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

November 15, 2016
Date of Report (Date of earliest event reported)

Flex Pharma, Inc.

(Exact name of registrant as specified in its charter)

Delaware	**001-36812**	**46-5087339**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

800 Boylston Street, 24th Floor Boston, MA	**02199**
(Address of principal executive offices)	(Zip Code)

Registrant's telephone number, including area code: **(617) 874-1821**

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligations of the registrant under any of the following provisions:

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

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

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

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

Item 7.01. Regulation FD Disclosure.

On November 15, 2016, Flex Pharma, Inc. will present a poster entitled "Chemical Neuro Stimulation of TRPV1 and TRPA1 Sensory Neurons Decreases Muscle Cramps in Humans" at the Society for Neuroscience Conference. The poster is furnished herewith as Exhibit 99.1.

The information contained in this Item 7.01 and Exhibit 99.1 attached hereto shall not be deemed "filed" for purposes of Section 18 of the Securities and Exchange Act of 1934, as amended, 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, except as expressly set forth by specific reference in such filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

The following exhibit relating to Item 7.01 shall be deemed to be furnished and not filed:

Exhibit No.	Description
99.1	Flex Pharma, Inc. Poster

SIGNATURES

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.

Flex Pharma, Inc.

Dated: November 15, 2016

By: /s/ Robert Hadfield
Robert Hadfield
General Counsel and Secretary

INDEX TO EXHIBITS

Exhibit No.	Description
99.1	Flex Pharma, Inc. Poster

Presentation Number: 537.15

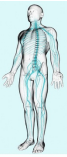
Chemical Neuro Stimulation of TRPV1 and TRPA1 Sensory Neurons Decreases Muscle Cramps in Humans

Glenn F. Short III, Laura B. Rosen, Robin Sutherland, Jian Liu, Jennifer M. Cermak, Gary Maier and Thomas Wessel

Flex Pharma, Inc. Boston, MA 02199

FLEXPharma

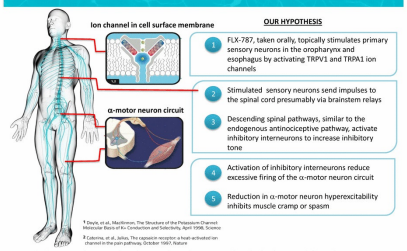
Novel Treatments for Neuromuscular Conditions



Summary

Chemical Neuro Stimulation is the treatment of neurological disorders by using small molecules applied topically to sensory neurons to alter the behavior of distinct neural circuits within the central nervous system. We have devised one such approach whereby the co-activation of TRPV1 and TRPA1 ion channels in the upper alimentary canal decreases muscle cramp frequency and severity. Based upon recent evidence that α-motoneuron hyperexcitability is the underlying cause of cramps and spasticity (1,2), we hypothesized that TRP activation could provide sufficient excitatory sensory input via the solitary tract to modulate descending spinal pathway signaling to increase interneuronal inhibition and dampen motor neuron hyperexcitability. We have generated data that suggests that either an oral solution containing a mixture of naturally-derived TRP activators (TRP-Stim) or FLX-787, a synthesized single molecule TRPV1/TRPA1 co-activator, stimulate TRP ion channels in the mouth, oropharynx and esophagus in a local, topical fashion to inhibit muscle cramping. Efficacy studies using an electrically-induced cramp (EIC) model demonstrated that both TRP-Stim and FLX-787 significantly reduced cramp intensity by as much as 3-fold relative to inactive control (p<0.01). Moreover, the pharmacokinetic profile of FLX-787 could not account for its EIC efficacy, as no systemic exposure of the parent form of FLX-787 in human plasma was observed. In both animals and humans, FLX-787 was found to undergo rapid phase 2 metabolism, resulting in extensive conjugation 15 minutes after ingestion, predominantly to glucuronide and sulfate metabolites. Even at doses up to 500 mg/kg/day in rats, the conjugates of FLX-787 accounted for >90% of circulating drug. To understand if topical exposure to mucous membranes in the mouth, oropharynx and esophagus mediates the TRP-Stim and FLX-787 effect (given the lack of systemic exposure to FLX-787), the TRP-Stim mixture was encapsulated in gelatin capsules. Ingestion of the encapsulated mixture afforded no EIC efficacy relative to vehicle control. These results suggest that the observed effect on electrically-induced muscle cramps does not depend on the systemic bioavailability of TRP activators but rather on topical exposure of sensory neurons and consequent neuronal signaling. Given that efficacy signals have also been observed in proof of concept (POC) nocturnal leg cramp (NLC) studies in humans with TRP-Stim or FLX-787, Chemical Neuro Stimulation may be a general approach to develop novel treatments for cramps, spasms and spasticity in clinical populations. Based upon these findings, we have initiated studies with FLX-787 in MS and ALS.

Topical Chemical Neuro Stimulation



[1] Doyle, et al., MacKinnon, The Structure of the Potassium Channel: Molecular Basis of K+ Conduction and Selectivity, April 1998, Science

[2] Caterina, et al., Julius, The capsaicin receptor: a heat-activated ion channel in the pain pathway, October 1997, Nature

Figure 1. Overview of suspected mechanism of muscle cramps and methods of cramp inhibition by activation of TRP ion channels. Muscle cramping is caused by the uncontrolled and repetitive firing of α-motor neurons in the spinal cord (1), resulting in maintained contraction of the muscle. Flex Pharma's proprietary products exploit a general principle of neural circuits whereby strong excitatory sensory input from one source enhances overall inhibitory tone by increased recruitment of inhibitory neurons, thereby reducing excitability in other parts of the circuit (3). Flex Pharma's products stimulate primary sensory neurons in the mouth, esophagus and stomach by activating TRPV1 and TRPA1 ion channels (4,5). When activated, these sensory neurons, which project both directly and indirectly to the spinal cord, enhance the inhibitory tone in spinal cord circuits to reduce repetitive firing of α-motor neurons which prevents or reduces the frequency and intensity of muscle cramps and spasms.

Methods

Muscle cramps were induced in the flexor hallucis brevis (FHB) muscle by electrical stimulation and monitored by EMG to quantify cramp intensity and duration (Figure 2). The subject's medial plantar nerve was electrically stimulated by transcutaneous nerve stimulation 2Hz above the experimentally determined cramp threshold frequency to elicit a reproducible and robust cramp. The muscle cramp intensity and duration were measured by EMG and quantified by calculating the area under the cramp curve (AUC) and cramp duration post cessation of electrical stimulation (Figure 3). Muscle cramp intensity and duration were found to vary from subject-to-subject, necessitating a pre-treatment EMG to serve as a subject-specific baseline control. After consumption of Flex Pharma products or vehicle control, the resulting EMGs were quantified for cramp AUC and duration and compared to baseline values. The time at which the subject received treatment or vehicle control is referred to as time point zero.

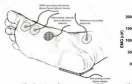


Fig 2. Muscle cramps were induced in the flexor hallucis brevis and monitored by EMG.

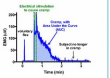
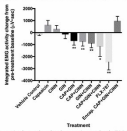


Fig 3. Exemplary EMG demonstrating cramp induction and observable AUC.

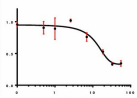
Results

Figure 4. Topical exposure to oropharynx and esophagus is required for cramp inhibition



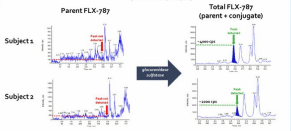
FLX-787 (29 mg), a single molecule that activates both TRPA1 and TRPV1, afforded a greater decrease in cramp intensity compared to other treatments tested. The effect of treatment was calculated based upon the AUC change from a pre-treatment baseline cramp. FLX-787 treatment led to a 5-fold reduction in AUC compared to its study specific vehicle control. Treatments that demonstrated a significant difference from study-specific vehicle controls are annotated with an asterisk (* p<0.05, ** p < 0.01).

Figure 5. FLX-787 Inhibition of electrically-Induced cramps in healthy subjects is dose-responsive



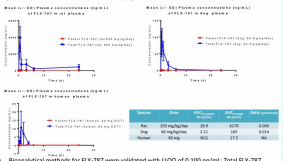
- Seven doses of FLX-787 ODT yield classic sigmoidal dose curve (n=5, p<0.05)
- Efficacy saturates at 32 mg FLX-787-ODT

Figure 6. FLX-787 is not systemically available in its parent form in humans



- 1h after PO administration of a liquid formulation containing 19 mg FLX-787
- The parent form of FLX-787 was not detected in plasma only conjugates (LLOQ 0.100 ng/mL).
- Suggests rapid first-pass phase 2 metabolism with no measurable systemic exposure of parent drug.

Figure 7. Primary systemic exposure of glucuronide and sulfate conjugates of FLX-787 in all species



- Bioanalytical methods for FLX-787 were validated with LLOQ of 0.100 ng/mL; Total FLX-787 methods are semi-quantitative with LLOQ of <0.200 ng/mL
- The variability noted in the metabolic conversion of FLX-787 to conjugates suggests rapid and extensive phase 2 metabolism (first pass likely at the enterocyte level)

NLC Exploratory POC Studies

❖ **Nocturnal leg cramps (NLC)**

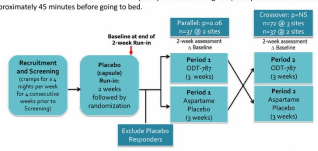
- 50% of those over the age of 50 suffer from NLC with increasing prevalence and frequency with age; Over 4 million in the US over age 65 suffer daily.
- Lack of clinical evidence that common "remedies" such as electrolyte replacement, bananas and hydration afford relief.
- Quinine, prescribed in the United Kingdom for NLC, is associated with thrombocytopenia, hypersensitivity reactions and QT prolongation and is no longer approved in the US for NLC.
- No approved drug alternative in US to treat NLC.

References

1. Minetto MA, Holobar A, Botter A, and Farina D. *Exerc Sport Sci. Rev.* 41(1): 3-10, 2013.
2. Milanov, I. Electromyogr Clin Neurophysiol. 50 (5): 73-9, 1994.
3. Okun, M. & Lamp; I. *Nature Neurosci.* 11: 535–537, 2008.
4. Bolshakov et al., *Am J Physiol Gastrointest Liver Physiol* 294: C1120–C1128, 2008.
5. Yu et al., *Am J Physiol Gastrointest Liver Physiol* 307: G34–G42, 2035.
6. El-Tawil S. et al., *Cochrane Database of Systematic Reviews* Issue 12, Art. No.: CD006644.2010.

Figure 8. Interim data analyses of NLC Exploratory POC Studies signal efficacy and carry-over effects

Multi-Center Trials in NLC: Two randomized, double-blind, placebo-controlled, cross-over studies to evaluate the effects of a FLX-787 on the frequency of nocturnal leg foot/cramps when self-administered approximately 45 minutes before going to bed.



- **Study 1**: Efficacy signals (cramp frequency, p=0.06) with ODT in sub-analysis (n=37) of parallel portion
  - Data from 1 site (n=35) excluded
- **Study 2** (dosing/formulation): Significant effect (cramp frequency, p<0.05) in first exposure parallel analysis (repeated, sequential, multiple crossovers, n=29 from prior NLC study)
  - Statistically significant on some, but not all crossover endpoints

Figure 9. Effect sizes calculated for the parallel portion of the cross-over studies are on-average larger than those reported in the quinine clinical literature

Endpoint	Treatment/Study	N_{total}	$N_{left\ size}$	p-value	Effect Size
Cramp Frequency	TRP-Stim	50	50	0.08	0.46
	FLX-787 (ODT)/Study 1*	72	37	0.06	0.77
	FLX-787 (liq)/Study 2	29	29	0.02	0.94

*The effect size of Study 1 is a sub-analysis (n=37) excluding data from 1 site (n=35).

- Exploratory ANOVA analyses across studies showed a period effect within each cross-over
- To avoid the potential influence of carry-over on effect size estimates were calculated from the first exposure in each study
- Average effect size of cramp frequency derived from quinine literature is **0.12** (95%CI[-3.5,-1.36]). (6)

Conclusions

- FLX-787 has demonstrated a sigmoidal dose-response curve in a human EIC-model in the absence of systemic exposure.
- Topical Chemical Neuro Stimulation of TRPA1/TRPV1 indirectly inhibits α-motor neuron hyperexcitability.
- FLX-787 has shown positive signals on cramp frequency in the parallel design portion of two exploratory human POC NLC studies.
- FLX-787 is well tolerated and safe, and no SAEs have been reported.
- Consistent with FDA guidance, future FLX-787 studies in NLC will be parallel design with emphasis on patient selection, data capture & monitoring.
- Clinical studies in MS and ALS are underway to explore the utility of FLX-787 in additional indications of different etiology where cramping and/or spasticity is prevalent.
- Planned initiation of IND-opening Phase 2 parallel design study in H1 2017.