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Abstract

In the event of a short-term, large-scale toxic chemical release to the atmosphere, shelter-in-place (SIP) may be used as an emergency response to protect public health. We modeled hypothetical releases using realistic, empirical parameters to explore how key factors influence SIP effectiveness for single-family dwellings in a residential district. Four classes of factors were evaluated in this case-study: (a) time scales associated with release duration, SIP implementation delay, and SIP termination; (b) building air-exchange rates, including air infiltration and ventilation; (c) the degree of sorption of toxic chemicals to indoor surfaces; and (d) the shape of the dose-response relationship for acute adverse health effects. Houses with lower air leakage are more effective shelters, and thus variability in the air leakage of dwellings is associated with varying degrees of SIP protection in a community. Sorption on indoor surfaces improves SIP effectiveness by lowering the peak indoor concentrations and reducing the amount of contamination in the indoor air. Nonlinear dose-response relationships imply substantial reduction in adverse health effects from lowering the peak exposure concentration. However, if the scenario is unfavorable for sheltering (e.g. sheltering in leaky houses for protection against a nonsorbing chemical with a linear dose-response), the community must implement SIP without delay and exit from shelter when it first becomes safe to do so. Otherwise, the community can be subjected to even greater risk than if they did not take shelter indoors.

Keywords: sorption, infiltration, air-exchange rate, toxic chemical, emergency response.

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1   Introduction

In the event of a sudden, large-scale release of a toxic chemical into the atmosphere, nearby populations may be advised to take shelter indoors. Shelter-in-place (SIP) requires simply being indoors, closing doors and windows, and turning off fans. Broadly, SIP offers protection through two modes. First, lower peak indoor concentrations compared to outdoor concentrations are encountered after a short-term release because building envelopes limit indoor-outdoor air exchange. Second, sheltering may reduce cumulative exposures through one or more mechanisms, including transformations of toxic chemicals on building surfaces and time-dependent manipulation of ventilation rates.

The effectiveness of SIP for protecting public health can depend upon many factors. Attributes of the release, such as the quantity emitted, toxicity of the chemical, release duration, and atmospheric transport and dispersion, influence the effectiveness of protection. Building characteristics, including leakiness of the building envelope and the interactions between the toxic chemicals and building surfaces, also play an important role. Finally, human factors such as delays in notification and response, and the timing of shelter termination, can impact the effectiveness of the strategy.

Most prior studies of SIP effectiveness focused on characterizing single residences (Engelmann, 1992; Siren, 1993). Existing software that assesses indoor concentrations during an atmospheric release event, such as ALOHA (Areal Locations of Hazardous Atmospheres), assumes a typical air leakage value for all low-rise buildings (US EPA, 2006). However, studies of air leakage reveal substantial variability among residential buildings (Chan et al., 2005). This variability can lead to considerable differences in their ability to protect building occupants against outdoor releases. Vogt et al. (1999) speculate that residences constructed before 1970 may be more leaky then newer residences, owing to changes in building codes and construction practices. As a result, older dwellings may be less fit to function as emergency shelters. This dependency is supported by the detailed analysis presented in Chan et al. (2005).

As toxic chemicals penetrate building envelopes, some fraction might be lost to surfaces of the air leakage pathways, thus reducing the exposure of building occupants. However, only limited evidence is available to assess the significance of this mechanism (Karlsson, 1994; Liu and Nazaroff, 2001). Exposure during SIP may also be influenced by the interactions of toxic chemicals with indoor surfaces, including building materials and furnishings. Such interactions can occur through several mechanisms including redox reactions, acid-base reactions, hydrolysis, and sorption. Of these, the most thoroughly studied is sorption, which refers to the reversible or irreversible binding of a chemical to the surface, or within the bulk, of a material (Karlsson and Huber, 1996; Singer et al., 2005a). Sorptive uptake can be fast relative to the air-exchange rate, thus it can effectively reduce the peak indoor concentration. The extent to which sorptive uptake occurs depends on properties of the toxic chemical in relation to the indoor materials. Because of differences in interior materials, variability in the rates of this process would be expected across a building stock.

SIP effectiveness is sensitive to time-scale parameters. If many residences have open windows and doors at the time of the release, SIP effectiveness can be impaired owing to the time required to alert the community to close all windows and doors. Rogers (1994) compiled interview data on when decisions were made by key officials to warn the public following a release event. After the decision to warn is made, there can be another time delay before people comprehend the warning and take appropriate action. Community receipt and response times of different emergency warning systems have been investigated using post-event survey data (Rogers and Sorensen, 1989; 1991). The time required for each step can vary considerably. The overall impact of delay in sheltering on SIP effectiveness also depends on the initial ventilation conditions of the dwellings.

The present paper is the second in a series that seeks to advance the understanding of community-scale SIP effectiveness and the factors that affect it. In the first paper (Chan et al., 2006), we conducted a parametric investigation using idealized representations of the system. Two new metrics were introduced to quantify SIP effectiveness – the casualty reduction factor

(CRF) and the safety factor multiplier (SFM). SIP effectiveness was assessed using three coupled models to quantitatively link release conditions with adverse health consequences: a Gaussian-plume dispersion model, an indoor-outdoor contaminant transport model, and a toxic-load dose-response model. This analysis revealed that SIP effectiveness varies significantly with the toxic load exponent in the dose-response model and the time-scale parameters, namely the release duration and the building air-exchange rate.

The purpose of this second paper is to assess SIP effectiveness in a residential community using more realistic transport and transformation models and input parameters. The study is restricted to single-family detached housing units, which represent the majority of the US housing stock. Hypothetical releases with various characteristics are simulated using an operational atmospheric transport and dispersion model driven by real meteorological data. Distributions of weather-driven air infiltration rates are predicted by incorporating variability in the air leakage among dwellings. Interactions of chemicals with indoor surfaces are modeled to capture the influence of sorptive interactions on SIP effectiveness. The effects of delays in initiating and terminating SIP are investigated. Because of the complexity of the simulations, this analysis encompasses only a limited sets of release conditions. However, the case studies are constructed such that many of the findings discussed here should apply to other residential communities taking shelter from large-scale outdoor releases.

2 Methods

2.1 Case Study

Three hypothetical releases of different duration (0.5, 1, and 2 h) are modeled for an urban residential district of a city that we will refer to as City A. Scenarios are chosen to represent large-scale toxic industrial chemical releases that pose significant health risks to downwind residences. The outdoor concentrations are simulated using an atmospheric dispersion model known as the Lagrangian Operation Dispersion Integrator (LODI), as operated by the National Atmospheric Release Advisory Center at Lawrence Livermore National Laboratory.

The model solves the 3-D advection-diffusion equation using a Lagrangian stochastic Monte Carlo approach (Ermak and Nasstrom, 2000). LODI can simulate the effects on pollutant concentrations of mean wind advection, turbulent diffusion, buoyant plume rise, and pollutant dynamics such as first-order chemical reactions and wet and dry deposition. In the simulations presented here, pollutant dynamics are not included, meaning that the toxic chemical is treated as a conserved species in outdoor air.

Fig. 1 depicts the predicted outdoor concentration for one of the hypothetical releases, which took place on a cold winter evening with moderate wind. The predominant easterly wind causes the plume to pass through a residential district west of the city. The other two releases are modeled to occur at the same location and begin at the same time. The plotted concentrations represent 5-minute average values for a grid cell resolution that varies from 34 m near the source to 1.8 km at the most distant location. The release is modeled to occur 5 m above ground to represent a slightly elevated point source (e.g. top of a ruptured tank). The outdoor concentrations in the presented analyses are evaluated at 10 m height. The mass release rate is assumed to be uniform throughout the release duration. Along the plume centerline, outdoor concentrations vary by several orders of magnitude over a downwind distance of 10 km.

The toxicity of the released chemical is approximately equivalent to chlorine gas. The released mass of each simulation is scaled to produce the same number of potential casualties estimated for outdoor exposure. Releases on the order of 1 to 10 tonnes are thus simulated. In the event that people are exposed outdoors, severe acute adverse health effects are predicted to occur up to a distance of 5 to 7 km downwind from the release location. Adverse effects of exposure to the plume in the crosswind direction extend from 1 to 3 km from the plume centerline. Even though all simulations are scaled to produce the same number of potential outdoor casualties, locations where the casualties occur differ among the cases because of changes in wind direction as the event proceeds. In all cases, the outdoor concentrations were simulated for 4 h from the start of the release. At the end of the simulation the toxic plume had advected beyond the model domain, and the outdoor concentrations of the dispersed plume were no longer dangerous.

2.2 Air Infiltration Rate Distribution

When doors and windows are closed, and fans are off, building air exchange occurs by uncontrolled air leakage across the building envelope, a phenomenon known as air infiltration. The LBL infiltration model (Sherman and Grimsrud, 1980) predicts the rate of airflow, Q (m^3 s^{-1}), through a building envelope driven by indoor-outdoor temperature difference, ΔT (K), and wind speed, U ($m\ s^{-1}$), for a small detached building.

$$Q = \text{ELA} \cdot \sqrt{f_s^2 \cdot |\Delta T| + f_w^2 \cdot U^2} \tag{1}$$

Here, f_s ($m\ s^{-1}\ K^{-0.5}$) and f_w (-) are the stack-effect factor and the wind-effect factor, respectively, and ELA (m^2) is the effective leakage area of the building. The extent of shielding from nearby obstacles and local terrain affect the value of f_w. For residential neighborhoods situated in an urban area, moderate shielding and urban terrain class are reasonable choices. Both f_s and f_w also depend weakly on the geometry and the distribution of leakage area over the building envelope. Under the assumption that half of the total leakage area is attributable to the vertical walls of the building, and that there is little difference between the air leakage associated with the ceiling and the floor, the values of f_s and f_w are each roughly 0.15 for a single-story house in a residential urban area with moderate shielding. The outdoor (T_o) and indoor temperatures (T_i) are assumed to be uniform within the model domain. For the present cases, T_o decreased from 4 ^{o}C at the start of the release to -1 ^{o}C by the end of the 4 h simulation, while T_i was assumed to be constant at 20 ^{o}C. The wind speed at most locations varied between 2 and 4 $m\ s^{-1}$, but some areas experienced high wind momentarily reaching 7 m s^{-1}.

Normalized leakage (NL) is ELA (expressed in units of cm^2) divided by the building floor area (m^2) and by a correction factor for building height. Air leakage measurements collected from houses across the US reveal that the NL of houses tends to increase with age, decrease with floor area, and to be higher for low-income than for not low-income households (Chan et al., 2005). Most single-family dwellings in the US have NL in the range 0.2 to 2 cm^2

m^{-2}. Based on these findings, we estimate the air leakage distribution of dwellings in City A by the characteristics of the housing stock. Census tract was chosen as the geographical unit of analysis because it permits good data access along with a reasonable compromise between the number of units to analyze and the spatial resolution of the results. City A is comprised of roughly 140 census tracts, each typically containing 1000 houses, with a density of 10 to 100 houses per km^2.

The procedure to predict the air leakage distribution of houses in a census tract is briefly summarized here; see §3.4.2 in Chan (2006) for details. Houses in each census tract are first divided into 2 sets based on household income: above and below the poverty limit. Each set is further divided into groups according to house year-built and floor-area. The distribution of NL for each group of houses is predicted by its age, size, and household income status using a regression model derived from analysis of US nationwide air leakage measurements (Chan et al., 2005). The number of houses in each group is extracted from data collected in the US Census Survey (US Census, 2002) and American Housing Survey (US HUD, 2002). The composite NL distribution for the census tract is the sum of all distributions weighted by the number of houses in each group. The composite distribution, which describes the air leakage of houses in each census tract, is then converted to units of ELA. Finally, the distribution of Q is predicted using Eq. (1) as a function of time at each grid cell in the model domain.

The predicted air infiltration rates, defined as Q normalized by the house volume V (m^3), vary with time following a pattern similar to the change of wind speed during the simulation (Fig. 2). In this case study, the neighborhoods that are located closer to the release site have higher poverty rates and tend to have older and smaller dwellings. Consequently, the median air infiltration rates predicted there are about twice as high as in locations that are substantially downwind. Besides these spatial differences, Fig. 2 also shows the predicted variability at the two locations. Because air leakage of houses is roughly lognormally distributed (Chan et al., 2005), leaky houses in the top 10th percentile are predicted to have air infiltration rates reaching

1–2 h^{-1}. These tail-end estimates are much higher than the median value (0.6 h^{-1}) predicted for houses in the model domain for this simulation.

2.3 Sorption on Indoor Surfaces

Sorptive uptake of toxic chemicals on indoor surfaces and desorption from indoor surfaces modify the time profile of indoor concentrations. The two-sink model of Singer et al. (2005a) captures the pollutant dynamics in a room by partitioning the chemical into three compartments: the room air, a surface sink, and an embedded sink. Chemicals in room air would first sorb onto the surface sink. Chemicals on the surface sink could then diffuse into the bulk material or through pores to hidden surfaces, which are collectively modeled as the embedded sink. Attachment to the surface and embedded sinks are each potentially reversible. The embedded sink interacts with the surface layer only, and not directly with the room air. In this model representation, the complex combination of materials that would be found in any real room is represented as a single effective material. Conservation of pollutant mass in the room air (C_{in}), on the surface sink (M) and in the embedded sink (E) yields three governing equations:

$$\frac{dC_{in}}{dt} = \frac{Q}{V} \cdot \left(C_{out} - C_{in}\right) - k_a \cdot C_{in} + k_d \cdot M$$
$$\frac{dM}{dt} = k_a \cdot C_{in} - \left(k_d + k_1\right) \cdot M + k_2 \cdot E \qquad\qquad (2)$$
$$\frac{dE}{dt} = k_1 \cdot M - k_2 \cdot E$$

The mass in each compartment is normalized by the indoor volume, V, such that C_{in}, M, and E are each expressed in units of g m^{-3}. The rate coefficients k_a, k_d, k_1, and k_2 all have units of h^{-1}.

The present analysis considers both strongly sorbing and moderately sorbing compounds. The rate coefficients used are shown in Table 1. Parameters for dimethyl methylphosphonate (DMMP), a surrogate for nerve gas sarin, are used to represent chemicals that sorb strongly onto surfaces (Singer et al., 2005b). Parameters for NH$_3$ are used to represent chemicals that sorb moderately (Karlsson and Huber, 1996). A one-sink model is adequate to describe a moderately sorbing toxic chemical, where k_1, k_2, and $E(t)$ are all set to zero.

2.4 Shelter-in-Place Response Time

Implementing SIP in response to a release includes three steps, each associated with a time delay: (1) the time required for officials to issue a warning to the public, (2) the time required for the warning to substantially disperse throughout the population, and (3) the time required for people to effectively respond. These delays can vary considerably depending on the release circumstances. Table 2 lists ranges and typical times for each step as distilled from the literature. Values chosen for step (1) span the range reported in six past accidents that involved an SIP response, according to a study by the National Institute of Chemical Studies (NICS, 2001). Values chosen for step (2) represent the time needed for 50% of the population to receive a warning given out by three methods: sirens plus telephones, sirens only, and media broadcast (Rogers and Sorensen, 1991). Values chosen for step (3) are derived from survey data for an evacuation event, from which Rogers et al. (1990) made adjustments to estimate SIP response time for cases of accidental chemical releases. In the analyses presented here, we assume that all households respond similarly to the warning message. To further simplify the treatment, only three SIP initiation times are modeled based on the total time required for the three steps: 0.25, 0.5, and 1 h from the start time of the release.

Before SIP is initiated, some households in a community could have elevated air-exchange rates owing to open windows or operation of exhaust fans or a central air handling system. The prevalence and extent of window opening in a community depends upon weather conditions. Studies indicate that as many as 35% of households have some windows open when outdoor temperatures are comfortable (Johnson and Long, 2005; Price and Sherman, 2006). Operation of exhaust or heating and cooling system fans can also increase air-exchange rates, but likely to a lesser extent than open windows (Wallace et al., 2002). In the analysis presented here, three cases are considered: (1) infiltration only; (2) 80% of households have an additional 0.3 h^{-1} air-exchange above infiltration to represent the condition where many residences have a central heating system operating or some other minor source of induced air exchange; (3) 40% of households have an additional 1 h^{-1} air-exchange above infiltration to represent the condition

where some residences have open windows. These cases represent a distillation of empirical

evidence regarding air-exchange rates in residences (Wilson et al., 1996; Howard-Reed et al.,

2002; Johnson et al., 2004); however, issues such as between-dwelling variability and interaction

of enhanced air-exchange with air infiltration are not considered.

2.5 Shelter-in-Place Effectiveness Metrics

Table 3 summarizes the model parameters used in this paper to assess SIP effectiveness.

For each simulation, SIP effectiveness was quantified in terms of two metrics: the casualty

reduction factor (CRF) and the safety-factor multiplier (SFM). In brief, CRF represents the

fractional reduction in the expected number of casualties for populations sheltering indoors as

compared with being exposed outdoors. The SFM is the multiplicative extent to which sheltering

enhances the safety factor (SF) of an exposed individual. The safety factor can be interpreted as

the maximum factor by which the exposure concentration could be multiplied without the

exposed individual being subjected to potential adverse health effects. CRF and SFM are

computed as follows (see Chan et al., 2006 for details):

$$\text{CRF} \;=\; 1 - \frac{\text{Population}\,(\text{TL}_{in} > \text{TLL})}{\text{Population}\,(\text{TL}_{out} > \text{TLL})} \tag{3}$$

$$\text{SFM} = \frac{\text{SF}_{in}}{\text{SF}_{out}} = \frac{\left(\dfrac{\text{TLL}}{\text{TL}_{in}}\right)^{\frac{1}{m}}}{\left(\dfrac{\text{TLL}}{\text{TL}_{out}}\right)^{\frac{1}{m}}} = \left(\frac{\text{TL}_{out}}{\text{TL}_{in}}\right)^{\frac{1}{m}} \tag{4}$$

CRF is defined at the community level, whereas SFM is evaluated at each grid cell. Both CRF

and SFM can be evaluated as functions of time as the release proceeds. High values of CRF

(upper limit = 1) and SFM (no upper limit) indicate effective SIP. In these equations, the toxic

load limit, TLL [$(\text{mg m}^{-3})^{m}$ h], represents a threshold, above which an adverse health effect might

result. The TLL used in this paper is comparable to that of acute exposure to chlorine leading to

severe health effects (NRC, 2003).

Both CRF and SFM require computation of the toxic load (TL), which incorporates the potentially nonlinear dose-response effect of acute exposures to toxic chemicals (ten Berge et al., 1986):

$$\mathrm{TL}(t) = \int_0^t \left(C(t') \right)^m dt' \qquad (5)$$

For an exposure event beginning at time $t = 0$, $C(t)$ is the time-dependent breathing-zone concentration, and m is an empirical parameter referred to as toxic load exponent. The toxic load accumulated if one were exposed to the outdoor or indoor concentration are referred to as TL_{out} or TL_{in}, respectively. At each grid cell, TL_{in} is computed using indoor concentrations evaluated at different percentiles of the air leakage distribution. Finally, we assume that the population density within any given census tract (obtained from US Census) is uniform when evaluating CRF and the distribution of SFM. Note that although we explore only three integer values of m (Table 3), empirically determined values of m can be non-integers. Also, the results reported for a particular integer value of m would be similar to the results for toxic chemicals with values of m in the vicinity of the reported value.

3   Results and Discussion

3.1 Effects of Air Leakage Variability

Large variability in air leakage across the housing stock means that while tighter residences might provide sufficient protection for occupants, leakier residences may not. The result of this variability is illustrated in Fig. 3. In this case, it is assumed that the chemical is conserved and nonsorbing, that people initiated SIP immediately upon chemical release, and that the dose-response relationship is linear ($m = 1$ in Eq. (5)). Adverse health effects are evaluated at the end of the 4-h simulation. All residents are assumed to remain indoors for the entire period. Fig. 3 shows that in areas within a few km downwind of the source, indoor toxic loads in most residences would exceed the toxic load limit, regardless of the leakiness of the building envelope. On the other hand, in areas further downwind of the release, most people would be effectively

protected except those sheltering in leaky residences. The fraction of the population at risk of adverse health effects at each grid cell is determined by the condition $TL_{in} > TLL$. We assume that the population in each grid cell is uniformly distributed among houses. As a result, the distribution of toxic loads for individuals matches that of indoor toxic loads. For example, if 10% of the houses have indoor toxic loads higher than the TLL, then we expect 10% of the population to be exposed to toxic loads exceeding the TLL.

Variability in air leakage among houses also explains differences in the calculated safety-factor multiplier. The distribution of SFM is evaluated at each grid cell that encounters non-zero outdoor concentration using Eq. (4). Fig. 4 shows the aggregate distribution of SFM across grid cells for the same 1-h release scenario considered in Fig. 3 evaluated at the end of the simulation. Houses that are classified as "tight", "typical", and "leaky" in Fig. 4 are those with air-leakage at the 5th, 50th, and 95th percentiles of the distribution, respectively. For the conditions of this hypothetical release, Fig. 4 shows that the tightest houses can provide effective protection (SFM ~ 2–3), whereas the leakiest houses provide minimal protection (SFM ~ 1). Owing to this difference in the level of protection offered among houses, those who shelter in leaky houses can be at risk of adverse health effects even far downwind of the release. The overall SFM distribution for the entire exposed community is skewed (see "All Houses" in Fig. 4). While some residences provide values of SFM exceeding 2, most only reach values between 1.1 and 1.6 given the release scenario and SIP strategy considered in this case. Thus, one must account for air-leakage variability to accurately describe SIP effectiveness for a residential community.

Fig. 5 shows the difference in potential casualty predictions for two cases: (a) all houses are assumed to have uniform air leakage equal to the median value in the census tract; and (b) the full distribution of leakage in the housing stock is taken into account. Casualty underpredictions on the order of 5–20% are observed for the uniform leakage case. This is a result of ignoring houses that are much leakier than the median. The difference between the two curves becomes small at times long after the release has stopped. This is expected for the case $m = 1$, because adverse health effects are determined by the time-integrated indoor concentration. For a

conserved pollutant, the eventual value of this integral is insensitive to the air-exchange rate so long as the rate does not change significantly with time. For chemicals with $m > 1$, casualty underpredictions from treating building air leakage as uniform at the median value can persist long after the release has ended.

3.2 Effects of Sorption and Response Time Delay

Sorption on indoor surfaces reduces peak indoor concentrations relative to the case of a nonsorbing chemical. For a 1-h release and for the sorption parameters considered here, the reduction in peak indoor concentration is in the range 15–65%. A slightly larger reduction is observed when the air infiltration rate of the house is lower. This is because fast exfiltration of the indoor air contaminant to the outdoors competes with sorption of chemicals onto surfaces, since both processes reduce indoor air concentrations. After the ambient plume has dispersed, reversibly sorbed chemicals slowly desorb from surfaces and reenter the room air. Compared to the case of nonsorbing chemicals, the indoor concentrations after the toxic plume has passed are less sensitive to the air-exchange rate when reversible sorption occurs. Similar low levels of residual chemicals are predicted in buildings with different air leakage. Strong sorption also causes the indoor concentration in leaky buildings to be consistently higher compared to buildings with lower air infiltration rate. This outcome differs from the case of nonsorbing chemicals, where the indoor concentrations in the leakiest buildings will decline the fastest after the outdoor plume has passed. The difference occurs because strong sorption causes a large amount of the toxic chemical to accumulate on surfaces in leaky buildings. Desorption then can mostly compensate for loss by exfiltration, keeping the indoor concentration high for a long time after the toxic plume has passed. As a result, the indoor concentrations are sustained at a slightly higher level relative to buildings with lower air infiltration rates.

As illustrated in Fig. 6, SIP is more effective for sorbing chemicals than for nonsorbing chemicals, and also more effective for chemicals that exhibit strong sorption than for moderately sorbing species. The upper frames show that the casualty reduction factors exceed 0.75 for

sorbing chemicals in all the scenarios modeled. The lower frames illustrate the interaction between the toxic load exponent (m) and the strength of sorption in influencing the safety factor multiplier. A key to SIP effectiveness, especially when the toxic load exponent is high, is to reduce the peak exposure concentrations. In response to ambient plumes of short duration (i.e., release duration shorter than the inverse of the building air-exchange rate), both slow air-infiltration and reversible sorption act to reduce peak indoor concentrations. Comparing like conditions in Fig. 6, the median SFM is increased by a factor of 2–3 for $m = 3$ compared with $m = 1$. For $m = 3$, the interquartile range of air-exchange rates influences SFM by a factor of 2–3 also. Finally, the difference between strong sorption and no sorption causes another factor of 2–3 difference in SFM. The overall result is that the safety factor multiplier spans well more than an order of magnitude for the range of scenarios displayed in Fig. 6.

The delay in SIP termination substantially affects SIP effectiveness only if $m = 1$ and if the chemical does not sorb onto indoor surfaces. Even if sorption of chemicals to indoor surfaces is reversible, the amount of a toxic chemical that desorbs from surfaces into indoor air is relatively small over the course of a few hours after the release has stopped. As a result, SIP effectiveness remains essentially unchanged with respect to SIP termination time for sorbing chemicals, as is illustrated by the CRF values shown in the top frames of Fig. 6.

If SIP is not implemented until after a release has already begun, toxic chemicals that entered the building at high concentration during a period of elevated air-exchange rate can be trapped indoors because people shut their windows or stop operating fans that had induced the additional air exchange above infiltration. The importance of prompt SIP initiation depends on a number of factors. The longer the release duration, the longer the toxic load is accumulated. As a result, the penalty of having an initial period of fast air exchange with the outdoors becomes less significant to the overall exposure for releases of long duration. Proximity of the exposed population to the release location also matters. At locations close to the release site, the outdoor concentrations rise rapidly and reach a peak value soon after the release begins. As a result, delaying SIP close to the release can cause significant increases in indoor concentrations and

worsen the health consequences. For a 1-h release, delaying SIP by 0.5-h can decrease the SFM

values (evaluated at $m = 1$ and no sorption) by 10–40% at locations where the plume arrives

within the first 5 minutes of the release. At locations further downwind where the arrival of the

plume lags the onset of the release, the SFM values are less affected by initiation time delay.

Fig. 7 shows the combined effect of initiation and termination delays on SIP effectiveness

for the case of a nonsorbing chemical. Two pre-sheltering conditions are investigated: (1) many

households operate their heating system or have other minor sources of induced air-exchange,

and (2) some households have open windows. Incidentally, the two pre-sheltering conditions

considered yield similar results, even though the fraction of affected residences and the amount

of additional air-exchange rate modeled are quite different. Fig. 7 shows that the drop in CRF

value as a result of delayed initiation of SIP is significant only when coupled with a long delay in

termination. Furthermore, it is only in the case where the dose-response is linear ($m = 1$) that the

combined effect of the two sources of time delay substantially impacts SIP effectiveness. Even

under these conditions, so long as SIP is implemented successfully before the release has stopped,

the degradation in effectiveness associated with initiation delay is predicted to be small. For

example, the same 1-h initiation delay lowers SIP effectiveness more significantly in the case of

shorter 0.5-h releases, relative to longer 2-h releases (compare the top-left frame with the

bottom-left frame of Fig. 7). If the initiation of SIP is long delayed, and if the released chemical

has a linear dose-response ($m = 1$) and is nonsorbing, then SIP must be terminated punctually at

the end of the release to avoid considerable loss in effectiveness. This is illustrated by the large

drop in CRF values in the top-left frame of Fig. 7 as a function of termination time.

3.3 Is Shelter-in-Place Always Beneficial?

There are some conditions under which SIP could be ineffective. Yet, three aspects of

SIP help counteract the risk of doing more harm than good. First is the effect of nonlinear dose-

response ($m > 1$). The middle and right-hand columns of Fig. 7 show that when $m = 2$ or 3, SIP

initiation time delay has little effect on the casualty estimates. The second factor is termination

time. Prompt termination can mitigate the impact of a delayed start. Terminating SIP soon after it is safe to do so substantially eliminates the loss in effectiveness caused by a delay in initiation time. By terminating SIP promptly, people would no longer be exposed to high levels of toxic chemicals trapped indoors. Consequently, even for the case $m = 1$ (Fig. 7, left frames), elevated amounts of chemicals entering indoors owing to late SIP initiation would not cause as much adverse health effects to the occupants if prompt termination can be achieved.

Sorption to indoor surfaces is the third factor that can potentially offset the loss in effectiveness caused by an initiation time delay. Fig. 8 shows the predicted potential casualties if people were to take shelter 1 h after the start of a hypothetical release for three sorption cases: none, moderate and strong. For these cases, adverse health effects are evaluated for $m = 1$ only, where SIP effectiveness is the most sensitive to the parameters considered. Before SIP is initiated, it is assumed that 40% of the residences have increased air-exchange (1 h^{-1} above infiltration) from open windows. For all release durations considered, sorption on indoor surfaces at a moderate rate is sufficient to sustain SIP effectiveness. Even in the worst-case scenario where SIP was not implemented until after the release has already stopped (i.e. a 0.5-h release duration with a 1-h SIP initiation time delay), as long as the toxic chemical is at least moderately sorbing, SIP would not cause more harm to the community relative to outdoor exposure (i.e. CRF > 0). The predicted SFM values also exceed 1 at all grid cells for these conditions, meaning that no one is expected to accumulate toxic load higher than the corresponding outdoor level even if they did not close their windows until an hour after the release has started. On the other hand, if the toxic chemical is nonsorbing indoors, about 50% of the affected population can be worse off than if they were exposed to the outdoor levels (i.e. $TL_{in} > TL_{out}$). For the community as a whole, however, some benefit from sheltering can still be realized, especially among parts of the neighborhood that are further downwind of the release site but are still within the area of impact. Residents sheltering in tighter buildings are also better protected. Factors such as the proximity of the population to the release location, and the speed of the advecting plume, also

affect how sensitive SIP effectiveness is with respect to the response time delay of the community.

4   Conclusion

In planning for emergency situations, model predictions can provide officials with expectations about the effectiveness of a response strategy. The analysis presented here explored several factors that can affect SIP effectiveness. Some of these factors are controllable, such as the response time of the community to take shelter; others are inherent of the situation, such as the release conditions and the chemical properties. We estimated the influence of these factors on SIP performance using a case-study approach. Distributions of air infiltration rates were modeled using housing characteristics from an urban residential area and local meteorology. The consequent variability in indoor concentrations was modeled both temporally and spatially for releases of different durations and with different dose-response relationships. Adverse health effects are estimated for the exposed community by taking this variability into account. Houses that are older, smaller, and occupied by low-income households tend to have higher air leakage, and as a result they provide less effective shelter. As part of a community preparedness effort, attention should be paid to these vulnerable dwellings, especially those located close to a potential release sources.

The influence of sorption of toxic chemicals to indoor surfaces and of time delays associated with implementing the SIP strategy was evaluated using two measures of effectiveness: casualty reduction factor (CRF) and safety-factor multiplier (SFM). Even for chemicals that sorb only moderately to indoor surfaces, sorption is sufficient to offset the need for timely termination of SIP, and to counteract the loss in effectiveness caused by SIP initiation delay. Yet, in certain scenarios, improving the response time of the community when instructed to take shelter can be key to a successful strategy against adverse health effects from exposure to a toxic outdoor release. For example, if the released chemical is nonsorptive and its dose-response is linear, emergency responders and authorities must (1) ensure that people take shelter

quickly, and (2) give all-clear signal as soon as it is safe. Otherwise, the community can run the risk of being worse off with SIP than without it.

The case study presented here considers a limited set of scenarios typical of a large-scale release in a moderately dense urban neighborhood. In situations where the parameters deviate far from those modeled in this analysis, SIP effectiveness might differ. The level of protection considered here is easily achievable under current conditions in typical residences. Proactive measures, such as using stand-alone air cleaners (Ward et al., 2005) or duct tape and plastic sheets (Jetter and Whitfield, 2005), might improve effectiveness. However, they can be costly and time consuming to deploy, which might limit their utility for community-scale application. Furthermore, this analysis is restricted to single-family detached dwellings. Other forms of residences, such as townhouses and apartment buildings, and other buildings, such as offices and schools, can have air leakage characteristics that are significantly different (Price et al., 2006; Sherman and Chan, 2006). The airflow in these buildings also tends to be more complex owing to their larger sizes and the flow resistance of internal partitions. These types of buildings constitute a large fraction of the building stock, especially in urban areas. A large proportion of a potentially exposed population might be at schools, workplaces, and commercial establishments rather than in their homes at the time of a release. For these reasons, knowing the characteristics of these buildings is also essential to evaluating SIP effectiveness for an exposed community. We intend to address these issues in a future article on SIP effectiveness in commercial buildings.

There are other aspects of this analysis that warrant more detailed consideration in the future. Some chemicals can have other important reactions or removal pathways indoors that are not considered in this analysis. Variability in human susceptibility in response to acute exposure to toxic industrial chemicals and chemical warfare agents (Griffiths and Megson, 1984; Sommerville, 2003) might substantially influence casualty estimates and predicted areas at risk of adverse health effects. Stochastic fluctuation in the outdoor concentrations not captured by the atmospheric dispersion model implies that SIP is likely to be more protective than the results presented in this analysis (Hilderman et al., 1999; Yee, 1999). The present analysis also assumed

that the entire community was already indoors at the time of the release and all would follow the instruction to shelter-in-place without exception. The validity of these two assumptions might vary depending on the time or day of the release, and the degree of emergency preparedness of the community. In situations where short initiation time delays are important, the distribution of response time in a community and the rate of non-compliance should be modeled explicitly.

Acknowledgement

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Figure Captions

Fig. 1. Outdoor ground-level concentrations of a toxic chemical during and after a hypothetical 2-h release in City A. The concentrations plotted are 5-minute averages predicted at 0.5, 1.5, and 2.5 h since the start of the release. Predictions were made in units of g m^{-3}, but values displayed have been normalized to the highest ground level concentration of the simulation. Also shown are boundaries of census tracts (thin grey lines), major roadways (thick grey lines), and a river (blue line).

Fig. 2. Predicted distribution of air infiltration rates at two downwind sites from the release source. The open symbols represent the predicted median values, which vary as a function of time in response to the driving forces for air infiltration: indoor-outdoor temperature difference and wind speed. The lengths of vertical bars, with tick marks showing the 5th, 10th, 90th, and 95th percentiles, reflect the predicted variability of the distribution. Elevated infiltration rates close to the release are a result of the smaller size, greater age, and higher prevalence of low-income housing in the urban core of City A than in the area further downwind.

Fig. 3. Predicted areas where indoor exposure is expected to exceed the toxic load limit (TLL) for a 1-h release of nonsorbing chemical under linear dose-response ($m = 1$). The fraction of the population at risk of adverse health effects is computed using indoor exposures estimated at specific percentiles of the air-leakage distribution. People are assumed to have implemented SIP immediately at the outset of the release and maintain it throughout the 4-h simulation.

Fig. 4. Predicted distribution of safety-factor multiplier (SFM) for people who shelter in houses at the tightest and leakiest 5th percentiles ("tight" and "leaky"), and in houses at the median of the air-leakage distribution ("typical"), for the entire 4-h simulation. SFM values are evaluated at each grid cell, and the distributions are weighted by the number of exposed individuals. The rightmost boxplot incorporates the variability in air-leakage of houses and gives the expected distribution for the entire exposed community. Simulation conditions are the same as in Fig. 3.

Fig. 5. Casualty estimates for a hypothetical release event as a function of time comparing population exposure outdoors and exposure indoors (1) if all houses have an air leakage equals to the median value, or (2) if houses have different air leakage described by a distribution similar to

that shown in Fig. 2. All casualty estimates, evaluated using a linear dose-response ($m = 1$), are normalized to the maximum outdoor values. For the indoor exposures, people are assumed to have implemented SIP at the start of the release and to have maintained SIP throughout the 4-h simulation. The toxic chemical is assumed to be nonsorptive.

Fig. 6. Influence of sorption to indoor surfaces on SIP effectiveness measured in terms of the casualty reduction factor (CRF) and safety-factor multiplier (SFM). Twenty-seven scenarios are modeled in a $3 \times 3 \times 3$ format: (a) release durations of 0.5, 1, and 2-h; (b) linear ($m = 1$) and nonlinear ($m = 2$ and 3) dose-response relationships; and (c) nonsorbing, moderately sorbing, and strongly sorbing chemicals. CRF values are estimated at different SIP termination times: 0.5, 1, and 2 h after the end of the release. The distributions of SFM are estimated at 2 h after the release has stopped. (See Fig. 4 for the interpretation of boxplots to express the SFM distributions.)

Fig. 7. Sensitivity of SIP effectiveness to initiation and termination time for various release scenarios. In each simulation, four initiation times are modeled: no delay (0 h), 0.25 h, 0.5 h, and 1 h delay after the start of the release. In each case, two sets of pre-sheltering air-exchange rates are considered: Case (1) 80% of houses have an increase in air-exchange rate of 0.3 h^{-1} above infiltration before SIP is initiated; and Case (2) 40% of houses have an increase in air-exchange rate of 1 h^{-1} above infiltration. Casualty reduction factors (CRF) are evaluated at 0.5, 1, and 2 h after the end of the release. The released chemical is assumed to be nonsorbing.

Fig. 8. Predicted SIP effectiveness in the event that sheltering is delayed by 1 h from the start of the release event. SIP effectiveness is presented in terms of CRF in relation to release duration, degree of sorption onto indoor surfaces, and termination time delay. The distributions of SFM are estimated at 2 h after the release has stopped. Before SIP is initiated, 40% of the residences are assumed to have an additional 1 h^{-1} air-exchange to represent the effect of induced ventilation through open windows. Since SIP effectiveness for the case of linear dose-response is most sensitive to initiation time delay (see Fig. 7), only these cases are considered in this comparison.
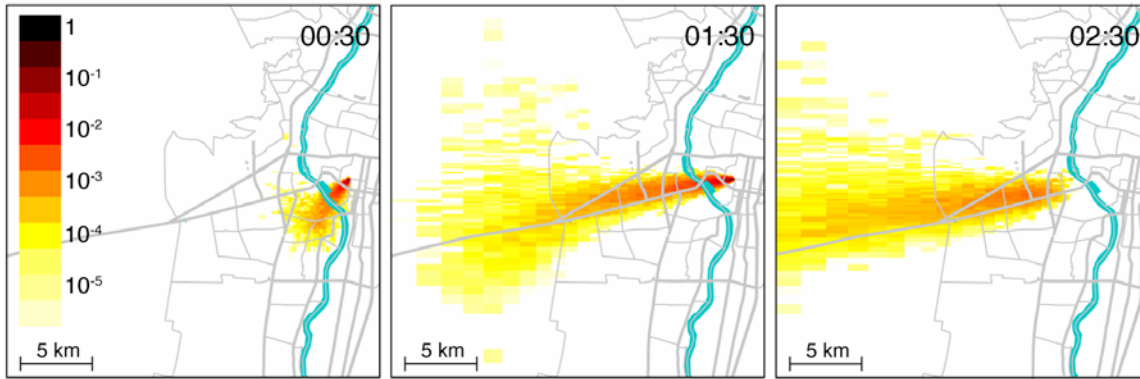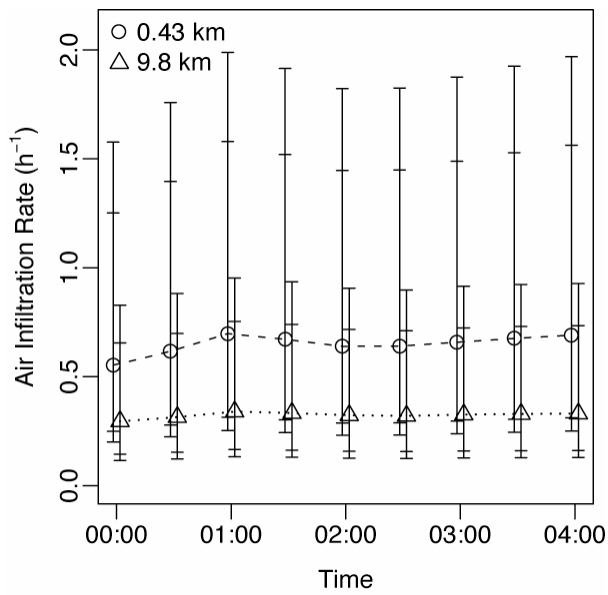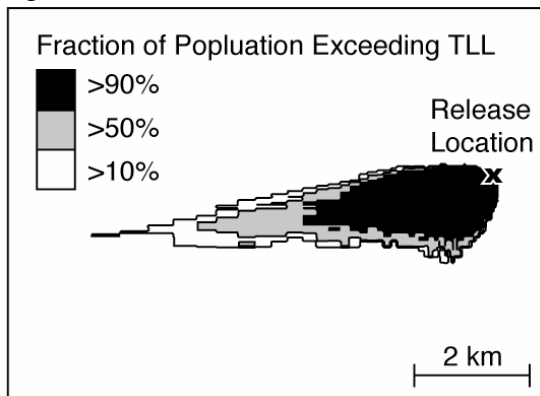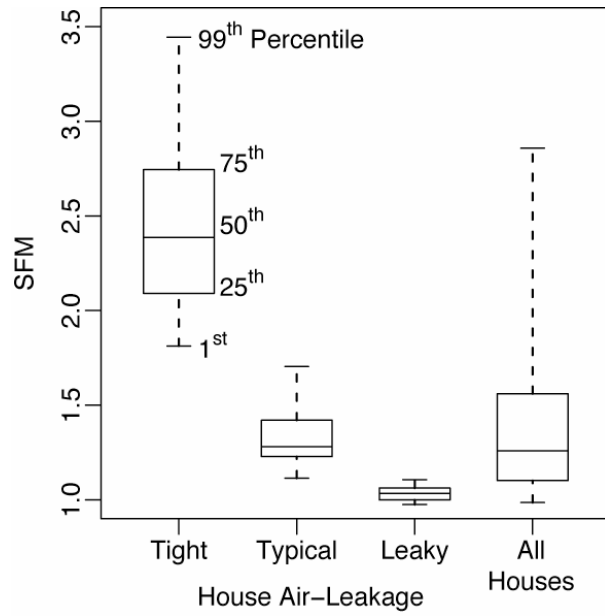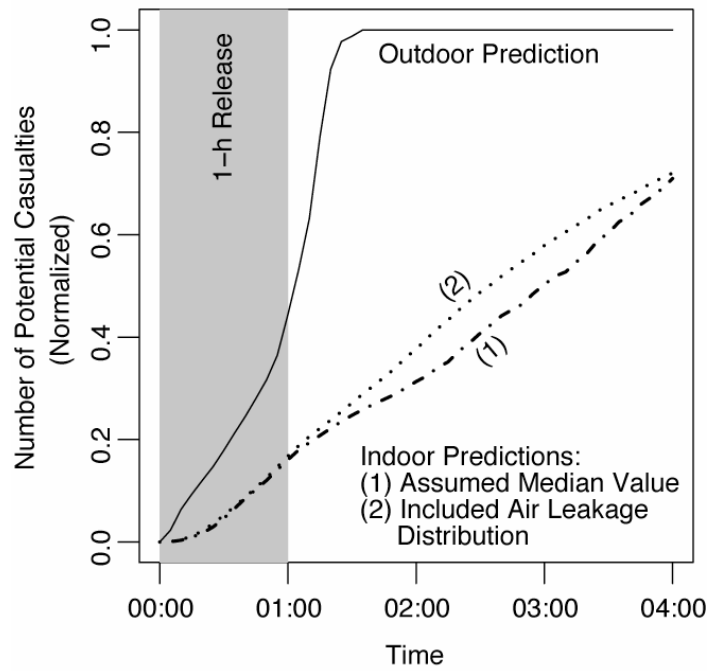
Figures

Fig. 1

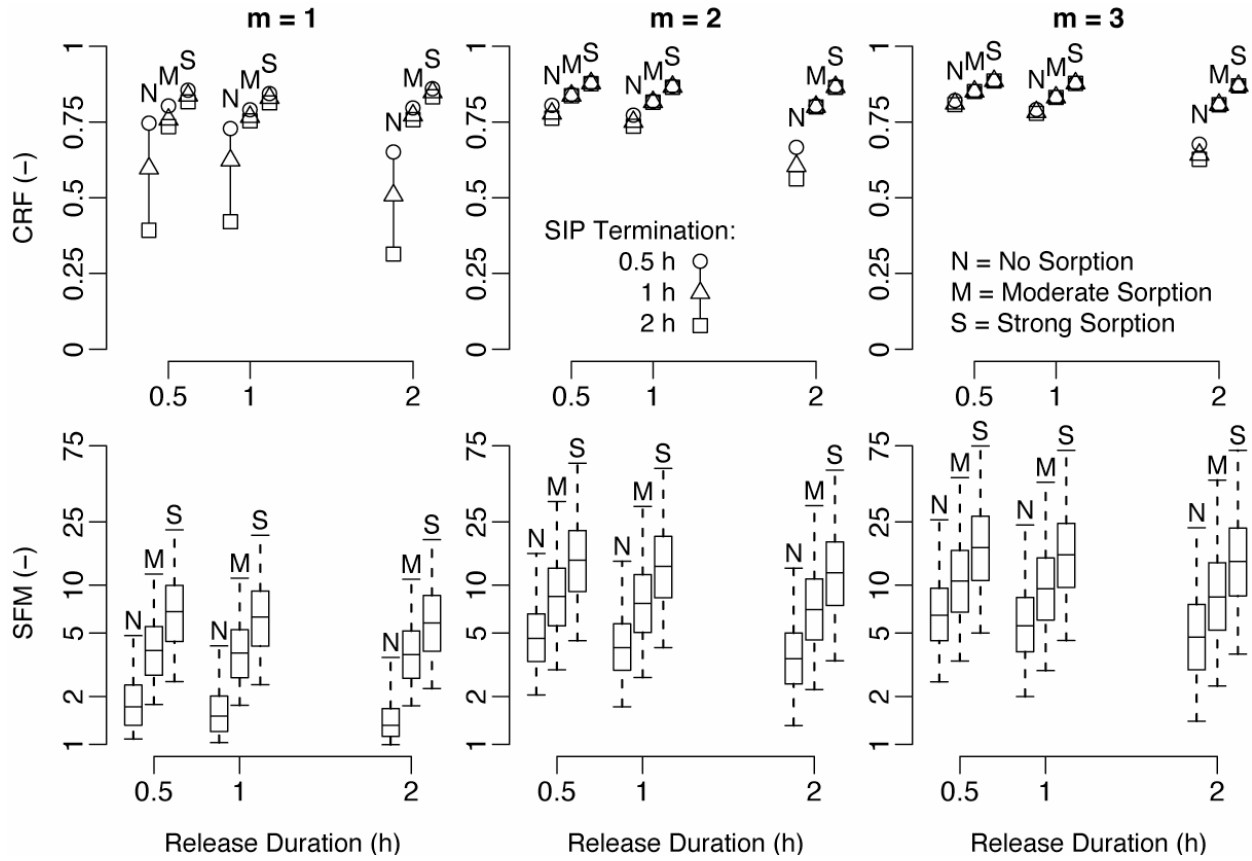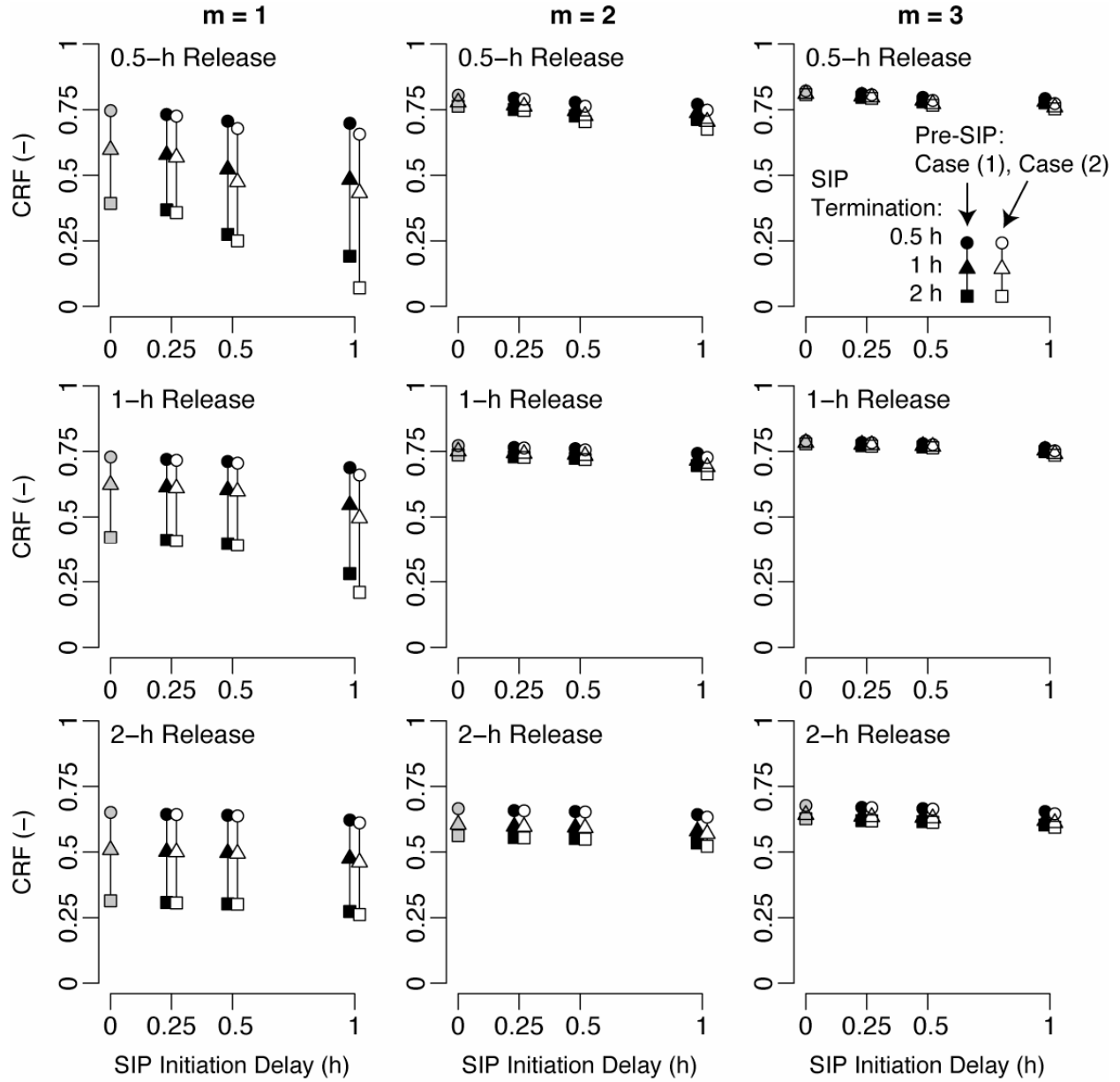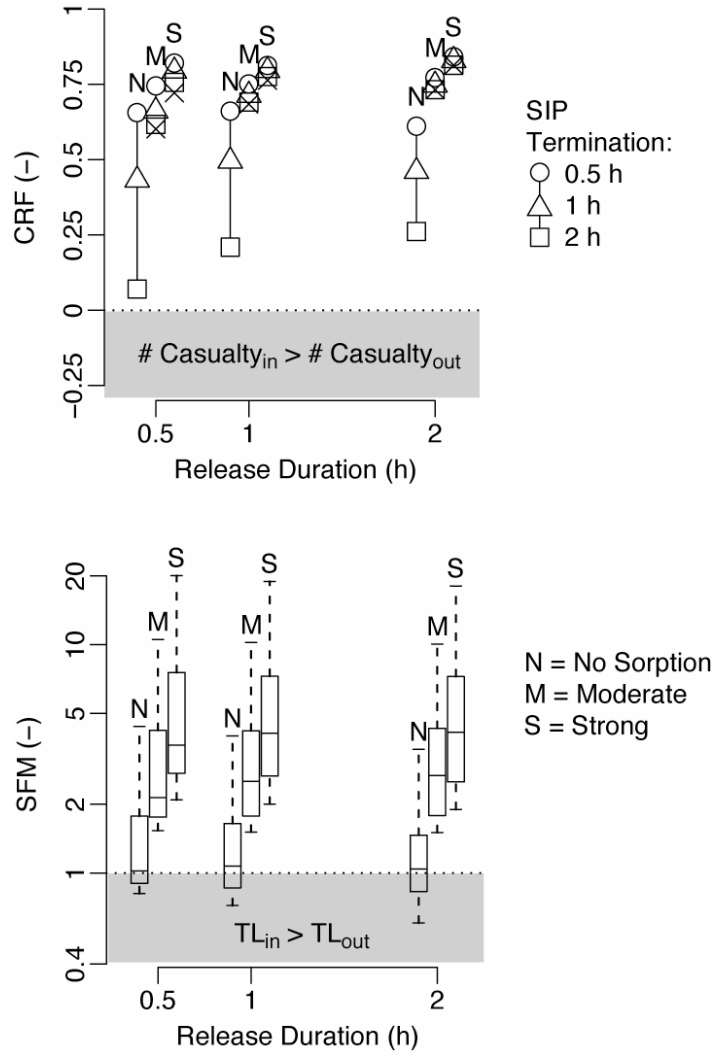Fig. 2

Fig. 3

Fig. 4

Fig. 5

Fig. 6

Fig. 7

Fig. 8

Tables

Table 1
Sorption and desorption rate coefficients (see Eq. (2))

	Surface sink		Embedded sink		Surface materials
	k_a (h^{-1})	k_d (h^{-1})	k_1 (h^{-1})	k_2 (h^{-1})	
Strong sorption [a]	5.0	0.86	0.72	0.12	Typical room in residences
Moderate sorption [b]	1.4 [c]	0.02	0	0	Painted walls, roof, and synthetic carpet

[a] Represented by dimethyl methylphosphonate (DMMP), using empirical data from Singer et al. (2005b).
[b] Represented by ammonia (NH_3), using empirical data from Karlsson and Huber (1996).
[c] A surface-to-volume ratio of 3.5 m^2 m^{-3} (Singer et al., 2005b) is assumed when converting the uptake rate coefficient from 1.1×10^{-4} m s^{-1}, as it is originally reported, to units of inverse time (h^{-1}).

Table 2
Estimated SIP initiation delays for emergency response in a community

Action		Time required (minutes)		
		Fast	Typical	Slow
(1)	Officials identify hazard and issue warning [a]	5	15	45
(2)	Population receives warning [b]	5	10	30
(3)	Population implements shelter-in-place [c]	5	10	20

[a] Source: NICS (2001).
[b] Source: Rogers and Sorensen (1991).
[c] Source: Rogers and Sorensen (1989); Rogers et al. (1990).

Table 3
Model parameters used to assess SIP effectiveness in case study

Parameters		Model values		
Release duration	T_r (h)	0.5	1	2
Toxic load exponent	m (-)	1	2	3
Sorption rate [a]		Nil	Moderate	Strong
SIP initiation delay [b]	T_i (h)	0.25	0.5	1
Additional air-exchange before SIP [c]	k (h^{-1})	Nil	+0.3 (80%)	+1 (40%)
SIP termination delay [d]	T_t (h)	0.5	1	2

[a] See Table 1 for the corresponding sorption/desorption rate coefficients.
[b] Initiation time delay measured from the start time of the release.
[c] Percentage in parentheses indicates the fraction of dwellings with elevated air-exchange.
[d] Termination time measured from the stop time of the release.