research and improvement. The main pathogenic mechanisms include preoperative increased pain sensitivity (possibly due to genetic or psychosocial factors), intraoperative nerve damage, and acute postoperative pain.

Treatment strategies include effective – perhaps preemptive – perioperative analgesia, nerve-sparing minimally invasive surgical techniques and, potentially, preoperative identification of high-risk patients with subsequent high intensity, early and prolonged (?) acute pain treatment. However, results from analgesic trials irrespective of the technique used (systemic/ regional analgesia) have so far been rather disappointing. This is most probably due to the use of relatively insufficient and short-lasting monotherapy with limited effects on peripheral and central neuroplasticity. There is therefore a need for better-designed future clinical trials which include: (1) detailed preoperative assessment of nociceptive function (heat pain, pain genes, psychosocial factors); (2) detailed intraoperative description of the handling of nerves, and (3) detailed early and late postoperative assessment with quantitative sensory testing and magnetic resonance imaging (to determine the relative role of nerve damage vs a continued inflammatory response in the surgical field), combined with multimodal effective and prolonged (maybe 2–4 weeks) treatment in well-defined patient populations.

Recommended reading

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SCHWARZ PHARMA Sponsored Breakfast Symposium: LACOSAMIDE: A NEW ANTICONVULSANT FOR THE TREATMENT OF PAINFUL DIABETIC NEUROPATHY

126
LACOSAMIDE: A NEW ANTICONVULSANT FOR THE TREATMENT OF PAINFUL DIABETIC NEUROPATHY

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The investigational anticonvulsant lacosamide selectively enhances slow inactivation of sodium channels and interacts with collapsin response mediator protein 2.

Lacosamide was potent and broadly efficacious in animal models for pain, CNS disorders and epilepsy, where it acted synergistically with other established anticonvulsants.

In preclinical studies, lacosamide showed a favourable safety profile, no evidence of teratogenicity and no indication of abuse liability or dependence.

In vitro, lacosamide is barely metabolized and binding to plasma proteins is very low (<15%). Phase I trials did not reveal appreciable drug–drug-interactions, suggesting that lacosamide has only low potential for drug–drug-interactions in clinical use.

Phase II and III clinical trials with lacosamide in painful diabetic neuropathy consistently showed clinically relevant pain reduction in short- and long-term use. Lacosamide proved to be well tolerated. Somnolence and behavioral or cognitive side effects, which are typical for anticonvulsants, were comparably low in these studies. Lacosamide acted weight neutral and did not cause edema, which is of particular interest for diabetics. The safety profile observed in a 2-year open-label trial was comparable to that found in short-term studies. Long-term analyses showed sustained patient satisfaction with lacosamide treatment and good adherence to therapy.

In conclusion, lacosamide is a potent and safe anticonvulsant which is effective in treating painful diabetic neuropathy. Its novel dual mode of action differentiates lacosamide from currently available anticonvulsants and could be the link to its favorable preclinical and clinical profile. Based on its mode of action, lacosamide might even have disease-modifying effects.

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NeurogesX Sponsored Breakfast Symposium: LOCALIZED APPROACHES TO THE MANAGEMENT OF NEUROPATHIC PAIN SYNDROMES

127
LOCALIZED APPROACHES TO THE MANAGEMENT OF NEUROPATHIC PAIN SYNDROMES: PHYSIOLOGIC RATIONALE AND CLINICAL RESULTS

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Pharmacologic management of neuropathic pain is fraught with challenges. Responses to pharmacothera-