imbcom pty ltd
the commercialisation company for the institute for molecular bioscience
ABN 30 094733 909

Phospholipase A2 Inhibitors for the Treatment of Dysmenorrhoea

We are seeking a partner to out-license this opportunity for further clinical validation.

Market Overview

Fifty percent of menstruating women suffer from dysmenorrhoea, the period pain accompanying menstruation. Clinical signs of dysmenorrhoea are known to correlate with plasma and menstrual fluid levels of inflammatory mediators, eicosanoids, which are synthesised in the uterus. Eicosanoids such as prostaglandins (PGs) and leukotrienes (LTs) are normally the physiological mediators of uterine contraction, but in the case of primary dysmenorrhoea, these chemicals are over-produced. Elevated concentrations of eicosanoids increase the force of the uterine contraction (cramping), constriction of blood vessels with resultant anoxia of tissues (pain), and the sensation of pain receptors in the pelvic nerve terminals to other pain–inducing chemicals and physical stimuli. They can enter the circulation and cause diarrhoea, headache, dizziness, nausea and inflammation. Eicosanoids have also been identified in the pathology of endometriosis.

Development Status

Eicosanoids are a family of polyunsaturated fatty acids formed from arachidonic acid. Arachidonic acid is derived mainly from the phospholipids of cell membranes by the enzyme phospholipase A2 (PLA_2). Arachodonic acid is then further metabolised by (1) cyclooxygenase to produce the classical prostaglandins or by (2) lipoxygenase to produce leukotrienes.

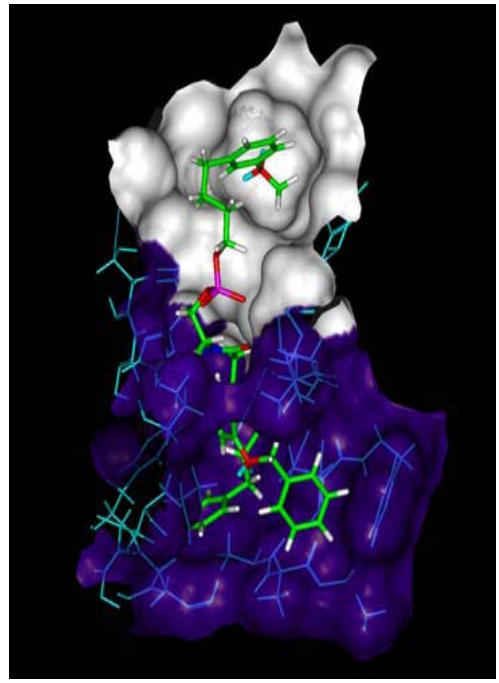
We have demonstrated that non-peptide inhibitors of human non-pancreatic $sPLA_2$ were up to 1000 times more effective in inhibiting uterine contractions compared to non-steroidal anti-inflammatory drugs (NSAIDs). These $sPLA_2$ inhibitors block the formation of mediators that cause uterine contractions, providing strong evidence that they will be effective in reducing or alleviating the symptoms associated with dysmenorrhoea.

Intellectual Property

Current treatment for dysmenorrhoea relies mainly on the use of aspirin or NSAIDs. NSAIDs exert their effects via inhibition of cyclooxygenase enzymes (COX-1 or COX-2), which synthesise the prostaglandin eicosanoids. A significant proportion of dysmenorrhoea sufferers' symptoms are not relieved by NSAIDS. Furthermore, some medical conditions may preclude use of some NSAIDs (eg aspirin-sensitive asthma, etc) and prolonged use of many non-specific NSAIDs can cause gastrointestinal side effects.

Business Opportunity

This IP is currently in National Phase Entry in the US, Europe and Australia. Each of these jurisdictions show significant market potential for this technology. We are currently seeking to out-licence this intellectual property.



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