemical extracts intended to maintain beneficial product properties while lowering formation of nitrite-derived pre-carcinogenic compounds, we tested a mitigation strategy directly at the food level. Biomarkers such as apparent total *N*-nitroso compounds excretion (ATNC: biomarker of overall *N*-nitroso compound exposure), colonic DNA adduct-related endpoints, DNA damage measures, and colon gene expression showed that genotype influenced both the genotoxic response to processed red meat and the degree of protection achieved with phytochemical enrichment. Using multi-gene approaches (*e.g.*, allele importance and gene scores) further supports

translation toward identifying susceptible subgroups and responders to mitigation strategies in food products (4,5).

In addition to studying naturally occurring dietary bioactives and mitigation strategies in food products, our food-safety work also includes food additives, ingredients used for technological purposes in foods, where careful evaluation of potential unintended health effects remains essential. Our recently finalized human dietary intervention study on titanium dioxide (E171), which is a food additive used as a whitening and opacifying agent in various food products, follows the same approach as our other work: realistic dietary exposure, sensitive molecular readouts, and a mechanistic framework to interpret heterogeneous responses. Rather than relying only on traditional clinical immune marker panels, we applied transcriptomics and oxidative-stress endpoints to detect early biological perturbations that may precede disease, an important step for next-generation risk assessment. We demonstrated that while these panels are largely unchanged, molecular readouts show that biology is shifting. This approach therefore helps to move food-safety evaluation toward identifying early mechanistic signals and recognizing that some individuals may respond more strongly than others (6).

Finally, the MiBlend project adds another perspective by showing that food safety and health benefits are also shaped by the way foods are produced and processed, before an intervention even starts. Antioxidant-rich foods are often delivered as complex mixtures (*e.g.*, smoothies or blended products), and ensuring microbiological safety typically requires processing steps such as pasteurization or high hydrostatic pressure. While it is well known that processing and storage can influence phytochemical levels, we found that in a complex fruit-and-vegetable mixture, these effects are highly compound-specific and not always intuitive. Processing can reduce some bioactives, preserve others, and sometimes even increase measurable concentrations, which can have an effect on the bioavailability. This first layer of complexity is crucial when linking foods to biological effects, because the exposure is partly defined by processing choices, storage duration, and even cultivar variation. Building on that optimized and well-characterized exposure, the MiBlend randomized cross-over trial tested multiple whole fruit and vegetable blends (450 g/day) representing distinct phytochemical profiles (*e.g.*, flavonoids, anthocyanins, carotenoids, glucosinolates, and combinations) and found improvements in plasma antioxidant capacity, reduced susceptibility to induced DNA damage, and benefits in microvascular markers. Different blends produced different “benefit signatures,” and transcriptomics indicated plausible mechanisms (DNA repair, signal transduction, transcriptional regulation). A genetic follow-up showed that inter-individual variability is not noise but informative biology: for example, XRCC1 and GSTP1 variants helped explain why some participants gained stronger DNA protection or vascular improvements from specific phytochemical patterns. From a food safety perspective, MiBlend shows how nutrigenomics can move beyond “eat more fruits and vegetables” to which composition works best for which subgroup, using both functional endpoints and mechanistic readouts (7-9).



Afb. 1. Figure illustrating the multidisciplinary nature of nutrigenomics.

Adapted from https://welocitygenetics.com/website/advantage-nutrigenomics

Together, these four examples illustrate how our nutrigenomics work contributes to modern food safety by (i) quantifying *both* benefit and risk signals in realistic dietary settings, (ii) explaining *why* they occur through mechanistic pathway readouts, and (ii) identifying *who* is most likely to benefit or be at risk, while recognizing that real-world factors like food processing can be decisive in shaping exposure and effect.

Going forward, our work within the Nutrigenomics theme will focus on strengthening the mechanistic, translational link between diet, molecular pathways, and inter-individual variability in health outcomes. We plan to do this by combining well-controlled nutritional interventions with multi-omics profiling and bioinformatics to identify response patterns within subgroups driven by genetic and epigenetic variation, metabolic and microbiome context, and relevant environmental exposures. Translation will be built in from the start by relevant biomarkers and strategies that can be validated across different subpopulations and converted into evidence-based strategies for targeted prevention and precision nutrition in practice. Currently, we are part of the NUTRIOME (10) project, a European MSCA doctoral network

that investigates why individuals respond differently to the same foods and meals at metabolic and immune levels. The project focuses on the postprandial phase, which is the state in which people spend most of their day, where circulating metabolites and immune responses fluctuate and strongly influence cardiometabolic health. By using multi-omics approaches (metabolomics, transcriptomics, epigenetics, metagenomics) and advanced data science, the network aims (i) to identify biological determinants of inter-individual variability in food responses, (ii) to uncover mechanisms linking dietary components to immune and metabolic regulation and (iii) to develop data-driven models to predict which meals are most beneficial for specific individuals or population subgroups. The ultimate goal is to enable tailored, stratified dietary advice for prevention of diet-related non-communicable disease.

Taken together, personalized prevention is an emerging field driven by rapidly accelerating advances in omics technologies, data science and *in vitro* modelling. The combined contributions of our nutrigenomics group help build the scientific foundation necessary to design prevention strategies that move beyond population-based approaches toward person-centered solutions.

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TERUG NAAR INHOUD

Let's get heavy metal poisoning for Christmas

End of the year celebrations have started, a caloric supernova, prime time for accruing those "holiday kilos"! With all what we eat during this period, we contribute to stealthy heavy metal bioaccumulation, starring the insidious "time bomb" cadmium (Cd).

By *Héloïse Proquin*

Cadmium's Origin Story

Discovered in 1817 by Friedrich Stromeyer, who excised Cd from a zinc ore sporting—naming it after ancient Greek "cadmeia" for zinc ore—Cd debuted as a jaunty yellow pigment (CdS) in 19th-century paints[1]. Cd exists in the earth's crust at about 0.1 part per million usually being found as an impurity in Zn or lead (Pb) deposits and therefore being produced primarily as a byproduct of Zn or Pb smelting. NiCd batteries once hogged global demand, anti-rust shields, PVC stabilizers, solar semiconductors, radiation blockers, pigments, polyvinyl chloride (PVC) stabilizers, radiation shielding, and semiconductors for solar cells joined also the party[1].

Silent Soil Saboteur: The Cadmium Crisis Threatening Global Croplands

Naturally frolicking in soils and phosphate fertilizers, Cd piles up like uninvited holiday guests, tainting cereal, bread, pasta, and potatoes[3]. Foodstuffs are the main source of cadmium exposure for the non-smoking general population. IARC classified cadmium and cadmium compounds as carcinogenic to humans (Group 1)[2]. EFSA estimated the European adult mean intake at 2.04 µg/kg body weight/ week (toddlers: 4.85, elders: 1.56), flirting with or breaching the 2.5 µg/kg TWI[4]. These levels are mainly due the consumption of veggies (Cd= 0.05 mg/kg), tubers like potatoes (Cd=0.10 mg/kg), cereals (Cd=up to 0.5 mg/kg)[5]. EFSA also showed that vegetarians, kids, smokers, and contaminated-zone dwellers double-dip exposure.

Toxic Tricks and Body Betrayal

Cd's credo in life is to be mutagen, repro-toxin, endocrine saboteur, kidney assassin, and osteoporosis enabler; eviction from the body crawls at a snail's pace, amassing in kidneys, liver, muscles for long-term sabotage[2,4].

Afb. 1. A crystal cadmium bar. Purity 99.999 %. Made by the flux process. As well as a 1 cm3 cadmium cube for comparison. By: Alchemist-hp (talk) (www.pse-mendelejew.de)

Chocolate's Cadmium Caper

In 2021, the French consumer group UFC-Que Choisir unwrapped an unsettling truth about dark chocolate: beneath its glossy finish lay a cadmium's holiday hijack[6]. Their analysis revealed cadmium concentrations ranging from 0.022 to 0.458 mg/kg—levels still within European safety limits, yet troubling for a treat often marketed as wholesome. Ironically, the worst performers tended to be organic or premium labels, many sourced from Latin America[7].

The culprit lies not in careless processing but in the land itself. Cocoa trees grown in the volcanic, metal-rich soils of Peru, Ecuador, and Colombia naturally draw cadmium from the ground, accumulating it in their beans. This geological inheritance turns fair-trade and organic chocolate—hailed for sustainability—into an inadvertent carrier of slow-release toxicity. By contrast, beans from West Africa or Asia, rooted in different geology, typically harbour far less cadmium.

To conclude, consuming about 55 grams of the most contaminated dark chocolate from the reported range could reach the maximum tolerable daily cadmium intake for an adult of 70kg. So, while each square of dark chocolate

may bring antioxidants, solace, or festive cheer, it can also deliver trace reminders of Earth's elemental mischief—a bittersweet chemistry lesson wrapped in foil.

Enjoy the holidays!

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All I Want for Christmas is... **Ultra-Processed Food?**

When walking through any supermarket in December, you will find a wealth of seductive holiday-themed products: reindeer-shaped cookies, Christmas tree-shaped pancakes, chocolates with golden glitters. But as nice as they may seem, they also have a naughty side. These Santa-shaped bao buns, snowman pastries or star-shaped gourmet-minis may actually be: ultra-processed foods…

By *Puck Roos*

What are ultra-processed foods?
In 2009, researchers from the University of Sao Paolo, Brazil, came up with a framework to group foods according to the extent of their processing: the Nova classification system. This system consists of four group. Group 1 contains unprocessed or minimally processed foods. These are fresh whole foods, such as eggs, fruits or meat. Foods can also be frozen, dried, pasteurised, or otherwise processed, as long as their natural structure is still preserved. No sugar, salt, oil or other ingredients are added during processing. Group 2 consists of processed culinary ingredients, for example olive oil, butter, honey. These are foods that have been extracted from the Group 1 foods, and are generally used to prepare dishes with the foods from Group 1. Group 3 consists of processed foods. These include foods from Group 1 to which sugar, salt, oil or other product have been added, such as tinned fish, cheese or canned fruits. Lastly, group 4 consists of ultra-processed foods (UPFs). UPFs have undergone sequential processing, such as fractioning crops into sugars, fats, or proteins, chemical modification, and industrial techniques, such as pre-frying. Additives are used to make the products taste, smell and look good. This yields products such as frozen pizzas, chicken nuggets or candy bars: attractive and convenient to eat. [1]

What are risks associated to UPFs?
Over the past decades, the share of UPFs has increased in diets worldwide, ranging from 9% of total energy intake in Iran to 60% in the USA.
However, despite their convenience, attractive packaging and seductive looks, UPFs come with a downside. Studies have linked UPF diets to numerous adverse health outcomes, including higher risks for all-cause mortality, cardiovascular disease and cardiovascular disease-related mortality, colorectal cancer, obesity, type 2 diabetes, hypertension, chronic kidney disease, depression, anxiety, wheezing, and adverse sleep outcomes [1-3].

Several mechanisms may underly these adverse health outcomes. A first mechanism could be nutrient imbalance. UPF-rich diets contain more nutrients that are linked to chronic diseases (such as sugar and fat), and fewer nutrients that are inversely linked to chronic disease (such as fibre, protein and vitamins) [1]. A second mechanism could be increased energy intake. Studies have compared high-UPF diets to no-UPF diets, in which both diets had similar nutrient profiles. Participants on the high-UPF diets consumed more calories. Furthermore, participants on the high-UPF diets used fewer chews per calorie, which may be due to the hyper-palatability of UPFs [1]. A third mechanism could be an increased intake of xenobiotics. Potentially toxic chemicals can be generated during manufacturing of UPFs or leach from the packaging. To illustrate, in an American study, people with higher UPF consumption had higher PFAS concentrations in their urine. Furthermore, UPFs often contain additives, such as emulsifiers, flavour enhancers, and sweeteners, which may be linked to chronic diseases [1, 4]. A last, mechanism could be disruption of the gut microbiome. The gut microbiome is important for metabolising nutrients, modulating the immune response, maintaining gut barrier integrity, and more. UPFs in general, or additives often found in UPFs, can shift the gut microbiome towards a more proinflammatory composition. This can lead to low-grade inflammation in the gut, which can increase intestinal permeability. Eventually, pro-inflammatory cytokines or pathogenic bacteria can be released into the bloodstream [4, 5]. Through these various mechanisms, UPF consumption can eventually result in adverse health outcomes.

So what can I do about this?

Now you may be wondering, what can I still eat? Well, not all UPFs are bad. For example, plant-based UPFs seem to be healthier than their milk- or meat-based counterparts. For example, soy milk consumption is associated with lower blood pressure, lower risk of gastric and breast cancer, and lower risk of type 2 diabetes than cow's milk consumption. Likewise, consumption of plant-based meat analogues is associated with lower body weight and cholesterol levels than real meat consumption. Lastly, margarine consumption is associated with improved lipid profiles and a lower risk of cardiovascular disease and mortality than butter consumption [6].

But if you are not a fan of plant-based products, or you want to cut down on UPF intake entirely, there are plenty of other options. The most straightforward action would be to buy fresh produce and make everything from scratch. YouTube and Instagram are full of influencers who are eager to show you their UPF swaps, some more time-consuming than others. But not all of us may have the time or skills to make homemade ice cream or energy bars. In that case, the Open Food Facts website and app may be helpful. You can search for a product or scan a barcode, and they will show you in which NOVA category the product falls, alongside other information on the ingredients. And finally, if cutting down on UPFs for Christmas is too much to ask, you can always add it to your New Year's resolutions.

By Open Food Facts. Design: Quentin Lagrange. - https://github.com/openfoodfacts/openfoodfacts-server/tree/main/html/images/logos, AGPL, https://commons.wikimedia.org/w/index.php?curid=127029017

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Methanol Poisoning from Alcoholic Drinks: **A Global Crisis**

Methanol poisoning associated with alcohol consumption remains one of the most severe yet preventable toxicological emergencies worldwide. Analysis of the Médecins Sans Frontières (MSF) methanol poisoning database covering the period 1998 to 2025 documents 1,039 incidents across more than 80 countries, affecting over 36,000 people and causing more than 14,427 deaths[1]. This corresponds to an overall case fatality rate of 40%. These figures almost certainly underestimate the true burden, as the dataset relies largely on media reports, scientific publications, and field communications, and many cases, particularly in low-resource settings, go undiagnosed or unreported.

By *Barae Jomaa*

Importantly, methanol poisoning in this dataset does not arise from a single mechanism. The recorded cases reflect a spectrum of exposure scenarios, including deliberate adulteration of alcoholic beverages with methanol, poorly produced illicit or home-distilled alcohol containing excessive methanol, and consumption of surrogate or non-beverage alcohols that contain methanol but are not intended for drinking.

Regardless of the way methanol ended contaminating the alcoholic drinks, exposure to high enough concentrations will cause nausea, vomiting and headaches due to metabolic acidosis, blindness due to optic nerve toxicity, and eventually multi-organ failure, especially respiratory or cardiovascular collapse, leading to death.

Methanol's toxicity is mediated by its liver metabolites. Alcohol dehydrogenase (ADH) converts methanol into formaldehyde, which is then rapidly oxidised to formic acid by formaldehyde dehydrogenase (ADH5/FDH), a glutathione-dependent enzyme. Formic acid disrupts mitochondrial respiration, causing severe metabolic acidosis and damaging the optic nerve, potentially leading to blindness. Antidotes include ethanol and fomepizole, which act by competitively inhibiting ADH, thereby blocking the formation of toxic metabolites and allowing the body to safely eliminate methanol[2] (Figure 1). In severe cases, hemodialysis is required to remove both methanol and accumulated formic acid.

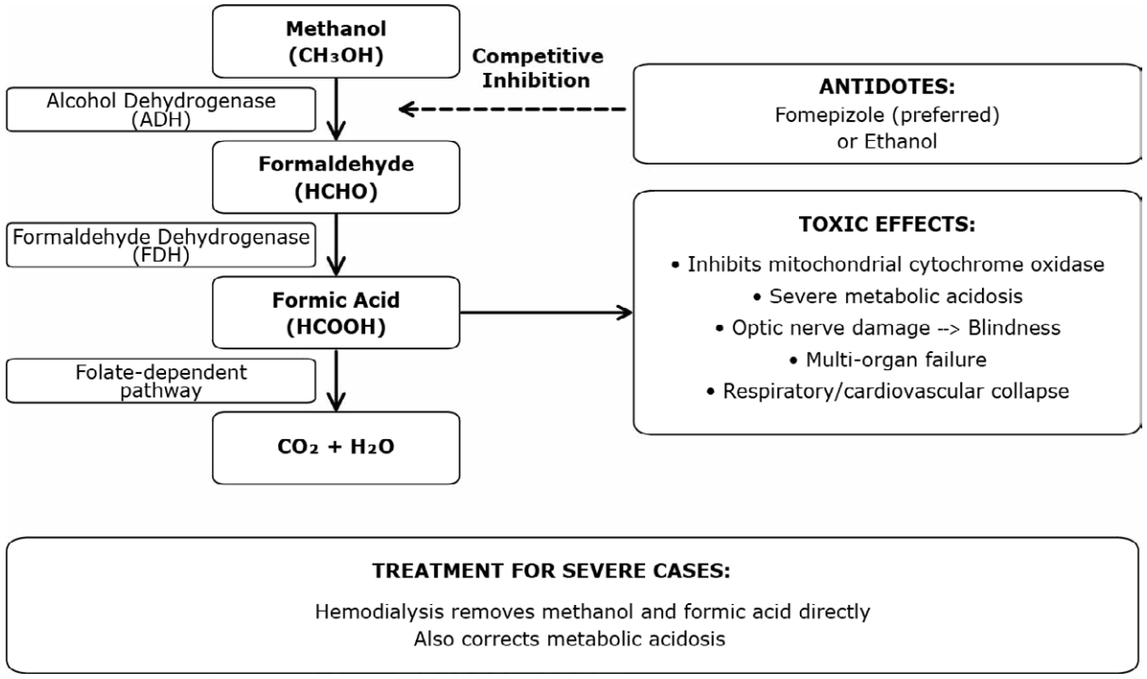


Figure 1. Methanol metabolism, toxicity mechanisms, and treatment strategies.

Figure 2. Press conference on the methanol crisis, São Paulo, Brazil, 6 October 2025. Officials from the Government of the State of São Paulo address the media at the Palácio dos Bandeirantes press room. Photo: João Valério / Governo do Estado de São Paulo (CC BY 4.0).

According to MSF, 30ml is the minimum fatal dose for an adult, and 10ml (2 teaspoons) is a dose that can cause blindness. It's like someone had turned off her bedside light, is how Canadian Ashley King described her experience of going blind after drinking contaminated alcoholic drinks on a 2011 vacation in Bali[3]. She is now dedicated to educating travelers on the dangers of methanol poisonings.

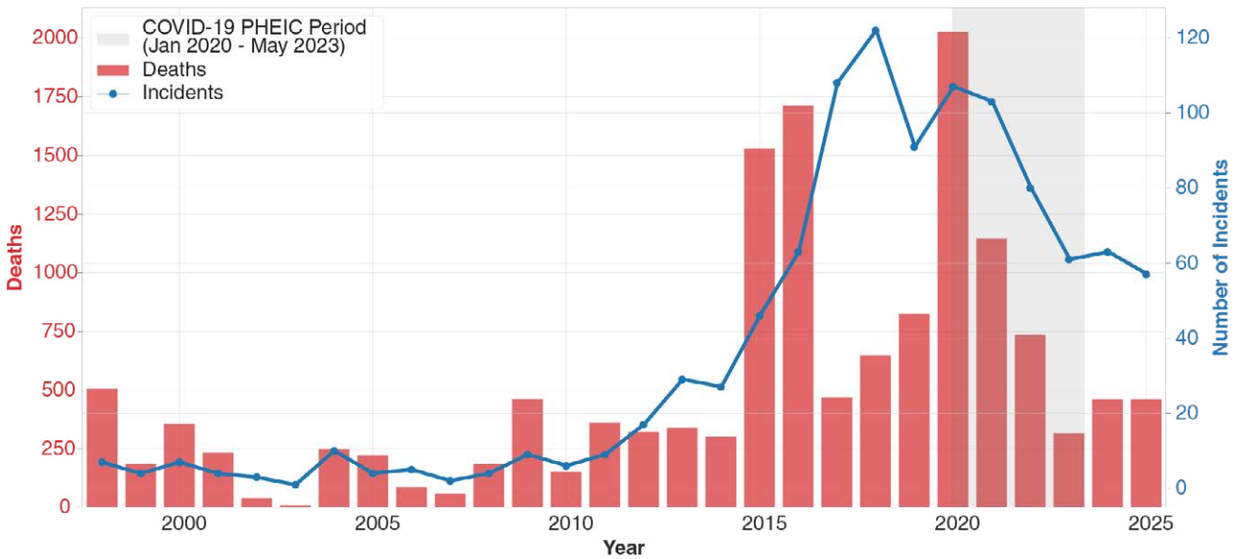
One of the methanol poisoning incidents that was widely circulated in the news in 2025 was the outbreak in Brazil, where consumption of illicit or adulterated alcoholic beverages led to multiple cases of severe poisoning, prompting emergency hospitalizations, public warnings, and a coordinated response by state authorities (Figure 2).

What emerges most clearly from the 27-year MSF dataset is not only persistence of incidents, but acceleration.

More than one-third of all recorded deaths occurred within the last six years of the observation period. Annual mortality has increased markedly since the late 1990s, with 2020 representing the deadliest year on record, coinciding with the COVID-19 pandemic. During this period, multiple factors amplified risk. These included economic hardship, disruptions to legal alcohol supply, increased reliance on illicit products, home distillation, and in some regions dangerous misinformation, including the belief that consuming alcohol or methanol could prevent viral infection. Mortality has remained elevated beyond the acute pandemic phase, indicating that underlying socioeconomic and regulatory drivers persist (Figure 3).

The burden of methanol poisoning is unevenly distributed. A small number of countries account for a disproportionate share of deaths, with India, Russia, and Indonesia together contributing nearly half of all fatalities in the MSF database. In India, outbreaks are predominantly linked to illicitly produced spirits consumed by economically marginalized populations. These products may contain methanol through deliberate substitution or through unsafe production practices. In Russia and parts of Eastern

Figure 3. Annual methanol poisoning deaths and incidents worldwide, 1998 to 2025. COVID-19 period is based on the World Health Organization (WHO) Public Health Emergency of International Concern (PHEIC) timeline from January 2020 to May 2023.



Europe, repeated mass poisonings have been associated with surrogate alcohols. These are industrial or household products not intended for consumption but ingested as ethanol substitutes, some of which contain very high methanol concentrations. Indonesia presents a contrasting pattern, with a high number of incidents but fewer deaths per incident. This suggests differences in methanol concentration, healthcare access, outbreak recognition, or reporting practices (Figure 4).

At the regional level, Asia accounts for approximately 60% of all recorded deaths, with particularly heavy burdens in South and Southeast Asia. The Middle East also contributes substantially, driven largely by outbreaks in Iran. During the COVID-19 pandemic, widespread misinformation led to intentional ingestion of methanol-containing products, resulting in hundreds of deaths in a short period. These patterns highlight the intersection of poverty, alcohol restriction or prohibition, regulatory gaps, availability of industrial methanol, and limited access to emergency medical care.

One of the most striking findings is the extreme variability in case fatality rates, which range from below 10% to 100% across individual incidents. Such variability strongly indicates that outcomes are not inevitable. High-fatality outbreaks are typically associated with delayed diagnosis, lack of antidotes, absence of hemodialysis, and limited clinician familiarity with methanol poisoning. Conversely, incidents involving large numbers of exposed individuals but relatively few deaths demonstrate that rapid recognition and appropriate treatment can dramatically reduce mortality, even when exposure is widespread (Figure 5).

Seasonal patterns further underscore preventability. Deaths peak predictably during periods associated with increased alcohol consumption, including New Year celebrations, spring festivals, and year-end holidays. These recurring temporal clusters likely reflect increased demand for cheap or illicit alcohol, greater circulation of unsafe products, and strained healthcare systems. They also offer opportunities for targeted prevention, public warnings, and preparedness.

The MSF dataset from 1998 to 2025 depicts a global toxicological crisis that appears to be worsening rather than

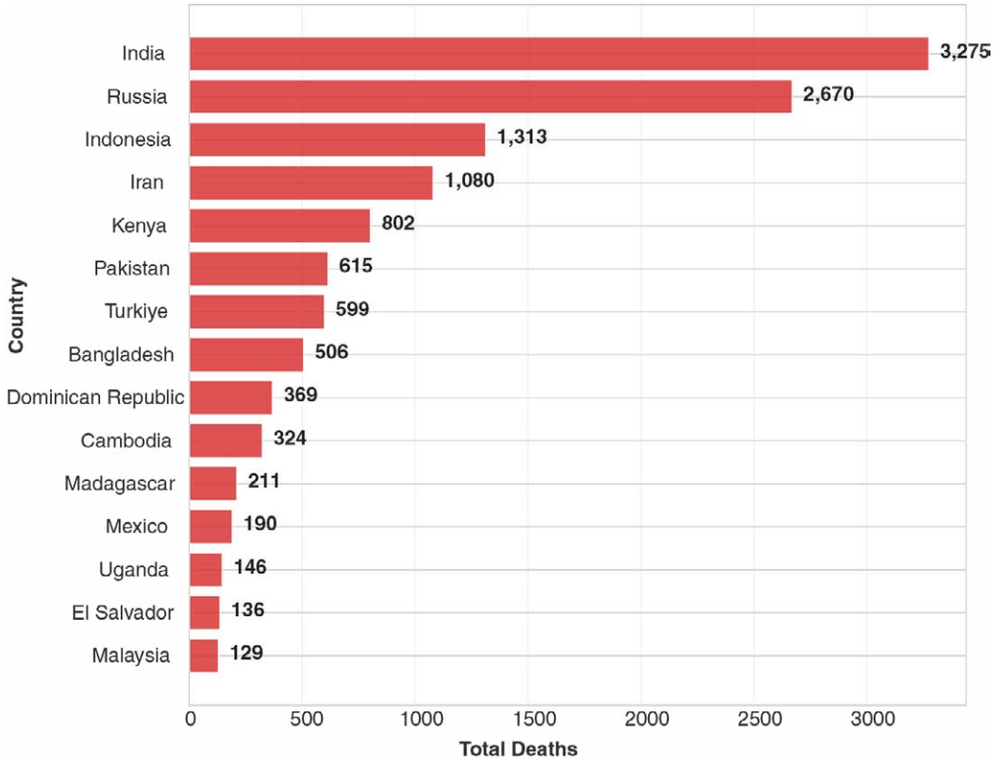


Figure 4. Countries with the highest methanol poisoning death tolls, 1998 to 2025.

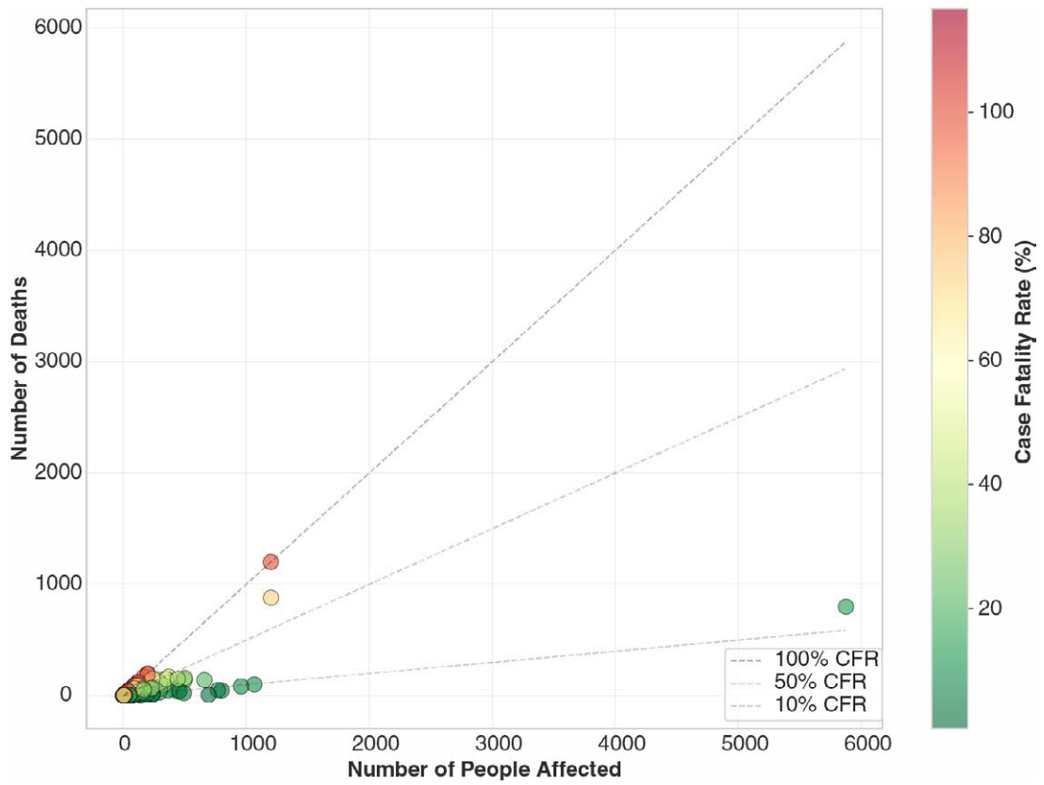


Figure 5. Relationship between number of people affected and deaths per incident, colored by case fatality rate.

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improving. Greater attention should be given to awareness campaigns, clinical preparedness, early recognition, access to antidotal therapy and dialysis, better surveillance, and broader public health and regulatory interventions addressing methanol availability and misuse. Methanol poisoning remains a largely preventable tragedy, and the accelerating trends demand renewed attention.

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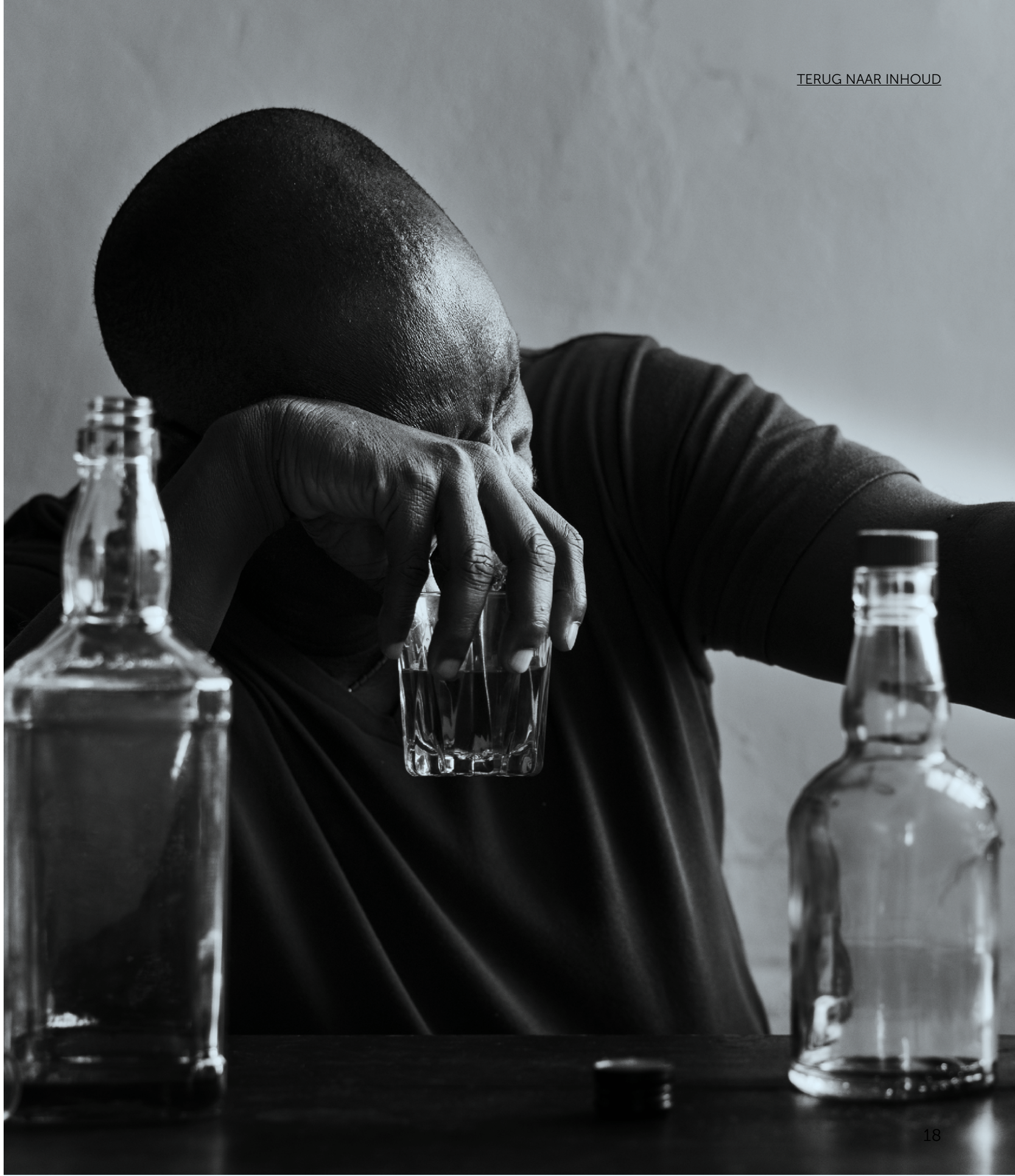


Santa's Naughty-or-Nice Food Chat Across the Atlantic

By *Marcha Verheijen and Héloïse Proquin*

Good morning! I wanted to wish you a very Merry Christmas day 🎄 🎄 ! How is it going in the US?

Good morning and a holly jolly 🎄 Christmas 🎄 to you! Everything is going well here 👍, I'm just enjoying my Christmas coffee ☕ and froot loops before I have to start preparing for the Christmas dinner tonight.

Ohh, froot loops... 😜 I miss them. We don't have them here. They say they can cause allergies or asthma because it contains Red 40, Yellow 5 (tartrazine) and Yellow 6 food dyes

I'm also getting ready for a busy Christmas day ✨. I first have to run to the store to buy some Kinder Surprise for the children.

They are so much fun 😜! But they are not allowed here 😳because they contain non-nutritive objects fully embedded in food. Can you send me some 🙏? My kids would 💙 love 💙 them.

Oh, that reminds me, I still have to get some twinkies. Do you want me to send you some 😊? You don't have them because next to the food colorants in froot loops, they also contain high-fructose corn syrup that have obesity and diabetes concerns.

Trading kinder surprise for twinkies sounds like a good plan! Let's do that. Gotta go now, talk to you later! 👏

Hey👏! Did you survive so far? We are now sitting by the tree. The kids have just unwrapped their presents🎁 and they are enjoying some Christmas cookies🍪 and Mountain dew or gatorade (and eggnog for us of course 😊)

Hi! Same here. The kids are bouncing around, overjoyed and hopped on sugar. 😁🙃 But no Mountain dew or gatorade for them. They contain brominated vegetable oil (BVO), which apparently can cause thyroid damage and neurological symptoms. So mine are enyoing some hot coco ☕. For the grownups, we had some specialized cheese treats 🧀🍷.

BTW, did you know that some US chocolate 🍫 milk brands use a seaweed extract called carrageenan that according to the EU may cause inflammation, heart disease, and even Alzheimer's 😳.

Really? EU sources suggest that eating about 55 grams of the most contaminated chocolate could expose you to the maximum daily limit of cadmium.

Remember the old days 💕 when dark chocolade was viewed as healthy 😈 because they contain flavonoids and antioxidants that lower blood pressure and improve brain functioning.

Yeah! The good old days !

So what do you have in store for the Christmas dinner? We had a starter with sliced meat: prosciutto, salami, coppa di Parma, and foie gras.

Sounds awesome😍 but I wouldn't be able to bring any piece of this at home 😟... The sliced meat is banned here because of swine vesicular disease (SVD) control, residue limits (e.g., nitrites), and processing methods....

For starter we will have breaded shrimp cocktail 🍤and thereafter continue with a main course of turkey🦃 stuffed with blueberries and instant mashed potatoes.

Sounds nice but with fresh blueberries? Or the artificial ones that contain the blue dye derived from petroleum with potential of causing nerve-cell degeneration and cancer. And, also, your instant mashed potatoes are linked to cancer because of the preservatives BHA and BHT.

Hmmm, I don't know🤷. I should check the can.

For our main course we had a nice goose with veggies and potatoes 🍷.

A European goose would also be prohibited because of growth hormones and medicine residues in animals. It's quite ironic🤔 because the US meat is banned in EU for the exact same reasons... 😒

Haha funny! 🤣 We did continue with a nice cheese plateau with fresh goat cheese, Roquefort and Camembert 🧀🍗.

All these good cheeses make me draw!😊 but none of them can be found here because the FDA has banned soft cheeses 😟 made from raw milk that are not aged for at least 60 days, a measure aimed at preventing the risk of food poisoning, particularly listeriosis.

Too bad! Then you have to come visit! For dessert, we had a nice baked Alaska 🍰.

We'll have a Cheesecake Trifle🍰 with coffee☕!

So nearly every part of diner is not allowed on the other side of the Atlantic, but our deserts are 😊

Yes, it's crazy🤯 that our 🎄 Christmas dinners🎄 are completely different just because of regulatory differences❗❗❗

Farewell Lecture

DNA, Danger and Disease: A Toxicological Journey

By *Prof. Frederik-Jan van Schooten - President Netherlands Society of Toxicology (NVT)*

On 10 November I gave my farewell lecture upon retiring from Maastricht University.
When I entered the field of toxicology four decades ago, I could not have predicted how deeply environmental contaminants would penetrate our bodies and ecosystems. In my lecture, I reflected on what I have learned, why toxicology matters more than ever, and what policymakers must understand if we aim to protect public health in the coming decades.

Herewith a short overview. The full lecture can be viewed via the following link:
https://www.maastrichtuniversity.nl/events/valedictory-lecture-prof-dr-frederik-jan-van-schooten

The exposome: the full picture we cannot ignore
If one concept captures today's environmental reality, it is the *exposome*. Every breath we take, every product we use, every meal we consume adds chemical traces to our bodies. PFAS in blood, microplastics in placenta and brain tissue, pesticides in produce, pharmaceutical residues in water, air pollution affecting billions - these exposures are not abstract risks. They are measurable, biologically active, and cumulatively significant.
These developments make one thing clear: understanding exposure and health is not optional. It is essential for public health and effective regulation.

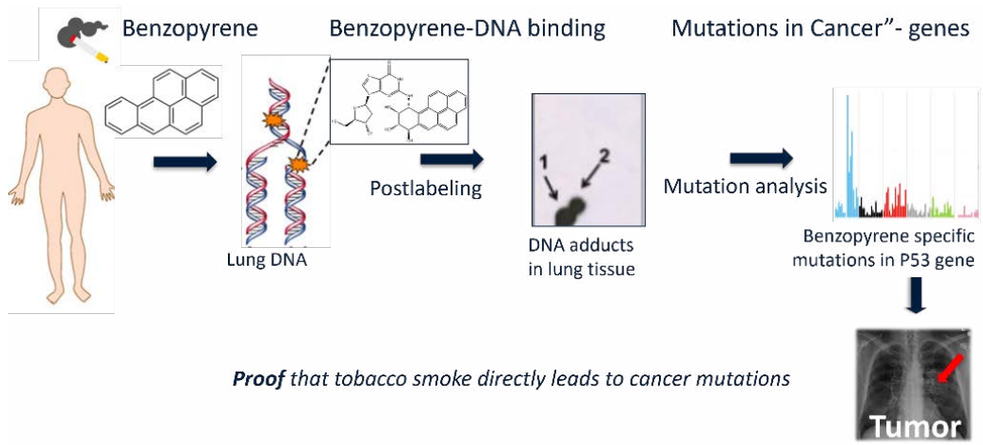
Figure 1: Already in the 1960s and 70s, the Club of Rome and Rachel Carson in Silent Spring warned that environmental pollution was outpacing our ability to control its consequences - a message echoed in countless later reports, showing that our technological development is advancing faster than our capacity to manage its harmful effects.

Science under pressure – and why independence matters
Despite growing awareness since the 1960s ***(Figure 1)***, many forms of pollution continue to increase. Global plastic production is rising towards one billion tons per year, pesticide use remains high, and air quality often fails to meet WHO guidelines. At the same time, doubt about risks is sometimes deliberately amplified to delay regulation - a strategy we have seen in tobacco, asbestos, climate, and chemicals. This underscores the need for independent, well-funded science. Recent political pressures on research institutes, both internationally and in the Netherlands, make this need even more urgent.

My scientific journey: using DNA as evidence

My own research career began with Biology at the Vrije Universiteit Amsterdam, followed by my PhD at Leiden and my early work at the Netherlands Cancer Institute. Genetic toxicology was still emerging at the time. We developed sensitive tools to detect DNA damage from environmental carcinogens such as benzo[a]pyrene- a key pollutant in air pollution and tobacco smoke. Biomarkers such as DNA-adducts provided molecular proof of harm. They helped demonstrate why smokers develop lung cancer ***(Figure 2)***, quantified risks for workers exposed to PAHs, and contributed to regulatory decisions. Even consumer products were not exempt: our studies showing excessive PAH uptake from coal-tar shampoos helped remove these products from retail shelves.

Figure 2: From DNA adduct to lung cancer. This provided direct molecular evidence that smoking not only correlates with lung cancer but actually causes it. The tobacco industry could no longer hide behind smokescreens or claim that it was merely a matter of epidemiological associations.



Tobacco and nicotine: industry design choices matter

A substantial part of my work focused on tobacco products. Real-world smoking behaviour, as we showed with colleagues at RIVM and NVWA, often results in far higher exposures than machine tests suggest- especially due to filter ventilation designed to "optimise" test outcomes. The resulting "cheat cigarettes" (*sjoemel sigaretten* in Dutch) expose smokers to misleadingly low tar and nicotine numbers. The research triggered parliamentary questions, public outrage, a criminal complaint against the tobacco industry, and a broad debate on testing and regulation - showing how science can reveal what is otherwise hidden and directly shape policy and public awareness.

Newer products such as IQOS, marketed as "less harmful," also raise concerns ***(Figure 3)***. In human lung cells we saw oxidative DNA damage after short exposures. For policymakers, the message is simple: product innovation in the tobacco sector does not automatically equal risk reduction. Regulation must be proactive, not reactive.

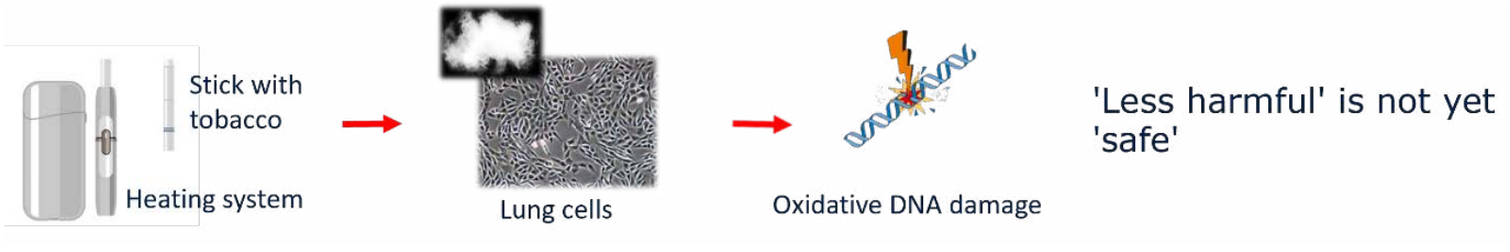


Figure 3: IQOS – "I Quit Ordinary Smoking" - is a heat-not-burn product in which tobacco is heated to about 350°C rather than burned, producing an aerosol with nicotine and other tobacco constituents but without conventional smoke. The emissions contained significant levels of free radicals and tobacco-specific nitrosamines, both genotoxic substances. Even one hour of exposure caused oxidative DNA damage, and higher concentrations led to DNA strand breaks and activation of DNA-repair genes. In short, IQOS is not without danger: even brief exposures can induce DNA damage in airway cells, with potential implications for lung cancer.

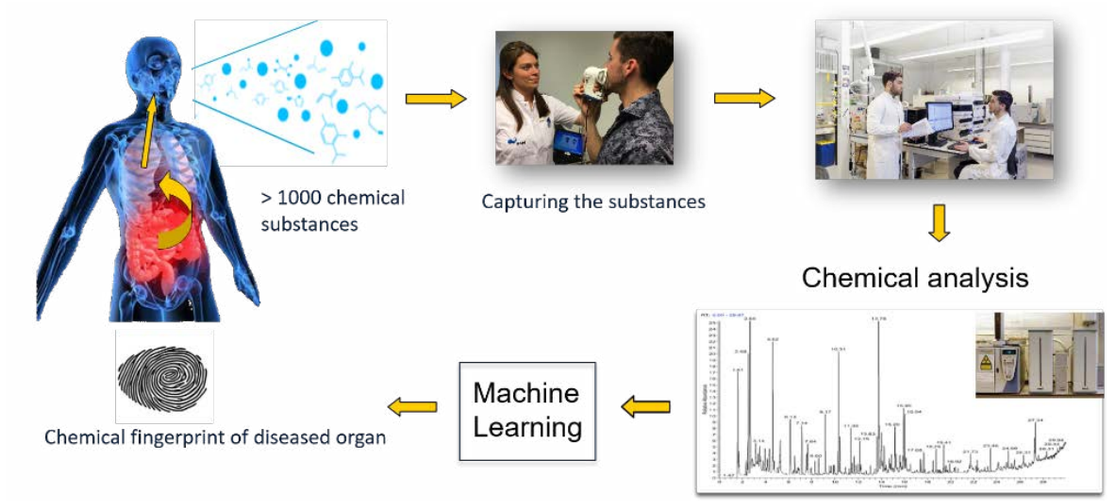


Figure 4: Disease processes in the body release volatile molecules into exhaled breath, which can be collected and chemically analysed using gas chromatography and mass spectrometry. Machine learning is then used to recognize patterns - identifying which molecules indicate illness or health - allowing us to generate molecular fingerprints that correspond to specific diseases or inflammatory states

Breath analysis: new tools for prevention and early diagnosis

In the early 2000s, I started a research line on "breathomics". Exhaled breath contains thousands of volatile compounds that reflect ongoing biological processes in lungs, liver, and gut ***(Figure 4)***. Working with colleagues across disciplines, we demonstrated that breath profiles can distinguish disease states ranging from asthma and COPD to inflammatory bowel disease and liver cirrhosis.

The next step lies in targeted probes - such as limonene for liver function - now moving toward clinical implementation. This type of innovation only succeeds when researchers, clinicians, industry partners and regulators work together. Breath analysis could ultimately help shift healthcare toward earlier, non-invasive detection - something every public health system urgently needs.

Training the next generation in environmental health

Alongside research, education has always been central to my work. In the 1990s I helped design the Environmental Health Sciences (Milieugezondheidskunde, MGK) programme. It integrated toxicology, epidemiology, environmental chemistry, law, economics, and public health - a combination we now desperately need again as chemical exposures rise and climate change reshapes disease patterns.

I also supervised forty PhD students, with twelve more currently underway to finish their thesis the coming years. Their projects span topics such as the absorption of microplastics through the lung and gut, dietary supplements that may enhance athletic performance but pose risks to the heart, the safety of novel laboratory-derived proteins, and the role of Maillard reaction products in processed foods in intestinal diseases. Their work drives the field forward. Young researchers need academic environments that safeguard independence, reduce unhealthy competition, and provide space for creativity. Investing in early-career scientists is, ultimately, an investment in long-term public health policy.

Leadership and management: what institutions need to thrive

During my years at Maastricht University I served as head of several departments, division leader within research institute NUTRIM, and board member and vice-dean during the transition to Faculty Health, Medicine and Life Sciences (FHML) and MUMC+. These experiences taught me that scientific excellence requires stability: trusted teams, reasonable workloads, and long-term structural funding. Without these basics, even the strongest research vision fails.

What policymakers should take away

Across all these experiences, the message that emerges is consistent:

- **Environmental exposures are universal, measurable and impactful.**
- **Delay - whether political or strategic - has real health consequences.**
- **Independent science must be protected, not weakened.**
- **Regulation must anticipate new risks, not wait for definitive harm.**
- **Investments in research and education pay off in prevention and reduced healthcare burden.**

Looking forward with cautious optimism

We face major challenges: persistent chemicals, changing diets, air pollution, climate-related health pressures, and widening health disparities. Yet I remain optimistic. The new generation of scientists is collaborative, technologically skilled, and motivated by societal impact.

The future will require collaboration across sectors, sustained investment in independent science, and the political courage to prioritise human and environmental health. Toxicology can serve as a compass in that journey - revealing what is hidden, quantifying what is uncertain, and making risks visible long before they become crises.

The retiring professor delivers his farewell lecture, with a reference to the NVT - note the logo on the slide. (Renskephotos)

A group of people in academic gowns. Try to spot the toxicology professors. (Renskephotos)

Jingle Jolt: **San Francisco Sues Food Giants for Ultra-Processed Health Hazards**

By *Héloïse Proquin*

Ho Ho Ho! San Francisco has filed a landmark lawsuit against major food companies, accusing them of producing and marketing ultra-processed foods that contribute to chronic health issues like obesity, diabetes, heart disease, and cancer, while burdening local governments with massive healthcare costs[1].

Lawsuit Details

The suit, filed on December 2, 2025, in San Francisco Superior Court by City Attorney David Chiu, targets ten corporations including Kraft Heinz, PepsiCo, Coca-Cola, Nestlé, and others behind products like Oreos, Cheerios, Lunchables, and sugary cereals. It alleges violations of California's Unfair Competition Law and public nuisance statutes through deceptive marketing that hides health risks, despite industry awareness dating back decades. The city seeks unspecified damages, civil penalties, restitution for healthcare expenses exceeding $100 billion annually nationwide, and court orders to stop deceptive practices and mandate corrective actions like consumer education.

In line with the current administration

The current Trump administration started a fight against obesity and other chronic disease by issuing a new directive that could restrict foreigners with conditions like diabetes or obesity from visiting or living in the United States[2]. Furthermore, left-leaning officials and the Trump administration have found rare agreement on concerns over ultra-processed foods. In April, Kennedy announced a U.S. ban on eight common artificial food dyes. He and the Make America Healthy Again initiative have urged companies to eliminate ingredients like corn syrup, seed oils, and artificial dyes, citing links to health issues. In response, some food companies have adjusted products; Coca-Cola, for instance, agreed this summer to switch to real cane sugar in U.S. drinks.

Health and Economic Impacts

Ultra-processed foods, defined as chemically altered products with minimal whole ingredients designed to trigger cravings, now make up 70% of the U.S. food market and disproportionately affect low-income and communities of colour. Officials claim these items fuel a public health crisis, with companies profiting while cities bear treatment costs for related diseases. Chiu stated the products are "engineered to be addictive" and "unrecognizable and harmful to the human body."

Industry Response

Food industry representatives, such as the Grocery Manufacturers Association, retort there's no "official naughty list" as there is no agreed scientific definition of ultra-processed foods and that classifying them as unhealthy ignores full nutrient content, potentially misleading consumers. They emphasize compliance with FDA safety standards for safe, affordable products. This is the first U.S. governmental lawsuit of its kind against ultra-processed food makers.

References:

[1] https://www.bbc.com/news/articles/c93wgeqpv0eo

[2] https://www.nbcnews.com/news/us-news/trump-directive-deny-visas-health-conditions-finances-rcna243420

Door Evan-Amos - Eigen werk, Publiek domein, https://commons.wikimedia.org/w/index.php?curid=13392219

Counterfeit Pills in the Netherlands: **At Least 13 Deaths Linked to Nitazene-Contaminated Oxycodone**

By *Barae Jomaa*

On the 13th of December 2025, NOS News reported that counterfeit medication sales in the Netherlands have caused at least 13 deaths, far exceeding previous estimates1. An Erasmus MC survey commissioned by NOS indicates that the official figures, including six confirmed fatalities and four serious poisonings reported by the Netherlands Forensic Institute (NFI), likely underestimate the true impact. Additional reports from the Netherlands Poisons Information Centre, the Trimbos Institute, and hospitals capable of testing for hazardous substances suggest at least six other suspicious deaths and over 20 serious poisonings where hospital intervention was critical.

The main substance implicated is nitazene, a potent synthetic opioid with a high risk of overdose. Although prohibited in pharmaceuticals, nitazene has been detected in counterfeit pills sold online as oxycodone. Hospital pharmacist and toxicologist Corine Bethlehem of Erasmus MC notes that nitazene was first found in a blood sample in October 2024, and the number of victims has sharply increased since then.

Many victims obtained these pills via websites such as Slaappillen.net. Two individuals connected to the site are in court, but they appear to have played a minor role. Authorities continue to investigate the main perpetrators, who seem to remain active. Bethlehem warns of the extreme dangers of buying medication outside licensed pharmacies and highlights underreporting due to limited testing in certain regions and among survivors.

The human toll is stark, exemplified by a 29-year-old Amsterdam woman who died after taking a pill she believed was oxycodone. Experts emphasize that reported cases likely represent only a fraction of the total impact. The combination of potent opioids, unregulated online distribution, and limited testing underscores the urgent need for improved monitoring, regulatory enforcement, and public education to prevent further harm.

Reference:

(1) NOS News. Consequences of selling counterfeit pills even more serious than expected: at least 13 deaths. NOS News. 2025 Dec 13. Available from: https://nos.nl/artikel/2594333-gevolgen-verkoop-neppillen-nog-ernstiger-dan-gedacht-zeker-13-doden

AIO toxafette - Matthias Hof

In the toxafette, PhD-students working in the toxicology field get the chance to open up about their experiences in performing research. Every issue a new candidate answers a series of questions, and then pass the baton to a fellow PhD-student. This time Matthias Hof, from the Dutch National Institute for Public Health and Environment (RIVM), tells us about his project.

Can you introduce yourself?
My name is Matthias Hof. I am a researcher at the Dutch National Institute for Public Health and Environment (RIVM) and I am also an external PhD student at the Institute for Biodiversity and Ecosystem Dynamics (IBED) at the University of Amsterdam. My research focuses on the assessment of mixtures in the living environment. My PhD is part of the project SPR ToxDown, which is funded by the strategic program of the RIVM.

How would you explain the subject of your research to a layperson?
With great difficulty! I have tested out different versions on my grandparents for the past three years, but I have not been very successful so far. My research focuses on the development of new indicators for the risk of mixtures of substances in the environment. Usually, substances are assessed one at a time, but in practice we are never exposed to just one single substance. The result is that we currently might underestimate the risks due to exposure to substances in the environment.
Currently, I am primarily focusing on mixtures in ambient air and human health. I am developing two indicators for mixtures in air, from two completely different angles. The first indicator is based on emission data, which we use to estimate concentrations and calculate mixture risks of multiple substances in air for the entire Netherlands. However, modelling approaches like this are always limited by the substances you know are in your mixture. It's not possible to include unknown substances. That is why we are also doing a pilot with applying effect-based methods (EBM) for the assessment of mixtures in air. In the attached picture you can see me and my co-promotor Milo de Baat installing passive air samplers in the city of Nijmegen. EBM are a lot more developed for the assessment of water quality (see for example the previous Toxafette by Jaimy de Schepper), but are rarely applied for the assessment of air as of yet.

How is your research related to the field of toxicology, and why did you choose this subject?
Already during my masters I was intrigued by the topic of mixture toxicity. There are already so many challenges and uncertainties in the assessment of individual substances… and then there are thousands of them at the same time? In a sense it seemed like an impossible task to assess these mixtures, so I was curious how toxicologists addressed this. I also enjoy finding or applying simple methods to address complex topics, which is often the first approach for mixture assessment. I kind of think of it as measuring the size of the forest first, before worrying about all the trees.

What was your motivation for starting a PhD program?
I actually started out after my Master's working as a "normal" employee at the RIVM four years ago. My first year I worked on a lot of different projects, including advanced materials, risk assessments of solar panels and wind turbines, and mixtures in freshwater. Certainly very interesting, but I also felt the desire to focus on and get good at a single topic. Luckily, I got the opportunity to do this PhD within SPR ToxDown.

How do you see the future of your research topic (follow-up research / social impact)? What do you hope for?
Considering mixtures in general, I hope that in the near future policy and regulation will better account for the fact that we and the environment are exposed to multiple substances at once. There is a huge task for scientist to improve the assessment of the risk and/or impacts due to exposure to these mixtures and communicate the results. However, I also think we should recognize that there will always be a certain level of uncertainty, which we also may not be able to quantify. We should be wary that this uncertainty does not lead to decision paralysis and procrastination with addressing mixtures in policy.
I also hope that our pilot with effect-based methods for air quality is successful, and that this is continued beyond our project. There is a lot that will need to be explored, e.g. testing and comparing various sampling methods, bioassay selection, developing effect-based trigger values for air. The water sector is a lot further in this regard, and can be a real inspiration.

Could you describe any experiences collaborating with people or organizations outside of academia to support your research? (Collaborative efforts)
Within SPR ToxDown we are focusing on air quality in Nijmegen as a case study. Nijmegen has an industrial site and port on the edge of the city, with a long history of odor complains and concerns from local citizens. We have regular meetings with members from the local municipality, GGD, and Omgevingsdiensten, who also share their expertise and data (e.g. emission permits) with us. We also try to inform and involve the local residents in our research. This year, we have held a workshop where we gathered input from citizens on which locations to sample at for our pilot with EBM. I was quite nervous at first: what if the residents don't understand that we're trying out a new method or are very skeptical in general? But the workshop went really well and we received great input!

What is the biggest challenge for you in doing PhD research? (Personal challenges)
So as I said one of the reasons for starting this project was that I would like to focus on a single subject. Ironically, my research now is actually very multidisciplinary and varied. I am doing data science, exposure modelling, field and lab work, and I've even dabbled in social scientific work performing and analyzing structured interviews of experts in the field, and of course the interaction with stakeholders from our case study. I really enjoy the variety and there's always something new to learn, but it's also quite challenging to always be doing something new.

What are your thoughts on using new technologies like artificial intelligence in toxicology research? Are you using any of these technologies in your work? (Research methodology)
I think they provide great opportunities! I think predictive models in general (AI or otherwise) are a great resource for toxicology, seeing the large data requirements for proper risk assessments. Personally, the release of language models such as ChatGPT has been a great help with coding for data science purposes. I am not trained as a data scientist, and it used to take me hours to find the right functions or fix errors. Now you can just ask!

What goals do you have regarding your career after finalization of your PhD? Would this be inside or outside academia, and why? Would you consider going abroad?
I would love to keep working at the RIVM and continue working on my current topics. I guess technically that would be outside academia? But it doesn't really feel like it.

Please answer the question from the last toxafette PhD-candidate: How does risk perception influence your research area?
Risk perception is very important for my research area. I believe a clean living environment should not only pose no health risk, but ideally it should also be perceived as such by the people living there. However, fundamentally absence of risk is impossible to prove so it might be hard to get to that point, at least not without social science. Collaborating with and listening to local stakeholders is essential both to increase trust in scientific results, and to improve the science itself, e.g. by adapting research questions to better address local concerns. Specifically for our project, I worry a bit about how to best communicate the results from EBM of air in Nijmegen. What if we see large differences in bioassay response between locations? Because it is such a new approach there is little to compare it to and provide context. However, up to now local residents have been very interested and understanding, so I'm sure we'll figure it out together.

The 17th International Congress of Toxicology

Beijing, China, October 15-18, 2025

TRAVELER:
Jiayi Yang[1,2]
[1]Institute for Risk Assessment Sciences, Utrecht University
[2]Farm Animal Health, Utrecht University

I am grateful to have the support from NVT travel grant to attend the ICT meeting in Beijing. It was amazing to return to the city where I did my bachelor after 5 years, this time presenting research from my PhD project.

I presented our research on the reproductive effects of polystyrene nanoplastics by using a bovine in vitro fertilization (IVF) model. The benefits of this model are (1) its larger similarities to human reproduction, particularly from the oocyte maturation to early embryo development phase compared to rodents or aquatic animals, and (2) the use of slaughterhouse-derived materials, which eliminates the need for experimental animals.

In our study, bovine cumulus-oocyte complexes collected from slaughterhouse ovaries were exposed to pristine polystyrene nanoplastics (50 nm or 200 nm) during 23 hours of in vitro maturation, followed by IVF and embryo culture. Nanoplastics uptake, oocyte maturation, and embryo developmental competence were assessed. Possible mechanisms of toxicity were assessed via transcriptomics and metabolic activity.

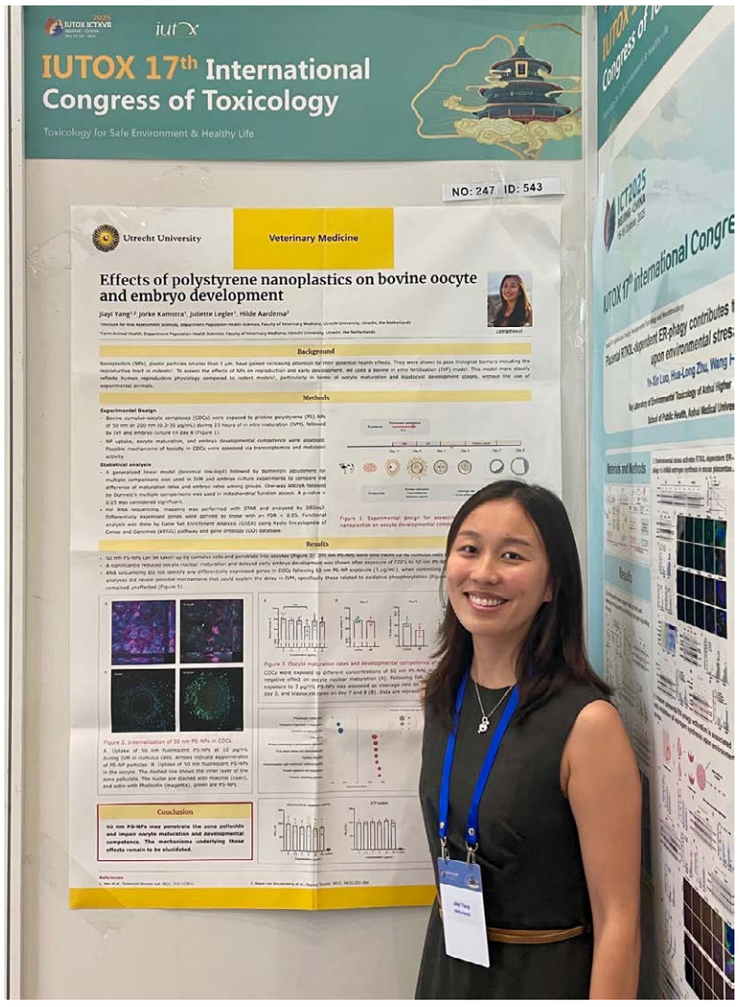
We showed the internalization of 50 nm nanoplastics in the oocyte, which coincided with a reduced oocyte maturation rate and delayed embryo development at a concentration of 3 μg/mL. However, these effects did not appear to be driven by major transcriptomic or mitochondrial functional alterations.

Highlights of the conference:
The conference sessions highlighted the latest advances, ranging from molecular mechanisms and safety assessment to environmental health and regulatory science. A great deal of attention was given to artificial intelligence-assisted toxicology and NAMs, reflecting a growing shift toward next generation risk assessment.

The session I really enjoyed focused on the potential assay interferences caused by nanomaterials. Such interferences can result in inaccurate toxicity predictions, hindering risk assessment. Although nanoplastics were not the focus, the talks made me reflect on the assays we used to test the toxicity of different nanoplastics. The session reinforced the importance of validating assay performance for specific nanomaterials or particles before conducting experiments.

My take home message from the ICT 2025 was that the future of toxicology will benefit from deeper cross-disciplinary integration, bringing together artificial intelligence, exposomics, microphysiological systems, to build more predictive science and ultimately improve population health.

Sustainability at ICT 2025:
Digital proceedings were used instead of the printed versions. The conference also offered shuttle buses from the venue to the gala dinner, which is in the center, reducing the use of individual taxi rides.



13th World Congress on Alternatives and Animal Use in Life Science

Rio de Janeiro | August 31st - September 4th, 2025

Presentation

At the WC13 in Rio I had the opportunity to present my project on thiabendazole (TBZ) in a 5- minute oral presentation and with a poster. TBZ is a widely used post-harvest fungicide and anthelmintic drug. It is quickly metabolized by humans with 84% being excreted via the urine within the first 24h. Despite the fast metabolism, its major phase I metabolite hydroxy-TBZ (OH- TBZ) has been found in over 90% of the population. *In vivo* experiments reported species differences in toxicity to TBZ in rats, mice and humans. TBZ is believed to be taken up by organic cation transporter (OCT) and upon entering the human body TBZ is first metabolized by CYP1A2 to OH-TBZ. Subsequently, it is further transformed by UGT, SULT and GST enzymes. This project aims to investigate interspecies differences in absorption, metabolism and excretion (ADME) in human and rat *in vitro* models of the gut microbiome, liver and kidney using LC-MS.
TBZ does not seem to get metabolized by the human gut microbiome. In primary hepatocytes the intrinsic clearance was more than double in humans compared to rats. Formation of OH-TBZ was faster and higher in human compared to rat hepatocytes. In kidney microsomes phase II metabolite OH-TBZ glucuronide was detected. In the kidney OCT does not seem to be the sole transporter involved in the uptake of TBZ. The experimental data will be integrated in PBK models to help to improve their accuracy.
Both the oral and poster presentation sparked valuable discussions about my project and potential collaborations with researcher from other groups.

Learnings from conference

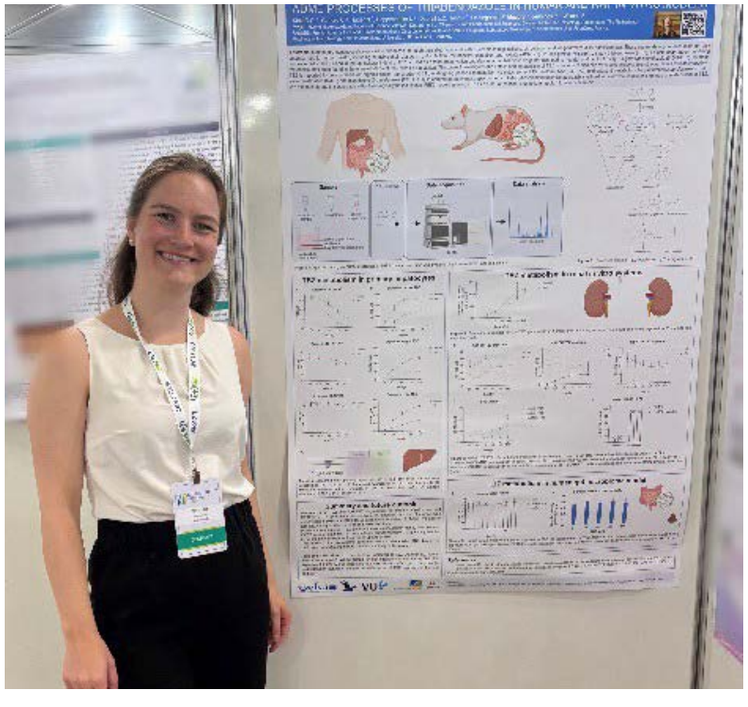
Reyk Horland, CEO of organ-on-a-chip (OoC) company TissUse, presented a liver ring trail in collaboration with multiple pharmaceutical companies. Aim of this project is to investigate the reproducibility of their OoC device between different labs. All chemicals and cells used in these experiments will be of the same batch. The measurements of end points will be conducted in the same lab. Digital twins of will be developed from the experiments to help translating the data to humans.
Another liver ring trail is underway that will compare the experimental results of 9 different OoC companies to multiple drugs to compare the different devices and to access their predictivity.
A researcher that inspired me was dr. Patrik Lundquist from Uppsala University. In his lab a new protocol to isolate primary enterocytes was developed. These cells show in vivo like expression of many ADME protein and functionality of many phase I and II metabolizing enzymes. His model could be useful for my further research as an accurate model to access xenobiotic metabolism in the intestine.
A value new technique presented by dr. Georg Aichinger from the ETH Zürich. His group works on gut microbiome models. Their data shows huge donor dependent differences to some xenobiotics when analyzed individually. This raises the question if personalized and population-specific risk assessment for the gut microbiome will be necessary in the future.

Take home message

The shift from animal experiments to NAMs for toxicity testing has not been fully realized yet, but there is considerable effort underway to improve current models and increase their predictivity and reproducibility.

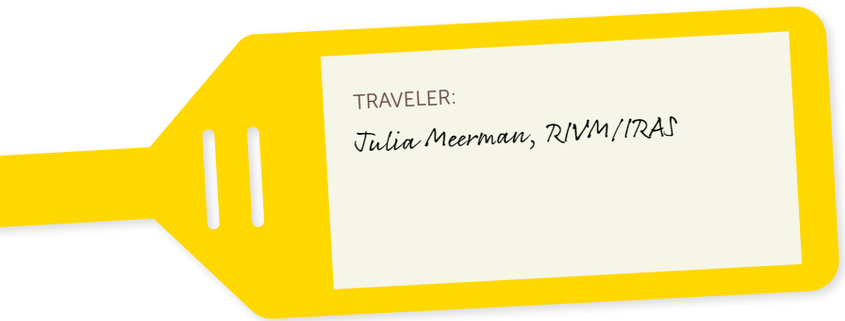
Climate efforts

The organizers offered a lot of vegetarian food. The food containers and cutlery were mostly made out of wood. My colleagues and I shared rides to and from the venue.



International Neurotoxicology Association Conference

September 7-14, Sommarøy, Norway



Thanks to the support of the NVT travel grant, I was able to join the biannual meeting of the international neurotoxicology association (INA). In addition to enjoying the beautiful view from the conference room and listening to exciting scientific presentations, I also had the opportunity to present my own work. Exposure to pesticides has been associated with an increased risk of developing Parkinson's disease later in life. Currently, neurodegeneration is not considered during safety assessment of novel pesticides. During our session, entitled 'Going the distance with neurodegeneration: From human biology to next generation risk and safety assessment of pesticides', our goal was to highlight facets of a Next Generation Risk Assessment (NGRA) approach to incorporate neurodegeneration in a novel regulatory safety assessment framework for pesticides.

My presentation focused on two practical aspects of using in vitro studies in this context. Our first study aimed to determine to what extent changing characteristics of exposure (e.g., exposure duration and frequency of exposure) had an impact on effects of pesticides measured in vitro. Overall, we found that an acute exposure (24 hours) was not the most informative for any of the pesticides tested. Moreover, the exposure scenario that was most devastating differed between the pesticides tested. To use in vitro tests for regulatory safety assessment of pesticides, relying solely on acute toxicity tests may not be sufficient and an optimal (set of) exposure scenarios will need to be established. Our second study covered in my presentation aimed to quantify part of the Adverse Outcome Pathway network previously established during my project. Here we found that not all pesticides activated the Key Events measured in the same chronological order, which may be due to differences in primary mechanism of action. As a safety assessment framework for pesticides should cover a broad range of chemicals with differing mechanisms of action, a tiered approach may be needed.

Although the INA is a relatively small organisation, the meeting covered a very broad range of topics related to neurotoxicity. The meeting started with a very insightful lecture on using the social exposome (i.e. neighbourhood (dis)advantages) to guide policy decisions, leading to an improved replacement plan of lead pipes in local neighbourhoods. It was really inspiring to hear how science can inform and improve regulatory decision making, leading to measurable improved individual outcomes (lower measured blood lead levels in this case). Staying on the topic of science and regulation, one of the speakers in our session covered some major challenges that we face when working towards NGRA-based safety assessment (i.e. Lack of consensus, different pace of science and regulation, and lack of shared understanding). I believe it was valuable to have this discussion in a room of (mostly) scientist, some of whom also work on methods with the aim to use them in a regulatory setting.

Finally, what I found very informative were the many presentations on more complex models for neurotoxicity. There were some interesting presentations on iPSC-based co-cultures that combine multiple neuronal cell types, which would allow for more relevant models to assess neurotoxicity. Another more complex model extensively covered was the zebrafish. It was interesting to hear how behavioural assessment batteries are being extended to cover a broad range of endpoints within the same test, allowing for assessment of endpoints that cannot be captured by cell model systems. These presentations stimulated me to consider how to incorporate these complex models within my project. Although the majority of the presentations were presented in the context of assessing endpoints relating to neurodevelopment, some may also prove useful for endpoints relating to neurodegeneration. On the one hand directly as model systems to use and on the other hand as learning opportunities, as some of the hurdles that were discussed concerning, for example, the interpretation of complex endpoints in a safety assessment context, may be shared between the two fields.

My "take home message" from this conference would be that it is crucial, when working towards solutions for complex regulatory problems, to keep the discussion between different parties involved going. Instead of building bridges later on, it may be more beneficial overall to prevent a gap from developing.

Conferences in general are crucial for advances in research, but are not very environmentally friendly. Some small, but important steps taken during this year's INA meeting were the use of reusable cardholders (no lanyards needed!) and the lack of single-use material for food and beverages in an effort to reduce waste.

Human Teratogens Course

February 23–25, 2026

This live, virtual continuing education course covers teratology principles, current knowledge and controversies, and counseling strategies. Session recordings available afterward. Free registration for individuals in low income and lower-middle income nations.

read more...

Society for Birth Defects Research & Prevention
EST. 1960 AS THE TERATOLOGY SOCIETY

Emerging Investigator Travel Award

ToxForum is offering multiple Emerging Investigator Travel Awards to their 2026 Winter Meeting, thanks to the David Miller Awards Fund. Nominations are accepted on a rolling basis, but the sooner they are received, the sooner the Awards Committee can select recipients who can then begin planning their meeting attendance. Winter Meeting registration opens November 19!

read more...

50 Years The Toxicology Forum

Inschrijving TiO

Voorletters	Achternaam	Opleider	Datum inschrijving
L.	Ockhuijsen	Dr.ir. P.T.J. Scheepers	04-12-2025
L.P.M.	van Bussel	Dr.ir. P.T.J. Scheepers	04-12-2025
L.J.	Habich-Reijntjes	Prof.dr. D.J. Touw	04-12-2025
T.	Sörmus	Prof.dr. R. Masereeuw	04-12-2025

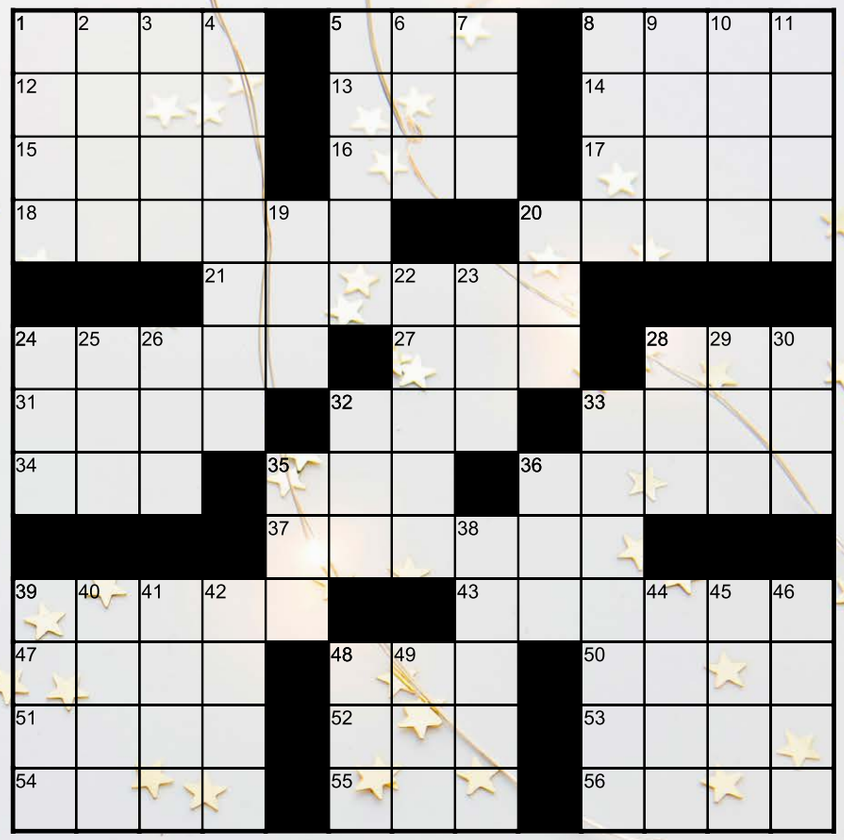
Rectificatie:
In de vorige TCDD (nr. 45) hebben we per abuis een onjuiste lijst van TiO-inschrijvingen opgenomen. De online versie is inmiddels gecorrigeerd.

TERUG NAAR INHOUD

Christmas Puzzle

Submit your answers for a chance to win a prize!

Send your solution to the editors of the TCDD via redactie@toxicologie.nl, stating 'result Christmas puzzle 2025'.



ACROSS
1. Gaiter
5. Took a seat
8. U.S. State
12. Wax
13. Reverential fear
14. Open country of S Africa
15. Related
16. An explosive
17. A Great Lake
18. Miserly
20. Unbolt
21. Eagles nests
24. Damn
27. Handwoven Scandinavian rug
28. Cot
31. Military detachment
32. New Guinea seaport
33. Vended
34. Beep horn
35. Obtain
36. Lapwing
37. Required
39. Hidden
43. Proverbs
47. Sewing case
48. Make lace
50. Bull
51. Foot part
52. Small truck
53. Therefore
54. Single items
55. - Kelly
56. Writing table

DOWN
1. Confidence trick
2. Small dog
3. Opera solo
4. Most taut
5. Woodland deity
6. Grain beard
7. 9th letter of the Hebrew alphabet
8. Baking chamber
9. Medicinal plant
10. Hip bones
11. River in central Europe
19. Soap ingredient
20. America (Abbr)
22. Angry
23. Optic organ
24. Young bear
25. Prefix, one
26. Tear
28. Bend
29. Biblical high priest
30. Once common, now banned, insecticide
32. Sheltered side
33. Calmed
35. Wildebeest
36. Prefix, foot
38. Outmoded
39. Mexican currency
40. English college
41. Govern
42. Ceases living
44. Pierce with horn
45. Work units
46. Cry-baby
48. Large barrel
49. Dined

The editorial team wishes you happy holidays!

TERUG NAAR INHOUD