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2014 Review on the Extension of the *AMedP-8(C)* Methodology to New Agents, Materials, and Conditions

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August 2015

IDA Document D-5226
Log: H 15-000873

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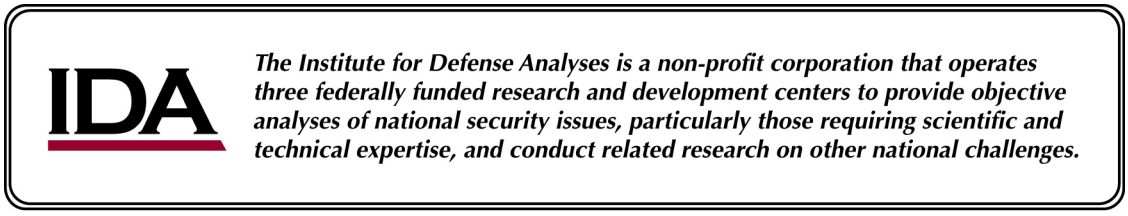

About This Publication

The work was conducted by the Institute for Defense Analyses (IDA) under contract HQ0034-14-D-0001, Project CA-6-3079, "CBRN Casualty Estimation Update of the Medical CBRN Defense Planning & Response Project," for the J-8 CBRN Joint Requirement Office and the US Army Office of the Surgeon General. The views, opinions, and findings should not be construed as representing the official position of either the Department of Defense or the sponsoring organization.

Acknowledgments

The authors would like to thank Dr. Sean Oxford for his help interpreting the results of this analysis. We are also grateful to Mr. Doug Schultz and Dr. Nathan Platt for reviewing, and Mrs. Amberlee Mabe-Stanberry for producing this document.

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Executive Summary

Background

In 2009, in support of the Office of The Surgeon General (OTSG), the Institute for Defense Analyses (IDA) produced the final draft of a North Atlantic Treaty Organization (NATO) planning guide documenting a methodology to estimate casualties from chemical, biological, radiological, and nuclear (CBRN) weapons. That document, *Allied Medical Publication 8 (C): NATO Planning Guide for the Estimation of CBRN Casualties*, (*AMedP-8(C)*), promulgated in March 2011, included the parameters to estimate casualties caused by three chemical agents, five biological agents, seven radioisotopes, nuclear fallout, or prompt nuclear effects.[1] Each year since 2009, OTSG has sponsored IDA to publish an annual review exploring and recommending extensions of this methodology to new agents, materials, and conditions.

The most recent version of the IDA-developed casualty estimation methodology is documented in Study Draft 2 of *Allied Medical Publication 7*.5 (*AMedP-7.5* SD2), an updated and renamed[2] publication of the NATO planning guide. In a continuing effort to identify additional improvements to this methodology, this year's annual review is a quick-look analysis comparing the methodology's outputs to those of Hazard Prediction and Assessment Capability (HPAC), a modeling and simulation tool developed by the Defense Threat Reduction Agency whose capabilities also include casualty estimation. The objective of this comparison is to identify additional improvements to the *AMedP-7.5* SD2 casualty estimation methodology.

Methodology

For this analysis, the IDA team evaluated four agents: anthrax, botulinum toxin, sarin (GB), and distilled mustard (HD), first using the default parameters and methods in HPAC and the *AMedP-7.5* SD2 methodology to compare total casualties (all individuals estimated to become ill regardless of their outcome) and fatalities (the subset of total casualties estimated to die) for a common scenario. In order to make meaningful comparisons of HPAC and the *AMedP-7.5* SD2 methodology, the IDA team then made incremental changes to the default casualty parameters and methods to control for all known data and methodological differences between the two methodologies (listed in the

[1] North Atlantic Treaty Organization (NATO), *AMedP-8(C): NATO Planning Guide for the Estimation of CBRN Casualties*, STANAG 2553 (Brussels: NATO, March 2011).

[2] The change in designation from *AMedP-8(C)* to *AMedP-7.5* reflects a change in NATO publication naming conventions, but the title of the document remains the same (*NATO Planning Guide for the Estimation of CBRN Casualties*).

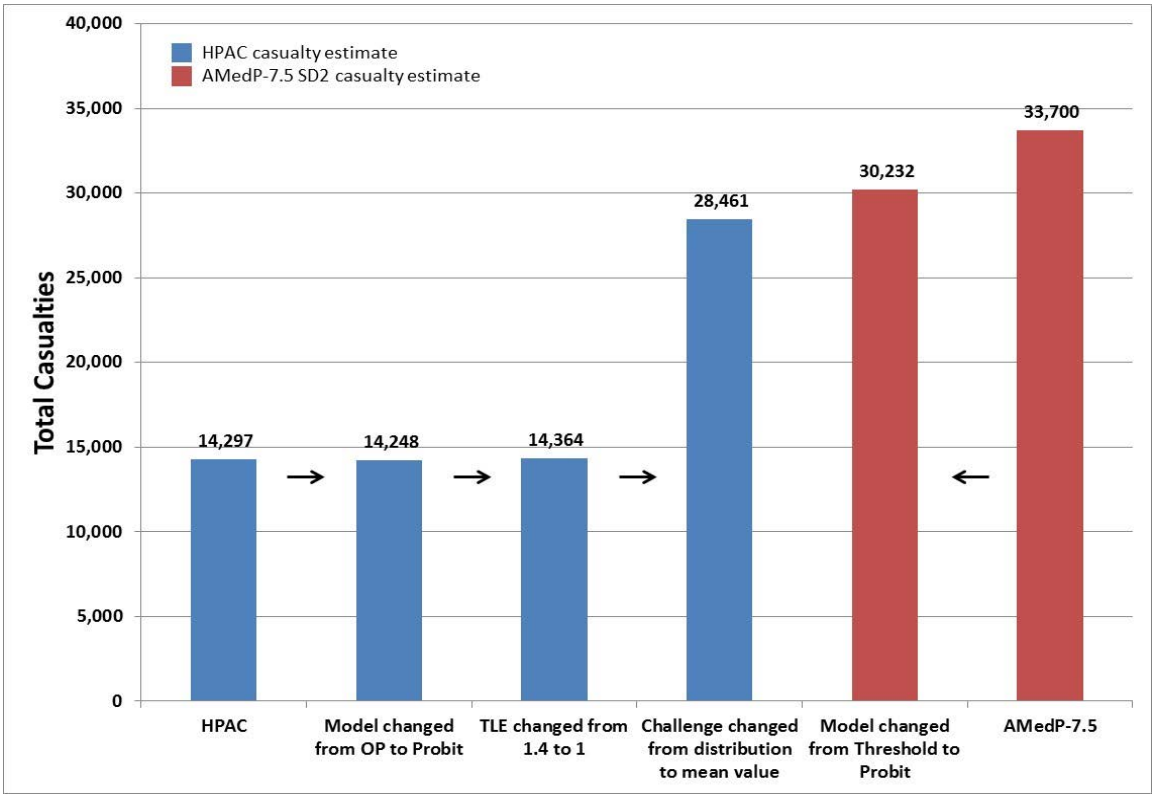
table below). With each change, the methodologies and their resulting casualty estimates converged, indicating that the identified data and methodological differences accounted for nearly all of the observed differences between the default casualty estimates for the two methodologies.

Data and Methodological Differences Controlled for in Analysis

Different Human Response Parameter Values			Different Effects and Mortality Models		Different Dose Representations
Median value (e.g., ID_{50})	Probit slope	Toxic load exponent	Threshold vs probit model	"OP" vs probit model	Mean value vs distribution

Results

The results of the analysis for each of the four agents are presented in figures similar to the one below, which displays the GB total casualties (mild or greater). The blue bars represent casualties computed using HPAC, whereas the red bars represent casualties computed using the *AMedP-7.5* SD2 methodology. The outermost bars correspond to the casualties estimated using the default parameters and internal methods for the two methodologies (HPAC on the left and *AMedP-7.5* SD2 on the right). Moving toward the center, each successive bar represents casualties estimated after a single data or methodological change relative to the outer adjacent bar of the same color. The bar labels specify the changes corresponding to each new estimate, and arrows indicate the progression from the default methodology estimates to the adjusted estimates. After controlling for all identified data and methodological differences in one methodology or the other, the two methodologies converged, and the resulting casualty estimates (represented by the adjacent blue and red bars) were very similar.



GB Total Casualties (Mild or Greater)

In this example, the total observed difference between the default HPAC and *AMedP-7.5* SD2 casualty estimates (the outermost bars) is 19,403. After accounting for four known data and methodological differences, the adjusted HPAC and *AMedP-7.5* SD2 estimates (adjacent blue and red bars) differed only by 1,771 total casualties, indicating that the changes explained 91% of the total observed difference. The following two tables show for each agent the total observed difference before accounting for any of the known data and methodological differences and the percent of that total observed difference explained by the known data and methodological differences.

Total Observed Difference between HPAC and *AMedP-7.5* SD2 Casualty Estimates before Accounting for Data and Methodological Differences

	Total Casualties		
Agent	**(Mild or Greater)**	**(Severe or Greater)**	**Fatalities**
GB	19,403	10,173	406
HD	8,420	612	119
Anthrax	38,806		38,813
Botulinum Toxin	29,581		18,170

Percentage of Total Observed Difference between HPAC and *AMedP-7.5* SD2 Casualty Estimates Explained by Data and Methodological Differences

Agent	Total Casualties (Mild or Greater)	(Severe or Greater)	Fatalities
GB	91%	99%	78%
HD	96%	94%	79%
Anthrax	95%		95%
Botulinum Toxin	95%		95%

The results in the second table indicate that by controlling for the data and methodological differences, the IDA team explained the vast majority of the total observed difference between the HPAC and *AMedP-7.5* SD2 casualty estimates. With the exception of the fatality estimates for the two chemical agents, the explained observed difference was more than 90% of the total observed difference. For GB and HD fatalities, this value was 78% and 79%, respectively, but the number of fatalities left unexplained was small in absolute numbers (90 for GB and 25 for HD).

Recommendation

One of the major methodological differences between the two methodologies was the use of a threshold dose-response model for chemical agents in the *AMedP-7.5* SD2 methodology and a variation of the probit model in HPAC. To control for this, the IDA team modified both methodologies to estimate chemical casualties using a probit model. The IDA team observed large variability in the magnitude of the effect of the change from the *AMedP-7.5* SD2 threshold model to the probit model. The degree to which the threshold model overestimates (or in one case underestimates) casualties relative to the probit model varies significantly by scenario. For GB, the threshold model predicts 11% more total casualties (mild or greater), 84% more total casualties (severe or greater), and 15% more fatalities than the probit model. Even greater variation occurs for HD, for which the threshold model predicts 31% more total casualties (mild or greater), 55% fewer total casualties (severe or greater), and 204% more fatalities than the probit model.

When the human response to a given challenge of agent varies within the exposed population, as is the case for the chemical agents in *AMedP-7.5* SD2, a probit model is often used to capture that variability in response. Because the probit models were fit directly to the raw data and the threshold model values were derived from the probit models, the probit models are generally better able to capture the underlying variability in human response than the threshold models. As no single threshold value consistently results in a good match between the threshold model and the probit model, the degree to which the *AMedP-7.5* SD2 methodology overestimates (or underestimates) chemical

casualties is highly dependent upon the scenario analyzed. In order to avoid this unpredictable variation from the probit model estimate and to more consistently and accurately predict the number of casualties from chemical agents, the IDA team recommends changing the threshold model in the *AMedP-7.5* SD2 chemical agent methodology to a probit model.

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Contents

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1. Introduction

A. Background

In 2009, in support of the Office of The Surgeon General (OTSG), the Institute for Defense Analyses (IDA) produced the final draft of a North Atlantic Treaty Organization (NATO) planning guide documenting a methodology to estimate casualties from chemical, biological, radiological, and nuclear (CBRN) weapons. That document, *Allied Medical Publication 8 (C): NATO Planning Guide for the Estimation of CBRN Casualties*, (*AMedP-8(C)*), promulgated in March 2011, included the parameters to estimate casualties caused by three chemical agents, five biological agents, seven radioisotopes, nuclear fallout, or prompt nuclear effects.[3] Each year since 2009, OTSG has sponsored IDA to publish an annual review exploring and recommending extensions of this methodology to new agents, materials, and conditions.

Table 1 summarizes the major topics addressed in each of the five annual reviews published to date, which include potential extensions to the originally published casualty estimation methodology. The IDA team has already incorporated many of these changes into the most recent version of the methodology, which is documented in Study Draft 2 of *Allied Medical Publication 7.5* (*AMedP-7.5* SD2), an updated and renamed[4] publication of the NATO planning guide. For a more complete discussion of the conclusions and recommendations from the prior annual reviews, the authors direct the reader to the 2013 annual review.[5]

[3] North Atlantic Treaty Organization (NATO), *AMedP-8(C): NATO Planning Guide for the Estimation of CBRN Casualties*, STANAG 2553 (Brussels: NATO, March 2011).

[4] The change in designation from *AMedP-8(C)* to *AMedP-7.5* reflects a change in NATO publication naming conventions, but the title of the document remains the same (*NATO Planning Guide for the Estimation of CBRN Casualties*).

[5] Lucas A. LaViolet and Carl A. Curling, *2013 Review on the Extension of the AMedP-8(C) Methodology to New Agents, Materials, and Conditions*, IDA Document D-4082 (Alexandria, VA: Institute for Defense Analyses, June 2014).

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Table 1. Summary of Prior Annual Review Topics

Year	IDA Publication Number	Focus of Annual Review
2009	IDA Document D-3945	• Identification of additional biological or chemical agents of concern • Estimation of level of effort to model a subset of identified agents
2010	IDA Document D-4131	• Incorporation of medical countermeasures into methodology • Alignment of *AMedP-8(C)* with Common User Database (CUD)
2011	IDA Document D-4486	• Identification of human response knowledge gaps • Estimation of level of effort to incorporate additional agents, new medical countermeasures, new outbreak data, and psychological casualties
2012	IDA Document D-4727	• Identification of new data to update existing agents or effects
2013	IDA Document D-4802	• Formal prioritization scheme for future enhancements to the methodology

B. Objective

The objective of this year's annual review is to identify additional improvements to the *AMedP-7.5* SD2 casualty estimation methodology. To meet this objective, the IDA team compared the methodology's casualty estimates to those of Hazard Prediction and Assessment Capability (HPAC) version 5.3.2, a modeling and simulation tool developed by the Defense Threat Reduction Agency (DTRA) whose capabilities also include casualty estimation. This comparison highlighted differences between two methodologies commonly used in the US to model the consequences of CBRN events. The IDA team further investigated these differences and considered whether each change that could be made to the *AMedP-7.5* SD2 casualty estimation methodology to more closely match the HPAC methodology would be an improvement.

Although HPAC is not available to all NATO nations, those other nations are likely to conduct a similar comparative analysis between the *AMedP-7.5* SD2 methodology (or its successor versions) and their national casualty estimation tools. This document may help OTSG anticipate and prepare appropriate responses to any questions posed by NATO nations that find differences in outputs between their national tools and the *AMedP-7.5* SD2 methodology.

C. Scope

This analysis is a quick-look comparison of two casualty estimation methodologies. It is not intended to be a verification and validation (V&V) of either methodology, nor is the primary focus to uncover every methodological detail that varies between the two. The IDA team referenced available HPAC documentation from HPAC training courses and prior V&V efforts and leveraged its existing knowledge of the *AMedP-7.5* SD2 methodology. However, the limited scale of this analysis precluded an exhaustive search for the sources of unexplained observed differences between the casualty estimates of the two methodologies.

While many biological and chemical warfare agents could be modeled using either methodology, the IDA team selected anthrax, botulinum toxin, sarin (GB), and distilled mustard (HD) for this analysis, because these four agents represent four different threat types (replicating organisms, toxins, nerve agents, and blister agents, respectively) and are of general concern to the defense community.[6] Due to the quick-look nature of this analysis, radiological and nuclear weapons were not considered, although this analysis could be extended to these other types of threats if required.

D. Document Organization

This document is organized into six chapters. Chapter 1 provides the objective and scope of the current analysis. Chapter 2 briefly introduces the HPAC and *AMedP-7.5* SD2 casualty estimation methodologies. Chapter 3 describes the data and methodological differences between the two methodologies. Chapter 4 presents the default casualty estimates for each agent using the two methodologies as well as the results of the analysis to attribute the large initial difference between these casualty estimates to the data and methodological differences identified in Chapter 3. Chapter 5 discusses the IDA team's observations relating to the analysis. Lastly, Chapter 6 recommends an improvement to the *AMedP-7.5* SD2 methodology based on the results of the analysis.

[6] Centers for Disease Control and Prevention (CDC), "Bioterrorism Agents/Diseases," accessed December 30, 2014, http://emergency.cdc.gov/agent/agentlist-category.asp; CDC, "Chemical Categories," accessed December 30, 2014, http://emergency.cdc.gov/agent/agentlistchem-category.asp.

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2. Overview of Casualty Estimation Methodologies

A. HPAC Casualty Estimation Methodology

DTRA developed HPAC as "a counter-proliferation/counterforce tool that predicts the effects of hazardous material releases (nuclear, biological, and chemical) into the atmosphere and their collateral effects on civilian and military populations."[7] HPAC's intended users are DTRA personnel who perform analyses in response to external requests (DTRA Reachback) and other highly skilled analysts in the U.S. government. Military analysts and planners outside of highly analytical organizations use the accredited DOD CBRN effects modeling and simulation software application, Joint Effects Model (JEM), which is a modified version of HPAC with less analytical flexibility, and rely on DTRA Reachback for more detailed analyses.[8]

To estimate casualties in HPAC, users must specify the spatial domain of interest, the specific incident release point, the munition and delivery system, the agent, the mass of agent load, the release altitude, and the weather, although default values are available for most required inputs. Users can also modify a number of other parameters, including the human response parameters associated with the agent, to customize the scenario.

HPAC estimates casualties based on population data files from Oak Ridge National Laboratory's LandScan™ 2012 dataset, which estimates the number of people (without designation of military or civilian status) at locations spanning the globe. Each LandScan population data file is represented as a grid of cells, each of which is 30 arc-seconds per side (approximately 1 km by 1 km) and associated with an estimated population uniformly distributed within that cell.[9] HPAC's dispersion model, Second-order Closure Integrated Puff (SCIPUFF), uses an adaptive grid to sub-divide the LandScan cells to varying resolution levels determined by the size and location of the cloud. SCIPUFF generates more sub-divisions (higher resolution) where the amount of agent changes rapidly. HPAC then matches the fraction of the population corresponding to the sub-

[7] The Analytic Sciences Corporation (TASC), *Accreditation Report for Joint Effects Model, Science and Technology Prototype/Hazard Prediction and Assessment Capability Version 5.1, Revision 2* (Chantilly, VA: TASC, 28 February 2013), 7.

[8] TASC, *Accreditation Report for Joint Effects Model, Science and Technology Prototype/Hazard Prediction and Assessment Capability Version 5.1, Revision 2* (Chantilly, VA: TASC, 28 February 2013), 7.

[9] "LandScan Documentation," Oak Ridge National Laboratory, accessed February 9, 2015, http://web.ornl.gov/sci/landscan/landscan_documentation.shtml.

divided LandScan cell with the amount of agent present at that location. SCIPUFF estimates both a mean concentration or dose value and, based on turbulence and uncertainties in the weather parameters, a variance about that mean.

Given a population and an associated concentration time (Ct) or dose value, HPAC's illness, infection, and injury (III) effects model and its mortality model determine how the probabilities of illness and death are calculated, respectively. The models used for the four agents included in the current analysis include "Probit," "OP" (organophosphate), "HD," and "Erfform."

Generally, a probit model describes a Ct- or dose-response relationship where the response (illness or death) is lognormally distributed as a function of Ct or dose.[10] Written in terms of the parameters of interest for a biological agent such as anthrax (median infective dose (ID_{50}) and probit slope), the probit model is described by Equation 1

$$\text{Probability of illness} = \Phi\left(\text{probit slope} * \log_{10}\left(\frac{\text{D}}{\text{ID}_{50}}\right)\right) \quad (1)$$

where Φ is the standard normal cumulative distribution function and D is the inhaled dose.[11] One would likewise calculate the probability of death and the probability of illness from a toxin using Equation 1 by replacing the ID_{50} with the median lethal dose (LD_{50}) or the median effective dose (ED_{50}), respectively. For the chemical agents described in this analysis, for which response is described as a function of Ct rather than dose, the equivalent terms are median effective concentration (ECt_{50}) and median lethal concentration (LCt_{50}).

HPAC's "Probit" model calculates probabilities by integrating the probit values over the distribution of Ct or dose values predicted by SCIPUFF. Equation 2 shows HPAC's calculation of the probability of illness from a biological agent, where pdf(D)*d*D is the probability of receiving a dose in the range from D to D + *d*D.[12]

$$\text{Probability of illness} = \int \Phi\left(\text{probit slope} * \log_{10}\left(\frac{\text{D}}{\text{ID}_{50}}\right)\right) * \text{pdf(D)}\, d\text{D} \quad (2)$$

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[10] Kim Vincent, "Probit Analysis," http://userwww.sfsu.edu/efc/classes/biol710/probit/ProbitAnalysis.pdf.

[11] Generally speaking, the probability of infection is not equivalent to the probability of illness unless all infected individuals become ill. However, the infectivity parameter values for the biological agents in this analysis were derived such that subclinical (or asymptomatic) infections were ignored. Everyone who is "infected" will become symptomatic.

[12] Jason Rodriquez, Gene E. McClellan, and Darren R. Oldson, *Chemical-Biological Operational Degradation Analysis (CODA)*, ARA-TR-09-SEASSP-00077.00002-004 (Arlington, VA: Applied Research Associates, September 2010), 12.

HPAC implements this using numerical integration by dividing the probability density function (pdf) into 30 bins, the details of which are specified in an appendix to one of the HPAC technical reports.[13]

The "OP" and "HD" models are variations of the "Probit" model that determine the probability of injury as a function of multiple routes of exposure. The "OP" model, used as the default III effects model for GB, converts the percutaneous vapor Ct and liquid dose values to equivalent inhaled vapor Ct values using the ECt_{50} and ED_{50} values for the different routes of exposure. The sum of the inhaled vapor Ct and the equivalent inhaled vapor Ct values from the percutaneous effects is then used in Equation 2.[14] The "HD" model likewise combines the HD percutaneous vapor and liquid challenges to calculate an equivalent percutaneous vapor Ct that is used in Equation 2 to estimate the probability of injury from percutaneous HD. The "HD" model uses Equation 2 a second time to calculate the probability of injury from inhaled HD vapor.[15] These two probabilities, P(Perc Injury) and P(Inh Injury), are then combined assuming independence according to Equation 3.[16]

$$P(\text{Injury}) = P(\text{Perc Injury}) + P(\text{Inh Injury}) - P(\text{Perc Injury})*P(\text{Inh Injury}) \quad (3)$$

HPAC's "Erfform" model, specific to anthrax mortality, integrates another dose-dependent function, described in Equation 4, over the dose distribution.[17]

$$\text{Probability of death} = \begin{cases} \text{Probability of illness}*0.86, & D \leq 10^5 \text{spores} \\ \text{Probability of illness}*\left(1\text{-}0.5\left(1+\text{erf}\left[\frac{2.3263-0.2959*\ln D}{\sqrt{2}}\right]\right)\right), & D > 10^5 \text{spores} \end{cases} \quad (4)$$

HPAC outputs its casualty estimate results in a table that reports three sets of values: "Best Estimate," "At 10.0% Risk," and "Worst Case." For this comparative analysis, the

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[13] Jason Rodriquez, Gene E. McClellan, and Darren R. Oldson, *Chemical-Biological Operational Degradation Analysis (CODA)*, ARA-TR-09-SEASSP-00077.00002-004 (Arlington, VA: Applied Research Associates, September 2010), 32–33.

[14] Jason Rodriquez, Gene E. McClellan, and Darren R. Oldson, *Chemical-Biological Operational Degradation Analysis (CODA)*, ARA-TR-09-SEASSP-00077.00002-004 (Arlington, VA: Applied Research Associates, September 2010), 18–19.

[15] Although HPAC refers to this as the probability of injury from inhaled HD vapor, it is derived from FM 3-11.9 parameters for ocular injury. FM 3-11.9, p. II-40. Jason Rodriquez, Gene E. McClellan, and Darren R. Oldson, *Chemical-Biological Operational Degradation Analysis (CODA)*, ARA-TR-09-SEASSP-00077.00002-004 (Arlington, VA: Applied Research Associates, September 2010), 20–21.

[16] Jason Rodriquez, Gene E. McClellan, and Darren R. Oldson, *Chemical-Biological Operational Degradation Analysis (CODA)*, ARA-TR-09-SEASSP-00077.00002-004 (Arlington, VA: Applied Research Associates, September 2010), 54.

[17] Jason Rodriquez, Gene E. McClellan, and Darren R. Oldson, *Chemical-Biological Operational Degradation Analysis (CODA)*, ARA-TR-09-SEASSP-00077.00002-004 (Arlington, VA: Applied Research Associates, September 2010), 26.

IDA team chose to compare the "Best Estimate" values from the casualty estimation table.

HPAC has the functionality not only to report casualties but also to specify the amount of agent present at various locations, which can be reported numerous ways (e.g., instantaneous liquid concentration, time-integrated vapor concentration). Users may assign samplers to particular locations of interest and specify that HPAC report the Ct or dose values for the samplers. This is a separate procedure from the HPAC casualty estimate, and the outputs do not include estimated casualties. Instead, the outputs consist of the mean and variance corresponding to the Ct or dose distribution at each sampler, which could be directly applied to casualty estimation.

B. *AMedP-7.5* SD2 Casualty Estimation Methodology

IDA developed the *AMedP-7.5* SD2 methodology for the purpose of estimating "the number, type, severity, and timing of CBRN casualties."[18] Although it has the capability to estimate the times at which individuals become ill/injured and die and the severity of the illness/injury over time, HPAC does not. For that reason, IDA only computed the comparable outputs, namely the numbers of estimated total casualties and fatalities, for this analysis.

The required inputs to the *AMedP-7.5* SD2 methodology are 1) a characterization of the population at risk in the scenario into icons, groups of individuals sharing a common location over time, and 2) an associated CBRN challenge value (Ct or dose for chemical and biological agents, respectively) for each icon. The generation of the Ct or dose values is external to the methodology and may be postulated or derived from some other tool. Of the agents included in this analysis, the models for GB, anthrax, and botulinum toxin consider only inhalation challenges, while the HD model accounts for multiple routes of exposure. As a result, the human response to HD is a function of three different Ct values: inhalation, ocular, and equivalent percutaneous. The equivalent percutaneous challenge is calculated as the sum of the percutaneous vapor Ct and the percutaneous liquid dose converted to an equivalent amount of vapor using the ratio of the HD severe ECt_{50} and ED_{50} values.[19]

Users may opt to specify additional icon attributes as inputs, such as breathing rates, individual protective equipment, or vehicle or shelter occupancy. The *AMedP-7.5* SD2 methodology uses these attributes to derive the effective CBRN challenge, the amount of agent that actually affects an icon and determines the human response to the agent. If

[18] NATO, *AMedP-7.5* SD2, 1-2, DRAFT.

[19] NATO, *AMedP-7.5* SD2, 4-14–4-15, DRAFT.

these optional inputs are omitted, then the Ct or dose values associated with each icon will be treated as the effective CBRN challenge values and will not be further modified.

Given an effective CBRN challenge value and a number of individuals at an icon, the *AMedP-7.5* SD2 methodology uses the probit model described in Equation 1 to determine the probabilities of illness and death for biological agents. It determines the chemical agent probabilities using a deterministic threshold model according to which all individuals above a threshold Ct value have a 100% probability of becoming ill or dying and all below the threshold have a 0% probability. Threshold values depend on the severity of injury (mild, moderate, or severe),[20] which are reported for the two chemical agents and three severity levels in Table 2. The threshold values are derived from the probit model estimates and are generally on the low end of the probit curve, which typically results in higher probabilities of illness and death than the probit model predicts. For HD, if the challenge value of at least one of the routes of exposure exceeds the corresponding threshold value, then all individuals receiving that challenge value are considered casualties. Mortality is only modeled to result from inhalation exposures, and therefore no lethal threshold values are present for other routes of exposure.

Table 2. *AMedP-7.5* SD2 Threshold Ct Values above Which Individuals Are Estimated to Become Ill (Total Casualties) or Die (Fatalities) (All Values Are in Units of mg-min/m^3)

Agent	Category	Route of Exposure	Mild	Moderate	Severe
GB	Total Casualties	Inhalation	0.2	1	12
	Fatalities	Inhalation	27	27	27
HD	Total Casualties	Ocular	4	4	70
	Total Casualties	Inhalation	50	100	150
	Total Casualties	Percutaneous	12	180	180
	Fatalities	Inhalation	250	250	250

The *AMedP-7.5* SD2 methodology estimates casualties by multiplying the Ct- or dose-dependent probabilities of illness and death by the number of people challenged by that amount of agent. This process is complicated by a unique feature of the *AMedP-7.5* SD2 methodology, namely a user-specified casualty criterion. In effect, the user selects the minimum symptom severity level at which someone is considered a casualty (mild, moderate, or severe), and anyone exhibiting symptoms at or above that severity level is counted in the casualty estimate.

For the chemical agents, if the user selects a casualty threshold of mild, the threshold Ct values associated with mild symptoms from Table 2 are used. If the casualty

[20] These severities are defined in NATO, *AMedP-7.5* SD2, 1-10, DRAFT.

threshold is severe symptoms, then the threshold Ct value associated with severe symptoms is used instead. In both cases, the probabilities are still 0% below the threshold Ct and 100% above. The probabilities of death are not dependent upon the casualty criterion. As HPAC only predicts mild and severe casualties, *AMedP-7.5* SD2 casualties are never estimated using the moderate casualty criterion in this analysis, as no comparison would exist to make between methodologies. For the two biological agents considered in this analysis, the threshold severity level is reached for all criteria, so the casualty criterion has no effect on the predicted number of casualties.

3. Methodology

A. Overview

For this analysis, the IDA team compared estimated casualties using HPAC version 5.3.2 and the *AMedP-7.5* SD2 methodology implemented in the computer programming language Python version 3.4.2. The IDA team's methodology for generating casualty estimates from the two methodologies is illustrated in Figure 1. The IDA team first specified a common scenario consisting of: 1) a chemical or biological incident and 2) a population by location. Once the IDA team input the chemical or biological incident details into HPAC, HPAC's transport and dispersion model converted the incident into a set of challenge values by location. The challenge by location from HPAC and the population by location specified by the scenario served as the two direct inputs to the casualty estimation portion of HPAC and to the *AMedP-7.5* SD2 methodology.

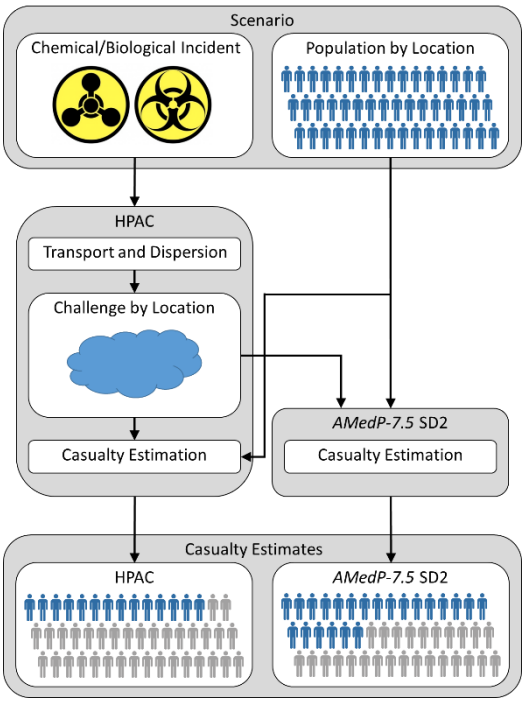

Figure 1. The IDA Team Used a Common Scenario to Generate Casualty Estimates Using HPAC and the *AMedP-7.5* SD2 Methodology

The casualty estimates generated by the two methodologies included both total casualties (all individuals estimated to become ill regardless of their outcome) and fatalities (the subset of total casualties estimated to die). For chemical agents, total

casualties were calculated in two ways: once using human response parameter values associated with mild effects (reported as "mild or greater") and once with values associated with severe effects (reported as "severe or greater"). The lethality parameter values were the same regardless of how casualties were defined (mild or greater vs severe or greater).

For each agent, the IDA team first estimated casualties using the default settings for the two methodologies. Then, in order to explain as much of the observed difference between the resulting casualty estimates as possible (hereafter referred to as the "observed difference"), the IDA team controlled for known differences in the default parameters and methods between the methodologies (hereafter referred to as "data and methodological differences") by making incremental changes to one methodology or the other. In order to isolate the effect of each change on the casualty estimates, the IDA team accounted for only one data or methodological difference at a time in a step-wise fashion, causing the models and parameter values used by HPAC and the *AMedP-7.5* SD2 methodology, and consequently their casualty estimates, to converge with each step. This chapter describes those changes made to HPAC and the *AMedP-7.5* SD2 methodology to account for the data and methodological differences the IDA team identified and controlled for.

B. Scenario Inputs

In order to make a valid comparison between the two casualty estimation methodologies, the IDA team used the same chemical and biological incident details and the same population at risk for both methodologies. The IDA team chose the incident release point and related cloud dispersion parameter values solely to expose a population large enough to produce sufficient casualties so that differences between the resulting HPAC and *AMedP-7.5* SD2 casualty estimates would be apparent. Because the attribution of these differing casualty estimates to data and methodological differences between the two methodologies is the focus of the analysis, the specific details pertaining to the release, such as weapon type, fill weight, and meteorological conditions, are inconsequential to the conclusions of this analysis as long as they were the same for both casualty estimation methodologies. The details of the chemical/biological incident are relegated to Appendix A.

Having chosen the incident details, the IDA team used HPAC's transport and dispersion model to generate the chemical or biological agent clouds and the associated challenge information (Ct values) over the area of interest, which was used as an input to both casualty estimation methodologies. In order for the *AMedP-7.5* SD2 methodology to use the HPAC cloud data, the IDA team converted the HPAC challenge outputs from units of kg-s/m^3 to the units appropriate for each chemical or biological agent. For the

biological agents, the IDA team used a breathing rate of 15 liters per minute[21] to convert to the required dose units and, for anthrax, the HPAC default biological conversion factor value of 10^6 organisms per microgram, assuming that 1 organism is equivalent to 1 spore.

In order to limit the dependency of the results on the population distribution as well as to ensure the same population at risk was used in both casualty estimation methods, the IDA team used a uniformly distributed population at risk of 288,000 people (1,000 people per LandScan cell). As the *AMedP-7.5* SD2 methodology uses discrete icons to represent the population at risk, the IDA team represented the uniform population of 1,000 people per LandScan cell as a grid of 400 evenly spaced icons, each designating the location of 2.5 people. The IDA team then created a grid of HPAC samplers with a uniform density of 400 samplers per LandScan population cell[22] and ran HPAC for each agent with its default parameter values to output the challenge value at each sampler location. Thus each icon of 2.5 people was associated with a unique challenge.

The IDA team's sampler grid approach to matching individuals to agent challenge values for use in the *AMedP-7.5* SD2 methodology is an approximation of HPAC's more complicated SCIPUFF adaptive grid matching methodology. To test the adequacy of this approximation, the IDA team compared the populations within the LCt_{90} contours using the two approaches for two agents.[23] For both of the agents tested, the population estimates were within 99.5% of each other. Given that the alignment of the agent cloud outputs to the population at risk is not identical for the two methodologies, an exact match between casualty estimates is unlikely, but the test results indicate that the contribution of any misalignment to the observed difference in the resulting casualty estimates should be small.

C. Data and Methodological Differences Controlled for in Analysis

Even using the common inputs described above, the casualty estimates for HPAC and the *AMedP-7.5* SD2 methodology differed substantially due to their different default human response parameter values and internal methods. The IDA team identified several such data and methodological differences, which are listed in Table 3 and described in

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[21] This is the same breathing rate used by HPAC's casualty estimation algorithms. Jason Rodriquez, Gene E. McClellan, and Darren R. Oldson, *Chemical-Biological Operational Degradation Analysis (CODA)*, ARA-TR-09-SEASSP-00077.00002-004 (Arlington, VA: Applied Research Associates, September 2010), 18.

[22] Due to the proximity of the release point to the equator, the cells were nearly square, with samplers approximately every 46 meters in both directions.

[23] When conducting this test, the IDA team calculated the *AMedP-7.5* SD2 in contour populations in Python by counting all individuals receiving challenges greater than or equal to the LCt_{90}. For comparison, the IDA team used the HPAC "within contour" (rather than the "statistical mean") computational method to display the populations within the LCt_{90} contours within HPAC. The distinction between these two methods is described later in this chapter.

detail in the following sections, and incrementally changed the two methodologies until they matched in terms of all identified data and methodological differences. As elaborated on in Chapters 5 and 6, one of these changes also represented a potential area for improvement of the *AMedP-7.5* SD2 methodology.

Table 3. Data and Methodological Differences Controlled for in Analysis

Different Human Response Parameter Values			Different Effects and Mortality Models		Different Representations of Challenge Values
Median value (e.g., ID_{50})	Probit slope	Toxic load exponent	Threshold vs probit model	"OP" vs probit model	Mean value vs distribution

Note that the number of data and methodological differences between the two methodologies for which the IDA team needed to control varied by agent and depended upon how closely the two methodologies already matched for each agent. For example, if the two methodologies used the same median and probit slope values, then the IDA team did not need to control for a difference in those values between methodologies. The discussion of each data or methodological difference listed below will explain for which agents the IDA team made a change to the default HPAC or *AMedP-7.5* SD2 data or methods.

1. Different Human Response Parameter Values

The first data or methodological difference the IDA team identified and controlled for in the analysis is the use of different human response parameter values. Table 4 lists the parameter values HPAC and the *AMedP-7.5* SD2 methodology use for the agents considered in this analysis, with the parameters that differ between the two methodologies shaded in gray. For the two biological agents, the default parameter values are significantly different. In contrast, with the exception of the GB toxic load exponent, the default chemical agent parameter values for the two methodologies are identical. HPAC models toxic load for GB using a toxic load exponent of 1.4, whereas the *AMedP-7.5* SD2 methodology does not include a toxic load model (which is equivalent to a toxic load exponent of 1). The *AMedP-7.5* SD2 methodology also excludes percutaneous challenge for GB, so no parameters are used. To account for the differences in parameter values between the two methodologies, the IDA team changed the HPAC default values to match those used in the AMedP-7.5 SD2 methodology (including a toxic load exponent of 1 for all agents).

Note that to determine human response for GB and HD, the *AMedP-7.5* SD2 methodology uses threshold Ct values that are based on the probit model parameter

values shown in Table 4, but these probit model parameter values are not explicitly used in the *AMedP-7.5* SD2 methodology.

Table 4. *AMedP-7.5* SD2 and HPAC Values for Agent-Specific Human Response Parameters

Agent	*AMedP-7.5* SD2 Values	HPAC Values
Anthrax (wet)[a]	ID_{50} = 17,000 spores, Probit = 0.79[b] Case fatality rate = 100%	ID_{50} = 8,900 spores, Probit = 1.43[b] 6 of 7 casualties die, increasing at high doses[c]
Botulinum toxin (wet)[a]	ED_{50} = 0.1 µg/man, Probit = 12.5[b] LD_{50} = 0.8 µg/man, Probit = 12.5[b]	ED_{50} = 0.24 µg/man, Probit = 2.9[b] LD_{50} = 1.2 µg/man, Probit = 2.9[b]
GB	$ECt_{50,inh,mild}$ = 0.4 mg-min/m^3, $Probit_{inh,mild}$ = 4.5[b] $ECt_{50,inh,sev}$ = 25 mg-min/m^3, $Probit_{inh,sev}$ = 12[b] $LCt_{50,inh}$ = 33 mg-min/m^3, $Probit_{inh}$ = 12[b]	$ECt_{50,inh,mild}$ = 0.4 mg-min/m^3, $Probit_{inh,mild}$ = 4.5[b] $ECt_{50,inh,sev}$ = 25 mg-min/m^3, $Probit_{inh,sev}$ = 12[b] $LCt_{50,inh}$ = 33 mg-min/m^3, $Probit_{inh}$ = 12[b]
	No percutaneous challenge	$ECt_{50,pc\text{-}v,sev}$ = 4,000 mg-min/m^3[d] $ED_{50,pc\text{-}l,sev}$ = 1,000 mg[d] $LCt_{50,pc\text{-}v}$ = 12,000 mg-min/m^3[d] $LD_{50,pc\text{-}l}$ = 1,700 mg[d]
	Toxic load exponent = 1 (no toxic load)	Toxic load exponent = 1.4
HD	$ECt_{50,oc,mild}$ = 25 mg-min/m^3, $Probit_{inh,mild}$ = 3[b] $ECt_{50,oc,sev}$ = 75 mg-min/m^3, $Probit_{inh,sev}$ = 3[b,e]	$ECt_{50,oc,mild}$ = 25 mg-min/m^3, $Probit_{inh,mild}$ = 3[b] $ECt_{50,oc,sev}$ = 75 mg-min/m^3, $Probit_{inh,sev}$ = 3[b,e]
	$ECt_{50,pc\text{-}v,mild}$ = 50 mg-min/m^3, $Probit_{pc\text{-}v,mild}$ = 3[b] $ECt_{50,pc\text{-}v,sev}$ = 500 mg-min/m^3, $Probit_{pc\text{-}v,sev}$ = 3[b] $ED_{50,pc\text{-}l,sev}$ = 600 mg[f] $LCt_{50,pc\text{-}v}$ = 10,000 mg-min/m^3[f] $LD_{50,pc\text{-}l}$ = 1,400 mg[f]	$ECt_{50,pc\text{-}v,mild}$ = 50 mg-min/m^3, $Probit_{pc\text{-}v,mild}$ = 3[b] $ECt_{50,pc\text{-}v,sev}$ = 500 mg-min/m^3, $Probit_{pc\text{-}v,sev}$ = 3[b] $ED_{50,pc\text{-}l,sev}$ = 600 mg[f] $LCt_{50,pc\text{-}v}$ = 10,000 mg-min/m^3[f] $LD_{50,pc\text{-}l}$ = 1,400 mg[f]
	$LCt_{50,inh}$ = 1,000 mg-min/m^3, $Probit_{inh}$ = 6[b]	$LCt_{50,inh}$ = 1,000 mg-min/m^3, $Probit_{inh}$ = 6[b]
	Toxic load exponent = 1 (no toxic load)	Toxic load exponent = 1

[a] The IDA research team selected the wet form of both biological agents, although this choice should affect only the dispersal and cloud formation, which was not the focus of the casualty estimation analysis.

[b] Probit slope units are probits/log(dose) for biological agents and probits/log(Ct) for chemical agents.

[c] $$\text{Probability of death} = \begin{cases} \text{Probability of infection} * 0.86, \mathrm{D} \leq 10^5 \text{spores} \\ \text{Probability of infection} * \left(1 - 0.5\left(1 + \text{erf}\left[\frac{2.3263 - 0.2959 * \ln \mathrm{D}}{\sqrt{2}}\right]\right)\right), \mathrm{D} > 10^5 \text{spores} \end{cases}$$
Jason Rodriquez, Gene E. McClellan, and Darren R. Oldson, *Chemical-Biological Operational Degradation Analysis (CODA)*, ARA-TR-09-SEASSP-00077.00002-004 (Arlington, VA: Applied Research Associates, September 2010), 26.

[d] These values are used to convert the percutaneous vapor and liquid values into the equivalent inhalation values. Jason Rodriquez, Gene E. McClellan, and Darren R. Oldson, *Chemical-Biological Operational Degradation Analysis (CODA)*, ARA-TR-09-SEASSP-00077.00002-004 (Arlington, VA: Applied Research Associates, September 2010), 18–19.

[e] The HPAC user interface refers to this parameter value as the severe ECt_{50} for inhaled vapor, although FM 3-11.9 refers to it as the severe ECt_{50} ocular value and does not provide a severe ECt_{50} for inhaled vapor. FM 3-11.9, p. II-40.

[f] These values are used to convert the percutaneous liquid values into the equivalent percutaneous vapor values. Jason Rodriquez, Gene E. McClellan, and Darren R. Oldson, *Chemical-Biological Operational Degradation Analysis (CODA)*, ARA-TR-09-SEASSP-00077.00002-004 (Arlington, VA: Applied Research Associates, September 2010), 18–19.

2. Different Effects and Mortality Models

The second data or methodological difference the IDA team identified and controlled for is the varied types of models used for estimating the probabilities of illness and death. As stated earlier, the default HPAC chemical agent III and mortality models ("OP" and "HD") account for multiple routes of exposure. As the *AMedP-7.5* SD2 GB model includes only inhalation challenges, the IDA team changed the default HPAC "OP" model (which includes percutaneous challenges) to the "Probit" model, which considers only inhalation challenges. Both the HPAC "HD" model and the *AMedP-7.5* SD2 HD model include percutaneous and inhalation/ocular challenges, so the IDA team did not need to change the HPAC HD model to "Probit."

In addition, the IDA team replaced the *AMedP-7.5* SD2 chemical agent threshold models, which were based on the probit model parameters used in HPAC, with the probit models themselves to make a direct comparison with HPAC. To further ensure comparability with HPAC results, the resulting probabilities of illness calculated for the various routes of exposure for HD using the *AMedP-7.5* SD2 methodology (with probit models instead of threshold models) were combined according to Equation 3.

Both methodologies already used the same model (probit) for the biological agents, so no changes were needed for a meaningful comparison. The exception to this was the anthrax mortality model. As the *AMedP-7.5* SD2 methodology models anthrax lethality as a case fatality rate of 100%, the IDA team ignored the HPAC mortality outputs and defined the fatalities as equal to the total casualties to align the two methodologies.

3. Different Representations of Challenge Values (Mean Value versus Distribution)

The third data or methodological difference the IDA team identified is the different treatment of the challenge values associated with each location. HPAC considers each location to have received a distribution of challenge values, whereas the *AMedP-7.5* SD2 methodology uses only the mean challenge value. As a result, the probability of illness/death calculations for the two methodologies differ. The *AMedP-7.5* SD2 methodology uses Equation 1 (with only the mean challenge value from HPAC), whereas HPAC uses Equation 2, which calculates the expected probability of illness/death by taking a weighted average across the entire distribution of possible challenge values.

The IDA team did not control for this methodological difference in the same manner as for the other data or methodological differences by making a direct change to either methodology. The change could not be made within HPAC because it does not provide a

way to alter the default calculations when outputting casualties. In order to change the *AMedP-7.5* SD2 methodology to account for this methodological difference, either the IDA team would need to solve for the parameters specifying the challenge distribution at each location needed to implement Equation 2 (something it only understood how to do late in the analysis) or HPAC would need to output the parameters directly. As a result, instead of making the change directly to either methodology, the IDA team used the outputs of HPAC's graphic user interface contour plotting feature to approximate the resulting difference in casualty estimates. Unlike the casualty estimation portion of the HPAC methodology, the contour plotting feature supported the option to select between the two representations of challenge values: the mean challenge value (via the "within contour" method) or the entire challenge distribution (via the "statistical mean" method).

To approximate the estimated total casualties and fatalities using this alternate approach, the IDA team first used HPAC's calculated in contour populations for contour levels ranging from the ECt_{01} to the ECt_{99}. Next, the populations between successive contours were calculated by subtracting the population within the higher contour value from the population within the lower contour value. For example, the number of people with challenges between the ECt_{01} and the ECt_{02} was calculated as the population within the ECt_{01} contour minus the population within the ECt_{02} contour. The IDA team then used the lower contour value to estimate the number of casualties for each group of individuals between contours. For instance, an estimated 1% of the individuals receiving a challenge between the ECt_{01} and the ECt_{02} were casualties. Finally, the IDA team added the estimated casualties for all groups between contours to determine an approximate value for the total casualties or fatalities for each agent, effect level (total casualties (mild or greater), total casualties (severe or greater), and fatalities), and computational method ("within contour" and "statistical mean"). These estimates should differ slightly from the casualties predicted by HPAC and the *AMedP-7.5* SD2 methodology due to the small variation of challenge within each population between contours.

For a given agent, HPAC could only output the in contour populations for a single challenge type. For the GB (Probit model), anthrax, and botulinum toxin models, which only predict casualties as a function of the inhaled Ct or dose, a single contour plot captured all necessary routes of exposure. However, a single contour plot was insufficient for generating the information on the multiple routes of exposure needed for the HPAC "HD" model. The additional effects of percutaneous vapor and liquid HD challenges therefore could not be incorporated into the approximation of HPAC's casualty estimate using the mean challenge rather than a challenge distribution; only inhalation/ocular challenges were used to estimate casualties.[24] As a result, the HD approximated casualty

[24] Three separate contour plots were generated in HPAC. The HD ocular ECt_{50} and probit slope values from Table 4 were used for calculating total casualties (mild and severe), while the inhalation LCt_{50} and probit slope values were used for calculating fatalities.

estimates are likely underestimates of what HPAC would predict if it could estimate casualties using the mean challenge value for all routes of exposure.

4. Results

A. Presentation of Results

The previous chapter described those data and methodological differences the IDA team identified and controlled for during this analysis. In order to isolate the effect of each change on the casualty estimates, the IDA team accounted for only one data or methodological difference at a time in a step-wise fashion, causing the models and parameter values used by HPAC and the *AMedP-7.5* SD2 methodology, and consequently their casualty estimates, to converge with each step.

This chapter presents the results of the analysis for each of the four agents in figures similar to Figure 2, which displays the total casualties for a notional example. The blue bars represent casualties computed using HPAC, whereas the red bars represent casualties computed using the *AMedP-7.5* SD2 methodology. The outermost bars correspond to the casualties estimated using the default parameters and internal methods for the two methodologies (HPAC on the left and *AMedP-7.5* SD2 on the right). Moving toward the center, each successive bar represents casualties estimated after a single data and methodological change relative to the outer adjacent bar of the same color. The bar labels specify the changes corresponding to each new estimate, and arrows indicate the progression from the default methodology estimates to the adjusted estimates. One methodological change could not be made directly in the *AMedP-7.5* SD2 methodology or HPAC's casualty estimation methodology. In order to approximate the adjusted casualty estimate after accounting for this change, the IDA team used another HPAC functionality, estimation of populations in various challenge contour levels, to indirectly assess the change in the casualty estimates resulting from this methodological change. The estimated casualties approximated this way are represented by the blue/green bar. After controlling for all identified data and methodological differences in one methodology or the other, the two methodologies converged, and the resulting casualty estimates (represented by the blue/green bar and the adjacent red bar) were very similar.

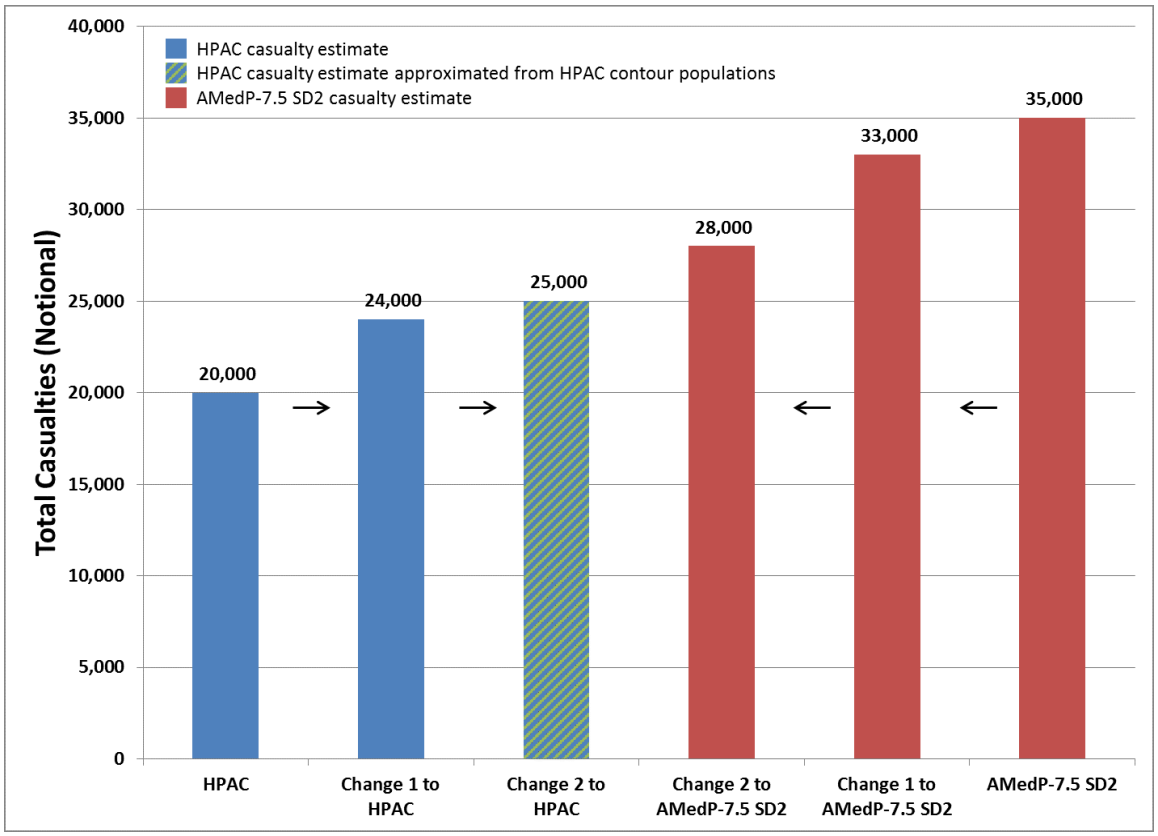

Figure 2. Total Casualties Notional Example

In the notional example in Figure 2, the "total observed difference" between the default casualty estimates was 15,000 casualties (20,000 for HPAC versus 35,000 for *AMedP-7.5* SD2). By making two changes to each methodology to control for known data and methodological differences, this total observed difference was reduced to an "unexplained observed difference" of 3,000 (25,000 versus 28,000). The difference between these two values, 12,000, is attributable to the data and methodological changes and is the "explained observed difference." The green/blue bar, which represents the HPAC estimate after controlling for all the changes made within HPAC, will be referred to as the "adjusted HPAC estimate." Likewise, the innermost red bar is designated the "adjusted *AMedP-7.5* SD2 estimate."

The results in this chapter will show a relatively small unexplained observed difference for the four agents analyzed, indicating that the changes made to control for the data and methodological differences identified in Chapter 3 accounted for the vast majority of the total observed difference.

B. Chemical Agent Casualty Estimates

1. GB

For the chemical agents, the IDA team made three comparisons: total casualties (mild or greater), total casualties (severe or greater), and fatalities. Figure 3 shows the results for GB total casualties (mild or greater), with the default HPAC estimate of 14,297 differing from the default *AMedP-7.5* SD2 estimate of 33,700 by 19,403. Changing the HPAC OP model to Probit and controlling for the toxic load exponent resulted in relatively minor changes to the casualty estimate, whereas using only the mean challenge value rather than a distribution significantly increased the HPAC casualty estimate (by approximately a factor of two). Changing the *AMedP-7.5* SD2 threshold model to a probit model resulted in fewer predicted casualties, bringing the *AMedP-7.5* SD2 estimate closer to the HPAC estimate. After controlling for these data and methodological differences, the unexplained observed difference was 1,771 total casualties, 9% of the total observed difference.

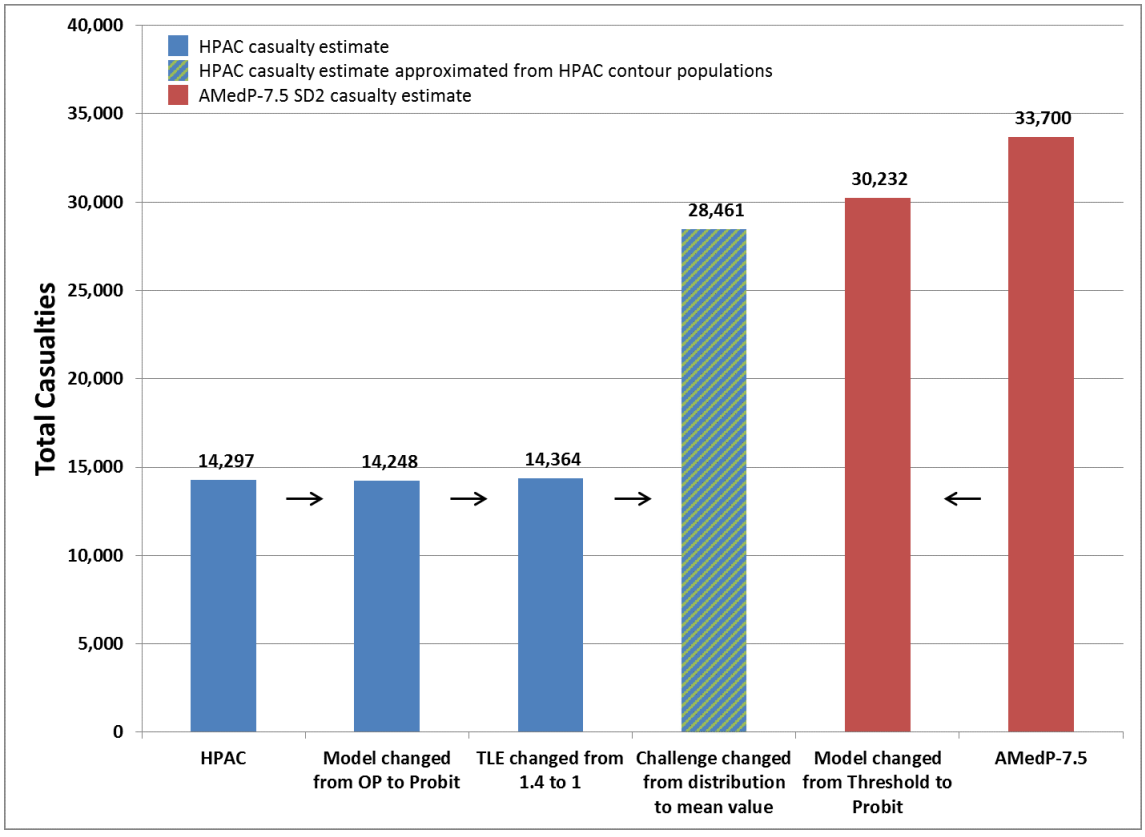

Figure 3. GB Total Casualties (Mild or Greater)

Inexplicably, the default HPAC GB total casualties (severe or greater) estimate is *higher* than the HPAC total casualties (mild or greater) estimate. Although the mild and

severe curves do cross (at approximately 298.86 mg-min/m^3; see Figure 4), the Ct distribution would need to be heavily skewed toward values higher than this crossover point to result in greater predicted casualties for the severe values. Realistically, as the crossover point is so far in the upper portion of both the mild and severe curves (where the probability of becoming a casualty is essentially 100% for both), even for the most extreme Ct distributions of only values higher than the crossover point, the mild and severe estimates should be equal; the severe estimate should never be higher than the mild estimate. This peculiarity in HPAC seems to be isolated to the OP model, as the switch from the default OP model to the probit model led to a significantly lower estimate of total casualties (severe or greater), one that is more in line with the other severe estimates.

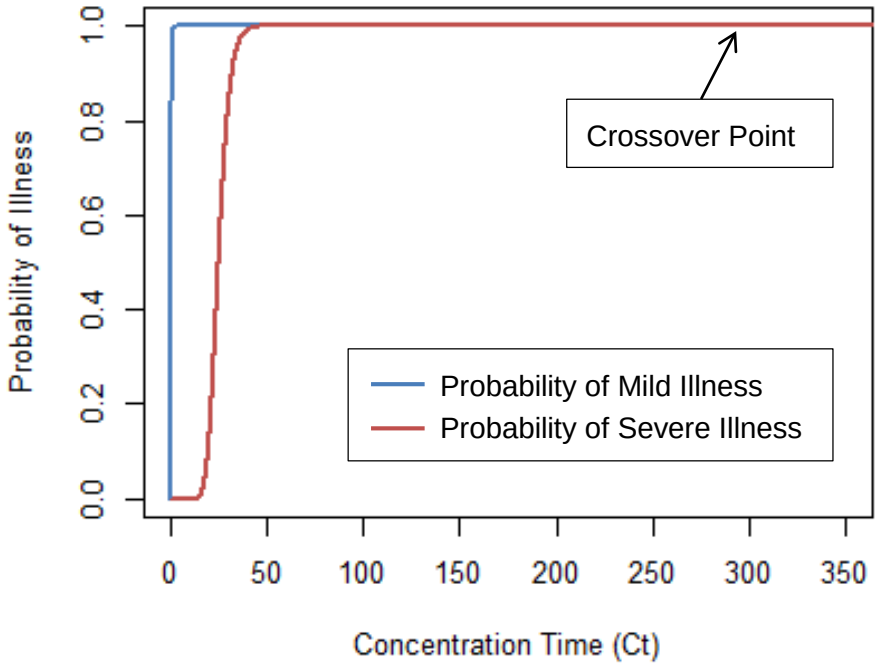

Figure 4. Comparison of GB Mild and Severe Probit Curves; Probability of Mild Illness > Probability of Severe Illness for Ct Values Less than the Crossover Point

As shown in Figure 5, the default HPAC estimate of total casualties (severe or greater) was 14,421, and changing to the probit model significantly reduced that value to a number lower than the equivalent value for the total casualties (mild or greater) and more in line with expectations. Adjusting the HPAC toxic load exponent from 1.4 to 1 increased the estimated total casualties as expected, and changing the challenge distribution to a mean value slightly decreased the estimated total casualties. The default estimate using the *AMedP-7.5* SD2 methodology was 4,248 total casualties (severe or greater), 10,173 lower than the default HPAC estimate. Converting the threshold model

to a probit model decreased the *AMedP-7.5* SD2 estimate to 2,306. Thus, the adjusted HPAC estimate (2,204) and the adjusted *AMedP-7.5* SD2 estimate (2,306) differed by only 102, and the IDA team was therefore able to explain 99% of the total observed difference between the default estimates with the four changes described.

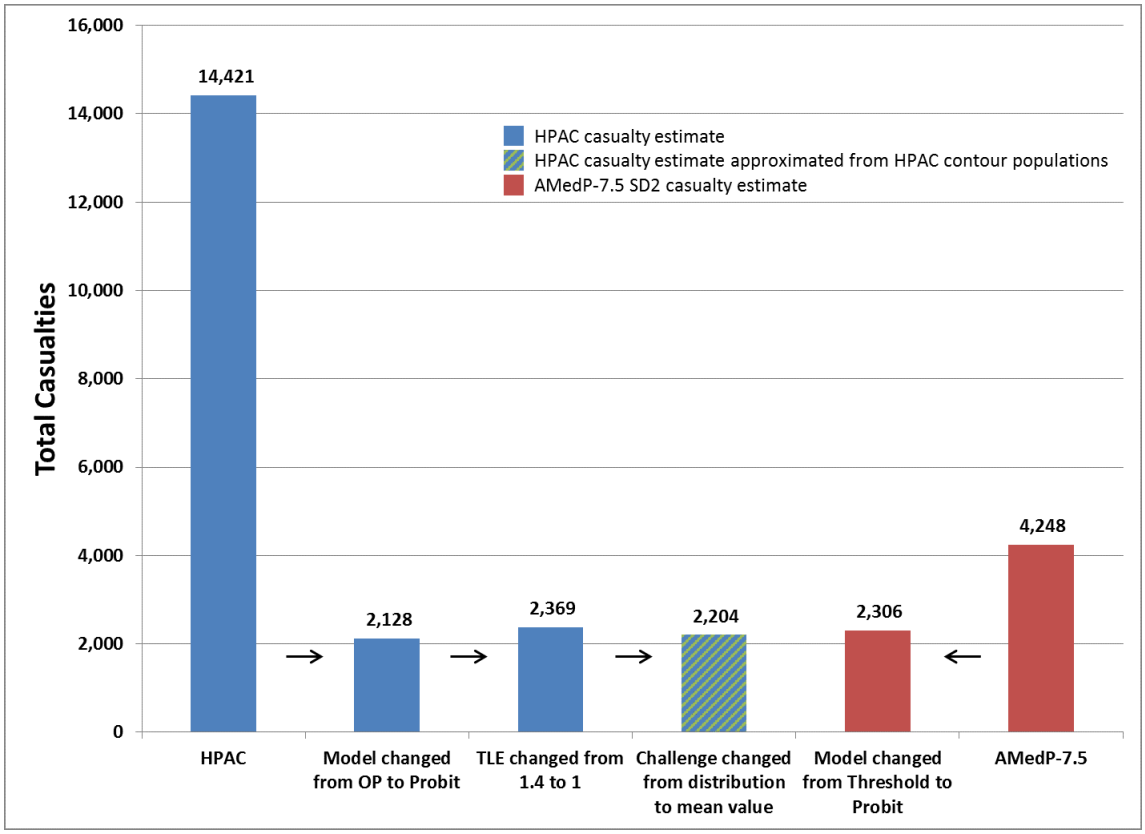

Figure 5. GB Total Casualties (Severe or Greater)

The total observed difference between the default HPAC GB fatalities estimate of 1,722 and the default *AMedP-7.5* SD2 estimate of 2,128 was 406. A slight decrease occurred in the HPAC estimate using the probit model instead of the OP model, but decreasing the toxic load exponent to 1 increased the estimate. Again, the change from a challenge distribution to a mean value decreased the estimated casualties, resulting in an adjusted HPAC estimate of 1,765. The adjusted *AMedP-7.5* SD2 fatalities estimate, which accounted for the change from the threshold model to the probit model, was 1,855. With the four data and methodological differences between the methodologies accounted for, the IDA team could explain 78% (all but 90) of the total observed difference between estimated fatalities. All GB fatalities estimates are shown in Figure 6.

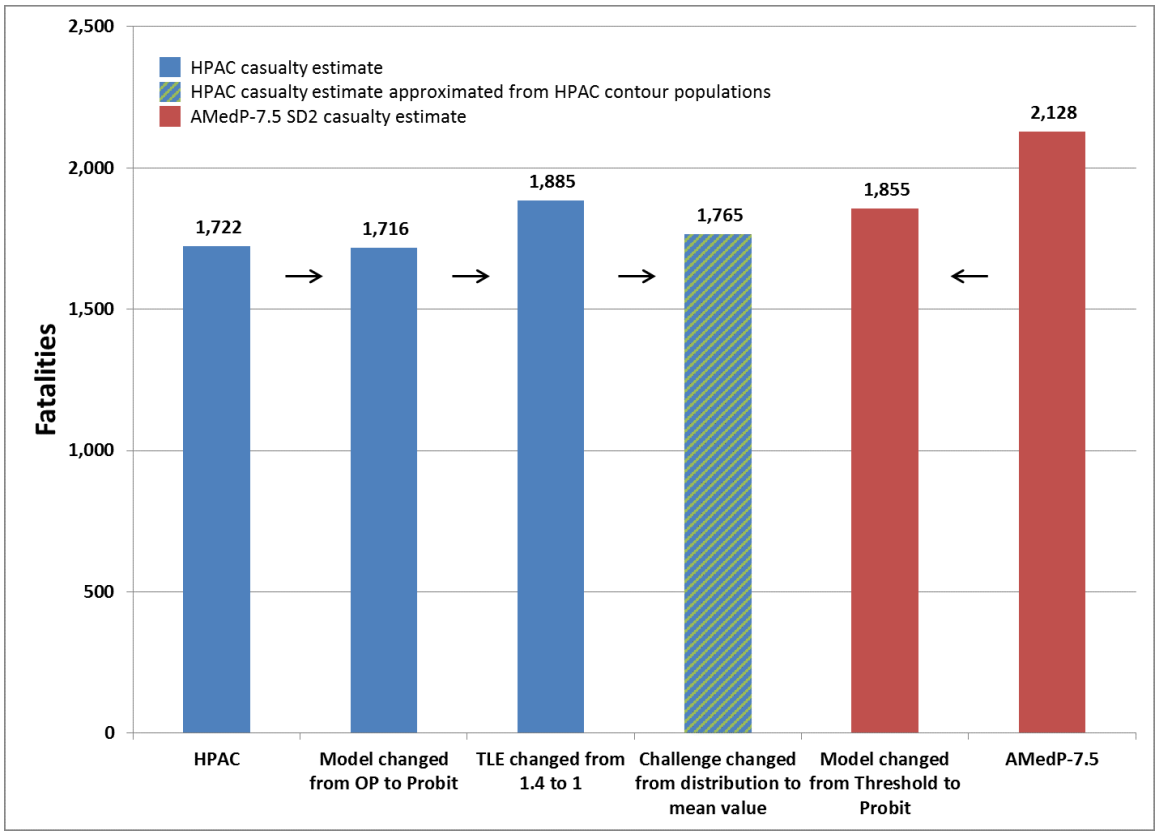

Figure 6. GB Fatalities

Table 5 summarizes the estimates of GB total casualties (mild or greater), total casualties (severe or greater), and fatalities for the different variations of the HPAC and *AMedP-7.5* SD2 methodologies. The unexplained observed difference is calculated by subtracting the smaller of the adjusted estimates (in the case of GB fatalities, the adjusted HPAC estimate) from the larger (in the case of GB fatalities, the adjusted *AMedP-7.5* SD2 estimate). For GB, the unexplained observed differences ranged from 90 (fatalities) to 1,771 (total casualties (mild or greater)) and from 1% of the total observed difference (for total casualties (severe or greater)) to 22% (for fatalities). As mentioned earlier, the total observed difference is the sum of the explained and unexplained observed difference.

Table 5. Explained and Unexplained Observed Differences in Estimated GB Total Casualties and Fatalities

	Total Casualties (Mild or Greater)	Total Casualties (Severe or Greater)	Fatalities
HPAC Estimate	14,297	14,421	1,722
AMedP-7.5 SD2 Estimate	33,700	4,248	2,128
Total Observed Difference	**19,403**	**10,173**	**406**
Adjusted HPAC Estimate	28,461	2,204	1,765
Adjusted *AMedP-7.5* SD2 Estimate	30,232	2,306	1,885
Unexplained Observed Difference	**1,771 (9%)**	**102 (1%)**	**90 (22%)**
Explained Observed Difference	**17,632 (91%)**	**10,071 (99%)**	**316 (78%)**

2. HD

As with GB, three sets of estimates were calculated for HD: total casualties (mild or greater), total casualties (severe or greater), and fatalities. As mentioned in the previous chapter, the HPAC casualty estimates approximated using the contour populations from HPAC are likely underestimates due to the exclusion of percutaneous challenges from the calculations. The comparison of estimated HD total casualties (mild or greater) is shown in Figure 7. A total observed difference of 8,420 total casualties (mild or greater) separated the default HPAC estimate of 3,733 and the default *AMedP-7.5* SD2 estimate of 12,153. Changing from a threshold to a probit model resulted in a significant decrease in the *AMedP-7.5* SD2 estimate. Using a mean challenge value rather than a challenge distribution also resulted in a decrease in the HPAC casualty estimate. The remaining unexplained observed difference between the adjusted estimates was 341, just 4% of the total observed difference.

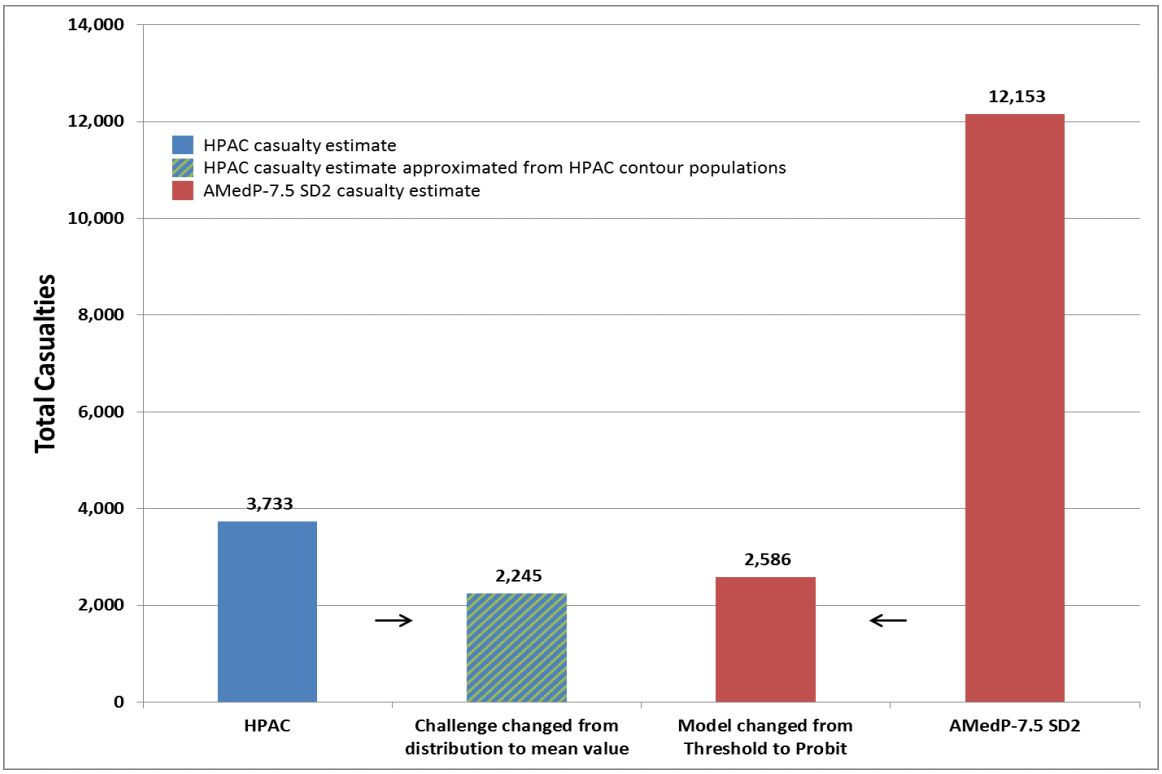

Figure 7. HD Total Casualties (Mild or Greater)

The estimates of total casualties (severe or greater) for HD are shown in Figure 8. The total observed difference between the default HPAC estimate of 1,350 and the default *AMedP-7.5* SD2 estimate of 738 is 612. Changing the *AMedP-7.5* SD2 threshold model to the probit model increased the *AMedP-7.5* SD2 estimate to 895. Controlling for the methodological difference of using different challenge values resulted in an adjusted HPAC estimate of 859. The unexplained observed difference between the two adjusted estimates is therefore 36, or 6% of the total observed difference.

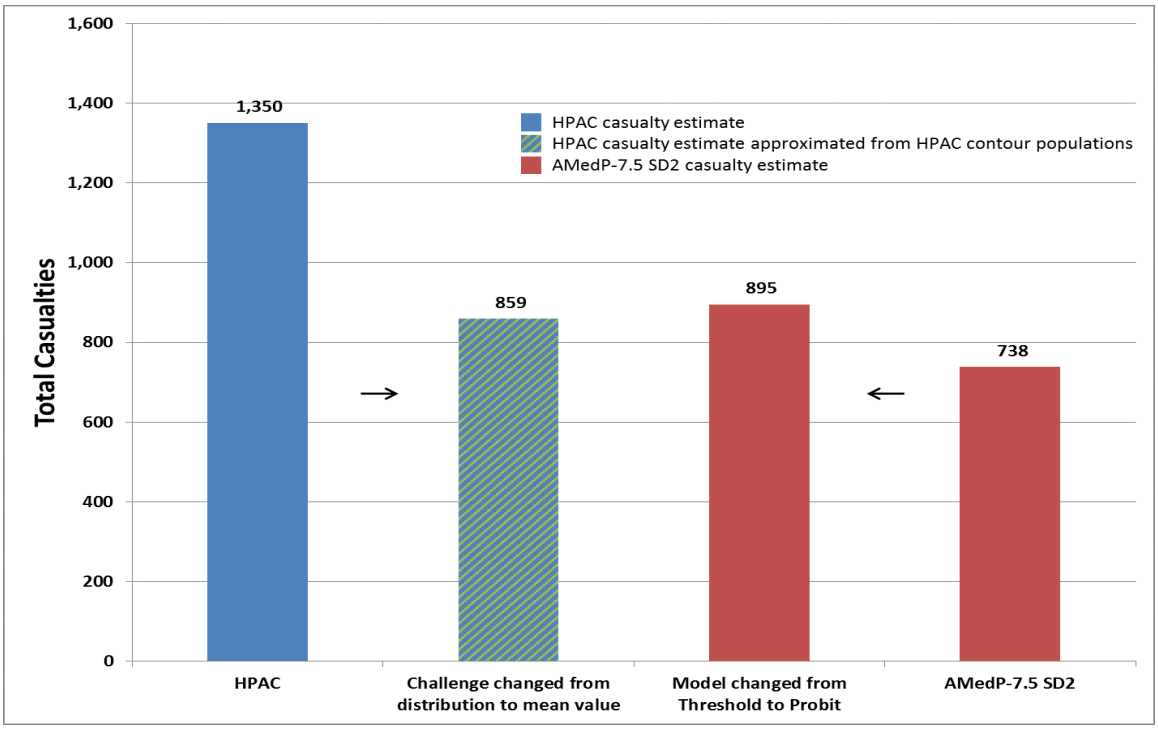

Figure 8. HD Total Casualties (Severe or Greater)

Figure 9 reports the fatality estimates for HD using the various methods. The default fatalities estimates for HPAC and the *AMedP-7.5* SD2 methodology are 136 and 255, respectively, a difference of 119 fatalities. After controlling for the threshold model and the challenge distribution versus mean value, the remaining unexplained observed difference was 25 fatalities (21% of the total observed difference).

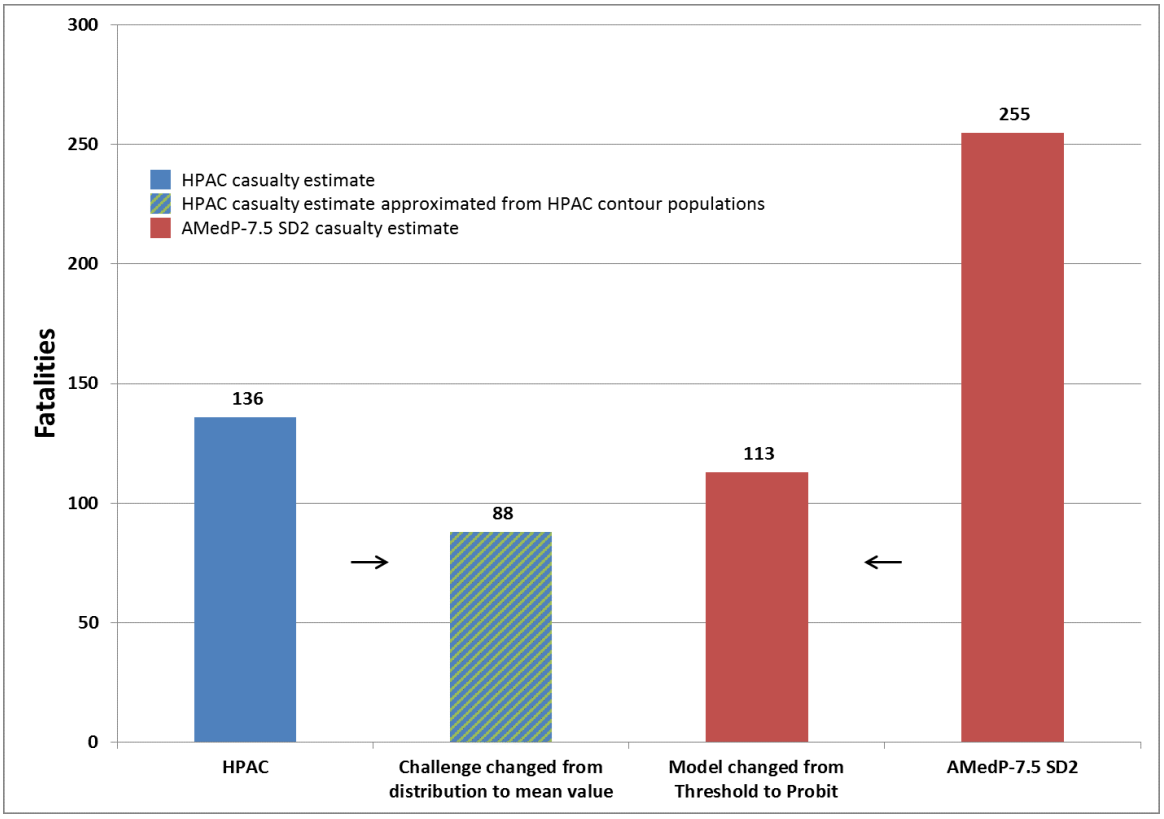

Figure 9. HD Fatalities

The total observed differences, unexplained observed differences, and explained observed differences for HD total casualties (mild or greater), total casualties (severe or greater), and fatalities are summarized in Table 6. The largest unexplained observed difference in terms of absolute numbers (341) was for the total casualties (mild or greater), but this value made up only 4% of the total observed difference. Conversely, the unexplained observed difference in fatalities was only 25 (the smallest absolute observed difference), but because the default estimates were quite close before any adjustments, the unexplained observed difference of 25 accounted for 21% of the total observed difference.

Table 6. Explained and Unexplained Observed Differences in Estimated HD Total Casualties and Fatalities

	Total Casualties (Mild or Greater)	Total Casualties (Severe or Greater)	Fatalities
HPAC Estimate	3,733	1,350	136
AMedP-7.5 SD2 Estimate	12,153	738	255
Total Observed Difference	**8,420**	**612**	**119**
Adjusted HPAC Estimate	2,245	859	88
Adjusted *AMedP-7.5* SD2 Estimate	2,586	895	113
Unexplained Observed Difference	**341 (4%)**	**36 (6%)**	**25 (21%)**
Explained Observed Difference	**8,079 (96%)**	**576 (94%)**	**94 (79%)**

C. Biological Agent Casualty Estimates

1. Anthrax

The results for the anthrax casualty estimates are shown in Figure 10 and Figure 11, which display the total casualties and fatalities, respectively. The total observed difference between the anthrax total casualties estimated using HPAC (2,777) and the *AMedP-7.5* SD2 methodology (41,583), shown in Figure 10, was 38,806. Changing the probit slope and ID_{50} values in HPAC to match those used in the *AMedP-7.5* SD2 methodology actually decreased the HPAC casualty estimate and *increased* the observed difference between the estimated total casualties. Changing the challenge distribution at each location to a mean value, however, greatly increased the HPAC estimate (by nearly a factor of 15) and resulted in an unexplained observed difference of 2,116. Thus, the data and methodological differences the IDA team controlled for explained 95% of the total observed difference.

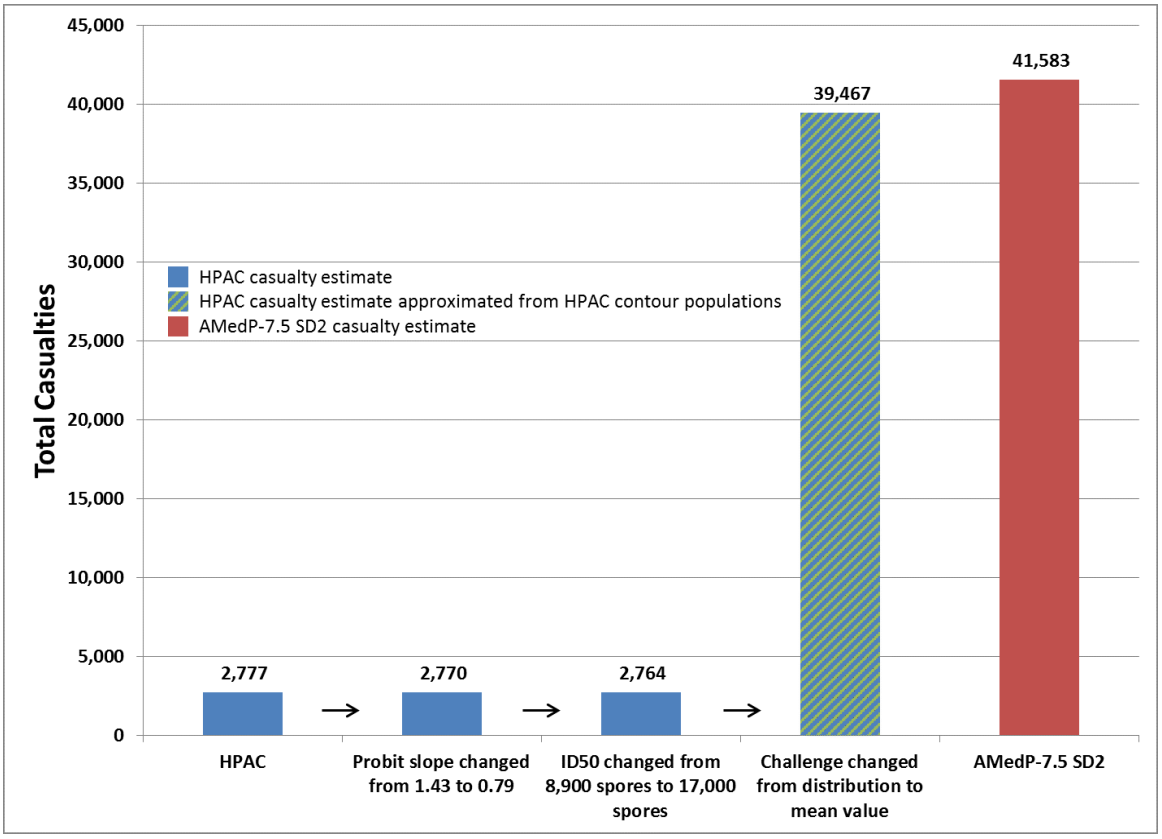

Figure 10. Anthrax Total Casualties

Because it uses a 100% case fatality rate, the *AMedP-7.5* SD2 methodology estimated the same number of anthrax fatalities (41,583) as total casualties, whereas the HPAC mortality model predicts fewer fatalities (2,770) than total casualties, as it allows for some survivors. As a result, a slightly greater total observed difference existed between the two methodologies for anthrax fatalities (38,813) than total casualties. As all other data and methodological differences between the two methodologies accounted for, shown in Figure 11, were the same as for total casualties, the unexplained observed difference remained 2,116.

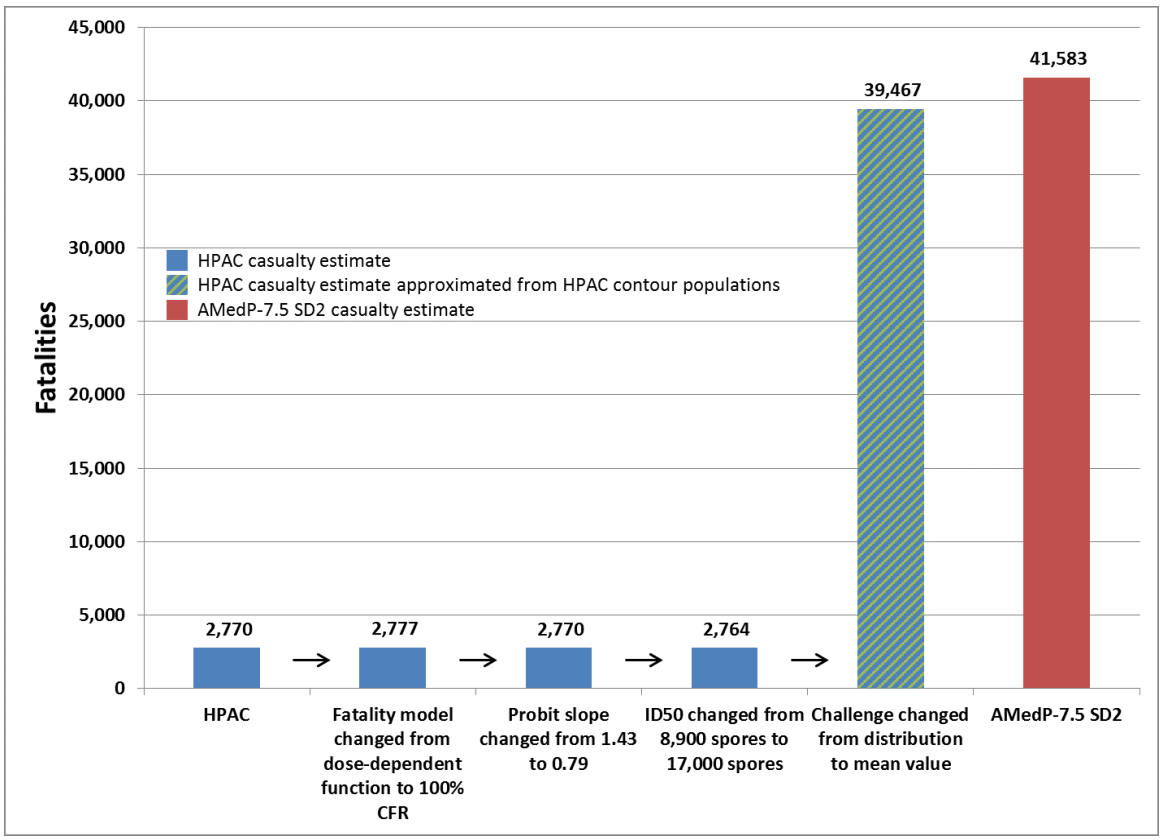

Figure 11. Anthrax Fatalities

Table 7 summarizes the observed differences between the HPAC and the *AMedP-7.5* SD2 estimates for both anthrax total casualties and fatalities, including the portions of the total observed differences that the data and methodological differences can and cannot explain. As with the chemical agents, the total observed difference between the default estimates represents the disparity that the IDA team hoped to explain by controlling for known data and methodological differences between the two methodologies. The observed difference between the adjusted HPAC estimate and the adjusted *AMedP-7.5* SD2 estimate is the portion of the total observed difference that was left unexplained (5% for both total casualties and fatalities).

Table 7. Explained and Unexplained Observed Differences in Estimated Anthrax Total Casualties and Fatalities

	Total Casualties	Fatalities
HPAC Estimate	2,777	2,770
AMedP-7.5 SD2 Estimate	41,583	41,583
Total Observed Difference	**38,806**	**38,813**
Adjusted HPAC Estimate	39,467	39,467
Adjusted *AMedP-7.5* SD2 Estimate	41,583	41,583
Unexplained Observed Difference	**2,116 (5%)**	**2,116 (5%)**
Explained Observed Difference	**36,690 (95%)**	**36,697 (95%)**

2. Botulinum Toxin

The botulinum toxin estimates of total casualties and fatalities are shown in Figure 12 and Figure 13, respectively. As for anthrax, the *AMedP-7.5* SD2 botulinum toxin total casualties estimate (31,117) was significantly higher than the HPAC estimate (1,536). Adjusting the probit slope and ED_{50} values in HPAC to match those used in the *AMedP-7.5* SD2 methodology marginally increased the HPAC casualty estimate. Replacing the challenge distribution with only the mean value in the casualty calculations resulted in a significant increase in the HPAC casualty estimate (by a factor of nearly 20). The net result was an unexplained observed difference of 1,565 total casualties (5% of the total observed difference).

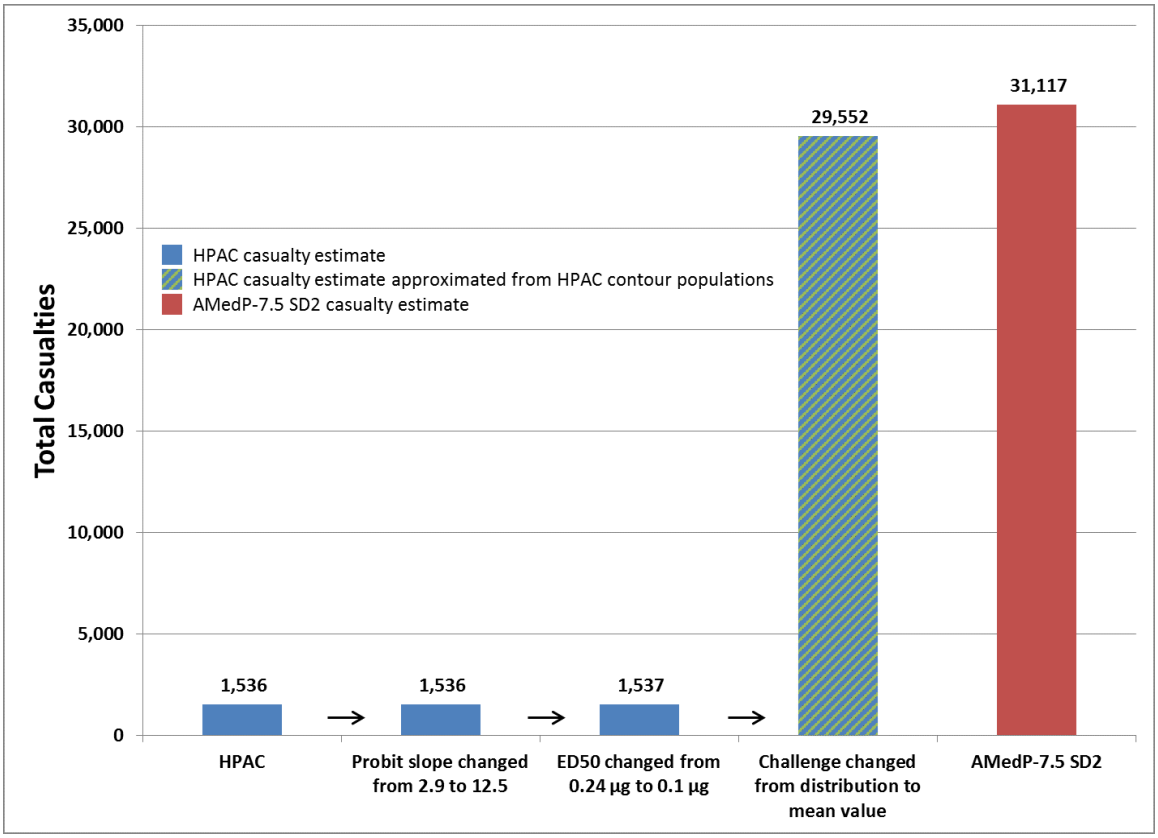

Figure 12. Botulinum Toxin Total Casualties

As shown in Figure 13, the default HPAC and *AMedP-7.5* SD2 botulinum toxin fatality estimates (1,531 and 19,701, respectively) varied by 18,170. Controlling for the different probit slope and LD_{50} values and the difference between the challenge distribution and mean value reduced this to an unexplained observed difference of 844 (5% of the total observed difference). Table 8 summarizes the botulinum toxin total casualties and fatalities estimates.

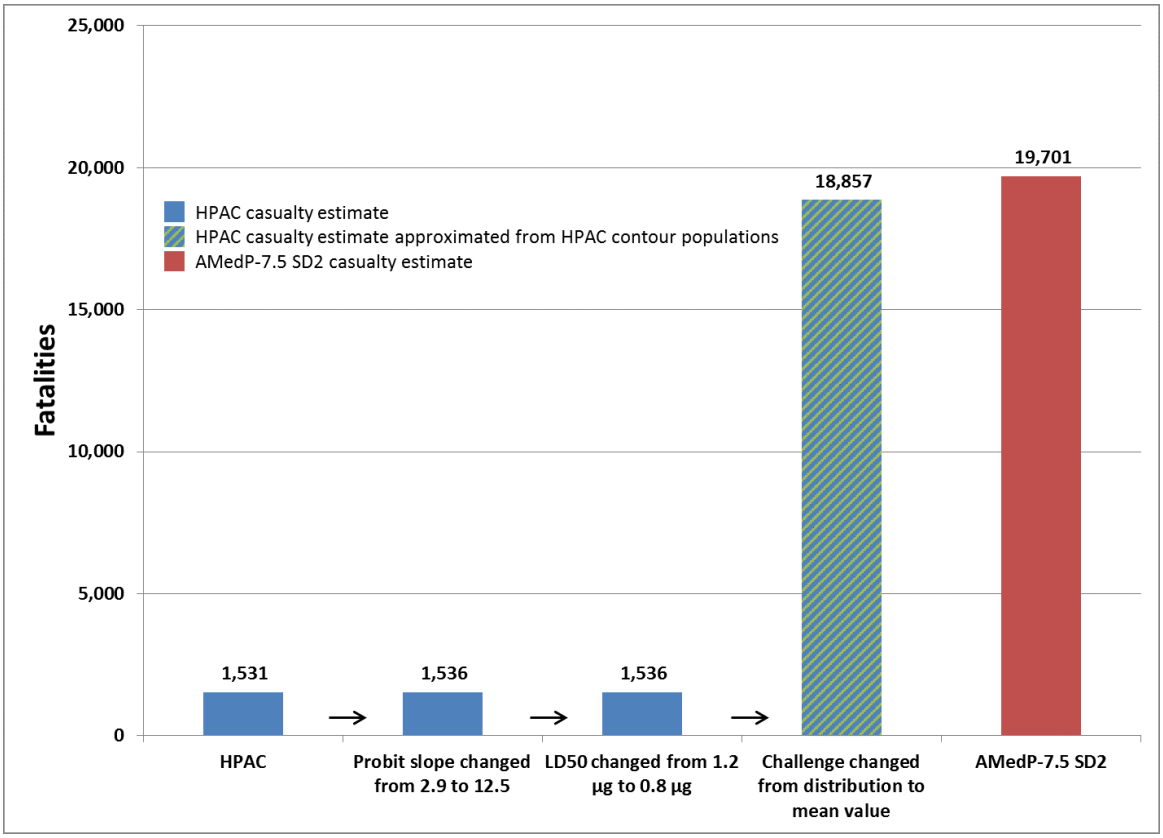

Figure 13. Botulinum Toxin Fatalities

Table 8. Explained and Unexplained Observed Differences in Estimated Botulinum Toxin Total Casualties and Fatalities

	Total Casualties	Fatalities
HPAC Estimate	1,536	1,531
AMedP-7.5 SD2 Estimate	31,117	19,701
Total Observed Difference	**29,581**	**18,170**
Adjusted HPAC Estimate	29,552	18,857
Adjusted *AMedP-7.5* SD2 Estimate	31,117	19,701
Unexplained Observed Difference	**1,565 (5%)**	**844 (5%)**
Explained Observed Difference	**28,016 (95%)**	**17,326 (95%)**

D. Summary of Results

The results in this chapter highlight the large total observed differences between the default HPAC and *AMedP-7.5* SD2 casualty estimation methodologies, which are summarized in Table 9 for each agent. The results of this analysis also indicate that by controlling for the data and methodological differences described in Chapter 3, the IDA team explained the vast majority of the total observed difference between the HPAC and

AMedP-7.5 SD2 casualty estimates. Table 10 summarizes the explained observed difference between the two methodologies' casualty estimates for all four agents as a percentage of the total observed difference. With the exception of the fatality estimates for the two chemical agents, the explained observed difference was more than 90% of the total observed difference. The percentage explained of the total observed difference between the two methodologies' estimates for GB and HD fatalities, 78% and 79%, respectively, can likely be attributed to a small sample size. The total observed differences for the two agents were 406 fatalities for GB and 119 fatalities for HD, which are much smaller than the total observed differences for the other casualty estimates relative to the uncertainty in the approximated casualty estimate of the HPAC fatalities using the mean challenge value rather than the challenge distribution.

Table 9. Total Observed Difference between HPAC and *AMedP-7.5* SD2 Casualty Estimates before Accounting for Data and Methodological Differences

	Total Casualties		
Agent	**(Mild or Greater)**	**(Severe or Greater)**	**Fatalities**
GB	19,403	10,173	406
HD	8,420	612	119
Anthrax	38,806		38,813
Botulinum Toxin	29,581		18,170

Table 10. Percentage of Total Observed Difference between HPAC and *AMedP-7.5* SD2 Casualty Estimates Explained by Data and Methodological Differences

	Total Casualties		
Agent	**(Mild or Greater)**	**(Severe or Greater)**	**Fatalities**
GB	91%	99%	78%
HD	96%	94%	79%
Anthrax	95%		95%
Botulinum Toxin	95%		95%

To get a sense of how large each unexplained observed difference was relative to the adjusted estimates, the IDA team computed the percentage difference between the two adjusted estimates, which is reported in Table 11. This metric, the quotient of the unexplained observed difference and the average of the two adjusted estimates, signifies how close the two adjusted estimates are. For instance, the GB total casualties (mild or greater) adjusted *AMedP-7.5* SD2 estimate of 30,232 and the adjusted HPAC estimate of 28,461 differ by about 6%. As shown in Table 11, the adjusted casualty estimates for GB, anthrax, and botulinum toxin were all within 6% of each other. Recall that the HPAC

adjusted estimate for HD was based on an approximated casualty estimate derived using only the in contour population for inhalation/ocular challenges. The missing contribution of the percutaneous challenges likely accounts for the larger percentage difference between the two methodologies' casualty estimates for HD. Overall, such close proximity between the adjusted estimates suggests that the changes made by the IDA team to control for the known data and methodological differences explained nearly all of the observed difference between the two methodologies' casualty estimates.

Table 11. Percentage Difference of Adjusted HPAC and *AMedP-7.5* Casualty Estimates (Unexplained Observed Difference/Average of Adjusted Estimate)

	Total Casualties		
Agent	**(Mild or Greater)**	**(Severe or Greater)**	**Fatalities**
GB	6%	5%	5%
HD	14%	4%	25%
Anthrax	5%		5%
Botulinum Toxin	5%		4%

5. Observations and Discussion

A. Impediments to an Exact Match between Casualty Estimates

This analysis highlights a number of data and methodological differences between HPAC and the *AMedP-7.5* SD2 methodology, namely different human response parameter values, different effects and mortality models, and different representations of the challenge values (i.e., full distribution versus mean value). The previous chapter's results illustrate that controlling for these data and methodological differences explained most of the total observed differences between the two methodologies' casualty estimates. Yet, this analysis also highlights that, at present, no means exist for HPAC to output the exact casualty estimates as the *AMedP-7.5* SD2 methodology nor a simple way for the *AMedP-7.5* SD2 methodology to exactly replicate HPAC's casualty estimates.

A direct comparison between the two methodologies faces two impediments. The first impediment, described in Chapter 3 under the "Scenario Inputs" section, is the inability to easily replicate HPAC's alignment of the challenge values to the number of people in the population receiving those challenge values. The second impediment is the third data or methodological difference described in Chapter 3: the use of a challenge distribution in HPAC and a single mean integrated concentration or dose value in the *AMedP-7.5* SD2 methodology. To address both issues, the IDA team approximated the HPAC methods, which prevented an exact match between the two methodologies' casualty estimates. The following sections describe the functionalities that would need to be added to the two methodologies to allow their casualty estimates to match exactly.

1. Reproducing *AMedP-7.5* SD2 Methodology Estimates in HPAC

a. Matching Challenge Values to Populations

The *AMedP-7.5* SD2 methodology allows users to specify the number and locations of individuals in a scenario. In order to determine the challenge values at the specified locations, the user can specify a sampler in HPAC at each location of interest, which outputs the challenge information. The user then inputs the challenge and associated population information into the *AMedP-7.5* SD2 methodology to calculate casualties. Currently, HPAC has no mechanism to calculate casualties for population distributions that are not uniform or specified at the resolution of the LandScan grid. In order for HPAC to calculate casualties for the customized population distribution, it would need to allow the user to associate the user-specified population at each location with the challenge location information that feeds into HPAC. This option would guarantee that

the matching of challenge values to populations would be identical in both methodologies.

b. Using Mean Challenge Value Rather Than Distribution

With HPAC's graphic user interface contour plotting feature, HPAC users can opt to report the estimated populations exposed to challenge levels at or above various threshold values in two different ways: using either the mean challenge values at each location of interest or the challenge distributions. If a similar option to use either challenge characterization were available in the casualty estimation portion of HPAC, then HPAC would be able to replicate the *AMedP-7.5* SD2 methodology's use of the probit model (Equation 1) to estimate the probabilities of illness and death using the mean challenge values. Instead, only the default option is available to use Equation 2 to calculate the probabilities of illness and death by integrating the challenge distribution.

2. Reproducing HPAC Estimates in *AMedP-7.5* SD2 Methodology

a. Matching Challenge Values to Populations

The *AMedP-7.5* SD2 methodology can estimate casualties given any combinations of challenge value and population exposed to that challenge. In order to ensure that it aligns challenge and population values in the same way as HPAC, a user would need to extract the exact values used in HPAC's calculations for the *AMedP-7.5* SD2 methodology to then use as inputs. To do this, one could use the HPAC output specifying the location of the adaptive grid points used in the scenario and the associated challenge values. In addition, this output also includes information that could allow the user to calculate the fraction of the LandScan cell that was exposed to that challenge. Based on that information and the known LandScan cell population values, the user could determine the challenge and population values needed as inputs to the *AMedP-7.5* SD2 methodology. While a script could be written to calculate the necessary population information for use in the *AMedP-7.5* SD2 methodology, it would be easier if HPAC simply output both needed values in tabular form.

b. Using Challenge Distribution Rather Than Mean Value

HPAC currently outputs the mean and variance associated with a clipped normal distribution of challenge values at each sampler location. However, those are not the parameter values needed to specify the pdf used in Equation 2 and calculate the probabilities of illness and death from distributions of challenge. HPAC uses the mean and variance values as inputs to a lookup table to approximate the necessary parameters for use in Equation 2. It was not until late in the analysis that the IDA team learned of a

way to calculate those parameters from the mean and variance output by HPAC.[25] As the approximation of the change from a challenge distribution to a mean value had already resulted in adjusted HPAC and *AMedP-7.5* SD2 casualty estimates that were very close, and the numerical calculation of the parameters for use in Equation 2 were not guaranteed to match those derived using the lookup table in HPAC, the IDA team decided not to invest further time to implement these calculations.

Another way to reproduce the Equation 2 calculation in the *AMedP-7.5* SD2 methodology would be to have HPAC output the values of the needed parameters after it finds them in the lookup table. An option for outputting either the pre- or post-lookup table parameter values would allow the IDA team and other analysts to reproduce HPAC's casualty estimates using the challenge distribution or alternatively to use only the mean value.

B. Significant Impact on Casualty Estimates of Change from Challenge Distribution to Mean Value

As shown in the previous chapter, a significant source of the observed differences between casualty estimates from the two methodologies is the use of a distribution of challenge values in the calculation of casualties in HPAC and a mean challenge value used in the *AMedP-7.5* SD2 calculation of casualties. The difference between casualty estimates using the two different representations of challenge was especially large for biological agents, with the ratio of casualties estimated using the mean value to casualties estimated using the challenge distribution approaching 15 for anthrax and 20 for botulinum toxin. For the chemical agents, the ratio was 2 for GB casualties (mild or greater) and 0.9 for GB casualties (severe or greater) and fatalities and 0.6 for all HD casualties.

While the magnitude of the change in the casualty estimates as a result of this methodological difference is noteworthy, the IDA team does not recommend changing the *AMedP-7.5* SD2 methodology to reflect a distribution of challenge values (rather than a point estimate) at each icon's location. The *AMedP-7.5* SD2 methodology is agnostic as to the choice of the transport and dispersion model used to determine the challenge to each icon, as long as it provides the required inputs. At the present, those inputs are point estimates of the challenge values at each icon's location. Because a transport and dispersion model (such as HPAC) that provides more than simply a point estimate of the challenge values may not be available to all NATO nations, the IDA team cannot

[25] Calculating these parameters using a numerical implementation of the multi-dimensional Newton's method for root-finding is referenced in Nathan Platt, William Ross Kimball II, and Jeffry T. Urban, "The Use of Probabilistic Plume Predictions for the Consequence Assessment of Atmospheric Releases of Hazardous Materials," *International Journal of Environment and Pollution* 55, no. 1/2/3/4 (2014): 3–12.

recommend changing the *AMedP-7.5* SD2 methodology to require challenge distributions for each icon, as that would potentially preclude some nations from using the methodology.

C. *AMedP-7.5* SD2 Threshold Model Results Highly Scenario-Dependent

When comparing estimates of chemical casualties, the IDA team observed large variability in the effect of the change from the *AMedP-7.5* SD2 threshold model to the probit model. As shown in Figure 14, the degree to which the threshold model overestimates (or in one case underestimates) casualties relative to the probit model varies significantly by scenario. For GB, the threshold model predicts 11% more total casualties (mild or greater), 84% more total casualties (severe or greater), and 15% more fatalities than the probit model. Even greater variation is present for HD, for which the threshold model predicts 31% more total casualties (mild or greater), 55% fewer total casualties (severe or greater), and 204% more fatalities than the probit model.

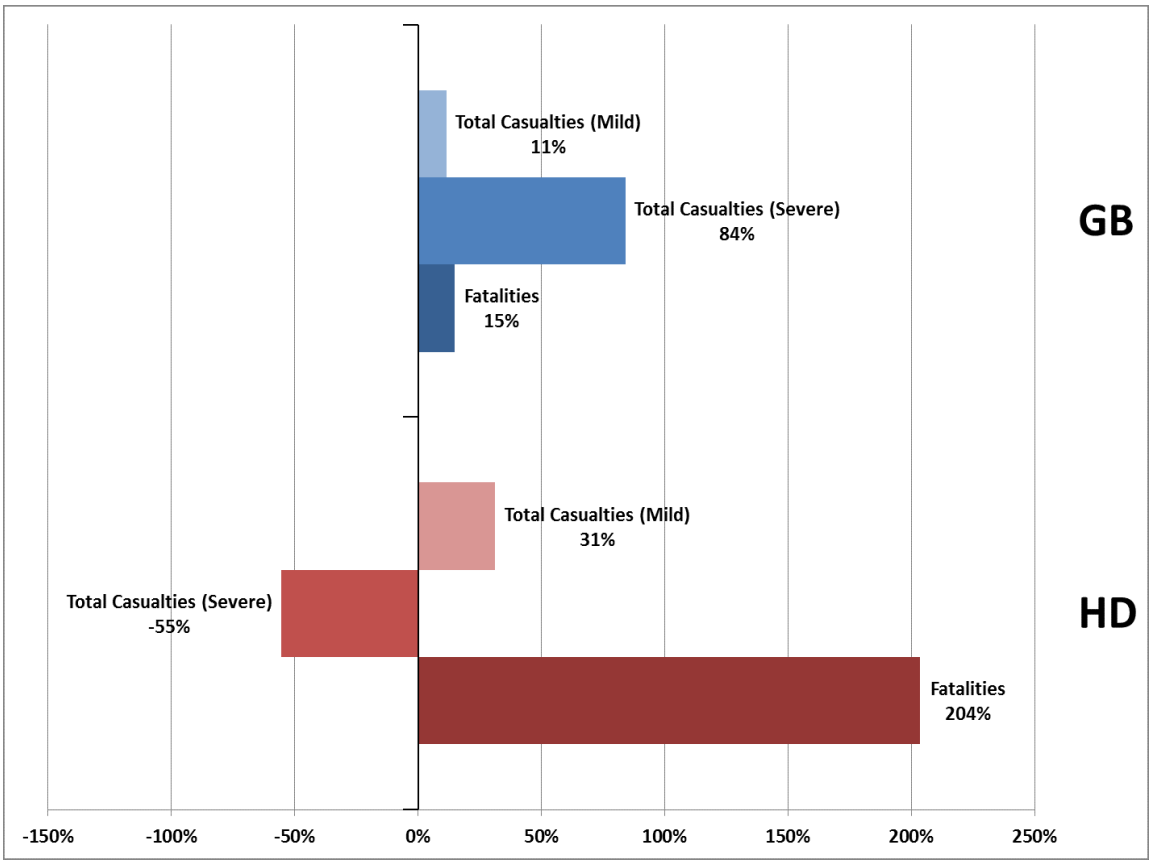

Figure 14. Percent Change from Probit Model to *AMedP-7.5* SD2 Threshold Model

The variation across agents and casualty types is a function of both the probit slope of the chemical agent and the distribution of Ct across the population at risk. This

dependency is illustrated by the four plots comparing the threshold model (shaded step function) and the probit model (solid colored curve) in Figure 15. In each plot, the probability of illness is shown as a function of Ct, and all parameters are the same across plots except the probit slope value, which varies from 3 to 12 probits/log(dose). For the probit model, the probability of illness increases gradually with larger values of Ct. In contrast, for the threshold model, the probability of illness changes abruptly from zero to one at the threshold value (the left edge of the shaded box), which was set to the same probability of illness value in all plots.

The area on each plot where the probit model curve has noticeably lower magnitude than the threshold function represents the Ct range for which the threshold model overestimates casualties. For instance, in the top left (blue) graph corresponding to a probit slope of 3 probits/log(dose), the threshold model overestimates casualties relative to the probit model for Ct values (of arbitrary units) between approximately 4 and 149. For values outside this range, the two models predict nearly the same results (namely, 0% or 100% probability of illness). As the probit slope increases, the Ct range over which the threshold model overestimates casualties decreases. For a probit slope value of 6 probits/log(dose), the corresponding Ct range in this example is approximately 10 to 61. For 9 and 12 probits/log(dose), the ranges are approximately 14 to 45 and 16 to 39, respectively.

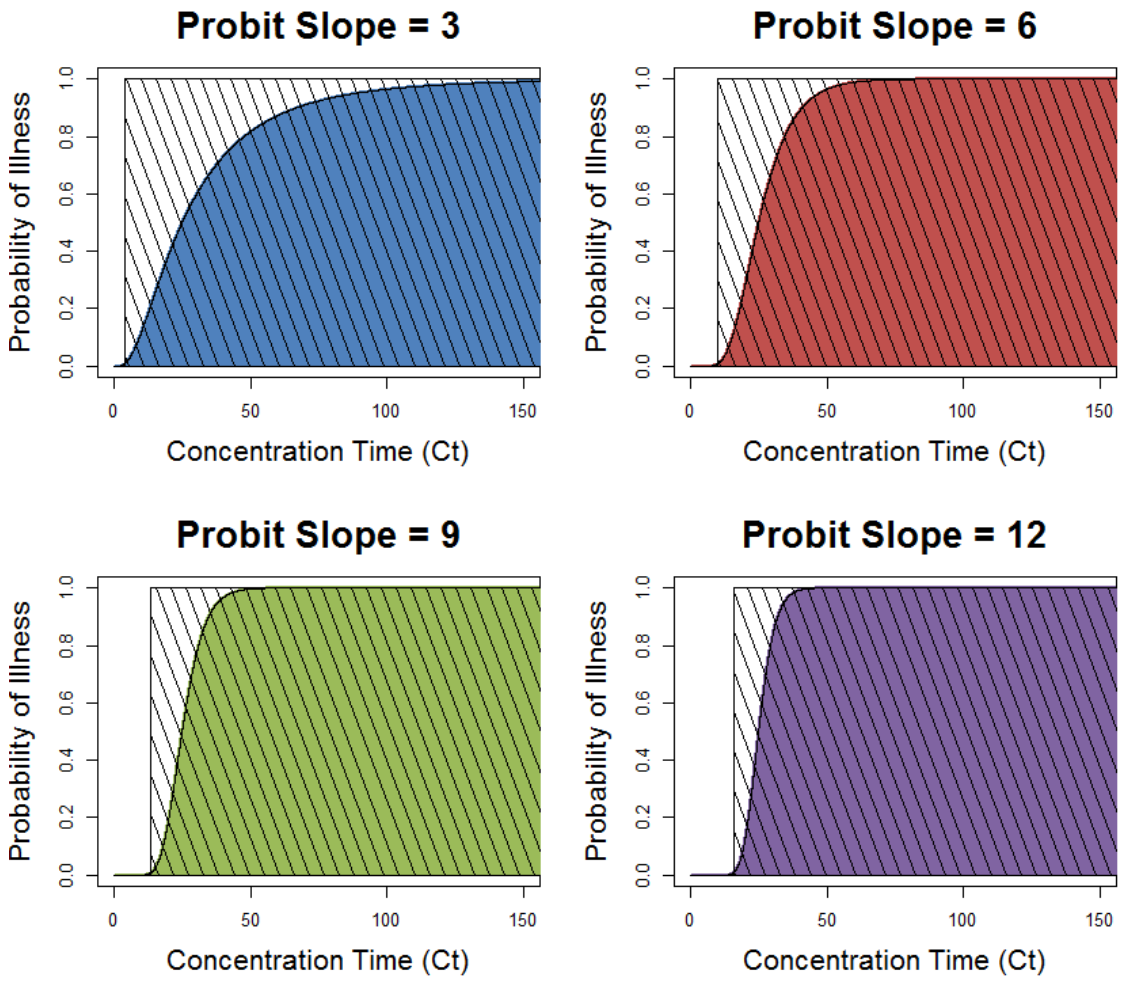

Figure 15. Effect of Probit Slope on Ct Range for Which Threshold Model and Probit Model Differ Significantly

For a given agent and effect (e.g., HD casualties (mild or greater)), the probit slope is fixed, so the observed difference between the threshold and probit model estimates depends upon the distribution of Ct values across the population at risk in the scenario. If a large proportion of exposed individuals were challenged with Ct values in the range where the two models differ, then the results would align worse than for a scenario in which most of the exposed individuals received challenges outside that range. In Figure 15, the threshold value was fixed at approximately the ECt_{01}, which corresponds to the *AMedP-7.5* SD2 HD mild casualty threshold of 4 mg-min/m^3 for a probit slope of 3 probits/log(dose). In this and most cases in the *AMedP-7.5* SD2 methodology, the threshold value is significantly below the ECt_{50}, which overwhelmingly results in higher casualty estimates than those the probit model predicts.

In the case of HD total casualties (severe or greater), however, the threshold model predicts fewer casualties than the probit model. This is due to the combination of a threshold value slightly below the ECt_{50} and a Ct distribution more heavily weighted where the probit model curve was greater than the threshold model in contrast to where the opposite was true. As illustrated in Figure 16, the threshold model predicts fewer casualties than the probit model below Ct values of 70 mg-min/m^3 and more casualties than the probit model above 70 mg-min/m^3. Therefore, the specific scenario analyzed must have included more individuals receiving challenges below this threshold level.

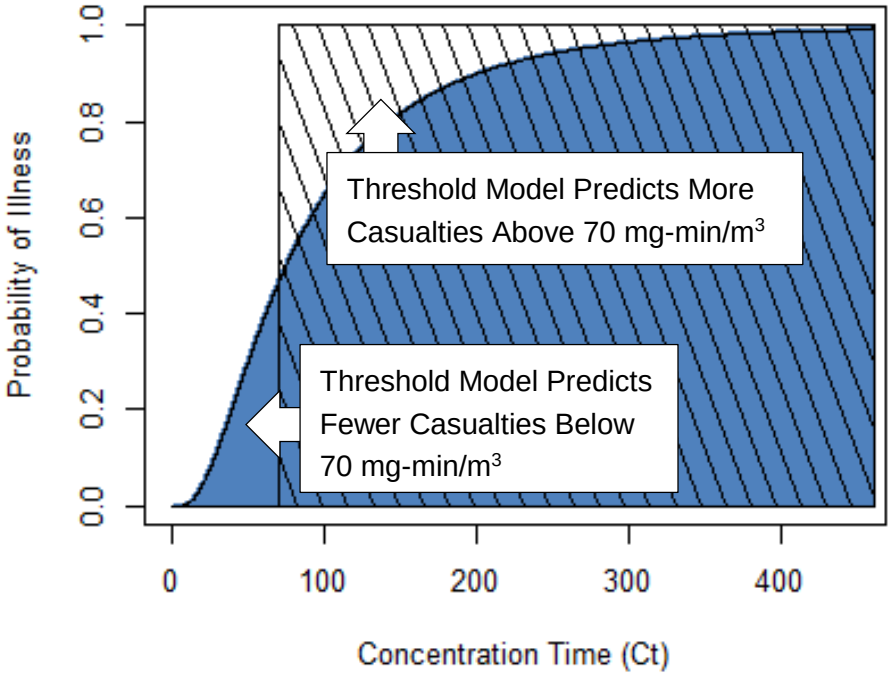

Figure 16. Comparison of Threshold Model and Probit Model for HD Severe Effects

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6. Conclusions and Recommended Change to the *AMedP-7.5* SD2 Methodology

The objective of this analysis was to identify potential improvements to the *AMedP-7.5* SD2 methodology. Chapter 3 described the known data and methodological differences between the two methodologies for which the IDA team controlled, namely different human response parameter values, different effects and mortality models, and different representations of the challenge values (i.e., full distribution versus mean value). Each data or methodological difference between the two methodologies that was highlighted in this analysis provided the IDA team the opportunity to assess its impact on the *AMedP-7.5* SD2 casualty estimates.

As observed in Chapter 4, controlling for the different human response parameter values in HPAC and the *AMedP-7.5* SD2 methodology had very little impact on the casualty estimates. The biological agent casualty estimates were only slightly affected, and with the exception of the GB toxic load exponent, the chemical agent human response parameters were already the same between the two methodologies. The IDA team does not recommend changing the *AMedP-7.5* SD2 human response parameter values, regardless of the impact of the change on the casualty estimates, because the IDA team has more confidence in the correctness of the *AMedP-7.5* SD2 values than the HPAC biological agent values, which were derived from *AMedP-8(B)*, a much earlier predecessor to the *AMedP-7.5* SD2 methodology.

In contrast to the human response parameter values, the different representations of challenge distribution had a very significant impact on the casualty estimates, especially for the biological agents. The ratio of casualties estimated using the mean challenge value to those estimated using the distribution of challenge values was nearly 15 for anthrax and approximately 20 for botulinum toxin. Despite the large effect on the casualty estimates, the IDA team does not recommend changing the *AMedP-7.5* SD2 methodology to require inputs in the form of challenge distributions for each icon, as it may preclude NATO nations without access to a transport and dispersion model capable of producing these inputs from using the *AMedP-7.5* SD2 methodology.

An assessment of the last methodological difference between the two methodologies, different effects and mortality models used to determine who became ill and died, led to a recommended improvement to the *AMedP-7.5* SD2 methodology. As observed in Chapter 4 and discussed in Chapter 5, the methodological difference between the use of the probit model in HPAC and the threshold model in the *AMedP-7.5* SD2 methodology for chemical agents has a major impact on the casualty estimates that varies

significantly by scenario. When the human response to a given challenge of agent varies within the exposed population, as is the case for the chemical agents in *AMedP-7.5* SD2, a probit model is often used to capture that variability in response. Because the probit models were fit directly to the raw data and the threshold model values were derived from the probit models, the probit models are generally better able to capture the underlying variability in human response than the threshold models. As no single threshold value consistently results in a good match between the threshold model and the probit model, the degree to which the *AMedP-7.5* SD2 methodology overestimates (or underestimates) chemical casualties is highly dependent upon the scenario analyzed. In order to avoid this unpredictable variation from the probit model estimate and to more consistently and accurately predict the number of casualties from chemical agents, the IDA team recommends changing the threshold model in the *AMedP-7.5* SD2 chemical agent methodology to a probit model.

Appendix A
HPAC Scenario Parameter Values

The IDA team used HPAC version 5.3.2 to generate chemical or biological agent clouds and the associated challenge information (Ct values) over the area of interest, which was used as an input to both methodologies. Except for the agent-specific human response parameters tested, HPAC scenario parameter values were the same for all agents and are those listed in Table A-1. All other HPAC parameters were set to the default values. The scenario location and release parameters were chosen solely to result in a significant number of casualties for analysis and do not reflect any known threat or U.S. military plans. The IDA team set the horizontal and vertical uncertainty to zero to reduce the variance of the predicted challenge values. The LandScan population associated with this location was replaced with a uniform population distribution, as described in Chapter 3, to further remove the dependency of the results on the chosen location.

Table A-1. HPAC Scenario Parameter Values for All Agents

Parameter	Value
Weather	Historical weather from October 15, 1990
Spatial domain	Southwest: 1.3 N, 103.7038 E Northeast: 1.4 N, 103.9083 E (Singapore)
Incident release point	1.318683 N, 103.8536 E
Munition and delivery system	Aerial spray
Mass of load	1200.0 kg
Altitude	50.0 m
Horizontal uncertainty	0.0 m
Vertical uncertainty	0.0 m

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Appendix B
Illustrations

Figures

Tables

Approved for public release; distribution is unlimited.

Appendix C
References

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Appendix D
Abbreviations

AMedP-7.5 SD2	*Allied Medical Publication 7.5* Study Draft 2
AMedP-8(C)	*Allied Medical Publication 8 (C)*
CBRN	Chemical, Biological, Radiological, and Nuclear
Ct	Concentration Time
DOD	Department of Defense
DTRA	Defense Threat Reduction Agency
GB	Sarin
HD	Distilled Mustard
HPAC	Hazard Prediction and Assessment Capability
IDA	Institute for Defense Analyses
III	Illness, Infection, and Injury
JEM	Joint Effects Model
kg	Kilogram
NATO	North Atlantic Treaty Organization
OTSG	Office of the Surgeon General (U.S. Army)
SCIPUFF	Second-order Closure Integrated Puff
U.S.	United States
V&V	Verification and Validation

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REPORT DOCUMENTATION PAGE	Form Approved OMB No. 0704-0188

1. REPORT DATE (DD-MM-YYYY)	2. REPORT TYPE	3. DATES COVERED (From – To)
XX-08-2015	Final	

4. TITLE AND SUBTITLE

2014 Review on the Extension of the *AMedP-8(C)* Methodology to New Agents, Materials, and Conditions

5a. CONTRACT NO.

HQ0034-14-D-0001

5b. GRANT NO.

5c. PROGRAM ELEMENT NO(S).

6. AUTHOR(S)

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5d. PROJECT NO.

5e. TASK NO.

CA-6-3079

5f. WORK UNIT NO.

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)

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8. PERFORMING ORGANIZATION REPORT NO.

IDA Document D-5226
Log: H 15-000873

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10. SPONSOR'S / MONITOR'S ACRONYM(S)

J-8/JRO CBRN and OTSG

11. SPONSOR'S / MONITOR'S REPORT NO(S).

12. DISTRIBUTION / AVAILABILITY STATEMENT

Approved for public release; distribution is unlimited.

13. SUPPLEMENTARY NOTES

14. ABSTRACT

This is the sixth in a series of annual reviews on the extension of the casualty estimation methodology originally described in Allied Medical Publication 8 (C): NATO Planning Guide for the Estimation of CBRN Casualties (AMedP-8(C)) and most recently published as AMedP-7.5 Study Draft 2 (SD2). The objective of this document, the 2014 review, is to identify additional improvements to the AMedP-7.5 SD2 casualty estimation methodology through a comparison of its casualty estimates for four agents (anthrax, botulinum toxin, sarin (GB), and distilled mustard (HD)) to the casualty estimates of Hazard Prediction and Assessment Capability (HPAC), a modeling and simulation tool developed by the Defense Threat Reduction Agency whose capabilities also include casualty estimation. By comparing every step of the two methodologies, the IDA team observed that the threshold dose-response model used in the AMedP-7.5 SD2 chemical agent methodology overestimates (or underestimates) chemical casualties in a way that is highly dependent on the scenario analyzed. In order to avoid this unpredictable variation from the probit model estimate and to more consistently and accurately predict the number of casualties from chemical agents, the IDA team recommends changing the threshold model in the AMedP-7.5 SD2 chemical agent methodology to a probit model.

15. SUBJECT TERMS

CBRN, AMedP-8, AMedP-7.5, Casualty Estimation, Human Response Model, HPAC, Probit Model

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NO. OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a. REPORT	b. ABSTRACT	c. THIS PAGE			Stewart, Harry M.
U	U	U	U	70	19b. TELEPHONE NUMBER (Include Area Code) harry.m.stewart4.mil@mail.mil

Standard Form 298 (Rev. 8/98)
Prescribed by ANSI Std. Z39.18

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