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

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The possibility that traumatic brain injury (TBI) occurring in a cold environment exacerbates brain pathology and oxidative stress was examined in our rat model. TBI was inflicted by making a longitudinal incision into the right parietal cerebral cortex (2 mm deep and 4 mm long) in coldacclimatized rats (5 °C for 3 h daily for 5 weeks) or animals at room temperature under Equithesin anesthesia. TBI in coldexposed rats exhibited pronounced increase in brain lucigenin (LCG), luminol (LUM), and malondialdehyde (MDA) and marked pronounced decrease in glutathione (GTH) as compared to identical 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 cold-exposed rats after injury as compared to room temperature. Nanowired delivery of H-290/51 (50 mg/kg) 6 and 8 h after injury in cold-exposed group significantly thwarted brain pathology and oxidative stress whereas normal delivery of H-290/51 was neuroprotective after TBI at room temperature only. These observations are the first to demonstrate that (i) cold aggravates the pathophysiology of TBI possibly due to an enhanced production of oxidative stress, (ii) and in such conditions, nanodelivery of antioxidant compound has superior neuroprotective effects, not reported earlier.

Blood-brain barrier

Brain edema

Cold environment

Glutathione

H-290/51

Lucigenin

Luminol

Malondialdehyde

Nanodelivery

Neuronal damage

Oxidative stress

Traumatic brain injury (TBI)

antioxidant

glutathione

H 290 51

h 290 51

lucigenin

luminol

malonaldehyde

nanowire

titanium dioxide

unclassified drug

antioxidant

H290-51

indole derivative

nanowire

neuroprotective agent

animal experiment

animal model

animal tissue

Article

blood brain barrier

brain region

cold acclimatization

controlled study

disease exacerbation

dose response

drug delivery system

drug effect

edema

enzyme activity

enzyme blood level

male

neuropathology

neuroprotection

nonhuman

oxidative stress

parietal lobe

pathophysiology

rat

room temperature

traumatic brain injury

animal

brain

cold

metabolism

oxidative stress

pathology

physiology

Sprague Dawley rat

traumatic brain injury

treatment outcome

Animals

Antioxidants

Brain

Brain Injuries, Traumatic

Cold Temperature

Indoles

Male

Nanowires

Neuroprotective Agents

Oxidative Stress

Rats

Rats, Sprague-Dawley

Treatment Outcome