Polydopamine based perfluorocarbon nanodroplets for medical ultrasound applications

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Polymer coatings are of central importance for a variety of biomedical applications. In recent years, polydopamine, a nature inspired polymer based on mussels has attracted considerable research interests in materials science and technology due to its highly attractive properties. In this presentation, I will present some of our recent efforts in engineering ultrasound responsive polydopamine nanodroplets based on the acoustic vaporization mechanism for biomedical applications. The physical properties of our polydopamine nanodroplets have been characterized and upon exposure to ultrasound, polydopamine based nanodroplets were able to undergo phase transition to generate microbubbles. These and other results will also be presented.

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Cold environment exacerbates brain pathology and oxidative stress following traumatic brain injuries. Potential therapeutic effects of nanowired antioxidant H-290/51

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Military personnel are the most vulnerable to TBI either during peace keeping or combat operations at extreme hot and cold environments. Although, some reports suggest that hyperthermia following TBI is harmful, studies conducted on the effects of cold environment on the pathophysiological outcomes of TBI are still lacking. We examined the effects of cold environment on TBI in our rat model with regard to generation of oxidative stress and brain pathophysiology. In addition, an effect of a potent antioxidant compound H-290/51 with or without TiO2 nanowired drug delivery on the pathophysiology of TBI in cold environment was also evaluated. Focal TBI was inflicted under Equithesin anesthesia in Wistar Male rats over the right parietal cortex by making an incision of 2 mm deep and 4 mm long after opening of the skull bone (ca. 4 mm diameter, area 12.56 mm2). The animals were allowed to survive 48 h after TBI. Animals were exposed either at 5°C for 3 h daily for 5 weeks before injury. The control groups were maintained at normal room temperature (21±1°C). In these animals some of the key oxidative stress parameters e.g., Leucigenin (LCG), Luminol (LUM), Malondialdehyde (MDA) and Glutathione (GTH) in the brain along with blood-brain barrier (BBB) breakdown, brain edema formation and neuronal injuries were measured. TBI in animals subjected to cold environments exhibited about 80 to 190 % increase in LCG, LUM and MDA and 220 % decrease in GTH in the brain as compared to rats subjected to TBI at room temperature. The magnitude and intensity of BBB breakdown to radioiodine and Evans blue albumin, edema formation and neuronal injuries were also exacerbated in TBI group in cold environment by 120 to 280 % from the injured group at room temperature. Nanowired delivery of H-290/51 (50 mg/kg) 6 to 8 h after TBI in cold group was able to significantly thwart brain pathology and oxidative stress whereas normal delivery of H-290/51 failed to achieve any reduction in these animals after TBI. These observations demonstrate that cold aggravates the pathophysiology of TBI and this could be partially due to an enhanced production of oxidative stress in cold environment, not reported earlier.

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