# Computational Modeling Related to Blast Exposure

## Computer Modeling of Blast-related Brain Injuries

This project is designed to determine the cause of mild traumatic brain injury (mTBI) due to blast overpressure (BOP) and, if possible, the human tolerance to BOP. The research, conducted at Wayne State University (Detroit, Michigan) consists of an experimental portion in which 12 porcine and six unembalmed post-mortem human surrogates were exposed to blast. The experimental effort is supplemented by a computer modeling section to extend the results of the tests to blast scenarios not easily obtained experimentally. During FY17 the study was completed and a detailed histological study of blast-exposed porcine brain is underway, with plans to publish the results once completed.

Results to date from this study show:

1. BOP at and above 300 kilopascals (peak) can cause mTBI in porcine, based on histological studies of the frontal brain. There were β-amyloid precursor protein (β-APP) reactive zones in both the gray and white matter and proliferation of astrocytes and microglia (Figure 1).
2. Quantitative electroencephalographs (qEEG) show evidence of brain injury almost immediately after the brain was exposed to BOP.
3. Injury occurs in both the gray and white matter of the porcine brain and is likely due to the dynamic pressure pulse because the computed strains in brain were less than three percent while the estimated injury level due to strain is about 20 percent.
4. Computed maximum brain strains due to blast exposure, based on both the porcine and the human brain models, were found to be on the order of five percent. This level of strain is well below the injury level, based on what published reports about brain strain is blunt impact (*Bain and Meaney 2000, Morrison et al. 2003*).
5. Axonal injury in blast-exposed porcine brain that is only subjected to very low strains requires reconsideration of the hypothesis that axonal stretch is the sole cause of diffuse axonal injury. Additional studies are recommended to determine the effect of pressure on brain cells, such as exposing isolated brain cells to blast (*Jean et al. 2014, Zhu et al. 2015*).
6. Open field blasts have a much shorter duration than those in a shock tube and is thus a more realistic simulation of blast. Longer duration blasts can cause more severe injuries to the brain and the results of shock tube studies are open to question (*Bauman et al. 2009, Vandevord et al. 2012*).

The researchers also developed and partially validated computer models for both the porcine and human brain. The validation was based on comparing the measured and computed pressures in various regions of the brain. Such models can be used to predict the response of the brain to a variety of blast scenarios. Based on these results, the following conclusions may be drawn:

1. There is firm evidence that open-field blasts can cause traumatic brain injury to the brains of anesthetized swine for peak pressures of 300 kilopascal or higher.
2. Both the gray and white matter are injured in the experiments carried out thus far. Blast overpressure appears to be the culprit as the computed brains strains are far below levels that can cause axonal injury.
3. More work is needed to establish a human level of tolerance to blast overpressure.
4. It is necessary to revisit the injury data from animals exposed to blasts in a shock tube.

The impact on the Service member can be anticipated as follows:

1. Protection from blast overpressure will require head coverings that can attenuate the magnitude of the blast wave.
2. The human head model needs to be run to study the effect of covering various portions of the head and to determine if the entire head needs to be covered by a shock deflecting and/or absorbing material.
3. It may be worth testing the use of Kevlar in helmets for head protection because it appears to protect the lung of dismounted soldiers exposed to blast overpressure.

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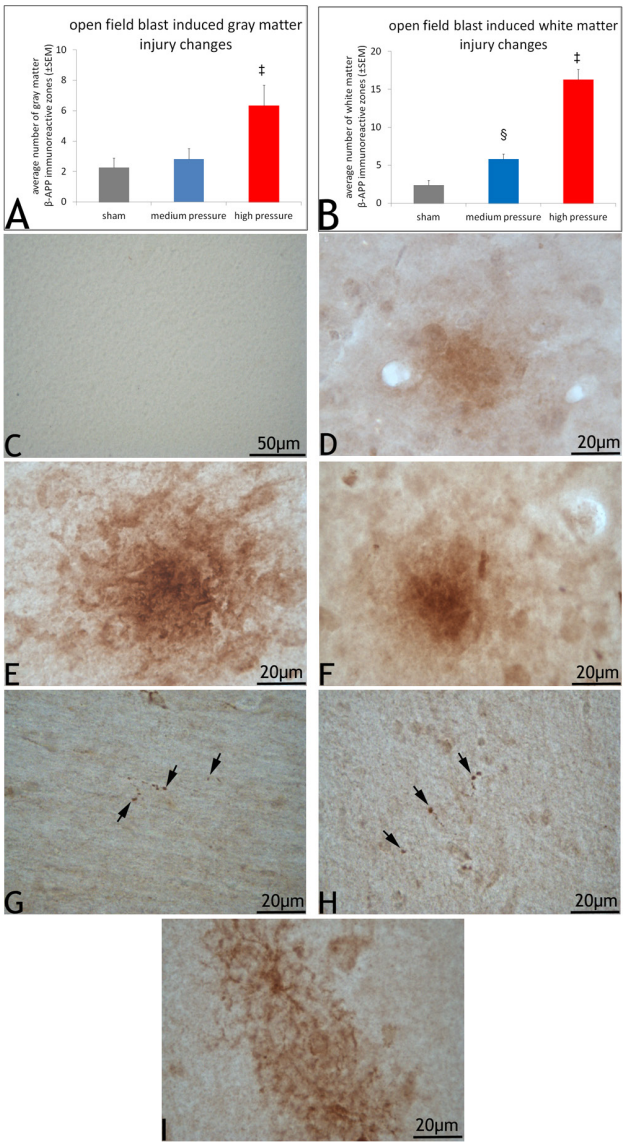


**FIGURE 1:** (A) shows average number (± standard error of the mean) of β-APP immunoreactive zones in the gray matter of blast and sham swine sections. The number of β-APP immunoreactive zones in the gray matter of high blast overpressure sections were significantly high compared to those in the sham and medium blast overpressure ($p<0.05$, LSD; One-way Analysis of Variance [ANOVA]). (B) shows average number (± standard error of the mean) of β-APP immunoreactive zones in the white matter of high blast overpressure sections. The number of β-APP immunoreactive zones in the white matter was significantly high compared to those in sham and medium blast overpressure ($p<0.05$, LSD; One-way ANOVA). Furthermore, the extent of white matter immunoreactive zones was also significantly high in the medium overpressure sections, compared to sham ($p<0.05$, LSD; One-way ANOVA. (C) shows a control section stained without the primary antibody, showing no apparent immunoreaction. (D) shows a sham section showing a less intense gray matter β-APP immunoreactive region. (E and F) show an intensely stained gray matter immune-reactive zone from representative high and medium blast overpressure swine sections. (G and H) show representative β-APP reactive reaction retraction balls in the cortical white matter tracts. It shows an immunoreactive zone with stellate-like profiles in the white matter tracts of a swine subjected to high blast overpressure. (I) shows an immunoreactive zone with stellate-like profiles in the white matter tracts of a swine subjected to high blast overpressure. (Figure used with permission from the authors)