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Sébastien Gorecki [a], Fabrice Nesslany [b], Daniel Hubé [c], Jean-Ulrich Mullot [d], Paule Vasseur [e], Eric Marchioni [f], Valérie Camel [g], Laurent Noël [h], Bruno Le Bizec [i], Thierry Guérin [a], Cyril Feidt [j], Xavier Archer [k], Aurélie Mahe [a], Gilles Rivière [a].

**a : French Agency for Food, Environmental and Occupational Health & Safety, 14 rue Pierre & Marie Curie, F-94700 Maisons-Alfort, France**
**b : Laboratoire de Toxicologie Génétique, Institut Pasteur de Lille, Lille, France**
**c : BRGM, French Geological Survey, 6 avenue Claude Guillemin, 45000, Orléans, France**
**d : Navy Expert Lab of Toulon, BCRM Toulon, BN/LASEM, 83800 Toulon, Cedex 9, France**
**e : CNRS UMR 7360, University of Lorraine, Metz, France**
**f : Université de Strasbourg, CNRS, UMR 7178, F67037 Strasbourg, France**
**g : UMR Ingénierie Procédés Aliments, AgroParisTech, Inra, Université Paris-Saclay, 91300 Massy, France**
**h : The French Directorate General for Food, Ministry of Agriculture, Agro-16 Food and Forestry, Paris, France**
**i : ONIRIS - LABERCA, Atlanpole – La Chantrerie, BP 40706, Nantes F-44307, France**
**j : Université de Lorraine, INRA, 2 avenue de la forêt de Haye, TSA 40602, 54518 Vandoeuvre Cedex, France**
**k : Central Laboratory of Police Prefecture (LCPP), 39 bis rue de Dantzig, 75015, Paris, France**

*Published in Science of the Total Environment. Available online: http://dx.doi.org/10.1016/j.scitotenv.2017.04.213*

**Keywords**

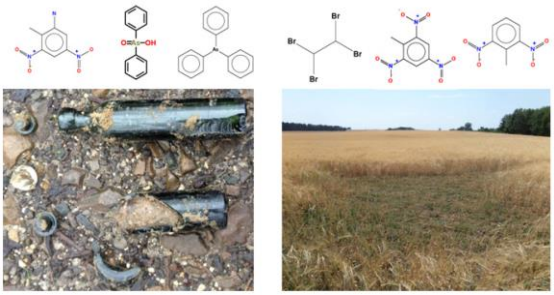
Chemical munitions, explosives, polluted site, food contamination, health risk assessment.

**Abstract**

Shells fired during World War I exhibited different explosive compounds and some of these weapons also contained a wide variety of chemical warfare agents. At the end of the war, for safety purposes, the large quantity of weapons remaining on the former front needed to be dismantled and destroyed. A large amount of the remaining shells was destroyed in specific sites which led to the contamination of the surroundings in Belgium and France.

In the 1920s, 1.5 million chemical shells and 30,000 explosive shells were destroyed in a place close to the city of Verdun, in the East of France. In this paper, the risk for human health related to the consumption of foodstuffs produced on this site was assessed. To this end, food products of plant and animal origin were sampled in 2015–2016 and contaminant analyses were conducted. Human exposure was assessed using a specifically built methodology. The contaminants considered in this study were trace elements (TEs -primarily Zn, As, Pb and Cd), nitroaromatic explosives (trinitrotoluene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, 2-amino-4,6-dinitroluene and 4-amino-2,6-dinitrotoluene), phenylarsenic compounds including diphenylarsinic acid and triphenylarsine, perchlorate, tetrabromoethane and vinyl bromide. Depending on the compound, different approaches were used to assess the risk for both adults and children. Exposure to these contaminants through the consumption

of foodstuffs produced locally on the considered site was unlikely to be a health concern. However, as for inorganic arsenic, given the presence of highly contaminated zones, it was suggested that cereals should not be grown on certain plots.



## Introduction

During the First World War (WWI), it is estimated that over 1 billion shells and projectiles were fired in Europe by the belligerents on the battlefield. The projectiles contained different high explosives and some of them were loaded with a wide variety of chemicals (toxic warfare agents, incendiary agents). As far as explosives are concerned, >400,000 tons of nitroaromatic compounds were manufactured by Germany, the most important being 2,4,6-trinitrotoluene (TNT), along with dinitrobenzene (DNB) and dinitrotoluene (DNT) isomers (Bausinger et al., 2007). Different chemical warfare agents were also massively produced and used on a large-scale on the battlefield for their lethal or incapacitating properties. Among the most commonly used chemicals in ammunitions were arsenical compounds (emetic agents), sulfur mustard (also called yperite, a blister agent), phosgene (a choking agent) and hydrogen cyanide (a blood agent) (Pitschmann, 2014). Additionally, about 11,000 tons of arsenical warfare agents were produced in Germany and France during WWI. Due to their vomiting properties, diphenylchloroarsine (“Clark 1”), diphenyloarsinous cyanide (“Clark 2”), phenylarsine dichloride (“Pfiffikus”) and dichloroethylarsine (“Dick”) were some of the most commonly used arsenicals, only used by the German armies for the loading of “Blue Cross” or “Green Cross 2,3” shells which appeared late in Great War (Bausinger and Preuss, 2005). Half of the poison-gas shells fired on the battlefield by Germans during the last year of WWI were Blue Cross shells. The French did not use organo-arsenical compounds to load chemical shells. However, arsenic trichloride ($AsCl_3$, “Marsite” for the French) just like tin tetrachloride ($SnCl_4$) (“Opacite” for the French) were mixed with suffocating agent as smoke-producing compounds in French chemical projectiles of the Great War (Belot, 1997 – unpublished).

At the end of the war, the large quantity of fired and unfired munitions remaining on the former front and innumerous ammunition dump sites in the former backlines needed to be broken down. Hundreds of thousand tons of these weapons were dumped in the Northern European seas after WWI, in shallow waters (Missiaen et al., 2010). Recent research shows that >1.7 billion tons of ammunitions remained at the Armistice in dumps as surpluses and coming from salvaging conducted by the allies (Hubé, 2016). In 1920,1.1 million tons had to be broken down for safety purposes and to recover scrap, metals, and some chemical constituting the shells. A large amount of these remaining shells was destroyed in specific sites what led to the contamination of the surroundings in Belgium and in France (Bausinger et al., 2007; Bausinger and Preuss, 2005). Bausinger and Preuss (2005) showed that the soil nearby a former ammunition destruction facility in Belgium was polluted by arsenic, copper, lead, nitroaromatic explosive compounds and their derivatives (ni-trobenzenes, nitrotoluenes), as well as triphenylarsine (a pyrolysis byproduct of organoarsenical agents).

These studies enabled to shed a light on the impact of ammunition destruction activities on soil contamination in Europe. In 2015, the French Bureau of Geological and Mining Research (BRGM) undertook an environmental assessment of a well-known former site used for the destruction of WWI chemical shells, called “La place à gaz” (BRGM, 2015) and previously studied by Bausinger and Preuss (2005). This site is a small zone located in the North East of France, close to the city of Verdun, in the Spincourt forest. “La place à gaz” was operated during 1926–1928 and over 200,000 German arsenical containing weapons were burnt there. However, historical research recently conducted by the BRGM revealed that this zone was part of a much larger-scale site, called the “Muzeray-Spincourt-Vaudoncourt” complex (also referred to as the “Clere & Schwander Meuse” complex). This site was operated from 1919 to 1925 by the historic French ministry of war ammunitions. One and a half million chemical shells and 300,000 explosive shells have been gathered there by the armies for destruction, making it the largest chemical ammunitions breaking down facility known on the former western front.

The “Clere & Schwander Meuse” site now covers >100 ha of farmlands and grasslands. The agricultural productions implanted in this zone are cereals for human consumption (soft winter wheat, winter and spring barley) and maize silage for the feeding of the dairy cow herds of two local farms. The analysis undertaken by the BRGM in the “Clere & Schwander Meuse” area revealed the presence in agricultural soils of nitrates, zinc, arsenic, organo-arsenical agents, tetrabromoethane (a solvent used for the manufacturing of arsenic warfare agents) and locally high levels of nitroaromatic compounds (BRGM, 2015).

Consequently, the French Agency for food, environmental and occupational health & safety (Anses) was asked to determine whether the consumption of plant and animal products originating from this area was safe for consumers. In this paper a specific methodology to assess the health risks related to the consumption of foodstuffs produced on areas with a battlefield history is proposed, and the results obtained in the particular case of the “Clere & Schwander Meuse” site are presented. To this end, food products of plant and animal origin were sampled in 2015–2016 and concentrations of organic and inorganic chemical contaminants were measured using developed specific analytical methods. Human exposure was assessed based on local individual consumption or/and national diet study. Risk was assessed for adults and children using health-based guidance values (HBGVs), margins of exposure or the toxicological concern concept in the case limited toxicological information were available.

## Material and Methods

### Selected contaminants

Based on the preliminary soil analysis undertaken by the BRGM the following substances were measured as a priority in different foodstuffs (see Section 2.3 for details):

Trace elements (TEs); primarily Zn, As, Pb and Cd; from shells, fuses and loading,

Nitroaromatic explosives: primarily trinitrotoluene (TNT, 2,4,6-trinitrotoluene), TNT impurities (2,4-dinitrotoluene (2,4-DNT) and 2,6-dinitrotoluene (2,6-DNT)), as well as 2-amino-4,6-dinitroluene (2-ADNT) and 4-amino-2,6-dinitrotoluene (4-ADNT), which are the primary metabolites of TNT in plants;

Primarily phenylarsenic compounds including diphenylarsinic acid (DPAA -oxidation compound of phenylarsine) and triphenylarsine (TPA -pyrolysis transformation compound of phenylarsine);

Perchlorate ion (high explosive used in some French artillery mines);

Tetrabromoethane (TBE), a solvent that was used to produce arsines as warfare agents

Vinyl bromide: given that this substance is a metabolite of tetrabromoethane in soil and that this substance has been classified as “probably carcinogenic to humans” (group 2A) according to the International Agency for Research on Cancer (IARC), it was deemed necessary to include this compound in the assessment.

### Hazard characterization

For TEs, health-based guidance values (HBGVs) selected in the context of the second French total diet study (TDS2) were used (Anses, 2011a). For the other contaminants, an in-depth literature search was carried out. For certain contaminants (e.g. nitroaromatic explosives), HBGVs were available, and the most robust were selected. For the other substances, since no HBGVs have been established to date, toxicological benchmarks have been proposed based on the available data. It should however be noted that these toxicological benchmarks were specifically determined for this study and are not intended to be used for another health risk assessment without an exhaustive analysis of the literature.

For diphenylarsinic acid and triphenylarsine, in order to allow the Threshold of Toxicity Concern (TTC) approach, the Cramer classification was determined with the Organization for Economic Co-operation and Development (OECD) Quantitative Structure-Activity Relationship (QSAR) Toolbox and Toxtree (version 2.5.0) software programs.

### Contamination data

#### 2.3.1. Sampling protocol

Samples of maize silage, barley and wheat were collected for analysis from seven growing plots located on the “Clere & Schwander Meuse” site. Regarding straw cereals (barley and wheat), in order to ensure representative samples for each plot, ten sampling points were randomly selected in the field using a “W” design (Belp, 1986). These ten sub-samples were pooled into equal shares to make composite samples before the analysis. Additional samples were taken in highly contaminated areas (referred to as “hot spots”) determined by the BRGM based on the preliminary soil analyses.

As for foodstuffs of animal origin, two dairy cattle farms were considered in this study because their animals were fed with maize silage grown on the “Clere & Schwander Meuse” site. Therefore, 32 animals including 16 dairy cows, one heifer, three young bulls and 12 calves from these two farms were slaughtered. For each of these animals, muscle, liver and kidney tissues were sampled for analyses. Composite samples of raw milk were constituted for analysis. All the samples were shipped in frozen state to the laboratories in charge of the analyses.

#### 2.3.2. Analytical methods

Arsenic and zinc in foodstuffs of plant origin were analyzed by inductively coupled plasma mass spectrometry (ICP-MS) after mineralization undertaken according to the NF EN 14084 standard (AFNOR, 2003a). Lead and cadmium in cereals were

analyzed by atomic absorption spectroscopy according to the NF EN 14082 standard (AFNOR, 2003b). For TEs in foodstuffs of animal origin (milk, muscle, liver and kidneys) were analyzed by ICP-MS according to the method validated by Millour et al. (2011). Details of the method are described in the supplementary material. The perchlorate analyses were carried out by liquid chromatography coupled to tandem mass spectrometry based on a previously reported method (FDA, 2005).

For the other compounds, specific analytical methods were developed and validated by the participating laboratories as summarized in Table 1 along with the reported analytical performances. Details of these methods are described in the supplementary material.

### 2.3.3. Contamination data

For each studied foodstuff/contaminant pair, as part of a "worst-case" scenario, the maximum contamination value obtained in this study was retained for risk assessment (Table 2).

Results below the limit of detection (LoD) or quantification (LoQ) were processed using the "substitution method" recommended by the World Health Organization (WHO) (WHO, 2013). It consists of defining the lower bound (LB) and upper bound (UB) for a measured value. The LB was calculated by considering that all values below the LoD were equal to zero and those between the LoD and LoQ were equal to the LoD. The UB was calculated by considering that all values below the LoD were equal to the LoD and those between the LoD and LoQ were equal to the LoQ.

### 2.3.4. Consumption data

The consumption data used for these two populations were taken from the second French consumption study (INCA2) (Anses, 2009). Exposure levels were estimated for children and adolescents between 3 and 17 years old and adults over the age of 17 years. In order to take into account local consumption habits, INCA2 respondents surveyed in the vicinity of Meuse, i.e. 136 individuals (77 adults and 59 children), were selected.

## [redacted]Exposure calculations

Based on the individual consumption data and contamination data, exposure was calculated using the following equation:

$$E_i = \sum_{k=1}^{n} \frac{C_{i,k} \times L_k}{BW_i}$$

where:

Ei is the total daily exposure of an individual i (µg kg body weight$^{-1}$ day$^{-1}$),

Ci,k is the daily consumption of the food k by an individual i (g day$^{-1}$),

Lk is the estimated level for the studied contaminant in the food k (mg kg$^{-1}$ fresh food),

BWi is the body weight of the individual i (kg),

n is the total number of foods consumed by the individual i.

Mean exposure levels for the population were calculated in addition to exposure levels for the most exposed individuals (95th percentile).

## [redacted]Scenarios considered for exposure calculations

Given that the foodstuffs produced on the "Clere & Schwander Meuse" site are intended to be distributed on a national scale, the exclusive consumption of locally produced both foodstuffs of animal origin and cereals is highly unlikely. Consequently, two protective and more realistic scenarios were considered for exposure calculations. On one hand, the exposure through the consumption of foodstuffs of animal origin only was considered (scenario A) and, on the other hand, the exposure through the consumption of wheat only was considered (scenario B). The Health Risk Assessment (HRA) was undertaken according to the procedure presented in Fig. 1.

Kidneys were not taken into account for the calculation of exposure since the 136 local individuals considered in this study declared not eating kidneys. Similarly, for maize silage and barley, several data were lacking to quantitatively assess the risk related to the consumption of these foodstuffs (spatial variability of contaminant levels in soil, soil- plant, plant-animal and soil-animal transfer rates). Consequently, these foodstuffs were not taken into account.

Scenario A: Exposure via the consumption of foodstuffs of animal origin produced on the "Clere & Schwander Meuse" site

For milk, meat, liver and wheat, individual consumption data from INCA2 were taken into account.

### 2.5.1. Trace elements (TEs)

It was assumed that the contaminants contained in the raw foodstuffs are fully transferred to finished products (with no dilution or concentration effect).

Total dietary exposure was calculated only for substances for which mean contamination levels measured in this study were higher than those previously reported in the framework of the TDS2 (it was indeed considered that for substances for which the contamination levels were below those of the TDS2, consumption of the foodstuffs produced on the "Clere & Schwander Meuse" site will not lead to overexposure compared to the general population studied in the TDS2). In this case, total dietary exposure was calculated by combining the contamination data for the total diet (excluding meat, liver and milk) from TDS2 with those measured in the present study (meat, liver and milk). When the calculated exposure levels (mean and 95th percentile) were higher than those of TDS2, a specific HRA was undertaken.

### 2.5.2. Nitroaromatic explosives, perchlorate ions, brominated compounds and arsines

For substances not considered in the TDS2 study (nitroaromatic explosives, perchlorate ions, brominated compounds and arsines), in order to take into account exposure related to the consumption of dairy products that could be produced from raw milk harvested on the “Clere & Schwander” site, contamination levels for dairy products like ultra-fresh dairy, butter and cheese were estimated using a table of dairy equivalents available on the website of the French National Federation of Dairy Cooperatives (Table 3).

Scenario B: Assessment of exposure via the consumption of wheat produced on the “Clere & Schwander Meuse” site

For wheat, which is integrated in the form of flour in many food-stuffs, the total daily consumption of this foodstuff was estimated for the French population using available information in the INCA2 database (flour content of foodstuffs and daily consumption of these foodstuffs). The total consumption of wheat was 125 g $day^{-1}$ on average and 220 g $day^{-1}$ at the 95th percentile for adults and 80 g $day^{-1}$ on average and 190 g $day^{-1}$ at the 95th percentile for children. Exposure levels were estimated by considering only the consumption of foodstuffs including wheat flour, taking into account mean wheat consumption of the INCA2 French population.

## [redacted]Health risk assessment (HRA)

For substances with threshold dose effects, HRA was performed either by calculating a margin of exposure (MOE) (US-EPA, 2012) or a hazard quotient (HQ). HQ was calculated for substances with an available health-based guidance value (HGBV). For 2,6-DNT and tetrabromoethane, MOEs were calculated based on their no observed adverse effect level (NOAEL). For carcinogenic substances with no-threshold dose effects (TNT, 2,4-DNT, 2,6-DNT and vinyl bromide), HRA was performed by calculating an individual excess cancer risk (IECR) (US-EPA, 2005) based on their respective slope factors and considering a lifetime of exposure (70 years).

# [redacted]Results

## [redacted]Hazard characterization

### 3.1.1. Trace elements

For TEs, health-based guidance values selected in the context of the TDS2 were used (Anses, 2011a).

### 3.1.2. .2,4,6-trinitrotoluene

TNT is currently classified by IARC as belonging to Group 3, “not classifiable as toits carcinogenicity to humans” (IARC, 1996). Available toxicity studies indicate that chronic oral exposure to TNT can lead to effects on the liver, kidneys and blood (ATSDR, 1995).

The United States Environmental Protection Agency (US EPA) set a Reference Dose at 0.5 Eıg kg $bw^{-1}$ $day^{-1}$ based on a study on dogs exposed for 26 weeks, considering hepatotoxic effects as the critical effect (US-EPA, 1988). Regarding non-threshold dose effects, the US EPA also established a slope factor of 0.03 (mg kg $bw^{-1}$ $day^{-1})^{-1}$ based on a two-year carcinogenicity study in rats, using combined urinary tract tumors as the critical effect (US-EPA, 1988). The risk related to dietary exposure to TNT was assessed using these two guidelines to take into account threshold dose effects on the one hand and non-threshold dose effects on the other hand.

### 3.1.3. 2,4-Dinitrotoluene

Toxicity studies indicate that chronic oral exposure to 2,4-DNT can lead to adverse effects on the liver, kidneys and blood. Carcinogenicity studies in rats revealed that 2,4-DNT induces tumors in the renal tubules, hepatocellular carcinomas, adenomas of the mammary gland, and fibromas and fibrosarcomas of the skin. Consequently, IARC classified 2,4-DNT as belonging to group 2B, “possibly carcinogenic” to humans (IARC, 1996).

The US EPA set a minimum risk level of 2 Eıg kg $bw^{-1}$ $day^{-1}$ based on a two-year study on oral exposure in dogs, considering hematological effects as the critical effect (US-EPA, 1992). Regarding the non-threshold dose effects of 2,4-DNT, a slope factor of 0.31 (mg kg $bw^{-1}$ $day^{-1})^{-1}$ was established by the Office of Environmental Health Hazard Assessment (OEHHA) based on the incidence of tumors of the liver and mammary gland (OEHHA, 2005). The risk related to dietary exposure to 2,4-DNT was assessed based on these two values to take into account non-threshold dose effects on the one hand and threshold dose effects on the other hand.

### 3.1.4. 2,6-Dinitrotoluene

2,6-DNT is classified by the IARC as belonging to group 2B (IARC, 1996). Only one carcinogenesis study is reported by ATSDR for 2,6-DNT that indicates that 2,6-DNT can cause hepatocellular carcinomas in rats exposed to oral intake for 52 weeks (ATSDR, 2013).

As part of the Superfund Program, the US EPA set a provisional Toxicological Reference Value (TRV) of 0.3 Eıg kg $bw^{-1}$ $day^{-1}$ for 2,6-DNT, based on a lowest observed adverse effect level (LOAEL) of 4 mg kg $bw^{-1}$ $day^{-1}$ (US-EPA, 2013). Regarding non-threshold dose effects, the US EPA set a provisional slope factor of 1.5 (mg kg $bw^{-1}$ $day^{-1})^{-1}$ based on a one-year study on oral exposure in rats, using the onset of hepatocellular carcinomas as the critical effect (US-EPA, 2013).

Given the provisional nature of the TRV of 0.3 Eıg kg $bw^{-1}$ $day^{-1}$and the uncertainties regarding the toxicity of 2,6-DNT, as reflected in the choice of a safety factor of 10,000 to derive this TRV (lack of toxicity studies on reproduction and development, few chronic studies), it was deemed preferable to assess the risk related to dietary exposure to 2,6-DNT by calculating a margin of exposure (MOE) based on the LOAEL of 4 mg kg $bw^{-1}$ $day^{-1}$. The risk related to dietary exposure to 2,6-DNT was also estimated based on the provisional slope factor to take into account possible non-threshold effects.

### 3.1.5. 2-amino-4,6-dinitrotoluene and 4-amino-2,6-dinitrotoluene

Since there were no available specific toxicological data for 2-ADNT or 4-ADNT, the reference value set for 2,4-DNT (2 Eɪg kg $bw^{-1}$ $day^{-1}$) (US-EPA, 1992) was found suitable to assess the risk related to dietary exposure to 2-ADNT and 4-ADNT due to molecular similarity. Given that 2-ADNT and 4-ADNT have the same toxicological benchmark and are the most commonly measured TNT metabolites in plants (Burken et al., 2000; Vanek et al., 2006), the risk was assessed for the sum of these two compounds.

### 3.1.6. Perchlorate ions

Perchlorate ions inhibit iodine uptake (Greer et al., 2002). In its Opinion of July 2011, Anses proposed a TRV for perchlorate ions by ingestion of 0.7 Eɪg kg $bw^{-1}$ $day^{-1}$ that was used in the present study to assess the risks related to dietary exposure to this substance. This TRV was based on the study by Greer et al. (2002) undertaken in healthy subjects exposed to perchlorate in drinking water for 14 days, in which a small decrease in thyroidal radioiodine uptake was measured (Anses, 2011b).

### 3.1.7. Diphenylarsinic acid

Neurotoxic effects were observed in people chronically exposed to diphenylarsinic acid through the ingestion of water from a contaminated well (Ishii et al., 2014). In these studies, this substance was found to have toxic effects on the cerebellum, brainstem and brain. Diphenylarsinic acid tends to persist in the brain over a long period of time, having long-term repercussions. Mental retardation associated with brain atrophy has been observed in some poisoned children. These neurotoxic effects have also been observed in animals (Negishi et al., 2013; Ozone, et al., 2010). However, these studies cannot be used to determine a toxicological point of departure (POD) since the experimental conditions and/or exposure levels associated with the observed neurological symptoms are not known. In the absence of chronic or sub-chronic oral toxicity studies for establishing a POD, the "Threshold of Toxicological Concern" (TTC) approach, which offers a "minimum threshold value", was deemed the only approach that could be used for this study. In this case, the specific value that could be used was that determined by the compound's classification in Cramer Class III (according to the OECD Toolbox software), with no alert for genotoxicity, corresponding to 90 Eig $person^{-1}$ $day^{-1}$ (i.e. 1.5 Eig kg $bw^{-1}$ $day^{-1}$ considering a person with a body weight of 60 kg) (EFSA, 2012).

### 3.1.8. Triphenylarsine

Toxicological data for triphenylarsine are limited. Consequently, an assessment was undertaken with the QSAR Toolbox and Toxtree software programs. No alerts for in vitro mutagenicity, in vivo genotoxicity or carcinogenicity were found with these softwares, which classified triphenylarsine in Cramer Class III. Consequently, for triphenylarsine, the risk assessment was undertaken based on the TTC of 1.5 Eig kg $bw^{-1}$ $day^{-1}$ as in the case of diphenylarsinic acid.

### 3.1.9. Tetrabromoethane

1,1,2,2-Tetrabromoethane was shown to cause neurotoxic, pulmonary and hepatotoxic effects to occupationally exposed workers by the inhalation route (Morrow et al., 1990; Van Haaften, 1969). In animals, the susceptibility of four-day-old newborn rats treated for 17 days with 1,1,2,2-TBE administered orally was studied and compared with that of young rats between the ages of five and six weeks at the beginning of a 28-day study. The young rats appeared to be more susceptible than the newborns to the toxic effects of TBE in the range of the tested concentrations. A NOAEL at 6 mg kg $bw^{-1}$ day-1, based on hepatotoxic effects was selected (Hirata-Koizumi et al., 2005). The risks related to dietary exposure to TBE were assessed by calculating a MOE based on this NOAEL.

### 3.1.10. Vinyl bromide

Like vinyl chloride, a chemical structure analogue, vinyl bromide is mutagenic both in vitro and in vivo. Vinyl bromide also induces DNA fragmentation in vivo in several organs (stomach, liver, kidneys, bladder, lungs and brain) in mice (IARC, 1986; NTP, 2014). Furthermore, vinyl bromide is suspected to be carcinogenic to humans based on the induction of tumors in multiple organs in rats (NTP, 2014). To date, there is no HBGV available for vinyl bromide. However, the available data described above demonstrate similarities with vinyl chloride in terms of metabolism, genotoxicity and carcinogenicity. Consequently, in the absence of a specific HBGV, risks related to dietary exposure to vinyl bromide were assessed using the slope factor for vinyl chloride (oral route) of 1.5 (mg kg $bw^{-1}$ $day^{-1})^{-1}$ set by the US-EPA (US-EPA, 2000).

## [redacted]Exposure and risk assessment

### 3.2.1. Scenario A: Exposure via the consumption of foodstuffs of animal origin

#### 3.2.1.1. Trace elements

The mean contamination levels measured in foodstuffs from animal origin produced on the "Clere 8r Schwander Meuse" site (meat, liver and milk) were compared with those previously reported in the context of the TDS2 (Anses, 2011a). These results were in the same order of magnitude. It was therefore considered that the consumption of milk, meat and offal produced on the "Clere 8r Schwander Meuse" site was not likely to result in overexposure to TEs in relation to the general population studied in TDS2.

#### 3.2.1.2. Nitroaromatic explosives, arsines, brominated compounds and per-chlorate ions

With the exception of perchlorate ions for which quantifiable levels were measured in milk, meat, liver and kidneys, and triphenylarsine for which quantifiable levels were measured in offal (liver and kidneys), none of the other substances were detected (Table 2). Exposure levels were calculated according to UB hypothesis (Table 4). Results of the risk assessment are

presented in Table 5 (for threshold effects) and Table 6 (for no-threshold effects).

For TNT, 2,4-DNT, 2-ADNT, 4-ADNT, diphenylarsinic acid, triphenylarsine, and perchlorate ions, the calculated exposure levels were lower than the toxicological values, regardless of the population.

For 2,6-DNT and tetrabromoethane, the lowest calculated MOEs were 24,000 and 288,000 respectively (for children – exposure at the 95th percentile as the UB). In light of these MOEs, exposure to 2,6-DNT and tetrabromoethane through milk, liver and meat produced on the "Clere & Schwander Meuse" site was unlikely to be of health concern.

For TNT, an individual excess cancer risk (IECR) slightly above $10^{-6}$ was calculated (for children, considering exposure at the 95th percentile as the UB). For other populations, the IECRs were below $10^{-6}$. For 2,4-DNT, 2,6-DNT and vinyl bromide, IECRs above $10^{-5}$ were calculated for all populations; the highest IECR was calculated for 2,6-DNT in children at the 95th percentile (as the UB) with a value of $2.5 \times 10^{-4}$.

It should be emphasized that the risk was assessed using a worst-case scenario since it was assumed that individuals consume, for their entire lifetime (70 years), only foodstuffs of animal origin produced on the "Clere & Schwander Meuse" site. Consequently, in light of the above results, it was considered that the consumption of foodstuffs of animal origin produced on the "Clere & Schwander Meuse" site was unlikely to be of health concern.

### 3.2.2. Scenario B: Exposure through the consumption of wheat

#### 3.2.2.1. Trace elements

Pb and Cd levels measured in wheat were compliant with their respective regulatory limits (EC, 2006). For other substances for which regulatory limits in wheat have not been set out, the health risk was assessed.

Exposure levels were calculated according to UB hypothesis, considering mean wheat consumption (125 g day-1for adults and 80 g day$^{-1}$ for children). The results obtained are presented in Table 7.

The corresponding health based guidance values were not exceeded for Zn. Therefore, exposure to this element through the consumption of wheat products is unlikely to be of health concern.

With regard to inorganic arsenic, depending on both the scenario and the population, the MOEs calculated with regard to the benchmark dose lower bound (BMDL01 0.3 to 8 pg kg bw$^{-1}$ day-1) ranged from 8 to 665 (Table 8). The average exposure levels calculated for adults and children in this study accounted for 5 and 7% of the mean exposure levels calculated in TDS2 for adults and children respectively. Although not quantified in the three composite samples taken randomly, it should be noted that arsenic was quantified in one "hot spot" sample (0.01 mg kg-1, Table 2).

However, the scenario considered to calculate exposure levels via the consumption of wheat and wheat products appears conservative. Indeed, it was assumed that all wheat products consumed by the considered populations (bread, pastries, biscuits, etc.) were prepared using only wheat produced on the "Clere & Schwander Meuse" site. Therefore, it was considered that the batch of wheat produced on this site is unlikely to pose a health risk. However, given the presence of hot spots detected on certain wheat plots, it appears likely that on certain plots, wheat (and probably any other crops) should not be grown.

#### 3.2.2.2. Nitroaromatic explosives, arsines, brominated compounds, perchlorate ions

None of these substances were detected in wheat (see Table 2). Exposure levels were therefore calculated according to the UB hypothesis, considering mean daily wheat consumption (125 g $d^{-1}$ for adults and 80 g $d^{-1}$ for children). Results of the theoretical exposure calculations are shown in Table 9. Results of the HRA are given in Table 10 (for dose-threshold effects) and Table 11 (for no-dose-threshold effects).

Considering dose-threshold effects, for TNT, 2,4-DNT, 2-ADNT and 4-ADNT, diphenylarsinic acid, triphenylarsine, and perchlorate ions, the calculated exposure levels were lower than the corresponding health based guidance values used for the HRA, regardless of the population. For 2,6-DNT and tetrabromoethane, the lowest calculated MOEs were 150,000 and 1,000,000 respectively (for children – exposure at the 95th percentile as the UB). In light of these MOEs, exposure to 2,6-DNT and tetrabromoethane through the consumption of wheat produced on the "Clere & Schwander Meuse" site was unlikely to be of h Considering no-dose-threshold effects, for TNT, IECRs of approximately $10^{-8}$ to $10^{-7}$ were calculated. As for 2,4-DNT, 2,6-DNT and vinyl bromide, IECRs slightly higher than $10^{-5}$ were calculated for overall populations. So, it can be concluded for these effects and pollutants that the consumption of wheat produced on the "Clere & Schwander Meuse" site was unlikely to be of health concern.

## Discussion

The Meuse site illustrates the potential long-term impact of the destruction and disposal of weapons on the contamination of both soil and the food chain. Other similar destruction sites after WWI, WWII and Cold war have already been identified elsewhere in France and Europe by the BRGM where similar health concern may be encountered. Itis also well established that chemical weapons were disposed in the sea after WWI and WWII (Bełdowski et al., 2016) what raises a more general question on the impact of the disposal and/or destruction of weapons on marine ecosystems. Given the nature of the chemical substances involved (arsenical compounds,

explosives and derivatives), their persistence and toxicity, further consideration related to this issue is needed.

A specific methodology was developed for the health risk assessment related to the consumption of foodstuffs produced on the “Clere & Schwander Meuse” site, enabling the proposal of risk management measures in that particular case. For wellknown contaminants (such as TEs), for which the dietary exposure of French population has already been assessed in the framework of a total diet study, it was determined if the consumption of locally grown foodstuffs could lead to overexposure compared to the general population. For unknown contaminants, such as explosives and chemical warfare agents, specific analytical methods were determined. Concomitantly, a review of the available toxicity data was performed in order to determine toxicological benchmarks robust enough to conduct a health risk assessment. The same logic could easily be applied to other sites polluted by both well-known and unknown contaminants.

It should however be highlighted that several uncertainties can be listed. First of all, for some chemical substances considered, in the absence of health based guidance values, toxicological benchmarks were selected by default (e.g. the TTC was used for triphenylarsine and the slope factor for vinyl chloride was extrapolated to vinyl bromide). Specific toxicological data would therefore be required to fully characterize the hazards related to these compounds. Moreover, other compounds such as thiodyglycol (a metabolite of sulfur mustard and cyanide) also could have been taken into account and this would require the development of ad hoc analytical methods. In the framework of this study, some analytical methods were specifically developed, but it was non-feasible to develop additional methods to investigate other possible pollutants. Lastly, given the persistence of these contaminants and their presence in soil leachate as well as groundwater samples, the contamination of surface water and groundwater would need further consideration. For this particular matter, detailed environmental investigations conducted by the BRGM are foreseen in 2017 on the “Clere & Schwander Meuse” complex to implement the risk assessment by the characterization of groundwater, surface water, flesh of fish and sediments of the river Othain of the ponds close to the site. Deep soil will also be sampled.

Lastly, this work also highlighted the fact that additional data are required on the transfer of these contaminants from soil to plant and from animal feed to animals, to fully assess the risks related to the consumption of foodstuffs grown in contaminated areas. Further research is needed to improve knowledge on the toxicity, space and time fate of compounds present in high explosives, propellants and chemical warfareagents to assess the risk for health, water resource and environment in relation to these sites, in the specific historical, regulatory and environmental frame of France.

[a] During analysis, since tetrabromoethane broke down to tribromoethene, tribromoethene was systematically analyzed.

[b] During analysis, triphenylarsine was oxidized into triphenylarsine oxide. Consequently, both compounds were systematically analyzed.

**Table 1. Analytical methods used for the determination of contaminants and performances**

| | Analytical methods | | (in ıg $kg^{-1}$ fresh product) | |
|---|---|---|---|---|
| Compounds | Sample treatment | Analysis | Foodstuffs of animal origin | Foodstuffs of plant origin |
| Tetrabromoethane | Extraction by dynamic (40 °C) headspace sampling on a | Gas chromatography with single quadrupole MS | LoQ = 60 | LoQ = 200 |
| Tribromoethenea | Carbotrap (25 °C) adsorbent. | detection (GC/MS) | LoQ = 0.5 | LoQ = 1 |
| Vinyl bromide | | | LoQ = 1 | LoQ = 3 |
| Triphenylarsine + Triphénylarsine oxide[b] | PLE extraction using a mixture (70:30, v:v) of toluene and acetone (three extraction cycles, 120 °C, 100 bar). Purification by gel permeation chromatography | Gas chromatography with detection by high-resolution mass spectrometry (GC/MS) | LoQ = 0.03 | LoQ = 0.10 |
| Diphenylarsinic acid | Extraction with tetramethylammonium hydroxide (60 min, 90 °C) | High-performance liquid chromatography (HPLC) with detection by ICP-MS | LoQ = 1 (milk) LoQ = 2 (meat, liver, kidney) | LoQ = 5 |
| TNT | Extraction with acetonitrile (foodstuffs of animal origin) in an Ultra-Turrax or with 50/50 methanol-water mixture (cereals) Purification by solid-phase extraction on an OASIS 200 mg cartridge. | Ultra-performance liquid chromatography combined with a high-resolution mass spectrometer (Orbitrap type) | LoD = 1 | LoD = 5 |
| 2,4-DIT | | | LoD = 10 | LoD = 50 |
| 2,6-DIT | | | LoD = 4 | LoD = 20 |
| 2-ADNT | | | LoD = 1 | LoD = 5 |
| 4-ADNT | | | LoD = 1 | LoD = 5 |

**Table 2. Maximum contamination levels observed in foodstuffs produced on the "Clere & Schwander Meuse" site (Value ± Uncertainty).**

| | TEs (mg·kg$^{-1}$) | | | | Explosives (µg·kg$^{-1}$) | | | | | | Arsines (µg·kg$^{-1}$) | | Brominated compounds (µg·kg$^{-1}$) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Foodstuff | Pb[a] | Cd[b] | As | Zn | 2,4-DIT | 2,6-DIT | TNT | 2-ADNT | 4-ADNT | Perchlorates | TPA + TPA oxide[c] | DPAA | TBE[e] | Vinyl bromide |
| Milk (*n* = 2) | <LiD (0.001) | 0.0012 + 0.0002 | 0.002 + 0.001 | 3.92 + 157 | <LID (10) | <LoD[d] | <LoD[a] | <LoD[a] | <LoD[a] | 6.3 + 3.1 | <LoQ (0.03) | <LoQ | <LoQ (0.5) | <LoQ |
| Muscle (*n* = 32) | <LoQ (0.003) | <LoD (0.0003) | 0.014 + 0.002 | 47.5 + 13.4 | <LoD (10) | <LoD[d] | <LoD[a] | <LoD[a] | <LoD[a] | 11 + 6 | <LoQ(0.03) | <LoQ [b] | <LoQ (0.5) | <LoQ[a] |
| Liver (*n* = 32) | 0.065 + 0.007 | 0.063 + 0.013 | 0.029 + 0.007 | 133.5 + 37.8 | <LoD (10) | <LoD[d] | <LoD (1) | <LoDa | <LoDa | 21 + 12 | 0.14 | <LoQ [b] | <LoQ (0.5) | <LoQ' |
| Kidneys (*n* = 32) | 0.072 + 0.012 | 0.410 + 0.082 | 0.202 + 0.048 | 25.6 + 7.2 | <LoD (10) | <LoD[d] | <LoDa | <LoDa | <LoDa | 2.9 + 18 | 0.03 | <LoQ [b] | <LoQ (0.5) | <LoQ' |
| Wheat (*n* = 4) | 0.029 + 0.012 | 0.044 + 0.014 | 0.01 + 0.004[e] | 52 + 21 | <LoD (10) | <LoD[d] | <LoD[a] | <LoD[a] | <LoD (1) | <LD (0.5) | <LiQ(0.10) | <LoQ [e] | <LoQ[a] | <LoQ[e] |
| Barley (*n = 10)* | 0.051 + 0.02 | 0.018 + 0.006 | 15 + 0.6 | 72 + 29 | <LoD (10) | <LoD[d] | <LoQ*[d] | <LoD[a] | <LoD[a] | <LD (0.5) | 0.87 | 23 | <LoQ | <LoQ[e] |
| Maize silage (*n*=6) | 0.36 + 0.15 | 0.19 + 0.06 | 0.06 + 0.02 | 180 + 72 | <LoD (50) | <LoD (20) | <LoDe | <LoDe | <LoDe | 7.5 + 4.4 | 8.07 | <LoQ e | <LoQ' | <LoQ[e] |

TE: Trace Elements.
When the results are <LoD or <LoQ, the respective values of the LoDs and LoQs are given in parentheses.
[a] Values compliant with the regulatory limits set for foodstuffs: 0.020 mg kg for raw milk, 0.010 mg kg⁻1 for meat, 0.50 mg kg⁻1 for offal and 0.20 mg kg⁻1 for cereals (Regulation (EC) No 1881/2006).
[b] Values compliant with the regulatory limits set for foodstuffs: 0.050 mg kg⁻1 for meat, 0.50 mg kg⁻1 for liver, 1 mg kg⁻1 for kidneys and 0.10 mg kg⁻1 for cereals (Regulation (EC) No 1881/2006).
[c]Arsenic was quantified in one "hot spot" wheat sample and was not quantified in the three composite samples (LoQ = 0.01 mg kg⁻1).
d The analysis showed that triphenylarsine is easily oxidized. Therefore, the two compounds were systematically tested for.
[e]During analysis, tetrabromoethane broke down to tribromoethene. Tribromoethene was systematically tested for with an LoQ of 0.5 µg kg–1.

**Table 3. Table of dairy equivalents.**

| Dairy products | Equivalent volume of whole milk (L) |
|---|---|
| 1 kg butter | 22 |
| 1 kg Emmental | 12 |
| 1 1 whole-milk yoghurt cheeses | 1 |
| Four 250 g Camembert | 8 |

**Table 4. Exposure levels calculated according to the UB hypothesis for nitroaromatic explosives, brominated compounds, perchlorate and arsines, taking into accoun t milk, meat and liverproduced on the "Clere & SchwanderMeuse" site aswell as other dairy products that couldbe made from this milk (butter, ultra-fresh dairy, cheese).**

| | | | HRA - threshold effects - HQ or MOE calculation | | | |
|---|---|---|---|---|---|---|
| | | | Children | | Adults | |
| Substances | Calculation type | Selected toxicological value in ng kg $bw^{-1}$ $day^{-1}$ | Mean exposure | Exposure at the 95th percentile | Mean exposure | Exposure at the 95th percentile |
| TNT | HQ | 500 | 0.006 | 0.011 | 0.004 | 0.011 |
| 2,4-DIT | HQ | 2000 | 0.014 | 0.027 | 0.009 | 0.027 |
| 2,6-DIT | MOE | 4,000,000 | >350,000 | >150,000 | >550,000 | >400,000 |
| 2-ADNT + 4-ADNT | HQ | 2000 | 0.003 | 0.005 | 0.002 | 0.005 |
| Diphenylarsinic acid | HQ | 1500 | 0.009 | 0.018 | 0.006 | 0.018 |
| Triphenylarsine | HQ | 1500 | 185 x $10^{-4}$ | 3.58 x $10^{-4}$ | 1.18 x $10^{-4}$ | 3.58 x $10^{-4}$ |
| Tetrabromoethane | MOE | 6,000,000 | >2000,000 | >1,000,000 | >3,000,000 | >2,500,000 |
| Perchlorate ions | HQ | 700 | 0.002 | 0.004 | 0.001 | 0.004 |

a Exposure levels for these two substances were added up since these compounds have the same TRV and are common metabolites of TNT during plant metabolism.

**Table 5. Risk assessment (tier 2A approach) according to the UB hypothesis: results of the Hazard Quotient and Marge Of Exposure calculations (substances with dose-threshold effects).**

| | | | Risk assessment | | | |
|---|---|---|---|---|---|---|
| | | | Children | | Adults | |
| Substances | Calculation type | Selected toxicological value (ng kg bw$^{-1}$ day$^{-1}$) | Mean exposure | Exposure at the 95th percentile | Mean exposure | Exposure at the 95th percentile |
| TNT | HQ | 500 | 0.034 | 0.083 | 0.019 | 0.083 |
| 2,4-DIT | HQ | 2000 | 0.085 | 0.208 | 0.048 | 0.208 |
| 2,6-DIT | MOE | 4,000,000 | 58,900 | 24,005 | 105,193 | 54,760 |
| 2-ADNT + 4-ADNTa | HQ | 2000 | 0.017 | 0.041 | 0.009 | 0.042 |
| Diphenylarsinic acid | HQ | 1500 | 0.012 | 0.030 | 0.007 | 0.030 |
| Triphenylarsine | HQ | 1500 | 3.40 x 10$^{-4}$ | 8.33 x 10$^{-4}$ | 190 x 10$^{-4}$ | 8.33 x 10$^{-4}$ |
| Tetrabromoethane | MOE | 6,000,000 | 706,795 | 288,066 | 1,262,312 | 657,118 |
| Perchlorate ions | HQ | 700 | 0.236 | 0.592 | 0.132 | 0.592 |

[a] Exposure levels for these two substances were added up since these compounds have the same TRV and are common metabolites of TNT during plant metabolism.

**Table 6. Risk assessment (tier 2A approach) according to the UB hypothesis: results of the individual excess cancer risk calculation (substances with no-dose-threshold effects).**

| | | Risk assessment – IECR result | | | |
|---|---|---|---|---|---|
| | Selected slope factor | Children | | Adults | |
| Substances | (in ng kg • bw$^{-1}$ day$^{-1}$) $^{-1}$ | Mean exposure | 95th percentile | Mean exposure | 95th percentile |
| TNT | 3 x $10^{-8}$ | 5.09 x $10^{-7}$ | 125 x $10^{-6}$ | 2.85 x $10^{-7}$ | 5.48 x $10^{-7}$ |
| 2,4-DIT | 31 x $10^{-7}$ | 5.26 x $10^{-5}$ | 129 x $10^{-4}$ | 2.95 x $10^{-5}$ | 5.66 x $10^{-5}$ |
| 2,6-DIT | 15 x $10^{-6}$ | 102 x $10^{-4}$ | 2.50 x $10^{-4}$ | 5.70 x $10^{-5}$ | 110 x $10^{-4}$ |
| Vinyl bromide | 15 x $10^{-6}$ | 2.55 x $10^{-5}$ | 6.25 x $10^{-5}$ | 143 x $10^{-5}$ | 2.74 x 10-5 |

**Table 7. Exposure to trace metal elements via wheat consumption estimated according to the UB hypothesis**

| Substances | Units | Children (*n* = 59) | | Adults (*n* = 77) | |
|---|---|---|---|---|---|
| | | Mean | 95th percentile | Mean | 95th percentile |
| Pb | μg kg • $bw^{-1}$ $day^{-1}$ | 0.11 | 0.22 | 0.07 | 0.10 |
| Zn | mg $day^{-1}$ | 5.84 | 5.84 | 9.13 | 9.13 |
| Cd | μg kg • $bw^{-1}$ $week^{-1}$ | 1.13 | 2.18 | 0.72 | 0.96 |
| Total As | μg kg • $bw^{-1}$ $day^{-1}$ | 0.054 | 0.054 | 0.018 | 0.024 |
| Inorganic As[a] | μg kg • $bw^{-1}$ $day^{-1}$ | 0.038 | 0.038 | 0.012 | 0.016 |

a Speciation hypothesis used: 70% of total arsenic in wheat is considered to be in inorganic form (EFSA, 2009)

**Table 8. Inorganic arsenic exposure levels and risk assessment considering wheat consumption**

| Population | Mean exposure | $95_{th}$ percentile | MOE for mean exposure | MOE for exposure at the 95th percentile |
|---|---|---|---|---|
| | (µg•kg $bw^{-1}$ $day^{-1}$) | (µgkg·$bw^{-1}$ day-1 | | |
| Adults | 0.012 | 0.016 | 25-665 | 19-505 |
| Children | 0.020 | 0.038 | 15-400 | 8-215 |

MOEs were calculated with regard to the BMDL01 of 0.3 µg kg $bw^{-1}$ $day^{-1}$ (EFSA, 2009).

**Table 9. Exposure estimated for nitroaromatic explosives, brominated compounds, perchlorateand arsines considering wheat consumption (tier 2B approach).**

| | Exposure level (ng kg $bw^{-1}$ $day^{-1}$) | | | |
|---|---|---|---|---|
| | Children (*n* = 59) | | Adults = 77) | |
| Substances | Mean | 95th percentile | Mean | 95th percentile |
| TNT | 2.8 | 5.4 | 177 | 2.36 |
| 2,4-DIT | 27.7 | 53.7 | 17.7 | 23.6 |
| 2,6-DIT | 11.1 | 215 | 7.1 | 9.4 |
| 2-ADNT + 4-ADIT[a] | 5.5 | 10.7 | 3.5 | 4.7 |
| Diphenylarsinic acid | 13.9 | 26.9 | 8.9 | 11.8 |
| Triphenylarsine | 0.277 | 0.537 | 0.177 | 0.236 |
| Vinyl bromide | 8.3 | 16.1 | 5.3 | 7.1 |
| Tetrabromoethane | 2.8 | 5.4 | 18 | 2.4 |
| Perchlorates | 14 | 2.7 | 0.89 | 118 |

a Exposure levels for these two substances were added up since these compounds have the same TRV and are common metabolites of TNT during plant metabolism.

**Table 10. Risk assessment: Results of the Hazard Quotient and Marge Of Exposure calculations (substances with dose-threshold effects). Scenario taking into account the consumption of wheatproduced on the "Clere & Schwander Meuse" site.**

| | | | HRA - threshold effects - HQ or MOE calculation | | | |
|---|---|---|---|---|---|---|
| | | | Children | | Adults | |
| Substances | Calculation type | Selected toxicological value (ng kg bw$^{-1}$ day$^{-1}$) | Mean exposure | Exposure at the 95th percentile | Mean exposure | Exposure at the 95th percentile |
| TNT | HQ | 500 | 0.034 | 0.083 | 0.019 | 0.083 |
| 2,4-DIT | HQ | 2000 | 0.085 | 0.208 | 0.048 | 0.208 |
| 2,6-DIT | MOE | 4,000,000 | 58,900 | 24,005 | 105,193 | 54,760 |
| 2-ADNT + 4-ADNTa | HQ | 2000 | 0.017 | 0.041 | 0.009 | 0.042 |
| Diphenylarsinic acid | HQ | 1500 | 0.012 | 0.030 | 0.007 | 0.030 |
| Triphenylarsine | HQ | 1500 | 3.40 x 10$^{-4}$ | 8.33 x 10$^{-4}$ | 190 x 10$^{-4}$ | 8.33 x 10$^{-4}$ |
| Tetrabromoethane | MOE | 6,000,000 | 706,795 | 288,066 | 1,262,312 | 657,118 |
| Perchlorate ions | HQ | 700 | 0.236 | 0.592 | 0.132 | 0.592 |

a Exposure levels for these two substances were added up since these compounds have the same TRV and are common metabolites of TNT during plant metabolism.

**Table 11. Results of the individual excess cancer risk calculations (substances with no-dose-threshold effects). Scenario taking into account the consumption of wheat produced on the "Clere &Schwander Meuse" site.**

| | | Risk assessment - IECR result | | | |
|---|---|---|---|---|---|
| | Selected slope factor | Children | | Adults | |
| Substances | (in ng kg • bw-1 day-1) -1 | Mean exposure | 95th percentile | Mean exposure | 95th percentile |
| TNT | 3 x 10-8 | 5.09 x 10-7 | 125 x 10-6 | 2.85 x 10-7 | 5.48 x 10-7 |
| 2,4-DIT | 3.1 x 10-7 | 5.26 x 10-5 | 129 x 10-4 | 2.95 x 10-5 | 5.66 x 10-5 |
| 2,6-DIT | 15 x 10-6 | 102 x 10-4 | 2.50 x 10-4 | 5.70 x 10-5 | 110 x 10-4 |
| Vinyl bromide | 15 x 10-6 | 2.55 x 10-5 | 6.25 x 10-5 | 143 x 10-5 | 2.74 x 10-5 |

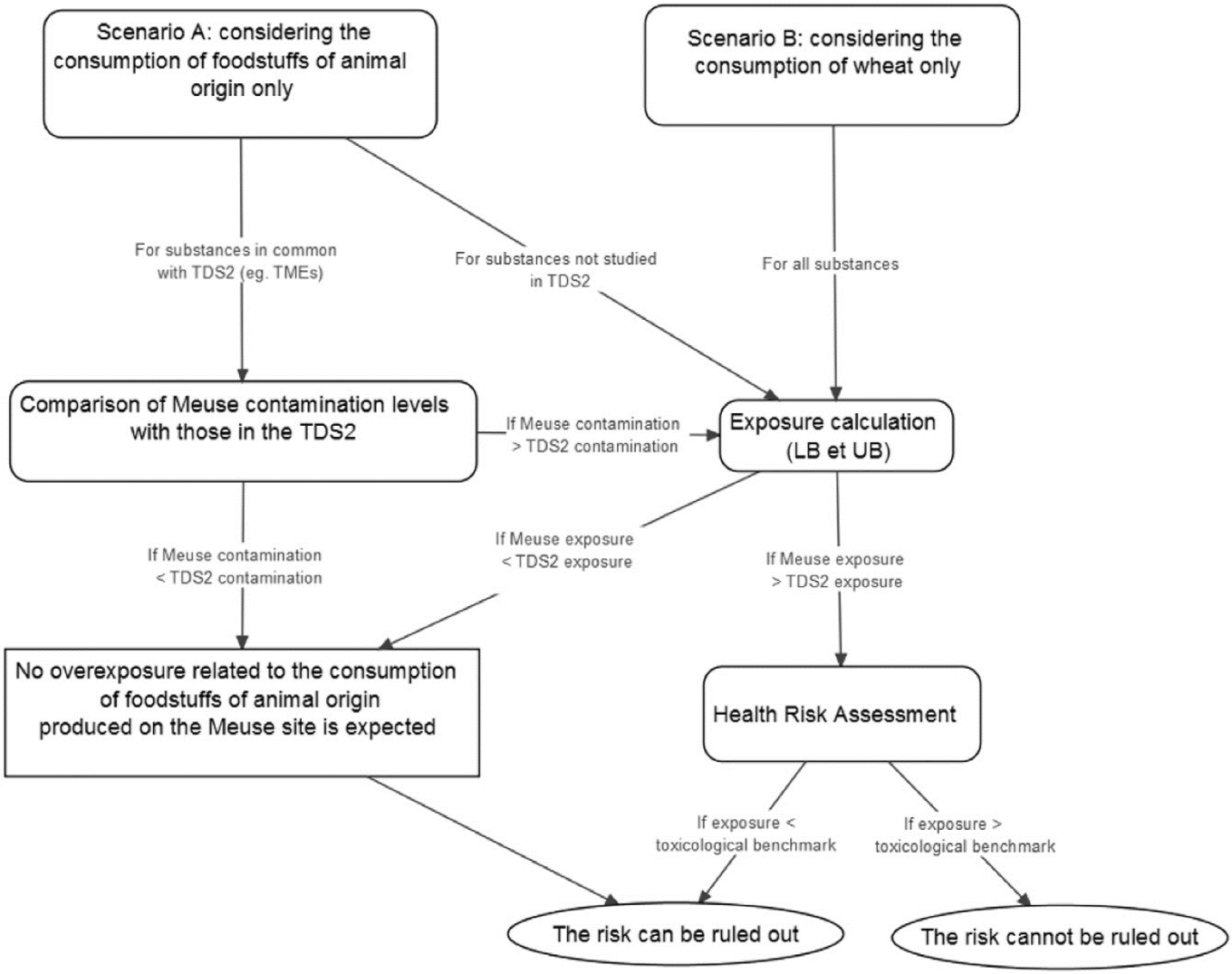



**Figure 1. Procedure followed for the Health Risk Assessment.**

Indication: Numbers correspond to the PCB congeners. Framed congeners are dioxin-like PCBs. Bold numbers were congeners transferred at a high level ranking from 38 to 78% and from 30 to 80% respectively for milk and eggs.

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