Human repeated blast lung injury assessments – A new framework for use behind Personal Protective Equipment (PPE)

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Abstract. Human injury criteria from blast are fundamentally multivariate depending on at least two physical input parameters. Exposure parameters for human injury risk used by Bowen [4] and numerous others are peak overpressure and positive-phase duration. With noisy field data or complex blast, determining an unambiguous positive-phase duration is difficult. Assessments of structural blast effects generally use positive-phase specific impulse and peak overpressure as more convenient damage metrics. Recently, Panzer et al [8] developed repeated blast lung injury assessments for lung injury and fatality using 2,349 live animal tests for peak overpressure and duration. These assessments are robust for repeated ideal blast exposures but are uncertain for complex blast. Existing complex blasts risk evaluations are sparse and are often inconsistent with well-established single blast risk. To provide a more robust framework for assessments behind armor, where substantial modifications of ideal or complex blast waveforms are expected, the risk functions of Panzer were recast in terms of positive-phase specific impulse and peak positive-phase overpressure. This ideal blast risk assessment is further extended to a repeated and complex blast framework suitable for use behind PPE. Identification is based on the observation that blast lung injuries are produced by both shock wave transmission through the torso and chest wall motion. Injurious portions of complex waveforms are isolated using chest wall acceleration thresholding of input pressure waveforms. This procedure removes the non injurious portions of the time nistory and restricts input to those impulsive waveforms that produce injurious chest wall motions. This approach assumes measurements are taken in a reflected conditions (e.g. on a simulated chest behind armor) but is extensible to incident or omnidirectional sensing. The resulting assessments are consistent with single ideal blast injury risk models of Bowen [4]and Bass [7] and repeated blast evaluations of Panzer et al [8] while extending use behind PPE.

1. INTRODUCTION

Blast-related injuries have increased as the consequence of increased explosive usage in modern warfare and terrorist actions (e.g. [1, 2]). The threat posed by improvised explosive devices (IEDs) is also increasing [3], highlighting the need for further research into the injury mechanisms, particularly those related to chest injuries behind armor. Blast waves are generated when explosives are detonated causing an outward increase in pressure. Ideal blast waves or Friedlander waves have been studied extensively in the past for their simplicity and straightforward experimental models (eg. [4]). Friedlander waves are produced in open areas allowing the blast wave to continuously propagate outward unimpeded. During an ideal wave a spike of increased pressure (peak overpressure - OVP) occurs at detonation and decays exponentially (positive phase) the decay passes ambient pressure creating a vacuum (negative phase) before returning to ambient pressure. Blast wave injury response is intrinsically multivariate. Exposure parameters for human injury risk used by Bowen et al. [4] and numerous others are peak OVP and positive-phase duration. With noisy field data or complex blast, determining an unambiguous positive-phase duration is difficult. Assessments of structural blast effects generally use positive-phase specific impulse and peak overpressure as more convenient damage metrics. However, it is often difficult in practice to unambiguously identify the positive phase duration, and blast risk functions for structures often use peak OVP and positive phase impulse to categorize risk (e.g. [5]). Generally, however, for ideal waves, any two of peak OVP, positive phase impulse, or positive phase duration can be used to characterize blast.

Complex blast waves, often generated within range of buildings, debris, and other structures that cause the blast wave to reflect and bounce while impinging on the torso, may be both highly directional and highly variable. This complicates analyses using most existing injury criteria. Behind personal protective equipment (PPE), even simple blast can become complex, and complex blast can become more complex. Estimates of injury risk become uncertain behind body armor and assessments of the local blast environment on the chest is not established. However, an approach based on surface pressure transducers, modified using paired accelerometers and reflected blast time histories, has some promise for assessing injury risk while wearing PPE (c.f. [6]).

To assess short duration ideal blast, Bass *et al*. conducted a study with 2,550 large animals for short duration blasts with less than 30 milliseconds positive phase duration [7]. The curves were compared to Bowen

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et al. [4] and matched the previously developed risk assessment for reflected ideal blast. However, the two studies did not show similar curves developed with the direction of the blast shock parallel and perpendicular to the blast. Recently, Panzer *et al* [8] extended the Bass risk assessment to repeated blast lung injury assessments for lung injury and fatality using 2,349 live animal tests. This study combines and extends short duration assessments of Bass *et al* and long duration assessments of Rafaels et al [9].

The risk model in Panzer *et al* was intended for use with ideal blast waves. Existing complex blast risk evaluations are sparse and are often inconsistent with well-established single blast risk. For example, the Axelsson/Yelverton thoracic response model assessment [10] is inconsistent with both the Bowen curves and the Panzer assessments for a single ideal blast, recommending higher injury tolerance values that are inconsistent with the existing data. Other models are often taken directly from automobile rate response models, well outside their range of applicability, and require instrumentation that is not compatible with many circumstances, including in vehicles and behind body armor. (c.f [11]).

The typical injury paradigm for the lung in automobile biomechanics (e.g. [12]) and for blast (e.g. [13]) is based on the hypothesis that displacement into the lung causes lung contusions. However, the compressive speed of sound in the lung is ~20 m/s. Since chest wall velocity and imputed shock wave propagation often exceeds 20 m/s in blast, there is a strong potential for compressive shock wave development in the lung [14] as shown in **Figure 1**. Existing thoracic response for blast lung injury models do not directly account for such compressive shocks (e.g. [10, 11]). There are several potential paths into the lung, in simple or complex scenarios either with or without body armor. In air, supersonic shocks generally propagate at velocities well over the 340 m/s quiescent speed of sound in air. When the air shock impinges on the torso, there are two alternatives, one is that the local air speed of sound is greater than 1500 m/s, the speed of sound in the flesh components of the thoracic wall. If so, a shock wave may be transmitted through the chest wall. If the speed of the incoming waves is subsonic in the chest wall, a piston-type excitation is transmitted across the torso. In both cases, the shock or high velocity piston loading may be propagated into the lung as a shock since the lung speed of sound is ~20 m/s for most normal physiological volume fractions of air and tissue. Generally, subsonic displacement requires a relatively large impulse over a long time and is not likely to produce lung injuries typical of blast. There are potential reflections and transmissions at each interface.

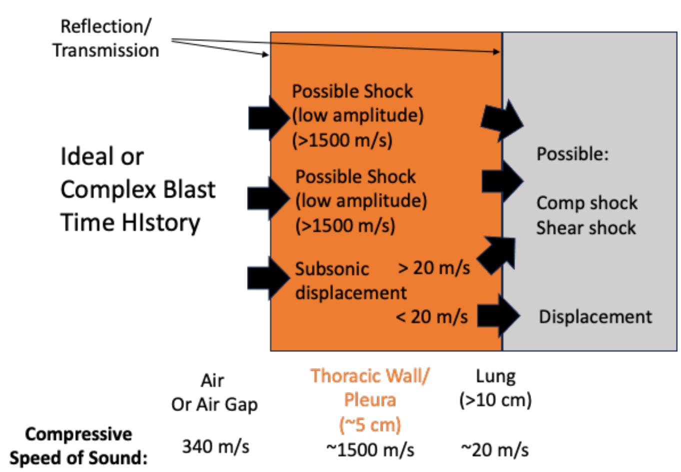

Figure 1. Routes of transmission of blast shock into the torso. Note: not drawn to scale.

Based on this paradigm of compressive shock damage in the lung, the purpose of this study is to establish a robust framework for assessments of primary blast injuries behind armor, where substantial modifications of ideal or complex blast waveforms are expected. We recast the risk functions of Panzer in terms of positive-phase specific impulse and peak positive-phase overpressure and use this analysis to framework for use in assessing injury risk based on analyses of compressive shock in the lung.

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2. METHODS

2.1 Multiple Blast Exposure Injury Criteria

The single and multiple blast injury assessment methodology of Panzer [8] was adapted to produce both single and multiple blast injury risk assessments in terms of positive phase reflected OVP and positive phase reflected impulse. All experiments in the dataset had peak OVP rise times of less than 0.3 ms, the typical rise time expected for simple blast injury [15, 16]. The study data input included 2,349 large animal experiments with a range of positive phase OVP and impulse. Substantial differences between small and large animals led to the restriction to species of > 15 kg body mass or high phylogenetic order, such as primates. For experiments where only one of incident pr reflected pressure measurements were reported, the Rankine-Hugoniot relations provided a conversion between incident and reflected pressure. CONWEP was used to assess peak overpressure and duration when only charge characteristics were reported. To convert duration to impulse, the Friedlander approximation was used between duration, OVP, and impulse. Since Bass *et al* [7] and Rafaels *et al* [9] found that animal orientation and the presence of a reflecting surface did not influence the model results, these were excluded.

Experiments with both single and repeated blasts were used. Repeated blasts included 2-100 exposures, with elapsed interval timing between 1-30 minutes. For this assessment, a dichotomous outcome variable was used. Fatality data were classified as dead, and injury data were classified as 'surviving'. Since data from 14 species of disparate sizes were used in this analysis, the scaling procedure of Bowen [4] was used. This is equivalent to the assumption that a single length scale characterizes all interspecies differences, and that mass density and effective modulus of elasticity was constant between species. Altitude correction was not used for short duration blasts given experimental evidence that this correction has negligible influence on injuries. Long duration scaling was used based on rodent experiments suggesting the scaling was required [4].

Fatality risk functions were used a logistic regression of a piecewise linear model with peak overpressure, scaled impulse, and number of exposures as parameters. This model incorporated multiple breakpoints in impulse that provide fidelity to a continuous nonlinear fit while maintaining a simple form. The location of these breakpoints were optimized for model fit to the data.

The basis for the model is the single exposure fatality risk function. The repeated injury risk model was solved at the same time as the repeated fatality model since multiple blast fatality data were sparse. In the original analysis [8], the effect of the repeated exposure was modeled as a declination function that scaled the risk. In the current study, the results are presented as a multiple injury risk model for integrated impulse over multiple exposures. Note that this model is strictly only valid for repeated ideal blasts without further analysis. The regression models had goodness of fit characteristics that replicated the original analysis for peak OVP and positive phase duration.

The optimizations were performed a using custom Matlab gradient descent solver (The Mathworks Inc., Natick, MA). The code had been previously verified by comparing the logistic regression results of a simplified dataset and model with those calculated in commercially available statistical software (JMP 8; SAS Institute Inc., Cary, NC)[8].

2.2 Mechanism of Wounding

The paradigm developed in this study assumes that compressive shocks are the principal source of blast wounding, particularly for short durations, but generally for all sharp rising peaks of any duration. For ideal shocks to form from local material acceleration (here, chest wall acceleration, a_{cw}) a distance/peak amplitude and time are required (Friedrichs, 1948). In n half-plane domain bounded an accelerating wall, the shock path (x) and shock pressure (p) are calculated as

$$x = x_\nu + c_0\left\{(t - t_\gamma) + \frac{3(\gamma+1)}{32\gamma}\frac{a_{CW}(t-t_\gamma)^2}{c_0} - \frac{45(\gamma+1)^2}{2048\gamma^2}\frac{a_{CW}^2(t-t_\gamma)^3}{{c_0}^2} + \cdots\right\} \quad (1)$$

$$p = p_0\left\{1 + \frac{3(\gamma+1)}{4}\frac{a_{CW}(t-t_\gamma)}{c_0} - \frac{63(\gamma+1)^3}{512\,\gamma}\frac{a_{CW}^2(t-t_\gamma)^2}{{c_0}^2} + \cdots\right\} \quad (2)$$

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where c_0 is the speed of sound in the material, g is the ratio of specific heats (1.4 for an adiabatic process), a_{cw} is the chest wall acceleration, p_0 is the ambient pressure, p is the shock pressure, x is the distance from the material wall, and x_g and t_g are the shock initiation distance and time from the material wall. These analyses can be combined with chest wall physical response data or models to assess the potential for lung shock injuries with accelerations that produce shocks in the lung parenchyma at distances shorter than the local depth of the lung.

Shock formation distance calculated using Equation (1) with typical parameters (c_0 = 20 m/s, p_0 = 101 kPa, g = 1.4) implies that shocks occur away from the pleural interface but well within typical porcine (or human) lungs (< 1 mm or less for higher severity blasts) based on typical human lung depths of ~180 mm or more. Previous experience in a pig lung model demonstrates this shock formation distance through alveolar damage [14]. Since pig lung size is approximately human lung size, this implies shock formation is occurring analytically at a distance that is plausible to explain the preliminary results. An important note is that this phenomenon does not scale with size/species, except with effects of size through the local applied acceleration and chest wall effective mass. Similarly, shock formation time calculated from Equation (2) using typical parameters (c_0 = 20 m/s, p_0 = 101 kPa, g = 1.4) shows that shock formation time is often 0.5 ms or less. This implies shock formation is occurring analytically at a time that shorter than that required to transit the model in large animals. Timing and wave reflections around the torso and back that are not considered in this analysis. The presence of strong reflections may make shock effects worse, but likely require strong enough shocks that the damage is fatal with or without strong reflections (i.e. the shock must still be a shock when reflected from opposite surfaces of the lung).

2.3 Complex Blast

To assess complex blasts using the hypotheses developed in this study, two aspects are important. The first is the single exposure blast assessment in terms of reflected impulse and reflected peak OVP as discussed above. The second is a relationship between applied thoracic impulse/pressure and chest wall motion, particularly local accelerations present in the shock model in Equations (1) and (2). For this aspect, we used the correlations of Boutillier et al [18] in the porcine thoracic model. These provide correlations between blast exposure (both reflected and incident impulse) at various levels and maximum chest wall acceleration, velocity for a range of severities. These severities are generally limited to lower impulse levels, but there are peak acceleration levels greater than 40,000 m/s^2. For example, for low impulse values, the correlation of max acceleration vs reflected impulse is

$$a_{max}=0.0644\ I_{ref}^2+78.225\ I_{ref}. \tag{3}$$

For complex blast, the ideal blast assessments must be adapted for the acceleration levels necessary to cause blast shock over the lung distance available without being separating discrete loading events during a complex blast time history. Based on the hypothesis in this study, this implies that the chest wall acceleration must be lower than the shock formation distance that would cause lung damage over the distance the wave must travel through the lung. Assessments are performed for both peak and mean acceleration levels from the Boutillier et al data. Assuming simple piston accelerative loading of the chest, mean acceleration is defined over a symmetric triangular rise and fall so that $a_{mean} = 0.41\ a_{max}$. Projected frontal area of the lungs is estimated using body surface area estimates assuming the torso 0.09% of total body area of 2.0 m^2 for adult males [cf. 19].

A limiting factor for shock development is the dimensions of the human lung. This is approximately 180 mm for human males [20]. Equation (1) is used to determine chest wall acceleration thresholds for alveolar injuries for 0% and 50% volume fractions damage. To assess threshold acceleration criteria and fatality criteria respectively. Effective chest wall mass, m_{eff}, is calculated from the Boutillier data for various combinations of peak OVP and positive phase impulse. So, for a given complex time history, a filter for chest wall acceleration values may be used to eliminate all non injurious thoracic wall response. Using the analyses the anthropometric parameters of adult humans, and the effective mass of the chest at various peak OVPs, the risk function for for multiple exposures in terms of peak OVP and positive phase impulse, may be used to assess the filtered complex blast including behind PPE.

3. RESULTS

The injury risk function for 5%, 50% and 95% risk of fatality in terms of peak reflected OVP and positive phase reflected impulse for single ideal blast exposure is shown in **Figure 2**. This reoptimization of the original Panzer model uses two breakpoints to obtain fidelity with the blast data at intermediate reflected impulse values between

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1,000 kPa ms and 10,000 kPa ms. The high impulse limit of the risk function is essentially flat with little dependence on peak reflected OVP suggesting that these blast injuries are not sensitive to time dependence represented by positive phase impulse. At shorter reflected impulse values, the risk functions for peak OVP have a step slope to high OVP implying the smaller the reflected impulse, the larger the peak reflected OVP risk tolerance at each risk level displayed. The model displays good model fit with error assessments commensurate with the original model analyses.

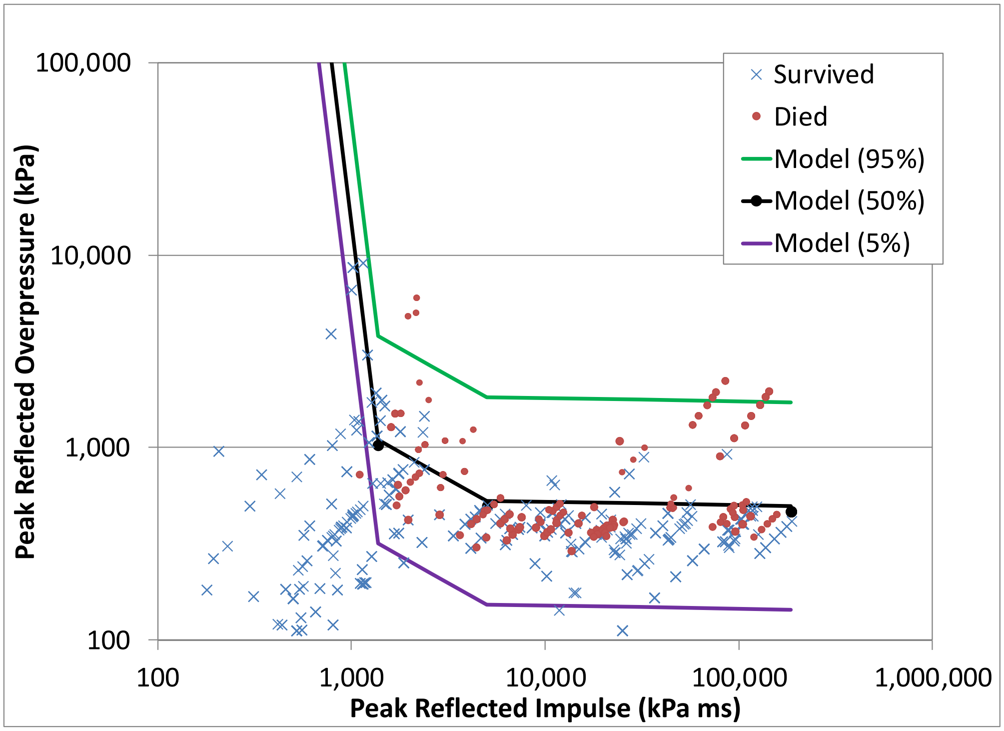

Figure 2. Single ideal blast exposure injury risk for 5%, 50% and 95% risk of fatality in terms of positive peak OVP and peak reflected impulse using the survival and fatality data in the current database. The survival and fatality experimental data for this model are shown in the plot.

The injury risk function for 5%, 50% and 95% risk of fatality in terms of peak reflected OVP and positive phase reflected impulse for multiple ideal blast exposure is shown in **Figure 3**. This reoptimization of the original Panzer model uses two breakpoints to obtain fidelity with the blast data at intermediate reflected impulse values between 1,000 kPa ms and 10,000 kPa ms. The high impulse limit of the risk function is essentially flat with little dependence on peak reflected OVP suggesting that these blast injuries are not sensitive to time dependence represented by positive phase impulse. At shorter reflected impulse values, the risk functions for peak OVP have a step slope to high OVP implying the smaller the reflected impulse, the larger the peak reflected OVP risk tolerance at each risk level displayed. This model includes the effect of the declination function from the original Panzer analyses [8] to provide analyses of complex blast through a filtering function that eliminates non injurious dynamic content. The model displays good model fit with error assessments commensurate with the original model analyses.

The shock formation distance using peak acceleration based on chest response porcine data of Boutillier [18], adult male anthropometric values, and the inversion of Equation 1 is shown in **Figure 4**. Using the 50% male lung depth as the threshold for fatality, the low risk (5%) value approach shock formation at the lung midpoint at high impulse. This suggests that the long impulse region may be conservative in this analysis.

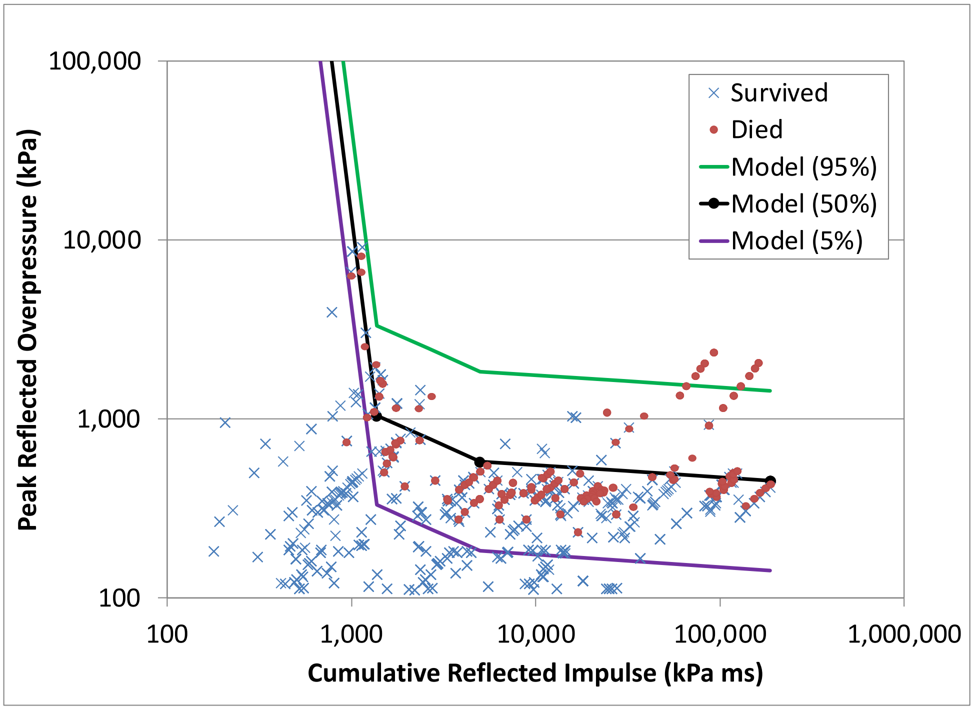

Figure 3. Multiple ideal blast exposure injury risk for 5%, 50% and 95% risk of fatality in terms of peak reflected OVP and cumulative reflected impulse using the survival and fatality data in the current database. The survival and fatality experimental data for this model are shown in the plot.

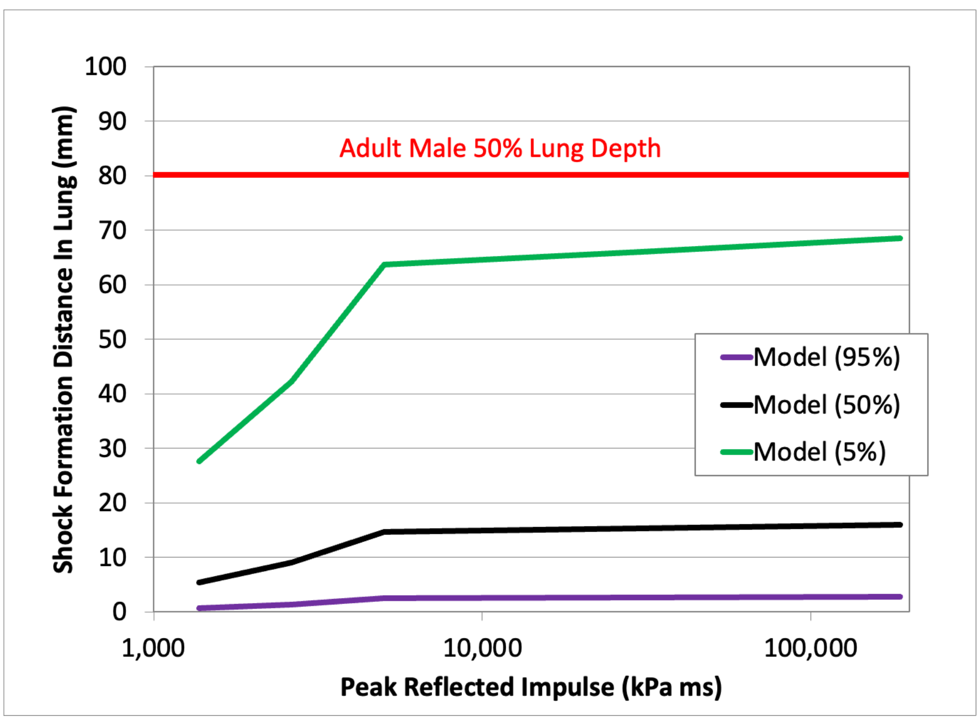

Figure 4. Internal lung shock wave development distance against peak reflected impulse. Accelerations are based on chest response porcine data of Boutillier (2017) and mid-size male anthropometric characteristics. Reference adult male lung depth is derived from Kramer et al [20].

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Inverting Equation (1) for chest wall acceleration, this implies that chest wall acceleration values below 1850 m/s^2 will not cause lung shocks over the anterior/posterior distance of the lung. So, pressure time histories that do not produce chest wall accelerations of this magnitude will not result in shock injuries to the alveoli. This value provides a threshold for lung damage. Assuming 50% lung damage is not immediately fatal, acceleration values below 3,700 m/s^2 will not cause fatalities. There is strong evidence for this association of large lung damage with survival in experience in Israeli bus bombings [21] where up to 70% lung damage was survivable under some circumstances. By extension, for complex blast, only the accelerative loading that is > 3700 m/s^2 chest wall acceleration can cause fatalities, with wounding possible down to ~1850 m/s^2. Below the 3700 m/s^2 threshold, the limiting probability for fatalities is approximately 2.5% for long duration/low pressure, suggesting that values below 0.05% are likely conservative.

The thoracic effective accelerating mass can be estimated using the response data of Boutillier as shown in **Figure 5** in terms of peak reflected OVP. Note that the effective mass takes three ranges depending on injury risk while align with approximately logarithmic dependence on peak reflected OVP so that

$$M_{eff} = -0.448\ ln(P_{refl}) + 0.433 \quad (4)$$

For lower applied impulse, the thoracic model response has higher effective mass, and for higher impulse, the thoracic model response has lower effective mass.

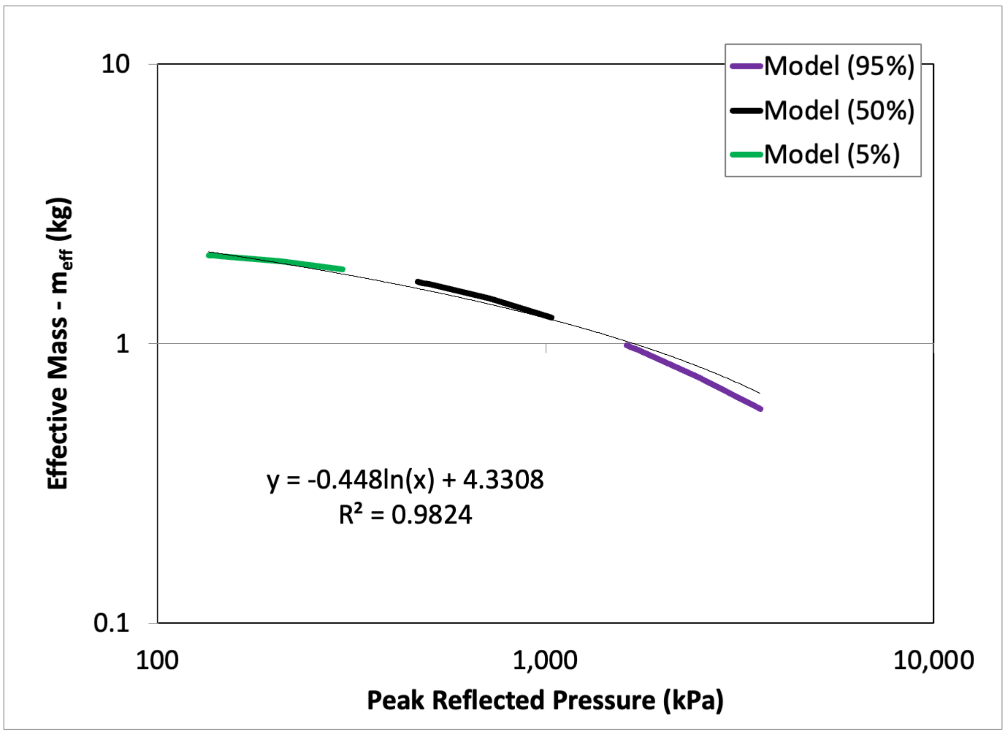

Figure 5. Effective mass of the chest wall vs peak reflected OVP based on the response data of Boutillier [18]

So, to calculate risk for complex blast fatalities, assuming a 50% shock depth to fatality and the effective mass shown in **Figure 5** with anthropometric parameters for a mid-size male we take a filter and calculate approach

1. Filter nonfatal exposures for the complex pressure/impulse time history based on threshold acceleration values derived from the thoracic response data of Boutillier [18], assuming arthrometric parameters for the appropriate human demographic such that

$$a_{threshold}(t) = P_{ref}(t)\ A_{lung}/m_{eff} \quad (5)$$

This results in a modified complex time history with non injurious components removed.

2. Use the risk assessments in **Figure 3** for cumulative impulse, $I_{cumulative}$, to assess injury risk against peak reflected OVP.

This combined assessment produces a complex blast injury assessment consistent with the single blast injury risk functions in **Figure 2** and **Figure 3** consistent with shock formation distance shown in **Figure 4**.

4. CONCLUSIONS

This study provides a generalizable paradigm for complex blast with PPE based on chest wall acceleration and velocity that predicts alveolar disruption characteristic of blast, not rib fractures and associated lung injuries associated with automobile crashes and other blunt trauma. Based on a filtering technique to remove non injurious components, it is extensible to different human anthropometries and human nonfatal injuries, as opposed to fatalities. This assessment is consistent with the available single and multiple exposure ideal blast data, in contrast with previous chest wall motion models intended for use with complex blast [10, 11]. In general, the injury risk function is insensitive to the high impulse, low overpressure regime. This is typical of nuclear blast exposures where tertiary effects become dominant.

Beyond this paradigm, we have provided injury risk assessments for multiple ideal blasts in terms of positive phase reflected pressure and reflected impulse. These are an extension of the multiple blast risk assessments of Panzer [8]. There are distinct advantages to using overpressure/impulse relations to assess blast lung risk, especially for use under PPE. Generally, reflected pressure measurements are available under PPE, but care must be taken to calibrate and account for gauge acceleration on nonrigid surfaces, and thoracic variation of input in a complex blast field. This will generally require multiple sensing locations for effective use of this technique.

In using this work in other contexts, care must be taken in interpreting response data, especially thoracic response data obtained by simple lumped mass approaches (e.g Axelsson [10]) or finite element models that have not been validated for larger accelerations. Chest wall velocity injury regimes from the Axelsson model predict blast lung injuries starting at 3-4 m/s chest wall velocity. These predictions stand in contrast with typical displacement-based automobile injury criteria for thoracic models that show no evidence of blast lung injuries [12].

The principal limitation of this study is that blast exposure is assumed to be directed towards the torso, and pressure/impulse exposure orthogonal to the local chest direction are not an important source of chest wall motion and injury. From the data, however, the differences between parallel and reflected are large, and tangential blast exposure is likely a small contributor to chest wall motion, especially behind PPE. Other limitations include limited thoracic response data in the low pressure/high impulse regime. In addition, the Boutillier response estimates are based on with local length of 9.5 mm and a density of ~9000 kg/m^3 [18]. Since the typical rib mass density is ~700 kg/m^3, this disparity may have influence on the local measurement of acceleration, reducing computed accelerations and velocities.

Future efforts will include expansion to additional anthropometries, and reduction in the approximations that lead from pressure to thoracic time histories. Other improvements include the implementation of a finite volume blast shock model for lung that can use arbitrary reflected OVP time histories to validate and extend the experimental thoracic model.

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