Continued

Fig. 5C

pERK
Western Blot

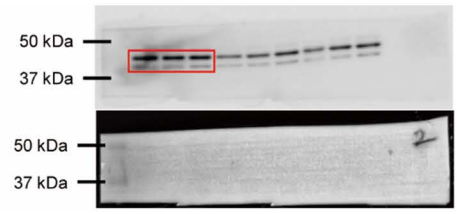


Photo image

ERK
Western Blot

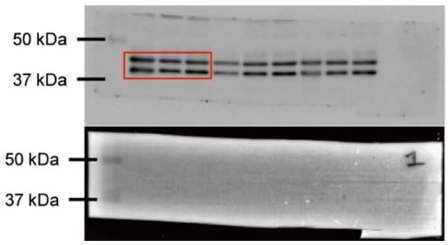


Photo image

β-actin
Western Blot

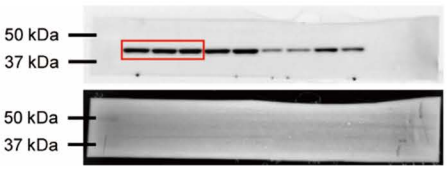


Photo image

Fig. 8A

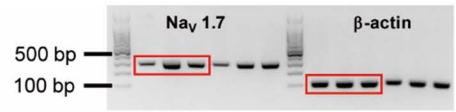


Fig. 8C

Na$_V$ 1.7
Western Blot

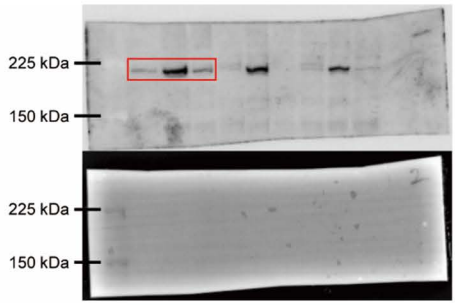


Photo image

β-actin
Western Blot

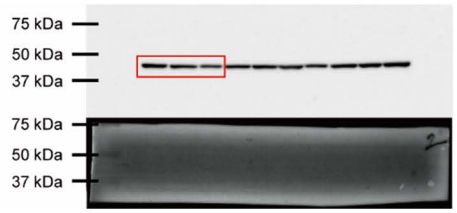
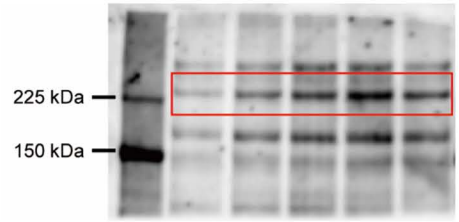


Photo image

Supplementary Fig. 1. Expanded views of western blot and PCR findings including weight marker in Figs 1, 4, 5, and 8. The red boxes highlight the areas used in the figures. Note that in Fig. 5A, as we did not save the photograph of membrane of these series, we provide the contrast-adjusted gel images, in which the marker bands appeared.

Fig. 1

Na_V 1.7
Western Blot



β-actin
Western Blot

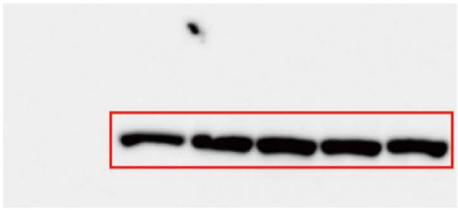
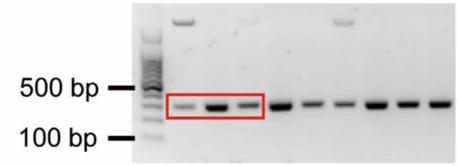


Fig. 4A

Na_V 1.7
PCR



β-actin
PCR

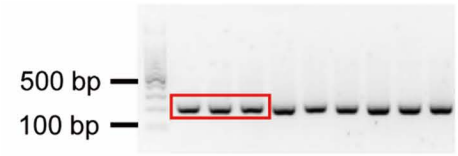


Fig. 4C

Na_V 1.7
Western Blot

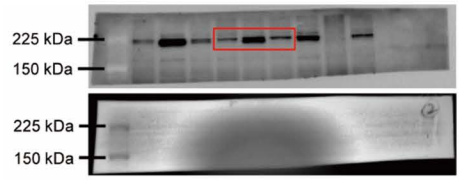


Photo image

β-actin
Western Blot

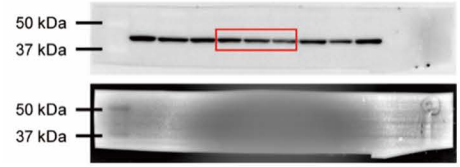
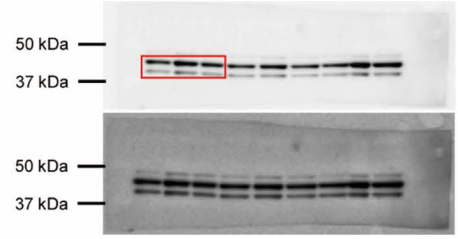


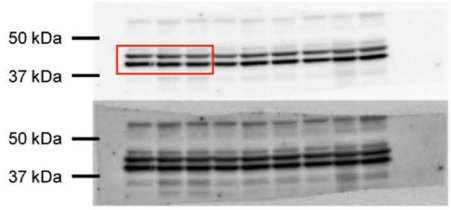
Photo image

Fig. 5A

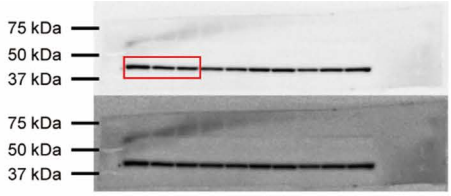
pERK
Western Blot

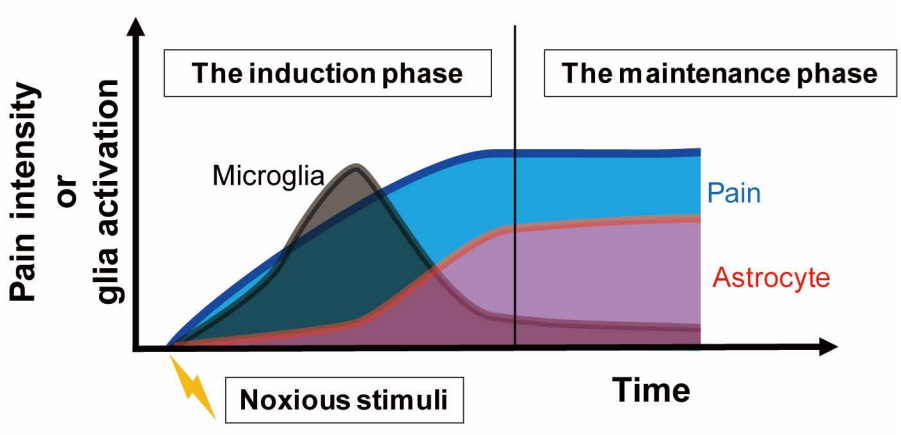


ERK
Western Blot



β-actin
Western Blot





Supplementary Fig. 2. Schematic representation of the phase change that occurs during neuropathic pain evolution. Noxious stimuli, such as RTX exposure, trigger microglial and astrocyte activation. Microglial activation gradually declines; however, astrocyte activation continues in the presence of noxious stimuli. Hence, microglia and astrocytes contribute to the induction and maintenance phases of neuropathic pain evolution, respectively.