ANSES Opinion
Request No 2015-SA-0149

The Director General

Maisons-Alfort, 9 June 2016

Opinion
of the French Agency for Food, Environmental and Occupational Health & Safety

on the health risk related to the consumption of plant and animal products produced on a site polluted by components of chemical munitions in the Meuse *département* of France

ANSES undertakes independent and pluralistic scientific expert assessments.
ANSES primarily ensures environmental, occupational and food safety as well as assessing the potential health risks they may entail.
It also contributes to the protection of the health and welfare of animals, the protection of plant health and the evaluation of the nutritional characteristics of food.

It provides the competent authorities with all necessary information concerning these risks as well as the requisite expertise and scientific and technical support for drafting legislative and statutory provisions and implementing risk management strategies (Article L.1313-1 of the French Public Health Code).

Its opinions are made public.
This opinion is a translation of the original French version. In the event of any discrepancy or ambiguity the French language text dated 9 June 2016 shall prevail.

On 15 July 2015, ANSES received a request from the Directorate General for Food (DGAL) to undertake the following expert appraisal: “Request for an opinion on the health risk related to the consumption of plant and animal products produced on a site polluted by components of chemical munitions in the Meuse *département* of France”.

1. BACKGROUND AND PURPOSE OF THE REQUEST

The French Bureau of Geological and Mining Research (BRGM) was mandated by the prefect of Meuse to undertake an environmental assessment of 'La Place à Gaz', a former site for the destruction of chemical shells from World War I; it is currently a clearing not used for agricultural purposes. For this expert appraisal undertaken between April 2014 and March 2015, a historical approach (archives) revealed the existence of a larger-scale site, the 'Muzeray-Spincourt-Vaudoncourt' or 'Clere & Schwander' complex. According to the experts, it was the largest site for the destruction of chemical munitions in Europe (see map in Annex 1). One and a half million chemical shells and 30,000 explosive shells were blasted or dismantled or even burned there in the 1920s.

The existence of the 'Clere & Schwander' site has been erased from collective memory, which is why it is now largely used for farming. The following are produced there: soft winter wheat, winter barley, spring barley, maize silage, grasses, milk and meat. Moreover, although water is not covered in this opinion, it should be noted that the first investigations of the BRGM (January 2015) revealed significant concentrations of diphenylarsinic acid and arsenic in surface water. In agricultural soils, signatures of nitrates, traces of thianes ('mustard' gas impurities), high

concentrations of zinc and arsenic, locally high levels of mobile nitroaromatic compounds and the near-systematic presence of tetrabromoethane were observed.

Based on the data in the literature on similar sites and information in the BRGM report on soil contamination on the 'Place à Gaz' and 'Clere & Schwander' sites in the Meuse *département*, ANSES was asked to determine whether the consumption of plant and animal products from this zone represents a non-negligible risk to consumers. Pending the results of the health risk assessment (HRA), the authorities decided, as a precautionary measure, to sequestrate the zone's agricultural products. For products of animal origin, two dairy cattle farms are currently affected by these measures. They are holdings having grasslands and/or producing fodder (maize silage in particular) on the 'Clere & Schwander' site.

An interim memo intended for the DGAL was written by ANSES on 28 July 2015. In this memo, the Agency concluded in particular that in the absence of data on the contamination of foodstuffs of plant and animal origin produced on the site of the 'Clere & Schwander' complex, it was not possible to undertake an assessment of the health risks for consumers. Pending such work and its conclusions, and considering the potential hazards and exposure, ANSES stressed the need to maintain the sequestration measures already in place for the affected products.

In order to undertake an HRA, ANSES indicated it was necessary to have data on the contamination of foodstuffs of animal and plant origin produced in the 'Clere & Schwander' complex based on a sampling plan representative of the situation. It was deemed necessary to test for the following substances in foodstuffs as a priority, in light of their established presence in soils and their toxicity:

- Trace metal elements (TMEs); primarily Zn, As, Pb and Cd;
- Nitroaromatic explosives: primarily trinitrotoluene (TNT), 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;
- Phenylarsines including diphenylarsinic acid and triphenylarsine;
- Polychlorodibenzodioxins (PCDDs), polychlorodibenzofurans (PCDFs) and polycyclic aromatic hydrocarbons (PAHs), since while not quantified, these toxic substances were detected in soils. Moreover, there may be larger quantities in certain soils, in particular in the Muzeray zone which, according to the BRGM, served as a burning site for munitions;
- Perchlorate ions;
- Tetrabromoethane.

On 31 July 2015, ANSES received another request from the DGAL to update its memo of 28 July 2015 to take into account the results of the first analyses undertaken in samples of raw milk from the farms affected by the sequestration measures (TMEs, PCDD/Fs and perchlorate ions). In an interim memo dated 8 August 2015, ANSES reiterated that certain substances found in large quantities in soil samples from the 'Clere & Schwander' site had not yet been measured in samples of raw milk (nitroaromatic explosives, phenylarsines, PAHs and tetrabromoethane). ANSES thus again indicated that it could undertake a quantitative health risk assessment once all contamination data for foodstuffs had been generated.

On 1 April 2016, ANSES sent the DGAL an additional interim memo on the assessment of health risks related to the consumption of milk produced on the 'Clere & Schwander' site. This Opinion sets out all of the information sent to the DGAL on 28 July 2015, 8 August 2015 and 1 April 2016.

2. ORGANISATION OF THE WORK

The internal expert appraisal was carried out in accordance with French Standard NF X 50-110 “Quality in Expert Appraisals – General Requirements of Competence for Expert Appraisals (May 2003)”.

It falls within the sphere of competence of the Expert Committee (CES) on Assessment of physico-chemical risks in foods (ERCA). ANSES entrusted the expert appraisal to the 'Meuse site' working group (WG) which met on 17 September 2015, 12 November 2015, 11 March 2016 and 9 May 2016. The methodological and scientific aspects of the WG's work were presented to the CES on 25 November 2015 and 24 March 2016. This Opinion was approved by the ERCA CES at its meeting of 25 May 2016.

ANSES analyses interests declared by experts before they are appointed and throughout their work in order to prevent risks of conflicts of interest in relation to the points addressed in expert appraisals. The experts’ declarations of interests are made public *via* the ANSES website (www.anses.fr).

3. ANALYSIS AND CONCLUSIONS OF THE ERCA CES

3.1. Hazard characterisation and choice of toxicological benchmarks for the health risk assessment

For TMEs, PCDD/Fs, PCBs and PAHs, the toxicological benchmark doses (TBMDs) chosen were based on the Toxicity Reference Values (TRVs) selected by ANSES for the second Total Diet Study (TDS2) (ANSES, 2011) and more recently the Infant Total Diet Study (iTDS – study in progress). In the context of the iTDS, ANSES undertook an exhaustive updated analysis (2015 included) of the toxicity data available for these substances, with the exception of tin, for which no TRV could be proposed due to a lack of data for establishing speciation hypotheses and of robust TRVs for the inorganic forms of tin. These TRVs (or toxicological benchmarks) were deemed sufficiently robust to undertake an HRA and appear in the table in Annex 2.

For the other contaminants, an in-depth literature search was carried out by the 'Meuse site' WG. The results of this literature search are set out in Section 3.2 of this opinion. For certain contaminants (e.g. nitroaromatic explosives), there are available TRVs, and those that were deemed sufficiently robust were used. For the other substances, since no TRVs have been established to date, toxicological benchmarks were proposed based on the data available at the time of the study. The ERCA CES would like to point out that, for these contaminants, the selected values and proposed toxicological benchmarks should not be considered TRVs. These values were chosen specifically for this formal request and are not intended to be used again for another HRA without an exhaustive updated analysis of the literature.

3.2. Estimation of exposure related to the consumption of foodstuffs produced on the 'Clere & Schwander' site

3.2.1. Estimation of contamination in the foodstuffs produced on the 'Clere & Schwander' site

3.2.1.1. Description of the sampling protocol

Foodstuffs of plant origin

Samples of maize silage, barley and wheat were taken for analysis from the nine growing plots covered by management measures (see Table 1). These nine plots are located on the Muzeray, Noire Fontaine and Vaudoncourt sites. The locations of these plots within the 'Clere & Schwander' complex are shown in Annex 3. It should be noted that, further to a new soil testing campaign undertaken by the BRGM in the summer of 2015 with the aim of refining the boundaries of the impact zone, the plots named GL_OH and ER_MA were released from sequestration during the study. These plots have therefore not been taken into account in this risk assessment.

Regarding straw cereals (barley and wheat), in order to ensure representative samples for each plot, the sampling protocol was as follows: ten sampling points were randomly selected in the field using a 'W' design (method described by Belp, 1986). These ten sub-samples were combined (pooled) into equal shares to make composite samples before the analysis.

Moreover, additional samples were taken in highly contaminated areas ('hot spots') determined by the BRGM based on the soil analyses. These additional samples came from the CO_OP (seven 'hot spot' samples) and SV_BL (one 'hot spot' sample) plots.

Given that some farmers wanted to preserve their harvest pending the analysis results, sampling methodologies were different for the SV_MA maize silage plot. For this plot, for which 'hot spots' were reported by the BRGM, maize was preserved by creating a specific ground-pile silo[1] isolated from the other plots.

Table 1: Description of the plots located in the 'Clere & Schwander' complex

Plot code	Surface area (in ha)	Crop	Type of sample
LP_OH	8.9	Winter barley	1 composite sample of 10 sub-samples
LP_BL	16.3	Wheat	1 composite sample of 10 sub-samples
LP_OP	5.16	Spring barley	1 composite sample of 10 sub-samples
FA_BL	6.26	Wheat	1 composite sample of 10 sub-samples
SV_BL	7.74	Wheat	1 composite sample of 10 sub-samples + 1 'hot spot' sample
CO_OP	10	Spring barley	1 composite sample of 10 sub-samples + 7 'hot spot' samples
GL_OH*	18	Winter barley	1 composite sample from a silo
SV_MA	5	Maize silage	Sampling at harvest: 1 composite sample of 10 sub-samples + 5 'hot spot' samples
ER_MA*	2.8	Maize silage	Sampling from a plot along a transect: 1 composite sample

*Plot released from sequestration during the study further to a new soil testing campaign undertaken by the BRGM in the summer of 2015.

Foodstuffs of animal origin

For products of animal origin, there are two affected dairy cattle farms. They are holdings having grasslands and/or producing fodder (maize silage in particular) on the 'Clere & Schwander' site. In order to assess the risks related to the consumption of foodstuffs of animal origin produced on the 'Clere & Schwander' site, 32 animals from the two farms affected by the sequestration measures were slaughtered. Nineteen were from holding A, which had a herd of 286 cattle and 13 were from holding B, which had a herd of 138 cattle. They were gradually slaughtered based on their stage of

[1] Fodder is placed directly on the ground, in a dry area. The silo is then covered with sheeting.

development. Sixteen dairy cows, one heifer, three young bulls and 12 calves were thus slaughtered. For each of these animals, muscle, liver and kidney samples were taken in sufficient quantities for the analyses. Some samples from the same class of animals were grouped together with no more than ten samples per pool. This was the case for some TME, PCDD/F, PCB and PAH analyses. All the other analyses were undertaken with individual samples from the same animal.

Lastly, several composite samples of raw milk were taken from these two farms for analysis. Two sampling campaigns were organised:

- A first campaign, on 20 July 2015: on each of the two farms, three one-litre samples were taken to test for TMEs, perchlorate and PCDD/Fs+PCBs+PAHs;
- A second campaign, on 8 October 2015: on each of the two farms, two one-litre samples were taken to test for explosives and chemical warfare agents or derivatives.

All the milk, meat and offal samples were shipped in frozen form to the laboratories in charge of the analyses.

The ERCA CES notes that for milk, only one sampling campaign was undertaken for each group of contaminants.

3.2.1.2. Analytical methods used

For TMEs (Pb, Cd, As, Hg, Al, Co, Cu, Zn, Sn, Sb, Ni), the laboratory of the Joint Laboratory Service (SCL) of Lille undertook the analyses in plants. Mineralisation was undertaken according to the NF EN 14084[2] standard and inductively coupled plasma mass spectrometry (ICP-MS) was used for analysis. The Maisons-Alfort Laboratory for Food Safety (LSA) undertook the analyses for foodstuffs of animal origin (milk, muscle, liver and kidneys) by ICP-MS according to the in-house method LSA-INS-0086 - ANSES Maisons-Alfort CIME 11[3]. For PCDD/Fs and PAHs, analyses were undertaken by LABERCA[4] according to in-house methods[5]. Since testing for TMEs, PCDD/Fs and PAHs was undertaken according to standardised analytical methods or methods previously assessed by ANSES, these analytical methods were not re-validated by the 'Meuse site' WG.

However, the analytical methods developed for perchlorate ions, TNT and its derivatives, arsines, tetrabromoethane and vinyl bromide were discussed in the WG. All these methods were deemed sufficiently robust to be able to undertake this HRA.

Diphenylarsinic acid:
Testing for diphenylarsinic acid was carried out by LEAV[6] after preparation (grinding and freeze-drying/drying) of the samples by LABERCA. The analysis consisted of extraction (60 minutes, 90°C) from the ground dry samples with a buffer solution, a centrifugation-filtration stage and high-performance liquid chromatography (HPLC) analysis with detection by ICP-MS (*m*/*z* 75).

[2] NF EN 14084 July 2003 - Foodstuffs - Determination of trace elements - Determination of lead, cadmium, zinc, copper and iron by atomic absorption spectrometry (AAS) after microwave digestion.
[3] ANSES Maisons-Alfort CIME 11: Determination of levels of heavy metals and minerals (lithium, boron, aluminium, sodium, magnesium, potassium, calcium, titanium, vanadium, chromium, manganese, iron, nickel, cobalt, copper, zinc, gallium, germanium, arsenic, selenium, strontium, molybdenum, silver, cadmium, tin, antimony, tellurium, barium, mercury, lead and uranium) in all foodstuffs - Mineralisation by closed-system microwave digestion and measurement by inductively coupled plasma mass spectrometry (ICP-MS).
[4] Laboratory for Residues and Contaminants in Food.
[5] LABERCA/HAP-AL.1.05 method for the analysis of PAHs and LABERCA/DGAl/DPCB-al.2.01 method for the analysis of PCDD/Fs and PCBs.
[6] Laboratory for the Environment and Food of Vendée.

Measurements were taken by external calibration. In order to verify the extraction quality, 2 µg $As_{equivalent}.L^{-1}$ of arsenocholine was systematically added to the sample before extraction.

Triphenylarsine:
Testing for triphenylarsine was undertaken by LABERCA. The analysis consisted of high-pressure extraction of fat with organic solvents (three cycles, 120°C, 100 bar) using a mixture (70/30 v:v) of toluene and acetone, a purification stage by gel permeation chromatography and analysis by gas chromatography with detection by high-resolution mass spectrometry (GC/HRMS) in 'Single Ion Monitoring' (SIM) mode. Standards (internal $^{13}C_{12}$ PCB-77 and external $^{13}C_{12}$ PCB-111) were used to monitor analysis quality.

Tetrabromoethane and vinyl bromide:
Testing was undertaken by LABERCA with dynamic headspace extraction, Carbotrap adsorbent trapping and thermal desorption before injection in cryofocused split mode (1:10, where 10% of the sample was injected in the column). The analysis was carried out by single quadrupole GC/HRMS using an ionisation source by electron impact. Signal acquisition was undertaken in SIM mode by monitoring vinyl bromide (*m/z* 106 and 108), tribromoethene (*m/z* 264; 266 and 185) and tetrabromoethane (*m/z* 267; 265 and 263). Measurements were taken by adding 100 ng of internal standards (1,1-dichloroethene *m/z* 61 and 96 for vinyl bromide and naphthalene-d8 *m/z* 136 for tetrabromoethane and tribromoethene). It should be noted that during analysis, tetrabromoethane broke down to tribromoethene. Tribromoethene was systematically tested for with a limit of quantification of 0.5 $\mu g.kg^{-1}$.

Perchlorate ions:

All the samples were tested for perchlorate ions by the Joint Laboratory Service (SCL) of Strasbourg. The method used was that developed by the American Food and Drug Administration (revision 2 dating from 2005) and used for the ANSES Opinion of 4 June 2014 on the presence of perchlorate ions in infant formula and drinking water in France (ANSES, 2014).

TNT, 2,4-DNT, 2,6-DNT, 2-ADNT and 4-ADNT:
Milk samples were processed by the Central Laboratory of the Prefecture of Police (LCCP). After solvent extraction (two successive extractions with acetone), followed by centrifugation, concentration and then dilution in water, the extracts were purified by solid-phase extraction on an OASIS cartridge (hydrophilic-lipophilic copolymer). The compounds of interest were then eluted with acetonitrile.

Muscle, liver and kidney samples were processed by LABERCA. They were ground and then extracted with acetonitrile in a dispersing and homogenising instrument (Ultra-Turrax). After centrifugation and concentration, the extracts were partially purified (delipidation by liquid-liquid extraction with hexane) and then concentrated. These extracts were then sent to the LCPP, which subjected them to a new purification stage (after dilution with water) by solid-phase extraction on an OASIS cartridge. The compounds of interest were then eluted with acetonitrile.

Cereal samples were ground and dried by LABERCA. After ultrasound-assisted solvent extraction (two successive extractions with a 50/50 methanol-water mixture) followed by centrifugation, the extracts were purified by solid-phase extraction on an OASIS cartridge. The compounds of interest were then eluted with acetonitrile. All the purified extracts were then diluted to one-half with water (to limit matrix effects), before being analysed by ultra-performance liquid chromatography (pentafluorophenyl phase) combined with a high-resolution mass spectrometer (Orbitrap type) with an APCI (atmospheric-pressure chemical ionisation) type interface. An internal standard (1,3-dinitrobenzene) was added to all the samples to ensure that the various stages were carried out properly during sample processing. Quantification (external calibration) was performed based on

the main ion for each compound; identification was undertaken based on the retention time on the one hand and the ratio between the quantifier ion and a confirmation (qualifier) ion.

3.2.1.3. Contamination data used for exposure calculations

For each studied foodstuff/contaminant pair, as part of a 'worst-case' scenario, the maximum contamination value obtained in this study was used. For the foodstuffs of plant origin, the levels measured in composite samples (taken according to the method of Belp (1986)) were taken into account for the calculation of exposure. These composite samples are considered representative of average contamination on each plot, which is not the case for the 'hot spot' samples.

Censored data[7] were processed using the 'substitution method' recommended by the World Health Organization (WHO) (WHO, 2013). It consists in defining the lower bound (LB) and upper bound (UB) for a measured value. The LB is calculated by considering that all values below the LD are equal to zero and those between the LD and LQ are equal to the LD; the UB is calculated by considering that all values below the LD are equal to the LD and those between the LD and LQ are equal to the LQ.

3.2.2. Consumption data used for exposure calculations

Exposure levels were estimated for the general population:

- Children and adolescents between the ages of three and 17 years;
- Adults over the age of 17 years.

The consumption data used for the general population were taken from INCA2, an individual and national study on food consumption (ANSES, 2009). This study was broken down into three waves between the end of 2005 and April 2007 in order to take into account seasonal variations. Two separate populations were included in the study: children between the ages of three and 17 years (1455 individuals) and adults between the ages of 18 and 79 years (2624 individuals). Food consumption was recorded with a consumption diary for seven consecutive days. This methodology was required to undertake both chronic long-term and acute short-term risk assessments. Every day was broken down into three meals and three between-meal snacks.

For each snack or meal, the participants had to describe in detail all of the foods and beverages consumed, estimate the quantity consumed using a manual of portion-size photographs, household measures or unit weights or volumes, and provide information about the type of product (industrial/home-made, fresh/tinned/frozen, enriched/light/or not). The information collected in the food consumption and supplement diaries was verified and harmonised by dieticians. Foods were codified using the INCA2 nomenclature with 43 groups created specifically for the study, also taking into account the previous version used in the INCA1 study. This nomenclature is compatible with that on the nutritional composition of foods managed by the ANSES Information Centre on Food Quality (CIQUAL).

In the framework of this study, in order to take into account local consumption habits, only survey respondents in the vicinity of Meuse[8] were selected, i.e. 136 individuals (77 adults and 59 children). It should be noted that for these individuals, consumption levels for meat, liver and dairy products were of the same order of magnitude as those for individuals living in the East Region and those recorded at national level.

[7] Censored data refer to results below the limit of detection (LD) or quantification (LQ).

[8] Since no data were specifically available for Meuse, consumption data collected in *départements* located near the study zone were taken into account: Meurthe-et-Moselle (54), Ardennes (08), Moselle (57).

3.2.3. Calculation of exposure

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

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

Where:
- E_i is the total daily exposure of an individual i (µg.kg body weight^{-1}.day^{-1}),
- $C_{i,k}$ is daily consumption of the food k by an individual i (g.day^{-1}),
- L_k is the estimated level for the studied contaminant in the food k (mg.kg^{-1} fresh food),
- BW_i is the body weight of the individual i (kg),
- and n is the total number of foods consumed by the individual i.

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

3.3. Scenarios considered for the calculation of exposure and assessment of health risks

The assessment of exposure was broken down into the two stages described below. Firstly (stage 1), all foodstuffs of plant and animal origin from the 'Clere & Schwander' zone were considered. Since the HRA showed that the reference values were exceeded for certain substances at the end of stage 1 (see results in Section 3.3.1.), the exposure scenario was then refined, considering only foodstuffs of animal origin on the one hand and only wheat on the other hand (stages 2A and 2B respectively).

Maize silage, barley and kidneys were not taken into account for the calculation of exposure, for several reasons explained below.

For kidneys, the 136 individuals from the INCA2 study considered in this study said that they do not consume kidneys. Furthermore, given the low consumption of kidneys in the general population (on average 0.08 and 0.01 g.day^{-1} for adults and children respectively, ANSES, 2009) and considering that contamination levels for kidneys were of the same order of magnitude as those measured in other matrices (see Annex 9), their non-inclusion is unlikely to modify the conclusions of this HRA.

Several data were lacking to quantitatively assess the risk related to the consumption of barley and maize silage (uncertainties related to toxicology, the spatial variability of levels in soil, and soil-plant, plant-animal and soil-animal transfer rates). Therefore, maize silage and barley were considered separately from other foodstuffs.

3.3.1. Stage 1: Assessment of exposure *via* the consumption of foodstuffs of animal and plant origin produced on the 'Clere & Schwander' site

First of all, exposure to contaminants *via* foodstuffs produced on the 'Clere & Schwander' site was calculated. In this scenario, it was assumed that the individuals consume only local milk, meat, offal and wheat produced on the 'Clere & Schwander' site, throughout their lifetime. This worst-case scenario was used as a first approach to have a first assessment of the risk related to the consumption of foodstuffs produced on the 'Clere & Schwander' site.

For milk, meat and offal, the individual consumption data collected during the INCA2 study were taken into account. For wheat, which is likely to be integrated in the form of flour in many of the products consumed in INCA2, additional work was undertaken to estimate total daily consumption of this foodstuff. To do so, the list of recipes from the INCA2 study was used to determine in which products wheat flour is likely to be integrated and in what proportions. For adults, consumption is 125 $g.day^{-1}$ on average and 220 $g.day^{-1}$ at the 95^{th} percentile. For children, consumption is 80 $g.day^{-1}$ on average and 190 $g.day^{-1}$ at the 95^{th} percentile. For the HRA, wheat consumption levels at the 95^{th} percentile were considered. Given uncertainties related to the use of wheat in the form of flour in various foods including processed foods (bread, biscuits, etc.), exposure through the rest of the normal diet was not taken into account.

3.3.2. Stage 2A: Assessment of exposure *via* the consumption of foodstuffs of animal origin produced on the 'Clere & Schwander' site

For foodstuffs of animal origin (meat, milk and liver), the HRA was undertaken according to the procedure presented in the flow charts found in Annexes 4 and 5. The procedure was tailored to the type of substance: substance considered in the TDS2 study or substance not considered in the TDS2 study. For substances in common with TDS2, exposure through total diet could be taken into account.

Substances in common with TDS2 (TMEs, PCDD/Fs, PCBs, PAHs)

For contaminants in common with TDS2 (TMEs, PCDD/Fs, PCBs, PAHs), the average contamination levels found in foodstuffs produced in the 'Clere & Schwander' complex were firstly compared with the average contamination levels observed in the TDS2 study. This comparison was possible for milk, meat and liver, which were sampled in the framework of TDS2.

Since foodstuffs were analysed as consumed in TDS2, it was assumed that the contaminants contained in the raw foodstuffs analysed in this study are fully transferred to finished products (with no dilution or concentration effect).

For substances whose average contamination levels were lower than those of the TDS2 study, overexposure related to the consumption of foodstuffs produced in the 'Clere & Schwander' complex is not expected, and the conclusions are the same as those given for TDS2.

Total dietary exposure was calculated only for substances whose average contamination levels observed in this study were higher than those found in TDS2. In this case, total dietary exposure was calculated by combining the contamination data for the normal diet (excluding meat, offal and milk) from TDS2 with the contamination data available for this study (meat, liver and milk). The exposure levels thus calculated were compared with the exposure levels (average and 95^{th} percentile) from TDS2 in order to determine whether the consumption of foodstuffs produced on the 'Clere & Schwander' site can result in overexposure to these contaminants.

When the calculated exposure levels were higher than those of TDS2, a specific HRA was undertaken. For substances with exposure levels lower than those of TDS2, the conclusions are the same as those given for TDS2.

Substances not considered in TDS2 (nitroaromatic explosives, perchlorate ions, brominated compounds and arsines)

For substances not considered in the TDS2 study (nitroaromatic explosives, perchlorate ions, brominated compounds and arsines), exposure levels related only to the consumption of meat, offal (liver and kidneys) and milk were calculated. Since contamination levels in the rest of the normal diet are not known, this assessment could lead to the underestimation of dietary exposure to these substances. Nonetheless, with the exception of perchlorate ions whose presence in certain common foods has been documented (EFSA, 2013; ANSES, 2014), the other substances are related quite specifically to the shell-disposal activities that took place in the studied zone; therefore, widespread contamination of the entire normal diet seems unlikely.

In order to take into account exposure related to the consumption of dairy products that could be made from raw milk produced on the 'Clere & Schwander' site, contamination levels for certain dairy products (ultra-fresh dairy, butter and cheese[9]) were estimated using a table of dairy equivalents available on the website of the French National Federation of Dairy Cooperatives[10].

Table 2: Table of dairy equivalents (source: French National Federation of Dairy Cooperatives)

Dairy products	Number of litres of whole milk
1 kg butter	22
1 kg Emmental	12
1 litre whole-milk yoghurt	1
Four 250g Camembert cheeses (i.e. 1 kg)	8

According to this table, since 22L of raw milk are required for example to make 1kg of butter, contamination levels in butter were assumed to be 22 times those measured in raw milk. It is therefore assumed that substances are fully transferred to by-products, with no dilution. This scenario is deemed conservative by the ERCA CES.

The exposure levels thus calculated were compared with the toxicological benchmarks used for this study.

3.3.3. Stage 2B: Assessment of exposure *via* the consumption of wheat produced on the 'Clere & Schwander' site

The exposure estimation procedure developed for foodstuffs of animal origin could not be applied for wheat. Given that foodstuffs were analysed as consumed in TDS2 and cereals are used as ingredients in a wide variety of products, the contamination levels measured in this study cannot be compared with the TDS2 reference levels.

[9] Consumption data for these foodstuffs are available in INCA 2.
[10] http://www.fncl.coop/filiere-laitiere/collecter-et-transformer-le-lait

Exposure levels were estimated by considering only the consumption of products made from wheat flour, taking into account average wheat consumption[11]. Exposure through the rest of the normal diet could not be taken into account.

3.4. Hazard characterisation: toxicological data for the main contaminants found in the soil of the 'Clere & Schwander' complex and choice of toxicological benchmarks

3.4.1. War explosives

3.4.1.1. 2,4,6-trinitrotoluene (TNT)

Toxicity studies undertaken in animals indicate that chronic oral exposure to this substance can lead to effects on the liver, kidneys and blood (ATSDR, 1995). TNT is currently classified by the IARC[12] as belonging to Group 3, "not classifiable as to its carcinogenicity to humans" (IARC, 1996). However, in 2008, OEHHA[13] listed TNT on the list of substances known to cause cancer in humans[14]. OEHHA based its assessment on the results of two carcinogenicity studies indicating the development of benign bladder tumours in female rats exposed to TNT for two years, as well as the results of several genotoxicity studies (positive *in vitro* and *in vivo* test results) and TNT's structural similarity to 2,4-dinitrotoluene and 2,6-dinitrotoluene (possibly carcinogenic according to the IARC).

Regarding the threshold effects of TNT, the US EPA[15] set a TRV (RfD[16]) of 0.5 µg.kg $bw^{-1}.day^{-1}$ based a study on dogs exposed for 26 weeks, using hepatotoxic effects as the critical effect. This TRV was determined based on a LOAEL[17] of 0.5 mg.kg $bw^{-1}.day^{-1}$, with a safety factor of 1000: ten for animal-human extrapolation, ten for human variability and ten to take into account the length of the study (26 weeks) for assessing chronic exposure and the use of a LOAEL instead of a NOAEL[18] (US-EPA, 1988).

Regarding no-threshold effects, the US EPA established a slope factor (SF) of 0.03 $(mg.kg\ bw^{-1}.day^{-1})^{-1}$ based on a two-year carcinogenicity study in rats, using combined urinary tract tumours as the critical effect (US-EPA, 1988).

In this study, the risk related to dietary exposure to TNT was assessed using these two approaches to take into account threshold effects on the one hand and no-threshold effects on the other hand.

3.4.1.2. 2,4-dinitrotoluene (2,4-DNT)

Toxicity studies undertaken in animals indicate that chronic oral exposure to 2,4-DNT can have effects on the liver, kidneys and blood. This substance is also responsible for carcinogenic effects in animals. Carcinogenesis studies undertaken in rats indicate that 2,4-DNT induces tumours in the

[11] For adults, average consumption is 125 $g.day^{-1}$. For children, average consumption is 80 $g.day^{-1}$.
[12] International Agency for Research on Cancer.
[13] Office of Environmental Health Hazard Assessment.
[14] OEHHA. Proposition 65 of the "Safe Drinking Water and Toxic Enforcement Act of 1986" http://oehha.ca.gov/prop65/prop65_list/Newlist.html.
[15] United States Environmental Protection Agency.
[16] Reference Dose.
[17] Lowest Observed Adverse Effect Level.
[18] No Observed Adverse Effect Level.

renal tubules, hepatocellular carcinomas, adenomas of the mammary gland, and fibromas and fibrosarcomas of the skin. Thus, 2,4-DNT is classified by the IARC as belonging to group 2B, 'possibly carcinogenic' (IARC, 1996).

Regarding the threshold effects of 2,4-DNT, the US EPA set a minimum risk level of two µg.kg $bw^{-1}.day^{-1}$ based a two-year study on oral exposure in dogs, using haematological effects as the critical effect. This value was established based on a NOAEL of 0.2 mg.kg $bw^{-1}.day^{-1}$, with a safety factor of 100: ten for transposition from animals to humans, and ten for inter-individual human variability (US-EPA, 1992).

Regarding the no-threshold effects of 2,4-DNT, an SF of 0.31 $(mg.kg\ bw^{-1}.day^{-1})^{-1}$ was established by the OEHHA[19] based on the incidence of tumours of the liver and mammary gland.

The risk related to dietary exposure to 2,4-DNT was assessed based on these two values to take into account no-threshold effects on the one hand and threshold effects on the other hand.

3.4.1.3. 2,6-dinitrotoluene (2,6-DNT)

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

Regarding the threshold effects of 2,6-DNT, as part of the Superfund Program[20], the US EPA set a provisional TRV (PPRTV)[21] of 0.3 µg.kg $bw^{-1}.day^{-1}$. This provisional TRV was determined based on a 13-week study on oral exposure in dogs, using haematological effects as the critical effect. It is based on a LOAEL of 4 mg.kg $bw^{-1}.day^{-1}$ (equivalent to 3 mg.kg $bw^{-1}.day^{-1}$ in humans after allometric adjustment), with a safety factor of 10,000: three for animal-human extrapolation, ten for human variability, ten to take into account the lack of developmental toxicity studies, three to take into account the use of a LOAEL and ten to take into account the short study period for assessing chronic exposure (US EPA, 2013).

Regarding no-threshold effects, as part of the Superfund Program, the US EPA set a provisional SF 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 µg.kg $bw^{-1}.day^{-1}$ and the various 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), the ERCA CES deemed it 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 $bw^{-1}.day^{-1}$. At the same time, the risk related to dietary exposure to 2,6-DNT was estimated based on the provisional SF to take into account no-threshold effects.

[19] OEHHA - 2005 Toxicity Criteria Database http://oehha.ca.gov/risk/pdf/cancerpotalpha81005.pdf.
[20] This programme, managed by the US EPA, is responsible for cleaning up polluted sites and soils, http://www.epa.gov/superfund.
[21] PPRTV: Provisional Peer-Reviewed Toxicity Value.

3.4.1.4. 2-amino-4,6-dinitrotoluene (2-ADNT) and 4-amino-2,6-dinitrotoluene (4-ADNT)

Only one acute toxicity study was undertaken in which lethal doses, 50% (LD_{50}[22]) were established for 2-ADNT and 4-ADNT in rats. They were 1400 $mg.kg^{-1}$ and 1000 $mg.kg^{-1}$, respectively for 2-ADNT and 4-ADNT (Ellis, 1980). Another study in which rats were exposed by gavage to a single dose of 2-ADNT, 4-ADNT, 2,6-DNT, 2,4-DNT or 2,4,6-TNT showed that these substances can cause hepatotoxic effects. In this study, the effects of 2-ADNT and 4-ADNT were not as strong as those observed after administration of 2,6-DNT, 2,4-DNT and 2,4,6-TNT (Deng *et al.,* 2011).

However, no sub-chronic or chronic studies have been identified to date and no TRVs have been established for these two substances.

That said, the RfD of 2 $\mu g.kg\ bw^{-1}.day^{-1}$ established for 2,4-DNT (US EPA, 1992) is used by the US EPA to set soil screening levels[23] for 2-ADNT and 4-ADNT. Likewise, for these two substances, the Finnish CEDA[24] uses this TRV to establish Maximum Acceptable Concentrations (MACs) in the soil not to be exceeded for military use of this soil (Koponen, 2015)[25].

Since there are no specific toxicological data for 2-ADNT or 4-ADNT, the RfD of 2 $\mu g.kg\ bw^{-1}.day^{-1}$ was used to assess the risk related to dietary exposure to 2-ADNT and 4-ADNT. 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 the two compounds.

3.4.1.5. Perchlorate ions

Although not quantified in the preliminary water analyses undertaken by the BRGM, perchlorate ions were considered relevant substances to be measured. In fact, a large quantity of perchlorate explosives was used in World War I. Moreover, high levels of chlorate and perchlorate ions were measured in samples of soil leachate from 'Place à Gaz' in 2007 (Bausinger *et al.*, 2007).

The toxic effects of perchlorate ions in humans have been established. They act on the thyroid, inhibiting iodine uptake (Greer *et al.*, 2002).

In its Opinion of 18 July 2011, ANSES proposed a TRV for perchlorate ions by ingestion of 0.7 $\mu g.kg\ bw^{-1}.day^{-1}$. This TRV was based on the study by Greer *et al.* (2002) undertaken in healthy subjects (21 women and 16 men) exposed to perchlorate in drinking water for 14 days, in which a decrease in thyroidal radioiodine uptake was measured. The dose of 7 $\mu g.kg\ bw^{-1}.day^{-1}$ caused only a marginal decrease in iodine uptake (1.8%), considered non-harmful, and was therefore used as the No Observed Effect Level (NOEL). The TRV was determined by applying an intra-specific safety factor of ten, to account for the most susceptible individuals (ANSES, 2011).

In this study, this TRV was used to assess the risks related to dietary exposure to perchlorate ions.

[22] LD_{50} is the amount of a substance, administered once, that kills 50% of the animals in the test group.
[23] Limit values in soil set by the US EPA in the context of the Superfund Program. These values are not regulatory but are established by the US EPA for the evaluation and rehabilitation of polluted sites and soils. These values are available at the following address: http://www.epa.gov/risk/risk-based-screening-table-generic-tables.
[24] Construction Establishment of Finnish Defence Administration.
[25] Article consulted at the following address: http://www.ecde.info/sites/default/files/docs/article_koponen.pdf.

3.4.2. Chemical warfare agents

3.4.2.1. Phenylarsines: diphenylarsinic acid and triphenylarsine

Diphenylarsinic acid

Diphenylarsinic acid is a soil degradation product of sternutatory arsines (diphenylarsine cyanide and diphenylarsine chloride) used as vomiting and sneezing agents in chemical weapons in warfare.

Only one acute toxicity study was undertaken in which diphenylarsinic acid was found to be toxic after oral exposure with an LD_{50} of 17 $mg.kg^{-1}$ in mice (Marhold, 1986)[26].

Regarding genotoxicity, the only study that has been undertaken was an *in vitro* trial in Chinese hamster V79 cells showing structural and numerical chromosome changes (Ochi *et al.*, 2004). However, the cell strain used was a *p53*[27]-deficient, highly karyotypically unstable murine line, which could directly impact the genotoxic response (Honma and Hayashi, 2011). This result is thus merely an alert for *in vitro* genotoxicity and will need to be confirmed by a study using genomically stable human cells, especially since the published experimental data are not fully correlated with *in silico* predictions. Toolbox and CAESAR quantitative structure-activity relationship (QSAR) models do not predict any alerts for *in vitro* mutagenicity, *in vivo* genotoxicity or carcinogenicity. In view of the available genotoxicity studies however, it is not possible to conclude as to the lack of genotoxicity.

In a short-term induction/promotion model in rats, diphenylarsinic acid induced carcinogenesis through a promotion mechanism (Wei *et al.,* 2013). Experimentally, however, only standard long-term carcinogenesis studies could confirm or invalidate possible carcinogenic potential.

Neurotoxic effects were observed in Japan in people chronically exposed to diphenylarsinic acid through the ingestion of water from a well contaminated by this substance. This substance was found to have toxic effects on the cerebellum, brainstem and brain (Ishii *et al.*, 2014). Diphenylarsinic acid tends to persist in the brain over a long period, 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. Ozone *et al.* (2010) observed a decrease in spatial learning ability in rats exposed orally for seven to 28 days. Furthermore, Negishi *et al.* (2013) noted that young rats exposed for six weeks from birth *via* breast milk from their mother and then drinking water (from the age of three weeks) had neurobehavioural abnormalities. These neurotoxic effects seem to be mediated by 'oxidative stress' (Ishii *et al.*, 2004, Kato *et al.*, 2007). According to a study undertaken in rats and cited by Kato *et al*. (2007), NOAELs were estimated at 0.3 and 0.8 $mg.kg\ bw^{-1}.day^{-1}$, respectively for males and females. According to the authors, these values are considered ten times higher than those for humans. However, they were taken from toxicological reports issued by the Japanese Ministry of the Environment[28] and no translation is available. These values could not be used in these conditions.
Lastly, in addition to these neurological effects, exposure to diphenylarsinic acid has also caused miscarriage (Ogata *et al.*, 2014).

[26] Consulted on the following site: http://chem.sis.nlm.nih.gov/chemidplus/rn/4656-80-8.
[27] The *p53* gene is recognised as a tumour suppressant.
[28] [Toxicological reports of DPA by Ministry of the Environment of Japan, URL: http://www.env.go.jp/en/].

In conclusion, unlike for inorganic arsenic, the carcinogenicity of diphenylarsinic acid is not known. Neurotoxic effects have been demonstrated in humans and animals but 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 TRVs and 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 Toolbox software), with no alert for genotoxicity, corresponding to 90 µg.person^{-1}.day^{-1} (i.e. 1.5 µg.kg bw^{-1}.day^{-1} considering a person with a normal body weight of 60 kg) (EFSA, 2012a).

Triphenylarsine

The literature search indicates there are almost no available toxicological data for triphenylarsine. Only one experimental study was found (National Research Council, 1954)[29]. In this study, triphenylarsine was toxic by intraperitoneal injection with an LD_{50} above 500 mg.kg bw^{-1} in mice. It should be noted that Regulation (EC) No 1272/2008 on classification, labelling and packaging of substances and mixtures does not mention the intraperitoneal route for classification purposes.

Haz-Map®[30], an occupational health database designed for the health and safety of professionals and consumers, describes this substance as being an irritant that is toxic by ingestion and inhalation.

An assessment was undertaken with the QSAR Toolbox and Toxtree (version 2.5.0) software programs. No alerts for *in vitro* mutagenicity, *in vivo* genotoxicity or carcinogenicity were found with these programs, which place triphenylarsine in Cramer Class III.

In the absence of consolidated toxicological information on triphenylarsine and considering that this substance has the same predictive profile as diphenylarsinic acid, the HRA was undertaken based on the TTC of 90 µg.person^{-1}.day^{-1} (i.e. 1.5 µg.kg bw^{-1}.day^{-1} considering a person with a normal body weight of 60 kg).

3.4.3. Other substances

3.4.3.1. Tetrabromoethane (TBE)

TBE is a solvent that was used to make arsines as warfare agents. TBE can take the form of two isomers: 1,1,2,2-tetrabromoethane (CAS 79-27-6) and 1,1,1,2-tetrabromoethane (CAS 25167-20-8). The latter has been studied less extensively than the former.

TBE is irritating to the skin and eyes. After inhalation, 1,1,2,2-tetrabromoethane is metabolised into tri- and dibromoethylene, eliminated through the lungs, and into dibromoacetic acid, glyoxylic acid and oxalic acid, eliminated through urine and faeces (Kennedy *et al.*, 1993). A 14% to 22% fraction of TBE is retained by the body depending on the level of exposure. It should be noted that of the metabolites that form, dibromoethylene is classified as “probably carcinogenic to humans” (2A) and dibromoacetic acid as “possibly carcinogenic to humans” (2B) by the IARC.

[29] Consulted at: http://chem.sis.nlm.nih.gov/chemidplus/rn/603-32-7.
[30] Consulted at: http://hazmap.nlm.nih.gov/category-details?table=copytblagents&id=9544).

In the workplace, 1,1,2,2-tetrabromoethane has had neurotoxic, pulmonary and hepatotoxic effects after exposure by inhalation. Experimentally, the no-effect concentration by respiratory route for five animal species was 1.1 ppm, i.e. 14 $mg.m^{-3}$ after 14 weeks (Hollingsworth *et al.*, 1963 cited by Unmack J., 2010). The renal toxicity characteristic of chlorinated analogues was not observed in rats, over a short 21-day period of oral exposure to 1,1,2,2- or 1,1,1,2-TBE (NTP, 1996). A significant increase in the incidence of papillomas of the forestomach was recorded in mice after repeated dermal applications (three/week) for one year (Van Duuren *et al.*, 1979). 1,1,2,2-TBE is mutagenic (Rosenkranz, 1977). However, TBE has not been tested for carcinogenicity in standard conditions and therefore does not have an IARC classification.

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 tested concentrations, from 3 to 200 $mg.kg\ bw^{-1}.day^{-1}$. The NOAEL was 6 $mg.kg\ bw^{-1}.day^{-1}$, based on hepatotoxic effects (Hirata-Koizumi *et al.*, 2005).

In the absence of a TRV, the risks related to dietary exposure to tetrabromoethane were assessed by calculating an MOE based on the NOAEL of 6 $mg.kg\ bw^{-1}.day^{-1}$.

3.4.3.2. Vinyl bromide

Analysis of the degradation of 1,1,2,2-tetrabromoethane in soils shows that it rapidly degrades to form tri, di-, or monobrominated ethylene intermediates resulting in vinyl bromide (Patterson *et al.*, 2007). Given that vinyl bromide is a metabolite of tetrabromoethane in soil and that this substance has been classified as “probably carcinogenic to humans” (group 2A) by the IARC, the experts decided to include this compound in the HRA.

Like vinyl chloride, its structural analogue, vinyl bromide is mutagenic *in vitro* to strains of *Salmonella* Typhimurium (with or without metabolic activation) and *in vivo* to *Drosophila melanogaster*. These two vinyl halides are also clastogenic in *Drosophila* germ cells. Lastly, vinyl bromide induces DNA fragmentation *in vivo* in several organs (stomach, liver, kidneys, bladder, lungs and brain) in mice (IARC, 1986, NTP, 2014). In light of all these experimental data, it can be concluded that vinyl bromide has genotoxic and mutagenic activity *in vivo*.

Furthermore, vinyl bromide is suspected of being carcinogenic to humans based on the induction of tumours in multiple organs in rats. Exposure to vinyl bromide in rats by inhalation leads to an increased incidence of hepatic hemangiosarcomas, Zymbal-gland[31] carcinomas, liver neoplastic nodules and hepatocellular carcinomas (NTP, 2014).

A near-unique characteristic of carcinogenesis by vinyl chloride is the induction of hepatic hemangiosarcomas, rare in animals, and the causal link established between exposure to vinyl chloride and excess risk of hepatic angiosarcomas in epidemiological studies (NTP, 1998). And yet in rats, vinyl bromide seems to be a more potent inducer of hepatic hemangiosarcomas than vinyl chloride. The fact that vinyl bromide and vinyl fluoride both induce the onset of hemangiosarcomas, rare in rat liver, and the formation of identical DNA adducts suggests a possible carcinogenesis mechanism common to these vinyl halides.

[31] Zymbal glands are modified sebaceous glands located at the base of the external ear that secrete (sebum) into the auditory canal.

In conclusion, the available data on vinyl bromide metabolism, the DNA-reactivity of its metabolites, and the spectrum of tumour induction suggest that vinyl bromide is a genotoxic carcinogen. The metabolism of vinyl bromide probably proceeds through the same pathway as that of the known human carcinogen vinyl chloride and the probable human carcinogen vinyl fluoride. The metabolism of vinyl halides results in the production of reactive metabolites that bind to proteins and nucleic acids. The three vinyl halide congeners (chloride, fluoride and bromide) are positive in genotoxicity assays. Inhalation exposure to each congener produces the same tumour spectrum and unequivocal cancer induction in rats and/or mice of both sexes.

To date, there is no official TRV available for vinyl bromide, whether for the general population or for susceptible populations. However, the available data described above clearly demonstrate similarities with vinyl chloride in terms of metabolism, and genotoxic and carcinogenic effects. Thus, by default, it seemed relevant to use the various TRVs (oral, no-threshold) for vinyl chloride for the HRA of vinyl bromide (see table in Annex 6).

The OEHHA's TRV was not used since it is based on a study on exposure by inhalation. The US EPA's TRVs of 0.75 and 1.5 $(mg.kg\ bw^{-1}.day^{-1})^{-1}$ are those used by the National Institute for Industrial Environment and Risks (INERIS) respectively for adulthood and lifetime exposure. They were preferred over RIVM's oral CR[32] of 6×10^{-4} $mg.kg\ bw^{-1}.day^{-1}$ since the US EPA extrapolated its TRV to estimate lifetime exposure using a PBPK model[33] (INERIS, 2010).

In the absence of a specific TRV, risks related to dietary exposure to vinyl bromide were assessed using the no-threshold TRVs for vinyl chloride (oral route) of 0.75 $(mg.kg\ bw^{-1}.day^{-1})^{-1}$ and 1.5 $(mg.kg\ bw^{-1}.day^{-1})^{-1}$.

3.5. Assessment of health risks related to the consumption of foodstuffs produced in the 'Clere & Schwander' complex

As explained in Section 3.1.4, the HRA was broken down into two stages. Firstly (stage 1), all foodstuffs of plant and animal origin from the 'Clere & Schwander' zone were considered. Given that the assessment showed a risk at the end of stage 1 (see results in Section 3.3.1.), the exposure scenario was then refined, considering only foodstuffs of animal origin on the one hand and only wheat on the other hand (stages 2A and 2B respectively).

3.5.1. Stage 1: Assessment *via* the consumption of foodstuffs of animal origin and plant origin produced on the 'Clere & Schwander' site

Exposure through the consumption of meat, milk, liver and wheat produced on the 'Clere & Schwander' site was first assessed for TMEs, PCDD/Fs and PAHs (see results in Annex 7). Toxicological benchmarks were exceeded for nickel and cadmium and the MOEs calculated for inorganic arsenic[34] and lead[35] were too low. Wheat products accounted for over 90% of exposure to these TMEs (see table in Annex 8). In light of these results obtained with a worst-case exposure scenario, the ERCA CES considered it was necessary to refine this exposure scenario. To do so,

[32] Oral cancer risk (CR) corresponds to an excess lifetime cancer risk with oral exposure. It is expressed as a dose, and not as a $(dose)^{-1}$, unlike an SF.
[33] Physiologically based pharmacokinetic modelling.
[34] Depending on the population (adults or children), the MOEs calculated with regard to the $BMDL_{10}$ of 0.3 to 0.8 $\mu g.kg\ bw^{-1}.day^{-1}$ (EFSA, 2014) ranged from 0.6 to 67.
[35] Regardless of the population (adults or children), the calculated MOEs were below the critical MOE of ten defined by EFSA (EFSA, 2010).

the HRA was undertaken by considering the consumption of foodstuffs of animal origin on the one hand and the consumption of wheat products on the other hand. Sections 3.1.4.2 and 3.1.4.3 describe the methodology used to undertake this HRA. The results of these HRAs are presented below.

3.5.2. Stage 2A: Exposure *via* the consumption of foodstuffs of animal origin (milk, meat, liver)

Substances in common with TDS2 (TMEs, PCDD/Fs, PCBs, PAHs)

For information, exposure levels for these substances were calculated for the total diet (milk, meat, liver from the 'Clere & Schwander' site + TDS2 normal diet) since contamination data for the normal diet are known *via* TDS2.

Firstly, the average contamination levels measured in foodstuffs of animal origin produced on the 'Clere & Schwander' site (meat, liver and milk) were compared with those measured in TDS2. The highest levels recorded in foodstuffs produced on the site appear in the tables in Annex 9. Tables comparing contamination levels for TMEs, PCDD/Fs and PAHs can be found in Annex 10.

Regarding TMEs, the average contamination levels recorded in the study were of the same order of magnitude as those of TDS2. The ERCA CES therefore considers that the consumption of milk, meat and offal produced on the 'Clere & Schwander' site is not likely to result in overexposure to TMEs in relation to the general population studied in TDS2.

As for PCBs, dioxins and furans, the average contamination levels measured in this study were slightly higher than those of TDS2, in particular for milk and liver. However, the measured levels were compliant with the maximum levels set in the regulations[36] for the three regulated parameters (sum of PCDD/Fs, sum of PCDD/Fs + DL-PCBs and sum of the six NDL-PCBs).

Regarding PAH4[37] and PAH11[38], the measured average contamination levels (according to the UB hypothesis) were higher than those measured in TDS2 for milk. It should be noted that in this study, the four PAHs were not quantified in raw milk. This difference in contamination levels can be attributed to lower analytical limits for milk in TDS2. The exposure levels calculated based on the results obtained (according to the UB hypothesis) in the present study were higher than those obtained in TDS2 (see Annex 11). A specific HRA was therefore undertaken for the Meuse site.

For PAH4, MOEs were calculated[39] based on the calculated exposure levels and the $BMDL_{10}$ of 0.34 mg.kg bw^{-1}.day^{-1} set by EFSA for the four PAHs (EFSA, 2008). The calculated MOEs were much higher than the critical MOE of 10,000 set by EFSA, regardless of the population (see Table 3). The exposure levels are therefore unlikely to be a health concern.

[36] Commission Regulation (EC) No 1881/2006 of 19 December 2006 setting maximum levels for certain contaminants in foodstuffs.
[37] Benzo(a)anthracene, benzo[a]pyrene, benzo[b]fluoranthene and chrysene. These are the four PAHs proposed by EFSA as markers of exposure to, and effect of, PAHs in food.
[38] In its 2003 report, AFSSA recommended the use of toxic equivalency factors (TEFs) based on the relative carcinogenic potency of the 11 most toxic and most representative PAHs with regards to food contamination (AFSSA, 2003).
[39] Calculated based on the ratio between exposure levels and the toxicological benchmark used. The result was compared with a critical MOE.

Table 3: Exposure levels calculated considering the consumption of milk, meat and liver (UB) as well as the normal diet and HRA results for PAH4

Population	$BMDL_{10}$	Average exposure ('Clere & Schwander' data)	95th percentile	MOE for average exposure	MOE for exposure at the 95th percentile
	(ng.kg bw^{-1}.day^{-1})				
Adults	340,000 (EFSA, 2008)	1.70	2.92	200,000	116,000
Children		2.98	7.43	114,000	45,000

For PAH11, the calculated exposure levels were at most 1.5 ng TEQ.kg bw^{-1}.day^{-1} (considering children at the 95th percentile as the UB) which is below the Virtually Safe Dose (VSD) of 5 ng TEQ.kg bw^{-1}.day^{-1} established by RIVM[40] (RIVM 2001). The exposure levels are therefore unlikely to be a health concern.

The results of the risk assessment show that for PAHs, regardless of the approach used (PAH4 or PAH11), the exposure levels are unlikely to be a health concern.

In conclusion, the consumption of foodstuffs of animal origin (meat, liver, milk) produced on this site is not likely to result in overexposure to TMEs. The PCDD/F and PCB contamination levels measured in samples of raw milk were compliant with the regulatory values. Regarding PAHs, considering the UB worst-case scenario, the results of the risk assessment show that the exposure levels are not a health concern.

Substances not considered in TDS2 (nitroaromatic explosives, arsines, brominated compounds, perchlorate)

With the exception of perchlorate ions for which quantifiable levels were measured in milk, meat and liver, and triphenylarsine for which quantifiable levels were measured in offal (liver and kidneys), none of the other substances were detected (see Annex 12). Exposure levels were therefore calculated according to the LB and UB hypotheses. All of the results of the theoretical exposure calculations can be found in Annex 13.

The HRA was undertaken by considering:

- the threshold effects of these substances on the one hand:
  - by calculating an MOE (for 2,6-DNT and tetrabromoethane);
  - by calculating the hazard quotient (HQ)[41] for the other substances;
- the no-threshold effects of these substances on the other hand, by calculating an Incremental Lifetime Cancer Risk (ILCR)[42] (for TNT, 2,4-DNT, 2,6-DNT and vinyl bromide).

The results are given in the tables of Annex 14 (for threshold effects) and Annex 15 (for no-threshold effects).

[40] An excess cancer risk of 10^{-6} could be calculated based on a VSD of 5 ng TEQ.kg bw^{-1}.day^{-1}.
[41] Calculated based on the ratio between the toxicological benchmark dose (TBMD) and exposure levels. Below one, the risk is deemed tolerable. Above one, the risk is deemed intolerable. This consists in comparing exposure levels to the TBMD used.
[42] ILCR = SF x Exposure. In this assessment, lifetime exposure was considered.

Substances with dose-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 TBMDs used for the HRA (HQs below one), regardless of the population.

For 2,6-DNT and tetrabromoethane, the lowest calculated MOEs were 24,000 and 289,000 respectively (for children – exposure at the 95th percentile as the UB). In light of these MOEs, the ERCA CES considers that exposure to 2,6-DNT and tetrabromoethane through milk, liver and meat produced on the 'Clere & Schwander' site is unlikely to be a health concern.

Substances with no-dose-threshold effects

For TNT, an ILCR slightly above 10^{-6} was calculated (for children, considering exposure at P95 as the UB). For other populations, the ILCRs were below 10^{-6}. For 2,4-DNT, 2,6-DNT and vinyl bromide, ILCRs above 10^{-5} were calculated for all the study populations; the highest ILCR was calculated for 2,6-DNT in children at the 95th percentile (as the UB) with a value of 2.5×10^{-4}.

3.5.3. Stage 2B: Exposure through the consumption of wheat

Substances in common with TDS2 (TMEs, PCDD/Fs, PCBs, PAHs)

First of all, it should be noted that the Pb and Cd levels measured in wheat were compliant with the regulatory levels[43]. For other substances for which regulatory levels in wheat have not been set out, an HRA was undertaken.

Exposure levels were calculated according to the LB and UB hypotheses, considering average wheat consumption (125 g.day^{-1} for adults and 80 g.day^{-1} for children). The results can be found in Annex 16.

The toxicological benchmarks were not exceeded for PCDD/Fs, PCBs, or for the following elements: Zn, Hg (considered in inorganic form), Ni, Cu, Co, Sb and Al. For PAH4, the calculated exposure levels were at most 1.72 ng.kg bw^{-1}.day^{-1} (considering children at the 95th percentile as the UB). Thus, the calculated MOE for the four PAHs was 198,000 with regard to the $BMDL_{10}$ of 0.34 mg.kg bw^{-1}.day^{-1} set by EFSA. Regarding PAH11, the calculated exposure levels were at most 1.11 ng TEQ.kg bw^{-1}.day^{-1} (considering children at the 95th percentile as the UB), which is below the VSD of five ng TEQ.kg bw^{-1}.day^{-1} established by RIVM (RIVM, 2001). Thus, for all of these substances, exposure through the consumption of wheat products is unlikely to be a health concern.

Lastly, for inorganic arsenic, MOEs were calculated. Depending on the scenario and the population in question, the MOEs calculated with regard to the $BMDL_{10}$ (0.3 to 8 µg.kg bw^{-1}.day^{-1}) ranged from eight to 665. The average exposure levels calculated for adults and children in this study accounted for 5% and 7% of the average exposure levels calculated in TDS2 for adults and children. Lastly, although not quantified in the three composite samples taken randomly (Belp method, 1986), it should be noted that arsenic was quantified in one 'hot spot' sample (0.01 ± 0.04 mg.kg^{-1}, see Annex 9).

[43] 0.20 mg.kg^{-1} for Pb and 0.10 mg.kg^{-1} for Cd (Regulation (EC) No 1881/2006).

Table 4: Exposure levels calculated considering wheat consumption (UB) and results of the MOE calculations undertaken with regard to the $BMDL_{10}$ of 0.3 and 8 µg.kg $bw^{-1}.day^{-1}$

Population	$BMDL_{10}$	Average exposure ('Clere & Schwander' data)	95th percentile	MOE for average exposure	MOE for exposure at the 95th percentile
	($\mu g.kg\ bw^{-1}.day^{-1}$)				
Adults	0.3 – 8 (EFSA, 2014)	0.012	0.016	25 - 665	19 - 505
Children		0.020	0.038	15 - 400	8 - 215

Substances not considered in TDS2 (nitroaromatic explosives, arsines, brominated compounds, perchlorate ions)

None of these substances were detected in wheat (see Annex 12). Exposure levels were therefore calculated according to the LB and UB hypotheses, considering average wheat consumption (125 $g.day^{-1}$ for adults and 80 $g.day^{-1}$ for children). All of the results of the theoretical exposure calculations can be found in Annex 17. The results of the HRA are given in the tables in Annex 18 (for dose-threshold effects) and Annex 19 (for no-dose-threshold effects).

Dose-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 TBMDs used for the HRA (HQs below one), regardless of the population and regardless of the level of wheat consumption.

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, the ERCA CES considers that exposure to 2,6-DNT and tetrabromoethane through wheat produced on the 'Clere & Schwander' site is unlikely to be a health concern.

No-dose-threshold effects

For TNT, ILCRs of approximately 10^{-8} to 10^{-7} were calculated. As for 2,4-DNT, 2,6-DNT and vinyl bromide, ILCRs slightly higher than 10^{-5} were calculated for all the study populations.

3.6. Conclusions of the ERCA CES

The conclusions of the ERCA CES are based on the HRAs undertaken, taking into account foodstuffs of animal origin on the one hand and wheat on the other hand (respectively stages 2A and 2B described above).

The CES would like to emphasise that:

- These assessments were undertaken by taking into account analytical results obtained for a given period on fully identified sites. These analytical data are therefore considered specific point data and it does not appear possible to transpose the results of these HRAs to other situations, whether spatially or temporally,

- For some substances not studied in TDS2, there is no TRV, and in this case, toxicological benchmarks were selected by default (e.g. the TTC was used for triphenylarsine and the TRV for vinyl chloride was extrapolated to vinyl bromide),
- No analytical assays were undertaken in drinking water; therefore, no HRAs were undertaken with this matrix. That said, there could be specific quantitative and/or qualitative contamination (e.g. potential presence of thiodiglycol, which is a metabolite of sulphur mustard) in water.

3.6.1. Foodstuffs of animal origin

For foodstuffs of animal origin, an HRA was undertaken, considering the consumption of milk, meat and liver (stage 2A).

For TMEs, PCDD/Fs and PAHs (substances studied in TDS2), the HRA shows that the exposure levels calculated *via* the consumption of foodstuffs of animal origin produced on the site are unlikely to be a health concern.

With regard to the threshold effects of TNT, 2,4-DNT, 2,6-DNT, 2-ADNT, 4-ADNT, diphenylarsinic acid, triphenylarsine, tetrabromoethane and perchlorate ions, the calculated exposure levels are unlikely to be a health concern.

In order to assess the risk of no-threshold effects occurring with TNT, 2,6-DNT and 2,4-DNT, ILCRs were calculated. For TNT, a maximum ILCR of 10^{-6} was calculated (for children, considering exposure at the 95th percentile). However, for 2,4-DNT, 2,6-DNT and vinyl bromide, ILCRs slightly higher than 10^{-5} were calculated for all the study populations.

However, it should be reiterated that the HRA was undertaken using a worst-case scenario with the following terms:

- It was assumed that individuals consume, for their entire lifetime, only foodstuffs of animal origin produced on the 'Clere & Schwander' site;
- No factor was considered for the dilution of these foodstuffs during industrial processing;
- For substances not studied in TDS2, contamination levels for dairy products (butter, cheese and ultra-fresh dairy) were estimated by using dairy equivalents, which can end up maximising the exposure levels calculated *via* the consumption of foodstuffs of animal origin;
- Levels for nitroaromatic substances and arsines and ILCRs were therefore calculated based on a UB scenario maximising contamination.

In light of the above results, the ERCA CES considers that the consumption of foodstuffs of animal origin produced on the 'Clere & Schwander' site is unlikely to be a health concern.

3.6.2. Foodstuffs of plant origin

The plant products covered in this study were wheat, barley and maize silage.

3.6.2.1. Barley and maize silage

Several data were lacking to quantitatively assess the risk related to the consumption of barley and maize silage (uncertainties related to toxicology, the spatial variability of levels in soil, and soil-plant, plant-animal and soil-animal transfer rates).

Nonetheless, levels of diphenylarsinic acid (whose genotoxic effects cannot be ruled out) and TNT (a genotoxic carcinogen) in barley demonstrated soil-to-plant transfer on the studied plots. As a result, in the absence of new data, the experts recommend not using these contaminated plots for agricultural purposes for human food or animal feed.

Levels of triphenylarsine (of up to 8.07 µg.kg^{-1}) were demonstrated in maize silage. Data on the metabolism of this compound in production livestock are lacking and those generated in this study cannot be used to estimate transfer rates between animal feed and foodstuffs of animal origin[44]. It therefore does not appear possible to determine concentrations of triphenylarsine in foodstuffs of animal origin after exclusive consumption of maize silage by production livestock.

It should be noted that these results are consistent with the soil analyses undertaken by the BRGM in 2015 (January and July) and demonstrate that soil-to-plant transfer is possible on the contaminated plots. In fact, quantifiable levels of nitroaromatic compounds and triphenylarsine were found in most of the soil samples taken from the studied barley plot. Likewise, the soil analyses undertaken by the BRGM on the maize plot showed quantifiable levels of nitroaromatic compounds and triphenylarsine.

3.6.2.2. Wheat

For wheat, an HRA was undertaken, considering the consumption of wheat products (stage 2B).

Substances in common with TDS2 (TMEs, PCDD/Fs and PAHs)

For inorganic arsenic, MOEs were calculated. Depending on the scenario and the population in question, the MOEs calculated with regard to the $BMDL_{10}$ ranged from eight to 665 and were of the same order of magnitude as those calculated in TDS2. For other TMEs, PCDD/Fs and PAHs, the calculated exposure levels are unlikely to be a health concern.

Substances not considered in TDS2 (nitroaromatic explosives, arsines, brominated compounds, perchlorate ions)

None of these substances were detected in wheat. An HRA was nonetheless undertaken for all of these substances (LB and UB scenarios). With regard to the threshold effects of these substances (TNT, 2,4-DNT, 2,6-DNT, 2-ADNT, 4-ADNT, diphenylarsinic acid, triphenylarsine, tetrabromoethane and perchlorate ions), the calculated exposure levels are unlikely to be a health concern. In order to assess the risk of no-threshold effects occurring, ILCRs were calculated for TNT, 2,6-DNT, 2,4-DNT and vinyl bromide. For TNT, a maximum ILCR of 10^{-7} was calculated (for children, considering exposure at the 95th percentile). However, for 2,4-DNT, 2,6-DNT and vinyl bromide, ILCRs slightly higher than 10^{-5} were calculated for all the study populations.

On the other hand, arsenic concentrations in the wheat samples taken according to the method described by Belp (1986) were below the limit of quantification and no traces of explosives or toxic

[44] Due in particular to a lack of knowledge of feed consumed by animals for which contamination data for meat are available.

warfare agents were detected. Moreover, the scenario considered to calculate exposure levels *via* the consumption of wheat and wheat products appears conservative. It was assumed that all wheat products (bread, pastries, biscuits, etc.) were prepared only from wheat produced on the 'Clere & Schwander' site. Therefore, the CES considers that the batch of wheat produced on this site is unlikely to pose a health risk. Regarding the hot spots[45] detected on certain wheat plots, and considering the source of contamination, it is likely that they are not randomly distributed. Therefore, the CES recommends locating and mapping the hot spots, in order to determine the relevance of defining a zone within which wheat should not be grown.

4. AGENCY CONCLUSIONS AND RECOMMENDATIONS

ANSES endorses the conclusions of the ERCA CES and the 'Meuse site' WG.

These conclusions rule out any risk related to the consumption of foodstuffs of animal origin and the consumption of wheat, but do not rule out the risk related to the consumption of barley and maize. These conclusions are valid only for the products that were sampled.

The Agency reiterates, in addition to these conclusions, the need to provide a more comprehensive picture of contamination for the various ecosystems potentially impacted by activities for shell disposal and the destruction of munitions stockpiled following World War I. This work should consist in particular of gaining a better picture of contamination levels in soils and in surface water or even in drinking water.

These additional investigations are essential, to increase the robustness of the HRA conclusion on the one hand, and to improve the management of agricultural production zones on the other hand. In this context, it should be noted that the Agency's work should not be considered a general conclusion as to the absence of risks for future agricultural products, in particular plant products from potentially contaminated zones.

Roger GENET

[45] Reminder: these are highly contaminated (by arsenic in particular) zones that were determined by the BRGM based on soil analyses.

KEYWORDS

Chemical weapons in warfare - explosives - polluted sites and soils.

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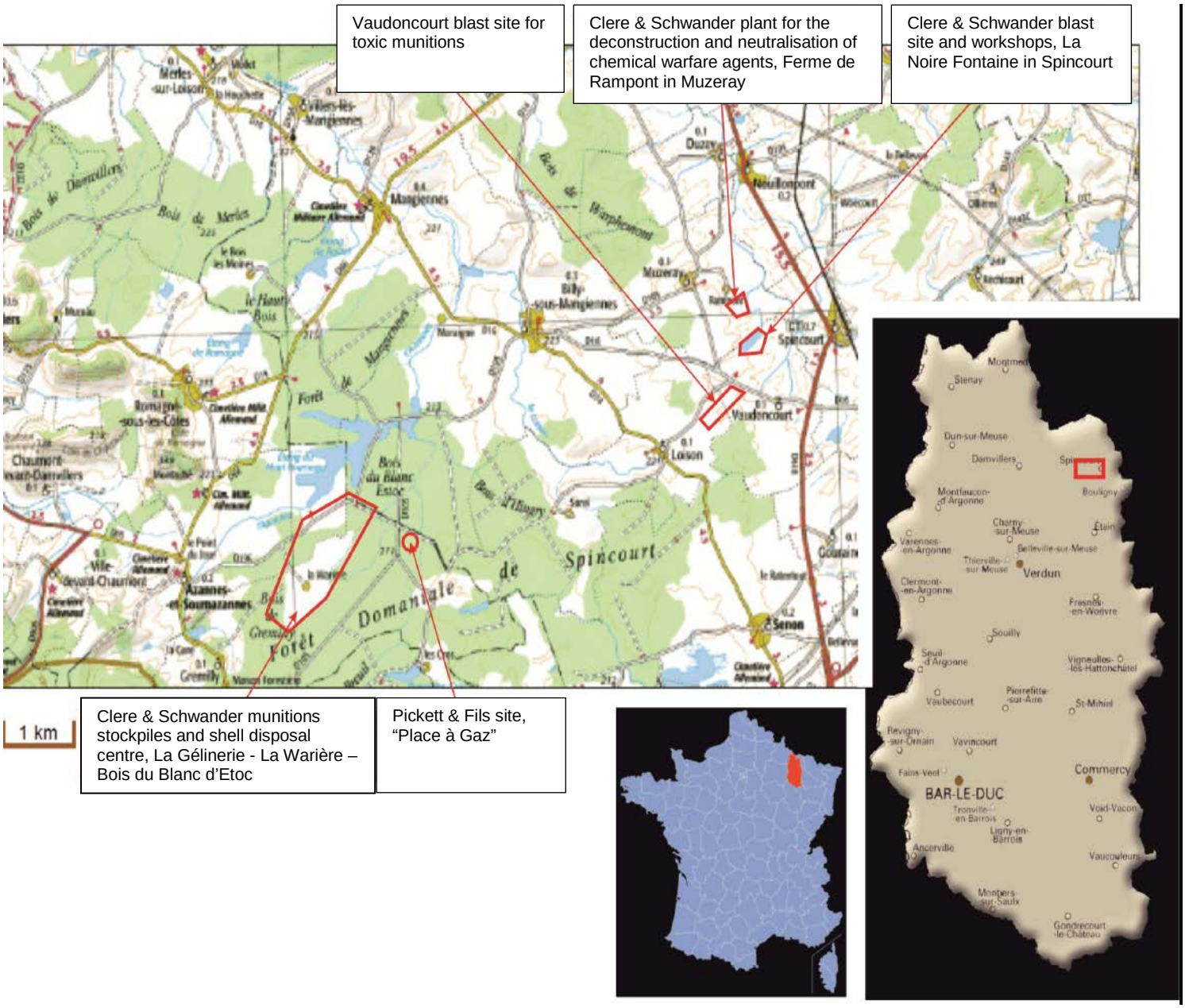
ANNEXES

Annex 1: Map of the 'Clere & Schwander' shell disposal complex



Annex 2: Summary of the toxicological benchmarks selected to undertake the HRA

Category of substances	Substances	Toxicological benchmark type - Source	Toxicological benchmark value	Study, critical effect	HRA approach
War explosives	TNT	SF (US EPA, 1989)	0.03 (mg.kg bw^{-1}.day^{-1})$^{-1}$	2 years, rats, oral route Combined urinary tract tumours (US-DOD, 1984)	Calculation of an ILCR
		RfD (US EPA, 1989) (ATSDR, 1995)	0.5 µg.kg bw^{-1}.day^{-1}	26 weeks, dogs, oral route Hepatic effects (Levine *et al.*, 1990)	Calculation of an HQ
	2,4 DNT	SF (OEHHA, 2005)	0.31 (mg.kg bw^{-1}.day^{-1})$^{-1}$	2 years, rats, oral route Combined liver and mammary gland tumours (US-DOD, 1979)	Calculation of an ILCR
		RfD (US EPA, 1992)	2 µg.kg bw^{-1}.day^{-1}	2 years, dogs, oral route Haematological effects (Ellis *et al.*, 1985)	Calculation of an HQ
	2,6 DNT	LOAEL (US EPA, 2013)	4 mg.kg bw^{-1}.day^{-1}	13 weeks, dogs, oral route Haematological effects (US Army, 1976)	Calculation of an MOE
		Provisional no-threshold TRV (US EPA, 2013)	1.5 (mg.kg bw^{-1}.day^{-1})$^{-1}$	1 year, rats, oral route Hepatocellular carcinomas (Leonard *et al.*, 1987)	Calculation of an ILCR
	4-ADNT and 6-ADNT	Threshold TRV (US EPA, 1992)	2 µg.kg bw^{-1}.day^{-1}	Read-across with 2,4 DNT	
	Perchlorate ions	Threshold TRV (ANSES, 2011)	0.7 µg.kg bw^{-1}.day^{-1}	14 days, humans, oral route Decrease in iodine uptake	Calculation of an HQ
Chemical warfare agents	Diphenylarsinic acid (DPAA)	TTC	1.5 µg.kg bw^{-1}.day^{-1}	Use of the TTC approach - threshold set for substances placed in Cramer Class III	
	Triphenylarsine (TPA)				
TMEs	Zinc	Tolerable upper intake level	25 mg.day^{-1} (adults) 10 mg.day^{-1} (4-6 years)		

Category of substances	Substances	Toxicological benchmark type - Source	Toxicological benchmark value	Study, critical effect	HRA approach
		(SCF, 2002)	13 $mg.day^{-1}$ (7-10 years) 18 $mg.day^{-1}$ (11-14 years) 22 $mg.day^{-1}$ (15-17 years)		
	Inorganic arsenic (As_i)	$BMDL_{01}$ (EFSA, 2009a)	0.3 - 8 µg.kg $bw^{-1}.day^{-1}$	Epidemiological study Lung, bladder and skin cancers	Calculation of an MOE Critical MOE not defined Taking into account the speciation hypotheses recommended by EFSA (EFSA, 2014) [46]
	Lead (Pb)	'Dietary intake value' (EFSA, 2010)	0.63 µg.kg $bw^{-1}.day^{-1}$	Value calculated from a critical blood-lead level based on nephrotoxic effects in human adults	HRA undertaken based on the two toxicological benchmarks Calculation of an MOE Critical MOE = 10
		'Dietary intake value' (HCSP, 2014).	0.5 µg.kg $bw^{-1}.day^{-1}$	Value calculated from a critical blood-lead level based on neurodevelopmental effects in human adults	
	Cadmium (Cd)	TWI (EFSA, 2009b)	2.5 µg.kg $bw^{-1}.week^{-1}$	Epidemiological studies - Nephrotoxicity	
	Methylmercury (Me-Hg)	PTWI (EFSA, 2012b)	1.3 µg.kg $bw^{-1}.week^{-1}$	Epidemiological studies – Neurodevelopmental toxicity	Taking into account the speciation hypotheses recommended by EFSA

[46] Speciation hypotheses for foods considered in this study: 70% of arsenic in inorganic form

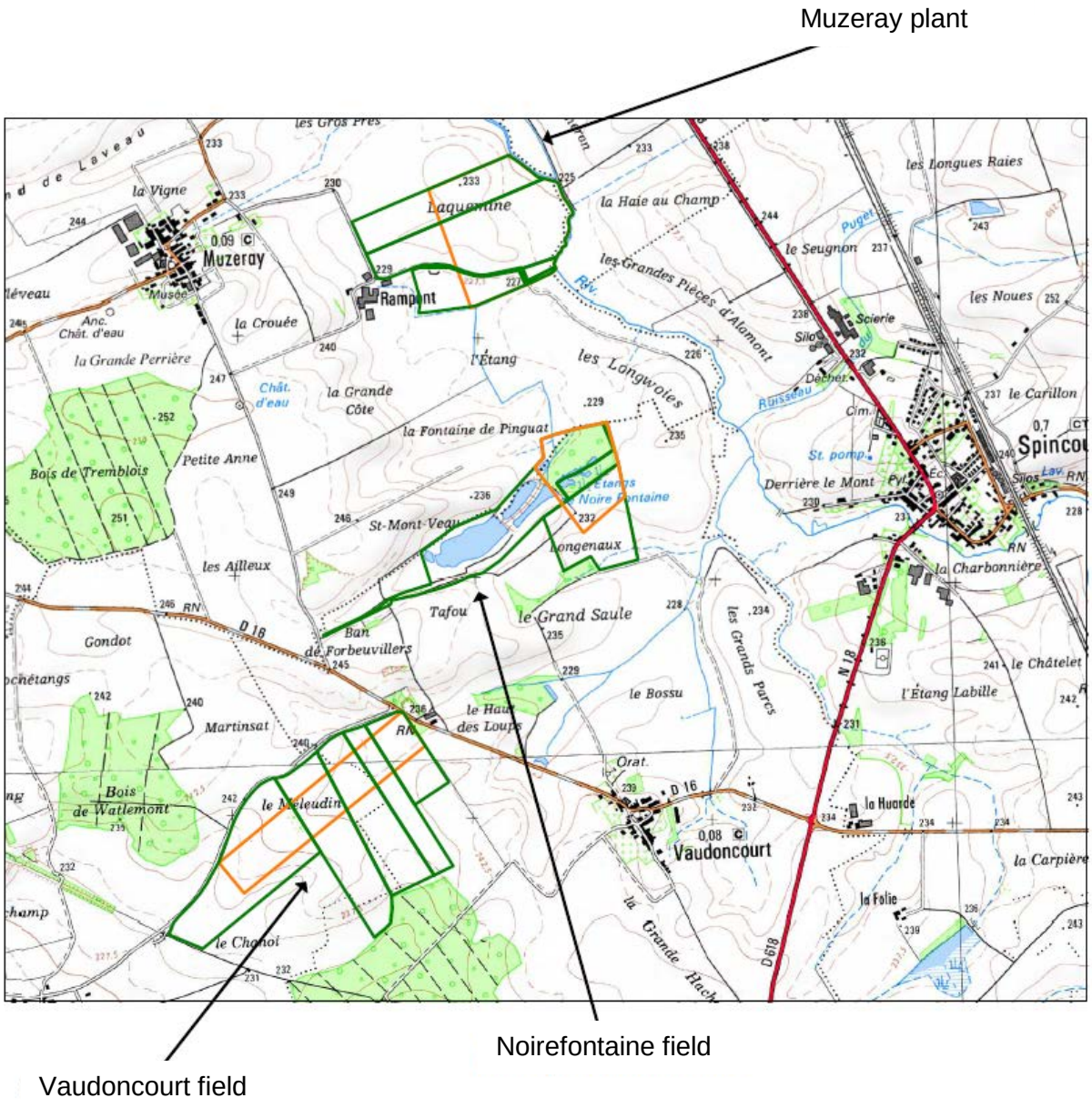
Category of substances	Substances	Toxicological benchmark type - Source	Toxicological benchmark value	Study, critical effect	HRA approach
	Inorganic mercury	PTWI (JECFA, 2011)	4 µg.kg bw^{-1}.$week^{-1}$	6 months, rats Nephrotoxic effects	(EFSA, 2012b)[47]
	Aluminium (Al)	PTWI (JECFA, 2006)	1 mg.kg bw^{-1}.$week^{-1}$	Rats Developmental toxicity	
	Cobalt (Co)	TDI (AFSSA, 2010)	1.6 µg.kg bw^{-1}.day^{-1}	Human exposure, 22 days - Polycythaemia	
	Copper (Cu)	USL (SCF, 2006)	5 mg.day^{-1}		
	Tin (Sn)	In the absence of speciation hypotheses and robust TRVs for the inorganic forms of tin, an HRA could not be undertaken for tin			
TMEs	Antimony (Sb)	TDI (WHO, 2003)	6 µg.kg bw^{-1}.day^{-1}	90 days, rats	
	Nickel (Ni)	TDI (EFSA, 2015)	2.8 µg.kg bw^{-1}.day^{-1}	2 generations, rats, oral route Developmental toxicity	
Other pollutants	PAHs	PAH4: $BMDL_{10}$ (EFSA, 2008)	PAH4: $BMDL_{10}$ = 0.34 mg.kg bw^{-1}.day^{-1}	2 years, mice, oral route Combined tumours in several organs	HRA undertaken using the two approaches
		PAH11: VSD (AFSSA, 2003)	PAH11: VSD = 5 ng.kg bw^{-1}.day^{-1}	2 years, rats, gavage (benzo[a]pyrene) Tumours (primarily the liver and forestomach)	
	PCDD/Fs	TRV (EPA, 2012)	0.7 pg WHO TEQ.kg bw^{-1}.day^{-1}	Epidemiological studies – Reprotoxicity	
	PCBs	TDI	10 ng.kg bw^{-1}.day^{-1} for	Monkeys,	

[47] Speciation hypotheses for foods considered in this study: 100% of mercury in the form of inorganic mercury

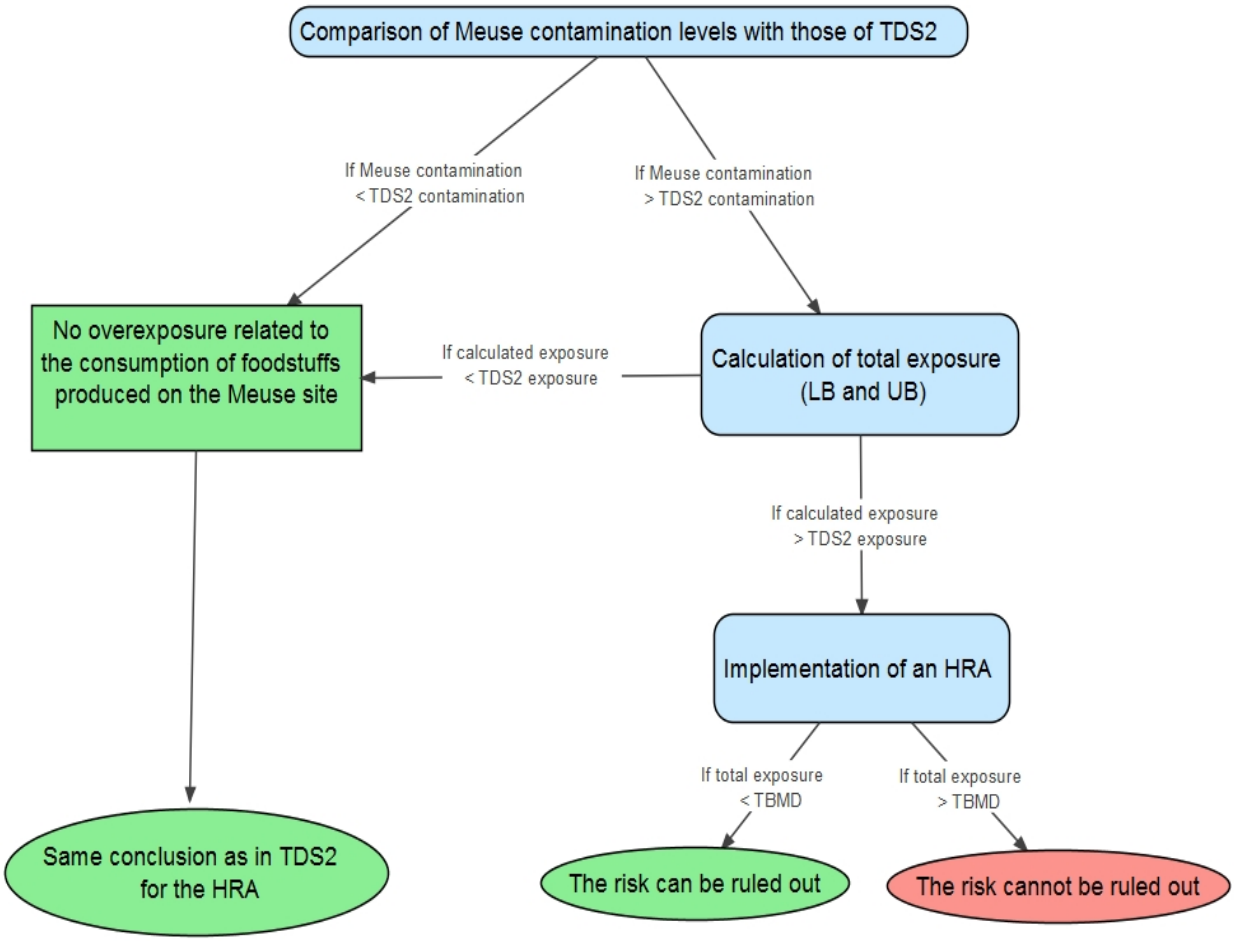
Category of substances	Substances	Toxicological benchmark type - Source	Toxicological benchmark value	Study, critical effect	HRA approach
		(AFSSA, 2007)	the six indicator NDL-PCBs[48]	Neurotoxicity	
	Tetrabromoethane (TBE)	NOAEL	6 mg.kg bw^{-1}.day^{-1}	28 days, rats, oral route Hepatotoxicity (Hirata-Koizumi *et al.*, 2005).	Calculation of an MOE Critical MOE = 10,000
	Vinyl bromide	SF for vinyl chloride (US EPA, 2010)	**0.75 (mg.kg bw^{-1}.day^{-1})$^{-1}$** (for exposure during adulthood) **1.5 (mg.kg bw^{-1}.day^{-1})$^{-1}$** (for lifetime exposure since birth)	Rats, 140 weeks, oral route Hepatocellular tumours (Feron *et al.*, 1981)	Calculation of an ILCR

[48] Six NDL-PCBs: PCB-28, 52, 101, 138, 153 and 180

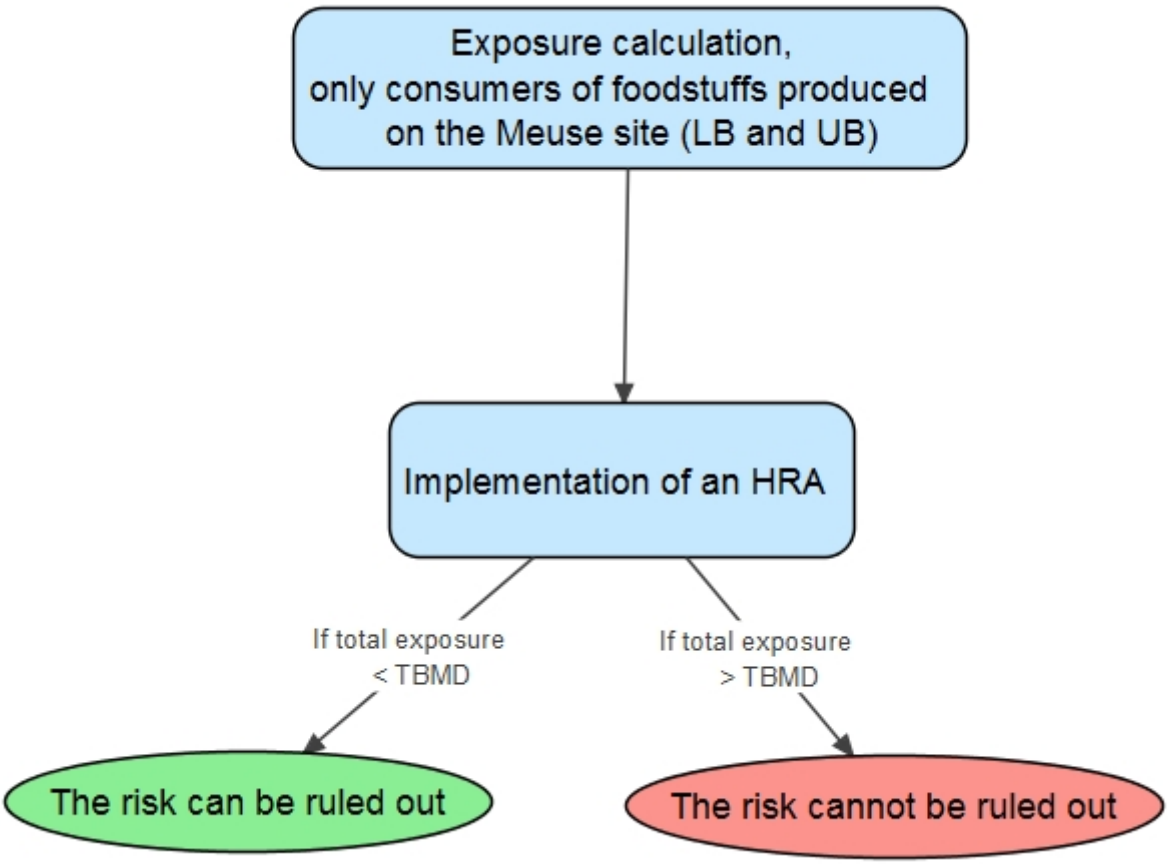
Annex 3: Locations of agricultural plots



Annex 4: Flowchart describing the approach used for the assessment of health risks related to the consumption of foodstuffs of animal origin – For substances considered in TDS2



Annex 5: Flowchart describing the approach used for the assessment of health risks related to the consumption of foodstuffs of animal origin – For substances not considered in TDS2



Annex 6: TRVs, no-threshold effects of vinyl chloride (oral route)

Organisation	Type of TRV	Value	Target organ/Critical effect
(US-EPA, 2000)	SF[49]	**0.75 (mg.kg bw^{-1}.day^{-1})$^{-1}$** (for exposure during adulthood) **1.5 (mg.kg bw^{-1}.day^{-1})$^{-1}$** (for lifetime exposure since birth)	Liver angiosarcomas, hepatocellular carcinomas and neoplastic nodules (Feron *et al.*, 1981)
(RIVM, 1999)	Oral cancer risk (CR)[50] (dose corresponding to an excess risk of 10^{-4})	6 x 10^{-4} mg.kg bw^{-1}.day^{-1}	Hepatocellular tumours (Feron *et al.*, 1981)
(OEHHA, 2010)	SF	0.27 (mg.kg bw^{-1}.day^{-1})$^{-1}$	Lung carcinomas. Value established based on a study on exposure by inhalation in mice (Drew *et al.*, 1983)

It should be noted that the US EPA also established a threshold TRV for oral exposure to vinyl chloride of 0.003 mg.kg bw^{-1}.day^{-1} based on hepatotoxic effects. However, this TRV was not used given that vinyl chloride is a genotoxic carcinogen.

[49] Values derived from a $BMDL_{10}$. These values are more conservative than those obtained by the US EPA with the linearised multistage (LMS) model.
[50] Oral CR corresponds to an excess lifetime cancer risk with oral exposure. It is expressed as a dose, and not as a (dose)$^{-1}$, unlike an SF.

Annex 7: Exposure levels calculated taking into account the consumption of milk, meat, liver and wheat produced on the 'Clere & Schwander' site, considering wheat consumption at the 95th percentile

Exposure levels calculated *via* milk + meat + wheat (considering wheat consumption at the 95th percentile)									
Substances	Units	Adults (n=77)				Children (n=59)			
		LB		UB		LB		UB	
		Avg exp	P95 exp	Avg exp	P95 exp	Avg exp	P95 exp	Avg exp	P95 exp
PCDD/Fs	pg.kg bw^{-1}.day^{-1}	0.06	0.11	0.07	0.13	0.23	0.60	0.26	0.69
PAH11	ng.kg bw^{-1}.day^{-1}	0	0	0.70	0.99	0.00	0.01	1.72	3.73
PAH4	ng kg bw^{-1}.day^{-1}	0	0	1.08	1.53	0	0	2.66	5.80
Six NDL-PCBs	ng.kg bw^{-1}.day^{-1}	0.20	0.40	0.20	0.40	0.76	2.23	0.76	2.23
Pb	µg.kg bw^{-1}.day^{-1}	0.13	0.17	0.13	0.17	0.27	0.52	0.28	0.55
Sn	µg.kg bw^{-1}.day^{-1}	0	0	0.07	0.17	0	0	0.30	0.87
Zn	mg.day^{-1}	18.19	21.43	18.19	21.43	16.23	18.61	16.23	18.61
Cd	µg.kg bw^{-1}.week^{-1}	1.27	1.72	1.28	1.72	2.75	5.34	2.75	5.34
Hg	µg.kg bw^{-1}.week^{-1}	0	0	0.25	0.35	0	0	0.62	1.40
Total As	µg.kg bw^{-1}.day^{-1}	0.16	0.22	0.16	0.22	0.36	0.74	0.36	0.74
Inorganic As	µg.kg bw^{-1}.day^{-1}	0.12	0.15	0.12	0.15	0.25	0.52	0.25	0.52
Cu	mg.day^{-1}	1.68	1.67	1.68	1.67	1.47	1.46	1.47	1.46
Co	µg.kg bw^{-1}.day^{-1}	0	0.00	0.03	0.05	0.01	0.01	0.08	0.17
Ni	µg.kg bw^{-1}.day^{-1}	1.18	1.58	1.22	1.66	2.50	4.85	2.67	5.36
Al	mg.kg bw^{-1}.week^{-1}	0.12	0.16	0.12	0.16	0.25	0.49	0.26	0.50
Sb	µg.kg bw^{-1}.day^{-1}	0	0	0.03	0.04	0	0	0.07	0.14

Annex 8: Contribution of foodstuffs of animal origin and wheat to exposure to TMEs, PCDD/Fs and PAHs. Exposure scenario taking into account the consumption of milk, meat, liver and wheat produced on the 'Clere & Schwander' site, considering wheat consumption at the 95th percentile

	Adults		Children	
Substances	Contribution of wheat	Contribution of foodstuffs of animal origin	Contribution of wheat	Contribution of foodstuffs of animal origin
PCDD/Fs	64%	36%	38%	62%
PAH11	93%	7%	79%	21%
PAH4	92%	8%	79%	21%
Six NDL-PCBs	41%	59%	23%	77%
Pb	98%	2%	96%	4%
Sn	42%	58%	22%	78%
Zn	88%	12%	85%	15%
Cd	99%	1%	97%	3%
Hg	88%	12%	74%	26%
Total As	95%	5%	91%	9%
Cu	95%	5%	93%	7%
Co	91%	9%	85%	15%
Ni	97%	3%	94%	6%
Al	99%	1%	99%	1%
Sb	98%	2%	96%	4%

Annex 9: Maximum contamination levels observed in foodstuffs produced on the 'Clere & Schwander' site (when the results are <LD or <LQ, the respective values of the LDs and LQs are given in parentheses)

	TMEs (mg/kg) – Maximum levels										
Matrix	**Pb[(1)]**	**Cd[(2)]**	**Hg**	**As**	**Zn**	**Cu**	**Sn**	**Co**	**Ni**	**Al**	**Sb**
Milk (n=2)	<LD (0.001)	0.0012 ± 0.0002	<LD (0.003)	0.002 ± 0.001	3.92 ± 1.57	0.074 ± 0.018	<LD (0.031)	<LD (0.001)	<LD (0.019)	<LQ (0.063)	<LD (0.0004)
Muscle (n=9)	<LQ (0.003)	<LD (0.0003)	<LD (0.005)	0.014 ± 0.002	47.5 ± 13.4	0.775 ± 0.132	<LD (0.05)	0.003 ± 0.001	<LD (0.03)	<LQ (0.1)	<LD (0.0006)
Liver (n=9)	0.065 ± 0.007	0.063 ± 0.013	<LD (0.005)	0.029 ± 0.007	133.5 ± 37.8	123 ± 30	<LD (0.05)	0.095 ± 0.019	0.103 ± 0.031	0.321 ± 0.077	0.002 ± 0.0004
Kidneys (n=9)	0.072 ± 0.012	0.410 ± 0.082	0.010 ± 0.003	0.202 ± 0.048	25.6 ± 7.2	4.04 ± 0.97	<LD (0.05)	0.041 ± 0.008	<LQ (0.06)	0.672 ± 0.114	<LD (0.0006)
Wheat	0.029 ± 0.012	0.044 ± 0.014	<LQ (0.01)	0.01 ± 0.04 [(3)]	52 ± 21	5.1 ± 2.1	<LQ (0.01)	<LQ (0.01)	0.27 ± 0.11	3.9 ± 1.6	<LQ (0.01)
Barley	0.051 ± 0.02	0.018 ± 0.006	<LQ (0.01)	1.5 ± 0.6	72 ± 29	5.8 ± 2.4	0.020 ± 0.008	0.022 ± 0.009	0.61 ± 0.25	19.9 ± 8	<LQ (0.01)
Maize silage	0.36 ± 0.15	0.19 ± 0.06	0.17 ± 0.07	0.06 ± 0.02	180 ± 72	101 ± 41	4.6 ± 1.9	0.04 ± 0.016	0.33 ± 0.14	0.39 ± 0.16	0.075 ± 0.03

(1) Values compliant with the regulatory limits set for foodstuffs: 0.020 mg/kg for raw milk, 0.010 mg/kg for meat, 0.50 mg/kg for offal and 0.20 mg/kg for cereals (Regulation (EC) No 1881/2006).
(2) Values compliant with the regulatory limits set for foodstuffs: 0.050 mg/kg for meat, 0.50 mg/kg for liver, 1 mg/kg for kidneys and 0.10 mg/kg for cereals (Regulation (EC) No 1881/2006).
(3) Arsenic was quantified in one 'hot spot' wheat sample and was not quantified in the three composite samples (LQ = 0.01 mg/kg).

	PCDD/Fs and PCBs				PAH4 (benzo(a)anthracene, benzo[a]pyrene, benzo[b]fluoranthene and chrysene)	PAH11 [4]
	Sum of PCDD/Fs (WHO TEQ 2005) [1]	**Sum of the six NDL-PCBs** *(PCB28, PCB52, PCB101, PCB138, PCB153 and PCB180)* [2]	**Sum of DL-PCBs (WHO TEQ 2005)**	**Sum of DL-PCBs + PCDD/Fs (WHO TEQ 2005)** [3]		
Milk (n=2)	0.206 ± 0.036 pg/g fat = 0.0072 pg/g wet weight	1.912 ± 0.434 ng/g fat = 0.0701 ng/g wet weight	0.332 ± 0.068 pg/g fat = 0.0120 pg/g wet weight	0.538 ± 0.104 pg/g fat = 0.0192 pg/g wet weight	<LQ (0.08 µg/kg wet weight)	0.05 µg TEQ/kg wet weight
Muscle (n=9)	0.256 ± 0.045 pg/g fat = 0.010 pg/g wet weight	4.384 ± 0.995 ng/g fat = 0.181 ng/g wet weight	0.255 ± 0.052 pg/g fat = 0.010 ng/g wet weight	0.511 ± 0.097 pg/g fat = 0.020 pg/g wet weight	<LQ (0.07 µg/kg wet weight)	0.04 µg TEQ/kg wet weight
Liver (n=9)	0.065 ± 0.012 pg/g wet weight	0.574 ± 0.130 ng/g wet weight	0.071 ± 0.015 pg/g wet weight	0.136 ± 0.026 pg/g wet weight	<LQ (0.07 µg/kg wet weight)	0.05 µg TEQ/kg wet weight
Kidneys (n=9)	0.365 ± 0.064 pg/g fat = 0.012 pg/g wet weight	2.572 ± 0.584 ng/g fat = 0.090 ng/g wet weight	0.350 ± 0.072 pg/g fat = 0.012 pg/g wet weight	0.715 ± 0.135 pg/g fat = 0.024 pg/g wet weight	<LQ (0.07 µg/kg wet weight)	0.04 µg TEQ/kg wet weight
Wheat	0.009 ± 0.001 ng/kg	0.015 ± 0.003 µg/kg	0.0024 ± 0.0005 ng/kg	0.011 ± 0.002 ng/kg	0.32 µg/kg wet weight	0.21 µg TEQ/kg wet weight
Barley	0.010 ± 0.002 ng/kg	0.034 ± 0.008 µg/kg	0.004 ± 0.001 ng/kg	0.014 ± 0.003 ng/kg	0.33 µg/kg wet weight	0.20 µg TEQ/kg wet weight
Maize silage	0.030 ± 0.005 ng/kg	0.128 ± 0.029 µg/kg	0.017 ± 0.004 ng/kg	0.047 ± 0.009 ng/kg	2.32 µg/kg wet weight	0.89 µg TEQ/kg wet weight

(1) Values compliant with the regulatory limits set for foodstuffs: 2.5 pg/g fat for raw milk and meat and 0.30 pg/g wet weight for liver (Regulation (EC) No 1881/2006)

(2) Values compliant with the regulatory limits set for foodstuffs: 5.5 pg/g fat for raw milk, 4 pg/g fat for meat and 0.50 pg/g wet weight for liver (Regulation (EC) No 1881/2006)

(3) Values compliant with the regulatory limits set for foodstuffs: 40 ng/g fat for raw milk and meat and 3ng/g wet weight for liver (Regulation (EC) No 1881/2006)
(4) Weighted sum (TEF, WHO 1998) for the 11 following congeners: Anthracene, Benzo(a)anthracene, Benzo(a)pyrene, Benzo(b)fluoranthene, Benzo(g,h,i)perylene, Benzo(j)fluoranthene, Benzo(k)fluoranthene, Chrysene, DiBenzo(a,h)anthracene, Fluoranthene, Indeno(1,2,3-cd)pyrene

Annex 10: Comparison of the average contamination levels obtained in foodstuffs of animal origin produced on the 'Clere & Schwander' site with the average contamination levels measured in the TDS2 study (UB values)

	TMEs (mg/kg) – Average values – UB hypothesis										
Matrix	**Pb**	**Cd**	**Hg**	**As**	**Zn**	**Cu**	**Sn***	**Co**	**Ni**	**Al**	**Sb**
'Clere & Schwander' milk (n=2)	0.001	0.0008	0.003	0.003	3.77	0.061	0.031	0.001	0.019	0.047	0.0004
TDS2 milk	0.0056	0.0011	0.005	0.012	3.73	0.09	0.011	0.0036	0.036	0.68	0.0006
'Clere & Schwander' beef muscle (n=9)	0.002	0.0003	0.005	0.011	45.3	0.791	0.05	0.003	0.033	0.072	0.0006
TDS2 beef (UB nat avg)	0.011	0.0014	0.005	0.025	53.89	0.759	0.0164	0.0079	0.0584	0.63	0.0015
'Clere & Schwander' liver (n=9)	0.028	0.043	0.005	0.026	85.6	105.5	0.05	0.077	0.042	0.168	0.001
TDS2 liver (UB nat avg)	0.02	0.0526	0.005	0.020	64.01	112.72	0.0181	0.0906	0.0769	0.584	0.0018

*substance not detected in any samples

	PCDD/Fs and PCBs – Average values – UB hypothesis				PAH4 (benzo(a)anthracene, benzo[a]pyrene, benzo[b]fluoranthene and chrysene)	PAH11
	Sum of PCDD/Fs (WHO TEQ 2005)	**Sum of the six NDL-PCBs** *(PCB28, PCB52, PCB101, PCB138, PCB153 and PCB180)*	**Sum of DL-PCBs (WHO TEQ 2005)**	**Sum of DL-PCBs + PCDD/Fs (WHO TEQ 2005)**		
'Clere & Schwander' milk (n=2)	0.006 pg/g wet weight	0.063 ng/g wet weight	0.010 pg/g wet weight	0.0163 pg/g wet weight	0.08 µg/kg wet weight	0.05 µg TEQ/kg wet weight
TDS2 milk (nat avg)	0.010 pg/g wet weight	0.044 ng/g wet weight	0.010 pg/g wet weight	0.010 pg/g wet weight	0.006 µg/kg wet weight	0.005 µg TEQ/kg wet weight
'Clere & Schwander' meat (n=32)	0.006 pg/g wet weight	0.067 ng/g wet weight	0.007 pg/g wet weight	0.014 pg/g wet weight	0.07 µg/kg wet weight	0.04 µg TEQ/kg wet weight
TDS2 beef (UB nat avg)	0.02 pg/g wet weight	0.235 ng/g wet weight	0.02 pg/g wet weight	0.05 pg/g wet weight	0.071 µg/kg wet weight	0.035 µg TEQ/kg wet weight
'Clere & Schwander' liver (n=32)	0.040 pg/g wet weight	0.409 ng/g wet weight	0.055 pg/g wet weight	0.092 pg/g wet weight	0.07 µg/kg wet weight	0.05 µg TEQ/kg wet weight
TDS2 liver (nat avg)	0.08 pg/g wet weight	0.262 ng/g wet weight	0.06 pg/g wet weight	0.14 pg/g wet weight	0.064 µg/kg wet weight	0.032 µg TEQ/kg wet weight

Annex 11: Results of exposure calculations for the four PAHs and 11 PAHs considering intake *via* foodstuffs of animal origin produced on the 'Clere & Schwander' site and the normal diet, and comparison with the exposure levels obtained in TDS2

All foodstuffs of animal origin produced on the site (milk, meat, offal) were considered for these calculations.

					LB scenario		UB scenario	
Substance	**Unit**	**Population**	**N**	**Food**	**avg**	**P95**	**avg**	**P95**
PAH4	ng.kg bw^{-1}.day^{-1}	Adults	77	Normal diet	1.468	2.788	1.608	2.858
PAH4	ng.kg bw^{-1}.day^{-1}	Adults	77	beef	0	0	0.026	0.074
PAH4	ng.kg bw^{-1}.day^{-1}	Adults	77	liver	0	0	0	0
PAH4	ng.kg bw^{-1}.day^{-1}	Adults	77	milk	0	0	0.053	0.246
PAH4	ng.kg bw^{-1}.day^{-1}	Adults	77	veal	0	0	0.005	0.025
PAH4	**ng.kg bw^{-1}.day^{-1}**	**Adults**	**77**	**Total**	**1.468**	**2.788**	**1.693**	**2.918**
PAH4 TDS2 - Adults (MB scenario)					**1.478**	**2.998**	**1.478**	**2.998**
PAH4	ng.kg bw^{-1}.day^{-1}	Children	59	Normal diet	2.164	5.525	2.418	6.129
PAH4	ng.kg bw^{-1}.day^{-1}	Children	59	beef	0	0	0.045	0.179
PAH4	ng.kg bw^{-1}.day^{-1}	Children	59	liver	0	0	0.002	0
PAH4	ng.kg bw^{-1}.day^{-1}	Children	59	milk	0	0	0.506	1.539
PAH4	ng.kg bw^{-1}.day^{-1}	Children	59	veal	0	0	0.006	0.044
PAH4	**ng.kg bw^{-1}.day^{-1}**	**Children**	**59**	**Total**	**2.164**	**5.525**	**2.977**	**7.433**
PAH4 TDS2 - Children (MB scenario)					***2.259***	***4.694***	***2.259***	***4.694***
PAH11	ng.kg bw^{-1}.day^{-1}	Adults	77	Normal diet	0.288	0.570	0.431	0.681
PAH11	ng.kg bw^{-1}.day^{-1}	Adults	77	beef	0	0.001	0.015	0.042
PAH11	ng.kg bw^{-1}.day^{-1}	Adults	77	liver	0	0	0	0
PAH11	ng.kg bw^{-1}.day^{-1}	Adults	77	milk	0	0.001	0.034	0.158
PAH11	ng.kg bw^{-1}.day^{-1}	Adults	77	veal	0	0	0.003	0.013
PAH11	**ng.kg bw^{-1}.day^{-1}**	**Adults**	**77**	**Total**	**0.288**	**0.571**	**0.482**	**0.742**
PAH11 TDS2 - Adults					**0.346**	**0.66**	**0.43**	**0.767**
PAH11	ng.kg bw^{-1}.day^{-1}	Children	59	Normal diet	0.419	1.189	0.699	1.701
PAH11	ng.kg bw^{-1}.day^{-1}	Children	59	beef	0.001	0.002	0.026	0.101
PAH11	ng.kg bw^{-1}.day^{-1}	Children	59	liver	0	0	0.001	0

PAH11	ng.kg bw^{-1}.day^{-1}	Children	59	milk	0.001	0.003	0.324	0.987
PAH11	ng.kg bw^{-1}.day^{-1}	Children	59	veal	0	0	0.003	0.024
PAH11	**ng.kg bw^{-1}.day^{-1}**	**Children**	**59**	**Total**	**0.421**	**1.192**	**1.053**	**2.724**
PAH11 TDS2 - Children					**0.548**	**1.131**	**0.680**	**1.349**

Annex 12: Maximum contamination levels measured in foodstuffs produced on the 'Clere & Schwander' site for nitroaromatic explosives, brominated compounds, perchlorate and arsines

Foodstuff	Explosives (in µg.kg^{-1})					Arsines		Brominated compounds (in µg.kg^{-1})		Perchlorates (in µg.kg^{-1})
	2,4-DNT	2,6-DNT	TNT	2-ADNT	4-ADNT	TPA + TPA oxide[51] (in µg.kg^{-1})	DPAA (in µg.kg^{-1})	TBE[52]	Vinyl bromide	
Milk (n=2)	<LD (10)	<LD (4)	<LD (1)	<LD (1)	<LD (1)	<LQ (0.03)	< LQ (1)	<LQ (0.5)	<LQ (1)	6.3 ± 3.1
Muscle (n=32)	<LD (10)	<LD (4)	<LD (1)	<LD (1)	<LD (1)	<LQ (0.03)	< LQ (2)	<LQ (0.5)	<LQ (1)	11 ± 6
Liver (n=32)	<LD (10)	<LD (4)	<LD (1)	<LD (1)	<LD (1)	0.14	< LQ (2)	<LQ (0.5)	<LQ (1)	21 ± 12
Kidneys (n=32)	<LD (10)	<LD (4)	<LD (1)	<LD (1)	<LD (1)	0.03	< LQ (2)	<LQ (0.5)	<LQ (1)	2.9 ± 1.8
Wheat	<LD (10)	<LD (4)	<LD (1)	<LD (1)	<LD (1)	<LQ (0.10)	< LQ (5)	< LQ (1)	< LQ (3)	<LD (0.5)
Barley	<LD (10)	<LD (4)	<LQ* (4)	<LD (1)	<LD (1)	0.87	23**	< LQ (1)	< LQ (3)	<LD (0.5)
Maize silage	<LD (50)	<LD (20)	<LD (5)	<LD (5)	<LD (5)	8.07	< LQ (5)	< LQ (1)	< LQ (3)	7.5 ± 4.4

* TNT was detected in only one sample of barley taken from a hot spot. This compound was not detected in any other samples.
** Diphenylarsinic acid was quantified in three samples taken from hot spots.

[51] The analysis showed that triphenylarsine is easily oxidised. Therefore, the two compounds were systematically tested for.
[52] During analysis, tetrabromoethane broke down to tribromoethene. Tribromoethene was systematically tested for with an LQ of 0.5 µg/kg.

Annex 13: Exposure levels calculated for nitroaromatic explosives, brominated compounds, perchlorate and arsines, taking into account milk, meat and liver produced on the 'Clere & Schwander' site as well as other dairy products that could be made from this milk (butter, ultra-fresh dairy, cheese)

Substances	Exposure levels calculated *via* dairy products and foodstuffs of animal origin (in ng.kg bw^{-1}.day^{-1})							
	Children (n=59)				Adults (n=77)			
	LB		UB		LB		UB	
	Avg exp	P95 exp	Avg exp	P95 exp	Avg exp	P95 exp	Avg exp	P95 exp
TNT	0	0	17.0	41.7	0	0	9.51	18.3
2,4-DNT	0	0	169.8	416.6	0	0	95.1	182.6
2,6-DNT	0	0	67.9	166.6	0	0	38.0	73.1
2-ADNT + 4-ADNT[53]	0	0	34.0	83.3	0	0	19.0	36.5
Diphenylarsinic acid	0	0	17.7	44.7	0	0	9.95	18.8
Triphenylarsine	0	0	0.509	1.250	0	0	0.285	0.548
Vinyl bromide	0	0	17.0	41.7	0	0	9.5	18.3
Tetrabromoethane	0	0	8.5	20.8	0	0	4.8	9.1
Perchlorate	165.1	414.4	165.1	414.4	92.7	175.7	92.7	175.7

[53] 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.

Annex 14: Results of the HQ and MOE calculations (substances with dose-threshold effects). Scenario taking into account the consumption of milk, meat and liver produced on the 'Clere & Schwander' site as well as other dairy products that could be made from this milk (butter, ultra-fresh dairy, cheese)

Substances	HRA - threshold effects - HQ or MOE calculation				Calculation type	Threshold TMBD in ng.kg $bw^{-1}.day^{-1}$
	Children		Adults			
	Average exposure	Exposure at the 95th percentile	Average exposure	Exposure at the 95th percentile		
TNT	0.034	0.083	0.019	0.083	HQ	500
2,4-DNT	0.085	0.208	0.048	0.208	HQ	2,000
2,6-DNT	58,900	24,005	105,193	54,760	MOE	4,000,000
2-ADNT + 4-ADNT[54]	0.017	0.041	0.009	0.042	HQ	2,000
Diphenylarsinic acid	0.012	0.030	0.007	0.030	HQ	1,500
Triphenylarsines	3.40×10^{-4}	8.33×10^{-4}	1.90×10^{-4}	8.33×10^{-4}	HQ	1,500
Vinyl bromide	/	/	/	/	...	/
Tetrabromoethane	706,795	288,066	1,262,312	657,118	MOE	6,000,000
Perchlorate ions	0.236	0.592	0.132	0.592	HQ	700

[54] 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.

Annex 15: Results of the ILCR calculations (substances with no-dose-threshold effects). Scenario taking into account the consumption of milk, meat and liver produced on the 'Clere & Schwander' site as well as other dairy products that could be made from this milk (butter, ultra-fresh dairy, cheese)

Substances	HRA - no-threshold effects - ILCR calculation				SF (in $ng.kg\ bw^{-1}.day^{-1})^{-1}$
	Children		Adults		
	Average exposure	P95 exposure	Average exposure	P95 exposure	
TNT	5.09×10^{-7}	1.25×10^{-6}	2.85×10^{-7}	5.48×10^{-7}	3×10^{-8}
2,4-DNT	5.26×10^{-5}	1.29×10^{-4}	2.95×10^{-5}	5.66×10^{-5}	3.1×10^{-7}
2,6-DNT	1.02×10^{-4}	2.50×10^{-4}	5.70×10^{-5}	1.10×10^{-4}	1.5×10^{-6}
Vinyl bromide	2.55×10^{-5}	6.25×10^{-5}	1.43×10^{-5}	2.74×10^{-5}	1.5×10^{-6}

Annex 16: Results of calculations of exposure *via* wheat consumption (TMEs, PCBs and PAHs), considering average wheat consumption (125 g.day^{-1} for adults and 80 g.day^{-1} for children)

Exposure levels calculated *via* wheat									
Substances	Units	Adults (n=77)				Children (n=59)			
		LB		UB		LB		UB	
		Avg exp	P95 exp	Avg exp	P95 exp	Avg exp	P95 exp	Avg exp	P95 exp
PCDD/Fs	pg.kg bw^{-1}.day^{-1}	0.02	0.03	0.03	0.04	0.04	0.07	0.04	0.08
PAH11	ng.kg bw^{-1}.day^{-1}	0	0	0.37	0.49	0	0	0.58	1.11
PAH4	ng.kg bw^{-1}.day^{-1}	0	0	0.57	0.75	0	0	0.89	1.72
Six NDL-PCBs	ng.kg bw^{-1}.day^{-1}	0.05	0.06	0.05	0.06	0.07	0.14	0.07	0.14
Pb	µg.kg bw^{-1}.day^{-1}	0.07	0.10	0.07	0.10	0.11	0.22	0.11	0.22
Sn	µg.kg bw^{-1}.day^{-1}	0	0	0.02	0.02	0	0	0.03	0.05
Zn	mg.day^{-1}	9.13	9.13	9.13	9.13	5.84	5.84	5.84	5.84
Cd	µg.kg bw^{-1}.week^{-1}	0.72	0.96	0.72	0.96	1.13	2.18	1.13	2.18
Hg[55]	µg.kg bw^{-1}.week^{-1}	0	0	0.12	0.17	0	0	0.19	0.38
Total As	µg.kg bw^{-1}.day^{-1}	0.018	0.024	0.018	0.024	0.028	0.054	0.054	0.054
Inorganic As[56]	µg.kg bw^{-1}.day^{-1}	0.012	0.016	0.012	0.016	0.020	0.038	0.038	0.038
Cu	mg.day^{-1}	0.90	0.90	0.90	0.90	0.58	0.58	0.58	0.58
Co	µg.kg bw^{-1}.day^{-1}	0	0	0.02	0.02	0	0	0.03	0.05
Ni	µg.kg bw^{-1}.day^{-1}	0.67	0.90	0.67	0.90	1.05	2.04	1.05	2.04
Al	mg.kg bw^{-1}.week^{-1}	0.07	0.09	0.07	0.09	0.11	0.21	0.11	0.21
Sb	µg.kg bw^{-1}.day^{-1}	0	0	0.02	0.02	0	0	0.03	0.05

[55] Speciation hypothesis used: 100% of total mercury is considered to be in inorganic form (EFSA, 2012)
[56] Speciation hypothesis used: 70% of total arsenic is considered to be in inorganic form (EFSA, 2014)

Annex 17: Exposure levels calculated for nitroaromatic explosives, brominated compounds, perchlorate and arsines, taking into account wheat produced on the 'Clere & Schwander' site

Substances	Exposure levels calculated *via* wheat and wheat products (in ng.kg bw^{-1}.day^{-1})							
	Children (n=59)				Adults (n=77)			
	LB		UB		LB		UB	
	Avg exp	P95 exp	Avg exp	P95 exp	Avg exp	P95 exp	Avg exp	P95 exp
TNT	0	0	2.8	5.4	0	0	1.77	2.36
2,4-DNT	0	0	27.7	53.7	0	0	17.7	23.6
2,6-DNT	0	0	11.1	21.5	0	0	7.1	9.4
2-ADNT + 4-ADNT[57]	0	0	5.5	10.7	0	0	3.5	4.7
Diphenylarsinic acid	0	0	13.9	26.9	0	0	8.9	11.8
Triphenylarsine	0	0	0.277	0.537	0	0	0.177	0.236
Vinyl bromide	0	0	8.3	16.1	0	0	5.3	7.1
Tetrabromoethane	0	0	2.8	5.4	0	0	1.8	2.4
Perchlorates	0	0	1.4	2.7	0	0	0.89	1.18

[57] 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.

Annex 18: Results of the HQ and MOE calculations (substances with dose-threshold effects). Scenario taking into account the consumption of wheat produced on the 'Clere & Schwander' site

Substances	HRA - threshold effects - HQ or MOE calculation				Calculation type	Threshold TMBD in ng.kg $bw^{-1}.day^{-1}$
	Children		Adults			
	Average exposure	Exposure at the 95th percentile	Average exposure	Exposure at the 95th percentile		
TNT	0.006	0.011	0.004	0.011	HQ	500
2,4-DNT	0.014	0.027	0.009	0.027	HQ	2,000
2,6-DNT	> 350,000	> 150,000	> 550,000	> 400,000	MOE	4,000,000
2-ADNT + 4-ADNT[58]	0.003	0.005	0.002	0.005	HQ	2,000
Diphenylarsinic acid	0.009	0.018	0.006	0.018	HQ	1,500
Triphenylarsines	1.85×10^{-4}	3.58×10^{-4}	1.18×10^{-4}	3.58×10^{-4}	HQ	1,500
Tetrabromoethane	> 2,000,000	> 1,000,000	> 3,000,000	> 2,500,000	MOE	6,000,000
Perchlorate ions	0.002	0.004	0.001	0.004	HQ	700

[58] 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.

Annex 19: Results of the ILCR calculations (substances with no-dose-threshold effects). Scenario taking into account the consumption of wheat produced on the 'Clere & Schwander' site

Substances	HRA - no-threshold effects - ILCR calculation				SF (in ng.kg $bw^{-1}.day^{-1})^{-1}$
	Children		Adults		
	Average exposure	P95 exposure	Average exposure	P95 exposure	
TNT	8.31×10^{-8}	1.61×10^{-7}	5.31×10^{-8}	7.08×10^{-8}	3×10^{-8}
2,4-DNT	8.59×10^{-6}	1.66×10^{-5}	5.49×10^{-6}	7.31×10^{-6}	3.1×10^{-7}
2,6-DNT	1.66×10^{-5}	3.22×10^{-5}	1.06×10^{-5}	1.42×10^{-5}	1.5×10^{-6}
Vinyl bromide	1.25×10^{-5}	2.42×10^{-5}	7.96×10^{-6}	1.06×10^{-5}	1.5×10^{-6}