Capsaicin impact on sensory neurons located in the nodose ganglion of a rat.

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The nodose ganglion (NG) is the inferior ganglion of the vagus nerve that transmits sensory information from the visceral organs to the hindbrain. It is essential in relaying information including distention of the heart, lungs, and stomach, elevation in blood pressure, and changes in blood oxygenation. The NG contains two types of neurons, A-type and C-type neurons. A-type neurons are myelinated, capsaicin resistant while C-type are unmyelinated, capsaicin sensitive neurons that contain capsaicin receptors called VR1 or TRPV1. Additionally in NG there are glial cells, called neuroglia or simply glia, which are non-neuronal that provide support and nutrition for neurons, form myelin, and maintain homeostasis. They are involved in signal transmission within the nervous system. This study determined if neuronal degeneration after capsaicin treatment is followed by compensatory replacement of C-type neurons through neurogenesis. Capsaicin, a neurotoxin, is the active component of chili peppers, producing a burning sensation in any tissue it comes into contact with. Through immunohistochemical and immunofluorescent techniques, we examined NG for incorporation of bromodeoxyuridine (BrdU), an analogue of thymidine, into newly synthesized DNA of neuronal nuclei during cell proliferation. Our data showed that VR1 staining in capsaicin treated rats decreased and there was BrdU incorporation after capsaicin treatment while none in vehicle treated rats. The results indicate that VR1 containing neurons are being destroyed due to the capsaicin treatment which is then followed by proliferation of new neuronal cells with BrdU incorporation in new replicated DNA. In conclusion, we can state that capsaicin induced neuronal destruction of primary sensory neurons of NG in adult rats is followed by neurogenesis.