UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): October 4, 2008

Sucampo Pharmaceuticals, Inc.

(Exact Name of Registrant as Specified in Charter)

Delaware	001-33609	13-3929237
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)
4520 East-West Highway, Suite 300 Bethesda, Maryland		20814
(Address of Principal Executive Offices)		(Zip Code)

Registrant's telephone number, including area code: (301) 961-3400

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (*see* General Instruction A.2. below):

o Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

o Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

o Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

o Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 8.01. Other Events.

On October 4, 2008, Sucampo Pharmaceuticals, Inc. delivered a scientific conference presentation at the 2nd World Conference on Magic Bullets (Ehrlich II) and American College of Gastroenterology that included written communication comprised of a presentation and poster. The presentation and poster at the 2nd World Conference on Magic Bullets (Ehrlich II) and American College of Gastroenterology are being furnished as Exhibits 99.1 and 99.2 to this Current Report on Form 8-K.

The information in this Item 8.01 and Exhibits 99.1 and 99.2 to this Form 8-K shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934 (the "Exchange Act") or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such a filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

99.1 Presentation for the 2nd World Conference on Magic Bullets (Ehrlich II), dated October 4, 2008

99.2 Poster for American College of Gastroenterology, dated October 4, 2008

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

SUCAMPO PHARMACEUTICALS, INC.

Date: October 7, 2008

By: /s/ JAN SMILEK
Name: Jan Smilek
Title: VP, Finance and Acting Chief Financial Officer

PROSTONES AS CIC-2 Cl^- CHANNEL ACTIVATORS FOR TREATMENT OF DISEASES AND DISORDERS

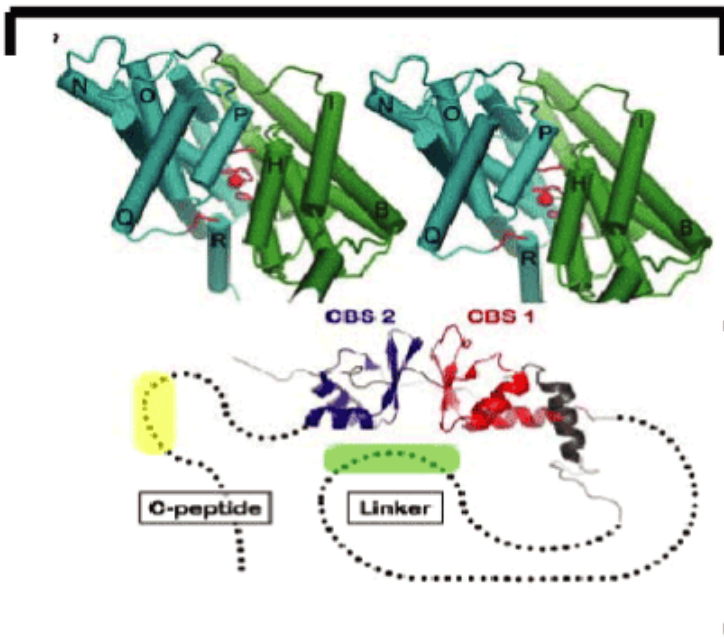
John Cuppoletti University of Cincinnati, Cincinnati,OH
and
Ryuji Ueno, Sucampo Pharmaceuticals Inc., Bethesda, MD

Prostones

- Prostones are derivatives of metabolites of prostaglandins.
- Lubiprostone is a prostone which is used to clinically to treat chronic idiopathic constipation and irritable bowel syndrome related to constipation (IBS-C).
- Lubiprostone is a ClC-2 chloride channel activator which increases salt and water secretion in the intestine.

ClC Cl^- CHANNEL IS THE TARGET FOR LUBIPROSTONE

ClC Cl^- CHANNELS ARE DIMERS



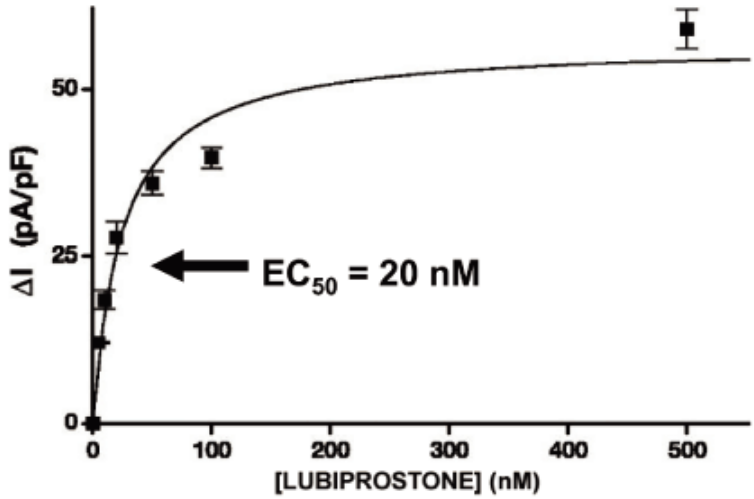
MEMBRANE DOMAIN[1]

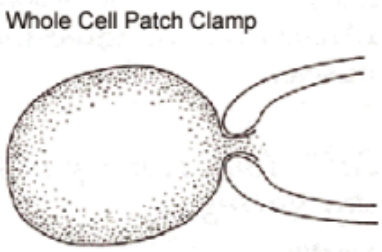
CYTOSOLIC C- TERMINAL DOMAIN[2]

[1]R.Dutzler, E.B. Campbell, M. Cadene, B.T. Chait and R.MacKinnon Nature 415, 287-294(2002)

[2]S. Alioth, S. Meyer, R. Dutzler and K. Pervushin Journal of Molecular Biology Vol. 369, Issue 5, 22 June 2007, Pages 1163-1169

LUBIPROSTONE POTENTLY ACTIVATES RECOMBINANT HUMAN CIC-2 CHLORIDE CHANNELS STUDIED BY WHOLE CELL PATCH CLAMP

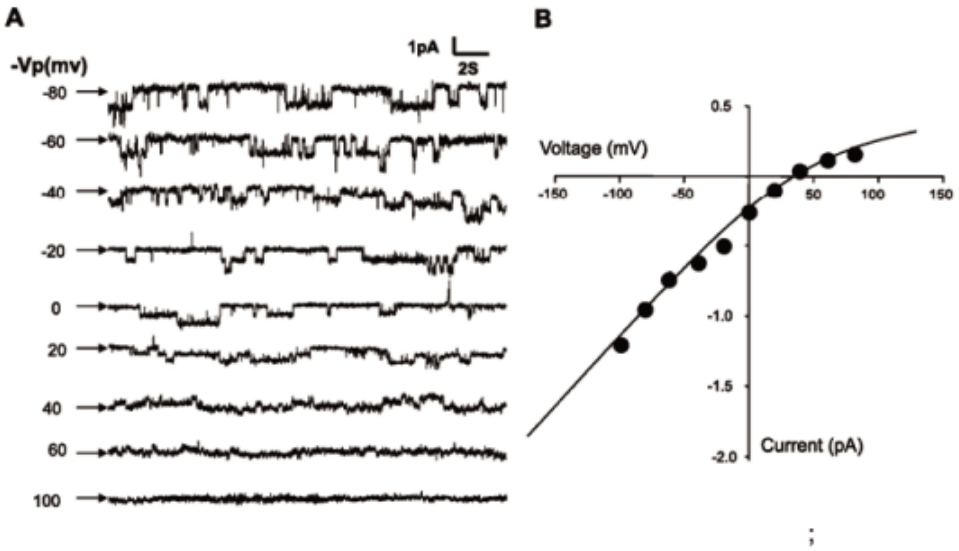




Cuppoletti, J. et al. Am J Physiol Cell Physiol 287: C1173-C1183 2004

AJP - Cell Physiology

LUBIPROSTONE ACTIVATES RECOMBINANT HUMAN ClC-2 Cl^- CHANNELS IN HEK293 CELLS AT SINGLE CHANNEL LEVEL RECORDED BY PATCH CLAMP



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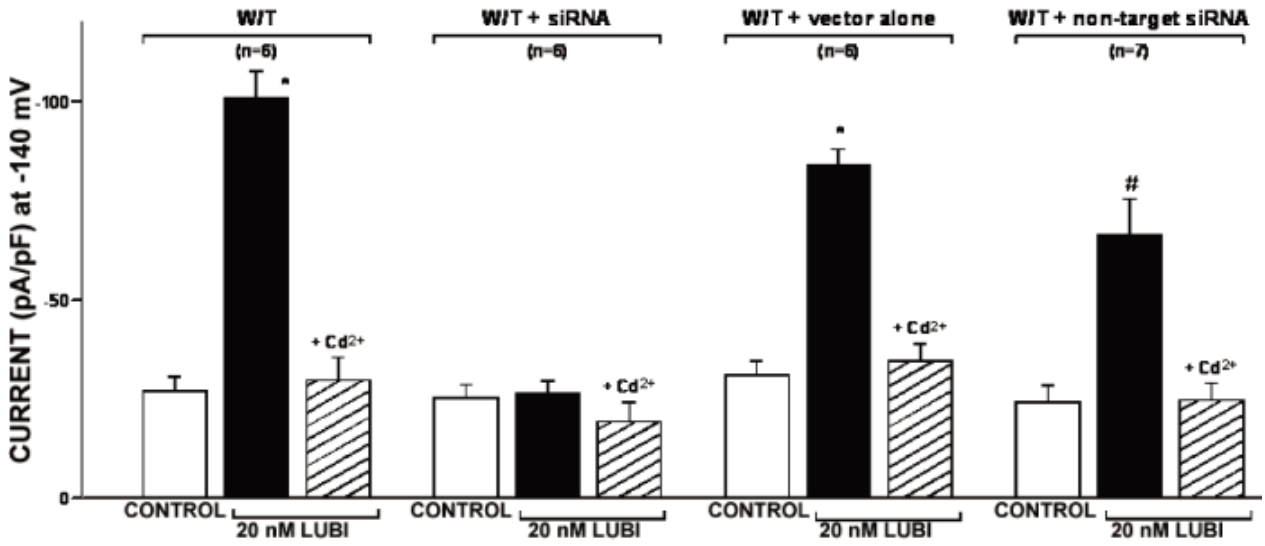
Bao, H. F. et al. Am J Physiol Gastrointest Liver Physiol 295: G234-G251 2008

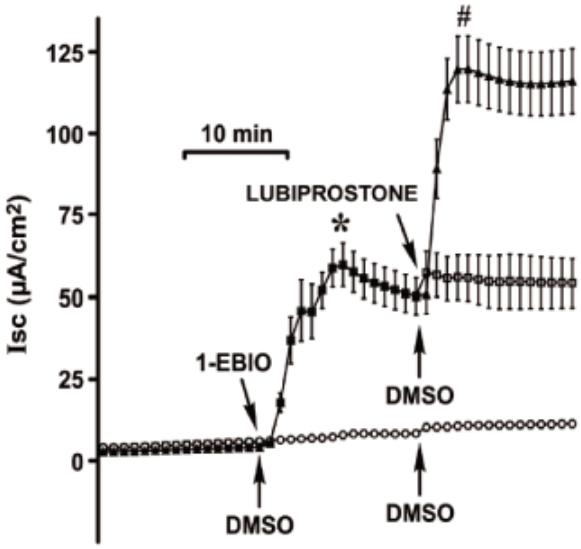
AJP - Gastrointestinal and Liver Physiology

siRNA TO ClC-2 ABLATES LUBIPROSTONE ACTIVATION OF CHLORIDE TRANSPORT BY RECOMBINANT HUMAN ClC-2 IN HEK293 CELLS

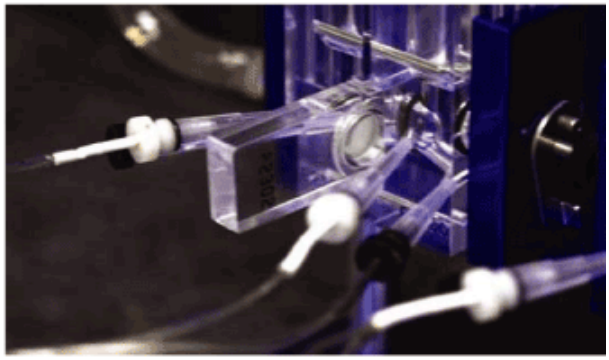


Cuppoletti J, Malinowska DH Tewari K, Chakrabarti J, Ueno R *Gastroenterology*, Vol. 134, Issue: 4, April, 2008. pp. A-582

LUBIPROSTONE ACTIVATES Cl^- TRANSPORT IN T84 INTESTINAL CELL CULTURES STUDIED BY SHORT CIRCUIT CURRENT WITH USSING CHAMBERS



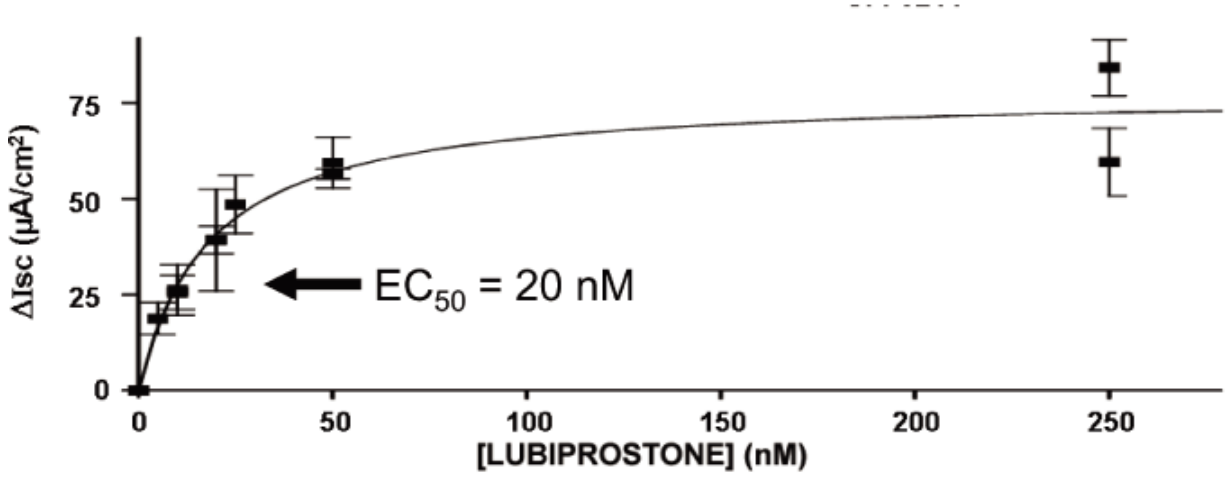
USSING CHAMBER



Cuppoletti, J. et al. Am J Physiol Cell Physiol 287: C1173-C1183 2004

AJP - Cell Physiology

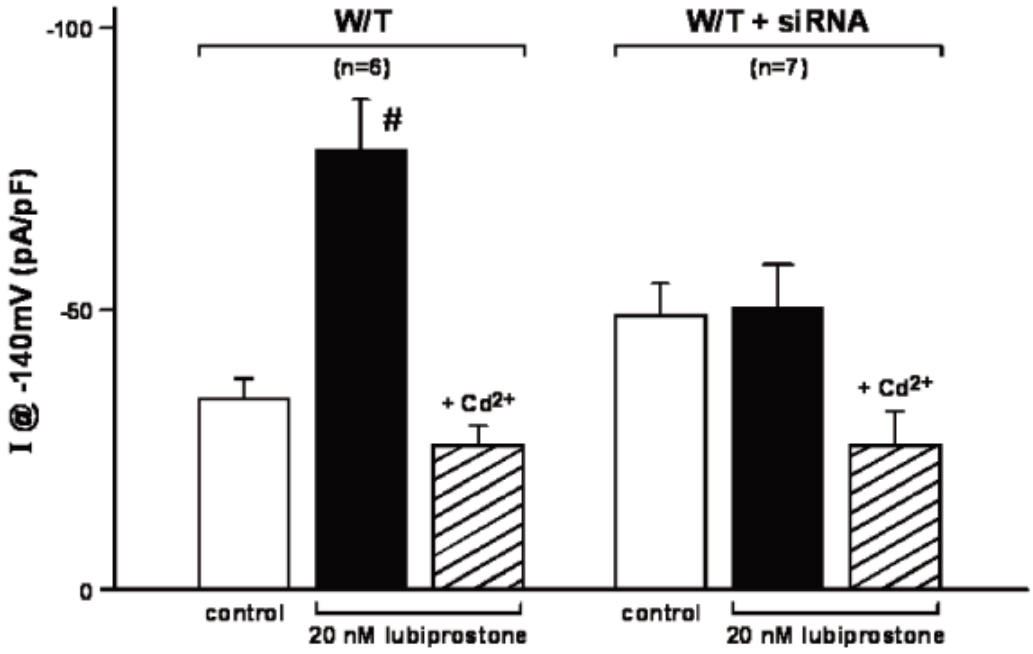
LUBIPROSTONE POTENTLY STIMULATES Cl^- TRANSPORT IN T84 INTESTINAL CELLS



Cuppoletti, J. et al. Am J Physiol Cell Physiol 287: C1173-C1183 2004

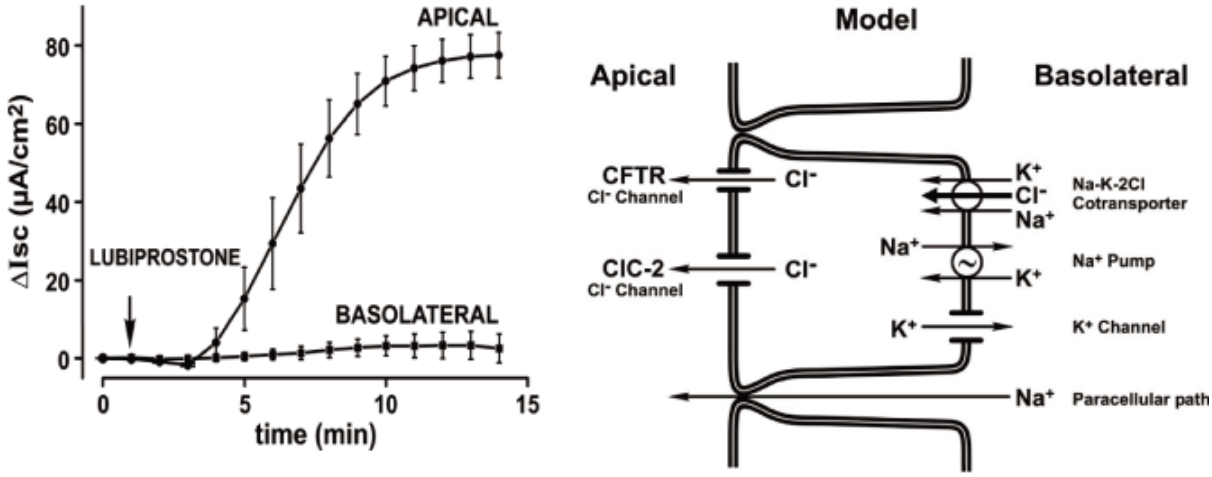
AJP - Cell Physiology

siRNA TO ClC-2 ABLATES STIMULATION OF Cl^- TRANSPORT BY LUBIPROSTONE IN T84 CELLS



Cuppoletti J, Malinowska DH Tewari K, Chakrabarti J, Ueno R *Gastroenterology*, Vol. 134, Issue: 4, April, 2008. pp. A-582

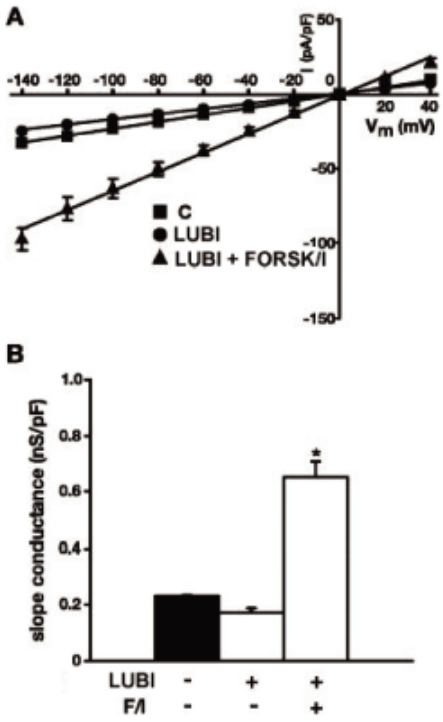
CIC-2 IS EXPRESSED ON THE APICAL MEMBRANE AS SHOWN USING NYSTATIN SOLUBILIZATION OF APICAL OR BASOLATERAL MEMBRANE



Cuppoletti, J. et al. Am J Physiol Cell Physiol 287: C1173-C1183 2004

AJP - Cell Physiology

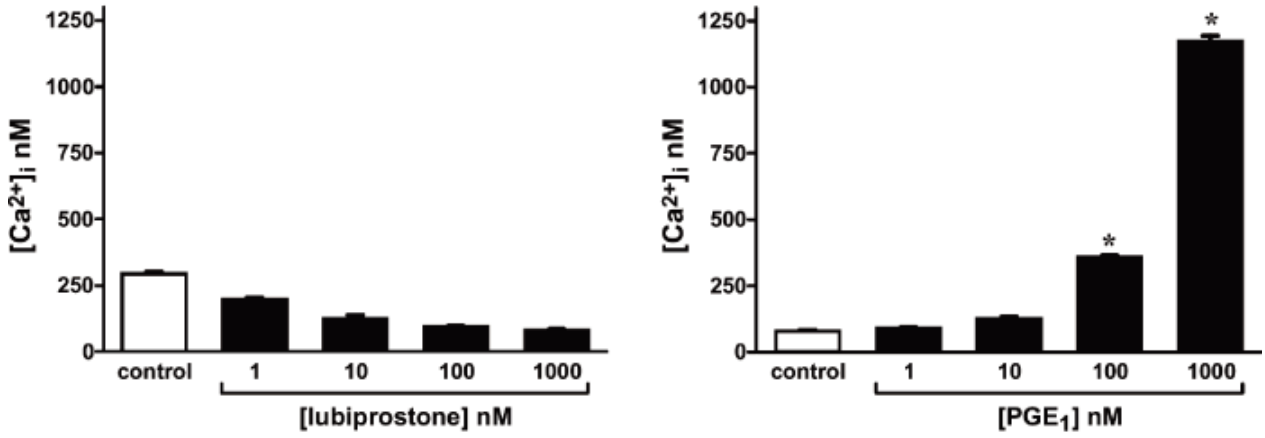
LUBIPROSTONE DOES NOT STIMULATE Cl^- TRANSPORT BY CFTR



Cuppoletti, J. et al. Am J Physiol Cell Physiol 287: C1173-C1183 2004

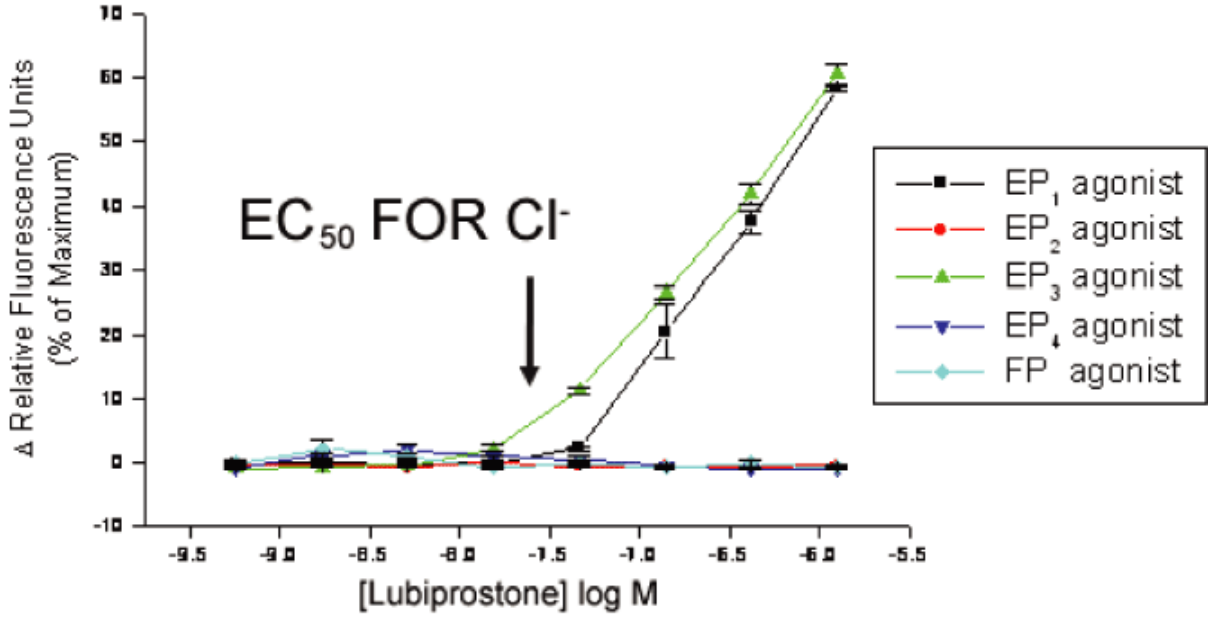
AJP - Cell Physiology

LUBIPROSTONE DOES NOT ACT THROUGH INCREASES IN INTRACELLULAR CALCIUM



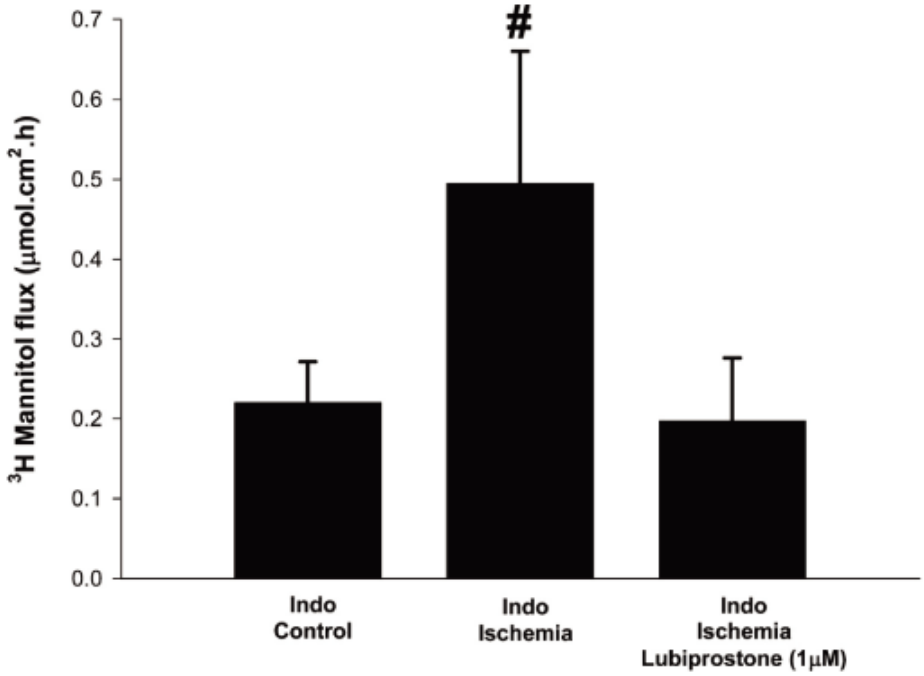
Cuppoletti J, Malinowska DH Tewari K, Chakrabarti J, Ueno R *Gastroenterology*, Vol. 134, Issue: 4, April, 2008. pp. A581-A-582

LUBIPROSTONE DOES NOT ACTIVATE Cl^- TRANSPORT THROUGH EP OR FP RECEPTORS



J.Cuppoletti, D.H.Malinowska, R.Ueno. ACG meeting, Orlando,Fl October 5, 2008

LUBIPROSTONE PROMOTES REPAIR OF BARRIER FUNCTION AFTER DAMAGE BY INDOMETHACIN AND ISCHEMIA



Moeser, A. J. et al. Am J Physiol Gastrointest Liver Physiol 292: G647-G656 2007;

AJP - Gastrointestinal and Liver Physiology

SUMMARY OF LUBIPROSTONE MECHANISMS OF ACTION

- LUBIPROSTONE TARGETS CIC-2 CHLORIDE CHANNELS IN THE INTESTINE AS SHOWN BY WHOLE CELL CURRENTS, SINGLE CHANNELS AND SHORT CIRCUIT CURRENT.
- LUBIPROSTONE POTENTLY ACTIVATES CIC-2 CHLORIDE CHANNELS
- LUBIPROSTONE DOES NOT AFFECT OTHER INTESTINAL CHLORIDE CHANNELS
- THE SITE OF ACTION OF LUBIPROSTONE IS THE APICAL MEMBRANE
- LUBIPROSTONE DOES NOT ACT THROUGH EP- OR FP-RECEPTORS, INCREASES IN cAMP, OR INCREASED CALCIUM
- LUBIPROSTONE INCREASES SALT AND WATER SECRETION IN INTESTINAL CELLS AND THIS IS THE BASIS FOR CLINICAL ACTION IN CONSTIPATION
- LUBIPROSTONE PROMOTES REPAIR OF EPITHELIAL BARRIERS AND THIS MAY CONTRIBUTE TO CLINICAL ACTION IN IBS-C

ACKNOWLEGDEMENT

- THESE STUDIES WERE SUPPORTED BY A GRANT FROM SUCAMPO PHARMACEUTICALS, INC., BETHESDA MD, USA

UNIVERSITY OF Cincinnati

SUCAMPO PHARMACEUTICALS, INC.

Exhibit 99.2

Prostaglandin Receptor Activation Properties of Lubiprostone

J. Cuppoletti*, D.H. Malinowska*, R. Ueno**
*Molecular and Cellular Physiology, University of Cincinnati, Cincinnati, OH; **Sucampo Pharmaceuticals, Inc., Bethesda, MD

Introduction

Lubiprostone is used clinically to treat chronic idiopathic constipation and irritable bowel syndrome with constipation[1]. It is a prostone (derived from metabolites of prostaglandins) that activates ClC-2 Cl^- channels with an EC_{50} of 20 nM[2] and increases intestinal fluid secretion. In the present study, the agonist and antagonist activity of lubiprostone on cloned human EP and FP receptors was examined to more directly determine the EC_{50} for lubiprostone binding to these specific receptors. This reduces the need to use either EP receptor antagonists or complex biological processes such as contraction to infer binding of lubiprostone to prostaglandin receptors.

Purpose

The aim of the present study was to determine the activities of lubiprostone on recombinant prostaglandin receptors.

Methods

Lubiprostone binding to recombinant EP_1, EP_2, EP_3, EP_4 and FP receptors was assayed by Millipore Corporation, Bioscience Division (St. Charles, MO), using Chemiscreen calcium optimized FLIPR cell lines containing high levels of the promiscuous G protein, Galpha15, to enhance coupling of the receptor to the calcium signaling pathway. These cells were transfected with cDNA containing either full-length human EP_1, EP_3, splice variant 6 of EP_3, EP_4 or FP receptors. Triplicate assays of lubiprostone effects were carried out with PGE_2 or $PGF_{2\alpha}$ as a positive control. In all cases, the readout was relative fluorescence units related to $[Ca^{2+}]_i$ through calcium release activated calcium ion channel (CRAC) measured by Fluo-4 relative fluorescence. To measure antagonist effects of lubiprostone, cells with cloned human receptors were first stimulated with either PGE_2 or $PGF_{2\alpha}$ to give about 80% of the maximum response and then lubiprostone was added.

Results

Positive controls: Measured agonist activity of PGE_2 on EP_1, EP_2, EP_3 and EP_4 receptor-expressing cells generated EC_{50} values of 7.46, 49.82, 3.86 and 31.18 nM respectively. Agonist activity of $PGF_{2\alpha}$ on FP receptor-expressing cells gave an EC_{50} of 3.40 nM.

Lubiprostone exhibited no agonist effects on cloned EP_2, EP_4 or FP receptors (Figure 1). There was weak agonist activity of lubiprostone on EP_1 and EP_3 receptors with EC_{50} values of 330 nM and 280 nM, respectively, which are 44 and 73 times higher than the agonist activity of PGE_2 activity on the respective EP receptors.

Lubiprostone did not demonstrate any detectable antagonist effects on EP_3 or FP receptors (Figure 2). However, there was weak antagonist effect of lubiprostone on EP_4 receptors (EC_{50} =127 nM).

Figure 1. Agonist Effect of Lubiprostone on Cloned EP and FP Receptors

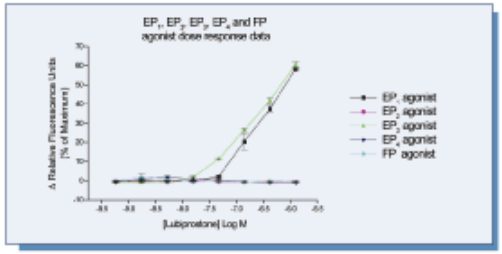


Figure 1. Dose response curve for agonist activity of lubiprostone on cloned EP_1, EP_2, EP_3, EP_4 and FP receptors expressed in cultured cells. Data represent the mean (± SE) of 3 determinations.

Figure 2. Antagonist Effect of Lubiprostone on Cloned EP and FP Receptors

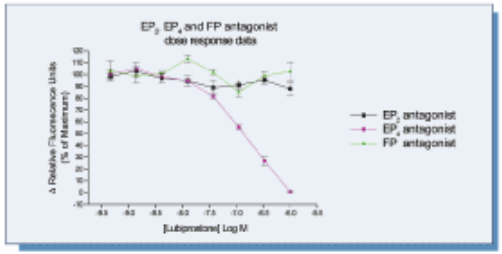


Figure 2. Dose response curve for antagonist activity of lubiprostone on cloned human EP_2, EP_4 and FP receptors expressed in cultured cells. Data represent the mean (± SE) of 3 determinations.

Summary

It has been shown in the present study that lubiprostone does not act as an agonist on cloned human EP_2, EP_4 or FP receptors. The lack of agonist activity of lubiprostone on cloned human EP_4 contradicts the finding that lubiprostone reduced electrically stimulated neural contractions in rat and human colon circular muscle with an EC_{50} at near nanomolar levels that were inhibited by an EP_4 (but not other EP) receptor antagonist[3] and implies that these reported effects are not due to EP_4 receptor occupation by lubiprostone.

Agonist activity of lubiprostone on cloned EP_1 was very low with an EC_{50}= 330 nM, a value 44 times higher than for PGE_2 on the EP_1 receptor. Moreover, this concentration is approximately 15 times higher than the EC_{50} for lubiprostone for activation of ClC-2 chloride channels[2]. Lubiprostone remains mostly in the lumen of the gut and does not enter the circulation[1]. This means that lubiprostone will not reach sufficiently high concentrations to activate EP_1 receptors in the stomach muscle layer. Since EP_1 antagonists do not affect the PGE_2 effects on the vagal nerve[4], lubiprostone is unlikely to act on the stomach through EP_1 (or other EP or FP) receptors causing vagal nerve stimulation. This study confirms the previous findings of Ueno *et al*[5] that demonstrated that lubiprostone does not have pharmacologically relevant activity on prostaglandin receptors.

Conclusions

At clinically relevant doses lubiprostone is unlikely to have significant PG receptor activity. The results demonstrate that activation of ClC-2 chloride channels underlying the clinical effects of lubiprostone is independent of EP or $FP_{2\alpha}$ receptor occupation.

References

1. Amitiza (package insert). Bethesda, MD: Sucampo Pharmaceuticals, Inc., 2008.
2. Cuppoletti J, Malinowska DH, Tewari KP, Li QJ, Sherry AM, Patchen ML, Ueno R. SPI-0211 activates T84 cell chloride transport and recombinant human ClC-2 chloride currents. Am J Physiol Cell Physiol. 2004 287(5):C1173-83.
3. Basil AK, Borman RA, Jones EM, McArthur Wilson RJ, Thangiah R, Sung EZ, Lee K, Sanger GJ. Activation of prostaglandin EP receptors by lubiprostone in rat and human stomach and colon. Br J Pharmacol. 2008 154(1):129-35.
4. Kee KK, Jones RL, Higgs MP, Tuddi JA. Excitatory action of prostanoids on the ferret isolated vagus nerve preparation. Eur J Pharmacol. 2004 Apr 20;491(1):37-41.
5. Ueno R, Engelke KJ, and Osanai H. Effects of lubiprostone, a novel GI chloride channel activator, on isolated smooth muscle. Neurogastroenterol Motil. 2005 17(4): 625-636.

Supported by Sucampo Pharmaceuticals, Inc. and Takeda Pharmaceutical Company Ltd.
Presented at The American College of Gastroenterology 2008 Scientific Meeting