## ABSTRACT

Objectives: Blast-induced traumatic brain injury (bTBI) has risen to a new level of importance and is recognized to be a major cause of injuries to the brain. A simplified, free field blast-injury model would facilitate studies to correlate biological outcomes with blast-injury mechanics to generate novel tolerance criteria for bTBI.

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An in vitro model of blast-induced traumatic brain injury

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Results: This in vitro blast-injury model was capable of producing 175 kPa overspressures, which elicited diffuse cell death in OHSC that increased over 24 hours following blast. Control cultures experienced minimal cell death. Hypothermia was significantly neuroprotective and prevented cell death in cultures exposed to 175 kPa or 325 kPa overpressures.

Conclusions: Our in vitro blast-injury model recapitulates the translation of a shock wave in air, such as that produced by an explosive device, into a pressure wave similar to that within the skull-brain complex in vivo. Our results suggest that OHSC are vulnerable to and directly affected by blast-injury. OHSC exposed to blast at 25 °C were protected from the injury with minimal resultant cell death. To better prevent and treat bTBI, both the initiating biomechanics and the ensuing pathobiology must be understood in greater detail. Future studies will elucidate the tolerance of OHSC to various parameters of blast-injury as well as the mechanisms influential in this blast-induced cell death response. A well characterized, in vitro model of bTBI, in conjunction with animal models, will be a powerful tool in developing strategies to mitigate the risks of bTBI.
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