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Organization and repair of the trigeminal system in the lamprey using fluorescent double labeling

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In the CNS, many sensory and motor systems are topologically organized relative to various body structures. For example, in humans, sensory inputs from the lower, middle, and upper parts of the body are received by the anterior, middle, and posterior somatosensory cortex, respectively. Following injury in the CNS of higher vertebrates, such as birds and mammals, there is very little regeneration and recovery of function. In contrast, in lower vertebrates, including lamprey, fish, and certain amphibians, substantial axonal regeneration occurs following CNS injuries, and there is virtually complete recovery of functions. However, it is not known whether axonal regeneration restores the topological organization that may have existed prior to injury. The trigeminal system is responsible for transmitting sensory and motor information from the head via axons in the trigeminal cranial nerve. In the lamprey following injury of the trigeminal nerve, sensory and motor axons regenerate. The purpose of the present study was to determine whether the trigeminal system of normal lamprey is topologically organized and whether regeneration of axons in injured trigeminal nerve restores this organization. In normal larval lamprey, Alexa 488 dextran amine (Alexa) and Texas red dextran amine (TRDA), two different anatomical fluorescent tracers, were applied to the medial and lateral parts of the head, respectively. This resulted in clear labeling of the medial and lateral parts of the trigeminal system in the brain with Alexa and TRDA, respectively. Thus, in normal lamprey, the trigeminal system appears to be topologically organized. If following injury of the trigeminal nerve, axonal regeneration restores this topological organization, this will indicate that regeneration in the lamprey is relatively precise and possibly controlled by specific guidance factors. Identification of these guidance factors would greatly improve our understanding of the mechanisms that regulate axonal regeneration following CNS injury in vertebrate animals, including humans.