Profile 2006

Contents

Santhera Profile

- Santhera is a Swiss specialty pharmaceutical company focusing on the discovery, development and marketing of small-molecule pharmaceutical products for the treatment of severe neuromuscular diseases.
- Santhera's vision is to become a global market leader in the treatment of neuromuscular diseases offering therapies for a number of indications in this area of high unmet medical need, which includes many severe orphan indications with no current therapy.
- Santhera has two compounds, SNT-MC17 (INN: idebenone) and JP-1730 (INN: fipamezole), in four indications in preparation of filing for marketing approval, in Phase III or Phase II clinical development.
- Santhera's goal is to build a diversified product portfolio by leveraging the Company's in-house neuromuscular disease expertise to
  a) actively manage the current portfolio by identifying new neuromuscular indications for its current or other well-characterized compounds, where overlapping disease mechanisms and scientific rationales exist;
  b) selectively in-license or acquire qualified compounds within its area of therapeutic expertise.

Santhera Share

Listing	SWX Swiss Exchange (main segment)
Ticker symbol	SANN
Swiss securities number	2714864
ISIN	CH0027148649
Common code	026905214
Number of registered shares (par value CHF 1.00)	3,099,156
Free float	38.3%

Share prices[1)]	
Year-end (December 29, 2006)	CHF 91.00
4-month high (February 9, 2007)	CHF 130.00
4-month low (December 5, 2006)	CHF 84.50

Market capitalization[1)]	
Year-end (December 29, 2006)	CHF 282.0 million
4-month high (February 9, 2007)	CHF 402.9 million
4-month low (December 5, 2006)	CHF 261.9 million

1) Closing prices from November 3, 2006 (IPO) to February 28, 2007; source: SWX

Letter to the Shareholders

Dear Shareholders,

On behalf of the entire team at Santhera we are very pleased to present our first Annual Report as a public company.

2006 was Santhera's most successful year to date. We achieved some remarkable milestones, in particular the very positive results from our collaborative study with the US National Institutes of Health (NIH) in Friedreich's Ataxia (FRDA), the start of an alliance with our Finnish partner Oy Juvantia Pharma Ltd (Juvantia) for a clinical program in Dyskinesia in Parkinson's Disease (DPD), and our successful listing on the SWX Swiss Exchange (SWX) in November.

We are currently focusing our clinical development on two compounds: SNT-MC17 (INN: idebenone) and JP-1730 (INN: fipamezole). The most advanced of Santhera's development programs, featuring our lead compound SNT-MC17 for the treatment of FRDA, has reached the watershed between Phase III clinical testing and filing for regulatory approval. The results of our recently completed clinical study, which we conducted in collaboration with the NIH, exceeded our expectations. Originally planned as a trial to increase the safety database, particularly at higher doses, the study showed a remarkable improvement in the neurological parameters and activities of daily living scores of FRDA patients after they had been treated with intermediate and high doses of SNT-MC17 over a period of just six months. Having discussed these data with representatives from member states of the European Medicines Agency (EMEA) and the US Food and Drug Administration (FDA) in late 2006 and early 2007, respectively, we now believe that it will be possible to accelerate our development timelines for SNT-MC17 in FRDA in both the EU and the US.

In the EU, a new guideline governing clinical trials in small patient populations came into force in February 2007. This directive enables Santhera to file for marketing authorization approval in Europe for SNT-MC17 for the treatment of FRDA on the basis of the data from the NIH study. We currently expect that this marketing approval could be received around mid-2008, which would be approximately six months ahead of our original schedule. If everything goes according to our revised plan, our marketing partner for SNT-MC17 in FRDA, Takeda Pharmaceutical Company Ltd of Japan (Takeda), could then be in a position to launch our first product in Europe in the second half of 2008. This would be a great achievement for both Santhera and all FRDA patients in Europe who are in great need of the first approved treatment for this devastating disease. In parallel with filing early, we will amend and continue the ongoing Phase III clinical trial in Europe to collect additional safety and efficacy data, particularly for high doses, in a larger patient population.

Michael Lytton Klaus Schollmeier

In the US, where there is no regulation comparable to the new EU guideline, we will have to conduct a pivotal clinical trial. However, based on recent discussions with the FDA, we should be able to conduct a shorter trial with fewer patients than originally planned, which may speed up the development timeline in the US considerably. Study enrollment is expected to start by mid-2007.

Part of our strategy is to leverage our in-house expertise in neuromuscular diseases to expand the pipeline. In doing so, we exploit the full potential of existing compounds for multiple indications or pursue additional drug candidates based on well-established scientific and medical rationales. The alliance with Juvantia is one example of how Santhera is able to strengthen its pipeline through partnerships. Together, we will further develop JP-1730 for DPD. This indication is a very severe side effect which frequently occurs when Parkinson's patients are treated with standard L-dopa therapy over a period of several years. The compound has already shown clinical proof-of-concept in an earlier Phase IIa study conducted by Juvantia in the US in collaboration with the NIH. In a limited number of patients, the study reported a reduction of dyskinesia symptoms after treatment with L-dopa, as well as a prolongation of L-dopa's therapeutic effects in Parkinson's. Santhera and Juvantia aim to confirm the earlier study results in a larger number of Parkinson's patients. A Phase IIb study is planned to start recruiting patients in the second half of 2007. We are currently performing additional preclinical tests and developing an improved formulation. Reflecting our view that JP-1730 could be a viable treatment option for DPD, we have safeguarded the proprietary rights to the compound by means of a contractual option to acquire Juvantia at a future point in time.

Our success to date has encouraged us to remain committed to our strategy of focusing on the discovery, development and commercialization of small-molecule pharmaceutical products for the treatment of severe neuromuscular diseases with high unmet medical needs. Our achievements in the past and the potential of our business strategy to create further shareholder value led to an international group of highly regarded investors supporting us in another private financing round. This preceded our initial public offering (IPO) that was one of the largest in the European biotech industry in 2006. Santhera's shares enjoyed a successful start to trading on the SWX on November 3, 2006. The proceeds from these financing activities allow us to further develop our product pipeline and to start building a specialized marketing organization in the US.

We achieved many more major milestones in 2006, although they attracted less attention. We successfully closed the enrollment for our proof-of-concept trial with SNT-MC17 in Duchenne Muscular Dystrophy (DMD), the most common form of muscle dystrophy. Regulatory approval was also received for a proof-of-concept trial for the third indication for SNT-MC17, Leber's Hereditary Optic Neuropathy (LHON), a distressing mitochondrial disease of the retina which leads to blindness within a short period of time.

Throughout the year, the Santhera team has worked hard on all fronts to prepare the Company for the next stage of growth and sustainability. Without our dedicated staff we would not have achieved our key strategic goals in 2006 and would not have been able to maintain our strong performance. Last year we succeeded in attracting additional talent to the Company in specific areas while being able to retain and enhance our existing in-house expertise. Together we can make something special happen. It is in this spirit that our sincerest thanks go out to everyone who contributes day-by-day to our progress and ultimately enables Santhera to launch successful new therapies for the benefit of all our stakeholders and, in particular, people suffering from these neuromuscular diseases.

In this Annual Report we are proud to present three distinguished personalities who, despite their Friedreich's Ataxia, live a rich life and provide an inspiring example for other patients: Friederike Giotas, a mother of two children from Munich, Nicholas A Johnson, a senior mechanical engineer from Boston, and Nadine Langenberg, a dedicated young woman from the German town of Dahlenwarsleben. We would like to thank them for helping us to visualize our ultimate aim: the improved treatment of patients with a severe neuromuscular disease.

Sincerely,

Michael Lytton
Chairman

Klaus Schollmeier
Chief Executive Officer

A Day in the Life of ...

"A day in the life of ..." intends to convey an insight into the everyday lives of three very special people. They all suffer from Friedreich's Ataxia, a rare form of a degenerative neuromuscular disease. Day after day, they fight and win against the odds associated with Friedreich's Ataxia, some days more, some days less. Normal activities of daily life – speaking, eating, drinking or washing – pose a serious challenge. Our three protagonists have managed to adapt to their disease and live as much as possible a "normal life." And daily, they overcome the restrictions imposed by Friedreich's Ataxia. We at Santhera dedicate all our efforts to helping the Friedreich's Ataxia community, the patients and their carers, to live a better life of higher quality. People like Friederike Giotas, Nicholas A Johnson and Nadine Langenberg are our incentive to bring SNT-MC17 to patients as first treatment for Friedreich's Ataxia, in Europe hopefully in 2008 and soon afterwards in the US.

Friederike Giotas, 51

Until I was 11 I lived in Greece. Then I moved to Germany, to Munich to be precise. The fact that I married Apostolos, a Greek, is coincidence. That I gave birth to a daughter at the age of 18 was not. With hindsight, this was a blessing, as I am not sure whether later I would have had the energy to bring up children. Our daughter is now 33. After finishing high school she moved to Greece. She wanted to live in a warm climate. Our son is 28. Since completing his studies in America, he has worked in a bank and lives with us. Which is a blessing. Indeed, my life has been full of blessings. I am in a wheelchair, I am ill, but – I am alive. And enjoy every day. Giving up is not part of my program. Or self-pity either. I know that there are a lot of people in the world who are worse off than I am. Everyone has a cross to bear, everyone.

At first I thought that I was so tired because I had my daughter when I was so young and continued to work part-time after she was born. What do I mean by tired? At times I was ready to drop, had no more strength, no energy, and experienced what it meant to be exhausted. Then the point came when exhaustion could no longer adequately explain what was happening to me. At the time I was working as a sales assistant, and one day I simply could not go on. I could not do anything. I could not lift my arms to stock the shelves; I could not lift my legs to climb the ladder. Apostolos was in Greece at that time, doing his military service.

I went to my family doctor, who immediately sent me to a neurologist. It did not take long before I was told that I had Friedreich's Ataxia. I was 21 at the time. But it took another 10 full years before I knew the implications. No one told me anything about the disease, no one advised me not to become pregnant again, and no one had any answers to my questions about the future. I found some of them in a medical book that I got hold of by chance and, more recently, on the Internet.

Until seven yeas ago I was able to do the housekeeping myself. I washed, ironed, cleaned, cooked, did the shopping – it took me longer than others, but I was able to do it. Six years ago the wheelchair arrived.

Part of my morning ritual is putting on my makeup. Every day, without exception. My life includes going to the cinema, to the opera, going for a stroll, visiting museums, sitting in cafes and watching people. I like doing this most of all in summer, when the outdoor restaurants are open. If people look at me, I never look away. On the contrary, I look people in the eye, thus telling them without words that they should not feel sorry for me. It helps; I have never had a bad experience.

If someone told me today that I should have to die tomorrow, I would count it as a blessing that I could live until tomorrow. It is true: my glass is never half empty. It is always half full!

Nicholas A Johnson, 43

At 19 years old, I was diagnosed with the neuromuscular disease Friedreich's Ataxia. As a young teenager, I was an all-star athlete. I was a singles champion in tennis and an all-star shortstop for Little League Baseball. I truly enjoyed sports and looked forward to any kind of physical activity. As a young teenager, I thought I was invincible as only young people can. Then, at the age of 19, the future path of my life would change dramatically.

I was in the consulting room of a neurologist, and he asked me to perform several simple physical tasks. He asked me to walk in a straight line, snap my fingers as fast as I could, touch my nose with my index finger with my eyes closed, and several other tests. Then he left the consulting room, saying "I'll be back in 15 minutes." Imagining the wide range of possible diagnoses the doctor could come back with, that quarter hour turned out to be the longest 15 minutes of my life.

When the doctor returned to the consulting room, he looked into my eyes for several minutes, sat down and said: "Nick, you have a neuromuscular disease called Friedreich's Ataxia. This disease will make your life extremely difficult in many ways. By the age of 30 you will need a wheelchair for mobility." At the time, I was in total shock about what he had just said. I truly did not believe a single word of it. After telling me that my life would be very difficult and that I would be in a wheelchair soon, he simply turned to the door to leave and said, "I am sorry; have a nice day."

He was an excellent physician, and many of his predictions were spot on. Later, through genetic testing, it was scientifically proved that I did have Friedreich's Ataxia. I went from being a great athlete to running slowly, to using a cane to walk, to using a walker, and at the age of 30 I started using a wheelchair.

Now, I have learned to compensate for my physical limitations with mental toughness. Despite all the adversities I have faced, I have managed to become a senior mechanical engineer. I have over 20 years of professional experience and work on projects worldwide. My professional colleagues seek my technical knowledge on many projects. I have established a reputation for being an "Engineer's Engineer."

At all times, there is a fierce, roaring, red-hot fire blazing deep inside me. It is absolutely essential for me that I make every possible effort to fulfill my potential in all ways. Every day I consistently make a new attempt to "push the envelope." Most people have various challenges they have to deal with in life. In many ways, I consider myself fortunate for the challenges I have been able to overcome in the face of adversity. I consistently make an effort to give a thousand percent. There are days on which I feel too tired to move at all. When asked where I get my energy from, on many occasions I say I get it from my wife Susan. I give everything possible, and I usually get everything back and more.

Besides my professional work as a senior mechanical engineer, I also volunteer for the MDA, the Muscular Dystrophy Association, an American organization that helps people with neuromuscular diseases. I am also a member of the board of director's for the Friedreich's Ataxia Research Alliance (FARA). These two organizations have agreed to form a partnership in an effort to fight against Friedreich's Ataxia. There is truly a lot of work to do, and I fight with every fiber of my being: address groups of people about the effects of the disease, help organize fundraising events, write articles, etc. In 2004 I was presented with the "National Personal Achievement Award", from the Muscular Dystrophy Association, for my accomplishments and for the way in which I deal with my disease.

My wife, Susan, sometimes says that I work too much, that I should try to relax more. But this is not my preference. I want to do what I can to improve society, want to make future opportunities for physically challenged people possible, day after day.

I met Susan, a dental hygienist, in the dentist's chair. She is beautiful, intelligent and has a great sense of humor too. We had known each other two years when we got married in 2000. I once joked with her that she fell in love with my teeth. She said: "A little. But above all, Nick, I was fascinated by your inner being; call it soul!"

Nadine Langenberg, 25

Sand between my toes – a wonderful feeling. A feeling that I sometimes wake up with in the morning. From a dream that takes me back to a place I used to really like. As a child, as a teenager. Together with my family. On one of those absolutely beautiful beaches in Sardinia. During the holidays.

Whether it is going to be one of my good days or not becomes clear shortly after I get up, at breakfast. On good days my hands don't shake as much, I don't spill my coffee, and I don't drop the bread on the floor. Well – everybody, even healthy people, has phases in which they feel better and phases in which they feel worse.

Many of my former friends no longer get in touch with me; I think I know why. My physical degeneration, my slowly, but steadily growing dependence on a wheelchair, my feet that roll inwards, my back that has given me the posture of a question mark – they could deal with all of this. But then, as my words began to slur and my movements became increasingly uncontrolled, they thought that my mental faculties were slowly beginning to deteriorate. That is perhaps the worst thing about this disease, the fact that people think I am feeble-minded. However, Friedreich's Ataxia does not affect my thought processes, my being or my core.

The disease was diagnosed in December 1996 at the university clinic in Magdeburg. I was 15 at the time, and although I now had a name for what was robbing me of my youthful power, I didn't have a prognosis. What would happen to me: no one could or would tell me then. The only advice I got was not to learn a skilled trade, but one where I would be able to sit. I trained as a commercial representative. It was a long and arduous journey, but with an enormous effort and an iron will I achieved my goal.

That was in 2000. Three years later I was sitting in a wheelchair. I never found a job. After a long struggle I was awarded a pension that just about enables me to live in my own flat. A flat with wide doors, no doorsill between the living room and the deck, and above all I'm allowed to keep a dog. A Shih Tzu. It's a "she" and is called Lizy. She spends one-half of the week with my parents and the other half with us. With Danny and me.

Danny has been my boyfriend going on for six years now. I met him at rehabilitation. We are a good team; he helps me where he can.

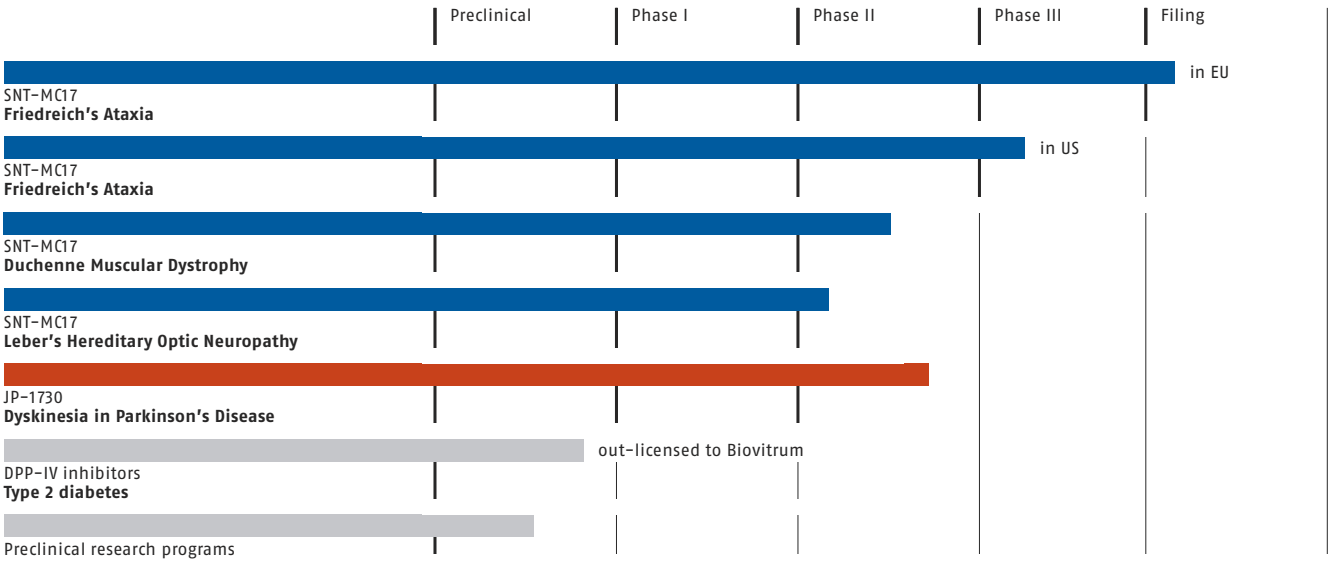
Three times a week I go to therapy; on the other days I spend most of my time one way or another with the disease.

I am the contact person for the "German Society for Hereditary Ataxia" (Deutsche Heredo-Ataxie-Gesellschaft) in Saxony-Anhalt. I also organize – in consultation with the others – our meetings. Everybody who is able to make it to the group comes. Many of the members of this self-help group are considerably worse off than I am. Despite this, I never wonder what will eventually become of me. Interestingly, I ignore the future. I live in the here and now; at least the disease has the advantage that I have to concentrate on the immediate present. Have to concentrate. That I should at some time again have the chance to experience such a moment on one of those absolutely gorgeous beaches on Sardinia, that is what I dream of. Sometimes even before I fall asleep.

Santhera's Product Pipeline

Santhera has developed a promising mid- to late-stage clinical pipeline of drug candidates focusing on the treatment of neuromuscular diseases and movement disorders. For the most advanced program the filing for market approval in Europe is currently in preparation.

Santhera's Product Pipeline



Products in clinical development

The Company's lead compound, SNT-MC17 (INN: idebenone), is currently being developed in three indications in which impaired mitochondrial function is a key contributor to the pathologies:

- Friedreich's Ataxia (FRDA);
- Duchenne Muscular Dystrophy (DMD);
- Leber's Hereditary Optic Neuropathy (LHON).

Based on promising data from a recent study conducted with the US National Institutes of Health (NIH), Santhera is currently preparing to file SNT-MC17 for the treatment of FRDA for marketing approval in Europe with a potential market launch in the second half of 2008. Meanwhile, the Company will continue its ongoing Phase III trial in Europe under an amended protocol. In the US, recruitment for a pivotal Phase III trial is expected to start in summer 2007.

Two Phase IIa trials are currently ongoing to obtain proof-of-concept for the efficacy of SNT-MC17 in two additional indications: DMD and LHON.

The Company's second clinical compound, JP-1730 (INN: fipamezole), is currently being prepared for a Phase IIb trial for the treatment of Dyskinesia in Parkinson's Disease (DPD). The study will be conducted in cooperation with Oy Juvantia Pharma Ltd of Finland (Juvantia), the compound's originator, in the US and potentially also in Europe, and is expected to start in the second half of 2007. The study is aimed at confirming efficacy data previously seen in a collaborative NIH/Juvantia trial.

Preclinical programs

Santhera's research and development is leveraging its expertise in neuromuscular disease for additional indications of existing compounds as well as identifying new candidates. Consequently, the Company's preclinical research programs focus on validated targets for which chemical templates or lead structures are available. To allow for rapid chemical optimization, Santhera employs state-of-the-art medicinal chemistry supported by in-house computational discovery skills and invivo biology, including disease-relevant models.

Santhera has several ongoing preclinical programs targeting neuromuscular indications. Furthermore, the Company has previously out-licensed a noncore preclinical program in metabolic diseases to Biovitrum AB of Sweden (Biovitrum).

Neuromuscular diseases

Neuromuscular diseases (NMDs) are characterized by high unmet medical need and usually affect relatively small patient populations, ranging from tens of thousands to only a few hundred patients worldwide. Over 200 distinct NMDs and movement disorders are known today, often characterized by devastating disabilities and leading to premature death. NMDs are mostly inherited, genetically determined disorders. People diagnosed with an NMD frequently suffer from progressive loss of muscle tissue, resulting in impaired movement control, loss of mobility and respiratory capacity as well as heart malfunction.

In many NMDs, well-organized advocacy groups gather patients and their families, as well as specialty physicians to act as central reference points and to offer comprehensive help and information about a specific disease.

Orphan drugs

Orphan drug legislation exists in both the US and the EU and is designed to encourage pharmaceutical companies to develop treatments for rare diseases or conditions. The FDA and the EMEA define orphan indications as affecting fewer than 200,000 individuals (US) or no more than 5 in 10,000 (EU). Products eligible for orphan drug status enjoy market exclusivity of up to 7 (US) and 10 (EU) years following the date of marketing approval. Additional incentives include tax credits, fee waivers for regulatory submissions and others.

Given their low prevalence, NMDs usually qualify as orphan diseases which means that most of Santhera's drug candidates can potentially benefit from these regulations. The Company has obtained orphan drug designation from health authorities in the US and EU for SNT-MC17 in all three indications (FRDA, LHON, DMD) currently in development.

Effective February 1, 2007, the EMEA adopted a guideline on clinical trials in small patient populations. Under this new regulation, even a single clinical trial with limited data can justify for filing and subsequent market approval if collectively all data look compelling. Based on the positive data from the recent study conducted in collaboration with the NIH, Santhera is taking advantage of this regulation for its early filing of SNT-MC17 in FRDA for marketing approval in the EU.

SNT-MC17 (INN: idebenone)

Mode of action

Idebenone is a small molecule optimized to facilitate the transport of electrons within mitochondria and to contribute to maintaining correct electron balance, which is necessary for the production of cellular energy.

Impairment of mitochondrial function can result from genetic mutations affecting the assembly of protein complexes of the electron transport chain in mitochondria, such as in FRDA and LHON. In addition, mitochondrial dysfunction can also occur secondarily, as in DMD.

Regardless of the primary origin, misdirected electron flux along the electron transport chain results in increased levels of cell-damaging reactive oxygen species and reduced proton flux and cellular energy production. Under conditions where energy output is impaired, idebenone can facilitate electron flux along the electron transport chain, decrease levels of reactive oxygen species, and increase proton flux and energy production. Overall, by stabilizing energy output and mitochondrial function, idebenone can support cell survival in particular for cells with high energy demand, such as muscle cells and nervous tissue. This biochemical function and resulting cellular survival is the underlying mechanism of action for the therapeutic benefit in various mitochondrial diseases, such as FRDA, DMD and LHON.

Nerve and muscle cells, including heart-muscle cells, are particularly energy-demanding and are, therefore, more prone to rapid cell damage or death due to mitochondrial dysfunction. Through preserving mitochondrial function and protecting cells from oxidative stress, it is believed that idebenone can prevent cell damage and increase the production of energy within impaired nerve and muscle tissue in FRDA and DMD patients. These properties could also be of therapeutic value for LHON patients whose retinal cells are damaged as a consequence of mitochondrial gene mutations, leading to blindness.

Friedreich's Ataxia

Friedreich's Ataxia (FRDA) is a rare but severe neuromuscular disease that results in the degeneration of an individual's nerve and muscle tissue, leading to severe impairment of movements and immobility. FRDA affects both Caucasian males and females equally. It is a chronic disorder and requires lifelong treatment. Average life expectancy for patients with FRDA is limited.

Causes

FRDA is an inherited disease caused by mutations in the gene encoding for the protein frataxin. This protein is essential for proper functioning of the mitochondria (the energy-production centers of a cell). Insufficient levels of frataxin affect the electron flux along the electron-transport chain of mitochondria, which in turn negatively impacts nerve and muscle tissues, as these cells are particularly energy demanding.

Diagnosis

Doctors usually diagnose FRDA by performing a careful clinical examination, which includes a medical history and a thorough physical examination. Additional tests that may be performed include an electromyogram, nerve conduction studies, an electrocardiogram, an echocardiogram as well as genetic testing to identify the affected gene. A genetic test which is carried out in certain specialized laboratories provides a molecular diagnosis.

Signs and symptoms

Disease symptoms may include any of the following:

- loss of motor coordination, impaired fine motor coordination;
- unsteady movements and walking;
- muscle weakness;
- vision impairment, hearing loss, and slurred speech;
- scoliosis (curvature of the spine);
- enlarged heart (hypertrophic cardiomyopathy).

Symptoms usually begin between the ages of 5 and 15 years, but can appear as early as 18 months or as late as 50 years of age.

Prognosis

Generally, within 10 to 20 years after the appearance of the first symptoms, patients with FRDA are confined to a wheelchair, and in later stages patients become completely incapacitated. Life threatening complications can occur in patients who develop cardiomyopathy, a cardiac complication that is frequently associated with FRDA.

Treatment

Current treatments are focused on support therapies, such as walking aids, wheelchairs, physical and speech therapy, and corrective surgery, as well as psychological support. At present, there are no specifically developed and approved pharmacological therapies available for the treatment of FRDA.

Clinical development

A recent clinical trial conducted in collaboration with the NIH has shown improvement in neurological parameters and activities of daily living scores of FRDA patients after treatment with SNT-MC17 for six months at daily doses of 900 mg and 2250 mg for adult patients. The results of the study were presented at the 3rd International Friedreich's Ataxia Scientific Conference in Bethesda, Maryland, US, in November 2006.

Based on the positive data from this trial together with the previously published data about the therapeutic effect on cardiomyopathy, Santhera intends to file for a marketing authorization approval for SNT-MC17 in FRDA in Europe in summer 2007. This filing, which will be possible under a new EU guideline for development of drugs to treat diseases in small patient populations, could lead to the product launch in Europe in the second half of 2008 by Santhera's marketing partner Takeda Pharmaceuticals of Japan (Takeda). The ongoing Phase III trial in Europe will be continued to collect additional safety and efficacy data, particularly for the high dose in a wider population of FRDA patients.

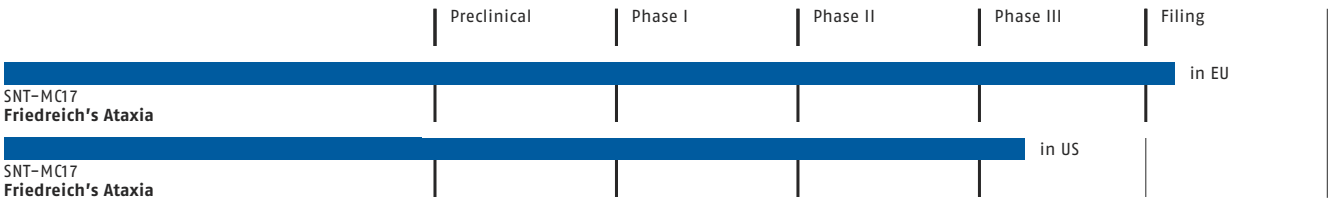
In the US, Santhera has submitted a clinical trial protocol to the FDA under its open IND (Investigational New Drug) and has asked for a Special Protocol Assessment. The design of this clinical trial reflects the major findings from the collaborative NIH trial regarding neurological endpoints and effective doses. Patient recruitment is expected to start in summer 2007.

Development and commercialization strategy

Santhera was granted orphan drug designations both by the EMEA and the FDA for SNT-MC17 for the treatment of FRDA. Upon successful registration and marketing clearance in Europe, SNT-MC17 will be distributed by Santhera's marketing partner Takeda. In the US, pending FDA approval, Santhera will sell SNT-MC17 through its own specialty marketing and sales organization.

Market opportunity

Published epidemiologic studies estimate that up to 20,000 people in Europe and North America suffer from FRDA. Based on the disease severity, prevalence, and the expected reimbursement level for pharmaceuticals that could reverse or slow the progression of FRDA, Santhera estimates the current market potential for such products to be approximately EUR 300 million in annual sales.



Duchenne Muscular Dystrophy

Duchenne Muscular Dystrophy (DMD) is the most common and devastating type of muscular degeneration and results in rapidly progressive muscle weakness. DMD affects males of all ethnicities worldwide. It is a chronic and disabling disorder which requires lifelong treatment. The average age of onset is between 3 and 5 years of age, with an average life expectancy of 30 to 35 years.

Causes

DMD is an X-linked recessive inherited disease, caused by mutations in the gene that encodes the protein dystrophin. Dystrophin is a mechanical linker between the contracting elements and cell surface proteins in each muscle cell thereby stabilizing the muscle cells during cycles of contraction and relaxation.

Diagnosis

Doctors usually diagnose DMD by carefully examining posture and gait, as the disease will have an effect on the way the affected boy stands, walks and runs, especially uphill or on steps. The diagnosis is usually confirmed by muscle biopsy, and some doctors also recommend electromyography.

Signs and symptoms

DMD is characterized by progressive muscle weakness and wasting throughout the body. Disease symptoms include:

- muscle weakness, initially in the legs and pelvis and spreading to shoulders and neck muscles, followed by arm muscles;
- muscle and skeletal deformities, including spinal deformation;
- respiratory failure and cardiac complications, both of which can be life threatening.

Prognosis

With disease progression, people with DMD suffer from severe medical conditions, and are often confined to a wheelchair during their teenage years. DMD results in muscle and skeletal deformities, including spinal deformation, and eventually leads to respiratory failure and cardiac complications that result in premature death.

Treatment

There is no effective treatment for DMD. Current treatments are focused on delaying or alleviating the disease's symptoms and include physical and occupational therapy, and the use of orthopedic devices such as walking aids, wheelchairs and ventilator support to prevent respiratory failure in the advanced stages of the disease. Pharmacological treatment with corticosteroids may delay disease progression and prolong mobility, but the significant adverse effects of chronic steroid use have prevented wide acceptance of this therapy.

Clinical development

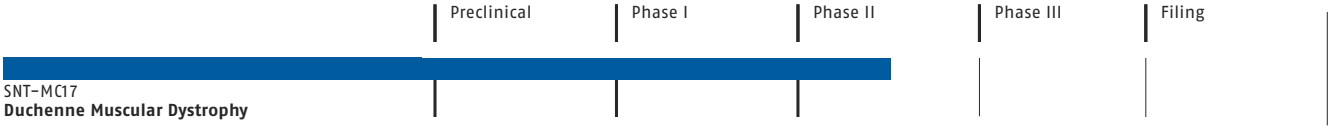
A single-center Phase IIa trial to establish the proof-of-concept for SNT-MC17 as a treatment of dilated cardiomyopathy and muscle weakness in DMD is running at the University of Leuven, Belgium. The study is a 12-month double-blind, randomized, placebo-controlled trial to assess the efficacy and tolerability of one dose level of SNT-MC17 compared to placebo in 8- to 16-year-old boys with DMD and early cardiac dysfunction. The primary endpoint of this trial is an assessment of the change in contractility of the heart muscle, which is an early predictor of cardiac failure in DMD. Secondary endpoints include the effects of SNT-MC17 on muscle strength. A total of 21 patients have been enrolled into this study: 14 patients receive SNT-MC17, while 7 receive placebo. Results from this trial are expected in the second half of 2007.

Development and commercialization strategy

Subject to a positive outcome of the Phase IIa trial, Santhera intends to seek protocol advice from the EMEA and FDA in order to prepare for a pivotal Phase III program. Santhera has already received orphan drug designation for SNT-MC17 in DMD in both the EU and the US. Upon marketing approval, Santhera will sell SNT-MC17 for DMD in the US on its own and through a partner in other markets.

Market opportunity

Based on published epidemiologic studies, an estimated 30,000 males suffer from the disease in the EU and the US. The market for pharmaceutical products that could treat DMD is estimated to be approximately EUR 400 million per annum, based on disease prevalence, severity and expected reimbursement levels.



Leber's Hereditary Optic Neuropathy

Leber's Hereditary Optic Neuropathy (LHON) is an inherited mitochondrial disease that results in the degeneration of nerve cells in the retina, leading to the rapid loss of central vision and blindness. LHON predominantly affects young adult men.

Causes

LHON is caused by mutations of the genetic code within the mitochondria, the energy-production centers in each cell, and is transmitted through the mother. The mutations ultimately lead to the reduction of cellular energy production, resulting in cell damage and death of optic nerve cells. The factor(s) determining why only optic nerve cells become affected is (are) still unclear.

Diagnosis

The diagnosis of LHON requires a neuro-ophthalmological evaluation and/or blood tests for DNA assessment available in specialized laboratories and hospitals.

Sign and symptoms

The symptomatic phase of LHON disease begins with the blurring of central vision. The effects are rapid and severe, with the damage to retina cells leading to blindness within a few months after the onset of symptoms.

Prognosis

After the initial symptoms appear, both eyes are usually affected within several months, leading to rapid loss of central vision and blindness. Within approximately 12 months of visual loss in one eye, over 97% of patients will experience vision loss in the second eye.

Treatment

There is no effective treatment for this disease.

Clinical development

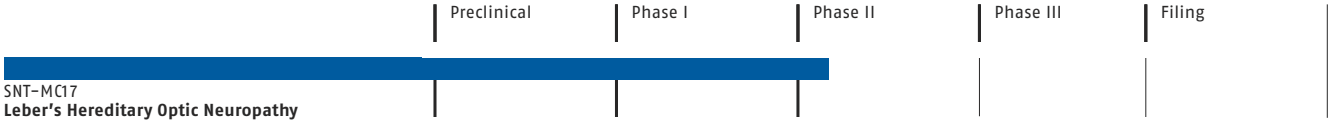
Santhera is currently enrolling patients in its Phase IIa proof-of-concept trial in Newcastle, UK, and Munich, Germany. The study, which is a double-blind, randomized and placebo-controlled trial, is designed to assess the efficacy of SNT-MC17 on the progression of vision loss in symptomatic LHON patients. Up to 60 LHON patients will be recruited for the study, and they will be treated for a period of nine months. A special statistical protocol allows for interim analysis after 12, 24, 36 and 48 patients have completed the trial.

Development and commercialization strategy

Following this trial, and subject to positive results, Santhera plans to initiate a Phase III program in the EU and the US. Santhera has received orphan drug designation for SNT-MC17 in the treatment of LHON in the EU and the US. Upon marketing approval for LHON, Santhera will sell SNT-MC17 itself in the US and through a partner in other markets.

Market opportunity

The prevalence of LHON was estimated in a recent study to be 3 per 100,000 individuals and is thus comparable in prevalence to inherited neuromuscular diseases such as DMD. As a genetic disease with well-known family histories, there is a potential that not only will LHON patients showing symptoms be users of a treatment, but that carriers may also take the product for disease prevention. The market for pharmaceutical products that could treat and ameliorate LHON is estimated by Santhera to be broadly comparable to that of DMD, based on estimated disease prevalence, severity and expected reimbursement levels.



SNT-MC17
Leber's Hereditary Optic Neuropathy

JP-1730 (INN: fipamezole)

Mode of action

Fipamezole is an antagonist of the adrenergic alpha-2 receptor and offers a novel and unique mode of action to treat DPD. The rationale behind the development of fipamezole is to increase noradrenergic release in certain areas of the brain, resulting in a rebalancing of the distorted brain network and alleviating symptoms of advanced Parkinson's disease (PD) such as dyskinesias, motor fluctuations and cognitive impairment. In addition, fipamezole is believed to extend the beneficial effects of commonly used levodopa (prolonged on-time) and other dopamine agonists without the negative side effects associated with these treatments. Such therapy is expected to improve the quality of life of Parkinson's patients.

Dyskinesia in Parkinson's Disease

Parkinson's disease (PD) is the second most common neurodegenerative disease, and affects movement as well as disorders of mood, behavior, thinking and sensation. Dyskinesia refers to an impairment of voluntary movement. Dyskinesia in Parkinson's Disease (DPD) result from chronic therapy, such as levodopa, the current standard treatment for PD.

Causes

The initial mechanisms responsible for levodopa-induced dyskinesia are not entirely clear, but appear to be related to widely varying blood levels of levodopa-derived L-dopa and possibly to storage and release of dopamine in brain areas where it should not normally occur. In PD, dopamine-producing neurons gradually die in an area called the substantia nigra, causing a shortage of dopamine. Dopamine helps to relay messages between areas of the brain that control body movement.

Diagnosis

DPD is characterized by typical chaotic movements occurring during treatment with levodopa.

Signs and symptoms

DPD is characterized by jerky involuntary movements of body, upper and lower limbs, tongue and head, typically during the peak on-time of the levodopa effect. These chaotic movements can become troublesome for both patients and caregivers.

Prognosis

Dyskinesia appears at the advanced stages of PD, usually after 3 to 7 years of levodopa treatment, and worsens over time. Early-onset Parkinson's patients are generally more prone to develop dyskinesia.

Treatment

There is no pharmaceutical therapy specifically approved for the treatment of DPD.

Clinical development

In 2006, Santhera and Juvantia entered into a strategic cooperation to advance the development of Juvantia's compound JP-1730 for the treatment of patients suffering from DPD. Santhera intends to fund a Phase IIb double-blind, placebo-controlled trial with JP-1730 in the US in 2007. The trial will further assess the compound's efficacy in the treatment of troublesome dyskinesias in a larger cohort of patients than the previous Phase IIa clinical trial conducted in the US by Juvantia in collaboration with the NIH. JP-1730 is also expected to prolong the action of levodopa, thereby having the potential to extend the patients' "quality on-time" and resulting in a clear benefit in quality of life.

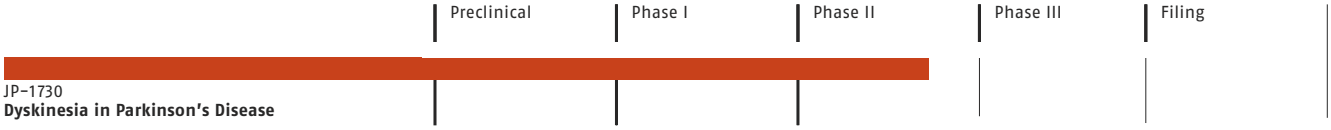
The intended trial will be a one-month efficacy trial taking advantage of Juvantia's open IND in the US. The previous Phase IIa clinical trial, which enrolled 21 patients, was a placebo-controlled, double-blind study and tested three different doses of JP-1730. The trial established clinical evidence that the compound has potential for the treatment of levodopa-induced DPD.

Development and commercialization strategy

Santhera will be responsible for conducting and funding development work of the Phase IIb clinical trial with the assistance of Juvantia. Santhera has a call option to acquire Juvantia by the end of 2008 after analyzing the data from the Phase IIb trial. A fast-track designation status has been granted by the FDA for JP-1730 in DPD.

Market opportunity

It is estimated that approximately 20% of all Parkinson's patients develop troublesome DPD within five years of initiating levodopa treatment. This represents approximately 200,000 DPD sufferers worldwide. The market for pharmaceutical products that could treat DPD is estimated to be approximately EUR 500 million per annum, based on disease prevalence, severity and expected reimbursement levels.



JP-1730
Dyskinesia in Parkinson's Disease

Management Discussion and Analysis

Highlights in 2006

February
Completion of patient enrollment for six-month Phase II clinical trial of SNT-MC17 in FRDA in collaboration with the NIH.

June
Transfer of German operations from Heidelberg to new premises in Liestal, Switzerland, completed.

July
Signing of collaboration agreement with Juvantia to advance development of JP-1730 in DPD with option to acquire Juvantia.

Completion of patient recruitment for twelve-month Phase IIa trial in DMD aimed at investigating the efficacy of SNT-MC17 in improving cardiac function and muscle strength.

September
Approval from UK and German regulatory authorities for a Phase IIa trial to obtain proof of concept that SNT-MC17 prevents or slows the progression of vision loss in LHON.

October
Santhera raises CHF 15.8 million in a private C financing round.

Preliminary results from collaborative NIH study show positive effects of SNT-MC17 in FRDA on neurological parameters and activities of daily living.

November
Successful completion of IPO at CHF 90.00 per share and first day of trading on the SWX.

Deutsche Bank, Santhera's global coordinator and lead manager for its IPO, fully exercises the over-allotment option (greenshoe).

Management Discussion and Analysis

Successful year for Santhera

2006 was a very busy and successful year for Santhera. Our development projects made significant progress, and we were able to publish positive results from our collaborative study with the NIH of SNT-MC17 in FRDA. Patient recruitment was successfully completed for a proof-of-concept study of our lead compound in DMD, and a clinical program was initiated in a third indication, LHON. On the partnering side, we entered a collaboration with Juvantia for a clinical program in DPD. As a result of another round of private financing and our IPO we were able to secure additional funding for the future successful development of our research and development pipeline.

Strong track record in financing with IPO and listing on the SWX

Since its inception in 2004, Santhera raised gross proceeds of CHF 87 million in three private financing rounds. From the beginning, the group of private equity investors of the predecessor companies participated in these private financings, demonstrating their confidence in the Company's business model and in its management. In October 2006, a series of new investors joined Santhera's base of shareholders in its pre-IPO financing round, raising another CHF 15.8 million with an option for an additional CHF 7.9 million in case no IPO could take place within a reasonable period of time. Additionally, in connection with the preceding round of financing in December 2005, the Company issued warrants that were converted in conjunction with the IPO.

On October 23, 2006, Santhera announced its intention to go public and offered 1,131,438 newly issued shares (including greenshoe shares) in an IPO in Switzerland and in an international private placement in the EU and the US to Qualified Institutional Buyers under Rule 144A. The offering was received by a strong demand from high-quality international investors and was oversubscribed. The shares were placed mainly with institutional investors in Switzerland, Germany, the US, the UK and Benelux at an offering price of CHF 90.00 per share valuing Santhera at CHF 279.0 million. The listing on the SWX Swiss Exchange took place on November 3, 2006, with an opening price of CHF 90.20. The successful IPO was underlined when Deutsche Bank, the Company's global coordinator, fully exercised its over-allotment option (greenshoe) of 147,579 shares at the offering price. Gross proceeds from the IPO, including proceeds from the greenshoe, amounted to CHF 101.8 million.

Since the listing and through February 28, 2007, there has been an average daily trading volume of 8,100 shares. The positive effects of the Company's engagement of two banks as market makers is reflected in a thin spread, usually of less than 1% between ask and bid.

Strong balance sheet with solid cash reserves at the end of 2006

With CHF 125.7 million cash and cash equivalents on hand at December 31, 2006, Santhera is well positioned to fund its current operations and strategic goals. This position reflects the cashburn and total funds raised in 2006. Santhera is investing these funds on the basis of investing guidelines to safeguard funds primarily for financing the Company's expanding pipeline and the planned setup of its specialized marketing and sales organization in the US.

Operating expenses focused on research and development

Santhera achieved revenues of CHF 0.8 million in 2006 primarily from research funding through a licensing agreement with Biovitrum. In 2005, Santhera received revenues from the two major partnerships with Takeda and Biovitrum in the amount of CHF 13.2 million.

Operating expenses amounted to CHF 29.4 million in 2006, an increase of CHF 4.1 million compared to 2005. These expenses were predominantly dedicated to research and development activities, accounting for CHF 18.0 million, or 61% of operating expenses. The increase in research and development expenses of CHF 3.5 million over the previous year reflects a shift from preclinical research to development, triggered by the clinical trial costs for SNT-MC17. These costs were for preparing and entering two Phase III trials in FRDA and two Phase II trials in two further indications, DMD and LHON.

General and administrative expenses of CHF 12.1 million in 2006 were significantly higher than in 2005 (CHF 6.0 million), mainly due to the costs of legal and other advisory services in connection with the private financing round, the process of going public, the negotiation of the Juvantia agreements and other business development activities. For the first time, Santhera also incurred marketing and sales expenses in the amount of CHF 0.3 million, currently reported as part of general and administrative expenses. Other operating income amounted to CHF 0.6 million compared with other operating expenses of CHF 4.7 million in 2005.

The total operating expenses in 2006 also include noncash-relevant expenses for share-based payments (nonvested stock options) in the amount of CHF 2.6 million and for warrants issued to investors of Juvantia, which accounted for CHF 0.9 million.

The cash-relevant operational key figure, the gross operating and investing cash flow, amounted to CHF –25.9 million in 2006 compared to CHF –22.5 million in 2005. This figure is based on the operating results, excluding noncash charges such as expenses for stock options, amortization and depreciation, issuance of warrants and net of gross profit.

Santhera is focusing on keeping its recurring general and administrative expenses to a minimum in order to support its investments in research and development, and in planned investments in its sale and marketing organization in the US. However, we have been investing in and are committed to corporate governance and risk management activities to ensure that they meet international standards. Our risk management is focused on the careful management of our cash burn and on minimizing our financial risks. The net financial result amounted to CHF 0.6 million in 2006, substantially higher than in 2005 (CHF –0.9 million), mainly due to higher interest income in 2006 and the issuance of warrants in connection with the financing round in 2005.

Over the course of 2006, the Company repaid loans from tbg Technologiebeteiligungsgesellschaft mbH of Germany (tbg), totaling CHF 2.6 million, substantially reducing our interest rate burden. The remaining outstanding sum under a second loan agreement with tbg amounts to CHF 1.4 million.

Cash-relevant incremental expenses directly attributable to equity financings are expensed through the equity statement and amounted to CHF 10.1 million in 2006 and CHF 0.3 million in 2005. The expenses in 2006 covered both the private financing round and the IPO.

Proven success in partnering

Santhera has a strong track record in executing its strategy by selectively partnering its marketing activities in certain markets, in adding development programs within its key expertise and in out-licensing noncore businesses.

In July 2006, Santhera entered into a cooperation agreement with Juvantia for the development of Juvantia's product candidate JP-1730 in DPD. This agreement is designed to generate the additional clinical data required prior to the commencement of pivotal clinical trials. In connection with this cooperation agreement, the Company and Juvantia's investors signed an option agreement that grants Santhera the right to purchase Juvantia if certain conditions are met. Santhera did not make any up-front payment in cash but instead granted an option premium through the issuance of 9,818 warrants to Juvantia's investors to acquire Santhera shares.

Under an agreement signed in July 2005, Santhera granted an exclusive license to Takeda to commercialize SNT-MC17 in Europe in the indication FRDA. Santhera will remain responsible for the development and registration of the product candidate. Takeda has paid EUR 5.0 million up-front, and Santhera is entitled to additional milestone payments of up to EUR 7.0 million and royalties once the product is marketed.

Also in July 2005, Santhera entered into a collaboration and license agreement with Biovitrum. Under this agreement, Biovitrum has been granted exclusive worldwide rights to Santhera's DPP-IV inhibitor program to select and develop compounds and commercialize future drugs for a range of metabolic diseases, including type 2 diabetes. Biovitrum paid Santhera an up-front licensing fee of EUR 3.0 million and provided Santhera with research funding of EUR 1.0 million. Upon initiation of Phase II clinical trials by Biovitrum, Santhera is entitled to a milestone payment in the amount of EUR 10.0 million. Santhera is entitled to additional milestone and royalty payments.

Outlook and financial management

Santhera's cashburn in 2007 is expected to increase compared to 2006, reflecting the successful development of our pipeline with SNT-MC17 in two Phase III and two Phase IIa trials, and JP-1730 in a Phase IIb trial. Cash will also be invested in ongoing preclinical research activities as well as in business development activities, with the goal of further growing the pipeline externally. In addition, by the end of 2007, the Company will begin to build up its marketing organization in the US.

As part of its business strategy, Santhera intends to focus on the North American markets while out-licensing marketing rights in Europe and other regions. As a result, milestone and up-front payments from such partnering agreements represent an important part of the Company's financing activities. First revenues from products can be achieved based on the planned launch of SNT-MC17 for FRDA in Europe by Takeda, expected in the second half of 2008.

Corporate Management

Executive Management




Klaus Schollmeier*, Chief Executive Officer
Since inception of Santhera in 2004
German citizen
PhD in biology, University of Dusseldorf, Germany. Over 16 years of experience in the pharmaceutical industry at BASF, Knoll and Abbott, with senior business and scientific management responsibilities.

Helmut Kessmann, Chief Business Officer
Since inception of Santhera in 2004
German citizen
PhD in biochemistry, University of Munster, Germany. Over 10 years of experience in biotech companies in senior business development and management positions, before in various positions in research at Ciba-Geigy (now Novartis).

Barbara Heller, Chief Financial Officer
Since 2005
Swiss citizen
MA (lic oec publ), University of Zurich, Switzerland. Over 15 years of experience in investment banking and corporate finance at Bank Vontobel and Bank Leu (CS Group) in various senior management positions.

Thomas Meier, Chief Scientific Officer
Since inception of Santhera in 2004
German citizen
PhD in biology and Lecturer in neurosciences, University of Basel, Switzerland. Distinguished scientific track record in the field of neuromuscular research; founder and CEO of MyoContract.

* Proposed to be appointed to the Board, subject to the approval by the Shareholders' Meeting, scheduled for April 23, 2007.

Board of Directors

Michael Lytton, Chairman
Since inception of Santhera in 2004
US citizen
JD, Harvard Law School, Massachusetts, US; MSc epidemiology and medical statistics, London School of Hygiene and Tropical Medicine, London, UK.
General partner of Oxford Bioscience Partners, Boston, Massachusetts, US.

Hans Peter Hasler, Vice Chairman
Since 2006
Swiss citizen
Federal Commercial Diploma, Berne, Switzerland; Marketing Manager Certificate, SIB, Switzerland.
Senior vice president and head of international business at Biogen Idec, Zoug, Switzerland.

Martin Gertsch
Since 2006
Swiss citizen
Swiss Certified Fiduciary; Swiss Certified Public Accountant.
CFO of ESBATech, Schlieren, Switzerland; former CFO of Straumann Group, Basel, Switzerland.

Rudolf Gygax
Since inception of Santhera in 2004
Swiss citizen
PhD in physical chemistry, University of Basel, Switzerland.
Managing director of Novartis Venture Fund, Basel, Switzerland.

Georg Nebgen
Since inception of Santhera in 2004
US citizen
PhD in pharmaceutical technology sciences, University of Bonn, Germany; MBA, University of St. Gallen, Switzerland.
Managing general partner and co-founder of NGN Capital, Boston, Massachusetts, US.

Timothy Rink
Since inception of Santhera in 2004
British citizen
MA, MD and ScD, University of Cambridge, UK.
Member of the scientific advisory board of Serono; former chairman and CEO of Aurora Biosciences.

Bernd Seizinger
Since inception of Santhera in 2004
German citizen
MD Ludwig Maximilians University; PhD, Max Planck Institute of Psychiatry, both Munich, Germany.
President and CEO of GPC Biotech, Martinsried/Munich, Germany.

Scientific Advisory Board

The Scientific Advisory Board provides additional research and development expertise relevant to Santhera's business. Its members are distinguished scientists and acknowledged specialists in their field of expertise:

Prof Katharine Bushby
University of Newcastle upon Tyne, UK

Prof Alfred Goldberg
Harvard Medical School, Boston, Massachusetts, US

Prof Karl Hofbauer
Biozentrum at the University of Basel, Switzerland

Dr med Bernd Löffler
Berlin, Germany

Dr Timothy Rink
Monaco

Prof Markus Rüegg
Biozentrum at the University of Basel, Switzerland

Prof Bernd Wetzel
Munich, Germany

Forward-looking statements

This Annual Report expressly or implicitly contains certain forward-looking statements concerning Santhera Pharmaceuticals Holding AG and its business. Such statements involve certain known and unknown risks, uncertainties and other factors, which could cause the actual results, financial condition, performance or achievements of Santhera Pharmaceuticals Holding AG to be materially different from any future results, performance or achievements expressed or implied by such forward-looking statements. There can be no guarantee that any of the development projects described will succeed or that any new products or indications will be brought to market. Similarly, there can be no guarantee that Santhera Pharmaceuticals Holding AG or any future product or indication will achieve any particular level of revenue. In particular, management's expectations could be affected by, among other things, uncertainties involved in the development of new pharmaceutical products, including unexpected clinical trial results; unexpected regulatory actions or delays or government regulation generally; the Company's ability to obtain or maintain patent or other proprietary intellectual property protection; competition in general; government, industry, and general public pricing and other political pressures. Santhera Pharmaceuticals Holding AG is providing the information in this Annual Report as of the date of the publication, and does not undertake any obligation to update any forward-looking statements contained herein as a result of new information, future events or otherwise.

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