Spinal cord dorsal horn inhibition is critical to the processing of sensory inputs, and its impairment leads to mechanical allodynia. How this decreased inhibition occurs and whether its restoration alleviates allodynic pain is poorly understood. Here, we show that the calcium binding protein parvalbumin (PV) controls the activity of inhibitory PV-expressing neurons (PVNs) by enabling them to sustain high-frequency tonic firing patterns. Upon nerve injury, PVNs transition to adaptive firing and decrease their PV expression. Interestingly, decreased PV is necessary and sufficient to the development of mechanical allodynia and the transition of PVNs to adaptive firing. This transition of firing pattern is due to the recruitment of calcium-activated potassium (SK) channels and blocking them during chronic pain restores normal tonic firing. Our findings indicate that PV is essential to the firing activity of PVNs and in preventing allodynia, these observations may lead to novel strategies for chronic pain relief.