

DOWNDOWN CHLORINE HAZARD ESTIMATES FOR THE 2015-2016 JACK RABBIT II CAMPAIGN

Nathan Platt¹, Kevin Luong², and Jeffry T. Urban¹

¹Institute for Defense Analyses, Alexandria, Virginia, USA

²University of California, Los Angeles, Department of Electrical Engineering, Los Angeles, California, USA

Abstract: Toxic industrial chemicals and chemical warfare agents present an acute inhalation hazard to civilians and military personnel. The US Department of Homeland Security (DHS), in cooperation with the US Defense Threat Reduction Agency, sponsored a two-year experimental campaign of large-scale outdoor chlorine releases at the US Army's Dugway Proving Ground named Jack Rabbit II (JRII).

This work presents estimates of the downwind inhalation health hazard to humans based on chlorine measurements made during the 2-year JRII campaign. For each downwind sampler arc, we calculated both the probability of adverse health effects using a US Department of Defense (DoD)-approved toxicological model and whether the chlorine exposure exceeds the US Environmental Protection Agency's Acute Exposure Guideline Levels (AEGLs) for chlorine. The toxicological model that we applied is the toxic load model, modified for use with time-varying exposures. We considered the probability of lethal, severe, or mild effects from chlorine exposure in both the general and military populations. We also considered AEGLs at all three levels of health effects severity.

We note that since our hazard distance estimates are produced from chlorine concentrations that were directly measured (i.e., not modelled), the accuracy of these estimates depends only on the quality of the health effects models we applied, and not on the quality of any atmospheric transport and dispersion model or container release (chemical source term) model.

Key words: Chlorine exposure health effects, toxic industrial chemicals, Jack Rabbit II, toxic load model, AEGLs

INTRODUCTION

Toxic industrial chemicals and chemical warfare agents present an acute inhalation hazard to civilians and military personnel. The US Department of Homeland Security (DHS), in cooperation with the US Defense Threat Reduction Agency's Joint Science and Technology Office for Chemical and Biological Defense (DTRA/JSTO-CBD), sponsored a two-year experimental campaign of large-scale outdoor chlorine releases at the US Army's Dugway Proving Ground named Jack Rabbit II (JRII). In 2015, the first year of the campaign, five instantaneous releases of pressurized liquefied chlorine from the bottom of a storage tank were conducted; release amounts were between 4.5 and 8.3 metric tons. To simulate effects of obstacles on dense gas transport and dispersion, a number of Conex containers were placed in a regular pattern within a "mock urban area" 122 meters square. In 2016, the second year of the campaign, four releases of pressurized liquid chlorine were conducted with release amounts between 8.3 and 17.7 metric tons. In this phase of the campaign, a mock urban area was not employed, and the angle of the release with respect to the ground was varied among releases. The JRII field experiments included a large number of sensors that measured chlorine concentrations, meteorological parameters, and chlorine tank release parameters. More than 200 samplers measured atmospheric chlorine concentrations continuously for up to 1.5 hours after the release. These samplers were deployed near the release site and in arcs at 0.2, 0.5, 1, 2, 5, and 11 km downwind. Figure 1 indicates locations of the surface samplers arranged on arcs for Trial 4 in 2015. Other JRII trials employed a similar arrangement of samplers on arcs, although the sampler density was changed between the 2015 and 2016 campaigns.

In this paper, we present estimates of the downwind inhalation hazard to humans based on the chlorine measurements made for the six JRII releases for which approximately 10 short tons (9071 kg) of chlorine were released. These include releases 2, 4, 5 from the first year of the campaign and releases 6, 7, and 8 from the second year. The release amounts were 8151 kg, 6970 kg, 8303 kg, 8380 kg, 9075 kg, and 9089 kg, respectively. Release 1 (4509 kg), release 2 (4512 kg), and release 9 (17706 kg) were excluded from this analysis to ensure that our hazard estimates were derived from comparably-sized releases.

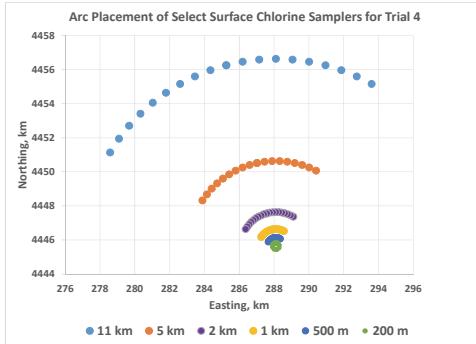


Figure 1. Locations of the arc-based chlorine samplers during JRII Trial 4.

HUMAN EFFECTS MODELLING

Several human health effects models were applied in this study, including (a) the toxic load model for human lethal chlorine effects on military and civilian populations developed by Sommerville, D.R, et al. (2010), (b) the toxic load model for both severe and mild chlorine inhalation effects developed by Winkel, D, et al. (2014), and (c) the Acute Exposure Guideline Levels (AEGLs) for chlorine used by the EPA (Acute Exposure Guidelines Levels for Selected Airborne Chemicals, Volume 4, 2004).

The toxic load is a generalized measure of exposure defined as $TL = C^n T$, where C is the constant atmospheric concentration of toxic chemical in the exposure, T is the duration of the exposure, and n is an experimentally-derived toxicological parameter called the toxic load exponent. When $n > 1$, short duration, high-concentration exposures to the chemical are more damaging than long, low-concentration exposures to the same amount of chemical; when $n < 1$, the reverse is true.

The typical toxicological response model used to relate toxic exposure (i.e., the toxic load) to a human population response is a probit model based on a log-normal distribution of the exposure. The toxic load-based probit model has three parameters: the median effective toxic load TL_{50} , the concentration-based probit slope b , and toxic load exponent n (Eq. 1):

$$Cas(TL) = \Phi\left(\frac{b}{n} \log_{10}\left(\frac{TL}{TL_{50}}\right)\right) \quad (1)$$

where $Cas(TL)$ is the fraction of the population receiving casualties at a certain level of effect (e.g., death or incapacitation) at toxic load value TL , and $\Phi(\bullet)$ denotes the standard normal cumulative distribution function. Following usual practice, we equate the casualty fraction with the probability that a random individual would experience that effect from the exposure. The median effective toxic load TL_{50} is the toxic load required to achieve a certain level of effect in 50% of the population. For chlorine, the toxic load model parameters shown in Table 1 were used to estimate various effects. Of note here, the Table 1 values for TL_{50} and b are valid only when n equals 2.75, the value we used for chlorine.

Table 1. Toxic load model parameters for Lethal (General and Military populations), Mild, and Severe effects.

Toxic load exponent, n	2.75	
Human Effect	TL_{50} , min-(mg/m ³) ^{2.75}	Probit Slope, b
Lethal, General Population	2.58E+10	6
Lethal, Military Population	6.79E+10	8
Mild	7500	2.6125
Severe	1.20E+08	7.975

Real-world exposures to airborne chemicals involve time-varying concentrations (not just constant C), thus the basic definition of the toxic load model must be extended to account for time-varying (i.e., fluctuating and intermittent) concentrations. In this paper, three proposed extensions of the toxic load model are considered (Eq. 2) (Czech, et al., 2011).

$$\begin{aligned}
\text{ten Berge:} \quad TL_{tenBerge} &= \int [c(t)]^n dt \\
\text{Average Concentration:} \quad TL_{AvgConc} &= \left(\frac{\int c(t) dt}{T} \right)^n T, \text{ where } T \text{ is the exposure duration} \\
\text{Peak Concentration:} \quad TL_{PeakConc} &= \frac{\int c(t) dt}{c_{Peak}^{1-n}}, \text{ where } c_{Peak} = \max \{c(t)\}
\end{aligned} \tag{2}$$

It can be shown that $TL_{AvgConc} \leq TL_{tenBerge} \leq TL_{PeakConc}$ when $n \geq 1$, hence these three model extensions might be considered to reasonably span the toxic loads resulting from time-varying exposures. We note that toxic loads calculated via the Average Concentration model are dependent on the exposure duration T , which we calculated as the difference between the plume arrival and departure times determined at a concentration threshold of 10% of the plume maximum. The ten Berge and Peak Concentration models do not require estimates of T . We computed the ten Berge and Peak Concentration toxic loads over the full duration of the concentration measurement at each sensor. For the sake of consistency, we also calculated the ten Berge and Peak Concentration toxic loads over the same exposure duration T that we used to calculate the Average Concentration toxic load. The choice of calculation method in this case had no significant impact on the modeled extent of the downwind chlorine hazard.

In addition to estimating the downwind hazard by predicting the probability of adverse health effects, one may estimate the downwind hazard by examining whether the exposure at a particular downwind distance exceeds a level of concern. The Acute Exposure Guideline Levels (AEGLs) developed in a collaboration led by the US EPA are designed for use by the emergency response community. For each chemical, AEGLs are specified in terms of threshold airborne concentration exceedance for five acute exposure durations and three levels of health effects. The three levels are (a) AEGL-1, the concentration above which a typical person might experience reversible non-disabling effects such as discomfort, irritation, or some asymptomatic non-sensory effects; (b) AEGL-2, the concentration above which a person might experience long-lasting, serious, or irreversible effects, or an impaired ability to escape; (c) AEGL-3, the concentration above which a person might experience life-threatening effects or death. Table 2 reproduces EPA's AEGLs for chlorine for the five acute exposure durations.

We apply AEGLs to the JRII chlorine concentration data by equating the plume passage time at a sensor location with the AEGL exposure duration and comparing the average measured concentration over the plume passage time to AEGL-1, AEGL-2, and AEGL-3 for that exposure duration. Since measured plume passage times generally do not correspond exactly to one of the five AEGL acute exposure durations, we use the fact that AEGLs are based on the toxic load model to perform a linear interpolation in $\log(\text{average concentration}) - \log(\text{exposure duration})$ space to determine the effective AEGL concentration threshold for arbitrary exposure durations.

Table 2. Acute Exposure Guideline Levels (AEGL) for airborne chlorine exposure in parts per million (ppm)

	Exposure Duration				
	10 min	30 min	60 min	4 hr	8 hr
AEGL 1	0.50	0.50	0.50	0.50	0.50
AEGL 2	2.80	2.80	2.00	1.00	0.71
AEGL 3	50.00	28.00	20.00	10.00	7.10

DOWNDOWN EXTENT OF HAZARD

The extent of the downwind chlorine hazard according to the ten Berge toxic load model is depicted in Figure 3. This figure shows the maximum probability of lethality (or "fractional lethality") for military (top left panel) and general population (top right panel), and the extent of downwind chorine hazard for severe health effects (bottom left panel) and mild health effects (bottom right panel) on each JRII sensor arc. As mentioned earlier, only the JRII releases in the range of 7 to 9 metric tons (8 to 10 short tons) of chlorine were considered. Each panel contains a set of bars for each JRII arc at 0.2, 0.5, 1, 2, 5, and 11 km from the release. Individual bars correspond to individual JRII trials. A pink or red circle above a bar indicates that at least one sensor on that arc reached its saturation limit: light pink circles indicate

instruments that saturate at 2000 ppm of chlorine and dark red circles indicates instruments that saturate at 50 ppm of chlorine. For the sake of brevity, only ten Berge toxic load model plots are provided here.

Figure 4 depicts the downwind chlorine hazard according to AEGLs. Again, six sets of bars represent the six JRII sensor arcs. The height of each bar indicates the highest AEGL that was exceed by any sensor on each arc. For example, a height of 2 at 1000 meters means that for that given JRII release, at least one of the sensors on the 1000-meter arc measured a chlorine exposure that was high enough to exceed AEGL-2 (severe effects), but that none of the sensors on that arc measured a chlorine exposure that was high enough to exceed AEGL-3 (lethal effects). As before, circles denote the presence of saturated sensors.

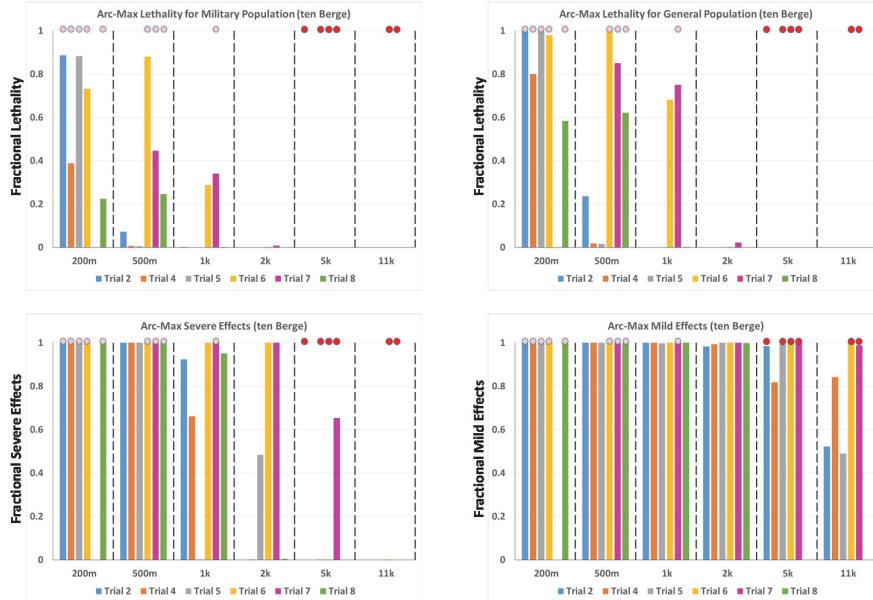


Figure 3. Arc-maximum fractional lethality for the military and general population, and the extent of downwind chlorine hazard for severe and mild effects estimated using ten Berge toxic load model for selected JRII releases.

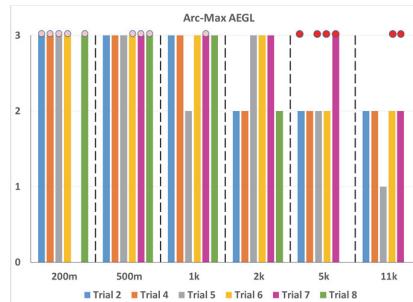


Figure 4. Arc-maximum Acute Exposure Guideline Levels (AEGLs) for selected JRII trials.

DISCUSSION

We used data from the JRII 2015 and 2016 campaigns to estimate the downwind extent of the health hazard for six sudden releases of 8–10 short tons of pressurized liquefied chlorine. The extent of the downwind hazard can be only roughly estimated due to the relatively sparse JRII sampler distributions.

We make the following observations about casualty-based estimates of the JRII downwind hazard using the toxic load model (see Figures 3):

- Lethal effects are expected up to 1 km from the release. However, for one release (Trial 7), both ten Berge and the Peak Concentration extensions of the toxic load model predict a non-negligible chance of death (2.3% and 8% respectively) at 2 km from the release.

- Severe effects are expected to be observed at the ranges of 2 to 5 km from the release. However, for one release (Trial 7), all extensions of the toxic load model considered in this paper predict severe effects at 5 km from the release.
- Mild effects are expected to be observed beyond 11 km from the release (i.e., beyond the farthest downwind observations made during the JRII campaign).

We make the following observations about AEGL-based estimates of the JRII downwind hazard (see Figure 4):

- AEGL-3 exposures are observed at 2–5 km from the release with some possibility of extending past 5 km (Trial 7).
- AEGL-2 exposures are observed up to 11 km range (i.e., up to the farthest downwind observations made during the JRII campaign).
- AEGL-1 exposures are observed beyond 11 km from the release (i.e., beyond the farthest downwind observations made during the JRII campaign).

Trial 7 resulted in significantly longer downwind hazard distances than the other trials with comparable release amounts. A possible explanation for the higher concentrations at longer range is that the 4.5 m/s wind speed during Trial 7 was relatively high among the JRII releases. However, Trial 2 did not exhibit a similar long downwind hazard despite a similar wind speed of 4.2 m/s. We considered several explanations for the difference in downwind hazard behavior between Trials 2 and 7. First, the Trial 2 release was directed downward from the container (compared to 135 degrees from vertical for Trial 7), potentially resulting in a wider initial cross-wind spread of chlorine due to impingement of the chlorine jet on the ground, and a corresponding decrease in high concentrations far downwind. Second, the Trial 2 release occurred in a mock urban area (whereas the Trial 7 release did not), which also may have resulted in a wider, lingering plume with decreased concentrations far downwind. Finally, Trial 2 occurred later in the morning than Trial 7 (0925 vs. 0756 local time), potentially allowing the development of a deeper atmospheric boundary layer that dispersed chlorine vertically, again leading to decreased surface concentrations downwind. More research is required to determine the cause of the atypical Trial 7 result.

We note that since our hazard distance estimates are based on directly measured airborne chlorine concentrations (i.e., not modelled concentrations), the accuracy of our results depends only on the accuracy of the health effects modelling (i.e., the toxic load model or the AEGL method), and not on the accuracy of any atmospheric dispersion model or container release (chemical source term) model.

Acknowledgments: This effort was supported by the US Defense Threat Reduction Agency (through Mr. Richard J. Fry as the project monitor) and the Institute for Defense Analyses' professional development program. The views expressed in this paper are solely those of the authors.

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