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Preprint · April 2024

DOI: 10.13140/RG.2.2.21010.88006

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Lessons learnt on exposure assessment process after a chemical incident: case study of the contamination patterns of acrylonitrile

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ABSTRACT

Background: A train which was transporting chemicals derailed and exploded on the 4th of May, 2013 in Wetteren, Belgium. **Objective:** To describe the trends of air distribution and concentration of acrylonitrile (ACN) in order to determine environmental contamination in the areas exposed to ACN. **Methods:** Ambient air monitoring was used to describe the exposure of ACN. Samples of ACN air concentrations from indoor and outdoor locations were collected during the three weeks following the train accident. A series of maps showing the distribution and concentration, and thus exposure hotspots, were produced with ArcGIS. Statistical hypothesis tests were used to establish whether differences existed between the ACN air concentration samples collected from these places. Potential risk levels were defined according to the “Intervention Values for Emergency Response” (French and Dutch limit values were available and used in 2013). **Results:** Of the 3006 geo-referenced samples, four areas presented high and alarming levels of ACN concentrations in the air (> 90 ppm) namely, near the train accident, in the sewers and nearby the Waste Water Treatment Plant (WWTP). Polluted environments which were categorised as having an immediate risk level were in sewers leading from the site of the train accident to the WWTP through the city (330 ppm), directly above manhole covers (196 ppm) and in private bathrooms and lavatories (98 ppm). Findings showed peaks of ACN concentration up to seventeen days after the release of the chemical as well as at a distance from the train accident. **Discussion and conclusion:** The data description analysis provides further information about the demarcation of risk areas and the routes of ACN distribution. Besides air contamination, water was a significant pathway for ACN and water must therefore be considered during the process of exposure assessment. The distribution of ACN concentrations collected in this study, which was based on environmental monitoring, were in line with previous studies conducted on human biomonitoring. The results are able to determine an anticipatory approach directly focusing on the identification of environmental areas at risk during a chemical exposure and to show that individuals were exposed to high levels of concentration in various places.

Key-words: exposure assessment, human health risk assessment, integrated approach, chemical accident, exposure pathways

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Contamination patterns of acrylonitrile exposure after a major chemical incident

1. INTRODUCTION

A train which was transporting chemicals including acrylonitrile (ACN), butadiene and triethylaluminum derailed and exploded on the 4th of May 2013 in Wetteren, Belgium. The largest quantity of one chemical product transported was ACN. Considerable amounts of water were used by rescue workers to control and prevent an explosion, which eventually resulted in water entering the sewer network crossing the city. More than 2000 residents, who were living nearby the train accident and along the sewers, were evacuated, and thousands of emergency professionals were called in. Several individuals complained and/or suffered from health symptoms and one resident, who was living along the sewer trajectory, died (Van Nieuwenhuyse, et al. 2014; De Smedt, et al. 2014; Simons, et al. 2016). This accident which had an environmental impact highlights the importance of assessing exposure during and following chemical accidents to monitor environmental contamination. In this context, air quality monitoring and human biomonitoring (HBM) studies were set up to assess the exposure of the local population, emergency responders and staff members working during the disaster.

ACN (C_3H_3N , also known as vinyl cyanide) is used as a monomer for the manufacturing of acrylic fibres, polystyrenes and adhesives. The main source of ACN exposure is tobacco smoke (De Jesús et al., 2021; Luo et al., 2020). At room temperature, it is a volatile, flammable, colourless and water-soluble liquid with a weak odour (PubChem, 2019; European Commission, 2004). This chemical can polymerize violently and form uninhibited monomer vapours. By considering this aspect, the Boiling Liquid Expanding Vapour Explosion (BLEVE) is an important indicator to consider in this case study. ACN may explode when it is stored in confined spaces, following a build-up of pressure in tanks, which may lead to a sudden rupture (Ning et al., 2011). The overpressure in the tank is the major hazard of BLEVE, leading to a fireball radiant heat and resulting in a blast (INERIS, 2017). Therefore, effective ventilation systems should be guaranteed during any ACN transport. The impact of the explosion results in human exposure mainly through inhalation. A biomarker for exposure to ACN is *N*-2-cyanoethylvaline (CEV), which is derived from a reaction of ACN with the *N*-terminal valine in haemoglobin. Hence, ACN exposure can be monitored in the blood for up to 4 months (126 days), which corresponds to the time it takes to fully regenerate red blood cells (Bader et al., 2014; Törnqvist et al., 1986). The CEV concentration normally decrease by 1/126 (or 0.8% per day). Importantly, human metabolism also results in the formation of cyanide. Depending on the extent of exposure, health consequences may vary mild irritation of the respiratory tract to fatigue, nausea and vomiting and to potentially convulsions, coma and even death. Table 1 summarises the Toxicological Reference Values (TRV) and health effects of ACN. The general population may be exposed to ACN through industrial emissions in the air, for example from incinerators (> 95% of human exposure) and from a Waste Water Treatment Plant (WWTP) (Carex, 2016). ACN has been classified as a possible carcinogen to humans by the International Agency for Research on Cancer (group 2B since 1999) and by the European Union (1B since 2008).

Table 1: Literature search of chemical properties to qualify the human health risk related to ACN exposures

Proprieties	Mean (range values)	References
BLEVE risk	Yes	Long G (WHO), 2002; Ning 2011
Water solubility at 25°C (g/litre)	73	INERIS 2008
Solubility	Miscible with organic solvents	Toxnet 2004 (American Cyanamid Co., 1959)

Half-life ($t_{1/2}$)		
air (h)	55 to 96	WHO, 2002
water (h)	170	WHO, 2002
soil (h)	170	WHO, 2002
sediment (h)	550	WHO, 2002
ACN found in general WWTP	0.1 mg/L	INERIS, 2008
TRV* Health effects		
Acute effects (human)		
Nonlethal effects (nervous system symptoms)	16-100 ppm (20-45min)	NRC, 2014 Wilson et al. 1948
IDLH**	85 ppm (1h)	Schwanecke 1966, OSHA, 2001, Ca 2007
Threshold (lethal value)	99.66 ppm (1h)	RIVM, 2015
	90 ppm (1h)	Dutch, 2013
	139 ppm (1h)	INERIS, 2008
Threshold (irreversible effects value)	58.89 ppm (1h)	RIVM, 2015
	22.67 ppm (1h)	Dutch, 2013
	22 ppm (1h)	INERIS, 2008
No symptoms of exposure	4.53 ppm (1h)	Dutch, 2013
	1.49 ppm (8h)	RIVM, 2015
Acute effects (animals)		
Nonlethal effects (nervous system symptoms)	8.6 ppm (8h)	RIVM, 2015
Threshold (lethal value)	13.59 ppm (8h)	RIVM, 2015
Chronic effects (human)		
Inhalation reference exposure level	$5.00 \times 10^{-3} \text{ mg/m}^3$	OEHHA, 2001
Reference Concentration	$2.00 \times 10^{-3} \text{ mg/m}^3$	US EPA, 1991
Unit Risk for Cancer	$6.8 \times 10^{-5} (\mu\text{g/m}^3)^{-1}$	US EPA, 1991
Unit Risk (at $1\mu\text{g/m}^3$)	$2.00 \times 10^{-5} (\mu\text{g/m}^3)$	WHO, 2017

* Toxicological Reference Values

** Immediately dangerous to life or health concentration, as defined by the National Institute for Occupational Safety and Health (NIOSH, US)

During and in the aftermath of the incident in Wetteren, a significant amount of data and information was collected by the professionals involved. Within a short timeframe, decisions had to be taken to identify the groups at risk and to rapidly take protective measures, such as setting up an evacuation action plan (Huizer et al., 2014; Heinälä et al., 2013) and performing environmental monitoring. According to the World Health Organization (WHO), Risk Assessment process allows the identification of adequate and informed decisions by policy makers and provides guidance in conducting a human risk assessment (Ritter et al. 2002; WHO, 2016). Although in the context of an incident, exposure assessment must be considered as an emergency response, more research is still needed to frame it for environmental disaster management (Hunault 2014; Svendsen et al. 2016). The purposes were (1) to explore the contact of the population to polluted atmosphere along with contaminated indoor and outdoor environments/materials, (2) to define the exposure routes considering the potential dispersion of ACN into the environment, and (3) and demonstrate that the linear model do not take account the multiple exposure during a chemical incident.

2. MATERIALS AND METHODS

2.1. Hazard identification: Environmental monitoring and data collection

To determine accurately the exposure levels and its duration, we questioned individuals involved during and after the incident, and we compiled and analysed all reports available. These documents were provided by the Belgium Crisis Centre of the Federal Public Service of the Interior and from the fire brigades.

Air samplings were performed with Dräger CDS kits, which provide rapid information on chemicals present in the environment. These essentially consist of a pump and colorimetric detector tubes, which deliver instant measurements. Once the main chemicals were identified, further quantification of the ACN concentration was performed by a photoionization detector, with a sensor at 11.7 eV (knowing that the ionization potential of ACN is 10.94 eV). These instruments provide a real-time data response and analysis. The same instruments were used for both indoor and outdoor ACN air measurements from the 5th to 24th of May, 2013. Data collection was organized at various locations around the train accident. The sampling protocol was adjusted according to the prevailing weather conditions (e.g. wind direction retrieved from nearby weather station).

2.2. Exposure descriptor: Delineation of areas

The explosion happened upstream from the city, in a green and sparsely populated area. Figure 2 shows the area that was investigated during the biomonitoring study and the rescue areas from where residents evacuated (De Smedt, et al. 2014; Simons, et al. 2016). In the authors' analysis, De Smedt et al. (2014) selected the study population based on the delineation by the Crisis Management Cell. The different zones corresponded to 1) the 250m perimeter of the evacuation zone which was determined hours immediately following the accident and to 2) a zone which included the streets parallel to the sewage system as well as the streets downwind from the train accident. In contrast to this study, the delineation areas in this paper have been determined by the environmental monitoring, in which ACN levels in the air (ppm) were objectified and quantified.

2.3. Human biomonitoring and data collection

Human biomonitoring campaigns were carried out between May 18–25 (i.e. days 14 till 21 after the train accident) and September 25-26 and 30th (i.e. days 144 till 149 after the train accident) with the assistance of the local general practitioners and the physicians of the Federal Public Service Health, Food Chain Safety and Environment. During the first HBM campaign, blood was analysed to identify and quantify N-2-cyanoethylvaline (CEV), a specific biomarker of ACN exposure. Knowing that ACN is present in tobacco smoke, and therefore the potential influence of the cotinine in the interpretation of CEV measurements, an urine sample was also collected (Van Nieuwenhuysse et al., 2014). A short questionnaire was also filled by 242 residents participants included (i) demographic variables, i.e. name, address, gender, day, month and year of birth; (ii) lifestyle variables, i.e. smoking status (non-smoker, ex-smoker, occasional smoker or daily smoker); and (iii) some specific variables related to the sampling, i.e. the day and the hour at which blood and urine sampling took place (De Smedt et al., 2014). During the second campaign of HBM, the follow-up concerned only 59 residents with CEV above the reference value which was 10 pmol/g globin for no-smokers and 200 pmol/g globin for smokers. The study protocol was approved by the Ethical Committee of Ghent University Hospital and an informed consent was signed by all participants prior to their participation in the study (De Smedt et al., 2014; Simons et al., 2016; Van Nieuwenhuysse et al., 2014).

2.4. Data analysis

The present study is a retrospective analysis of environmental data collected from May 4th to May 24th and HBM data from May 18th to 25th and the follow-up from September 25 to 30th, 2013 after a major chemical incident. Trends of exposures within a defined population and area were described (Figure 2) in the following steps.

Step 1 Environmental descriptive analysis: Compilation of available reports allowed to assign the missing information on air sampling to approximate location and hours of ACN measurements. Variables of interest in the environment were time, sampling locations and corresponding ACN concentrations in the air. CEV and cotinine levels were analysed from the HBM. We proceeded by geocoding ACN concentrations monitored in the sampling locations, namely houses, public buildings, WWTP, sewers, trucks and streets. Additional variables were created to distinguish whether the areas exposed were indoors or outdoors and the pathways of contamination by ACN. We used Google Maps and ArcGIS 10.3 for mapping data.

Step 2 Analysis by mapping environmental and health data: Health risk estimation was based on the intervention reference values for ACN in the environment available and used during the incident (in 2013). Categorisations were made to characterize different areas by referring to the “Intervention Values for Emergency Response”. In this sense, we categorized ACN concentrations expressed in ppm by referring to Dutch intervention values for one-hour exposure. Both TRV from RIVM and INERIS present a similar ACN concentration for the “risk level” (RIVM 2013; INERIS 2008).

Table 2: Intervention reference values for ACN in the environment (RIVM 2013; INERIS 2008):

	RIVM (2013)		INERIS (2008)
Safety threshold (ST)	< 4 ppm		< 2 ppm
Vigilance level (VL)	4 ≤ ppm ≤ 21	(10 mg/m ³ = 4.53 ppm)	> 17 ppm
Risk level (RL)	22 ≤ ppm ≤ 90	(50 mg/m ³ = 22.67 ppm)	> 22 * ppm
Immediate hazard level (IHL)	> 90 ppm	(200 mg/m ³ = 90.68 ppm)	

*INERIS TRV corresponding to an “Irreversible effects” (1 hour of exposure).

Association analysis between environmental and health data were based on CEV concentrations in blood and values measured after D-126. This approach referred to a linear model with the expression $CEV = \text{measured CEV} / (1 - t \times 0.008)$, where “t” is the number of days between the accident and the blood sampling (Granath et al., 1992; Simons et al., 2016).

Extrapolated CEV = CEV measured during the first campaign of HBM / (1 - 0,008) * Δt (Day of sampling D₀ – 4may T₀)

Reference values used in this analysis were for 10 pmol/g globin (Kraus et al., 2012), and smokers: 100 pmol/g globin (De Smedt et al., 2014).

Step 3 Descriptive analysis: we recoded variables of interest to evaluate the mean distributions across indoor and outdoor locations. Secondly, we recoded a new variable within the objective to quantify the ACN concentration in building (houses). For all variables, we excluded “undefined places” during statistical tests.

New variables created	included
Indoor	WWTP, sewage disposal, train, private houses, lavatories, public buildings
Outdoor	Streets, sewer systems
Houses	Private houses, lavatories, public buildings

We performed a descriptive analysis by using non-parametric data analysis. General descriptive data are presented with median (minimum – maximum). The differences between the mean ranks measured were evaluated by the Mann-Whitney tests to explore the distribution of medians across independent samples. The Kruskal-Wallis test, was used to analyse whether there is a difference in the median values between locations of measurements listed above.

In order to analyse associations between environmental and health data, regression analysis were performed. The dependent variable was CEV (extrapolated and follow-up), and independent variable was ACN in air were defined. P values < 0.05 were considered statistically significant. The SPSS

software version 27.0 (SPSS Inc., Chicago, IL, USA) was used. Finally, Scilab 6.1.1 was used to geographically illustrate and combined the results.

3. RESULTS

Exposure descriptors: Delineation of chemical exposure

3.1. Description of maximum values for acrylonitrile

During the monitoring campaign, which had run for three weeks (from 4th to 24th of May, 2013), 3384 determinations of ACN concentration in the ambient air had taken place. After the geocoding process, during which data points with missing information were omitted, 3006 determinations were withheld. By mapping these latter concentrations (*Figure 3*), we observed that most samples were collected near the train accident and all the way down along the major sewer system to the WWTP. Four hotspots-areas can be characterized as being highly exposed to ACN (*Figure 1*). According to the Table 2, which categories ACN exposure values and the associated human health risk, Zones 1 and 2 were qualified as “immediate hazard level” and corresponded to the evacuation areas. Zones 3 and 4 correspond to areas with a “risk level” for human health and were not evacuated after the accident.

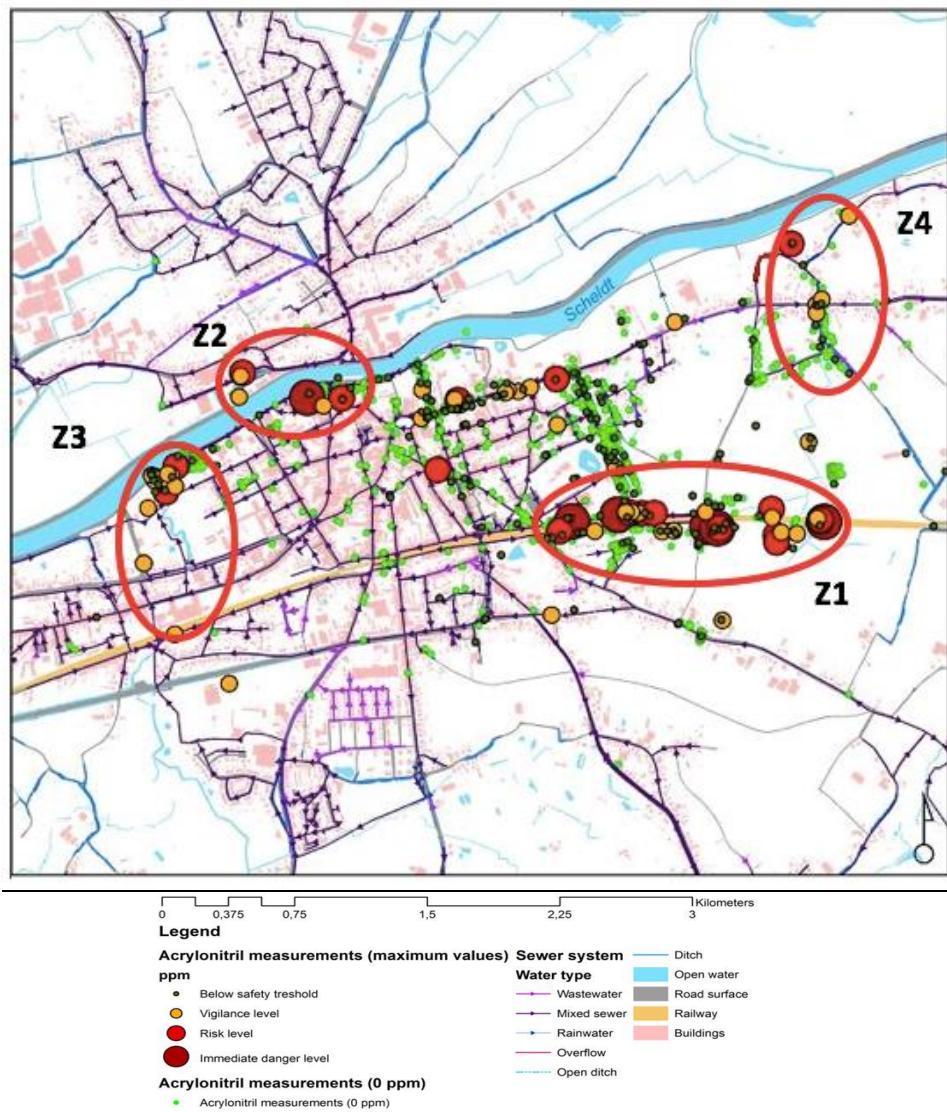


Figure 1: Map showing the delineation areas depending on the ACN concentration in the air (ppm): Chemical incident at Wetteren

From all measurements (N=3006), the maximum values were localised inside the sewer system (330 ppm), in the streets (196 ppm), in public and private buildings (bathroom), (107 ppm), and in the lavatories (98 ppm) (Table 3).

Table 3: Acrylonitrile air concentration (ppm), descriptive data

	N	Mean (SD)	SE	Min	Max	Mean Rank
Undefined	334	0.78 (4.38)	.13	0	50	1418.53
WWTP	99	1.13 (17.56)	.24	0	16	1476.24
Sewage Disposal	16	1.56 (3.42)	.56	0	24	1398.97
Train	29	6.33 (33.89)	.43	0	33	1855.86
Private Houses	373	0.48 (23.24)	.13	0	54	1320.85
Lavatories	5	40.84 (5.59)	.91	8.7	98	2972.3
Public Buildings	41	4.50 (9.93)	.37	0	107	1937.24
Streets	1722	0.59 (5.98)	.06	0	196	1435.15
Sewer Systems	387	4.50 (3.46)	.12	0	330	1976.95
Total	3006					

SD: Standard Deviation

SE: Standard Error

Table 3 shows eight independent groups to analyse. We determined the distribution of medians across locations. Results supported an evidence that the ranks of ACN monitored between each location was significantly different ($p < 0.000$, $N=3006$, $P\text{-value}: 0.000$, $df8$).

3.2. Acrylonitrile air concentration distribution (ppm)

Referring to indoor and outdoor air concentrations, we explored the mean distributions of ACN concentration for two independent groups. The statistical analysis of rank values is summarized in Table 4 and in which outdoor environment presented greater ACN air concentration than indoor. Also, ACN air concentration medians in the sewage systems was significantly higher than in the streets. There is no evidence to support a difference in rating ACN concentration between houses and streets.

Table 4: Mean ranks distribution quantified during measurement period

Indoor and Outdoor places	N	Mean Rank	Sum of Ranks	Z	P-value
ppm Indoor	563	1268.67	714263.50		
Outdoor	2109	1354.61	2856864.50		
Total	2672			-2.97	.003
Streets	1722	983.65	1693843.50		
Sewer Systems	387	1372.48	531151.50		
Total	2109			-14.07	.000
Houses	419	1048.19	439190.00		

Streets	1722	1076.55	1853821.00		
Total	2141			-1.15	.250

*Mann-Whitney Test (2-tailed): p-value < 0.05

3.3. Trend of ACN concentration (ppm/day)

ACN values monitored from the 25th of May, 2013 remained at zero ppm (below the safety threshold) and consequently not presented. Further description of contaminated areas is represented on Figure 4.

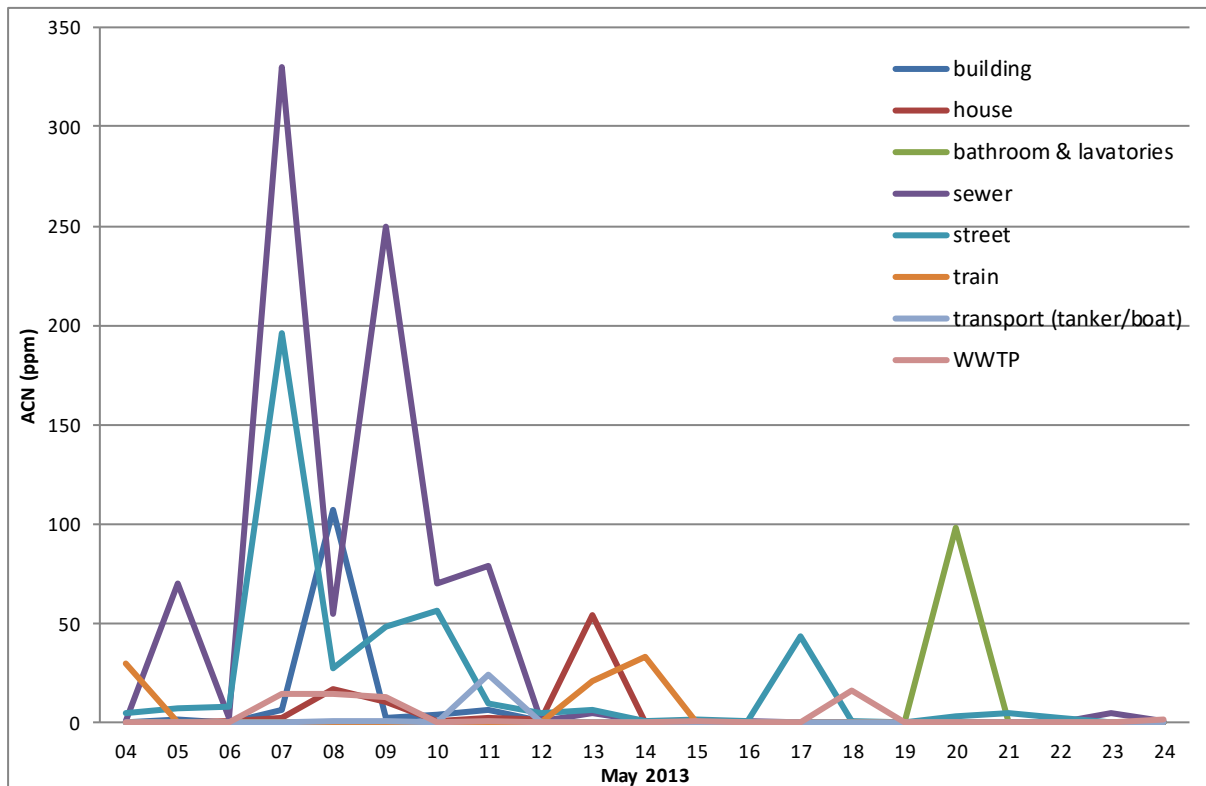


Figure 2: Trend of ACN air concentration (ppm) according to location sampling

Figure 4 illustrates the distribution of maximal ACN air concentrations (in ppm) during the three-week monitoring period. It shows the irregularity in ACN levels. The concentration decreases steadily, but until the 20th of May (day 17), the levels remain elevated and classified as “at risk”. Three peaks are observed during this period, respectively on the 7th of May (day 4) and the 9th of May (day 6) being the highest, and on day 17 (≥ 90.68 ppm) which corresponded to an “immediate hazard” for human health. On the 4-5th of May, hotspots were located around the train wagons, near the WWTP and in the sewer. Figure 3 shows sites exposed to ACN; from the 5th of May, ACN monitoring was extended across the sewer network, to also include private houses and public buildings. ACN peak concentrations in the sewer (near the site of train accident), streets and public buildings, respectively were 330, 196 and 107 ppm. At the WWTP, a maximum concentration corresponding to “vigilance level” ($4 \leq \text{ppm} \leq 21$) was observed. On the 9th of May, a high ACN concentration was localized in front of manholes (in the street). In general, street concentrations gradually decreased. However, on the 20th of May, ACN concentration reached an “immediate hazard level” in a private lavatory (≥ 90.68 ppm).

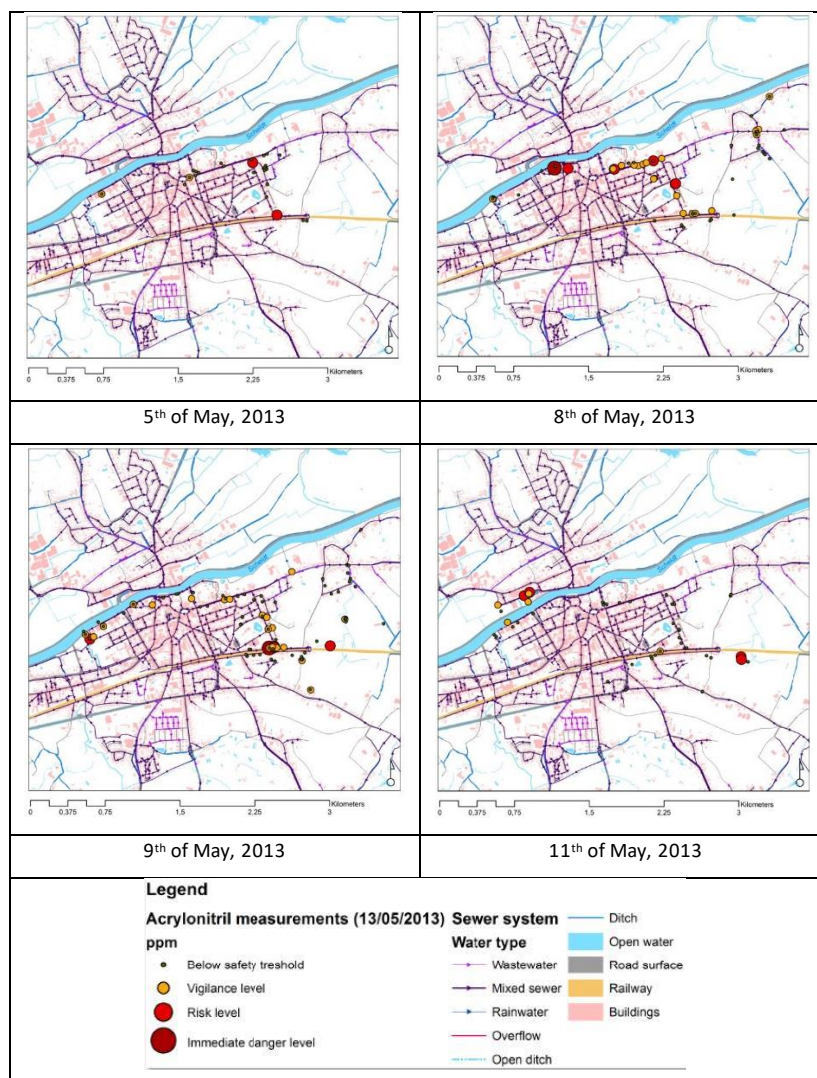


Figure 3: Exposure pathway of ACN in the environment

The table below shows the mean rank values depending on the identify Health risk zones identified. We can identify areas with the highest ACN measured in air and the association with the CEV concentration. Residents in Zone 1 (496.53 pmol/g globin), Zone 2 (261.26 pmol/g globin), Zone 3 (264.02 pmol/g globin), Zone 4 (319.82 pmol/g globin). The zone 5 represents area including the streets parallel with the sewage system and downwind of the train accident, where the maximum ACN concentration measured in the air was maximum 98 ppm (which correspond to a “Risk level”), and the maximum CEV measured associated to this day was 12614.865 pmol/g globin. Residents in zones 4 and 5 had been evacuated after the train accident.

Table 5: Extrapolated CEV at accident datum depending on the health risk zones (n=243)

		Mean (SD)	Mean Rank	Min	Max
N(232)					
Zone 1	70	68.876 (121.02)	6.00	1.126	496.530
Zone 2	3	181.155 (120.62)	211.00	42.431	261.261

Zone 3	14	100.794 (106.16)	49.50	1.126	264.020
Zone 4	19	70.171 (102.27)	10.00	2.315	319.820
Zone 5	128	296.466 (1225.16)	17.00	1.126	12614.865

3.4. Mapping environmental and health data

A visual map (Figure 4) was produced and shows that ACN in air and CEV in blood presents the same patterns of distribution in the space. The concentration thresholds used in this analysis refer to the VEC extrapolated on the day of the railway incident for non-smokers (10 pmol/g globin) and smokers (> 100 pmol/g globin).

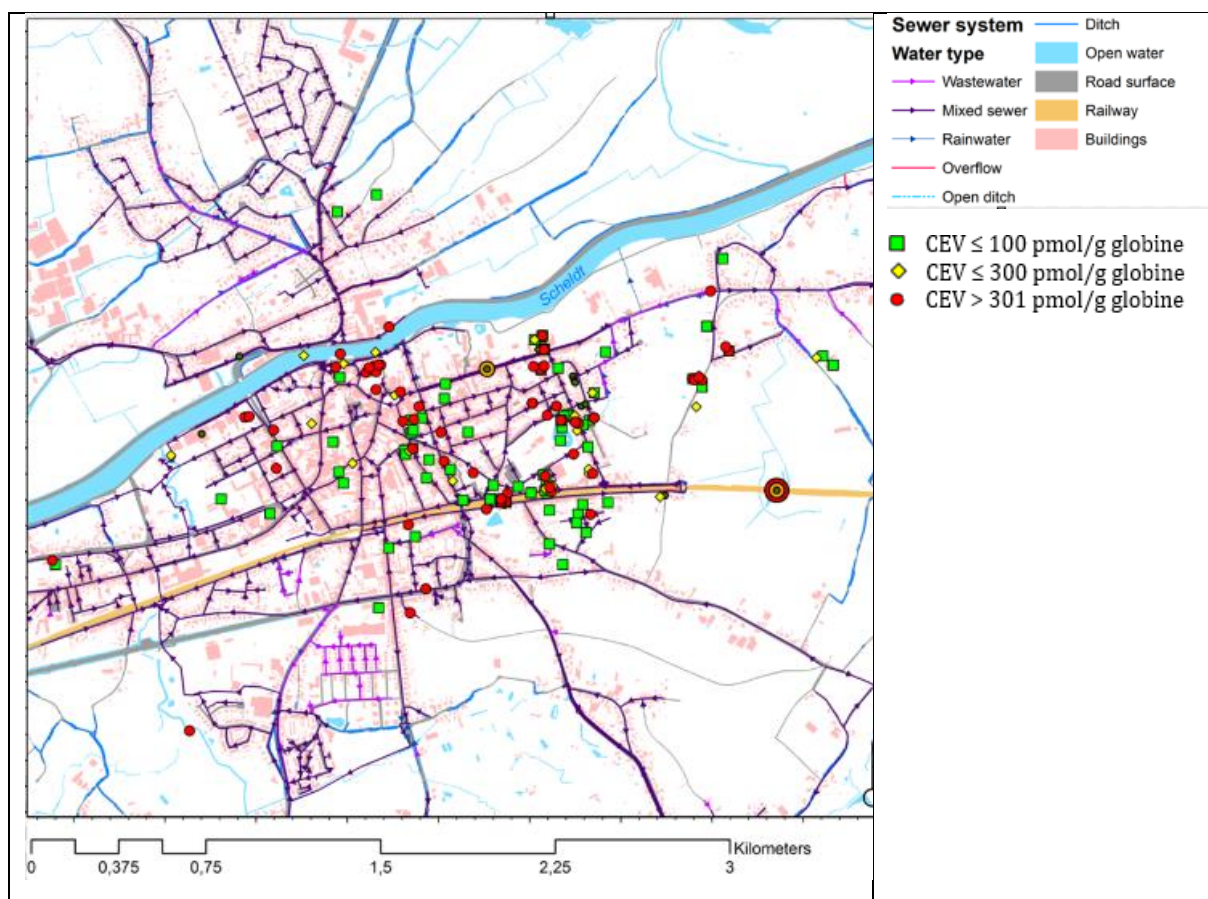


Figure 4: mapping CEV resulting the HBM campaign depending the tobacco status

The expression of the linear model used to interpret HBM results was $CEV = \text{measured CEV} / (1 - t \times 0.008)$ (Granath et al., 1992; Simons et al., 2016). Among the HBM follow-up, 57 participant were involved. The model analysis over the time was performed on the data (n=31) that showed a detectable CEV result in the blood. Results showed a decrease in the CEV values between the three time of measurements; day of the incident (D0), day of the first HBM campaign (D21) and day of the HBM follow-up (D44). Secondly, the decrease was linear for non-smokers, while the concentration trends for smokers did not show a significant difference from the extrapolated data to the time theoretical time of elimination (149 days after the accident).

4. DISCUSSION & CONCLUSION

The objectives of this retrospective study were to explore environmental contamination by identifying ACN-polluted areas in order to identify parameters that influence the environmental dispersion. An exposure description based on data retrieved during an environmental monitoring campaign following a major chemical accident was performed. Due to the crisis situation and to the interpretation of the retrospective data, some information may be lacking thus possibly limiting a complete comprehensive analysis. Instead of being systematic, ACN sampling localisation was rather ad hoc which is an inherent limitation. Indeed, the air sampling protocol was continuously adjusted according to the evolving situation and the meteorological conditions, so that neither the monitored locations nor the number of samples taken were similar during the study period. This aspect strongly impacts the interpretation of the trends in ACN concentration of this study.

Exposure description improves the identification and the delineation of chemical contamination; the chemical contamination zones identified in this study were larger than those delineated by firemen. Intervention areas corresponded to the major sewer network, even though the urban pipe infrastructures have several subsystems interlinked to ensure a continuous drain.

Data collection sites presented high levels of ACN in the air, which was found and quantified up to seventeen days after the chemical accident. Hotspots were localized following the gravitational sewer network from the train accident to the WWTP. Indeed, far away from the train accident, remote areas, i.e. zones 3 and 4, were also revealed as “highly polluted”. Zone 4 is localized in green areas (parks), receiving a local water stream and is close to the river. This area did not follow the gravitational influence from the major sewer system. Wind direction was south-west during days following the train accident. Natural circumstances such as heavy rain and floods, or due to a deficient sewer system, might lead to an overflow of the major sewers. Such situations occurred once tons of water were used to extinguish the blazing tank (Fradette, 2014; McLellan et al. 2015). Moreover, there was a correlation between intense rain and higher air concentration the following day in these zones. According to the historical Belgian forecast reports as well as confirmed by the accident reports from the Belgium Crisis Centre of the Federal Public Service of the Interior, the 7th of May 2013 was a night of intense rain-precipitation quantity was recorded at 16.3 mm- this likely contributed to the sudden drop in maximal sewer concentrations values on the 8th of May. During the study period, the average wind speed was only 10.92 m/s (Météo Belgique, 2013). Hence, it could be suggested that the wind had less impact than previously assumed on the environmental ACN distribution, and, moreover, that water transport had a greater role. This observation was also demonstrated by Ritter et al. (Ritter et al. 2002).

The observed trend of ACN concentration in this study confirms the persistence of this molecule in water (solubility 73.5 g/L at 20°C) as discussed after a biodegradation study done by Donberg et al. from Michigan University (Donberg PA et al, 1992). ACN vapours are heavier than air, its density is 1.83 (relative to air), lighter than water (density is 0.81 g/ml at 20°C) and, thus, might contaminate the environment over far distances and for a long-time duration due to its particular density (NTP, 2016; INERIS, 2008; INCHEM, 2002). According to WHO, the half-life of ACN in the atmosphere is estimated to be between 55 and 96 hours, which, from a theoretical basis, may result in a potential transport of up to 2000 km from the emission site. In water, where ACN is biodegradable by microorganisms, the half-life is between 30-552 hours, with a mean of 170 hours, i.e., 7 days. However, depending on the modelling method used, this mean value may vary from 13 years (under acidic conditions) to 188 years (under basic conditions). ACN is also biodegradable in soil and sediment with a half-life of 170 hours. The soil could be indirectly contaminated, indeed, as ACN vapours are heavier than air, the soil could absorb it (CDC, 2001; INERIS, 2017). Besides the literature review, continuous air monitoring showed that individuals could have been exposed to significant levels of ACN during longer periods of time, i.e., 480 hours in the air, than the estimated half-life in the air according to the literature (WHO 2002). ACN which reached the WWTP was destroyed faster than in the natural environmental compartments.

ACN does not seem to have any inhibitory effect on activated sludge systems and other microbial populations and will eventually be degraded, i.e., 95-100% (WHO, 2002). And as seen previously, likewise the instability of ACN in the air and the distribution through the water may explain the uncontrolled release.

As individuals were mainly exposed to ACN by inhalation, environmental contamination had been tracked by air monitoring in various places inside buildings, houses, in the streets and in sewage and wastewater treatment plants. Knowing the ACN pathways, the risk of the potential exposed population could be monitored more efficiently. Due to the chemical properties, the water source has an impact on the contamination of the environment. This study recommends to include water as a notable contamination pathway in exposure assessment for CAN and suggests that all the environmental media should be included in the exposure analysis.

ACN distribution analyses were made across every monitored place, and no significant difference was found in air concentration between private/public buildings and the streets. Outdoor environments, which refer to the streets, were significantly more exposed to ACN than general indoor locations. The specific in-house air measurements stemmed from individual complaints. These analyses led to the identification of highly polluted zones with uncontrolled chemical release that had not been foreseen.

Furthermore, such analysis requires a systematic and pre-defined design for data collection for a long enough period to include potential contamination such uncontrolled release of chemicals absorbed by the environment. It has been shown that methods geared towards a systematic data collection are needed to support the structural characterization and management of the risk of exposure (Dotson et al. 2015). In particular, in an environmental disaster context, continuous environmental monitoring, as suggested by Behbod et al., is recommended (Behbod et al, 2017). In order to anticipate the consequences of a chemical contamination, it may be useful to undertake a complete environmental analysis from soil, water, and air to vegetation, building materials and debris, as well as to envision several scenarios using Monte Carlo analysis (Huizer, 2014).

Our findings are based on the analysis of data used and referred to during the Wetteren chemical accident in 2013. By categorizing ACN levels based on the Dutch Intervention Values for Emergency Response, the potential risk for residents was assessed and polluted areas were identified. In general, the majority of locations that were monitored had *no significant level* of ACN (equal to zero ppm). Despite this, hotspots were identified with significant “risk” and “immediate hazard” levels of ACN exposure. These areas might likely correspond to individuals with health risks due to the chemical exposure. With regards to the literature review, when our analysis was adjusted to the updated intervention reference values proposed by the RIVM (2015)- fixed at 58.89 ppm (for one-hour exposure), the delineation of risk areas was different and presented a slightly different number of locations classified as “vigilance levels” rather than “risk levels”. Therefore, the reference values used in this study are more protective of residents and emergency workers, knowing that exposure is greater in occupational settings than in the general population. Accordingly, this analysis demonstrates the necessity of updating Guideline Reference values over the monitoring period, i.e., from 24 hours, and adapted to the group of population exposed.

Consideration was given to the use of a linear model to estimate the CEV value on the day of the chemical incident from the initial HBM results. According to this model, a high level of CEV on the day of sampling is representative to a CEV level following the chemical incident, without any influence from the time parameter. In the analyses, the difference in CEV data from participants between the first and second HBMs was nearly identical in concentrations. Moreover, these participants were hospitalised or located directly in the evacuated zone. However, the linear relationship was shown for CEV concentrations in non-smokers participants. These results suggest that the incident was the source of exposure to ACN for non-smokers. This finding cannot be extrapolated to all participants because the analysis was carried out on a very small number of participants (n=31).

Simon K. et al. discussed uncertainties encountered while using self-report exposure assessment after an accidental release of chemicals in the environment (Simons et al. 2016). Authors agreed that those issues in the exposure reconstruction can be diminished by using human biomonitoring (Leng et al. 2014; De Smedt, et al. 2014; Van Nieuwenhuysse et al. 2014, Simons, et al. 2016). When the air concentration of ACN exceeds 3 ppm, the biomarker CEV can be detected in the blood (Bader and Wrbitzky, 2006), while ACN becomes detectable by smell from 17 ppm (INERIS, 2008). In addition, overall, the ACN pattern shown in this analysis was in line with the results of the human biomarker analysis implemented two weeks after the train accident (De Smedt, et al. 2014; Van Nieuwenhuysse et al. 2014, Simons, et al. 2016). Acrylonitrile air distribution trend is similar to the N-2-cyanoethylvaline (CEV) biomarker distribution trend showed in the human biomonitoring study. Indeed, contaminated areas, illustrated in this study, correspond to locations where people presented significant biomarkers of exposure levels exceeding the intervention reference value. Both analyses, i.e., environmental and human biomonitoring, highlighted hotspots presenting to the highest ACN exposure areas. Notwithstanding, this study confirmed a number of additional hotspots. The environmental monitoring gives further indication of the distribution of the pollutant and is a reliable tool during accidental chemical release. To our knowledge, this analysis is the first exploration of descriptive environmental data following a contamination by ACN in the framework of a chemical disaster.

In summary, this research shows the effectiveness of an environmental description to characterize the exposure of ACN. From the present train accident, it appeared that direct measurement in several environmental media were essential to limiting the environmental contamination and therefore the human exposure. Since, we have shown that individuals may be exposed to high levels of concentration in various places. The risk management after chemical disasters not only requires a harmonized protocol in the framework of chemical accident management but also an integrated environmental and human health risk assessment.

Summary of lessons learnt:

- Early environmental assessment allows delineation of polluted and risk areas;
- Continuous air quality measurements are a complementary tool to HBM in order to characterize the risk and to identify the group(s) that may be exposed;
- Identification of potential sources of contamination and pathways of human exposure is a key element for an optimal public health management;
- Exposure assessment involves a dynamic analysis over time and space during a chemical accident;
- Collaboration and coordination in data management is crucial.

6. ACKNOWLEDGMENT

We thank Sciensano, The Scientific Institute of Health (Belgium), the Crisis centrum at Federal Public Service Health, Food Chain Safety and Environment, Brandweer Vereniging Vlaanderen (BVV), partners and co-authors involved in the risk management for providing the opportunity to do this research via data and support.

There is no conflict of Interest.

7. BIBLIOGRAPHY

- Acrylonitrile (CICADS 39, 2002) (2017) Retrieved 23 April 2017, from <http://www.inchem.org/documents/cicads/cicads/cicad39.htm#6>
- Arnold, S. M., Angerer, J., Boogaard, P. J., Hughes, M. F., O'Lone, R. B., Robison, S. H., & Schnatter, A. R. (2013) The use of biomonitoring data in exposure and human health risk assessment: benzene case study. *Critical Reviews in Toxicology*, 43(2), 119–153. <https://doi.org/10.3109/10408444.2012.756455>
- ATSDR - Toxicological Profile: Acrylonitrile. Retrieved 23 April 2017, from <https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=447&tid=78>
- Bader M, Wrbitzky R. (2006) Follow-up biomonitoring after accidental exposure to acrylonitrile:- implications for protein adducts as a dose monitor for short-term exposures, *Toxicol Lett.* 162(2-3):125-31.
- Behbod, B., Leonardi, G., Motreff, Y., Beck, C. R., Yzermans, J., Lebret, E., ... Pirard, P. (2017) An International Comparison of the Instigation and Design of Health Registers in the Epidemiological Response to Major Environmental Health Incidents. *Journal of Public Health Management and Practice: JPHMP*, 23(1), 20–28. <https://doi.org/10.1097/PHH.0000000000000489>
- Birnbaum, M. L., Daily, E. K., O'Rourke, A. P., & Loretta, A. (2014) Disaster Research/Evaluation Frameworks, Part 1: An Overview. *Prehospital and Disaster Medicine*, 1–12. <https://doi.org/10.1017/S1049023X14000211>
- Birnbaum ML, Daily EK, O'Rourke AP. (2015) Research and Evaluations of the Health Aspects of Disasters, Part III: Framework for the Temporal Phases of Disasters. *Prehosp Disaster Med.* 30(6):628-32. doi: 10.1017/S1049023X15005336.
- Bongers, S., Janssen, N. A. H., Reiss, B., Grievink, L., Lebret, E., & Kromhout, H. (2008) Challenges of exposure assessment for health studies in the aftermath of chemical incidents and disasters. *Journal of Exposure Science and Environmental Epidemiology*, 18(4), 341–359. <https://doi.org/10.1038/jes.2008.23>
- Carex Canada. (2016) Surveillance of environmental & occupational exposure for cancer prevention. Available on <https://www.cerexcanada.ca/en/acrylonitrile/#sources>
- Centers for Disease Control and Prevention (2001), International Chemical Safety Cards (WHO/IPCS/ILO) | CDC/NIOSH. Acrylonitrile ICSC:0092, Clifton Road Atlanta, USA. Available from <https://www.cdc.gov/niosh/ipcsnfrn/nfrn0092.html>
- De Smedt, T., De Cremer, K., Vleminckx, C., Fierens, S., Mertens, B., Van Overmeire, I., Van Nieuwenhuysse, A. (2014) Acrylonitrile exposure in the general population following a major train accident in Belgium: A human biomonitoring study. *Toxicology Letters*, 231(3), 344–351. <https://doi.org/10.1016/j.toxlet.2014.09.009>
- Donberg PA., Odelson DA., Klecka GM., Markham DA. (1992) Biodegradation of acrylonitrile in soil. *Environ Toxicol Chem*, 11: 1583-94
- Dotson, G. S., Hudson, N. L., & Maier, A. (2015) A decision support framework for characterizing and managing dermal exposures to chemicals during Emergency Management and Operations. *American Journal of Disaster Medicine*, 10(3), 237–258. <https://doi.org/10.5055/ajdm.2015.0206>
- European Commission. European (2004) Union Risk Assessment Report: acrylonitrile, Volume 32. EUR 20857. Environment and quality of life series

- European Union, Regulation(EC) No 1272/2008 of the European Parliament and the Council of 16 December 2008 on classification, labelling and packaging of substances and mixtures, amending and repealing Directives 67/548/EEC and 1999/45/EC, and amending Regulation (EC) No 1907/2006
- Gentry, R., Franzen, A., Van Landingham, C., Greene, T., & Plotzke, K. (2017). A global human health risk assessment for octamethylcyclotetrasiloxane (D4). *Toxicology Letters*, 279 Suppl 1, 23–41. <https://doi.org/10.1016/j.toxlet.2017.05.019>
- Government of Canada, E. and C. C. C. (2007) ARCHIVED - Environment and Climate Change Canada - Pollution and Waste - Fact Sheet - Acrylonitrile. Retrieved 23 April 2017, from <http://www.ec.gc.ca/planp2-p2plan/default.asp?lang=En&n=30F84DFF-1>
- Granath, F., Ehrenberg, L., Tornqvist, M., 1992. Degree of alkylation of macromolecules in vivo from variable exposure. *Mutat. Res.* 284, 297–306.
- Heinäälä M, Gundert-Remy U, Wood MH, Ruijten M, Bos PM, Zitting A, Bull S, Russell D, Nielsen E, Cassel G, Leffler P, Tissot S, Vincent JM, Santonen T. (2013) Survey on methodologies in the risk assessment of chemical exposures in emergency response situations in Europe. *J Hazard Mater* 244-245:545-54. doi: 10.1016/j.jhazmat.2012.10.068.
- Huizer, D., Ragas, A. M. J., Oldenkamp, R., van Rooij, J. G. M., & Huijbregts, M. A. J. (2014) Uncertainty and variability in the exposure reconstruction of chemical incidents--the case of acrylonitrile. *Toxicology Letters*, 231(3), 337–343. <https://doi.org/10.1016/j.toxlet.2014.07.019>
- Hunault, C. C., Boerleider, R. Z., Hof, B. G. H., Kliet, J. J. G., Meijer, M., Nijhuis, N. J., ... Meulenbelt, J. (2014) Review of acute chemical incidents as a first step in evaluating the usefulness of physiologically based pharmacokinetic models in such incidents. *Clinical Toxicology*, 52(2), 121–128. <https://doi.org/10.3109/15563650.2013.878866>
- International Agency for Research on Cancer, Acrylonitrile. IARC Monogr Eval Carcinog Risks Hum. 1999;71(pt 1):43-108.
- INERIS (2008), Portail Substances Chimiques. Acrylonitrile Available form : <https://substances.ineris.fr/fr/substance/332>
- INERIS (2017, Portail Substances Chimiques Acrylonitrile) Available from <http://www.ineris.fr/substances/fr/substance/332>
- Jardine, C., Hrudey, S., Shortreed, J., Craig, L., Krewski, D., Furgal, C., & McColl, S. (2003) Risk management frameworks for human health and environmental risks. *Journal of Toxicology and Environmental Health. Part B, Critical Reviews*, 6(6), 569–720. <https://doi.org/10.1080/10937400390208608>
- Knol, A. B., Briggs, D. J., & Lebreton, E. (2010a). Assessment of complex environmental health problems: framing the structures and structuring the frameworks. *The Science of the Total Environment*, 408(14), 2785–2794. <https://doi.org/10.1016/j.scitotenv.2010.03.021>
- Leng, G., & Gries, W. (2014). Biomonitoring following a chemical incident with acrylonitrile and ethylene in 2008. *Toxicology Letters*, 231(3), 360–364. <https://doi.org/10.1016/j.toxlet.2014.06.027>
- Long, G., Meek, M. E., Cureton, P. M. (2002) World Health Organization., International Labour Organisation., United Nations Environment Programme., & Inter-Organization Programme for the Sound Management of Chemicals. Acrylonitrile. Geneva: World Health Organization.
- Meteo Belgique (2013), Bilan climatologique du mois de mai 2013. Retrieved September 2018, from <https://meteobelgique.be>

- McLellan, S. L., Fisher, J. C., & Newton, R. J. (2015) The microbiome of urban waters. *International Microbiology: The Official Journal of the Spanish Society for Microbiology*, 18(3), 141–149. <https://doi.org/10.2436/20.1501.01.244>
- Ning, X. A., Zhou, J. B., Liu, J. Y., Yang, Z. Y., & Zhang, N. (2011) Analysis of Accidental Consequence of Butadiene Tank. *Applied Mechanics and Materials*, 71–78, 2243–2248. <https://doi.org/10.4028/www.scientific.net/AMM.71-78.2243>
- NRC (National Research Council) (2014) *Acute Exposure Guideline Levels for Selected Airborne Chemicals, Vol. 17*. Washington, DC: National Academy Press 2014. Doi 10.17226/18796
- NTP (National Toxicology Program) (2016) *Report on Carcinogens, Fourteenth Edition*.; Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service. <https://ntp.niehs.nih.gov/go/roc14/> (EndNote XML)
- PubChem (2019) National Center for Biotechnology Information. PubChem Compound Database; CID=7855, <https://pubchem.ncbi.nlm.nih.gov/compound/7855> (accessed Feb. 19, 2019).
- Ritter, L., Solomon, K., Sibley, P., Hall, K., Keen, P., Mattu, G., & Linton, B. (2002). Sources, pathways, and relative risks of contaminants in surface water and groundwater: a perspective prepared for the Walkerton inquiry. *Journal of Toxicology and Environmental Health. Part A*, 65(1), 1–142
- RIVM. (2015). Ministerie van Volksgezondheid, Welzijn en Sport, Risico's van stoffen, Acrylnitril IVW, Netherlands National Institute for Public Health and the Environment, BA Bilthoven, The Netherlands.
- Simons, K., De Smedt, T., Stove, C., De Paepe, P., Bader, M., Nemery, B., Van Nieuwenhuysse, A. (2016). Short-term health effects in the general population following a major train accident with acrylonitrile in Belgium. *Environmental Research*, 148, 256–263. <https://doi.org/10.1016/j.envres.2016.03.031>
- Svensden ER, Yamaguchi I, Tsuda T, Guimaraes JR, Tondel M. (2016) Risk Communication Strategies: Lessons Learned from Previous Disasters with a Focus on the Fukushima Radiation Accident. *Curr Environ Health Rep*. 3(4):348-359
- Törnqvist, M., Mowrer, J., Jensen, S., Ehrenberg, L. (1986) Monitoring of environmental cancer initiators through hemoglobin adducts by a modified Edman degradation method. *Anal. Biochem.* 154,255–266
- Toxnet (2004), Hazardous Substances Data Bank. Acrylonitrile, Environmental fate and exposure, Retrieved in 2017, from (<http://toxnet.nlm.nih.gov>)
- U.S. EPA. Guidelines for Human Health Risk Assessment. U.S. Environmental Protection Agency, Washington, DC.
- U.S. EPA. Guidelines for Risk Assessment. U.S. Environmental Protection Agency, Washington, DC.
- U.S. EPA. (1992) Guidelines for Exposure Assessment. (EPA/600/Z-92/001). Washington, DC.
- U.S. EPA. (2000) Health Effects Notebook for Hazardous Air Pollutants. U.S. Environmental Protection Agency, Washington, DC. Available from: <https://www.epa.gov/sites/production/files/2016-09/documents/acrylonitrile.pdf>
- U.S. EPA. (2011) Exposure Factors Handbook 2011 Edition (Final Report). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-09/052F.
- Van Nieuwenhuysse, A., Fierens, S., De Smedt, T., De Cremer, K., Vleminckx, C., Mertens, B., ... Van Looco, J. (2014). Acrylonitrile exposure assessment in the emergency responders of a major

- train accident in Belgium: A human biomonitoring study. *Toxicology Letters*, 231(3), 352–359. <https://doi.org/10.1016/j.toxlet.2014.08.013>
- Wang, L., Zhong, B., Vardoulakis, S., Zhang, F., Pilot, E., Li, Y., ... Krafft, T. (2016) Air Quality Strategies on Public Health and Health Equity in Europe-A Systematic Review. *International Journal of Environmental Research and Public Health*, 13(12). <https://doi.org/10.3390/ijerph13121196>
- WHO, (2002) International Programme on Chemical Safety, Acrylonitrile. Concise International Chemical Assessment. Genève: World Health Organization. CICAD 39. [MO-128534] <http://www.inchem.org/documents/cicads/cicads/cicad39.htm>
- WHO, (2017) Evolution of WHO air quality guidelines: past, present and future. Copenhagen: WHO Regional Office for Europe.
- Wilson, R.H., G.V. Hough, and W.E. McCormick. (1948) Medical problems encountered in the manufacture of American-made rubber. *Ind. Med. Surg.* 17(6):199-207.
- Wyke, S., Peña-Fernández, A., Brooke, N., & Duarte-Davidson, R. (2014) The importance of evaluating the physicochemical and toxicological properties of a contaminant for remediating environments affected by chemical incidents. *Environment International*, 72, 109–118. <https://doi.org/10.1016/j.envint.2014.05.002>
- Yee, S. H., Bradley, P., Fisher, W. S., Perreault, S. D., Quackenboss, J., Johnson, E. D., ... Murphy, P. A. (2012) Integrating human health and environmental health into the DPSIR framework: a tool to identify research opportunities for sustainable and healthy communities. *EcoHealth*, 9(4), 411–426. <https://doi.org/10.1007/s10393-012-0805-3>