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): November 1, 2016

RESPIRERX PHARMACEUTICALS INC.
(Exact name of registrant as specified in its charter)

Delaware	**1-16467**	**33-0303583**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

126 Valley Road, Suite C **Glen Rock, New Jersey** **(Address of principal executive offices)**	**07452** **(Zip Code)**

Registrant's telephone number, including area code: (201) 444-4947

(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:

[ ] 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 8.01 Other Events.

RespireRx Pharmaceuticals Inc. (the "Company") has received a copy of the Final Progress Report (the "Project Report") with respect to the project titled "Cannabimimetic treatment of obstructive sleep apnea: A proof of concept trial" (the "Project"), grant number 5UM1HL112856. The Company received this Project Report from the National Heart, Lung and Blood Institute of the National Institutes of Health in response to a Freedom of Information Act ("FOIA") request. A copy of the Project Report is attached as Exhibit 99.1 to this Current Report on Form 8-K.

As part of this Project, the University of Illinois and three other centers conducted a six week, placebo-controlled Phase 2B clinical trial investigating the effects of dronabinol in patients with obstructive sleep apnea (the "Clinical Trial"). The Company has licensed from the University of Illinois certain patent rights pertaining to the use of cannabinoids, including dronabinol, for the treatment of sleep-related breathing disorders such as obstructive sleep apnea. The Company did not manage or fund this Clinical Trial; this Clinical Trial was fully funded by the National Heart, Lung and Blood Institute of the National Institutes of Health and is being managed by University of Illinois researchers.

The Project Report describes preliminary data from three groups of patients who received either of two doses of dronabinol (2.5 or 10 mg) or placebo. The identities of the groups described in the Project Report remain blinded, but the Project Report indicates that significant group differences were observed in three of four primary outcome measures, including the Apnea-Hypopnea Index (AHI) and Epworth Sleepiness Scale (ESS). Unblinding of the group identities is expected to occur when the investigators have completed all planned primary and secondary analyses. The investigators have indicated that they "anticipate submitting an abstract detailing at least the top-line findings in December 2016, for presentation at the *Sleep 2017* international conference of the Associated Professional Sleep Societies." The investigators also stated they anticipate that they "will submit a peer-review manuscript detailing these findings in the Winter of 2017."

The Company is not involved in the management of the Clinical Trial and takes no position on the information contained in the Project Report. The Company is providing the Project Report, which is publicly available, to update investors and shareholders with respect to the information available on the Clinical Trial, which the Company has discussed in its periodic filings with the Securities and Exchange Commission, including its most recently filed Quarterly Report on Form 10-Q for the quarterly period ended June 30, 2016, as amended.

Item 9.01 Financial Statements and Exhibits

(d) Exhibits.

A list of exhibits that are furnished and filed as part of this report is set forth in the Exhibit Index, which is presented elsewhere in this document, and is incorporated herein by reference.

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.

Date: November 1, 2016

RESPIRERX PHARMACEUTICALS INC.
(Registrant)

By: */s/ James S. Manuso*
James S. Manuso
President and Chief Executive Officer

EXHIBIT INDEX

Exhibit Number	Exhibit Description
99.1	Final Progress Report, Grant Number 5UM1HL112856

GRANT NUMBER:	5UM1HL112856
PROJECT TITLE:	Cannabimimetic treatment of obstructive sleep apnea: A proof of concept trial
GRANTEE ORGANIZATION:	University of Illinois at Chicago
PROJECT PERIOD:	05/15/12 06/30/16
CONTACT PI:	David W. Carley

FINAL PROGRESS REPORT

Original Aims of the Project and Progress Toward These Aims

Overview

Sleep related breathing disorders (SRBD), especially obstructive sleep apnea (OSA), represent an important health problem, conferring substantial cognitive/behavioral symptoms and increased risk of motor vehicle accident, hypertension, myocardial infarction, stroke, diabetes and death on at least 3% of the US population. Identifying novel treatments for OSA would be of great public health significance, because fully effective and acceptable OSA treatments are lacking. A critical need remains for NIH supported, mechanistically driven proof-of-concept clinical studies to evaluate novel therapeutic strategies. Despite basic research advances regarding the pathogenesis of OSA, generally effective drug treatments have not been identified. Based on our animal and preliminary human data, we propose to test the innovative hypothesis that cannabimimetic drugs are both effective in reducing sleep apnea severity and disease modifying in protecting against cardiovascular and neurological sequelae of OSA. Peripherally, afferent vagal activation increases apnea propensity by reducing upper airway muscle activation and by destabilizing respiratory pattern. We hypothesize that cannabimimetic drugs blunt vagal activity via CB1 receptors in the nodose ganglia, reducing apnea propensity. We previously showed that dronabinol, a direct CB1 and CB2 receptor agonist, reduced apnea severity in an animal model and we here present preliminary data supporting a similar effect in patients with OSA. Centrally, 5-HT and NA help to maintain activation of upper airway muscles, and release of these monoamines is reduced during sleep. Cannabimimetics increase synaptic concentrations of 5-HT and NA in the brainstem, and we hypothesize that this maintains greater upper airway muscle activity during sleep, again reducing apnea propensity. Apneas cause repetitive asphyxia and arousals, both of which are potential sources of hypertension and neurological compromise. We hypothesize that cannabimimetics are disease modifying: reducing sympathetic activation, lowering blood pressure and producing neuroprotection via reduced excitotoxicity. Project 1 will be a randomized, placebo-controlled parallel groups proof-of-concept clinical trial of dronabinol in patients with OSA. Subjects will be randomized to receive either placebo or dronabinol for a period of 6 weeks. The overarching goal will be to establish the safety, tolerability and therapeutic efficacy of dronabinol in OSA, and the co-primary efficacy endpoints will be: reduction in apnea/hypopnea index (AHI), subjective and objective improvement in daytime alertness at the end of treatment. Secondary endpoints will include improved oxygenation, sleep quality, blood pressure control and time-on-treatments effects for these endpoints. Project 2 will

employ anesthetized and chronically instrumented conscious behaving animals to directly test the mechanisms of dronabinol action schematized in figure 1. For example, we will characterize dronabinol's dose-dependent inhibition of afferent vagal reflexes elicited by pharmacological and mechanical stimuli. We will use specific CB1 and CB2 antagonists to confirm the receptor targets for reduction of apnea propensity and we will establish the central versus vagal-reflex impact of cannabimimetics on upper airway muscle activity during sleep. We will test the hypothesis that cannabimimetics lower blood pressure by reducing sympathetic activity. Taken together, these, projects will provide critical evidence regarding the potential efficacy and mechanisms of action for cannabimimetic treatment of OSA. By providing a path toward the first viable OSA pharmacotherapeutic, the proposed studies could have a tremendous impact on clinical practice.

Project 1

We will conduct a multi-center, randomized, double-blind, placebo-controlled proof-of-concept trial of dronabinol, a non-selective CB1 and CB2 receptor agonist. Subjects with OSA will be randomized to 1 of 3 treatment groups (N=40 per group): placebo, 2.5 mg/day dronabinol, or 10 mg/day dronabinol. Each subject will undergo a 2-week dose escalation phase followed by a 4-week treatment phase. Subjects in the placebo or 2.5 mg/day groups will receive placebo or 2.5 mg/day dronabinol, respectively, throughout the dose escalation and treatment phases. Subjects randomized to the 10 mg/day group will receive 2.5 mg/day dronabinol for 7 days, followed by 5 mg/day for 7 days, followed by 10 mg/day throughout the 4-week treatment phase. All investigational drug-product will be over-encapsulated in unlabeled gelcaps (for blinding) and administered orally, 30 minutes before bedtime. By implementing the above paradigm, this project will accomplish 3 specific aims: Aim 1 will establish the safety and tolerability of repeated doses of dronabinol in patients with OSA, based on adverse events, medication utilization compliance, and patient-reported satisfaction with the treatment. Aim 2 will test the hypothesis that repeated doses of dronabinol reduce OSA severity, quantified by the AHI measured during laboratory polysomnography (PSG), as a primary endpoint. This Aim also will determine the ability of dronabinol to improve secondary endpoints including (for example) arterial oxygen saturation, sleep efficiency, arousal index, and limb movement index. We also will test the hypothesis that dronabinol increases the sleep-related dip in blood pressure, a physiologic response often lost in OSA. Aim 3 will test the hypothesis that repeated doses of dronabinol reduce objective and subjective symptoms and signs of OSA. As primary endpoints, we will assess overall improvement using the Clinician's Global Impression of Change (CGI-C), objective alertness using the Maintenance of Wakefulness Test (MWT) and subjective alertness using the Epworth Sleepiness Scale (ESS). We also will determine the impact of dronabinol on sustained attention and motor performance; quality of life; and diurnal blood pressure.

Project 1 Progress

In view of higher than anticipated screen failure rates, and with approval of the NIH program officer, we elected to continue recruiting and following new subjects until the very end of the project period 06/30/16. This had the desired effect of increasing the total number of subjects randomized into the protocol, but also pushed several essential activities into the post-funding period, including: final data querying, reconciliation and database lock; implementation of the statistical analysis plan; interpretation of results; and dissemination of findings in oral and peer-reviewed written forms. As of this report, data querying and reconciliation have been completed, the database has been locked and transmitted to the study statistician, and topline analyses have been conducted as specified in the written statistical analysis plan. These analyses have been

conducted in "blinded" fashion, with individual subjects coded only as "Group A", "Group B" or "Group C" for treatment. Treatment group assignment will remain blinded until we have completed all planned primary and secondary analyses. We anticipate submitting an abstract detailing at least the top-line findings in December 2016, for presentation at the *Sleep 2017* international conference of the Associated Professional Sleep Societies. We also anticipate that we will submit a peer-review manuscript detailing these findings in the Winter of 2017.

As summarized in the Inclusion Data Record for this project, we enrolled a total of 417 subjects into the protocol; meaning that 417 individuals provided written informed consent to participate. Of these 417, 81 (19.4%) individuals met all inclusion/exclusion criteria and were eligible to be randomized. Seventy-seven individuals agreed to continue and were randomized into the protocol. Although the study was designed to fully balance the three treatment groups (placebo, 2.5 mg/day dronabinol, 10 mg/day dronabinol) because the full planned number of randomized subjects was not achieved, slight imbalances are present in the final dataset: Group A, N=24 (31.2%), Group B, N=27 (35.0%), Group C, N=26 (33.8%). Of the 77 subjects randomized to treatment, 56 completed the full 6-week treatment protocol.

Baseline characteristics of the subjects randomized to treatment are summarized below.

Baseline	**N**	**Group A**	**Group B**	**Group C**	**p-value**
AHI, mean(SD)	74	27.55(12.56)	26.04(11.92)	23.93(9.60)	0.5509
ESS, mean(SD)	75	10.09(3.45)	12.78(3.78)	11.35(3.94)	0.0486
MWT, mean(SD)	77	20.14(14.30)	14.99(9.40)	21.83(13.36)	0.1213
Treat Satisfaction (Placebo run-in)	74	22	27	25	0.298
Dissatisfied	13	5	6	2	
At all Satisfied	61	17	21	23	
Age, mean(SD)	77	51.86(9.18)	52.67(8.74)	55.27(9.89)	0.3982
Gender	77	24	27	26	0.6055
Male	56	19	18	19	
Female	21	5	9	7	
Race	77	24	27	26	0.0332
White	39	18	12	9	
Black or African American	34	6	13	15	
Asian	3	0	2	1	
Unknown	1	0	0	1	
Ethnicity	77	24	27	26	0.2073
Hispanic or Latino	10	5	1	4	
Not Hispanic or Latino	66	19	25	22	
Unknown	1	0	1	0	
BMI, mean(SD)	77	32.06(5.78)	33.45(4.85)	33.89(6.28)	0.4947
Systolic Pressure, mean(SD)	77	127.75(11.00)	129.89(14.96)	134.46(15.35)	0.2261
Diastolic Pressure, mean(SD)	77	80.71(9.56)	81.22(9.89)	85.23(11.78)	0.2449

In terms of biometrics, the subject population was middle aged, predominantly male non-hispanic white or African American individuals who were obese but normotensive (with or without medication). Age, gender distribution, ethnicity, body mass index (BMI), systolic blood pressure and diastolic blood pressure all were equivalent among the three treatment groups. Group A was somewhat skewed toward white versus African American subjects in comparison to Groups B and C. Regarding primary outcome measures, subjects had moderate to severe obstructive sleep apnea (as required by study inclusion criteria), and clinically significant subjective (Epworth Sleepiness Scale; ESS) and objective (Maintenance of Wakefulness Test; MWT sleep latency) sleepiness. Apnea-hypopnea index (AHI) and MWT were equivalent among all treatment groups. Small and not clinically significant differences in pretreatment ESS were observed between the treatment groups, with group B exhibiting the greatest subjective sleepiness.

Topline analysis of primary outcome measures for subjects who completed participation per protocol is presented below:

Outcome	N	Group A	Group B	Group C	p-value
6-week change in AHI, mean(SD)	56	-1.71(11.74)	-5.21(9.52)	7.99(13.16)	0.0032
6-week change in ESS, mean(SD)	54	-0.26(2.94)	-4.00(5.13)	-1.47(3.29)	0.0163
6-week change in MWT, mean(SD)	56	-3.70(9.73)	1.40(11.71)	-2.50(13.09)	0.3604
Treatment Satisfaction at Week 6	55	19	20	16	0.040
At all Dissatisfied	12	7	1	4	
At all Satisfied	43	12	19	12	

Three of the four primary outcome variables exhibited significant differences among treatment groups. The decrease in OSA severity (change from baseline in AHI) was significantly greater for Group B than either Group A or Group C. The greatest difference (-13.2/hr) was observed between Groups B and C. For patients with mild or moderate severity of OSA, this difference in treatment response is clinically meaningful. Based on ESS, all Groups tended to show increased subjective alertness, but this effect was again greatest for Group B and the inter-group differences were statistically significant and potentially clinically meaningful for individuals with moderate sleepiness as exhibited by the study subjects at baseline. Conversely, objective sleepiness did not improve and the treatment effect did not differ between groups, based on the MWT. Regarding treatment satisfaction, 95% of subjects in Group B expressed overall satisfaction with the treatment as compared to only 63% and 75% in Groups A and C, respectively ($p = 0.04$). Thus, overall 3 of the 4 primary outcome variables exhibited significant differences among treatment groups, with Group B demonstrating the greatest improvements in OSA severity and subjective daytime alertness as well as the greatest satisfaction with the treatment.

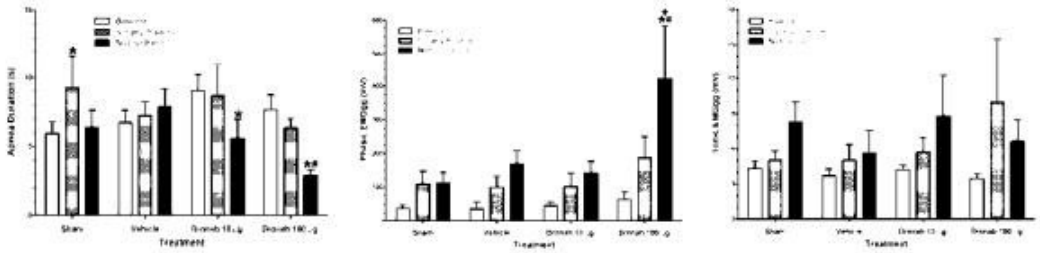
Project 2

Aim 1 will define the cannabinoid receptor-dependent and dose-dependent inhibition of afferent vagal reflexes in anesthetized rats. We will employ systemic and local nodose-ganglion injections of specific CB1 and CB2 receptor agonists and antagonists as well as infra-nodose and supra-nodose vagotomy to test the hypothesis that cannabimimetic inhibition of vagal afferent respiratory reflexes results from activation of CB receptors in the nodose ganglia. These same manipulations also will be used to define the receptor-, dose- and site-dependent cannabimimetic

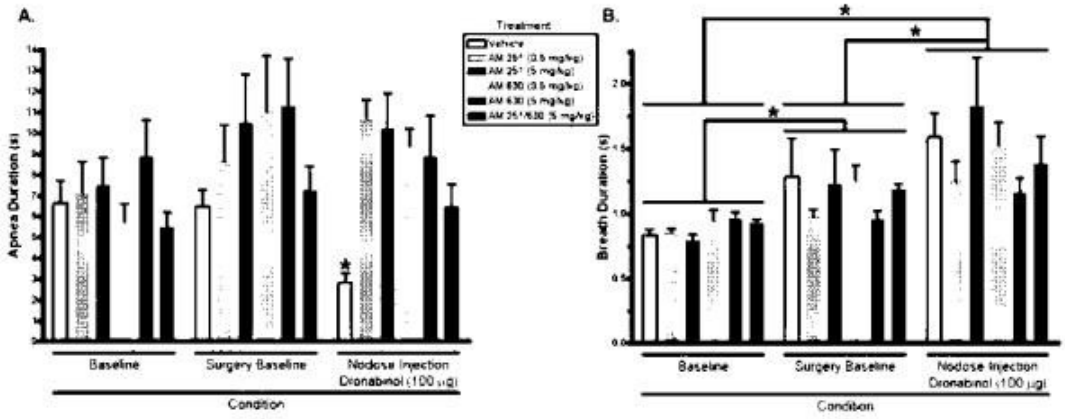
modulation of genioglossus electromyographic activity (EMGgg). The approach will allow us to differentiate CB1 versus CB2 effects mediated specifically by the nodose ganglia, as we hypothesize, versus central or other systemic influences. Aim 2 will test the hypothesis that cannabimimetic drugs act centrally to reduce blood pressure via reduced sympathetic outflow. Specifically, we will employ selective CB1 and CB2 receptor agonists and antagonists administered by systemic or intracerebroventricular injection to anesthetized rats to define the receptor-, dose- and site-dependent cannabimimetic impact on renal sympathetic nerve activity and systemic blood pressure. Aim 3 will employ chronically instrumented conscious animals to confirm the receptor targets for reduction of sleep apnea propensity by dronabinol. In combination with dronabinol, we will use specific CB1 and CB2 receptor antagonists administered by either systemic or intracerebroventricular injection to test the hypothesis that CB1 receptors both in the CNS and peripheral nervous system modulate apnea propensity. We will also establish the receptor and dose-dependent impact of dronabinol on EMGgg during wakefulness and sleep. These studies will be performed using a placebo-controlled repeated-measures crossover design in which each animal will serve as its own control.

Project 2 Progress

In order to test the hypothesis that dronabinol acts specifically within the nodose ganglia to inhibit vagally-mediated apnea (Specific Aim 1), we developed and published in the Journal of Visual Experimentation[1] (PMCID: 4354328) an innovative method to surgically dissect and locally inject the nodose ganglia. Using this method, we have completed placebo controlled studies confirming our *a prior* hypothesis. Intraganglionic injections of dronabinol at doses ranging from 10 µg (per ganglion) to 100 µg yielded a dose-dependent shortening of serotonin-induced apnea in anesthetized rats. In addition, 100 µg dronabinol injections significantly increased respiratory phasic activity of the genioglossus muscle – a key upper airway dilating muscle. These findings, published in Respiratory Physiology and Neurobiology[2] (PMCID: 3880550) are significant to the field because they support a plausible mechanism by which cannabimimetic pharmacotherapy may provide effective treatment for obstructive sleep apnea, an effect previously demonstrated in our small scale pilot study, and which is the focus of Project 1 of the present award.



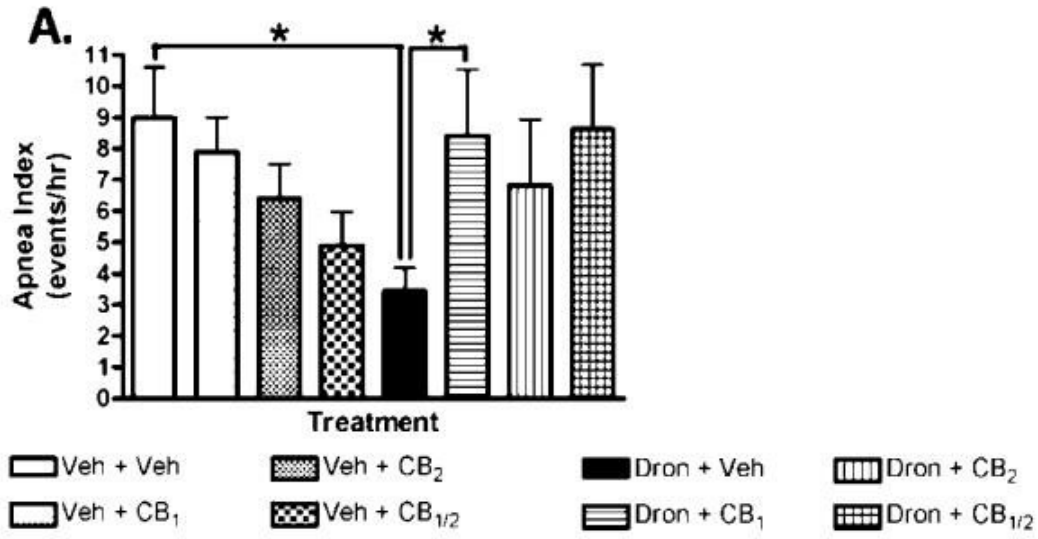
Utilizing pretreatment with selective antagonists, respectively, we extended the above findings to show that both CB1 and CB2 receptors contribute to the anti-apnea effect of dronabinol in the nodose ganglia of anesthetized rats. These findings are summarized in the figure below. Conversely, the CB antagonist drugs did not impair the potentiation of upper airway dilator muscle (genioglossus) activity by dronabinol. These findings, which further highlight the therapeutic potential of dronabinol and other cannabimimetics in obstructive sleep apnea were published in PLOS One[3] (PMCID: 4211887).



To evaluate the impact of cannabimimetic drugs acting in the central nervous system (Aim 2), we made stereotaxically guided injections into the cerebrospinal fluid of anesthetized rats. Here, we showed that in contrast to its effects when injected into the nodose ganglia, CNS administration of dronabinol had no ability to attenuate vagally-induced apneas, nor did it potentiate upper airway dilator muscle activity[4] (PMCID: 4852437). These results further refine our understanding of potential cannabimimetic mechanisms of action against apnea. They also have potential clinical implications in that developing a cannabimimetic drug that does not penetrate the blood brain barrier may preserve the therapeutic (anti-apnea) effects while dramatically reducing undesired psychogenic and appetite stimulating side effects.

Most recently, we have conducted studies of spontaneously occurring sleep-related apnea in chronically instrumented animals (Aim 3). Adult male Sprague-Dawley rats were anesthetized and implanted with bilateral stainless steel screws into the skull for electroencephalogram (EEG) recording and bilateral wire electrodes into the nuchal muscles for electromyogram (EMG) recording. Each animal was recorded by polysomnography on multiple occasions separated by at least 3 days. Using a fully nested, repeated measures crossover design, each rat was recorded following each of 8 intraperitoneal injections: vehicle; vehicle and CB_1 antagonist (AM 251); vehicle and CB_2 antagonist (AM 630); vehicle and CB_1/CB_2 antagonist; dronabinol; dronabinol and CB_1 antagonist; dronabinol and CB_2 antagonist; dronabinol and CB_1/CB_2 antagonist. As shown in the figure below, dronabinol suppressed spontaneous sleep-related apnea frequency, and this effect was reversed by pretreatment with a selective antagonist for CB1 receptors. Dronabinol also decreased the time spent in rapid eye movement sleep, an effect which was not altered by CB1 or CB2 receptor antagonists, alone or in combination (data not shown).

These results have been submitted for peer review by the journal *Sleep*, and a revised version of the manuscript is now under consideration.



Publications Resulting From the Project

1. Calik, MW, M Radulovacki, DW Carley. A method of nodose ganglia injection in Sprague-Dawley rat. *J Vis Exp*, 2014 Nov 25;(93):e52233. doi: 10.3791/52233. PMCID: 4354328
2. Calik, MW, M Radulovacki, DW Carley. Intranodose ganglion injections of dronabinol attenuate serotonin-induced apnea in Sprague-Dawley rat. *Resp Physiol Neurobiol*, 190:20-24, 2014. PMCID: 3880550
3. Calik, MW, DW Carley. Cannabinoid type 1 and type 2 receptor antagonists prevent attenuation of serotonin-induced reflex apneas by dronabinol in Sprague-Dawley rats. *PLOS ONE*, 9(10): p.e111412, 2014. PMCID: 4211887
4. Calik, MW, DW Carley. Intracerebroventricular injections of dronabinol, a cannabinoid receptor agonist, does not attenuate serotonin-induced apnea in Sprague-Dawley rats. *J Neg Res Biomed*, 2016; 15:8 doi: 10.1186/s12952-016-0052-1. PMCID: 4852437
5. Farabi, SS, B Prasad, L Quinn, DW Carley. Impact of dronabinol on quantitative electroencephalogram (qEEG) measures of sleep in obstructive sleep apnea syndrome. *J Clin Sleep Med*, 10:49-56, 2014. PMCID: 4242689