We expect that by targeting FABPs endocannabinoid levels will increase within the brain, essentially resulting in anti-pain and anti-inflammatory effects.

**Background**

The global analgesic pharmaceutical market, (>30 billion) is responding to high market demand for pharmacological compounds that work on the pain center of the brain. One innovative approach under review is altering the levels of anandamide (AEA), an endocannabinoid and neurotransmitter that regulates analgesia, vasodilatation, appetite and pleasure. Stony Brook researchers have identified the precise molecular mechanisms behind AEA transport and metabolism. Understanding this mechanism has lead to the creation of novel pharmacological compounds and new therapeutic targets for treatment of a wide variety of disorders, including addiction, pain, inflammation and appetite regulation.

**Technology**

Dr. Dale Deutsch and Dr. Iwao Ojima at Stony Brook University have worked together to identify a novel pain target and create lead compounds rationally designed to inhibit the targeted pathway. Dr. Deutsch has uncovered that endocannabinoid inactivation is modulated by FABP 5 and 7, transport proteins for anandamide, which deliver anandamide from the cell surface to their internal cellular binding proteins. Dr Ojima has created novel small molecules, which reversibly bind to FABP 5 and 7 and effectively block the transport of endocannabinoids. Taken together, this novel target and new class of small molecule inhibitors may be further developed as a treatment for neuropathic and inflammatory pain.

**Patent number/Publication:**
- Issued patent 8,980,820 (method); PCT/US2013/051337 (compounds, US and EU)

**Advantages**
- Patented targets with known crystal structure
- New composition of matter tested in the lab
- Increased efficacy over known FABP inhibitors
- Animal data

**Applications**
- Neuropathic pain
- Inflammatory pain

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