Since the discovery that LPA receptor signaling initiates neuropathic pain (Nature Medicine 2004), we have studied to probe that this finding is generalized in various chronic pain diseases. Current studies revealed that LPA1 signaling is involved in the development of chronic pain diseases. We have obtained findings that LPA1 receptor KO mice lose the chronic pain in partial sciatic nerve ligation-induced neuropathic (NeuP), paclitaxel treatments-induced NeuP, diabetic NeuP, central post stroke pain, spinal cord injury-induced NeuP, repeated acid saline-induced and intermittent psychological (empathy) or cold stress-induced fibromyalgia-like pain models. In some animal models, we also obtained the evidence that LPA1 signaling is also involved in the maintenance of chronic pain, since LPA1 antagonists reversed the established pain. Throughout the pharmacotherapeutical studies, we found that peripheral pain memory is maintained or feed-forwarded by LPA signaling in the spinal cord, and supra-spinal pain memory is additionally reinforced by arising pain itself, since repeated and complete medicinal treatments could completely cure the pain diseases even after the cessation of treatments. I will present key findings underlying my hypothesis and expect fruitful discussion.