Xenon Pharmaceuticals Inc.
Form 10-K
March 12, 2015

UNITED STATES

SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 10-K

(Mark One)

x ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934
For the fiscal year ended December 31, 2014

or

¨TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934
For the transition period from to

Commission file number: 001-36687

XENON PHARMACEUTICALS INC.

(Exact Name of Registrant as Specified in its Charter)

Canada	98-0661854
(State or other jurisdiction of incorporation or organization)	(I.R.S. Employer Identification Number)

200 – 3650 Gilmore Way

Burnaby, British Columbia V5G 4W8

Canada

(Address of Principal Executive Offices, including zip code)

(Registrant's Telephone Number, Including Area Code): (604) 484-3300

Securities registered pursuant to Section 12(b) of the Act:

Title of Each Class	Name of Exchange on Which Registered
Common Shares, no par value per share	The NASDAQ Stock Market LLC
	(The NASDAQ Global Market)

Securities registered pursuant to Section 12(g) of the Act: None

Indicate by check mark if the registrant is a well-known seasoned issuer, as defined in Rule 405 of the Securities Act. Yes ¨ No x

Indicate by check mark if the registrant is not required to file reports pursuant to Section 13 or 15(d) of the Act. Yes ¨ No x

Indicate by check mark whether the registrant: (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days. Yes x No ¨

Indicate by check mark whether the registrant has submitted electronically and posted on its corporate Web site, if any, every Interactive Data File required to be submitted and posted pursuant to Rule 405 of Regulation S-T (§232.405 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit and post such files). Yes x No ¨

Indicate by check mark if disclosure of delinquent filers pursuant to Item 405 of Regulation S-K is not contained herein, and will not be contained, to the best of the registrant's knowledge, in definitive proxy or information statements incorporated by reference in Part III of this Form 10-K or any amendment to this Form 10-K. ¨

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer, a non-accelerated filer or a smaller reporting company. See definitions of "large accelerated filer", "accelerated filer" and "smaller reporting company" in Rule 12b-2 of the Exchange Act.:

Large accelerated filer ¨ Accelerated filer ¨

Non-accelerated filer x Smaller reporting company ¨

Indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-2 of the Securities Exchange Act of 1934). Yes ¨ No x

The aggregate market value of the voting and non-voting common shares held by non-affiliates of the registrant on November 5, 2014 (including common shares issued in the registrant's initial public offering), based on the closing price of $10.50 per share for the registrant's common shares as reported by The NASDAQ Global Market, was approximately $111 million. The registrant has elected to use November 5, 2014 as the calculation date, which was the initial trading date of the registrant's common shares on The NASDAQ Global Market, because on June 30, 2014 (the last business day of the registrant's most recently completed second fiscal quarter), the registrant was a privately-held company. Common shares held by each executive officer and director and by each person who owns 5% or more of the outstanding common shares, based on filings with the Securities and Exchange Commission, have been excluded from this computation since such persons may be deemed to be affiliates of the registrant. This determination of affiliate status is not necessarily a conclusive determination for other purposes.

The number of outstanding common shares of the registrant, no par value per share, as of March 9, 2015 was 14,221,600.

DOCUMENTS INCORPORATED BY REFERENCE

Portions of the registrant's definitive Proxy Statement to be filed with the Securities and Exchange Commission in connection with the registrant's 2015 Annual Meeting of Shareholders, which will be filed subsequent to the date hereof, are incorporated by reference into Part III of this Form 10 K. Such Proxy Statement will be filed with the Securities and Exchange Commission not later than 120 days following the end of the registrant's fiscal year ended December 31, 2014.

XENON PHARMACEUTICALS INC.

FORM 10-K

For the Fiscal Year Ended December 31, 2014

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PART I

Forward-Looking Statements

Certain statements contained in this Annual Report on Form 10-K may constitute forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended and Canadian Securities laws. The words or phrases "would be," "will allow," "intends to," "may," "believe," "plan," "will likely result," "are expected to," "will continue," "is anticipated," "estimate," "project," or similar expr or the negative of such words or phrases, are intended to identify "forward-looking statements." You should read these statements carefully because they discuss future expectations, contain projections of future results of operations or financial condition, or state other "forward-looking" information. These statements relate to our future plans, objectives, expectations, intentions and financial performance and the assumptions that underlie these statements. These forward-looking statements include, but are not limited to:

- our ability to identify additional products or product candidates using our Extreme Genetics discovery platform;
- the initiation, timing, cost, progress and success of our research and development programs, preclinical studies and clinical trials;
- our ability to advance product candidates into, and successfully complete, clinical trials;
- our ability to recruit sufficient numbers of patients for our future clinical trials for orphan or more common indications;
- our ability to achieve profitability;
- our ability to obtain funding for our operations, including research funding;
- our ability to receive milestones, royalties and sublicensing fees under our collaborations, and the timing of such payments;
- the implementation of our business model and strategic plans;
- our ability to develop and commercialize product candidates for orphan and niche indications independently;
- our commercialization, marketing and manufacturing capabilities and strategy;
- our ability to find families to support our Extreme Genetics discovery platform;
- our ability to discover genes and drug targets;
- our ability to protect our intellectual property and operate our business without infringing upon the intellectual property rights of others;
- our expectations regarding federal, state and foreign regulatory requirements;
- the therapeutic benefits, effectiveness and safety of our product candidates;
- the accuracy of our estimates of the size and characteristics of the markets that may be addressed by our products and product candidates;
- the rate and degree of market acceptance and clinical utility of Glybera and future products, if any;
- the timing of, and our and our collaborators' ability to obtain and maintain regulatory approvals for our product candidates;
- our ability to maintain and establish collaborations;
- our use of proceeds from our initial public offering and the concurrent private placement completed in November 2014;
- our expectations regarding market risk, including interest rate changes and foreign currency fluctuations;
- our belief in the sufficiency of our cash flows to meet our needs for at least the next 12 to 24 months;
- our ability to engage and retain the employees required to grow our business;
- our future financial performance and projected expenditures;
- developments relating to our competitors and our industry, including the success of competing therapies that are or become available; and
- estimates of our expenses, future revenue, capital requirements and our needs for additional financing.

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These forward-looking statements are subject to certain risks and uncertainties that could cause actual results to differ materially from those anticipated in the forward-looking statements. Factors that might cause such a difference include, but are not limited to, those discussed in this report in Part I, Item 1A — "Risk Factors," and elsewhere in this report. Forward-looking statements are based on our management's beliefs and assumptions and on information currently available to our management. These statements, like all statements in this report, speak only as of their date, and we undertake no obligation to update or revise these statements in light of future developments. In this report, "we," "our," "us," "Xenon," and "the Company" refer to Xenon Pharmaceuticals Inc. Unless otherwise noted, all dollar amounts in this report are expressed in United States dollars.

Item 1. Business

Overview

We are a clinical-stage biopharmaceutical company discovering and developing a pipeline of differentiated therapeutics for orphan indications that we intend to commercialize on our own, and for larger market indications that we intend to partner with global pharmaceutical companies. We have built a core enabling discovery platform for the discovery of validated drug targets by studying rare human diseases with extreme traits, including diseases caused by mutations in ion channels, known as channelopathies. We have an integrated platform that includes in-house capabilities for human genetics, small molecule drug discovery, as well as preclinical and clinical development.

Our business was founded on our proprietary discovery platform, which we refer to as Extreme Genetics. Extreme Genetics involves the study of families where individuals exhibit inherited severe traits, or phenotypes. By identifying and characterizing single-gene defects responsible for these phenotypes, we gain insights into human disease biology to better select targets for therapeutic intervention. Our Extreme Genetics discovery platform has yielded the first approved gene therapy product in the European Union, or the EU, a broad development pipeline and multiple pharmaceutical partnerships. We believe that our Extreme Genetics discovery platform enhances the likelihood of discovering a drug target that has a major effect in humans. From these discoveries, we can gain an improved understanding of how a drug that modulates the target might act when given to a human.

Our pharmaceutical partners include Teva Pharmaceutical Industries, Ltd., or Teva (through its subsidiary, Ivax International GmbH), Genentech, Inc., or Genentech, and Merck & Co., Inc., or Merck (through its affiliate, Essex Chemie AG). Our pharmaceutical collaborations have generated in aggregate over $150.0 million in non-equity funding to date with the potential to provide us with over $1.0 billion in future milestone payments, as well as royalties and co-promotion income on product sales.

To date, our Extreme Genetics discovery platform has yielded:

- Glybera, developed by our licensee uniQure Biopharma B.V., or uniQure, the first, and currently the only, gene therapy product approved in the EU for the treatment of the orphan disorder lipoprotein lipase deficiency, or LPLD. We believe that uniQure's commercialization partner, Chiesi Farmaceutici S.p.A., or Chiesi, plans to launch Glybera in the first quarter of 2015;
- TV-45070 (formerly XEN402), a product candidate with four Phase 2 proof-of-concept clinical trials completed. Our partner Teva is conducting a 300-patient, randomized Phase 2b clinical trial in osteoarthritis, or OA, of the knee, with data expected in the third quarter of 2015 and is planning a Phase 2b clinical trial in patients with postherpetic neuralgia, or PHN, with patient enrollment expected to begin in March 2015;
- GDC-0276, a product candidate being developed in collaboration with Genentech for the treatment of pain. In September 2014, Genentech initiated a Phase 1 clinical trial for GDC-0276. The Phase 1 clinical trial has recently been expanded and is expected to complete enrollment in the second half of 2015. GDC-0276 is a selective, oral Nav1.7 small-molecule inhibitor being developed for the treatment of pain; and

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proprietary preclinical programs including a sodium channel inhibitor for the orphan disorder Dravet Syndrome, or DS, and XEN801, a stearoyl Co-A desaturase, or SCD1, inhibitor for the treatment of acne. We anticipate filing an investigational new drug, or IND or IND equivalent application for XEN801 in the second quarter of 2015 and an IND for our DS program in 2016.

The selection of suitable families with rare phenotypes is integral to our successful identification of single-gene defects. Such families are rare and dispersed throughout the world, which makes accessing and studying such families a challenge. We have developed internal clinical genetics expertise allowing us to identify and access rare families. To date, we have established a global network that has included more than 30 clinical collaborations in multiple countries. We collect DNA and detailed clinical information from the selected families to which we then apply our in-house genetics, molecular biology and bioinformatics capabilities to identify the single-gene defect. Using these genetic insights, we apply our in-house, small-molecule expertise as well as access other therapeutic modalities, with the goal of developing novel medicines.

A significant focus of our Extreme Genetics discovery platform has been human channelopathies. This focus has enabled us to develop strong capabilities in small-molecule ion channel drug discovery. Our ion channel discovery capability is based on our understanding of the genetics of channelopathies combined with our proprietary biology and medicinal chemistry assets and know-how. We have been able to discover new binding sites on ion channels which, in turn, has led to the discovery of highly-selective voltage-gated ion channel inhibitors, which may have safety and efficacy advantages over non-selective inhibitors.

While the pharmaceutical industry has shown interest in channelopathies, a general inability to target ion channels selectively with a pharmaceutical agent has been a limitation to the development of effective therapeutics. The efficacy of non-selective ion channel inhibitors has generally been limited by the adverse events observed at high doses due to the broad non-selective binding of such agents. We believe we have developed a core competence in developing highly-selective small-molecule ion channel inhibitors, and we believe we can use this know-how to develop a pipeline of novel ion channel inhibitors for diseases in areas of high unmet medical need.

We discovered that deficiency of the voltage-gated sodium channel Nav1.7 is present in the rare human disease called congenital indifference to pain, or CIP. Individuals with CIP are unable to feel pain. This relationship indicated that Nav1.7 may be a key mechanism for the development of novel analgesics. We are pursuing this mechanism in separate partnerships with Teva and with Genentech.

Similarly, with our collaborators from McGill University, we identified the genetic link between rare human epilepsies and mutations in the Nav1.1 sodium channel. These genetic epilepsy discoveries helped to define our therapeutic selective ion channel strategy for Dravet Syndrome. We believe that our Extreme Genetics discovery platform provides the opportunity to validate additional ion channel targets for both prevalent and orphan indications.

We were incorporated in the Province of British Columbia on November 5, 1996 under the predecessor to the Business Corporations Act (British Columbia) under the name “Xenon Bioresearch Inc.” We continued from British Columbia to the federal jurisdiction pursuant to Section 187 of the Canada Business Corporations Act, or the CBCA, on May 17, 2000 and concurrently changed our name to “Xenon Genetics Inc.” We registered as an extra-provincial company in British Columbia on July 10, 2000 and changed our name to “Xenon Pharmaceuticals Inc.” on August 24, 2004. We have no subsidiaries. Our principal executive offices are located at 200 – 3650 Gilmore Way, Burnaby, British Columbia, Canada V5G 4W8, and our telephone number is (604) 484-3300. We are a reporting issuer in British Columbia, Alberta and Ontario, but our shares are not listed on any recognized Canadian stock exchange. Our common shares trade on The NASDAQ Global Market under the symbol “XENE.”

This Annual Report on Form 10-K includes our trademarks and registered trademarks, including the Xenon logo, “Extreme Genetics” and other trademarks or service marks of Xenon. Each other trademark, trade name or service mark appearing in this Annual Report on Form 10-K belongs to its holder.

Where You Can Find Additional Information

We make available free of charge through our investor relations website, http://www.xenon-pharma.com, our annual reports, quarterly reports, current reports, proxy statements and all amendments to those reports as soon as reasonably practicable after such material is electronically filed or furnished with the SEC. These reports may also be obtained without charge by contacting Investor Relations, Xenon Pharmaceuticals Inc., 200 – 3650 Gilmore Way, Burnaby, British Columbia, Canada V5G 4W8, e-mail: investors@xenon-pharma.com. Our Internet website and the information contained therein or incorporated therein are not intended to be incorporated into this Annual Report on Form 10-K. In addition, the public may read and copy any materials we file or furnish with the SEC at the SEC’s Public Reference Room at 100 F Street, N.E., Washington, D.C. 20549 or may obtain information on the operation of the Public Reference Room by calling the SEC at 1-800-SEC-0330. Moreover, the SEC maintains an Internet site that contains

reports, proxy and information statements, and other information regarding reports that we file or furnish electronically with them at www.sec.gov.

Our Pipeline

The following is a summary of our current product pipeline:

Approved Product

Glybera

Glybera is the first and currently the only gene therapy product to receive commercial approval in the EU. It is specifically indicated for the treatment of a subset of adult patients with the orphan lipid disorder lipoprotein lipase deficiency, or LPLD, confirmed by genetic testing and suffering from severe or multiple pancreatitis attacks, despite dietary fat restrictions. LPLD is a severe metabolic disease of inadequate lipid metabolism resulting in pancreatitis and in some cases, death. Together with collaborators from the University of British Columbia, or UBC, we demonstrated that humans with a single gene variant of the lipoprotein lipase, or LPL, gene called LPL^{S447X}, resulted in increased LPL enzyme activity leading to reduced triglyceride levels. Under our sublicense and research agreement with uniQure, we collaborated with uniQure and UBC on preclinical activities, and thereafter uniQure developed an LPL gene therapy product, Glybera, which contains the LPL^{S447X} variant. We believe that the introduction of the therapeutic LPL^{S447X} gene through administration of Glybera provides a clinical benefit for a subset of LPLD patients.

Glybera is the first product whose active ingredient was derived from our platform to receive commercial approval. The goal of Glybera therapy is to treat LPLD in order to achieve sustained improvement of clearance of triglyceride-rich lipid particles known as chylomicrons, and to significantly reduce the risk of pancreatitis attacks in patients suffering from multiple recurrent pancreatitis and abdominal pain events. Glybera was developed by our licensee, uniQure. In 2012, Glybera was approved in the EU, and in July 2013, uniQure announced that it had entered into a partnership with Chiesi Farmaceutici S.p.A., or Chiesi, for the commercialization of Glybera in the EU and more than a dozen other countries including Brazil, China, Mexico and Russia. We believe that Chiesi plans to launch Glybera in the first quarter of 2015 in Europe, and that uniQure is pursuing a U.S. product approval strategy, with plans to file a Biologics License Application, or BLA, with the U.S. Food and Drug Administration, or the FDA, following receipt of the results from a planned Phase 4 trial expected to begin in mid-2015. Glybera has received both fast track and orphan drug designations for the treatment of LPLD in both the EU and the U.S.

We are eligible to receive mid single-digit royalties on net sales of the licensed products, for sales made by uniQure and its affiliates. The royalty rates are reduced to a low single-digit for sales made by uniQure and its affiliates in countries where a licensed technology or a licensed product is not covered by a valid patent claim. With respect to uniQure's sublicense to Chiesi, we are eligible to receive a percentage in the low twenties of all non-royalty compensation relating to the licensed technology or products that uniQure receives from Chiesi (for example upfront payments and milestone payments), a percentage in the low twenties of any royalties that uniQure receives from Chiesi based on sales of technology or products covered by the licensed patents, plus a mid single-digit percentage of certain further royalties that uniQure receives from Chiesi based on sales of our licensed technology or products after the expiration of all licensed patents covering the product.

Product Candidates in Development

TV-45070 for the Treatment of Pain

TV-45070 (formerly XEN402) is a small-molecule inhibitor of the sodium channel Nav1.7 and other sodium channels, including those that are expressed in the pain-sensing peripheral nervous system. TV-45070 has potentially broad application in nociceptive pain, mediated by damage or injury to tissues, including the pain sensitivity caused by inflammation, and neuropathic pain mediated by damage, dysfunction or injury of nerves. TV-45070 is partnered with Teva. Pursuant to the terms of the agreement, Teva is obligated to complete three Phase 2 or later stage clinical trials. Using a topical ointment formulation of TV-45070, Teva has initiated a 300-patient, randomized Phase 2b clinical trial in OA of the knee with data expected in the third quarter of 2015. Teva is also developing topical TV-45070 in neuropathic pain indications, and is planning a Phase 2b clinical trial in patients with PHN with patient enrollment expected to begin in March 2015. We selected Nav1.7 as a drug target after we discovered that the Nav1.7 protein is deficient in the rare human disease called congenital indifference to pain, or CIP, where humans suffering from CIP are unable to feel pain. We have observed promising evidence of activity for TV-45070 in four Phase 2 proof-of-concept clinical trials, including two trials in the orphan disease erythromelalgia, or EM, one trial in PHN and one trial in dental pain, a form of nociceptive pain.

In December 2012, we entered into a collaborative development and license agreement with Teva, through its subsidiary, Ivax International GmbH, or Ivax, pursuant to which we granted Teva an exclusive worldwide license to develop and commercialize TV-45070. Prior to our entry into the collaborative development and license agreement with Teva, we submitted INDs to the FDA for oral TV-45070 for the indication of dental pain (July 2009) and topical TV-45070 for the indication of acute and chronic pain, including neuropathic and inflammatory pain (July 2010). Teva submitted an IND to the FDA for topical TV-45070 for the symptomatic treatment of OA (November 2013). Under the terms of the agreement, Teva made an upfront payment to us of $41.0 million. In addition, we are eligible to receive potential milestone payments totaling up to $335.0 million, comprised of a $20.0 million clinical milestone payment, up to $285.0 million in regulatory milestone payments, and a $30.0 million sales-based milestone payment.

If TV-45070 is approved, we are also eligible to receive royalties in the low teens to the low twenties on net sales of the licensed products for the timeframe that such products are covered by the licensed patents and in certain other instances. We also have an option to co-promote products in the U.S.

GDC-0276 and Other Selective Inhibitors of Nav1.7 for the Treatment of Pain

In December 2011, we entered into a collaborative research and license agreement with Genentech and its affiliate, F. Hoffmann-La Roche Ltd, or Roche, to discover and develop selective oral inhibitors of Nav1.7 for the treatment of pain. Based on our discovery of Nav1.7 deficiency underlying CIP, we believe that Nav1.7 is a highly-validated target for the treatment of pain. Our Genentech collaboration is focused on discovering and developing selective oral Nav1.7 inhibitors, which is in contrast to our Teva partnership that is focused on developing a topical drug that targets a number of different sodium channels, including Nav1.7. The first small molecule, preclinical product candidate that was selected for development under our collaboration is GDC-0276. In September 2014, Genentech initiated a Phase 1 clinical trial for GDC-0276. The Phase 1 clinical trial has recently been expanded and is expected to complete enrollment in the second half of 2015. GDC-0276 is a selective, oral Nav1.7 small-molecule inhibitor being developed for the treatment of pain.

Under the terms of the agreement, Genentech paid us an upfront fee of $10.0 million, a $5.0 million milestone payment for the selection of GDC-0276 for development and an $8.0 million milestone payment upon the approval by Health Canada of the Clinical Trial Application, or CTA, for GDC-0276. We are also eligible to receive pre-commercial and commercial milestone payments with respect to the licensed products totaling up to an additional $613.0 million, comprised of up to $45.5 million in preclinical and clinical milestone payments, up to $387.5 million in regulatory milestone payments, and up to $180.0 million in sales-based milestone payments for multiple products and indications. In addition, we are also eligible to receive royalties based on net sales of the licensed products, which range from a mid single-digit percentage to ten percent for small-molecule inhibitors for the timeframe that such products are covered by the licensed patents and a low single-digit percentage thereafter, plus a low single-digit percentage for large-molecule inhibitors of Nav1.7.

Chronic pain conditions, such as severe cancer pain and neuropathic pain, are generally recognized as unmet medical needs providing potential commercial opportunities for a new oral pain drug. Currently available pain drugs often have either a lack of meaningful pain relief or dose-limiting side effects for many patients. An orally administered selective Nav1.7 inhibitor could present a novel mechanism for the treatment of moderate to severe pain as a single agent or in combination with existing analgesics that work through different mechanisms.

Product Candidates in Discovery

Selective Small-Molecule Sodium Channel Inhibitors for the Treatment of Dravet Syndrome

We are developing selective inhibitors of the voltage-gated sodium channel Nav1.6 as a treatment for the orphan disease Dravet Syndrome, or DS. We have developed considerable expertise in voltage-gated sodium channel biology and have accumulated significant experience in the development of selective sodium channel inhibitors. We are leveraging this expertise and chemistry know-how for the development of selective inhibitors of Nav1.6 for the treatment of DS.

DS is a severe form of childhood epilepsy that typically causes mental retardation and, in approximately 10% of cases, premature death before the age of 12 years. The frequency of DS in the U.S. has been estimated to be one in 20,000 to 40,000 births, which, when applied to U.S. federal census data, correlates to approximately 7,500 to 15,000 patients with DS in the U.S.

With our collaborators from McGill University, we identified the genetic link between rare human epilepsy and mutations in the Nav1.1 gene. It is now estimated that approximately 80% of DS cases are believed to be due to mutations in one copy of the Nav1.1 voltage-gated sodium channel that cause a partial loss of Nav1.1 function. Nav1.1 plays a critical role in the normal functioning of inhibitory pathways in the brain. The lack of fully functioning Nav1.1 and inhibitory pathways allows the brain excitatory pathways to be unopposed resulting in the severe seizures of DS. The brain excitatory pathways are preferentially mediated by the voltage-gated sodium channel Nav1.6, and therefore if we are able to selectively inhibit Nav1.6 with a small-molecule compound, we expect to taper this neuronal excitation and thereby treat DS. To further support inhibiting Nav1.6 as a potential therapeutic approach to treat DS, published data have shown that seizures and premature death observed in a DS mouse model can be corrected when these animals are bred with a Nav1.6 knockout mouse.

DS is one of the most resistant epilepsies to treatment. Some benefit has been reported for drugs that increase the activity of the inhibitory brain pathways such as benzodiazepines and Stiripentol, while non-selective sodium channel blockers such as lamotrigine are contraindicated as they may worsen seizures due to further inhibition of Nav1.1. Other intractable childhood seizures that have been associated with genetically-linked partial loss of function of Nav1.1 or gain of function of Nav1.6 may benefit from a selective inhibitor of Nav1.6 include intractable childhood epilepsy with generalized tonic-clonic seizures and sporadic infantile epileptic en