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TCDD

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SPECIAL THEME

War Toxicology

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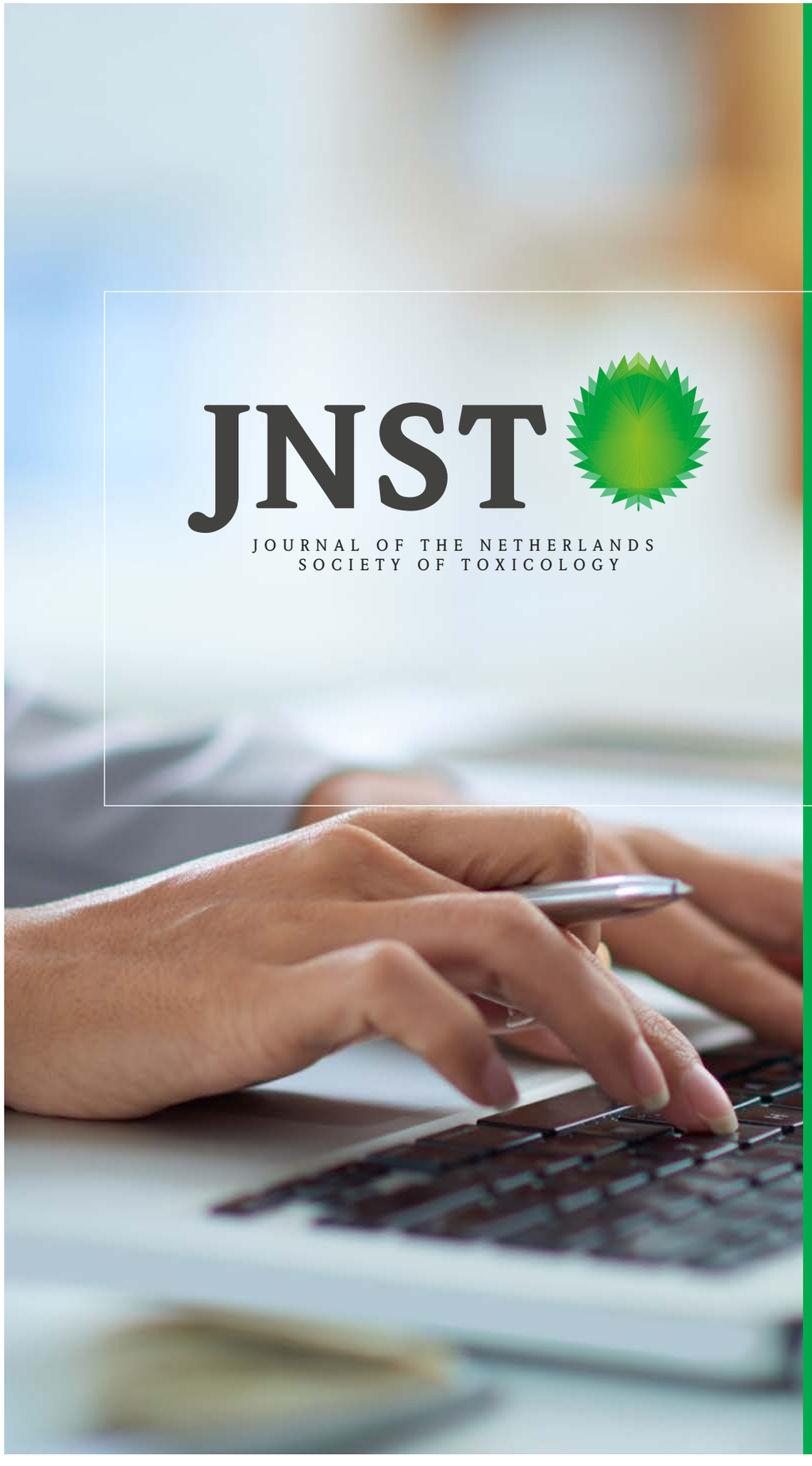

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TERUG NAAR INHOUD

Dear readers,

It is my pleasure to introduce myself as the newest member of the TCDD editorial team. For my first issue, I am joining a conversation that feels both timely and deeply important: the toxicological consequences of war. At a moment when the conflict involving the United States and Iran has again placed the human and environmental costs of war in the headlines, this issue looks beyond the immediate violence and considers the toxicological consequences that may persist long after active hostilities end. War is often described in terms of its immediate human, political, and military costs, but here we look further at the chemical, environmental, biological, and even intergenerational traces that conflict leaves behind. These legacies may be less visible than destroyed buildings or battlefields, but they can remain present in soil, air, water, bodies, and communities long after the fighting has stopped.

In this issue, our contributors explore war toxicology from several perspectives. We look back to the heavy metal contamination still present on former First World War battlefields, where the “iron harvest” continues to bring shells and fragments to the surface. We examine the toxic emissions of military burn pits and their long-term health consequences for exposed personnel and nearby populations. Other pieces consider epibatidine as both poison and potential painkiller, and the molecular memory of war through trauma, epigenetics, Agent Orange, and inherited biological effects. My own article, “The Invisible Toxic Legacy of War,” reflects on the difficulty of studying toxic exposures in conflict settings, where evidence is often incomplete, but the risks may be profound. Together, these stories show that war does not end toxicologically when the weapons fall silent. Its effects can persist, transform, and reappear across generations.

Beyond the special theme, this issue also brings toxicology into current public debates, from burnt food and carcinogenic compounds to the unexpected discovery of asbestos in coloured play sand for children. These stories remind us that toxicology is not only a scientific discipline, but also a way of making hidden risks visible. I hope you enjoy this issue, and I look forward to contributing to the TCDD editorial team.

Sincerely,

Devon Barnes

Call for Contributions – Special Issue on Pharmaceutical Toxicology

We are pleased to announce that the upcoming issue of *TCDD* will be dedicated to Pharmaceutical Toxicology. This special issue will explore topics such as regulatory toxicology, ethnicity and genetic diversity in drug development, and beyond.

Interested in contributing? We welcome your ideas and submissions. Get in touch with us at redactie@toxicologie.nl.

Submission deadline: 11 September 2026

Photo by Jess Bailey on Unsplash

News from the board

Summer is just around the corner! A time that often invites a bit of reflection, while at the same time looking ahead to what is coming. For the NVT, the coming months will once again bring us together around science, exchange, and community.

NVT Annual Meeting – 23 & 24 June, Amersfoort
On 23 and 24 June, we will meet at *De Eenhoorn* in Amersfoort for the **47th NVT Annual Meeting**. These two days are always a highlight for our members: a chance to share scientific insights, and to reconnect, meet new colleagues, and strengthen our network. Registration is open, with an **early bird discount available until 22 May**. We encourage you to register in time, particularly if you would like to participate in one of the workshops.

You can stay at the *Amrâth Berghotel Amersfoort* (€125 per night, including breakfast). Rooms have been reserved for us, but availability is limited. Booking early is advisable.

More information about the programme will follow soon via the website. We very much look forward to seeing many familiar and new faces in Amersfoort.

General Assembly – 4 June (hybrid)
This year, the General Assembly (GA) will be organized separately, on **Thursday 4 June (15:30–17:00)**. By doing so, we hope to create more time during the annual meeting itself for poster viewing, informal discussions, and networking. The moments where collaboration often begins.

- In person at RIVM in Bilthoven (limited capacity, first come, first served)
- Or online via Teams (link will be shared together with the meeting documents)

Board Vacancies
As our society continues to evolve, so does the composition of the board. From June onwards, we are looking for new board members. This is partly because Anene Kienhuis will be stepping down, and partly because we want to follow up on the outcomes of our future-proofing efforts.
We are therefore looking for:

- one board member with a focus on communication/ sections
- one board member with a focus on sponsorship

Joining the board is a great opportunity to contribute to the direction and vitality of our NVT community. If you are interested, please reach out via secretaris@toxicologie.nl.
Or contact individual board members for more information.

Website update
Behind the scenes, we are steadily building our renewed website. While the foundation is in place, several elements, such as the homepage and events page, are still being refined. We recently tested the events module in a smaller setting, which provided useful insights for future implementation. We are now exploring how best to use this for upcoming meetings. In parallel, a new email style is being developed to align with the look and feel of the website.

If all goes well, we hope to present - and possibly launch - the new website during the Annual Meeting. A small but important step in keeping our communication and society future-proof.

Finally
The months ahead offer many opportunities to connect, reflect, and move forward together as a community. For many of us, the NVT truly feels like a family.

I wish you all a wonderful and inspiring summer! I hope to see many of you at the General Assembly and, of course, in Amersfoort.

On behalf of the Board,

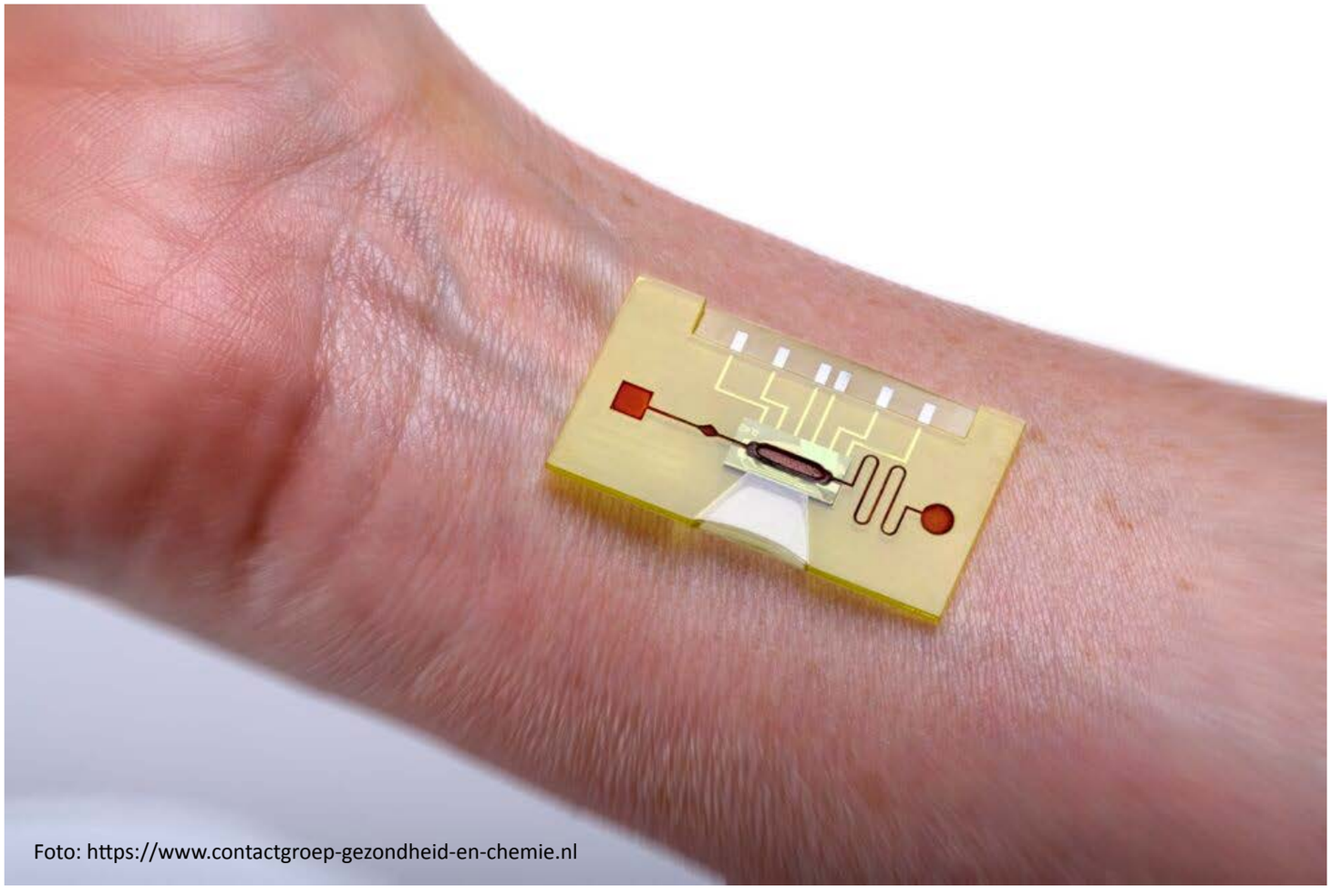
Frederike-Jan van Schooten

SECTIE
ARBEIDSTOXICOLOGIE

Presentaties online beschikbaar van het middagsymposium

“De toekomst van biomonitoring op de werkplek: een onmisbare stap vooruit?”

Op 26 maart 2026 organiseerde de sectie Arbeidstoxicologie in samenwerking met de Contactgroep Gezondheid en Chemie (CGC) een middagsymposium over nieuwe ontwikkelingen op het gebied van biomonitoring.



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Tijdens deze bijeenkomst kwamen achtereenvolgens een introductie over biomonitoring, recente ontwikkelingen in beleid en onderzoek, methoden en richtlijnen, praktijkvoorbeelden en een blik op biomonitoring in de toekomst en de rol van vroegsignalering aan bod. De presentaties van dit symposium zijn binnenkort te vinden op de website van de CGC: 26 maart 2026-de toekomst van biomonitoring op de werkplek

Op deze webpagina zal ook een uitgebreid verslag van de bijeenkomst beschikbaar komen.

Het volgende symposium van de CGC is op 18 juni 2026. Dit wordt georganiseerd in samenwerking met de Nederlandse Vereniging voor Arbeids- en Bedrijfsgeneeskunde en heeft als titel “Reanimatie PAGO 2.0”.

In memoriam: **Prof. dr. Wout Slob**

Wout Slob (1954-2026) was a dedicated scientist whose contributions to biology and quantitative risk assessment have left a lasting impact. He studied Biology at VU University Amsterdam, specializing in theoretical biology, and earned his PhD in 1987 with groundbreaking work applying statistics to biological research.

By Héloïse Proquin

Wout began his professional journey at RIVM in 1986, where he served as a consulting statistician until 1995. Over time, his interests evolved toward toxicology and risk assessment, fields in which he became deeply involved. From 1996 onward, he specialized in advanced methods for the risk assessment of substances, including dose-response modelling, exposure modelling, probabilistic risk analysis, and toxicokinetic modelling. His expertise was sought after by many international committees, where he played a pivotal role.

Among his notable achievements, Wout developed the PROAST software program for analysing dose-response data, which is widely used both nationally and internationally in risk assessment and scientific research.

From 2000 to 2010, Wout served as a professor of quantitative risk assessment at the Institute of Risk Assessment Sciences (IRAS) at Utrecht University, inspiring students and colleagues alike with his knowledge and passion.

Wout Slob will be remembered for his expertise in dose-response modelling, benchmark dose analysis, probabilistic risk assessment, exposure modelling, and experimental design and statistical analysis. His legacy continues through the many professionals and researchers he mentored, and through the invaluable tools and methods he developed. He could engage in heated debates and spoke his mind bluntly, but always with integrity. Wout had been retired for several years but remained active in the international field. Just a week before, some colleagues were still discussing publications and new ideas with him.

Wout's funeral took place on March 25 in a private ceremony.

He will be deeply missed by family, friends, colleagues, and the scientific community.

Some old colleagues liked to share some experience with him:

Henk van Loveren
I had the pleasure as president of the Netherlands Society of Toxicology (NVT) to nominate Wout to hold the prestigious Bo Holmstedt Lecture at the 2019 EUROTOX meeting in Helsinki and present his scientific advancements there. And he was selected. But…. Wout is Wout. And he was very hesitant, did not see the real necessity, his work was sufficiently known. What I learned is that Wout has no vanity whatsoever. In the end, he decided to go there and do the lecture. And I guess this was because of the mutual respect we felt for each other, and he did not want to let me down. That was also Wout. Well, Wout crossed his asymptote. I know that statistically that is not possible, but who would have thought Wout would die this early?

Guangchao Chen, RIVM

I started my current position at RIVM in early 2019 as a Biostatistician — Toxicology, specializing in Benchmark Dose (BMD) modelling. The main reason I was hired for this role was that Prof. dr. Wout Slob was set to retire in a few years. This gave me the opportunity to learn from him as much as possible and, together with other RIVM BMD teammates, eventually take over his tasks after his retirement. From my very first day at RIVM, Wout became my mentor. BMD modelling is a highly interdisciplinary field, spanning statistics, toxicology, and other areas. At the beginning, the challenge for me was that statistics was not my strongest suit, as my background was primarily in chemistry and toxicology. To address this, Wout had already prepared a plan for my education in statistics at the university level even before I started. Following his suggestions, I returned to study several key subjects at the VU Amsterdam.

Wout was a very honest and direct person, and he did not shy away from speaking his mind bluntly. Working with him was not always easy, especially during my junior years. There were moments when I doubted myself due to the high pressure and the complexity of the field. However, Wout's high standards and direct feedback consistently strengthened my technical understanding and expertise. Over the years, he generously shared his knowledge and extensive experience with me and other BMD teammates. Even after his retirement, he remained active in the field and continued mentoring us. Two weeks before I heard the very sad news of his unexpected passing, we met on a Friday at the office to discuss some scientific issues. Earlier that week, before he passed away, we were still communicating via email about our plans for this year. Little did I know that Friday would be the last time I saw him.

Wout was a dedicated scientist whose achievements in quantitative risk assessment continue to shape the field. He was an inspiration to students and colleagues alike, through both his expertise and his unwavering passion. To me personally, he has been a mentor who significantly shaped the path of my professional growth, and I am profoundly grateful for his guidance. His dedication, passion, and example have given me a strong sense of responsibility—a responsibility that drives me to continue on the path he pursued with such devotion. As the old saying goes, we stand on his shoulders. And we carry on.

I will deeply miss him.

The Toxic Legacy of the Great War: Heavy Metal Contamination on WWI Battlefields, a Century Later

By *Jelmer Faber*

Every spring, farmers in the Flemish fields near Ypres still turn up what locals call the “iron harvest”: shells, bullets, and fragments of a war that ended over a century ago. But alongside this visible harvest lies a silent one: lead, copper, zinc, and arsenic that seeped into the soil when millions of rounds were fired and have not left.

An Industrial War, an Industrial Contamination

The Western Front was a static, industrialised trench system stretching over 700 kilometres through Belgium and France. For four years, an estimated one billion artillery shells were fired [1]. Bullets were lead; shell casings were copper-zinc brass; percussion caps used mercury fulminate; and approximately 5% of all shells contained chemical agents, including organoarsenical compounds. When the guns fell silent, hundreds of thousands of tonnes of metallic debris had been deposited into the soil, and an estimated 200 to 300 million unexploded shells remained buried [2].

The French government defined the Zone Rouge as an area of over 1200 km^2 in northeastern France that was “completely devastated... impossible to clean. Human life impossible.” Roughly 100 km^2 remains under permanent restriction today.

What Is in the Soil, and Why Does It Stay?

Scientific studies over the past two decades have confirmed measurable enrichment of multiple heavy metals in battlefield soils. The dominant contaminants and their sources are:

- Lead (Pb) and Copper (Cu): from bullets, shrapnel, and brass cartridge cases
- Zinc (Zn): alloyed with copper in brass casings
- Antimony (Sb): hardening agent in lead-antimony bullets
- Arsenic (As): released from the thermal destruction of organoarsenical chemical weapons

Conical lead bullets. By Jacek Halicki - Own work, CC BY-SA 4.0, https://commons.wikimedia.org/w/index.php?curid=131737068

A landmark geostatistical study mapped copper concentrations across the entire province of West Flanders (3144 km^2) and found a regional enrichment of approximately 6 mg/kg Cu in topsoil around Ypres. At local hotspots, copper, lead and zinc concentrations exceeded EU soil sanitation thresholds [3]. At former ammunition burning grounds near Verdun, arsenic concentrations in the topsoil reached up to 176 mg/kg, far above any safe threshold [4], [5].

Heavy metals do not degrade. In the clay-rich, often waterlogged soils of Flanders and the Somme, lead and copper bind strongly to organic matter and iron oxides, maintaining their presence in the root zone indefinitely. Arsenic behaves more dynamically: under reducing conditions in wet soils, the more toxic and mobile arsenite form (As III) is favoured, raising leaching risk [5]. Meanwhile, each buried corroding shell continues to act as a slow-release source of metals.

Human and Ecological Exposure

The contaminated land of Flanders and northern France is among the most intensively farmed in Europe. Studies

have demonstrated uptake of lead and copper into edible plant tissues in crops grown on former battlefield soils [6]. The greatest concern is for root vegetables, leafy crops, and home gardens where tilling and direct soil contact are highest.

Children are the most vulnerable group: they ingest soil directly through play, and their developing systems are disproportionately sensitive. Chronic low-level lead exposure is associated with neurodevelopmental impairment, with no identified safe threshold [7], [8].

Arsenic is classified as a Group 1 human carcinogen by IARC, linked to cancers of the skin, bladder, lung, and kidney, as well as cardiovascular disease and adverse developmental outcomes [8], [9].

Ecological effects are also documented. Specialists observe reduced diversity of fungi, lichens, and plant species in the most affected departments, though disentangling battlefield contamination from modern agricultural and industrial pressures remains challenging.

Remediation: A Generation-Scale Challenge

Systematic remediation of WWI battlefield contamination is practically impossible at landscape scale. Contamination is spatially heterogeneous, concentrated around shell craters and burning grounds but also diffusely distributed across a vast agricultural area. Excavation is complicated by the ever-present risk of detonating buried munitions.

Technologies such as the Davinch system (Detonation of Ammunition in Vacuum Integrated Chamber) are deployed for recovered shells [2]. Phytoremediation, using hyperaccumulating plant species to extract metals from soil, is also being explored. In practice, risk management relies on monitoring, land use controls in the Zone Rouge, and soil testing in residential gardens. Remediation is measured in generations, not years.

Conclusion

Over a century after the Armistice, the toxicological legacy of the First World War is not a historical footnote. It is an ongoing exposure reality for communities, farmers, and ecosystems across northeastern France and Belgium. The heavy metals deposited in these soils are not fading; they are persisting, redistributing, and continuing to emerge from hundreds of millions of corroding shells. The Zone Rouge is the most dramatic symbol of this legacy, but the contamination extends far beyond its boundaries. For toxicologists, it is a powerful reminder that the consequences of war extend far beyond the ceasefire, sometimes for centuries.

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The Hidden Dangers of Burn Pits in the Military

By *Héloïse Proquin*

Military burn pits emerged as a waste-disposal shortcut in deployed bases lacking regular trash systems, incinerators, or landfills. They enabled rapid burning of mixed waste—plastics, food scraps, medical waste, batteries, chemicals, rubber, and base trash—to maintain hygiene and security in austere settings.

Primarily documented in US operations (e.g., Joint Base Balad), pits were shared infrastructure. NATO allies—including UK, French, Dutch, and other EU forces—in Afghanistan/Iraq (2001–2021) operated on these bases, facing exposure and likely adding waste [1,2,3,4].

Marines burning waste in burn pits in the 1st Marine Division Support Area in Saudi Arabia during the Gulf War in 1991. By Leonard J. DeFrancisci, WikiMedia

EU militaries used similar open burning historically (e.g., 1990s Balkans, early deployments), but phased it out sooner under environmental laws.

Toxic Emissions Profile

These open-air pits produced dense, complex smoke from incomplete combustion of hazardous materials like fuel, munitions, paints, and human waste, rather than clean flames. Smoke plumes travelled far, posing repeated exposure risks to personnel nearby.
Key releases included:

- Particulate matter (PM): PM2.5 and PM10 carrying toxins [2].
- Gases and VOCs: Benzene, carbon monoxide, nitrogen dioxide, volatile organic compounds (VOCs) [2].
- Carcinogens and metals: Dioxins/furans, polycyclic aromatic hydrocarbons (PAHs), hexachlorobenzene, lead, mercury [1,2,3].
- Other by-products: Heavy metals and combustion residues from plastics, fabrics, wood, and metals[1,2,3].

These mixtures irritated airways acutely and contributed to chronic effects, as lower-temperature smouldering amplified toxin formation.

Associated Health Risks

Exposure correlates with elevated risks of respiratory, cardiovascular, and other conditions. Studies link pits to toxic smoke inhalation causing breathing issues and long-term diseases like asthma, chronic obstructive pulmonary disease (COPD), chronic bronchitis, emphysema, constrictive bronchiolitis, sinonasal disease, hypertension, and ischemic stroke [1,2,3].
A key study, “Deployment to Military Bases with Open Burn Pits and Respiratory and Cardiovascular Disease,” analysed ~500,000 US Veterans from Iraq/Afghanistan wars via health data and declassified records[1]. It confirmed higher disease rates at pit-equipped bases.

Current Status and EU Transition

Burn pit use has dropped sharply post-2009 policies, though limited in remote US deployments. No global ban exists.
EU forces adhere to strict rules like the Waste Shipment Regulation, banning uncontrolled burning. The European Defence Agency pushes waste reduction, reuse, and biotech alternatives. In the Netherlands, the Ministry of Defence’s KPU and Category Management apply circular economy principles—repairing/reusing uniforms, helmets, and gear for navy, army, air force, and police—to minimize waste [4].

Burn pits solved wartime logistics but at high health costs from toxic emissions. Global shifts to sustainable disposal underscore the need for monitoring exposed veterans and enforcing greener practices in all militaries. More epidemiological research is needed—not only for military personnel but also for local populations near these pits who faced prolonged, uncontrolled exposure without protective measures or medical tracking.

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Epibatidine: **Poison or Pain Killer?**

On the 16th of February 2024, Russian opposition leader Alexei Navalny died in a Siberian penal camp. According to the Russian media, he took a short walk, felt unwell, collapsed and never regained consciousness. Two years later, a group of European countries concluded, based on analysis of biological material from his body, that Navalny was poisoned with epibatidine [1].

By *Puck Roos*

Epibatidine: a poisonous compound from frog skin

Epibatidine is an alkaloid compound that is found on the skin of poison dart frogs, which are found in Central and South America. In the 1970s, epibatidine and other alkaloid compounds were isolated from the skin of frogs in Western Ecuador. When these samples were tested in mice, they produced a Straub-tail reaction, in which the tail becomes erected across the back of the mice in an S-curved shape. This reaction is also seen upon exposure to morphine and other opioids, which sparked the interest of the researchers. On a subsequent trip, the researchers collected more samples. Epibatidine was only found on the skin of some dart frog populations, and not on the skin of frogs raised in captivity. This resulted in the hypothesis that the frogs do not produce epibatidine themselves, but that they sequester epibatidine in their skin glands from a (yet unknown) dietary source. Out of 750 frog skin extracts, the researchers isolated 60 mg of a mixture of alkaloids. The alkaloid that induced the Straub-tail reaction was isolated and later named epibatidine. Out of 750 frog skins, <500 µg of relatively pure epibatidine could be isolated. Later, in the 1990s, the structure of epibatidine was elucidated, and the compound could be synthesised chemically [2].

A non-opioid analgesic?

Despite the Straub-tail reaction that was observed in mice after exposure to epibatidine, similar to their response to morphine and other opioids, epibatidine had low affinity to the opioid receptors, around 9000-fold lower than morphine. Furthermore, the opioid receptor antagonist naloxone did not inhibit the effects of epibatidine [3]. Epibatidine turned out to be an agonist of the nicotinic acetylcholine receptor (nAChR), which transmits signals from presynaptic to postsynaptic neurons and mediates muscle contraction in skeletal muscles. *In vitro*, epibatidine induced neuronal depolarization and muscle contraction, which were blocked by nicotinic antagonists [4]. *In vivo*, epibatidine induced analgesic effects in mice in a tail flick test, which measures the animals' reaction to heat. These effects were thought to be mediated via nAChRs in the brain, because they were blocked by a nicotinic antagonist that can enter the central nervous system (CNS), but not by a nicotinic antagonist that passes poorly into the CNS. Furthermore, while epibatidine was more potent than nicotine or morphine, it had low affinity for other neuronal receptors (incl. opioid, serotonergic or dopaminergic receptors), making it a very selective nAChR agonist.

Hence, epibatidine was studied in the 1990s as a promising alternative to opioid analgesics. In fact, it was so promising that Paul Simon referred to it as “the antidote for pain” in his song *Señorita with a Necklace of Tears* [5].

Toxicology for dummies: the dose makes the poison

Unfortunately, epibatidine turned out to not be as “soothing as the rain”. While epibatidine caused analgesic effects in mice at low doses (10-30 µg/kg), it caused convulsions and death at only slightly higher doses (50-100 µg/kg) [4]. In other studies, it also caused respiratory paralysis [6]. The authors hypothesized that this narrow therapeutic window may be due to the ability of epibatidine to activate multiple subtypes of the nAChR, both in- and outside of the CNS. As a result, epibatidine is no longer being investigated as potential pharmaceutical compound. However, analogues have been developed that may be less toxic.

One of these analogues is tebanicline, which was tested up to Phase II clinical trials. In a Phase II clinical trial in patients with diabetic peripheral neuropathic pain, tebanicline significantly reduced pain in all treatment groups. However, the proportion of patients that dropped out due to adverse effects was also significantly higher in the treated groups (28% at the low dose to 66% at the high dose) compared to the placebo group (9%). These adverse effects included nausea, vomiting, dizziness, and abnormal dreams [7]. Eventually, development was stopped due to this high incidence of adverse effects. To date, no other epibatidine analogues have been successfully developed into a pharmaceutical compound.

Literature on human exposure to epibatidine itself is limited. One case report was published in 2010, which describes a lab technician who developed a rash shortly after working with epibatidine [8]. The lab technician was diluting epibatidine samples while wearing gloves and a lab gown, but without respiratory protection or protection to the eyes and face. Thirty minutes later, he developed an itchy rash on his arms, legs, and torso. He showered, but the rash persisted, after which he went to the emergency room. He had no clinical symptoms other than the rash (electrocardiograph, chest radiograph, serum electrolytes, liver biochemistry, full blood count, and immunological investigations were normal). He received prednisolone and chlorpheniramine, was discharged 24 hours after presenting at the hospital, and the rash had resolved in 1 week. The author hypothesised that the rash had been caused by epibatidine exposure via the oral or dermal route. The causal relationship, however, was only supported by the temporal relationship between the epibatidine exposure and the rash, and by the lack of an alternative explanation for the rash.

Conclusions

How epibatidine ended up in a Siberian prison, thousands of kilometres from the native habitat of the poison dart frogs, we may never know. What we do know is that the once promising pain killer proved not to be so promising after all. With its narrow therapeutic window, it is a good reminder for toxicologists that the dose indeed makes the poison. Who knows, perhaps in the future, epibatidine analogues could turn this frog poison into “a cure for all the suffering that mankind must endure”.

“Nothing but good news
There is a frog in South America
Whose venom is a cure
For all the suffering that mankind
Must endure
More powerful than morphine
And soothing as the rain
A frog in South America
Has the antidote for pain
That’s the way it’s always been
And that’s the way I like it”

- Paul Simon (2000), Señorita with a Necklace of Tears

Poison dart frog (Epipedobates tricolor). By Pauln - Own work, CC BY-SA 3.0, https://commons.wikimedia.org/w/index.php?curid=6038258

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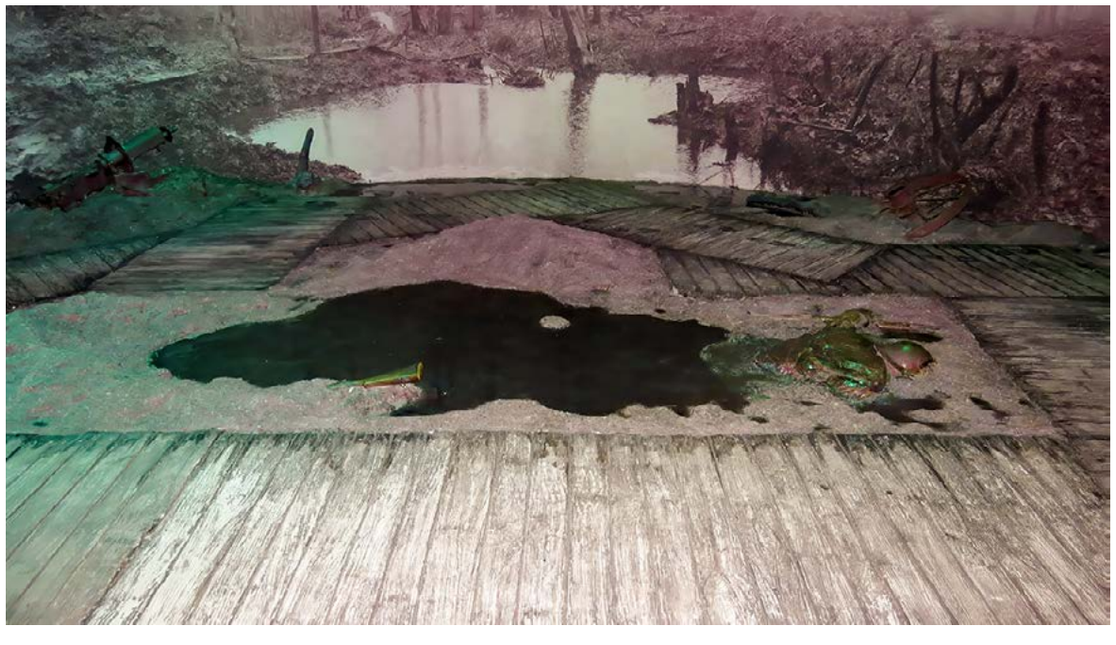
The Molecular Memory of War

By *Barae Jomaa*

It was in the late fall of 2007 when I went to the Canadian War Museum in Ottawa. It was certainly an experience to remember. The museum is large and full of tanks, planes, machine guns and other war paraphernalia. But that's not what I remember the museum for; I remember it for its very deep and engaging exhibit covering shell shock. Shell shock. It struck me how the sound of shells apparently imprints lasting psychological harm which we now know as post-traumatic stress disorder (PTSD). The very visual display at the museum tries to capture perhaps a smidgen of what was experienced by the nearly 10,000 Canadian soldiers diagnosed with shell shock during the First World War (1). Some years later, in 2015, I watched a PBS story about the children of Holocaust survivors that suffered from the psychosomatic effects of the trauma experienced by their parents. Could it be then that the effects of war trauma can be inherited through mechanisms that are still not fully understood, such as epigenetic modification?

The PBS story provided a firsthand account from the individuals affected that a scientific article cannot reproduce. Nonetheless, the story was based on research conducted by Yehuda and colleagues titled *Holocaust Exposure Induced Intergenerational Effects on FKBP5 Methylation* (2). FKBP5 encodes a co-chaperone protein that acts as a negative regulator of the glucocorticoid receptor (GR), the receptor that mediates the effects of cortisol on stress, metabolism, and the immune response. Yehuda's group measured cytosine methylation at CpG sites near the intron 7 glucocorticoid response element of *FKBP5* in 32 Holocaust survivors and 22 of their adult offspring, with matched controls. At one site (bin 3, site 6), survivors showed *higher* methylation than controls, while their offspring showed *lower* methylation, an inverse pattern that the authors attributed specifically to preconception parental trauma rather than the offspring's own life experiences (2). Methylation at this region also tracked with wake-up cortisol levels, suggesting functional relevance to HPA-axis tone (2).



The human cost of conflict: Display depicts a casualty from the Battle of Passchendaele in 1917. Canadian War Museum, Ottawa, Ontario, Canada (image source: Robert Lindsell)

An earlier study by the same group reported elevated 11β-hydroxysteroid dehydrogenase type 2 (11β-HSD-2) activity in Holocaust survivor offspring, the enzyme that inactivates cortisol to cortisone (3). This was interpreted as an intergenerational signature, particularly tied to maternal Holocaust exposure during childhood, and consistent with the broader pattern of altered glucocorticoid metabolism documented across this cohort (3, 4).

Something parallel was observed in the 1944 "Hunger Winter" in the Netherlands. Children conceived during the

A toxic mix: A military helicopter spraying Agent Orange during the Vietnam War. Huey Defoliation, National Archives: 111-CC-59948. 1963. This image is in the public domain.

famine carried an adverse metabolic profile decades later (higher BMI, dyslipidemia, impaired glucose handling, and elevated cardiovascular and schizophrenia risk) and showed differential DNA methylation at imprinted and metabolic loci, including *IGF2* (5, 6). More recent epigenetic clock analyses of the same cohort have shown a faster pace of biological aging in those exposed in utero, six decades after the event (7).

Such inherited responses are not unique to humans. In a now-classic experiment, Dias and Ressler conditioned male mice to fear acetophenone (a chemical with a sweet, cherry-blossom-like odour) by pairing it with a mild foot shock. Both their F1 offspring and F2 grandoffspring showed heightened sensitivity to the same odour without ever having encountered it, accompanied by altered methylation of the *Olfr151* odorant-receptor gene in the fathers' sperm and corresponding changes in olfactory neuroanatomy (8).

The Vietnam War's herbicide campaign provides further evidence that epigenetic effects resulting from war exposures may be passed on to the next generation. Agent Orange, dropped from helicopters to clear the dense forests and allow US forces better visibility of their targets, consisted of 2,4-D and 2,4,5-T. However, the toxicity of the herbicide to humans belongs to its contaminant: 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). With a human half-life of roughly 7–11 years and a binding affinity for the aryl hydrocarbon receptor (AhR) that drives both xenobiotic metabolism and chromatin remodelling, TCDD is uniquely positioned to leave long-lasting epigenetic imprints (9).

The most direct evidence for paternal transmission comes from the men of Operation Ranch Hand, the U.S. Air Force unit that handled and sprayed Agent Orange in Vietnam. The first scan of their sperm methylation, drawing on the Air Force Health Study, came up largely empty: when stringent statistics were applied across the whole genome, no single methylation site reliably separated men with high dioxin exposure from those with low exposure (9). A more focused look at the same data did identify altered methylation at *H19*, an imprinted gene region normally shielded from environmental influence, echoing findings in dioxin-exposed boys in Russia (9).

The picture changed when the dataset was reanalysed with a different lens. Instead of looking for sites that shifted in the same direction in every exposed man, the new approach searched for sites where exposure produced unusually erratic methylation, what the authors called "stochastic epimutations." Through this lens, 437 genes carried epimutation burdens that rose in step with the men's serum dioxin levels, and the affected genes clustered around two themes: embryonic patterning and reproduction. The top hits were *HOXA5*, *HOXA3*, *WT1*, *HOXA4*, and *ASCL2*, with hypermethylation increases of 28–43% per unit of serum dioxin (10). *HOX* genes are master regulators of how an embryo lays out its head-to-tail body plan, so finding them silenced in the sperm of veterans decades after the war provides a biologically plausible bridge between paternal exposure and the developmental concerns long voiced by veterans' families.

The most striking recent human evidence arrives from outside military medicine but speaks directly to it. Mulligan and colleagues sampled three generations of Syrian refugee families (grandmothers exposed to the 1982 Hama violence, their daughters, and their grandchildren) and identified 14 differentially methylated positions (DMPs) associated with germline exposure and 21 with direct exposure, with most

DMPs showing consistent directionality across germline, prenatal, and direct exposures, and prenatal exposure linked to epigenetic age acceleration in children (11). This is, to date, one of the clearest pieces of human evidence that war-related stress leaves an inheritable epigenetic signature.

We have all read about the atrocities of war and are reminded of them in the news as wars rage in Ukraine and the Middle East. Wars are always justified by the parties involved as a necessary outcome to combat some bad actors. What the increasing body of scientific evidence tells us is that the effects of war are not only experienced by soldiers and innocent bystanders who are unfortunate enough to be in an active warzone but also by their descendants. The longstanding legacy of war is physical, psychological and multigenerational. It is written in our cells. As scientists we may have limited direct influence on war, but our role is critical in researching the effects of war on human health and in developing targeted treatments for those affected.

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The Invisible Toxic Legacy of War

Wars rarely end when the fighting stops. For toxicologists, the formal end of conflict often marks the beginning of a different kind of problem: damaged environments, altered exposure pathways, weakened infrastructure, and risks that are harder to see as they become less visible. The public image of toxic harm in war still tends to centre on acute poisoning, chemical weapons, and other spectacular events. Those hazards matter. But they are not the whole story, and in many settings, they are not even the main one (1).

By *Devon Barnes*

What war leaves behind is a more diffuse burden. Urban destruction, damaged industrial sites, munitions residues, widespread fires, and failing water systems generate overlapping chemical and microbiological hazards. During the Gulf War oil fires, hundreds of burning oil wells released vast quantities of particulates and combustion products, illustrating the scale at which conflict can shape regional air quality (2). Similarly, post-conflict assessments following the Kosovo conflict identified industrial “hot spots,” where bombing of chemical and petrochemical facilities led to localised contamination of air, soil, and water (3). These risks do not occur in isolation. They accumulate over time.

Post-conflict environments often contain complex mixtures of airborne particulates, industrial residues, and structural debris, illustrating the diffuse and persistent nature of toxic exposure after war.

Beeld:
Sayedqudrathashimy1991, CC BY-SA 4.0 via Wikimedia Commons

War toxicology often must account for environmental contamination, infrastructure failure, occupational hazards, and chronic exposures that persist beyond active conflict. War does not simply release hazardous substances. It creates hazardous conditions. That shift expands both what counts as evidence and which populations come into view. Yet the discipline may not be well suited to these settings. Toxicology typically relies on defined substances, measurable doses, stable populations, and baseline data. War erodes each of these. Exposures are mixed and poorly documented, populations are mobile, and monitoring may be absent, delayed, or politically constrained. During the ongoing conflict in Ukraine, efforts to assess environmental exposures remain preliminary, with a comprehensive assessment only likely to follow once access improves (4). At the same time, displacement and damage to healthcare and other civilian infrastructure have complicated the broader public health response. As a result, the evidence most needed for toxicological judgment is often the least available.

That difficulty should not be mistaken for insignificance. Some of the most consequential harms are also the hardest to attribute with confidence. What is easiest to document in war is not always what matters most in the long term, and the burden is often unevenly distributed. In both

conflict and post-conflict settings, chemical exposures are often prolonged and overlapping. Children and pregnant individuals may face heightened vulnerability, responders and clean-up workers may encounter poorly characterised occupational risks, and displaced populations may return to environments that remain contaminated (5). In such contexts, exposure is not a single event but can become an ongoing part of daily life.

The question then becomes what burdens persist, and in whom. Some effects emerge only after considerable delay, when attribution becomes difficult. Respiratory, developmental, reproductive, and carcinogenic risks may all form part of this legacy. The long-term consequences of the Vietnam War, particularly the persistence of dioxin contamination following the use of Agent Orange, continue to shape discussions of delayed and transgenerational toxicological risk, as dioxin exposure can induce heritable epigenetic changes and disease across multiple generations (6). If toxicology focuses only on what can be demonstrated quickly, it will miss much of what war does in the long-term. This creates a difficult position for toxicologists. The field is rightly cautious, but in conflict settings, caution can begin to resemble silence. Waiting for ideal evidence may leave exposed populations without recognition or protection, while overstating the evidence risks undermining trust. The task is to work within that tension and define uncertainty clearly while still offering useful, evidence-based judgment. This requires a broader toxicological approach that draws on environmental, occupational, and public health perspectives and engages more closely with fields such as epidemiology, environmental forensics, and humanitarian health (7).
No single method will resolve the evidentiary problem, but multiple imperfect sources of evidence, interpreted together, may support more timely and protective decisions.

A thematic issue on war toxicology is therefore more than an opportunity to examine toxic agents used in conflict. It is an invitation to consider the wider range of exposures and consequences and ask what toxicology is for when its usual conditions of clarity have broken down. War exposes the fragility of public health systems, the vulnerability of civilian environments, and the persistence of harm after attention has moved elsewhere. It shows that the toxic damage is often slow, diffuse, and embedded in the aftermath rather than during the conflict itself and is deserving of sustained toxicological attention. Wars do not end toxicologically when ceasefires are declared, front lines shift, or headlines fade. Their legacy remains in the air, water, soil, dust, work, housing, and disease risk. To study war toxicology is therefore not simply to document incidents of poisoning, but to make visible the long afterlife of conflict in environmental, biological, and public health terms, and to recognise it as a central concern of toxicology.

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Burnt food and cancer risk highlighted by Dutch toxicologist

By *Barae Jomaa*

In an article by Luca ten Hoor for RTL Netherlands (26 March 2026), toxicologist and Wageningen University associate professor Nynke Kramer discusses the health risks of consuming burnt or heavily charred foods. During hightemperature cooking methods such as grilling, baking, and barbecuing, carcinogenic substances can form from fats, proteins, and sugars, with higher temperatures and longer cooking times leading to greater formation.

Kramer draws particular attention to polycyclic aromatic hydrocarbons (PAHs), which are produced during the incomplete combustion of organic material, especially fat. Burnt meat, especially when barbecued and exposed to smoke, can contain high PAH levels. Kramer notes that exposure from eating a heavily burnt sausage may be comparable to smoking an entire pack of cigarettes, as some of the same carcinogens are involved.

In addition, acrylamide can form in carbohydraterich foods through reactions between sugars and amino acids, contributing to the appealing brown color but posing carcinogenic concerns based on animal studies. The type of food strongly influences exposure: charred fatty meats generally contain higher levels of carcinogens than burnt vegetables.

Kramer emphasizes the toxicological principle that *"the dose makes the poison"*. Light browning results in much lower exposure than blackened food. Practical advice includes avoiding excessive temperatures and cooking times, limiting smoke exposure during barbecuing, and scraping off burnt portions. Severely burnt food, however, is best not consumed.

The article can be found here: Toxicoloog over aangebrand voedsel: 'Vergelijkbaar met roken van heel pakje sigaretten'

Pizza pepperoni. By Apalapala, CC BY-SA 3.0 <https://creativecommons.org/licenses/by-sa/3.0>, via Wikimedia Commons

Hidden asbestos in coloured sand for children: who would have thought?

The issue first drew major attention in Australia in November 20251, where asbestos was detected in coloured play sand sold for children, leading to recalls and temporary disruptions at schools and childcare centres. Starting in 2026, similar concerns then spread to the UK[2], the Netherlands[3], Belgium[4], France[5] and other European markets as testing and precautionary reviews found contaminated products or prompted warnings.

By *Héloïse Proquin*

Why asbestos got in

Asbestos fibres may end up in sand because the sand is extracted from mines where asbestos fibres are naturally present in the soil or in rocks, or due to contamination from the use of asbestos-containing equipment. It is highly unlikely that asbestos fibres are intentionally added[6].

What are the rules about asbestos

According to the REACH Regulation nr. 1907/2006, Annex XVII, point 6, the manufacture, placing on the market, and use of asbestos fibres and of articles and mixtures to which certain asbestos fibres have been intentionally added are prohibited. This applies to crocidolite, amosite, anthophyllite, actinolite, tremolite, and chrysotile[7]. These fibres are classified as Carcinogen 1A (and STOT RE Cat. 1), without a specific concentration limit. The generic classification threshold for Carcinogen Category 1A substances is 0.1% by weight[8].

There is also specific legislation in the EU regarding toys: namely the Toy Safety Directive 2009/48/EC[9]. Article 10 of the Toy Safety Directive sets out an essential safety requirement for toys:

toys and the chemical substances they contain must not, when used in accordance with their intended purpose or in a manner that can be expected given children's behaviour, pose a risk to the safety or health of users or third parties.

Health Mechanism

Asbestos toxicity is strongly influenced by fibre shape and size, with long, thin fibres (especially >8 µm in length and <0.25 µm in diameter) considered most carcinogenic because they are difficult for lung macrophages to engulf, although shorter fibres may also contribute to cancer. When inhaled, asbestos fibres reach the alveoli, where macrophages attempt to phagocytose them, releasing reactive oxygen and nitrogen species, cytokines, and growth factors that trigger inflammation and tissue damage[10]. These reactive species can damage DNA, proteins, and cell

membranes, explaining asbestos' mutagenic effects, while signalling molecules such as IL-1, TNF-α, TGF, PDGF, and IL-8 promote fibroblast proliferation and collagen deposition, leading to lung fibrosis. Other immune cells also contribute to this inflammatory process[10].

Dose-Response Risk

Epidemiological data from occupational cohorts (e.g., IARC Monograph 100C) show no safe threshold[8]; even low exposures (<0.1 f/cm³-years) elevate relative risks by 1.5–5x for lung cancer, with synergism alongside tobacco smoke. Regulators enforce stringent limits (0.01 f/cm³ TWA under EU rules) because linear no-threshold (LNT) models predict cumulative lifetime risk from trace airborne fibres in scenarios like sand play.

Conclusions about the coloured play sand

RIVM concluded in their report that "It should be noted that there is no safe exposure to asbestos. The risk to develop an asbestos related health effect depends on the level of exposure. For this reason, contamination of toy sand with asbestos fibres is undesirable and should be prevented as much as possible. We conclude that the risk of asbestos-related diseases from playing with toy sands lies at or below the negligible risk level. The asbestos exposure from most products is very low and does not pose a concern. However, there are four loose decorative sand products with notably higher levels of contamination compared to the other products. It is important to prevent further exposure to these products as in a hypothetical worst-case scenario where a child is always playing with one of these products they may lead to an unacceptably high risk."[11]

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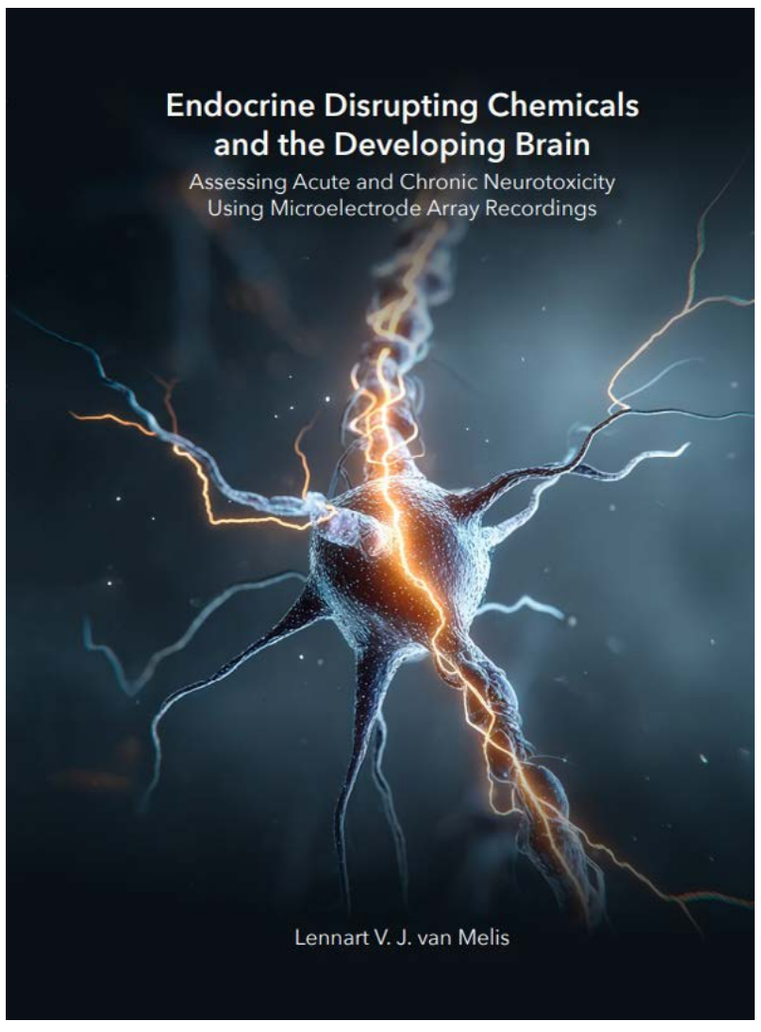
Endocrine disrupting chemicals and the developing brain -

Assessing acute and chronic neurotoxicity using microelectrode array recordings

By *Lennart van Melis*



This thesis investigates how exposure to endocrine-disrupting chemicals (EDCs) affects the development and function of neuronal networks. Using in vitro neuronal cultures and micro-electrode array technology, it demonstrates that many EDCs alter network activity under both acute and chronic exposure conditions, sometimes in different or opposing ways. The findings highlight the importance of studying long-term exposure, considering sex as a biological variable, and using network-level approaches in neurotoxicology. Overall, this work contributes to improved testing strategies for assessing chemical effects on the developing brain.

n this thesis, I investigated how exposure to endocrine-disrupting chemicals, or EDCs, affects the development and function of neuronal networks. EDCs are widespread in our environment and are increasingly associated with neurological disorders, including neurodevelopmental and neurodegenerative diseases. At the same time, the mechanisms through which these substances affect the nervous system remain insufficiently understood, particularly at the level of functional neuronal networks. Brain development is a complex process in which neurons organize into dynamic networks that form the basis for cognitive functions and behaviour. Disruption of this process may have long-lasting consequences. Within toxicology, there is therefore a growing need for methods that go beyond single-cell measurements and instead capture changes in network activity.

In my research, I used primary cortical neurons derived from male and female rat pups, which develop into spontaneously active networks *in vitro*. Using micro-electrode arrays (MEA), I measured the electrical activity of these networks. This technique allows for the analysis of parameters such as firing rate, (network) burst patterns, and synchronization, providing insight into how neuronal networks function and develop.
A central aim of this thesis was to systematically investigate the effects of different classes of EDCs, including insecticides, bisphenols, PFAS, phthalates, and flame retardants. Both acute and chronic exposure paradigms were included, allowing for the identification of immediate as well as developmental effects.

The results show that most of the tested substances affect neuronal activity and network development. These effects

were observed under both acute and prolonged exposure conditions, but differed in nature and direction. In some cases, acute exposure increased activity, while chronic exposure decreased it, or vice versa. This highlights the importance of including multiple exposure scenarios in toxicological assessments. In addition, I investigated the role of sex differences in sensitivity to these substances. While differences were observed between neuronal cultures of male and female origin, these effects were not consistent across all compounds and experimental conditions. This suggests that sex is a relevant but complex factor that requires further attention in neurotoxicology.

An important finding of this thesis is that the observed effects are likely largely driven by direct interactions of the EDCs with neurons, rather than mediated through endocrine mechanisms. This may be related to the model system used, which focuses on differentiated neurons. Hormonal influences are likely more prominent at earlier stages of development, which are less well represented in this model. Furthermore, the results indicate that some substances exert effects at concentrations that are close to, or even below, levels encountered in daily human exposure. This emphasizes the relevance of these findings for risk assessment and public health, and highlights the importance of sensitive and biologically relevant testing methods.

This thesis contributes to the further development of *in vitro* approaches in neurotoxicology. By using MEA technology, neuronal networks can be studied at an integrated functional level, providing additional information beyond traditional endpoints. This approach aligns with the broader shift towards animal-free and mechanism-based testing strategies. In addition, the findings underscore the importance of combining different models and approaches. No single model system can fully capture the complexity of brain development and function. Integrating data from multiple experimental systems, therefore, remains essential for robust risk assessment.

My interest in this field originated from a broad fascination with brain function, which began during my bachelor's in psychology and further developed through my bachelor's in biology and a research master in cognitive neuroscience. This interdisciplinary background informed my approach to studying neuronal networks from both a functional and toxicological perspective. The PhD project, conducted at the Institute for Risk Assessment Sciences (IRAS) at Utrecht University within the European ENDpoiNTs project, provided not only in-depth scientific knowledge but also essential skills in experimental design, data analysis, and scientific communication. It also taught me to evaluate the strengths and limitations of different model systems critically and to place findings within a broader scientific context.

In conclusion, my research demonstrates that EDCs can significantly affect neuronal network activity and development, with effects depending on exposure duration, concentration, and, to some extent, sex. At the same time, the findings highlight the need to integrate multiple approaches to understand the complexity of neurotoxicity better. With this work, I hope to have contributed to a better understanding of how chemical exposures affect the nervous system and to the development of more predictive and relevant testing strategies in toxicology.

AIO toxafette - Imke Bruns

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 passes the baton to a fellow PhD-student. This time Imke Bruns, from Leiden University tells us about her project.

Can you introduce yourself?
My name is Imke, and I am currently in the final year of my PhD at the Leiden Academic Centre for Drug Research (LACDR). My research focuses on evaluating the human relevance of renal test systems. Outside of my PhD, I enjoy staying active and creative: I play volleyball, love reading and cooking, and spend as much time as possible in nature; hiking or photography.

How would you explain the subject of your research to a layperson?
Before new chemicals can be introduced to the market, they are typically tested in animals. This process is costly, time-consuming, and raises ethical concerns. As a result, there is a growing effort to develop alternative testing methods that do not rely on animal experiments. One promising approach is to use human-derived cells (e.g. kidney cells grown in the laboratory) and study how they respond to chemical exposure. However, before such models can replace animal testing, we need to ensure that they accurately reflect what happens in the human body. In my research, we measure changes in gene expression within these cells, capturing information from around 20,000 genes at once. Because this is highly complex data, we group genes into “modules” based on similar expression patterns. As these modules show similar expression patterns, we assume they are involved in similar biological processes and can therefore annotate them with specific functions (e.g. oxidative stress, DNA damage, mitochondrial stress, etc.). We then compare these modules to those derived from real human kidney biopsy samples. This allows us to assess how closely our cell models mimic actual human biology. If the similarity is high, it increases our confidence that these systems can be used to predict human responses.

How is your research related to the field of toxicology, and why did you choose this subject?
Before starting my PhD, I performed all my internships in cancer research, focusing on topics such as resistance to PD-L1 inhibition therapies and gene expression in individuals with a high polygenic risk for breast cancer. However, I have always had a strong affinity for animals (I even wanted to become a veterinarian). Toxicology turned out to be the perfect intersection of my interests: it allows me to work with human clinical data while contributing to the development of alternatives to animal testing. When I came across this project, I was immediately enthusiastic!

What was your motivation for starting a Ph.D. program?
I have always loved research. I find it inspiring to contribute (even in a small way) to expanding the boundaries of knowledge and working towards a better understanding of the human biology. What particularly appealed to me about a PhD was the opportunity to work on a project over a longer period of time. During my master’s research project, I found it difficult to stop after only nine months because there was so much more to find out, which made me eager to pursue a PhD.

How do you see the future of your research topic (follow-up research / social impact)? What do you hope for?
I hope that my research contributes to building sufficient confidence in alternative test methods so that we can gradually reduce (and hopefully eventually replace) animal testing. Existing regulations, such as the ban on animal testing for cosmetics in Europe, show that meaningful change is possible when there is enough motivation. Rather than replacing animal testing all at once, I believe in a stepwise transition. For example, these new models could first be used to identify early biological responses that may later lead to toxicity. If we can demonstrate that these systems reliably reflect human biology, they can help us screen out harmful chemicals earlier in the process.

What are your thoughts on using new technologies like artificial intelligence in toxicology research? Are you using any of these technologies in your work?
I am very enthusiastic about the use of artificial intelligence in toxicology. This field often revolves around understanding biological pathways and cause-and-effect relationships; areas where AI can be particularly powerful. For example, large language models (LLMs) are designed to predict what comes next based on patterns in data. In a similar way, AI could help predict which genes or biological processes are involved in certain toxic responses. I believe this has great potential for improving and expanding adverse outcome pathways (AOPs). As a side project, I am currently exploring the application of language model-inspired approaches to human kidney single cell data. My goal is to identify key genes involved in processes such as the transition from healthy to damaged kidney cells. While this work is still in progress, I am excited to explore how these methods could be integrated into toxicology research.

How do you ensure that others can replicate your experiments and achieve the same results?
As a computational biologist, most of my work is performed in R (and sometimes Python), which allows for a high level of reproducibility. I use Git for version control and track changes and ensure that all steps in my analysis are transparent. In my code, I aim to clearly document not only what I did, but also why certain choices were made, so others can easily follow my reasoning. In addition, I work with virtual environments to fix package versions. Since packages are frequently updated and may change in functionality or compatibility, this ensures that anyone attempting to reproduce my work uses the exact same computational setup and obtains consistent results.

How do you combine your PhD project with your personal life? Are there choices you have to make?
I believe maintaining a good work-life balance is very important, although it can be challenging during a PhD. One of the difficulties is that a PhD project is never truly “finished”. There is always more that could be done, so it is important to set clear boundaries for yourself. I generally aim to work between 9.00 and 18.00. During busier periods, I sometimes work longer hours, but I try to keep this temporary. What helps me is having fixed activities outside of work: I have volleyball practice twice a week, which motivates me to leave on time. In the weekends, I make sure to plan fun activities with friends, which helps me recharge and start the new week with fresh energy and motivation.

What is the best advice that you have received as a PhD student or would like to give to another PhD student?
My advice would be closely related to the question above (maintaining a good work-life balance): be mindful of your time and allow yourself regular breaks. It may feel like there is always more work to do, but working continuously does not necessarily make you more productive or creative. Make time for activities you enjoy, whether that is spending time with friends, going away for the weekend, reading, or just relaxing. While it might seem unrelated to your PhD, these moments are essential for staying motivated and productive in the long run.
Also, somewhere halfway through your PhD, it may feel overwhelming when you realize how much still needs to be done in a limited amount of time. My advice is to take it step by step. In my experience, things start to come together towards the end, even if it does not always feel that way earlier on. And finally: enjoy the process. A PhD offers a lot of freedom and the opportunity to work at the forefront of research, so make the most of it! J

What goals do you have regarding your career after finalization of your PhD? Would this be inside or outside of academia, and why? Would you consider going abroad?
I currently expect that my future career will be outside of academia. However, I really enjoy my current working environment and the opportunity to be involved in research, so I would like to stay in a research-oriented role, for example as a bioinformatician or computational biologist. I am also very open to going abroad. I see the period after my PhD as a great opportunity to gain international experience, and I would really enjoy living in another country for some time. Due to COVID-19, I could not do an internship abroad, so this is something I would still very much like to explore. My PhD contract ends at the end of this year, so I am excited to see what opportunities the future will bring.

Please answer the question from the last toxafette PhD-candidate: How can we better bridge the gap between toxicology and fields like physics and engineering to tackle public health problems?
I think that bridging toxicology with fields such as physics and engineering is essential to tackle complex public

health challenges. These disciplines bring complementary expertise; engineering enables the development of advanced in vitro models like organ-on-a-chip systems, while physics contributes to imaging technologies and modelling approaches. To strengthen this connection, I think it is important to encourage interdisciplinary collaborations, both in research projects and in education. Ultimately, combining mechanistic biological insights with technological innovation will allow us to develop more predictive and human-relevant models for assessing chemical safety.

IUTOXtalks: a FREE 4-part webinar series, **Register Today!** IUTOXtalks Returns!

IUTOX is excited to present the 2026 programme of IUTOXtalks, a 4-part webinar series.

IUTOX organises a session each year at the SOT Annual Meeting under the heading **Global Collaboration Coffee**, a panel discussion session addressing a topical issue or concern in toxicology. The 2026 topic of **“Persistent and Pervasive: Tackling PFAS Through Science and Policy—A Global Perspective”** will continue with a series of 4 webinars, free for all participants, taking this important topic to a global audience.

IUTOX*talks* will run in April, June, September, and November 2026 and each talk will have 2 speakers from the Global Coffee Collaboration presenting their view (20 minutes each) followed by discussion and a Q&A session with the audience.

Read more

47th annual meeting of the Dutch Society of Toxicology (NVT)

We are pleased to invite you to the 47th annual meeting of the Dutch Society of Toxicology (NVT), which will take place on June 23 and 24 at De Eenhoorn in Amersfoort.

We would like to share several important updates and deadlines:

Abstract submission
The abstract submission deadline has been extended by two weeks!

- Abstracts can be submitted until **April 30**

Registration
Registration for the meeting is open! Early bird registration is available until May 22. We recommend registering before this date to secure your place, particularly for the workshops, as availability cannot be guaranteed afterwards. More information on workshops, keynote speakers and sessions are currently in preparation and will be updated on our website!

Accommodation
If you require accommodation, we strongly recommend booking your hotel in advance. Rooms are available at a rate of €125 per night for a single room, including breakfast. Please note that any unbooked room allocations will be released on May 22. More information can be found **here**.

For further updates, please visit our **website**.

If you have any questions, please feel free to contact us at nvtmeeting@gmail.com.

We look forward to welcoming you in Amersfoort!

The organizing committee of 2026:
Sanah Majid Shaikh, Kirsten Lassing, Emmely Wagener, Can Jiang, Judith de Heer, Tim Verbruggen, Thijs Benschop, Bensu Tangil, Sara van Kaam, Milou Hendriks, Lucy Sinke Laura Hondebrink, Joanne Salverda, Daan Touw, Hans Bouwmeester

P.S. Updates about the Annual Meeting will also be shared via LinkedIn. Follow our page to stay informed: https://www.linkedin.com/in/nvt-annual-meeting-78b52329a/

NVT Business Meeting 2026

The business meeting will not take place during the annual meeting this year, but will be held in a separate hybrid session so that everyone can focus on viewing posters and networking during the annual meeting. The business meeting will be held on Thursday 4 June from 15:30 to 17:00. Approximately 30 members may attend the meeting in person at the RIVM in Bilthoven. Places will be allocated on a first-come, first-served basis; once full, no further places will be available. The remaining members may join online via Teams. The link will be sent with the meeting documents two weeks before the meeting.

To join, please contact secretaris@toxicologie.nl

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