

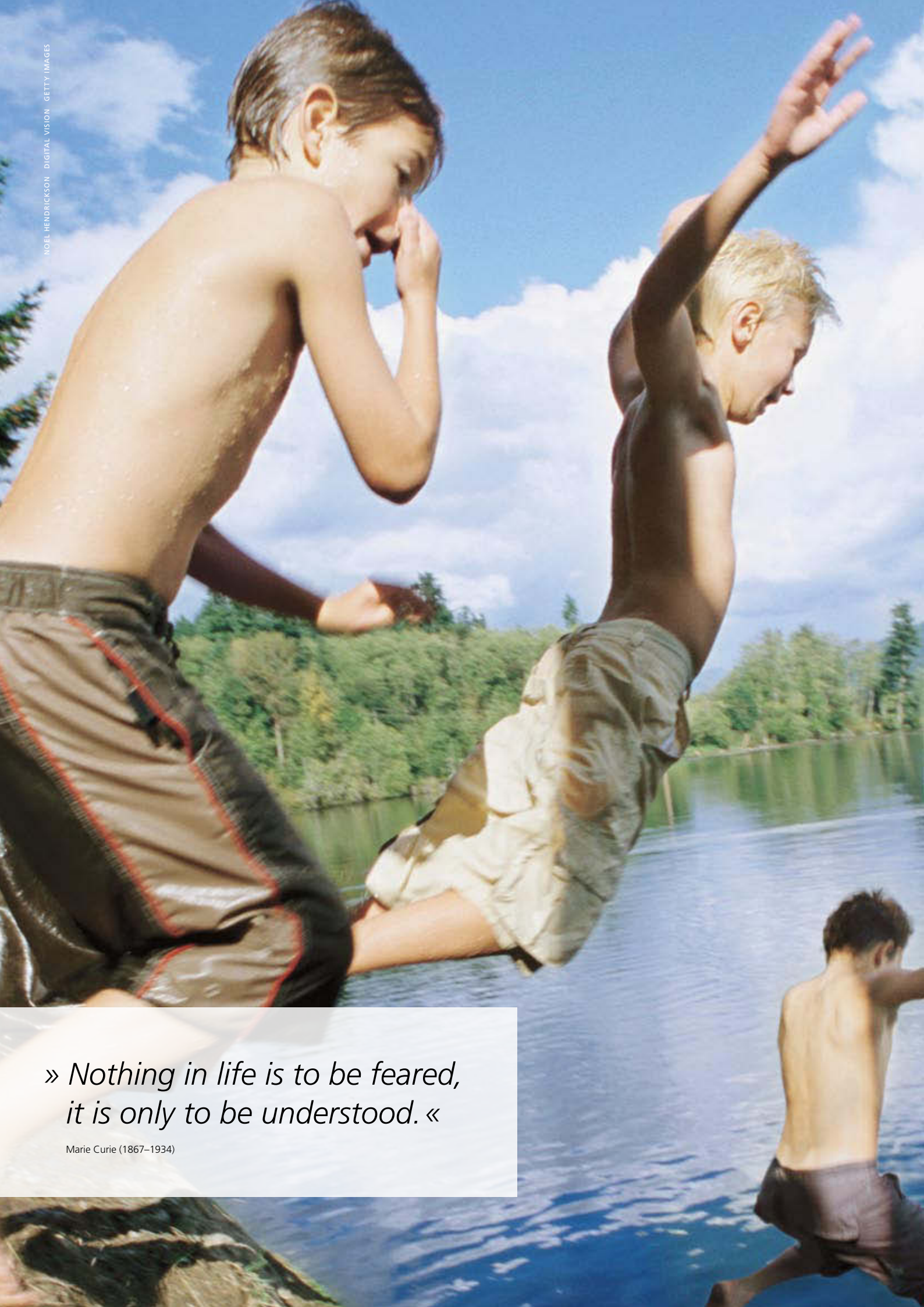


Diamyd Medical

Annual Report 2006/2007

*» You must be the
change you want to
see in the world. «*

Mahatma Gandhi



*» Nothing in life is to be feared,
it is only to be understood. «*

Marie Curie (1867–1934)



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Diamyd Medical in brief

Diamyd Medical is a Life Science company developing therapeutic products for diabetes and its complications. The Company licenses two proprietary technology platforms: the exclusive therapeutic rights to the gene coding for human Glutamic Acid Decarboxylase 65 kDa isoform (GAD65) including its uses for the treatment of diabetes and a Nerve Targeted Drug Delivery System (NTDDS).

Diamyd Medical's lead product is Diamyd®, a GAD65 based therapeutic vaccine for patients with newly-diagnosed type 1 diabetes and for the subset of patients with autoimmune type 2 diabetes known as LADA (Latent Autoimmune Diabetes in Adults). Treatable patients represent, in total, approximately 20% of the worldwide diabetes patient population.

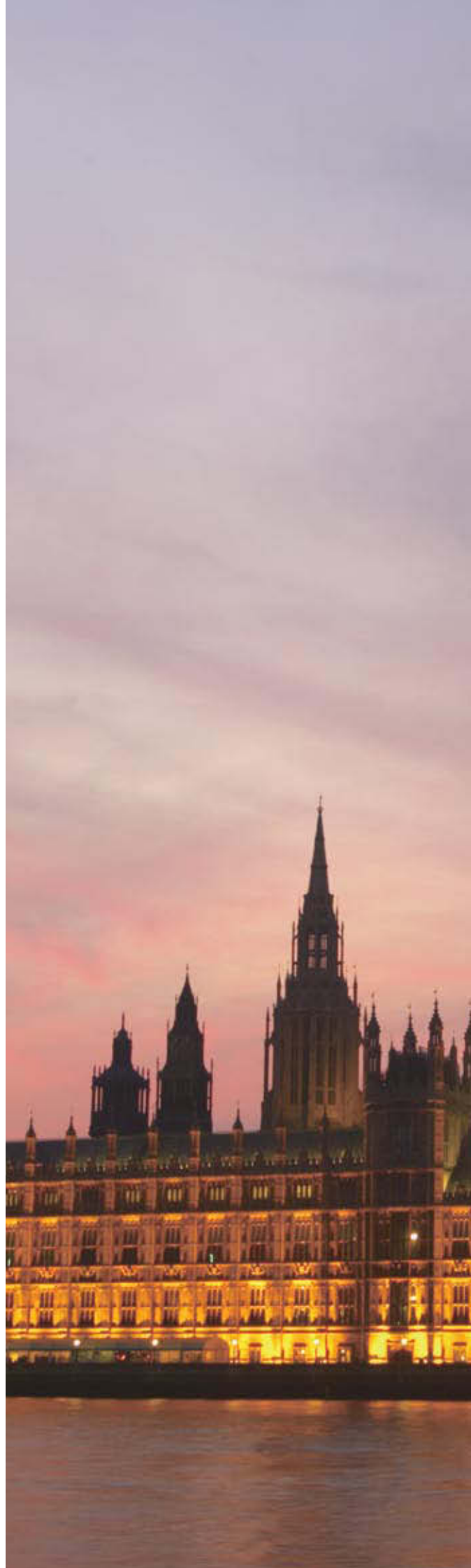
Diamyd® has demonstrated statistically significant efficacy in preserving insulin production in a Phase II clinical trial in 70 children and adolescents with type 1 diabetes. No serious adverse events associated with the therapy were observed, and the treatment was well received by patients and doctors. Additionally, analysis of individual patient immunological data showed that patients treated with Diamyd® had an up-regulation of certain beneficial immunological markers in response to the GAD65 protein. These immunological markers remained up-regulated even 15 months after the first injection.

The results of the study demonstrate that Diamyd® offers the potential to improve treatment of type 1 diabetes and a Phase III program is planned for both the US and Europe.

In addition to its role as a major autoantigen in autoimmune diabetes, GAD65 plays an important role in the nervous system by converting the excitatory neurotransmitter glutamate to the inhibitory neurotransmitter GABA. Through this mechanism, Diamyd Medical is developing products to treat chronic pain caused by diseases such as diabetes. In these products, GAD or enkephalin, another neurotransmitter, is delivered locally to the nerves that exert pain using a proprietary Nerve Targeted Drug Delivery System (NTDDS). In 2007, the lead NTDDS product, NP2, which produces enkephalin locally in the nerves to treat cancer pain, was advanced through preclinical safety testing and GMP manufacture and a Pre-IND meeting was held with the US FDA. The Company plans to file the IND for the NP2 Phase I safety study in cancer pain in the near term.

GAD65 can also be used to treat central nervous system diseases such as Parkinson's disease and epilepsy. During 2006, Diamyd Medical out-licensed the use of the GAD65 gene to Neurologix Inc., Fort Lee, New Jersey, U.S.A, for the treatment of Parkinson's Disease.

Diamyd Medical has offices in Stockholm, Sweden and in Pittsburgh, Pennsylvania, U.S.A. Diamyd® is manufactured by Protein Sciences, Inc. in Meriden, Connecticut, U.S.A.



» *The preliminary results with GAD are very exciting. GAD therapy is one of the most promising developments in trying to prevent inflammatory damage to insulin secreting cells.* «

PROFESSOR DAVID LESLIE, Professor of Diabetes and Autoimmunity at the Royal London and St. Bartholomew's School of Medicine, University of London





» If we can repeat our excellent Phase II results in the larger Phase III studies, it is likely that Diamyd® will become the drug of choice due to its favorable efficacy and safety profiles. «

ANDERS ESSEN-MÖLLER

CEO comments

This year we have focused our efforts on advancing our therapeutic diabetes vaccine Diamyd® towards initiation of our planned pivotal Phase III trials for recent onset type 1 diabetes patients. At the same time, and although no deal was reached, discussions and negotiations with regard to partnering Diamyd® with multinational pharmaceutical companies have been intense.

This year, our first NTDDS gene therapy project, NP2, also moved towards the clinic. A Phase I clinical study should be initiated in the near term. This may be followed by discussions with several potential partners regarding partnerships for nerve targeted delivery of these companies' various proprietary molecules.

Competition around therapies to treat type 1 diabetes patients to maintain the patients' own ability to produce insulin is heating up at the time of this writing. Two major partnering deals were recently announced involving T-lymphocyte targeting antibodies. Competing technologies however, often seem to have less specific effects on the immune system and may therefore be associated with more side effects than Diamyd®.

Diamyd Medical, being one of the few companies that have shown proof of concept with a beta cell specific autoantigen tolerization approach, should be well positioned as one of the most attractive candidates in this area of beta cell preservation. Diamyd® is without comparison very easy to administer, it has so far had a clean safety profile, and it has met with high acceptance by doctors, patients and parents. If we can repeat our excellent Phase II results in the larger Phase III studies, it is likely that Diamyd® will become the drug of choice due to its favorable efficacy and safety profiles.

Looking at the development status for our type 1 diabetes vaccine Diamyd®, we were pleased with our January End of Phase II Pre-IND meeting with the FDA in Washington DC. We believe that the outcome of the meeting was positive and that two successful independent Phase III trials, each with about 300 patients, may lead to product registration. The active substance of Diamyd® for the trials has been produced at Protein Sciences, and is at this time subject to testing. Formulation and vialing is planned to be performed in Europe. An application to conduct a Phase III study in the US is planned to be submitted in the near term.

In addition to having taken over responsibilities for manufacturing and preclinical activities for Diamyd®, our scientists in the Pittsburgh office are diligently progressing towards a Phase I trial using our proprietary NTDDS-enkephalin drug candidate for cancer pain. Once this Phase I trial is initiated we believe that the NTDDS platform technology will be used for delivery of several molecules, including GAD, to the nervous system for the treatment of various neurological diseases.

A setback was experienced in June, as we were forced to invalidate our Phase II study in LADA type 2 diabetes patients after an audit noted non-GCP conformances at the pharmacy responsible

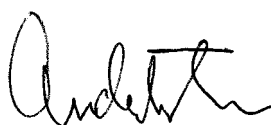
for the labeling and randomization of the study drug. On a more positive note, the positive 15 month results that were previously reported from our study in 70 children with type 1 diabetes, were still significantly positive after 21 months. This together with an increasing interest from US diabetes trial organizations such as NIDDK/TrialNet has recently reinforced the confidence in Diamyd®.

This year marked the turning point for our understanding of the mechanism of action of Diamyd®. Scientists in Linköping, Sweden, have reported positive results after analyzing immunological parameters from the type 1 diabetes trial previously reported. In fact, the data illustrated that virtually all patients treated with Diamyd® responded with an up-regulation of certain beneficial cytokines at restimulation with GAD65 even 15 months after the first vaccine administration.

In our view, this immunological data directly confirms the positive clinical results seen in the type 1 diabetes trial and further contributes to the large body of accumulated scientific evidence pointing to GAD65, the active ingredient of Diamyd®, as an effective and safe immunomodulator that prevents the immune system from destroying the insulin secreting beta cells. As a result, we continue to believe that additional applications for Diamyd® may include: **a)** treating LADA patients (patients with an autoimmune form of type 2 diabetes); **b)** treating pancreatic islet cell transplantation patients prior to transplantation to prevent recurrent autoimmune destruction of the islet cells; and **c)** preventing type 1 diabetes in at risk individuals.

We aim to continue discussions and negotiations with potential partners while simultaneously advancing our Diamyd® and NTDDS projects and investigating alternative ways to finance the planned studies. My belief in the success of Diamyd® has significantly increased during the past year, while of course the Company must still be regarded as high risk. We look forward to continued success in this coming year as well as making a difference when it comes to finding a cure for type 1 diabetes and neurological diseases.

Finally, I would like to thank our shareholders, directors, employees, scientific and other advisers, and not least our patients for making the work at Diamyd Medical so exciting and positive. Without you, our achievements so far would not have been possible. Respectfully, I ask for your continued support during this exciting journey and hope that the time of fruition will soon come into sight.



Anders Essen-Möller,
CEO and President of Diamyd Medical.

Product portfolio

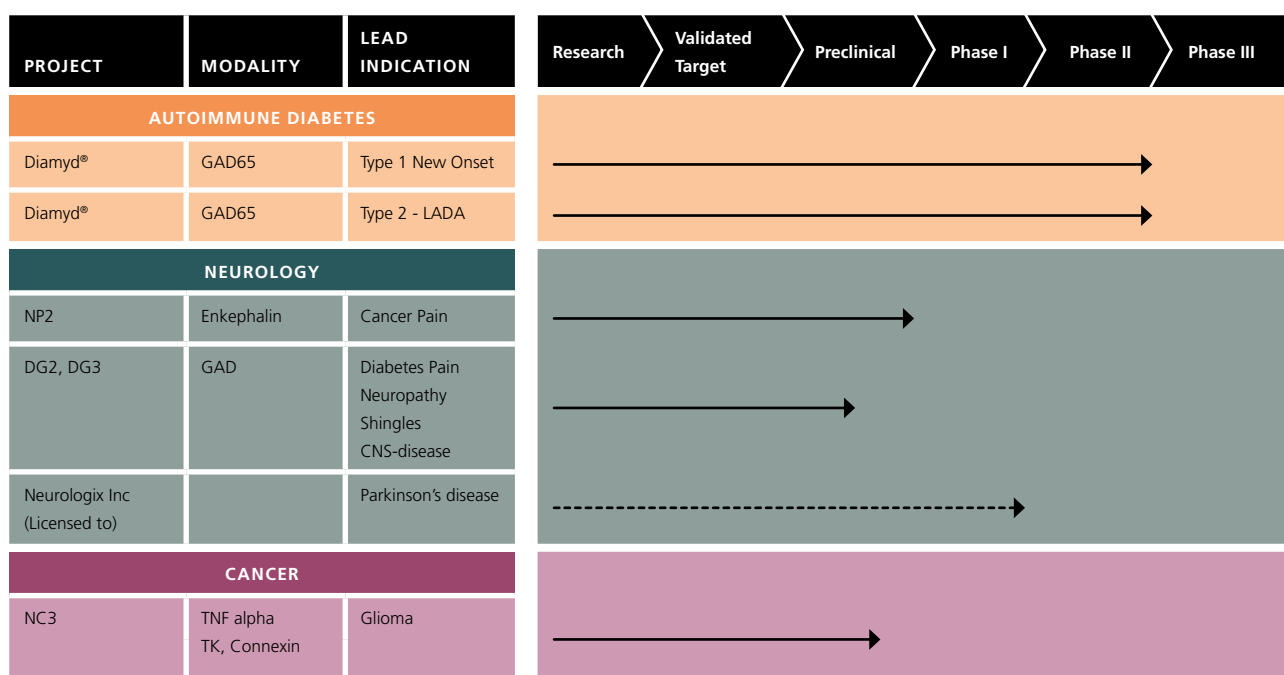
Diamyd Medical currently develops therapeutics from two independent platform technologies. One of these platforms relies on the GAD molecule and the other on a viral delivery system to deliver proteins to nervous tissue.

GAD

Glutamic Acid Decarboxylase (GAD) is being developed into a therapeutic vaccine for autoimmune diabetes, Diamyd®, and is the Company's furthest advanced candidate product. The endogenous human protein GAD may also have therapeutic effects in several neurological disorders including neuropathic pain, Parkinson's disease, migraine headaches, epilepsy, schizophrenia, bipolar disorders and anxiety. Neurologix Inc. has licensed GAD from Diamyd Medical on a non-exclusive basis for the development of new therapies for Parkinson's disease.

NTDDS

The Nerve Targeted Drug Delivery System (NTDDS) is used to deliver genes or therapeutic proteins to the nervous system. Products in the NTDDS portfolio utilize enkephalin or GAD to target chronic pain caused by diabetes, cancer and other diseases. Other NTDDS projects in pipeline involve neuroprotection and treatment of cancer.





Therapeutic areas

Diamyd Medical's furthest developed projects are focused on the treatment of autoimmune diabetes and chronic pain.

DIABETES AND ITS COMPLICATIONS

Scientific Background

Diabetes is a chronic condition characterized by elevated blood sugar (glucose) levels. After a meal, glucose is liberated from dietary carbohydrates within the small intestine and then absorbed into the blood. Elevated blood glucose levels stimulate release of insulin from pancreatic beta cells. Insulin acts on e.g. muscle and fat cells throughout the body to enable glucose uptake, utilization and storage. The blood glucose level is thereby reduced. However, if blood glucose levels cannot be controlled due to insufficient insulin secretion (as in type 1 and type 2 LADA diabetes) or if sensitivity to insulin is decreased (as in type 2 diabetes) diabetes follows. There are two principal forms of the disease.

I. Type 1 diabetes is the result of a deficiency of insulin due to an autoimmune attack on the patient's own insulin-producing pancreatic beta cells. At the time of clinical manifestation of the disease, typically in childhood, patients generally have about 10% of their beta cells remaining. The few remaining beta cells are incapable of producing enough insulin to maintain normal blood glucose levels and external insulin must be injected several times per day.

After presentation of disease, the autoimmune attack continues against the remaining insulin-producing beta cells, which in time will be completely destroyed. Maintaining tight control over blood glucose levels through monitoring and treatment with insulin will slow the progression of long-term complications. Nevertheless, most type 1 diabetes patients will suffer serious complications affecting blood vessels, nerves, kidneys and other organ systems due to the life-long nature of the disease.

II. Type 2 diabetes begins as a syndrome characterized by decreased sensitivity to insulin and the patient fails to respond appropriately to its own insulin. The exact mechanism is unknown, but in some patients insulin receptors are abnormal, while in others, the insulin signaling mechanism is defective. Type 2 diabetes generally occurs in adulthood with an estimated 90–95% of the world-wide diabetes patients having type 2 diabetes.

Approximately 10% of the type 2 diabetes patients have an autoimmune form of the disease similar to type 1 diabetes. While the progression of the autoimmune attack is slower than in type 1 diabetes, the patients will eventually require exogenous insulin as well. These patients are referred to as having LADA – Latent Autoimmune Diabetes in Adults. LADA patients are identified by the presence of antibodies against GAD.

Diabetes Complications

Poor control of diabetes, and long-term fluctuating and ill-controlled glucose balance leads to adverse health from pathological changes in a number of organs that may eventually lead to death. Major complications of type 1 and type 2 diabetes include:

Cardiovascular disease

Cardiovascular diseases are the major cause of death in diabetes and people with diabetes without previous heart attacks have been shown to have an increased risk of heart attacks.

Nephropathy

Diabetes is a major cause of renal failure requiring either dialysis or kidney transplantation.

Neuropathy

Diabetes leads to a wide range of effects on the peripheral nervous system. The most common manifestations of diabetic neuropathy are sensory loss in the feet and severe pain.

Retinopathy

In many cases long-term diabetes patients experience visual loss. Since type 2 diabetes often remains undiagnosed for several years, a significant number of people, even in developed countries, already have diabetic retinopathy and other complications at the time of diagnosis.

Diabetes Patient Population

It has been estimated by International Diabetes Federation that the number of diagnosed and undiagnosed adult individuals with diabetes is about 246 million persons worldwide. In 2003 it was 194 million and it is expected to reach 380 million by 2025. The incidence of diabetes in 2007 has been estimated to be 7 million new individuals. About 3–10% of the individuals diagnosed with diabetes have type 1 diabetes, with rates varying by country and ethnicity. About the same amount of patients have autoimmune type 2 diabetes, i.e. the LADA form of the disease.

According to the International Diabetes Foundation, the annual, worldwide healthcare cost for diabetes is estimated to 232 billion USD in 2007 (including treatment of complications of diabetes). The costs associated with diabetes in the western world are about 7% of the total health care budgets, or more than 100 billion USD in the United States alone. Each year, 3.8 million deaths are linked directly to diabetes-related causes.

CHRONIC PAIN

In the U.S.A., nearly one third of the population experiences severe chronic pain at some point in life, and, according to the American Pain Society, only one in four patients with chronic pain receives adequate treatment. The worldwide prescription pain market exceeded 19 billion USD in 2004 (*Source: "Peripheral Neuropathy and Neuropathic Pain" BioPharm Reports 2007*).

Diamyd's pain products are being positioned to compete in all major pain therapeutic categories including acute and chronic inflammatory, cancer and neuropathic pain. In particular, neuropathic pain is a poorly treated disease caused by nerve damage resulting from a number of diseases including diabetic peripheral neuropathy, HIV/AIDS neuropathy, spinal cord injury, and postherpetic neuralgia among others. In fact, peripheral neuropathy (PN) and neuropathic pain are inextricably linked with an estimated 170 to 270 million individuals globally affected by PN and over 100 million patients suffering from neuropathic pain, mostly due to PN. Diabetes, cancer and HIV alone are expected to increase the prevalence of PN and neuropathic pain by more than 10% by 2012.

The global market for neuropathic pain therapies was approximately 3.5 billion USD in 2006 and will double to >7 billion USD by 2016 (*Source: Datamonitor*). In the Diamyd pain portfolio, GAD seems especially well suited for the treatment of neuropathic pain and thus, the growing market demand will be suitable for Diamyd's first-to-market NTDDS pain products.

GAD

Glutamic Acid Decarboxylase isoform 65 kDa (GAD65) is a human enzyme that converts the excitatory neurotransmitter glutamate to the inhibitory transmitter GABA in nerve cells. In this context GAD65 may have an important role in several central nervous system-related diseases, e.g. Parkinson's disease and chronic pain. GAD65 is also present in insulin-producing beta cells in the pancreas although its role there is not fully understood. However, it is clear that GAD65 is one of the major targets when the immune system attacks the beta cells in autoimmune diabetes, i.e. type 1 diabetes and LADA. Diamyd Medical has an exclusive world-wide license from the University of California at Los Angeles regarding the therapeutic use of the GAD65 gene.

Diamyd®

Diamyd® is a therapeutic vaccine candidate which has shown clinical trial efficacy in preserving beta cell function in patients with type 1 diabetes and in LADA patients. No serious adverse events related to Diamyd® treatment have been reported in any study. The active substance in Diamyd® is recombinant human GAD65. In type 1 diabetes and LADA, where the immune system attacks the body's own insulin secreting cells, administration of Diamyd® can arrest or slow down the disease process. This allows the patients to maintain their own insulin secretion, which in turn leads to improved metabolic control and reduced hypoglycemic risk and reduces long-term diabetic complications. Furthermore, it may allow for regeneration of beta cells in a non-autoimmune environment, thus setting the stage for a cure of the disease.

Diamyd® Mechanism of Action

Studies in animal models for autoimmune diabetes as well as results from clinical trials have demonstrated that the destruction of the beta cells in autoimmune diabetes is mediated by the cellular arm of the immune system involving antigen-specific killer T-cells. The antibody arm of the immune system, the humoral arm, has more of a "bystander" role and is not actively involved in the destructive process itself. Diamyd® treatment is thought to induce immune tolerance to GAD65 and thereby intervene in the autoimmune process.

Several findings point to a mechanism of action of Diamyd® where the subcutaneous deposit of GAD65 in alum is processed by antigen-presenting cells to provide peptide fragments of GAD65 containing regions (determinants) recognized by T-cells. Presentation of those GAD65 determinants with tolerizing potential results in induction and proliferation of a subset of GAD65-specific regulatory T-cells. These regulatory T-cells down regulate antigen-specific killer T-cells that would otherwise attack the insulin-producing beta cells. This immunomodulation by Diamyd® vaccination is proposed to induce immune tolerance capable of preventing beta cell destruction in patients with autoimmune diabetes.

THE STORY OF GAD

2007	Preparation of Phase III clinical trials with Diamyd® in type 1 diabetes patients
2006	Positive results from Phase IIb trial in children with type 1 diabetes
2004	Start of Phase IIb trial in children with type 1 diabetes Start of Phase IIb clinical trial with Diamyd® in LADA patients
2003	Positive results from Phase IIa clinical trial with Diamyd® in LADA patients
2000	Start of Phase IIa clinical trial with Diamyd® in LADA patients
1999	Phase I clinical trial with Diamyd® with positive results Scientists show that suppression of GAD expression prevents diabetes
1998	Pre-clinical studies with Diamyd® Scientists confirm that transfer of GAD reactive T cells also transfers diabetes and that induction of regulatory T cells inhibits ongoing autoimmune diabetes
1997	Dose regimen for Diamyd® defined
1996	Scientists show that GAD vaccination extends the "honey moon" period and survival time of transplanted insulin producing beta cells
1994	Diamyd Medical licenses Intellectual Rights and initiates development of a GAD based diabetes vaccine
1993	Two research teams establish GAD as the first known target of T cells in diabetic NOD mice Both teams prevent diabetes by inactivation of GAD reactive T cells
1991	Molecular biologists clone and sequence GAD
1990	The 64K protein is identified as GAD
1988	Researchers find 64K antibodies in diabetic NOD mice
1987	Research shows that 64K antibodies predict type 1 diabetes in humans
1984	Scientists find antibodies to 64K protein in diabetic BB rats
1982	Scientists find antibodies to human beta cell 64K protein in diabetic children
1978	Research begins on beta cell antigens in diabetes
1974	Evidence is found that type 1 diabetes is an autoimmune disease

» For the first time it is possible to hold back type 1 diabetes «

For over 30 years we have studied different kinds of treatments aiming to intervene in the autoimmune process that characterize type 1 diabetes, and now it looks like we, with the vaccine Diamyd®, finally have found an easy and effective treatment that can hold back the disease. In the early 1970's, together with Åke Lernmark and Steinum Baekkeskov, we discovered that GAD, the active substance in Diamyd®, plays a role in type 1 diabetes, after having analyzed results from a study in Linköping where type 1 diabetes children were treated with plasmaphoresis. The GAD genes were then isolated in Allan Tobin's laboratory at University of California, Los Angeles, and in 1996 the doctors Daniel Kaufman and Jide Tian (UCLA) showed that GAD therapy can prevent type 1 diabetes in mice. Their studies formed the basis for the subsequent clinical studies with GAD that have been carried out by Diamyd.

It feels great that the GAD protein, which was discovered in our Linköping children, today is a key component in the most promising treatment of type 1 diabetes under development.

In 2004 we started a study where we gave seventy children between ten and eighteen years old who were recently diagnosed with type 1 diabetes, two injections of either the Diamyd® vaccine or placebo to see if treatment with GAD could improve the patient's own insulin production. The first results, fifteen months after the first injection, showed that the children treated with Diamyd® only experienced half the reduction in their own insulin production, measured by C-peptide, compared to those given placebo. Recently we also received the result from the 21 month follow up, which showed that the statistically significant treatment effect still remained.

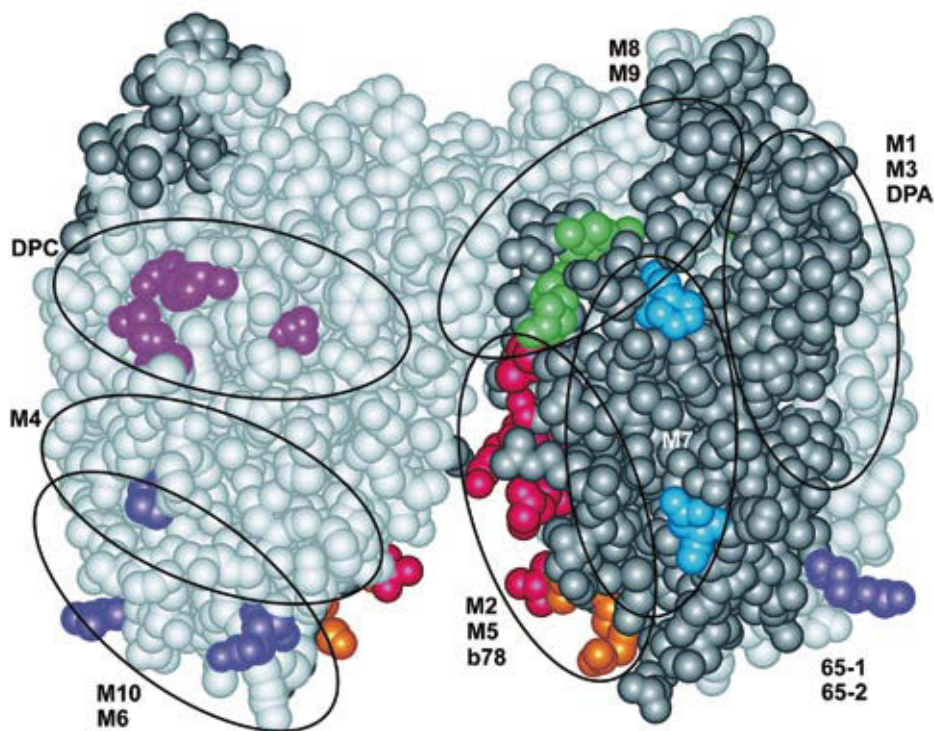
I have personally seen and analyzed the clinical results thoroughly and the clinical relevance for the patients that have received Diamyd® is clear. Maintaining insulin production is extremely important for those with type 1 diabetes because it helps them to better control the disease and reduce the risk of long term complications. For the first time we have found a substance that, without any side effects, can intervene in the immunological process and thereby save the beta cell function in patients with type 1 diabetes. This is fantastic! In addition the vaccine is very simple to administer and both children and their parents find the treatment easy – just two injections in children that are used to several injections a day.

At Linköping University Hospital we have also studied the immunological changes in the patients in this study. We found that fifteen months after the treatment there was a highly significant immunologic difference between patients that received Diamyd® and those who were given placebo. This shows that vaccination with Diamyd® gives a lasting and specific effect on the immune system, which can be the explanation for the protective effect on the insulin production.

The results from the study are exceptional and together with the immunological analyses it provide very strong evidence that this treatment has the potential to become a future vaccine for type 1 diabetes patients with the intention to preserve beta cell function. I am therefore very positive and full of expectation about the planned Phase III program that is intended to include patients that have only had the disease for three to four months. The earlier the vaccine is given, the better the effect should be. The next logical step, after the Phase III program, would be to investigate whether the vaccine can also be used to prevent diabetes.



JOHNNY LUDVIGSSON, at Linköping University Hospital, is Professor of Pediatrics and has been involved in type 1 diabetes research since the 70's. Today, among other things, he is leading a large prospective study in 17 000 children of which the etiology of type 1 diabetes is the focus (ABIS – All Babies in Southeast Sweden). In addition Ludvigsson is heading a large research team within diabetes autoimmunity at Linköping University Hospital, and is the chairman of Diabetes Research Centre, Linköping University. Professor Ludvigsson is also Principal Investigator for Diamyd Medical's Phase II type 1 diabetes study investigating the effect and safety of the Diamyd® vaccine in 70 children.



GAD65 molecule with different epitopes marked (Baekkeskov S., Schwatr H.L., J. Mol. Biol., 287, 5, 1999, 983–999)

Intervention with Diamyd® in recently diagnosed type 1 diabetes patients

In August 2006 Diamyd Medical announced positive results from a randomized, double-blind, placebo-controlled Phase II clinical trial in 70 children and adolescents with recent onset type 1 diabetes. Enrolled patients had duration of disease of a maximum of 18 months and were GAD65 antibody positive. A low dose of Diamyd® (20 µg) was injected twice, 30 days apart, in a typical prime and boost vaccine regimen. Patients are followed for 30 months, with data presented after 15 months, 21 months and 30 months.

The key efficacy measurement in the type 1 diabetes trial was meal-stimulated C-peptide. Since insulin and C-peptide are produced simultaneously in equal amounts, and C-peptide is easier to measure, meal-stimulated C-peptide level is the most appropriate parameter to follow in a type 1 diabetes trial where the aim is to measure the beta cell's ability to produce insulin.

In September 2006, Professor Johnny Ludvigsson, Linköping University Hospital, Sweden and Principal Investigator for the trial, reported positive 15 month results at the European Diabetes meeting EASD in Copenhagen in Denmark. One year later, in September 2007, Professor Ludvigsson reported continued positive results after 21 months at the International Society for Pediatric and Adolescent Diabetes (ISPAD) meeting in Berlin, Germany.

Major conclusions of the trial were:

- Diamyd® demonstrated efficacy in slowing the decline of C-peptide levels after a stimulated meal at 15 months. C-peptide levels in both groups experienced a decline, but the decline was significantly inhibited in the Diamyd® group ($p = 0.01$). The statistically significant protective effect remained also 21 months after the first injection ($p = 0.02$).
- The relative insulin requirements in the Diamyd®-treated group increased less than in the placebo group.
- The positive treatment effect was most pronounced in patients treated shortly after diagnosis. Diamyd®-treated patients with a disease duration of less than 3 months at intervention ($n = 4$) actually showed improved C-peptide levels at 15 months, whereas placebo treated patients ($n = 7$) showed a decline in C-peptide levels. The subgroup was too small for statistical calculations.
- Immunological analyses confirmed that treatment with Diamyd® causes a specific immune response to GAD65 that remained even 15 months after treatment. The immunological effect was observed in virtually all patients receiving Diamyd®, but not in patients receiving placebo. The immune system of Diamyd®-treated patients showed a highly statistically significant increase in the secretion of several immunomodulatory substances, dominated by regulatory cytokines, and increased activity of T-cells.

– In addition the results strongly support that administration of Diamyd® in children and adolescents is safe. There were no serious adverse events associated with the Diamyd® therapy.

The results provide strong support that the administration of Diamyd® is effective in preserving insulin-producing function in type 1 diabetes patients. The maintenance of endogenous insulin production is important as it helps patients to better control their disease and reduce long-term complications. Overall, the Company believes that Diamyd® will be able to offer a compelling, first in class therapeutic for insulin-producing beta cell preservation in type 1 diabetes due to the efficacy, safety and ease of use.

The next step in the clinical development of Diamyd® for treatment of type 1 diabetes will be to initiate large clinical trials in the U.S.A. and Europe. The Company is in the final stages of preparing for these trials. In Europe, Professor Ludvigsson will act as the lead investigator for the multi-center trial and Professor Jerry Palmer, Head of the Diabetes Endocrinology Research Center at the University of Washington in Seattle, WA will lead the US clinical program. Testing Diamyd® in a large, international patient population is aimed to support registration and market approval applications. The trials are planned to be conducted at roughly 30 sites in the US and Europe respectively, and will each include approximately 300 type 1 diabetes patients. Enrollment time is estimated to be approximately 9 months and results will be evaluated after 15 months. The patients will then be followed for an additional 15 months. The primary endpoint is planned to be meal-stimulated C-peptide, as a direct marker of endogenous insulin secretion. Trends in blood glucose levels and insulin dose will also be monitored.

Separately, the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) plans an international clinical study with the Diamyd® diabetes vaccine in 126 new onset type 1 diabetes patients. The trial is intended to include extensive immunological studies to clarify the mechanism of action and to evaluate the correlation between the clinical and immunological outcomes of Diamyd® treatment in recent onset type 1 diabetes patients. The study is proposed by the NIH/NIDDK sponsored global network TrialNet, a group of the world's foremost experts and key opinion leaders in type 1 diabetes.

Intervention with Diamyd® in type 2 LADA patients

Diamyd® is also being developed for the treatment of type 2 diabetes patients who have antibodies specific for GAD and therefore have a form of autoimmune diabetes known as Latent Autoimmune Diabetes in Adults (LADA). This form of the disease is characterized by a slow-progressing autoimmune attack on the insulin-producing beta cells.

Diamyd Medical has previously conducted a successful small scale, dose-finding Phase II clinical trial in 47 type 2 LADA patients. Positive

outcomes were reported after 24 months. A broad dose range was investigated with respect to safety and efficacy. The results indicated that the preparations were clinically safe and the 20µg dose of Diamyd® was found to be the most effective and was selected for further development. This dose was found to significantly improve both meal-stimulated C-peptide and HbA1c levels at two years after treatment. Five year follow up results are due mid-2008.

It is important to note that the statistically positive effect on C-peptide (or insulin) levels observed in the 20 µg dose group was associated with an elevated number of T-cells that can down-regulate an autoimmune attack on insulin-producing beta cells. This outcome likely enhances insulin-producing beta cell survival and function, and thereby gives rise to improved insulin (C-peptide) secretion.

To confirm the positive results obtained from the above type 2 LADA trial, another Phase II clinical trial was initiated in 160 type 2 LADA patients. In June 2007 that study was invalidated because of inconsistent and contradictory efficacy data combined with critical observations during a formal independent audit at the central pharmacy handling the investigational product (Diamyd® and placebo). The audit concluded that it was impossible to guarantee absolute identity of the content of each vial of the investigational product administered to the patients and that the risk of a possible mix up between active drug and placebo was obvious. Importantly, however, no serious adverse events related to the treatment were reported for any patient in the trial. The study continues with regard to safety.

GAD for treatment of Parkinson's disease

During August 2006 Diamyd Medical and Neurologix Inc. entered into a licensing agreement where Diamyd out-licensed the GAD65 patents to Neurologix for the development of a GAD-based therapy to treat Parkinson's disease on a non-exclusive basis. A Phase I trial with patients having Parkinson's disease was completed by Neurologix in 2006. Primary objectives of the trial regarding safety and tolerability, were successfully met. There were no adverse reactions reported. The patients registered a clinical improvement on the Unified Parkinson's Disease Rating Scale (UPDRS) compared to baseline ($p < 0.005$). Nine of the 12 patients showed an average improvement of 37%, and five of these patients had substantial improvement of between 40% and 65%. Neurologix expects to begin Phase II studies in Parkinson's within the near term.

Manufacturing of Diamyd®

In December 2005, Diamyd Medical entered into a manufacturing agreement with Protein Sciences, Meriden, CT, U.S.A, for production of clinical grade Drug substance (GAD protein) for future clinical trials.

» Diamyd® has a lasting impact on the cause of type 1 diabetes «

I have been involved in immunological research in type 1 diabetes for 20 years and it is no doubt that understanding the pathogenesis of this debilitating disease is a very complex matter, but also that it holds the key to finding a cure. Diamyd's approach of treating type 1 diabetes by intervening at the immunological level makes perfect sense as the cause of the disease is an attack by the patient's own immune system on the insulin producing beta cells in the pancreas.

Immunomodulation, aiming at teaching the immune system to leave the insulin producing cells alone, has been on the scientific community's agenda for many years. So far it has not been achieved, but now we have evidence that treatment with Diamyd® has both a lasting and selective impact on the immune system as well as a protective effect on the insulin production. Those are criteria for a valid immunomodulating therapy.

I was not an investigator in the successful study with Diamyd® in Swedish children, but after reviewing the findings I must congratulate Professor Ludvigsson and his team on their pioneering clinical studies that led to this breakthrough.

The immunological data from that study shows that in Diamyd® treated children, the part of the immune system that can counteract the attack on the insulin producing cells has been engaged and persists at least 15 months after the treatment. No corresponding effect can be seen in the placebo treated patients. The beneficial impact of Diamyd® on the immune system is very specific, which minimizes the risk for adverse side effects of the treatment. Prior attempts of intervening in the immune process have failed either due to dangerous side effects, lack of specificity or because a lack of clinical effect.

From our studies in clinical islet transplantation, we recently learned that the currently applied therapies in that context seem to cope with rejection, but are insufficiently targeting the cause of type 1 diabetes: the immune response against islets. This observation underscores the need of therapies selective for islet autoimmunity, and it is here that Diamyd® may offer a solution.

This is the first time that biomarkers have been identified that correlate with immunological efficacy using an autoantigen specific therapy in the clinic. A perhaps even more pressing need is the definition of immune correlates with clinical efficacy. An extremely encouraging message from the clinical studies of Professor Ludvigsson is indeed the evidence of immune markers that correlate with clinical efficacy, providing another novelty in immune intervention therapy in autoimmune diseases. Perhaps the most astonishing finding is the persistence of immune changes many months after therapy, which implies a lasting impact of Diamyd® on autoimmune deviation. This represents a breakthrough in antigen-specific immunotherapy.

I am looking forward to the 30 month results from the study with great excitement. In addition, the clinical study with Diamyd® planned by TrialNet/NIDDK will clarify the effect of the vaccine on the immune system in greater detail. This will help us understand even better how Diamyd® intervenes in the type 1 diabetes disease process and how Diamyd® can be used to prevent type 1 diabetes in individuals with high risk of developing the disease.



PROFESSOR BART ROEP, head of the Division of Autoimmune Diseases at the Leiden University Medical Center in The Netherlands, is an internationally renowned immunologist. His primary interest is the pathogenesis of type 1 diabetes in humans, which was the topic of his dissertation already in 1992. He has focused on the role of autoreactive T cells in diabetes assessing human cellular immune responses, autoantigen identification, islet allograft rejection and the design and immunological monitoring of immunointervention strategies in clinical type 1 diabetes.

Clinical development of Diamyd®

An overview of the completed or ongoing clinical trials with the therapeutic vaccine Diamyd® is presented below:

	1. Skin prick test trial, Sweden, 1995	2. A Phase I randomized, double-blind, placebo controlled, rising dose trial, UK, 1999	3. A Phase IIa, randomized, double-blind, placebo-controlled, group-comparison, dose-finding trial, Sweden, 2000	4. A Phase IIb, randomized, double-blind, placebo-controlled, multi-centre trial, Sweden 2004	5. A Phase IIb, randomized, double-blind, placebo-controlled, multi-centre trial, Sweden 2004
Participant	Type 1 diabetes patients and healthy controls	Male healthy volunteers	Type 2 LADA patients	Type 1 diabetes patients	Type 2 LADA patients
Age, years	Adolescents	24–45	30–70	10–18	30–70
Number of patients	N=15	N=24	N=47	N=70	N=160
Trial period	28 days	10 weeks	Main trial period: 6 months. Completed April 2003. Follow up: 4.5 years. Ongoing	Main trial period: 15 months. Completed August 2006. Follow-up: 15 months. Ongoing	Main trial period: 18 months. Ongoing
Purpose	Safety	Safety, tolerability	Dose-finding, safety, efficacy	Safety, efficacy	Safety, efficacy
Comments	Administration of 1 µg rhGAD65.	Single dose, 20, 100, 250 and 500 µg rhGAD65 or placebo.	Prime and boost of 4, 20, 100, 500 µg Diamyd® or placebo, given on two occasions, 4 weeks apart. Up to two extra boosts in the 500 µg Diamyd® group.	Prime and boost of 20 µg Diamyd® or placebo, given on two occasions, 4 weeks apart.	Prime and boost of 20 µg Diamyd® or placebo given on two occasions, 4 weeks apart.
Safety outcome	None of the subjects showed any adverse reactions.	Subcutaneous administration of all doses of rhGAD65 was well tolerated.	Subcutaneous administration of Diamyd® was well tolerated in all dose ranges over the trial period and after 2 years.	Subcutaneous administration of 20 µg Diamyd® was well tolerated.	Subcutaneous administration of 20 µg Diamyd® was well tolerated.
Efficacy outcome			Statistically significant increases in C-peptide levels in conjunction with an increase in the CD4 ⁺ CD25 ⁺ subset of down regulating T-lymphocytes were seen in the 20 µg Diamyd® dose group alone. During follow-up the decreasing trend in HbA1C became statistically significant in the 20 µg Diamyd® dose group alone. These data were used to rationalize the selection of the 20 µg Diamyd® dose group alone for further clinical investigation.	Although the mean meal stimulated secretion of C-peptide declined in both trial groups, there was a significantly smaller decline in the Diamyd® group 9, 15 and 21 months after the first injection compared to placebo.	Efficacy results invalidated in June 2007 due to violation of GCP guidelines at the pharmacy responsible for the randomization process. Study continues with regard to safety.

» Proud to take Diamyd® to the next level «

I have followed "The Story of GAD" right from the beginning. GAD is a key antigen in type 1 diabetes and antibodies to GAD are present in most patients with this disease and are also markers for increased risk of future diabetes in non-diabetic individuals. Administration of GAD prevents type 1 diabetes in animal models and recent data shows slowing of beta cell loss in GAD-vaccinated type 1 diabetes patients. I am proud to be part of the team that will bring this project to the next level in my role as lead investigator for the planned US Phase III clinical trial in type 1 diabetes patients.

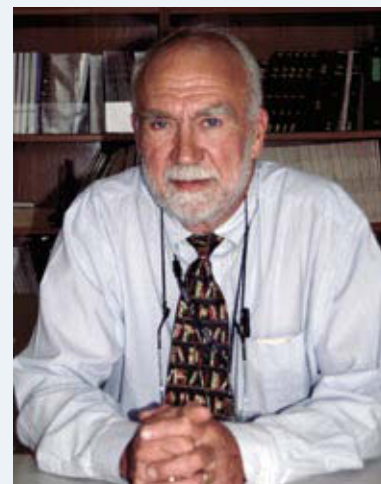
The onset of human type 1 diabetes is the clinical manifestation of the beta cell failure caused by autoimmune destruction of the insulin producing pancreatic beta cells. This results in a life long dependence on daily insulin injections and in many patients acute and late complications of this disease. The severity of these complications makes the development of a therapy that preserves beta cell function, such as Diamyd®, of great urgency for patients and caregivers.

I have had the privilege of reviewing the results from Diamyd Medical's Swedish Phase II type 1 diabetes trial in depth. The statistically significant better preservation of endogenous C-peptide production in the Diamyd®-treated group compared to placebo, shows that the drug is clearly effective in preserving beta cell function. This is an important breakthrough for autoantigen specific therapy. Control of blood glucose requires continual adjustment of insulin secretion and the body's own insulin production is superior to insulin injections in managing swings in blood glucose levels. Any intervention which can stop or delay the complete loss of residual beta cell function is significant and clinically meaningful. This is particularly true for a treatment with such a strong safety profile and ease of administration as Diamyd®. Regardless of the patients' need for insulin injections, even relatively modest preservation of the patient's physiological ability to secrete insulin is known to improve metabolic control and lower risk for hypoglycemia and chronic complications.

C-peptide is a proxy for endogenous insulin as it is secreted in equal amounts to insulin by the beta cells. Measurement of insulin itself in patients with type 1 diabetes is confounded by the insulin from their injections. Meal stimulated C-peptide levels is in my opinion the most accurate parameter to show whether a drug can maintain the critical function of pancreatic beta cells in type 1 diabetes patients. I am pleased with Diamyd Medical's decision to use meal stimulated C-peptide as the primary endpoint in the upcoming Phase III trials and that the FDA supports the decision.

I will also be the lead investigator in a separate clinical study with Diamyd®, which will be run by TrialNet/NIDDK. That study will put more emphasis on the immunological mechanism behind the clinical effects of Diamyd® treatment. A better understanding of the immunological effects could serve as the basis for designing trials to investigate if Diamyd® can prevent type 1 diabetes in individuals who are at high risk of developing the disease.

Type 1 diabetes is a severe burden to children and their families, representing one of the most common chronic childhood diseases. I look forward to contributing to the success of future clinical studies with the hope that Diamyd® may help patients with type 1 diabetes achieve better control of their diabetes and to experience fewer long-term complications. Eventually Diamyd® may be used to prevent the disease.



PROFESSOR JERRY PALMER, Head of the Diabetes Endocrinology Research Center at the University of Washington in Seattle, will be the Lead Investigator in the planned Diamyd Medical Phase III clinical trial in the US. Professor Palmer is one of the world's leading authorities in type 1- and in type 2 diabetes, and has extensive experience in clinical trials. Professor Palmer's research focuses on the immunology of the type 1 diabetes disease process in humans. The dual role of T-cells in mediating the beta cell destruction of this disease, but also in mediating protection against beta cell destruction in other individuals is being investigated.

NTDDS

Replication Defective Nerve Targeting

Drug Delivery System (NTDDS)

The NTDDS is based on a highly engineered replication defective virus that does not multiply once injected into the patient. Further, the NTDDS has a natural affinity for nerve cells that result in a highly targeted delivery vehicle. When delivered to the nerve cell, the NTDDS remains stable for several weeks while it produces therapeutic biologics from inserted genes directly into the nerve junctions (synapses). A key advantage of the NTDDS lies in the fact that the localized therapy will not circulate throughout the body, and, therefore, will be less likely to produce any systemic side effects that are common in current pain and CNS disease therapies. Additionally, the NTDDS does not integrate into a host cell's chromosome and the risk of adverse events is further reduced.

NTDDS has the capacity to deliver multiple genes and development of several products for treatment of pain and other nervous system diseases are anticipated. The same delivery system can also be used to deliver cancer killing compounds through direct injection into solid tumors.

NTDDS Lead Products for Chronic Pain – NP2 and GAD

The NTDDS lead projects are therapeutics for treating pain using Enkephalin (NP2) and GAD (DG2 and DG3). There is an extensive body of preclinical safety and efficacy data for NP2 and related products. In particular, extensive rodent and primate studies show that related NTDDS products are well tolerated. The results of these studies have been published in the peer reviewed literature and presented at numerous international conferences and the results demonstrate that a simple injection of the NTDDS product into the skin:

- Effectively alleviates pain in a variety of acute and chronic pain animal models for weeks;
- Is safe and targets sensory neurons in animals;
- Provides a therapeutic effect, even in the face of tolerance to morphine;
- May have an additive effect when used with morphine;
- Can be re-administered effectively; and
- Reduces chronic pain without inducing tolerance or inducing side effects.

NP2 was the subject of a pre-investigational new drug (IND) meeting with the U.S. Food and Drug Administration in August, 2007. GMP-grade clinical material for the Phase I NP2 safety trial has been produced in 2007 and Diamyd plans to file the NP2 IND application in the near term with the Phase I trial to follow. The proposed Phase I clinical trial will be conducted at the University of Michigan in Ann Arbor. Dr. David Fink, Professor and Chair of the Department of Neurology, at the University of Michigan will be the principle investi-

gator. The trial will be designed as a dose-escalation study and is intended to test the safety of NP2. In total, 12-24 patients who suffer from severe cancer-related pain are planned to be enrolled.

The NTDDS product producing GAD locally in the neurons has demonstrated efficacy in treating chronic neuropathic pain in animal models resulting from nerve damage caused by diseases such as diabetes and spinal cord injury. Diamyd plans to initiate preclinical safety testing and GMP manufacturing for either its DG2 or DG3 product in 2008.

GAD and enkephalin delivered by the NTDDS for pain control – Mechanism of action

Pain is transmitted through a series of neurons that run from the skin to the brain. Pain signaling can be inhibited in several ways at the synapse between the peripheral and central nervous systems. This synapse provides input from the skin or organs via the first order neuron. The output from this synapse, the second order neuron, is within the spinal cord and projects into the brain to complete the pain pathway. Interneurons represent a feedback loop from the brain to modify the pain transmission through the release of compounds that dampen the signals when the brain senses pain.

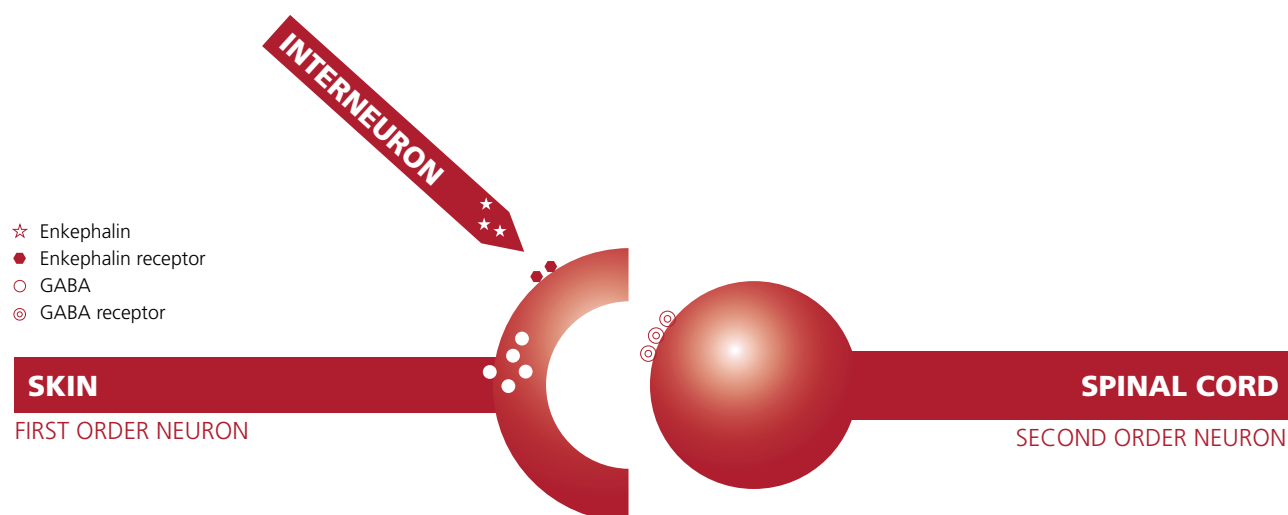
Major compounds that dampen pain transmission are enkephalins and GABA. These transmitters are expressed in all synapses, however, depending on location in the body, there might be differences in their effectiveness. For example, while GABA dampens spinal cord injury pain signals, enkephalins may be more efficient in inflammatory pain and cancer pain.

In normal first order peripheral neurons, GAD is synthesized and converts glutamate into GABA, an inhibitory neurotransmitter. Release of GABA from the first order neuron into the synapse will bind the GABA receptor present on the second order spinal neuron and stimulate intracellular signaling to dampen pain transmission. Through a separate mechanism, enkephalin is naturally released from the interneuron to bind to enkephalin receptors on the surface of the second order neuron to block pain transmission to the spinal cord.

Thus, in Diamyd Medical's approach, GAD and enkephalin are produced by the NTDDS in the first order neuron and released directly into the synapse to block or dampen transmission of the pain signals. Finally, since the GAD and enkephalin act on separate pain receptors, development of two NTDDS pain products, GAD for neuropathic pain caused by diabetes and enkephalin for treatment of acute pain caused by cancer or arthritis are, therefore, complementary.

Glioma Cancer Therapy – NC3

Through the acquisition of the NTDDS-project, Diamyd acquired NC3, a late-preclinical stage product for the treatment of brain cancer. The novel approach to treating glioblastoma multiforme involves a multigene (TNF α , TK, ICPO, Cx43) therapeutic NTDDS delivery system. The product has been engineered to provide high



levels of expression for up to 7 days of the therapeutic genes, which will act in concert to destroy malignant cells while sparing surrounding normal neurons: ICPO has been shown to arrest tumor cell division and enhance vector transgene expression, TK activation of ganciclovir kills infected tumor cells; connexin 43 enhances bystander killing of uninfected neighboring tumor cells and tumor necrosis factor alpha enhances tumor cell sensitivity to radiosurgery. Ultimately, while the initial indication will be malignant glioma, it is anticipated that NC3 may be useful for the treatment of numerous solid tumors.

The NC3 product is currently in rodent toxicology testing that is being funded with grants from the US National Institutes of Health.

Other NTDDS Products

The NTDDS technology can also be leveraged for targeting other peripheral and central nervous system diseases. Diamyd has generated compelling preclinical data in animal disease models suggesting that the NTDDS products expressing neurotrophic factors are effective as therapies for preventing nerve damage caused by peripheral neuropathy. Peripheral neuropathy is a debilitating disease affecting millions of patients who suffer from diabetes, viral infections, high-dose chemotherapy and other nerve damaging conditions. There is presently no effective treatment and a therapy that could act locally to protect neurons would be of enormous clinical importance. Additionally, utilizing the NTDDS to deliver proteins such as GAD to the central nervous system may be effective for the treatment of Parkinsons, epilepsy and other neurological diseases. These additional products are in the early stages of preclinical development and no timeline for clinical development is currently estimated.

» The potential applications for the NTDDS platform are limited only by our imagination «

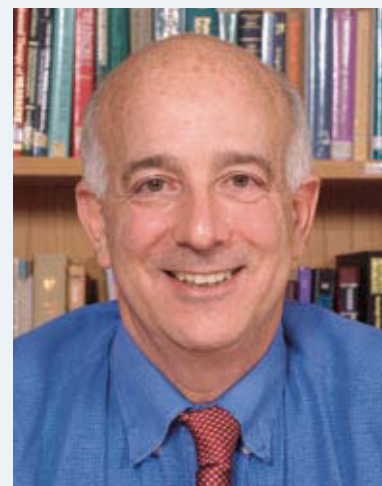
For the past 15 years, my research has focused on the development of new methods to treat diseases of the nervous system, ultimately resulting in the novel nerve targeting delivery system (NTDDS) that is now being developed by Diamyd. Through a collaboration with Dr. Joseph Glorioso and his research team that includes many of the scientists now working for Diamyd, we have demonstrated in animal models the potential of the NTDDS technology to treat diseases of the peripheral nervous system including chronic pain and sensory polyneuropathy, as well as diseases of the central nervous system including cancer and Parkinson's disease.

The advantage of Diamyd's NTDDS technology over conventional therapies is the ability to express the active agent directly in neurons. In the treatment of pain, for example, effective use of conventional drug therapies is often limited by unwanted off-target adverse effects resulting from actions of the pain-relieving drugs at sites unrelated to pain. Local delivery to the desired site using Diamyd's NTDDS technology circumvents this problem, producing a sustained, pain-relieving effect.

In published experiments, we have reported that delivery of a variety of different small protein molecules using this technology can relieve pain in animal models of pain due to inflammation, tissue injury, nerve damage or cancer. In particular, the NP2 product now being readied for human clinical testing has been demonstrated to be effective in models of pain resulting from cancer and arthritis. I am very excited about the potential for other NTDDS products, for example the NTDDS system expressing GAD to treat the common condition of pain resulting from diabetic neuropathy.

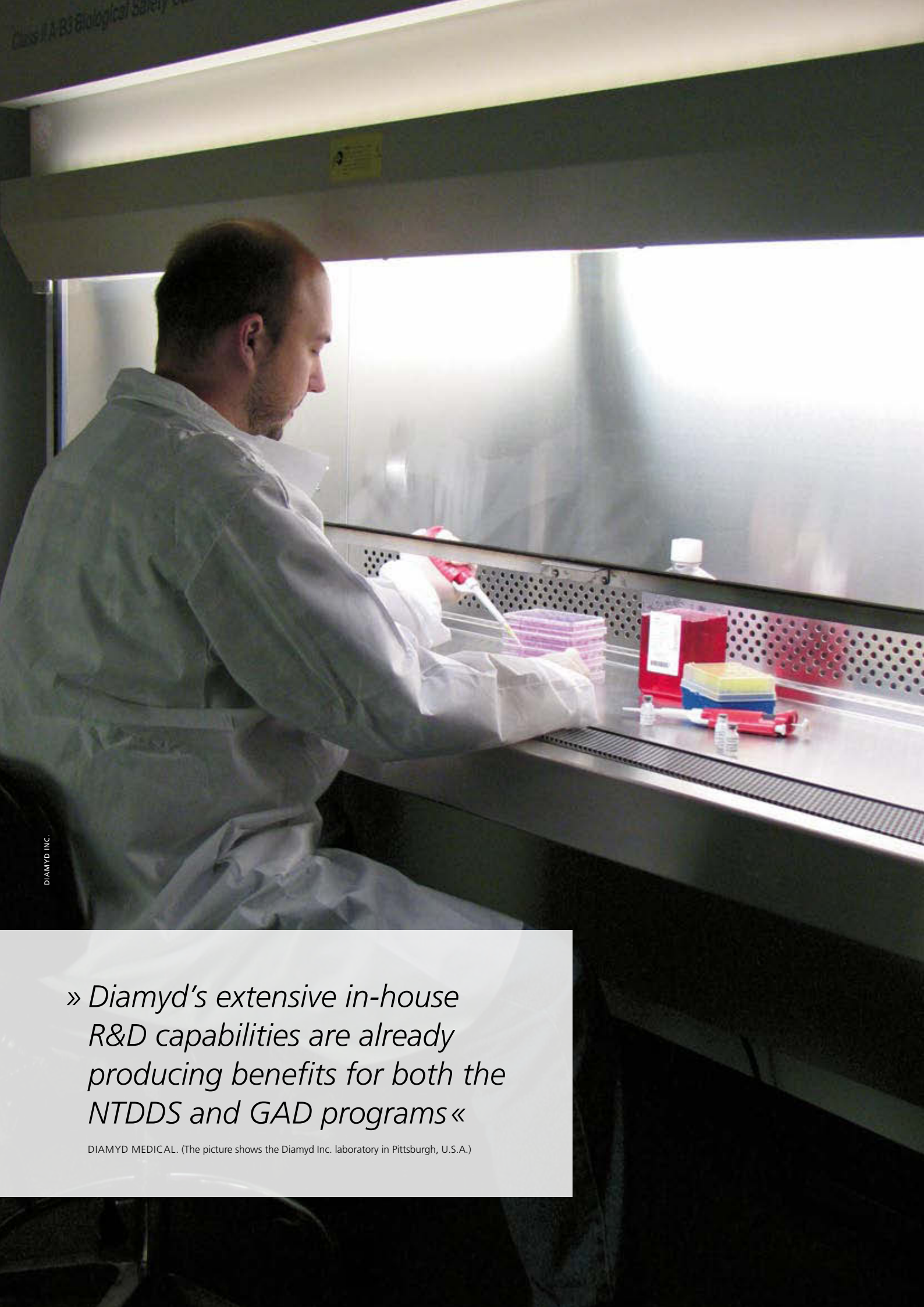
It is with great enthusiasm that I look forward to the next phase of development of this research program: initiation of human clinical trials. I am extremely pleased that Diamyd has committed the resources and successfully moved NP2 to the brink of clinical development and am looking forward to acting as the principal investigator on the first clinical study at the University of Michigan.

As a practicing neurologist, my scientific and clinical interest in the potential applications of the NTDDS technology is not limited to pain. For example, we have published extensively on the potential of the use of other agents based on the NTDDS platform to prevent or treat peripheral neuropathy. Neuropathy, a debilitating disease characterized by nerve damage resulting in loss of sensation in the hands and feet is a common complication of diabetes for which there currently is no effective therapy. I am optimistic that once we have established the safety and efficacy of this broad delivery platform, applications will be limited only by our imagination.



DAVID J. FINK, M.D. is the Robert Brear Professor and Chair of the Department of Neurology at the University of Michigan. Dr. Fink is a leading researcher in the development of gene transfer for the treatment of neurological disease. He is the author of more than 110 peer-reviewed articles, 30 book chapters, and is an inventor on 2 issued patents. He serves on the editorial boards of the journals *Gene Therapy*, *NeuroTherapeutics*, and the *International Review in Neurobiology*, as well as on the Board of Councilors of the American Neurological Association.

Dr. Fink is an inventor on some of the patents licensed to Diamyd. He has sponsored research agreements with Diamyd but does not hold an equity position in, or consult for, the Company.



DIAMYD INC.

» *Diamyd's extensive in-house R&D capabilities are already producing benefits for both the NTDDS and GAD programs* «

DIAMYD MEDICAL. (The picture shows the Diamyd Inc. laboratory in Pittsburgh, U.S.A.)



Intellectual property rights

The Intellectual Property Rights of Diamyd Medical should allow the Company to exclusively commercialize its projects. Preservation of patent rights is critical for commercial development of therapeutics for which the economic investment is considerable.

The Company has licensed patents to the therapeutic use of the gene coding for GAD65 and for the treatment of diabetes with GAD65 protein from the University of California, Los Angeles, CA (UCLA) and the University of Florida, Gainesville, FL (UF). The GAD65 gene and its protein also have potential for therapeutic applications in other metabolic and neurological diseases. The licensed patents protect the Company's use of GAD65 until the year 2021 in the U.S.A. and until the year 2016 (including extensions) in Europe.

In addition to the exclusive rights to therapeutic use of GAD65, the Company licenses non-exclusive rights to GAD-based diagnostic applications. Also, U.S.A. patent rights covering a method for identifying non-insulin dependent diabetes patients who are at risk of developing insulin dependent diabetes (type 2 LADA patients) are exclusively licensed from the University of Washington, Seattle, WA.

The Company has been granted its own patent in the U.S.A. and Europe pertaining to a modified GAD molecule that lacks enzymatic activity but retains the protein's immunological potency.

Diamyd Medical has an exclusive worldwide license from the University of Pittsburgh to an intellectual property portfolio covering its NTDDS technologies and lead products. Core patents relate to the modified NTDDS delivery system and were issued between 1997 and 2001. Several additional US and international applications have been filed as recently as 2005 and relate specifically to NP2 and the manufacturing reagents and cell lines. The issued and pending patents protect the NTDDS delivery technology, methods of manufacturing, and proprietary reagents.

In addition to the exclusive rights to the NTDDS delivery technology Diamyd Medical has an exclusive in-license agreement with Centre National de la Recherche Scientifique (CNRS) in Paris for the rights to a patent portfolio covering therapeutic use of GAD via viral vectors. The portfolio consists of active applications in Europe and the U.S.A. with one granted patent in Europe. This granted European patent covers the use of GAD65 and GAD67 in gene therapy for treatment of neurodegenerative diseases using adeno-associated virus.

Competition

Diamyd Medical competes with other life science companies that, in general, are developing therapies for type 1 and type 2 diabetes and more specifically, are developing drugs to prevent or inhibit the autoimmune attack against insulin-producing beta cells.

Diamyd Medical selected the full-length GAD molecule as the active substance in its diabetes therapeutic. Alternative strategies being tested by competitors are based on molecules and antigens other than GAD. These include, but are not limited to, IA-2 (tyrosine kinase), oral insulin, insulin peptide ligands, proinsulin, heat shock protein peptide ligands and anti-lymphocyte antibodies, among others. The following section gives an overview of some of these alternative techniques that are being developed for treatment of autoimmune diabetes.

Eli Lilly and Company has recently teamed up with **MacroGenics, Inc.** for development of its Teplizumab (also called MGA031 and hOKT3γ1(Ala-Ala) anti-CD3 monoclonal antibody. Teplizumab binds to an epitope of the CD3-epsilon chain expressed on mature T lymphocytes and, by doing so, may modulate the pathological immunologic responses underlying multiple autoimmune diseases. MacroGenics is evaluating three dosing regimens in a global pivotal Phase II/III study in type 1 diabetes patients who are up to 12 weeks from diagnosis. Teplizumab is reported to increase the abundance of regulatory T cells and induce tolerance to antigens under attack by the immune system. Side effects such as transient flu-like symptoms and Epstein-Barr Virus reactivation have been reported from earlier studies.

GlaxoSmithKline has recently teamed up with **Tolerx, Inc.** for development of the Otelixizumab (TRX4) anti-CD3 monoclonal antibody. These antibodies target the CD3 receptor that is present on all T lymphocytes. It is reported, after two Phase II studies, that a single 6 day course therapy with Otelixizumab preserves insulin producing beta cells and decreases the required need for administered insulin. Otelixizumab administration was associated with transient flu-like symptoms and Epstein-Barr Virus reactivation.

Merck KGaA has via its acquisition of **Serono S.A.** entered into a partnership with **NovImmune** regarding the NI-0401 anti-CD3 antibody. Phase I/II studies are ongoing for a Crohn's Disease application, but other autoimmune diseases, like type 1 diabetes, may be investigated in the future.

Biogen Idec, in partnership with **PDL Biopharma, Inc.**, is developing Daclizumab (**Zenapax®**) for autoimmune diseases. Daclizumab is a marketed therapeutic based on a murine-human chimaerised monoclonal antibody to the IL-2Rα CD25 receptor of T lymphocytes. It is used as an immunosuppressant to prevent rejection in organ transplantation. An ongoing clinical study by TrialNet in the U.S.A is evaluating Daclizumab alone and in combination with CellCept® to see if immunosuppression can stop the immune system from destroying beta cells in new onset type 1 diabetes.

Rituximab/Mabthera® is an anti-CD20 monoclonal antibody targeting B lymphocytes. This antibody is marketed for other purposes than type 1 diabetes by companies such as **Genentech**, **Biogen Idec** and **Roche**. While originally approved for treatment of B cell lymphomas, and subsequently for rheumatoid arthritis, clinical studies are underway to evaluate the antibody's effect in relapsing multiple sclerosis and type 1 diabetes.

Andromeda Biotech Ltd. a fully owned subsidiary of **Clal Biotechnology Industries Ltd.** in Israel is developing **DiaPep277®**, a 24 amino acid synthetic peptide immunomodulator for the treatment of type 1 diabetes. The product encompasses an immunodominant epitope from the autoantigen heat shock protein 60 (hsp60) and is currently in Phase III clinical development in a global study being carried out in Europe, South Africa and Israel.

The Company's understanding is that Diamyd® has a significant advantage compared to other potential therapies listed above in that it is aimed specifically to tolerize self-reactive T-cells that attack the islet beta cells. This enhances the likelihood of both a positive outcome as well as reduced side effects with Diamyd® treatment. Furthermore, if any of the above-mentioned, or any other unnamed alternative strategy, should be shown to be successful, this does not mean that GAD65 administration would be excluded. Rather, it could be an attractive complement in a combination therapy in which the different treatment strategies confront various pathways of the disease process.

Board, team and auditors

THE BOARD

ANDERS ESSEN-MÖLLER, born 1941, Stockholm, M.Sc. in Engineering, is founder and CEO of Diamyd Medical AB and member of the Board since 1996. Essen-Möller was also founder of Synectics Medical AB, which was sold to Medtronic, Inc., in 1996. Other assignments: Chairman of Arnea AB. Holdings: 561,671 A-shares, 229,298 B-shares, 30,000 subscription options 2004/07 and 10,000 Employee options 2007/2010.

JOSEPH JANES, born 1965 (U.S.A.), Chairman of the Board since 2006 and a board member since 2005. Juris Doctor from The American University, Washington College of Law, Washington, DC. Holdings: 3,877 B-shares and 20,000 subscription options 2004/07.

BJÖRN O. NILSSON, born 1956, Sollentuna, Ph.D., Assoc. Adj. Professor, member of the Board since 2005. Other assignments: Member of IVA, Board member of BioInvent International AB (publ). Dr. Nilsson holds a position as Senior Vice President of Biovitrum (publ). Holdings: 5,000 subscription options 2004/07.

PETER ROTHSCHILD, born 1950, Lidingö, M.Sc. in Economics, member of the Board since 2001. Other assignments: CEO of BioGaia AB. Holdings: 5,000 subscription options 2004/07.

HANS WIGZELL, born 1938, M.D. and Ph.D., member of the Board since 2006. Other assignments: Currently Senior Scientific Advisor for the Swedish Government, The Karolinska Institute, Biocon (India) and HBM Partners (Switzerland). Wigzell is Director of the Board of Directors for Karolinska Innovation AB (Sweden), Karolinska Development I and II AB (Sweden), Biovitrum AB (Sweden), Raysearch AB (Sweden), Probi AB (Sweden) and Intercell (Austria). Holdings: none.

KEY EXECUTIVES

ANDERS ESSEN-MÖLLER is Chief Executive Officer (See the Board)

MICHAEL CHRISTINI is President of Diamyd Inc. Michael Christini received a J.D from Case Western Reserve Law School. Michael Christini founded Nurel Therapeutics in 2003 and joined Diamyd upon its acquisition in December 2005. Prior to joining Nurel, Mr. Christini was an attorney with the US National Institutes of Health and the US Federal Trade Commission in Washington, DC, and with the law firm Cooley Godward, LLP in San Francisco, CA. Holdings: 53,565 B-shares, 5,000 subscription options 2004/07.

CECILIA DRIVING is Chief Financial Officer. Cecilia Driving has received a Master of Laws and a B.Sc. in Business Administration from the University of Stockholm. Cecilia Driving joined the Company in 2007. Holdings: 10,000 Employee options 2007/2010.

ERIKA HILLBORG is Director of Clinical Trials. Erika Hillborg received a M.Sc. in Biomedicine from the Karolinska Institute in Stockholm, and a Science Journalist degree from Uppsala University. Erika Hillborg has worked for the Company since 2006. Holdings: 5,950 B-shares, 15,000 subscription options 2004/07 and 10,000 Employee options 2007/2010.

JOHN ROBERTSON is Director of Research and Development. Dr. Robertson holds a Ph.D. and has worked for the Company since its start. Robertson has an international background in biotechnology and toxicology from academia (Karolinska Institute in Stockholm, Pasteur Institute in Paris, NIH in Washington) and experience in drug development from industry (Inveresk Research International in UK, Schering Agrochemicals in the U.K). Holdings: 26,652 B-shares, 15,000 subscription options 2004/07 and 10,000 Employee options 2007/2010.

PETER ZERHOUNI is Business Developer. Mr. Zerhouni received a M.Sc. in Biology and a B.Sc. in Economics & Business Administration from Lund University and UC Berkeley. From 1999 to 2006 Mr. Zerhouni held various positions at ING Bank in Brussels and Amsterdam, most recently within Structured Finance. Mr. Zerhouni joined the Company in 2006. Holdings: 10,000 Employee options 2007/2010.

EMPLOYEES

MARINA ANNERHALL is Economic Assistant. Marina Annerhall received an Engineer degree from the University of Samarkand. Annerhall has worked for the Company since 2007. Holdings: 10,000 Employee options 2007/2010.

NATALIE JELVEH is Financial Controller. Natalie Jelveh received an M.Sc. in Economics and Applied Biotechnology from Södertörn University College. After graduation she has been involved with sales and finance issues. Natalie Jelveh has worked for the Company since 2006. Holdings: 10,000 Employee options 2007/2010.

DAVID KRISKY is Director of Protein Manufacturing, Diamyd, Inc. Dr. Krisky received his M.D. from the University of Pittsburgh and his Ph.D. in Molecular Genetics and Biochemistry from the University of Pittsburgh and has worked for the Company since 2007. Prior to joining Diamyd, Dr. Krisky was an Assistant Professor in the Department of Pathology at the University of Pittsburgh School of Medicine. At the University of Pittsburgh, Dr. Krisky conducted research in gene vector engineering and manufacturing process development and also served as the Director of the Human Gene Therapy Applications Lab. At Diamyd Dr. Krisky manages the external collaborations relating to the manufacture, formulation and testing of the GAD65 product and also continues his research on HSV gene vector engineering and manufacturing. Holdings: 1,500 B-shares.

JAMES B. WECHUCK is Director of Virus Manufacture, Diamyd, Inc. Dr. Wechuck received his Ph.D in Chemical Engineering, University of Pittsburgh and has worked for the Company since 2006. Prior to joining Diamyd, Dr. Wechuck was a Research Assistant Professor in Bioengineering, University of Pittsburgh and Director of Manufacturing at the University of Pittsburgh Human Gene Therapy Applications Laboratory. Dr. Wechuck's experience includes training and working in a cGMP environment for clinical manufacturing of cell and viral products. At Diamyd, Dr. Wechuck leads the process development, testing and clinical manufacturing of the NTDDS products. Holdings: 3,000 B-shares, 10,000 subscription options 2004/07.

DARREN WOLFE is Director of Preclinical Development, Diamyd Inc. Dr. Wolfe received his Ph.D. in Molecular Biology and Biochemistry, Pennsylvania State University and has worked for the Company since 2006. Dr. Wolfe was a Research Assistant Professor in the Department of Molecular Genetics and Biochemistry at the University of Pittsburgh. Dr. Wolfe manages internal and external preclinical product development efforts. Dr. Wolfe has more than 12 years experience in developing Diamyd's unique therapeutic NTDDS platform and is an inventor on several licensed patents. Holdings: 2,400 B-shares, 10,000 subscription options 2004/07.

AUDITORS

Öhrlings PricewaterhouseCoopers AB has been elected for four years at the annual shareholder's meeting 2006.

EVA RIBEN has been authorized public accountant since 1982 and is Diamyd's auditor since 2006. Eva Riben is the auditor in charge.

LISELOTT STENUDD has been authorized public accountant since 1986 and is Diamyd's auditor since 2006.

Scientific and medical advisory board

The Company has appointed a Scientific and Medical Advisory Board consisting of highly regarded researchers from the U.S.A., The Netherlands, England and Sweden. The following individuals comprise the advisory board:



PROFESSOR MARK ATKINSON, U.S.A., Ph.D., born 1961, is an Eminent Scholar in Diabetes Research at the University of Florida. Dr. Atkinson was amongst the first of researchers to identify the value of measuring immune responses against GAD in persons with type 1 diabetes.

Dr. Atkinson holds positions on a number of scientific advisory boards/research panels including the Juvenile Diabetes Research Foundation (JDRF), the American Diabetes Association (ADA) and the National Institutes of Health (NIH) in the U.S.A. His research efforts have resulted in his being one of the 5 most cited authors in diabetes research as well as his receipt of the highest awards for research accomplishments provided by the JDRF and ADA. Professor Atkinson's current research extends to understanding the immunological mechanisms underlying the formation of diabetes, with his primary research goal involving the development of an effective method for preventing and reversing type 1 diabetes. Professor Atkinson has been a member of the Scientific and Medical Advisory Board since 1997.



PROFESSOR JOSEPH GLORIOSO, U.S.A., Ph.D., is the McElroy Professor and Chairman of the Department of Molecular Genetics and Biochemistry at the University of Pittsburgh, Pennsylvania, U.S.A. Dr. Glorioso is the founder and Director of the University of Pittsburgh Molecular Medicine Institute.

Dr. Glorioso is a recognized expert on herpes simplex virus and gene therapy and is the former president of the American Society for Gene Therapy. Dr. Glorioso is an inventor on several of the Diamyd NTDDS platform patents and has been a member of the Scientific and Medical Advisory Board since 2006.



PROFESSOR DANIEL KAUFMAN, U.S.A., Ph.D., born 1956, is Professor in the Department of Molecular and Medical Pharmacology at the UCLA School of Medicine in Los Angeles, California, U.S.A. Professor Kaufman's current research is focused on GAD and its relation to

diabetes. In a research paper in November 1993, Professor Kaufman demonstrated that the administration of GAD to mice that would otherwise develop type 1 diabetes prevented the outbreak of this disorder. Professor Kaufman was the first to clone a GAD gene and his lab was the first to demonstrate that a GAD treatment could inhibit diabetes in mice with established autoimmune responses. Professor Kaufman was a member of the group associated with Professor Allan J. Tobin, which was the first to submit a patent application for the full cDNA code for GAD, the patent portfolio that Diamyd Medical licenses. Professor Kaufman has been a member of the Scientific and Medical Advisory Board since 1996.



PROFESSOR ALLAN J. TOBIN, U.S.A., Ph.D., born 1942, is Managing Director of MRSSI Inc. MRSSI advises the High Q Foundation and CHDI, organizations dedicated to finding therapeutics for Huntington's disease. Previously, Professor Tobin was Eleanor Leslie Chair of Neuroscience

and Director of the Brain Research Institute at UCLA, Los Angeles, U.S.A. Professor Tobin is also Scientific Director Emeritus of the Hereditary Disease Foundation, which organized the identification of the gene that causes Huntington's disease. Professor Tobin has specialized in the use of molecular methods for synthesis, function and break-down of GABA, which serves as the major inhibitory signal in the brain and the pancreas. Professor Tobin has been a member of the Scientific and Medical Advisory Board since 1996.



PROFESSOR LARS KLARESKOG, Sweden, M.D., Ph.D., born 1945, is Professor of Rheumatology and Head of the Rheumatology Research Laboratory at the Center for Molecular Medicine at Karolinska University Hospital/Karolinska Institute, Sweden. Professor Klareskog's research is specifically aimed at the origin and treatment of autoimmune disorders.

Professor Klareskog has been a member of the Scientific and Medical Advisory Board since 1996.



PAUL KORNBLITH, U.S.A., M.D. is a Director of Pennsylvania BIO (Western PA) and a consultant to the Pittsburgh Life Sciences Greenhouse. Dr. Kornblith is the Founder and Chairman Emeritus, Precision Therapeutics, Inc. and Chairman, Celsense, Inc. Dr. Kornblith is an

expert in neurology and neuro-oncology. He is the former Assistant Professor and Director of Neuro-oncology at Harvard University, the former Chief of Surgical Neurology, NIH-NIHDS/NCI and the former Vice Chairman and Professor of Neurosurgery, University of Pittsburgh. Dr. Kornblith joined the Diamyd Medical's Scientific Advisory Board in 2006.



PROFESSOR DAVID LESLIE, U.K., M.D., Ph.D., born 1949, is Professor of Diabetes and Autoimmunity at the Royal London and St. Bartholomew's School of Medicine, University of London. He has been involved in diabetes research and clinical studies since 1975. Professor Leslie has been

Director of the British Diabetic Twin Study since 1982, the world's largest twin study of its type and Principle Investigator of the European Action LADA consortium. By studying twins, Professor Leslie has been able to show the possibilities for predicting and preventing Autoimmune diabetes. Professor Leslie has been a member of the Scientific and Medical Advisory Board since 1999.



PROFESSOR ÅKE LERNMARK, U.S.A., Med. D., born 1945, is the Robert H. Williams Professor of Medicine at the University of Washington in Seattle. He is also Professor of Experimental Diabetes at Lund University in the Department of Clinical Sciences at the University Hospital MAS

in Malmö, Sweden. Professor Lernmark focused his research on diabetes and at an early stage identified the antigen that later proved to be GAD. He and his colleagues were the first to clone GAD65 from human islets and used biochemical methods to be the first to define antibodies against GAD65 in patients with type 1 diabetes. Professor Lernmark was first to use molecular methods to identify HLA genes that are necessary, but not sufficient to develop the disorder. Professor Lernmark has been a member of the Scientific and Medical Advisory Board since 1996.



PROFESSOR BART O. ROEP, The Netherlands, M.D., Ph.D., is associate professor of medicine and head of the Division of Autoimmune Diseases at the Leiden University Medical Center in The Netherlands. He has focused on the role of auto-reactive T cells in diabetes assessing human cellular

immune responses, autoantigen identification, and islet allograft rejection and the design and immunological monitoring of immunointervention strategies in clinical type 1 diabetes. Professor Roep holds positions on a number of scientific advisory boards/research panels including the Juvenile Diabetes Research Foundation International (JDRF), the Dutch National Research Council, the European Union, the European Foundation for Diabetes Research (EFSD) and the National Institutes of Health (NIH) in the U.S.A.. Professor Roep has been a member of the Scientific and Medical Advisory Board since 2006.



PROFESSOR RICHARD J. WHITLEY, U.S.A., M.D. is the Loeb Professor of Pediatrics; Professor of Pediatrics, Microbiology, Medicine, and Neurosurgery at the University of Alabama at Birmingham, Alabama, U.S.A. Dr. Whitley was a co-founder of Aviron (acquired by Medimmune)

and is a Scientific Advisory Board Member for Gilead. Dr. Whitley's primary research focus is on the molecular pathogenesis of herpes simplex virus infections. Dr. Whitley is also responsible for the National Institute of Allergy and Infectious Diseases Collaborative Antiviral Study Group, a multicenter collaboration to improve the treatment of human herpes simplex virus. Dr. Whitley has been a member of the Scientific and Medical Advisory Board since 2006.



» *The highly successful Diamyd trial in adolescent type 1 diabetes patients is a breakthrough in islet-specific immune intervention therapy in the clinic* «

PROFESSOR BART O. ROEP, head of the Division of Autoimmune Diseases
at the Leiden University Medical Center in The Netherlands

Administration report 2006/2007

The Board of Directors and Chief Executive Officer for Diamyd Medical AB (publ), Registration number 556530-1420 with its domicile in Stockholm, Sweden, hereby presents its Administration Report regarding the operations of the Group and Parent Company for the fiscal year beginning September 1, 2006 and ending August 31, 2007.

Operations

The Company currently develops therapeutics from two independent platform technologies. One of these platforms relies on the GAD65 molecule and the other on a viral delivery system of proteins to nervous tissue. Therapeutics for conditions other than diabetes are also being developed using this system.

Group structure and ownerships

The Diamyd Group consists of the Parent Company Diamyd Medical AB (publ) and three wholly-owned subsidiaries, Diamyd Diagnostics AB, Diamyd Therapeutics AB (Sweden) and Diamyd Inc., PA, U.S.A.

Important events during the fiscal year

In December 2006, an End of Phase II Briefing Package was filed with the US Food & Drug Administration (FDA) and a subsequent End of Phase II/Pre-IND meeting was held in January 2007. The meeting concluded, according to Diamyd Medical's interpretation, that two successful independent Phase III trials, each with about 300 patients, may lead to product registration of Diamyd®. Diamyd Medical plans to submit an application to conduct a Phase III study in the US with the FDA in the near term.

In December 2006, the Company signed an exclusive in-license agreement with Centre National de la Recherche Scientifique (CNRS) in Paris for the rights to a patent portfolio covering therapeutic use of GAD via viral vectors. The portfolio consists of active applications in Europe and the US with one granted patent in Europe. This granted European patent covers the use of GAD in gene therapy for treatment of neurodegenerative diseases using adeno-associated virus.

In May 2007, scientists in Linköping, Sweden, reported preliminary positive results after analyzing immunological parameters from the previously reported type 1 diabetes trial. The data illustrated that patients treated with Diamyd® responded with an up-regulation of certain beneficial cytokines at restimulation with GAD65, 15 months after the first administration.

In June 2007, a Phase II study in 160 LADA type 2 diabetes patients was invalidated after an audit noted violations of GCP guidelines at the pharmacy responsible for the labeling and randomization of the study drug.

In August, 2007, Diamyd Inc., in Pittsburgh held a meeting with the FDA. Diamyd Inc., intends to file an Investigational New Drug (IND) application with the FDA in the near term to initiate a Phase I trial with the proprietary NTDDS-enkephalin drug candidate for cancer pain.

The warrant program DIAM 1999/2006 was fully exercised.

Shareholders invested an additional MSEK 49.2 into Diamyd Medical.

In December 2006, a Swedish institutional investor acquired 70,000 new B-shares in Diamyd Medical at 145 SEK (US\$21) a share, which corresponded to the current market price. The shares were issued with the support of an authorization given at the Annual General Meeting to issue up to 600,000 shares. The transaction generated MSEK 10 in new funding for the Company.

Diamyd Medical's Convertible Promissory Note in Protein Sciences Corporation, CT, U.S.A., was converted into shares as of December 31, 2006. Protein Sciences is manufacturing Drug Substance for the diabetes vaccine Diamyd®. After conversion, Diamyd Medical's stake in Protein Sciences is approximately 6.7% of capital and votes on a fully-diluted basis.

In April 2007, Diamyd's ADRs were listed on the new OTCQX list as a further step in the Company's strategy to increase visibility with American investors.

At the extra shareholders' meeting of Diamyd Medical, held in Stockholm, Sweden, in May, 2007, the shareholders adopted an employee option program. To secure the employee option program it was decided to issue 250,000 warrants. Each warrant shall entitle the holder to acquire one (1) series B-share within three (3) years at a predefined price. The Company shall retain warrants to cover the costs and taxes that the Company will be liable for at execution of the warrants. At full execution, the dilution is calculated to approximately 2.5%.

Negotiations with possible partners have continued, while simultaneously alternative ways to finance the upcoming planned studies have been investigated.

Events subsequent to the period

In September 2007, further evidence for lasting immunologic efficacy of the Diamyd® diabetes vaccine, was presented at the European Association for the Study of Diabetes (EASD) conference in Amsterdam. The analyses were conducted and presented by the team of Professor Johnny Ludvigsson, Linköping University, Sweden. The presentation confirmed that treatment with Diamyd® causes a specific immune response to GAD65 that remained even 15 months after treatment. The immunological effect was observed in patients receiving Diamyd®, but not in patients receiving placebo.

In September 2007, Diamyd Medical announced that the statistically significant protective effect of the experimental Diamyd® vaccine on insulin secretion in type 1 diabetes patients remains 21 months after the first injection.

In September 2007, Diamyd Medical reported that the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) has announced a planned international clinical study with the Diamyd® diabetes vaccine in 126 new onset type 1 diabetes patients on the NIH clinical trials webpage, www.clinicaltrials.gov. The study is

proposed by the NIH/NIDDK sponsored global network TrialNet, a group of the world's foremost experts and key opinion leaders in type 1 diabetes.

The active substance of Diamyd® has been produced at Protein Sciences, Meriden, CT, U.S.A, and is at present subject to testing. Formulation and vialing will subsequently be performed in Europe.

Diamyd Medical increased the investment in Protein Sciences Corporation, Meriden, CT, U.S.A., with another MUSD 1. The investment was made in the form of convertible debt. Diamyd Medical has previously invested MUSD 3, that have been converted into a holding of 6.7% of the shares after full dilution in Protein Sciences. The latest investment makes Diamyd Medical the second largest shareholder in Protein Sciences.

THE DIAMYD GROUP'S SALES AND COSTS

The Group's sales

The Group's sales amounted to kSEK 531 (4 323) and consisted of sales of GAD-protein to researchers. Last year a down payment from a licensing agreement where Diamyd Medical outlicensed the use of GAD65 to Neurologix Inc., NJ, U.S.A. was paid. The Group's sales fluctuate from quarter to quarter as most customers are researchers that purchase the products at initiation of the studies. Of the Group's sales, kSEK 289 (317) originates from Diamyd Inc.

The Group's costs

The Group's running costs were MSEK 56.0 (44.1). The increase in running costs originates from increased investments in Diamyd Inc. and process development and production of the vaccine at Protein Sciences.

The Group's net loss

The Group's net loss after net interest income/expense was MSEK 53.2 (37.7).

The Group's cash flow

The Group's operating cash flow was MSEK 55.7 (-10.8). The Company's cost structure is dominated by costs for research and development which in conjunction with low sales has led to a negative cash flow for the Group since the start. With the current operations, the Company will be long-term dependent on obtaining additional financial resources prior to reaching a cash flow break even.

The Group's liquid assets

The Group's liquid assets, amounted to MSEK 68.8 (13.2) as of August 31, 2007. The Group's liquid assets are being placed mainly in short term funds that are bank guaranteed. During the years 1996-2007, the Group has obtained MSEK 343 in cash through offerings to the public market.

The shareholders' equity for the Group

The shareholders' equity for the Group as of August 31, 2007 was MSEK 105.1 (95.6), giving an equity ratio of 92.0% (91.0%).

Investments

During the fiscal year the investment in Protein Sciences Corporation has been converted to shares and is now a holding. No additional and substantial investments, other than as noted above, have been made during the fiscal year.

External research and development cost

The Group's costs for research and development were MSEK 29.0 (23.2). The research and development costs during the fiscal years from 1996/1997 until 2006/2007 have been 22%, 16%, 48%, 61%, 62%, 38%, 46% 45%, 61%, 51% and 52%, respectively, as a percentage of the Group's total costs. As these costs have been for research purposes, in accordance with IFRS/good accounting practice, these costs are accounted as current costs for the projects.

Financial statements from other holdings

Diamyd Medical holds an ownership of 19% in Mercodia AB (Sweden). The net sales in Mercodia 2006 was MSEK 55.3 (48.2) and the reported profit was MSEK 7.8 (7.2). The value of Diamyd Medical's part of equity in Mercodia is MSEK 4.8 (3.5). The Company has received dividends from Mercodia of kSEK 350 (250) this year.

Diamyd Medical holds an ownership of 6.7% after full dilution in Protein Sciences Corp. (U.S.A.). The net sales in Protein Sciences 2006 was MSEK 63.2 (22.1) and the reported loss was MSEK 6.5 (19.4). The value of Diamyd Medical's part of equity in Protein Sciences is MSEK 2.8 (-).

Staff

The Group had eleven (nine) employees on August 31, 2007.

Six are men and five are women. The staff is employed within the subsidiaries Diamyd Therapeutics AB (seven) and Diamyd Inc. (four). Employee costs were MSEK 13.5 (9.9).

Diamyd Medical AB (publ) – Parent Company

The Parent Company's net sales were SEK 0.00 (0.00) as all sales are managed by the affiliated companies. The net loss of the year before final accounting and taxes was MSEK 47.6 (41.3). Investments for the year were MSEK 0.00 (40.9). Change in liquid assets was MSEK 54.7 (-16.1).

SUMMARY OF DIAMYD MEDICAL'S DEVELOPMENT

Diamyd® Clinical Trials: Type 1 Diabetes

A Phase II trial in 70 children and adolescents with type 1 diabetes is ongoing. GAD-antibody positive type 1 diabetes patients having presented with disease within 18 months were included in the study. Significant efficacy was demonstrated in preserving beta cell function after 15 and 21 months. No serious adverse events related to Diamyd® treatment have been reported. The treatment consisted of only two injections of Diamyd® and was well received by patients, their doctors and family members. In addition, immunology data clearly shows that a response to the Diamyd® vaccine is still present at 15 months in patients vaccinated with Diamyd®. The trial is still in a follow-up phase with results due during the first quarter of 2008.

Diamyd Medical plans to initiate an international Phase III program for type 1 diabetes in the near term.

Diamyd® Clinical Trials: Autoimmune Type 2 Diabetes (LADA)

A Phase II study in 160 type 2 diabetes LADA patients was invalidated during the past year. Five year follow up results are expected in mid 2008 from a Phase IIa trial in 47 LADA patients. Previously it was reported that the most efficacious dose (20 µg) significantly improved both meal-stimulated C-peptide levels and HbA1c at two years after treatment. No serious adverse events related to Diamyd® treatment have been reported in any study.

NTDDS

Diamyd Inc. in Pittsburgh is developing a replication deficient viral delivery system for proteins, in particular, for targeting nervous tissues, the proprietary Nerve Targeted Drug Delivery System (NTDDS). The NTDDS lead projects are therapeutics for treating pain using enkephalin (NP2) and GAD (DG2, DG3). Diamyd plans to file an IND-application with the US FDA to initiate a Phase I trial to test the safety of NP2 in the near term.

GAD and other neurological diseases

Apart from being a major autoantigen in autoimmune diabetes, GAD65 is also an enzyme that converts the excitatory neurotransmitter glutamate into the inhibitory neurotransmitter GABA. Several neurological and movement related disorders may be due to disturbances in the glutamate-GABA balance, and GAD65 may come to play an important role as a component in future medications for treatment of such diseases.

Diamyd Medical has sublicensed rights to the GAD65 gene to Neurologix, Inc. for the development of a GAD-based therapy to treat Parkinson's disease. A Phase I trial with patients having Parkinson's disease has been completed. Primary objectives of the study regarding safety and tolerability were successfully met. Additionally, indications of efficacy were shown. Neurologix, Inc. expects to begin Phase II studies in Parkinson's disease later this year.

Principles for compensation for management

Principles for compensation and other terms of employment for the President and Chief Executive Officer of Diamyd Medical AB are that the compensation is according to market conditions and is a mix of salary, pension benefits or other benefits as well as terms for notice. The President and Chief Executive Officer has been entitled to participate in the employee option program decided at the shareholder's meeting. Only the President has been in the management category. The board of directors proposes that the same principles shall include other key executives for the coming year.

Environmental & ethical policies and quality assurance

Diamyd Medical strives for continual improvement in the fields of environmental & ethical behavior and quality assurance. The use of different animal models for trials of safety and efficacy of Diamyd® is a requirement from the authorities, and is conducted by professional partners regulated by the authorities.

Share Option Plans

DIAM KO 2004/2007

In December 2004, the Company issued 200,000 promissory notes combined with detachable warrants. The subscription price when subscribing warrants to shares using the warrants will be SEK 50 and conveyance of the options shall be estimated to market price, based on Black & Scholes valuation formula. At end of fiscal year 2005/2006 all options were subscribed.

DIAM KO 2007/2010

In May 2007, the Company issued 250,000 warrants. The subscription price when using the warrants will be SEK 146. The cost of the options shall be estimated to market price, based on Black & Scholes valuation formula. According to IFRS 2 and URA 46 the costs for the employee option programs issued in May had an impact on the result with MSEK 0.1 as well as the equity in the Company with MSEK 0.5. At end of fiscal year 2006/2007 80,000 options were outstanding.

Risk Factors

There is no guarantee that Diamyd Medical's research and development will result in commercial success. There is no guarantee that the planned clinical trials will be allowed or that trials conducted by Diamyd Medical can demonstrate sufficient safety and efficacy to obtain the necessary approvals from regulatory authorities, or that they will result in marketable products. There is no guarantee that Diamyd Medical will be able to produce GAD in sufficient amounts and with sufficient quality.

There can be no guarantee that Diamyd Medical will develop products that can be patented, that granted patents can be retained, that future inventions will lead to patents, or that granted patents will be sufficient to protect Diamyd Medical's rights. There may be a need to turn to the capital market for financing in the future. Both the size and the timing of the Company's potential future capital requirements are dependent on a number of factors, including opportunities to enter into collaboration or licensing agreements and the possibility of achieving success in research and development projects undertaken. Generally a biotechnology company such as Diamyd Medical is associated with high risk.

The term "Risk Factors" comprises both internal and external factors that could materially impact Diamyd Medical. The process of developing a pharmaceutical substance is connected with substantial uncertainty as this process deals with novel, unpredictable, complex parameters and biological organisms. An investment in a research and development company, such as Diamyd Medical, results in a high degree of financial risk. Below, are some of the risks and uncertainties that might be of importance when judging whether to invest in Diamyd Medical. The risk factors are not ordered in any particular manner, nor are they fully described.

Risk with regard to the safety and efficacy of Diamyd®

Present data from clinical trials indicate that Diamyd® is safe to administer to humans. Data also demonstrate that Diamyd® has efficacy in patients with autoimmune diabetes. Earlier preclinical data confirm these indications. The results are, however, based on a limited number of people and animals. Uncertainty remains regarding whether safety and efficacy persist when Diamyd® is administered to a larger population of patients. There is a risk that Diamyd® will not have efficacy or induce severe adverse reactions and that Diamyd® then cannot be approved for further clinical testing or finally for market approval.

Risk associated with the manufacture of Diamyd®

Diamyd® is manufactured in cultured cells where the active pharmaceutical ingredient, rhGAD65, is coded with a gene. The manufacturing is highly advanced and demands strict control of a number of parameters in order to be reproducible with regards to quality and quantity. Diamyd Medical progressively scales up the manufacturing capacity to meet an increased demand of Diamyd®. It is uncertain whether Diamyd Medical will successfully manage to manufacture adequate amounts of Diamyd® which could lead to delays in the clinical development of Diamyd®. Diamyd Medical has entered into a collaboration with Protein Sciences Corporation regarding the manufacture of Diamyd®. There is a risk that Protein Sciences will not manage to manufacture Diamyd® according to the demand, and there is a risk that the regulatory authorities will not approve Diamyd® manufactured by Protein Sciences for use in clinical trials in the U.S.A. or in Europe.

Risk regarding intellectual property portfolio

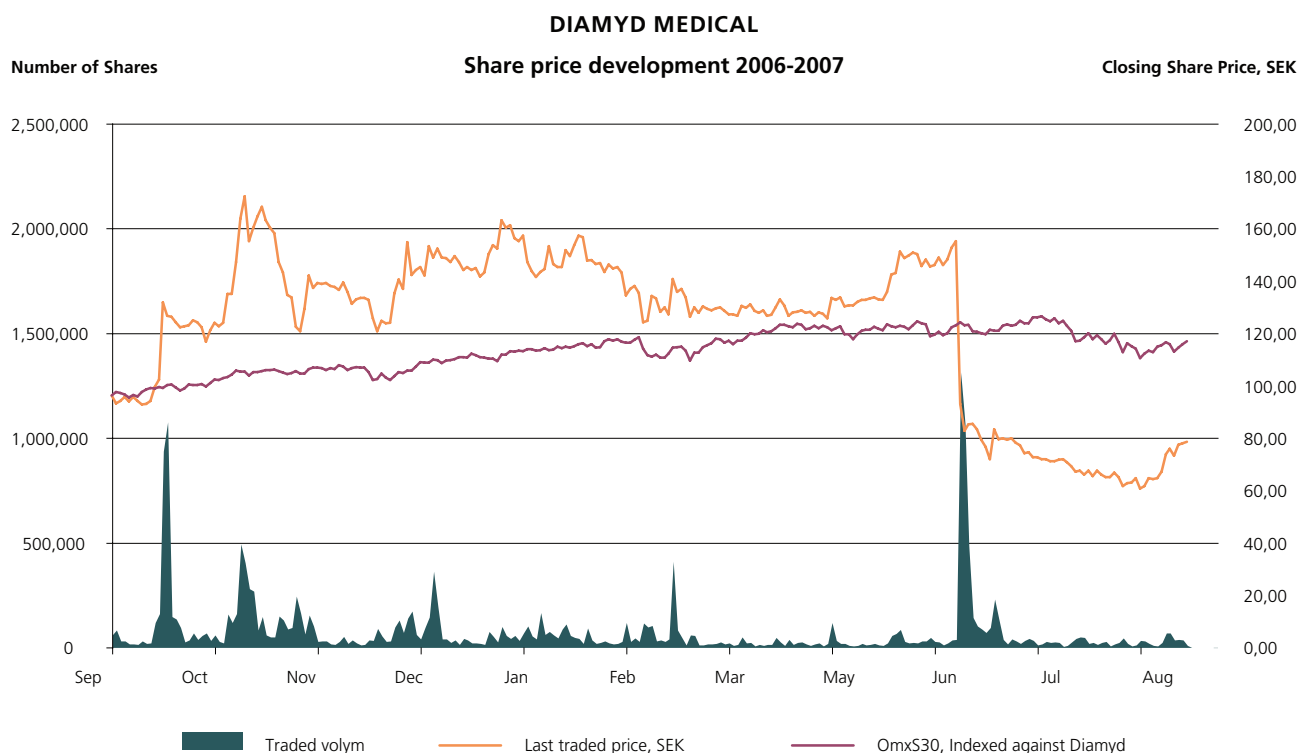
There is a risk that Diamyd Medical's in-licensed intellectual property rights may be insufficient for the Company to commercialize its projects. As Life Science is an area where many patents exist and continues to be granted, the Company is continuously working to protect and expand its rights. There is also a risk that the Company may fail to fulfill its responsibilities under its licenses and that therefore these licenses may be canceled or have limited or no value.

Future competition

A number of other therapies are being developed for the same indications as Diamyd®. There is uncertainty whether competing therapies will succeed and how they might influence sales of Diamyd®.

Uncertainty with future partnerships and deals

Business operations are dominated by transactions that create value. Partnerships are common transactions for sharing the risk of developing a project. There is an uncertainty whether the Company will successfully enter into a transaction that generates value growth for Diamyd Medical.



Financial risks and policies

Diamyd Medical is currently not profitable. The Company is dependent, for the long term, on investments in its operations to make profit in the future. There is a financial risk that the Company will not succeed to secure the adequate financial resources necessary to fully develop its products.

Insurance risks

Diamyd Medical's insurance covers areas such as property, operation breakdown, personal and property damage, legal protection, operational liabilities, clinical trials, business trips, CEO and Board liabilities and product liability. The increased presence in the U.S.A. brings increasing insurance risks. There is no guarantee that Diamyd Medical's insurance is completely adequate to address all risks.

Market development

An investment in Diamyd Medical incurs future uncertainty. There is a risk that an investment in Diamyd Medical will lose all or any part of its value.

ONE YEAR SHARE DATA

On August 31, 2007 the assets of Diamyd Medical were divided among 9,210,807 (8,264,016) shares of series B (1/10 vote) and 561,671 (471,200) shares of series A (one vote). At the end of the fiscal year the share capital in Diamyd Medical was SEK 9,772,478 (8,735,216). There is no limitation on the number of votes a shareholder can emit on a shareholder's meeting.

Shares from series B are listed on the Nordic Stock exchange (ticker symbol SE NOMX:DIAM B) and can also be traded on the new OTCQX list as an American Depositary Recipient (ADR) with Bank of New York as PAL (ticker symbol US ADR:DMYDY). Remium Securities AB is liquidity provider for Diamyd Medical.

The share value of Diamyd Medical was SEK 78.25 (97.00) at end of the fiscal year, which converts to a market capital of MSEK 720.7 (847.3). During the fiscal year the share value decreased 17.8% (+81.3%). In comparison the index OMX Stockholm 30 (OMXS30) increased 22.8% (17.0%). The highest paid share price during the period was SEK 172 (117.5) and the lowest share price during the period was SEK 60.50 (36.20). The average share value during the fiscal year was SEK 124.16 (54.62) and 18,829,365 (7,556,650) Diamyd Medical shares were traded to a total transaction value of MSEK 2,338 (592.8).

Share capital development:

Year	Event	Terms (ration, share price)	Number of shares (increase)	Series A (increase)	Series B (increase)	Share capital (accumulated, SEK)
1996	Company founded*	–	252,575	150,000	102,575	126,288
	Stock dividend**	–	512,500	–	512,500	765,075
96/97	Rights issue	–	771,335	785	770,550	1,536,410
97/98	Rights issue	–	1,536,410	150,785	1,385,625	3,072,820
99/00	Rights issue	–	768,205	75,390	692,815	3,841,025
01/02	Rights issue	1:1, 40 SEK	762,214	94,240	667,974	4,603,239
02/03	Exercise of warrants	1:1, 22,10 SEK	12,232	–	12,232	4,615,471
02/03	Exercise of warrants	1:1, 21,79 SEK	13,106	–	13,106	4,628,557
02/03	Exercise of warrants	1:1, 56,00 SEK	7,801	–	7,801	4,636,378
02/03	Rights issue	2:1, 40 SEK	2,318,189	–	2,318,189	6,954,567
03/04	Directed issue	40 SEK	1,390,913	–	1,390,913	8,345,480
04/05	Exercise of warrants	1:1, 18,36 SEK	72,563	–	72,563	8,418,043
05/06	Non-cash issue	55,00 SEK	317,173	–	317,173	8,735,216
06/07	Warrants	–	912,262	90,471	821,791	9,647,478
06/07	Exercise of warrants	1:1, 50 SEK	30,000	–	30,000	9,677,478
06/07	Exercise of warrants	1:1, 50 SEK	25,000	–	25,000	9,702,478
06/07	Directed issue	145,75 SEK	70,000	–	70,000	9,772,478
Total			9,772,478	561,671	9,210,807	*** 9,772,478

* Share nominal value SEK 0,50 **Share nominal value SEK 1 *** Share Capital divided by the number of Shares is SEK 1

The ten largest shareholders as of August 31, 2007

Name	Series A	Series B	Capital (%)	Votes (%)
Lindkvist, Bertil	–	3,717,303	38%	25,07
Essen-Möller Grp	561,671	211,902	8%	39,31
Östersjöstiftelsen	–	541,608	6%	3,65
Nordnet Pensionsförsäkring AB	–	171,074	2%	1,15
Försäkringsaktiebolaget, Avanza Pension	–	156,630	2%	1,06
SIS Segaintersettle AG/Zürich, W8IMY	–	148,842	2%	1
Merrill Lynch, Pierce, Fenner & Smith, W9	–	119,376	1%	0,81
Gålöstiftelsen	–	114,100	1%	0,77
SEB Trygg Life Ireland Assurance	–	100,000	1%	0,67
Fjärde AP-Fonden	–	99,000	1%	0,67
Total	561,671	5,379,835	61%	74%

Shareholders

On August 31, 2007 the number of shareholders was 4,655 (3,731). The ten largest shareholders held Diamyd Medical shares corresponding to 61.0% (60.8%) of the capital and 74.0% (73.6%) of the votes. According to a shareholders agreement between the main holders Bertil Lindqvist and Anders Essen-Möller, the sole series A holder, Anders Essen-Möller, may not transfer any share of series A to a third party (inheritance transfer excepted) unless the third party buyer commits to first purchase all series B-shares on the same terms and conditions as for the transfer of the series A share. In line with the Articles of Association, an owner of series A shares may freely convert these to series B shares.

Key personnel

The Company is dependent on key personnel. There is a risk that the Company's business and product development will be delayed or halted if key personnel, for any reason, cannot conduct his/her duties. Furthermore, there is a risk that the Board of Directors, the management or key personnel by acting incorrectly may negatively impact the value or perception of the Company.

Litigation

The Company continues its legal dispute with Mercodia AB (Sweden) regarding an amount of kSEK 305 which is not considered in the accounts.

Board Activities

The Board consists of five members. The Board of Directors works according to an established work plan which controls the frequency and agenda of Board meetings, distribution of material to attending members as well as the tasks presented to the Board for information purposes or for decision making. Part of the work plan controls the division of labor among the Board, the Chairman of the Board and the CEO, as well as defining the CEO's authority and salary. The Chairman of the Board prepares Board meetings together with the CEO. Apart from deciding on the Company's strategy, business ideas, scientific and financial plans, the Board also monitors the Company's operations and development. The CEO and management team report on operations at the Board meetings, including development and progress within research and business areas as well as presenting financial reports. The Board makes decisions regarding important issues such as significant contracts, budgets, financial policy and large investments. According to the work plan at Diamyd Medical, the Board will stage at least 4 Board meetings per calendar year apart from the constitutional Board meeting. The Board held 15 minute-recorded meetings during the most recent fiscal year.

The Company's appointed accountants report their accounting directly to the Board. Questions arising from accounting are considered to be of such importance to warrant handling by the Board and not by a separate audit committee.

The Company has neither a nomination committee nor a dividends committee, but such issues are dealt with by the Board. Currently, the Code of Conduct does not apply to Diamyd Medical.

Payments to the Board members amounted to kSEK 495 of which kSEK 165 was for the Chairman of the Board in accordance with the Bulletin from the Annual General Meeting of Shareholders 2006. Payment to the accountants has been according to a fixed retaining fee.

The group's future development

Diamyd Medical intends to continue to focus on building shareholder value. Partnership discussions are ongoing regarding commercialization of Diamyd®. At the same time preparations for the Phase III program continues. Market approval of the product is conditioned of completed successful Phase III-trials. The cash is estimated to last until the end of 2008 as long as the Phase III program is not initiated.

PROPOSAL FOR THE TREATMENT OF THIS YEAR'S LOSS

The parent company's accumulated loss according to the balance sheet:

SEK	
Loss from previous year	75,607,207
Net loss of the year	47,641,257
Accumulated loss	123,248,464

The Board of Directors proposes that SEK 123,248,464 is transferred from share premium reserve non-restricted and statutory reserves of the parent company to cover this years loss of SEK 123,248,464.

The Company's outcome of the fiscal year and financial balance per August 31, 2007 is presented in attached Income Statement, Balance Sheet, Cash Flow Statement and compilation over changes in Shareholders equity with pertaining notes, which constitute an integrated part of this annual report.

DIVIDENDS

The Board of Directors proposes no dividends are issued as a result of operations during the fiscal year 2006/2007.



Group's Consolidated Income Statement

kSEK		12 months Sep-Aug 2006/2007	12 months Sep-Aug 2005/2006	12 months Sep-Aug 2004/2005
	Note			
OPERATING INCOME				
Net sales	1	531	4,323	883
Other operating income		540	126	48
Total operating income	2	1,071	4,449	931
OPERATING EXPENSES	2			
Raw material and consumables		-18	-166	-775
External research and development cost		-29,049	-23,167	-24,676
Patents- and licenses expenses		-1,908	-1,471	-1,719
Personnel	5, 6	-13,554	-9,876	-8,698
Other external expenses	7, 8, 9	-10,941	-8,680	-4,055
Depreciation, patents	10, 14	-403	-656	-751
Depreciation, equipment	11	-146	-115	-147
Total operating expenses		-56,019	-44,131	-40,821
OPERATING LOSS		-54,948	-39,682	-39,890
Financial income and expenses				
Dividends from from other bonds		350	250	152
Other interest income and similar items	12	2,574	1,808	3,195
Other interest expense and similar items	12	-1,447	-56	-26
Total financial income and expenses		1,477	2,002	3,321
Loss before taxes		-53,471	-37,680	-36,569
Taxes	13	266	-	-63
NET LOSS FOR THE PERIOD	10	-53,205	-37,680	-36,632
Earnings per share before dilution, SEK	3	-5.5	-4.4	-4.4
Earnings per share after dilution, SEK	3	-5.5	-4.4	-4.4
Number of shares	3	9,772,478	8,735,216	8,418,043
Average number of shares	3	9,659,558	8,582,797	8,410,787
Number of shares after dilution	3	9,750,960	9,544,076	8,442,800



Group's Consolidated Balance Sheet

kSEK	Note	Aug 31 2007	Aug 31 2006	Aug 31 2005
ASSETS				
NON-CURRENT ASSETS				
Intangible assets	10, 14	16,885	17,715	1,309
Tangible assets	11	414	133	220
Financial assets	15	21,418	800	800
Total non-current assets		38,716	18,648	2,329
CURRENT ASSETS				
Inventory	16	11	12	8
Trade receivables		86	148	450
Other receivables		3,107	2,879	1,536
Prepaid tax		789	326	168
Prepaid expenses and accrued income	17	2,709	2,600	5,447
Other investments	32	–	21,735	–
Short-term investments	18, 19	–	45,551	91,374
Liquid assets	4, 20	68,803	13,190	24,161
Total current assets		75,505	86,441	123,144
TOTAL ASSETS		114,221	105,089	125,473
SHAREHOLDERS' EQUITY AND LIABILITIES				
Shareholders' equity				
Issued capital	21			
	28, 29, 30	9,772	8,735	8,418
Other capital contributions		349,995	288,938	271,571
Other reserves		311	160	30
Accumulated losses		-254,944	-202,231	-164,551
Total shareholder's equity	10	105,134	95,602	115,468
Current liabilities				
Trade payables		4,016	1,624	2,508
Other payables	22	220	2,114	1,745
Prepaid income and accrued expenses	23	4,851	5,749	5,752
Total current liabilities		9,087	9,487	10,005
TOTAL SHAREHOLDERS' EQUITY AND LIABILITIES		114,221	105,089	125,473

Information regarding Groups assets pledged, see Note 33.



Change in Shareholder's Equity (Group)

kSEK	Share Capital	Other Capital Contributions	Other reserves	Accumulated losses	Total
Opening balance, September 1, 2004	8,345	271,144	28	-127,919	151,598
Translation gain	–	–	2	–	2
Total revenues and costs posted directly to shareholders' equity	–	–	2	–	2
Net loss for the period	–	–	–	-36,632	-36,632
Total revenues and costs	–	–	2	-36,632	-36,630
New share issue	64	-64	–	–	–
New share issue	9	131	–	–	140
Paid option premiums, note 29	–	360	–	–	360
Closing balance, August 31, 2005	8,418	271,571	30	-164,551	115,468
Adjustment according to IAS 39	–	–	530	–	–
Opening balance, September 1, 2005	8,418	271,571	560	-164,551	115,998
Translation gain	–	–	207	–	207
Revaluation of short-term investments	–	–	-607	–	-607
Adjusted financials, note 10	–	–	–	970	970
Total revenues and costs posted directly to shareholders' equity	–	–	-400	970	570
Net loss for the year	–	–	–	-38,650	-38,650
Total revenues and costs	–	–	-400	-37,680	-38,080
Option premiums, note 29	–	240	–	–	240
New share issue	317	17,127	–	–	17,444
Closing balance, August 31, 2006	8,735	288,938	160	-202,231	95,602
Opening balance, September 1, 2006	8,735	288,938	160	-202,231	95,602
Revaluation of short-term investments	–	–	77	–	77
Translation gain	–	–	74	–	74
Total revenues and costs posted directly to shareholders' equity	–	–	151	–	151
Net loss for the period	–	–	–	-53,205	-53,205
Total revenues and costs	–	–	151	-53,205	-53,054
New share issue	912	48,332	–	–	49,244
Option premiums, note 29	55	2,695	–	–	2,750
New share issue	70	10,030	–	–	10,100
Employee option, note 30	–	–	–	492	492
Closing balance, August 31, 2007	9,772	349,995	311	-254,944	105,134



Parent Company's Income Statement

kSEK		12 months Sep-Aug 2006/2007	12 months Sep-Aug 2005/2006	12 months Sep-Aug 2004/2005
OPERATING EXPENSES				
Employee benefit expenses		–	–	-522
Other external expenses	7, 8, 9	-17,019	-5,789	-394
Total operating expenses		-17,019	-5,789	-916
OPERATING LOSS				
		-17,019	-5,789	-916
FINANCIAL INCOME AND EXPENSES				
Results from group participation	24	-32,005	-37,495	-37,287
Dividends from from other bonds		350	250	152
Interest income and similar items	12	2,459	1,708	3,114
Interest expense and similar items	12	-1,426	-1	-13
Total financial income and expense		-30,622	-35,538	-34,034
Loss before taxes				
		-47,641	-41,327	-34,950
Taxes	13	–	50	–
NET LOSS FOR THE PERIOD		-47,641	-41,277	-34,950



Parent Company's Balance Sheet

kSEK	Note	Aug 31 2007	Aug 31 2006	Aug 31 2005
ASSETS				
Non-current assets				
<i>Intangible assets</i>				
Licences and similar assets	10, 14	16,627	16,627	–
<i>Financial assets</i>				
Shares in group companies	26	1,701	1,209	1,209
Receivables at group companies	27	6,784	178	1,563
Other long term bond holdings	15	21,418	800	800
Total non-current assets		46,530	18,814	3,572
CURRENT ASSETS				
Other receivables		398	670	345
Prepaid expenses and accrued income	17	1,424	1,636	4,115
Other investments	32	–	21,735	–
TOTAL TRADE AND OTHER RECEIVABLES		1,822	24,041	4,460
Short-term investments	18, 19	–	45,551	91,374
Liquid assets	4	59,631	4,915	21,022
TOTAL CURRENT ASSETS		61,453	74,507	116,856
TOTAL ASSETS		107,983	93,321	120,428
SHAREHOLDERS' EQUITY AND LIABILITIES				
SHAREHOLDERS' EQUITY				
Restricted equity				
Issued capital	28, 29, 30	9,772	8,735	8,418
Statutory reserve		141,673	141,673	–
Share premium reserve restricted		–	–	141,433
Non-restricted equity				
Share premium reserve non-restricted		78,184	17,127	–
Profit or loss brought forward		-75,607	-34,899	–
Net loss		-47,641	-41,277	-34,950
Total shareholder's equity		106,381	91,359	114,901
Long term liabilities to subsidiary	25	181	231	4,149
CURRENT LIABILITIES				
Trade payables		630	124	40
Other payables		72	840	839
Prepaid income and accrued expenses	23	719	767	499
Total current liabilities		1,421	1,731	1,378
TOTAL SHAREHOLDERS' EQUITY AND LIABILITIES		107,983	93,321	120,428
Assets pledged		157	–	–
Contingent liabilities		–	–	–



Change in Shareholder's Equity (Parent)

kSEK	Share capital	Statutory reserve	Share premium reserve restricted	Share premium reserve non-restricted	Accumulated losses	Total
Opening balance, September 1, 2004	8,345	–	157,860	–	-16,853	149,351
New share issue	64	–	-64	–	–	–
New share issue	9	–	131	–	–	140
Option premiums	–	–	360	–	–	360
Result allocation	–	–	-16,853	–	16,853	–
Net loss for the period	–	–	–	–	-34,950	-34,950
Closing balance, August 31, 2005	8,418		141,433		-34,950	114,901
Adjustment according to IAS 39		–	–	–	530	530
Opening balance, September 1, 2005	8,418	–	141,433	–	-34,420	115,431
Reclassification	–	141,433	-141,433	–	–	–
New share issue	317	–	–	17,127	–	17,444
Option premiums	–	240	–	–	–	240
Group contribution received	–	–	–	–	177	177
Tax effect received group contribution	–	–	–	–	-49	-49
Net loss for the period	–	–	–	–	-42,247	-42,247
Revaluation of short-term investments	–	–	–	–	-607	-607
Adjusted financials, note 10	–	–	–	–	970	970
Closing balance, August 31, 2006	8,735	141,673	–	17,127	-76,176	91,359
Opening balance, September 1, 2006	8,735	141,673	–	17,127	-76,176	91,359
New share issue	912	–	–	48,332	–	49,244
Option premiums, note 29	55	–	–	2,695	–	2,750
New share issue	70	–	–	10,030	–	10,100
Revaluation of short-term investments	–	–	–	–	77	77
Employee option, note 30	–	–	–	–	492	492
Net loss for the period	–	–	–	–	-47,641	-47,641
Closing balance, August 31, 2007	9,772	141,673	–	78,184	-123,248	106,381

Cash Flow Statement

kSEK	Group			Parent		
	12 months	12 months	12 months	12 months	12 months	12 months
	Sep-Aug 2006/2007	Sep-Aug 2005/2006	Sep-Aug 2004/2005	Sep-Aug 2006/2007	Sep-Aug 2005/2006	Sep-Aug 2004/2005
Cash flow from operations before changes in working capital						
Operating loss	-54,948	-39,682	-39,890	-17,019	-5,789	-916
Interest received	3,051	4,304	1,444	2,920	4,219	1,396
Interest paid	-26	-56	-26	-5	-1	-13
Dividend received	350	–	152	350	–	152
Non-cash flow items						
Depreciation	549	771	898	–	–	–
Changes in accrued interest	-477	-2,496	1,751	-461	-2,511	1,718
Other non-cash flow items	568	1,933	–	-255	1,934	–
Income tax paid	-205	-158	-119	-20	–	–
Net cash flow from operating activities before changes in working capital	-51,138	-35,384	-35,790	-14,490	-2,148	2,337
Cash flow from investing activities						
Increase (-) Decrease (+) Inventory	-1	-5	82	–	–	–
Increase (-) Decrease (+) Receivables	-278	2,040	-4,455	503	2,404	-2,871
Increase (+) Decrease (-) Liabilities	-138	680	3,892	458	-417	2,268
Net cash flow from operating activities	-51,555	-32,669	-36,271	-13,529	-161	1,734
Cash flow from investing activities						
Group's contributions	–	–	–	-32,005	-37,316	-37,287
Purchase of intangible assets	–	-436	–	–	–	–
Purchase of tangible assets	-435	-28	-30	–	–	–
Purchase of financial assets	45,551	22,077	-1,766	45,551	22,077	-1,766
Net cash flow from investing activities	45,116	21,613	-1,796	13,546	-15,239	-39,053
Cash flow from financing activities						
Change in long-term liabilities	–	-768	–	-7,395	1,385	7,866
Option premiums	–	–	–	–	-3,150	–
New share issue	62,094	1,058	500	62,094	1,058	500
Change in short term	–	–	–	–	–	–
Net cash flow from financing activities	62,094	290	500	54,699	-707	8,366
Total cash flow for the period	55,655	-10,766	-37,567	54,716	-16,107	-28,953
Cash and cash equivalents at beginning of period	13,190	24,161	61,730	4,915	21,022	49,975
Net foreign exchange difference	-42	-205	-2	–	–	–
Cash and cash equivalents at end of period	68,803	13,190	24,161	59,631	4,915	21,022

Accounting principles

GROUP

Diamyd has prepared its consolidated financial statements in accordance with IFRS, International Financial Reporting Standards, as endorsed by the EU. The same principles were applied to the Annual Report for 2005/2006. In addition to IFRS, the Group also observes RR's (Redovisningsrådet, the Swedish Financial Accounting Standards Council) recommendations RR 30 (Supplementary accounting regulations for groups) and The Swedish Annual Accounts Act (Årsredovisningslagen). The Diamyd group presents its income statement by cost class, implying that operating costs are divided between external research and development costs, other external costs and personnel costs as well as depreciation, amortization and write-downs. The essential significance of the new IFRS standards is stated in the following headings, in which the principles of annual reporting are reviewed in more detail.

PARENT COMPANY

Diamyd Medical AB, with residence in Stockholm, continues to apply those accounting principles relevant to legal entities that prepare consolidated financial statements and are listed on a stock exchange. Briefly, this implies the continued applications of RR's recommendations to the extent that they are applicable to a group parent company. Thus Diamyd Medical AB observes RR 32:05, Accounting for legal entities. The parent company's accounting principles are consistent with the group unless stated otherwise below. Pursuant to RR 32:05, the Parent Company will structure its reports in accordance with all applicable IFRS/IAS unless the standards state an exception from their application. The board of directors has on the 20th of November 2007 approved this Consolidated Financial Statement to be publicized.

BASIS FOR VALUATION

The Group uses acquisition values for balance sheet items unless otherwise stated.

CONSOLIDATED FINANCIAL STATEMENTS

The Group comprises Parent Company Diamyd Medical AB, the wholly owned Swedish subsidiaries Diamyd Therapeutics AB and Diamyd Diagnostics AB and the American subsidiaries Diamyd Inc. PA and the American subsidiary Diamyd Inc. NJ under liquidation. The consolidated financial statements have been prepared pursuant to IAS 27 and IFRS 3 on consolidated financial statements and by adopting the purchase method of accounting, implying that subsidiary equity is eliminated at the time of acquisition. Moreover, the preparation of Diamyd's consolidated financial statements conforms to the stipulations of IAS 27 and IFRS 3 including elimination of intra-group receivables and liabilities as well as intra-group revenues and costs, implying that the consolidated income statement and consolidated balance sheet are prepared without intra-group trans-

actions. The untaxed reserves of individual companies are divided between shareholders' equity and deferred tax liabilities.

Subsidiary is companies (including companies with special purposes) which the Group has the right to establish financial and operative strategies in a way that usually follows with a holding in shares of more than 50% of the total voting rights. Occurrence and effects of potential voting rights that is present to utilize or convert, the existence and effect of potential voting rights that are currently exercisable or convertible are considered when assessing whether the Group controls another entity. Subsidiaries are fully consolidated from the date on which control is transferred to the Group. They are de-consolidated from the date that control ceases.

The purchase method of accounting is used to account for the acquisition of subsidiaries by the Group. The cost of an acquisition is measured as the fair value of the assets given, equity instruments issued and liabilities incurred or assumed at the date of exchange, plus costs directly attributable to the acquisition. Identifiable assets acquired and liabilities and contingent liabilities assumed in a business combination are measured initially at their fair values at the acquisition date, irrespective of the extent of any minority interest. The excess of the cost of acquisition over the fair value of the Group's share of the identifiable net assets acquired is recorded as goodwill. If the cost of acquisition is less than the fair value of the net assets of the subsidiary acquired, the difference is recognised directly in the income statement.

FOREIGN CURRENCY TRANSLATION

Functional currency and presentation currency

Diamyd has a foreign subsidiary; Diamyd Inc. Items included in the financial reports for this entity within the Group are valued in the currency used in the primary economic environment in which the Company operates (presentation currency). Swedish krona (SEK), the parent company's functional and presentation currency is utilized in the consolidated financial statements.

TRANSACTIONS AND BALANCES

Foreign currency transactions are translated to the functional currency using the exchange rates prevailing at the dates of the transactions. Foreign exchange gains and losses resulting from the settlement of such transactions, and from the translation at year-end exchange rates of monetary assets and liabilities denominated in foreign currencies, are recognized in the Income statement. Gains and losses on trading receivables and liabilities are recognized net under other operating income or other operating costs.

GROUP COMPANIES

The results and financial positions of all the group entities (none of which has the currency of a hyperinflationary economy) that have a functional currency different from the presentation currency are

translated into the presentation currency as follows: **(I)** assets and liabilities for each balance sheet presented are translated at the closing rate at the date of that balance sheet; **(II)** income and expenses for each income statement are translated at average exchange rates (unless this average is not a reasonable approximation of the cumulative effect of the rates prevailing on the transaction dates, in which case income and expenses are translated at the rate on the dates of the transactions), and **(III)** all resulting exchange differences are recognized as a separate component of equity.

FINANCIAL INSTRUMENTS, ACCOUNTING, DISCLOSURE AND CLASSIFICATION

At every balance sheet date the Group makes the estimate if there exist objective evidence for write-down requirement for a financial assets or a group of financial assets. Regular purchases and sales of financial assets are recognised on the trade-date – the date on which the Group commits to purchase or sell the asset. Investments are initially recognised at fair value plus transaction costs. Financial assets are derecognised when the rights to receive cash flows from the investments have expired or have been transferred and the Group has transferred substantially all risks and rewards of ownership.

Financial assets that can be sold

Financial assets that can be sold is assets that is not derivative and assets that are identified as sellable or not classified in some other category. They are included in non-current assets as long as management does not have the intention to dispose of the assets within 12 months after balance sheet date. Investments in companies, with an exception of companies classified as associated companies, are disclosed through equity at market value. Dividends on shares that can be sold is accounted as a dividend from other bonds when the Groups right to receive the payment is established.

If the market for a financial asset is not active (and for unlisted securities), the Group establishes fair value by using valuation techniques. These include the use of recent arm's length transactions, reference to other instruments that are substantially the same, discounted cash flow analysis and option pricing models, making maximum use of market inputs and relying as little as possible on entity-specific inputs.

In the case of equity securities classified as available for sale, a significant or prolonged decline in the fair value of the security below its cost is considered as an indicator that the securities are impaired. If any such evidence exists for available-for-sale financial assets, the cumulative loss – measured as the difference between the acquisition cost and the current fair value, less any impairment loss on that financial asset previously recognised in profit or loss – is removed from equity and recognised in the income statement. Impairment losses recognised in the income statement on equity instruments are not reversed through the income statement.

Short-term investments in the balance sheet are classified as financial assets available-for-sale and fair value adjustments are accounted for directly in equity through the change in shareholders' equity, except for write-downs, currency gains (losses), until the financial assets are derecognised at which date the accumulated gains or losses that had previously been recognised in equity are accounted in profit and loss.

No futures were utilized in the year. More information on financial risks and investments is in Note 19, Financial risks. Diamyd adopted IAS 39 in its Consolidated Financial Statements from 1 January 2005 onwards.

Loans and receivables

Accounts receivable are non-derivative financial assets, with determined or determinable payments that are not listed on an active market. Their distinguishing feature is that they arise when the group provides funds, goods or services direct to a customer without any intention to trade in the arising receivable. They form part of current assets, apart from items with maturities more than 12 months from the balance sheet date, which are classified as fixed assets. Trade receivables are recognised initially at fair value and subsequently measured at amortized cost using the effective interest method, less provision for impairment. A provision for impairment of trade receivables is established when there is objective evidence that the Group will not be able to collect all amounts due according to the original terms of the receivables. The amount of the provision is the difference between the asset's carrying amount and the present value of estimated future cash flows, discounted at the original effective interest rate. The amount of provision is accounted in the income statement.

Other receivables, and where applicable interim receivables, are accounted pursuant to the same principles.

Other investments are classified as loans and receivables referring to convertible debts, which has been valued at amortised cost.

STOCK OPTION PLANS

Scope

As of balance sheet date, Diamyd has one outstanding stock option plan. Upon conversion/exercise, liquid funds would increase by the exercise/conversion price and the share capital by a nominal SEK 1 per share, with the remaining deposited amount increasing equity. For more details on the various effects of each plan and the number of outstanding stock options, please refer to note 30, Warrants and stock options.

Accounting policies for stock option plans

Diamyd reports its employee option plan in accordance with IFRS 2 and IFRIC 11. The recommendation implies that Diamyd values the current plan 2007/2010 at the time of issuance at actual value and

then distributes the value over the earnings period as a personnel cost. This remuneration to personnel implies that Diamyd issues its own capital instrument (warrants to which personnel are entitled, pursuant to the plan's agreements) and thus, for the cost associated with each period, achieves the corresponding decrease in accumulated losses. The stock options attributable to personnel in the subsidiary Diamyd Therapeutics AB., are accounted in accordance with IFRIC 11. Here, Diamyd's issue of a capital instrument is defined as a shareholder contribution to the subsidiary from the Parent Company, implying that this is reported as investment in a subsidiary. Like other contributions, the investment is then evaluated for any write-down requirement. If there is a need to write down shares in a subsidiary, the effect is that a financial cost is posted to Diamyd AB's Income Statement.

Social security costs on stock option plans

For each outstanding plan, Diamyd makes provisions for social security costs for each outstanding plan at the end of each accounting year. The provision for social security costs is calculated according to URA 46, with the application of the same valuation model used when the options were signed. The provision is revalued on each reporting date on the basis of a calculation of the charges that may be payable when exercise takes place. Diamyd effects the valuation according to the Black & Scholes model, which takes into account factors including the share price, remaining time until exercise, volatility and risk-free interest rates. Payments of social security costs in connection with employee exercise of options are offset against the provision made according to the above. To cover the social security costs inherent in its stock option plans, Diamyd is disposing a number of options intended for conversion into shares, which are then sold to finance payment of the associated social security costs. Because a taxable benefit (the difference between the redemption/exercise price and market value of shares) arises when stock options are exercised, Diamyd can cover the social security costs on the taxable benefit by converting a portion of its options to shares and then divesting them. However, the personnel cost arising in the income statement, which is provisioned progressively pursuant to URA 46, will not be offset by a cost reduction (income), but the effect arises in cash flow terms exclusively.

SHARE OPTION PLANS

Options attached to future issues of shares in the Company and the value of which has been appointed to the market value are recognized as equity when the purchase price has been transferred.

The shares in the share option plan of the Company are classified as marketable securities (IFSR 2) and are exercised at market value. As the share option plan is given the market value, the only effect of the share option plan on the accounts is with respect to received funds for execution of options and conversion to share capital. Calculated dilution effects are presented under Earnings per Share

and appointed market value is calculated in accordance with the Black-Scholes valuation model, see Note 29 for significant assumptions. Received revenues, net after direct transaction expenses, are recognized as Share Capital and Non-Restricted Reserves when the options are exercised.

INTANGIBLE ASSETS

Intangible assets refers to license rights, acquired directly or via a business combination. Expenses for acquiring patent licenses are shown as an asset if the patent is the basis for a product or if the license is judged to have a market value equivalent to the value shown. Amortization is calculated using the straight line method, starting with the acquisition value and the estimated economic life expectancy. Patent maintenance costs are continually reported. The research activities Diamyd conducts are associated with sufficient uncertainty that IAS 38's capitalization criteria cannot be considered satisfied. Accordingly, all research and development costs are written off as they arise.

Property, plant and equipment

Depreciation

Diamyd calculates depreciation using the straight-line method to allocate the cost of property, plant and equipment over its estimated useful life. In accordance with IAS 16 for property, plant and equipment and IAS 38 for intangible assets, depreciation/amortization according to plan is calculated on original acquisition values with depreciation rates based on estimates of the assets' financial life-span.

IMPAIRMENT

Review of tangible and intangible fixed assets for impairment is performed whenever internal or external indications of a potential write-down requirement arise pursuant to IAS 36. For assets with an unlimited useful life, including goodwill, this examination is performed per balance sheet date, irrespective of whether there is an indication of decreased value or not. Examination is performed through an estimation of the recoverable amount. The recoverable amount is the higher of an asset's value in use and fair value less costs to sell. If the recoverable value is lower than the asset's carrying amount, an impairment loss is recognized.

EQUITY

Changes in equity, with comparative years, in the group and Diamyd Medical AB in the context of accounted profits, deficits brought forward, appropriation of profits, exchange rate differences and share capital is accounted on pages 36 and 39. Consolidated shareholders' equity is reported in accordance with the Swedish Financial Accounting Standards Council's instructions URA 47 on reporting equity in groups.

REVENUE RECOGNITION

Revenue comprises the fair value of the services sold exclusive of value added tax and discounts and after elimination of sales within the Group. The Group recognizes revenue when the amount of revenue can be reliably measured, it is probable that future economic benefits will flow to the entity and specific criteria have been met for each of the Group's activities as described below.

The income amount is not regarded to be measurable in a reliable manner until all commitments regarding sales have been met or expired. The Group bases its assessments on historical outcomes and, in this context, considers the type of customer, the type of transaction and special circumstances in each individual case. Following specific criterions most also be fulfilled before revenues will be accounted.

Sales of Goods

Revenue is recognized when the significant risks and rewards of ownership of the Goods have passed to the buyer and the amount of revenue can be measured reliably.

Interest income

Revenue is recognized as the interest accrues and can be reliably measured (taking into account the effective yield on the asset).

Revenue from out-licensing of technology

Revenue from out-license is recognized to the extent that it is probable that the economic benefits will flow to the Group as it accrues and can be reliably measured.

Dividend

Dividend income is accounted for when the right to receive dividend is established.

SEGMENT INFORMATION**Primary reporting format – business segments**

The Diamyd Medical Group has divided sales into two business segments: GAD technology and NTDDS technology. The grouping has its basis in risk and opportunity in the different projects.

Secondary reporting format – geographical segments

Geographic Regions provide products and services within a financial environment that has risks and opportunities other than other financial environments.

PENSION LIABILITY AND PENSION COSTS

Diamyd applies IAS 19, all company employees are provided with individual pension schemes which are chargeable and for which the Company has an agreement with an insurance company to administer these schemes. For defined contribution plans, the Group pays contributions to publicly or privately administered pension insurance

plans on a mandatory, contractual or voluntary basis. The Group has no further payment obligations once the contributions have been paid. The contributions are recognised as employee benefit expense when they are due. Prepaid contributions are recognised as an asset to the extent that a cash refund or a reduction in the future payments is available.

INCOME TAX

Deferred tax is reported in total in accordance with the balance sheet method, on all temporary differences that arise between the tax base on assets and liabilities and the reported values in the consolidated financial statement. The deferred tax is not reported if it occurs from a new transaction if it is not a business combination, at the time for the transaction and it will not affect the reported or the fiscal value. Deferred tax assets is calculated according to applicable tax rate (and laws) that has been adopted or announced on closing day and is expected to be in force when the deferred tax will be realized or the deferred tax liability will be settled.

Deferred tax is calculated on temporary differences which arise on participation in group companies and in associated companies except when the timing of the deferred tax assets can be controlled by the Group and it is likely that the temporary difference will not be reversed in the foreseeable future.

Pursuant to IAS 12, deferred tax assets should only be reported to the extent that it is likely that deductions will be utilized. Note 13 reports items including the estimated deductible deficits accumulated in the Group. The taxable deficits of the Group have no expiry. The treatment of potential deferred tax on temporary differences is reported and explained in Note 13. The various constituent items of consolidated total tax are also explained. The positive tax amount is pursuant to a revaluation of the deficits in the subsidiary Diamyd Inc.

RELATED-PARTY TRANSACTIONS

Diamyd accounts remuneration and benefits to senior executives pursuant to IAS 19 Employee benefits and IFRS 2 Share-based payment. Other information on closely related parties is accounted pursuant to IAS 24. A specification of the various amounts is in Note 6.

SIGNIFICANT ESTIMATES

When the Board and President prepare reports in accordance with generally accepted accounting principles, certain assessments and assumptions must be made that affect the values recorded in the final accounts. These assessments and assumptions constitute the basis of the recorded values of assets, liabilities, revenues and expenses in those cases where these cannot be determined simply through information from other sources. The areas which contain a high degree of assessment, which are complex or in which assumptions and estimations are of considerable importance comprise above all the Diamyd Group's non-current assets and a current investment in a convertible bond that is not subject to trade.

- *Intangible assets:* Licenses and intellectual property rights are valued at acquisition value with straight line amortisation during the estimated economic life time. The write-down requirement is assessed on the basis of the net expected future cash flow.
- *Financial assets:* Holdings in other companies than associated companies is to be write-down if there is a ascertained remaining decreased value. These assets are valued on available information continuously.

CASH FLOW STATEMENT

The cash flow statement has been prepared in accordance with IAS 7 and with application of the indirect method. Recognized cash flow only encompasses those transactions implying payments made or received. In accordance with IAS 7, liquid funds include cash and bank balances, plus short-term investments such as commercial papers, fixed-income and bond funds with maximum duration of three months.

THE INTRODUCTION OF NEW ACCOUNTING POLICIES

At the time of the preparation of the consolidated financial statements as of 31 August 2007, a number of standards and interpretations have been published that have yet to take effect. A preliminary assessment of the impact the introduction of standards and statements may have on Diamyd's financial reporting follows:

IFRS 7 Financial Instruments: Disclosures: This standard, which takes effect in 2007 and the complementary Amendment to IAS 1: Presentation of Financial Statements – Capital Disclosures (takes effect 1 January 2007), which is applicable to Diamyd, will not have any impact on the accounting of assets and liabilities, but will only exert an impact through more stringent standards applying to supplementary disclosures.

IFRS 8 Operating Segments: This standard takes effect on 1 January 2009 and applies to financial years that begin from this date. The standard relates to the breakdown of a company's operations into different segments. According to this standard, the Company should use this structure as the starting point for its internal reporting structure, determining which segments will be included in reporting. Diamyd currently presents reports for two segments, discontinued operations and continuing operations. Because this division will eventually disappear, the Company will review the reporting structure so that it satisfies these requirements as of the 2009 financial year.

IFRIC 10 Interim Financial Reporting and Impairment: This interpretation statement comes into force on 1 November 2006 and applies to financial years that begin after this date. The interpretation prohibits the impairment losses recognized in an interim period from being reversed at a subsequent balance sheet date. The group will apply IFRIC 10 from 1 January 2007, but it is not expected to have any impact on the Group's accounts.

IFRIC 11 IFRS 2 Group and Treasury Share Transactions: This interpretation statement comes into force on 1 March 2007 and applies to financial years that begin after this date. The interpretation clarifies

the classification of share-related remuneration in which the Company buys back shares to fulfill its commitments as well as disclosing stock option plans in subsidiaries that apply IFRS. The company applied this principle in relation to the reporting of stock options in subsidiaries; see Note 30.

Interpretations to existing standards that is not yet effective and not relevant for group's operations.

*IFRIC 12 Service concession arrangements:** The interpretation is mandatory from 1 January, 2008 and is applicable to periods beginning on or after 1 January 2008. The interpretation applies to contractual arrangements whereby a private sector operator participate in development of infrastructure to provide services for the public sector for a specific time. The company receives payment for this service during the contractual time. The group will apply IFRIC 12 as of 1 January 2008, but this is expected not to have any effect on the group consolidated statement.

*IFRIC 13 Customer Loyalty Programmes:** The interpretation is mandatory from 1 July 2008 and is applicable to periods beginning on or after 1 July 2008.

*IFRIC 14 IAS 19 The Limit on a Defined Benefit Asset, Minimum Funding Requirements and their Interaction:** The interpretation is mandatory from 1 July 2008 and is applicable to periods beginning on or after 1 July 2008.

* These standards/interpretations are not adopted by the EU at this date.

LEASING

Leasing where a substantial part of the risks and advantages of ownership are retained by the lessor is classified as operating lease. Payments that are made during the lease period are expensed in the income statement.

INVENTORIES

Inventories are stated at the lower of cost and net realizable value.

PROVISIONS

For legal or informal undertakings entered into, allocations are continually made on the basis of an assessment of the value of the undertaking. In cases where it has not been possible to make a reliable assessment or where there is uncertainty about the undertaking, the undertaking/relationship is shown as a contingent liability.

EVENTS AFTER THE BALANCE SHEET DATE

Any events after the end of the financial year that confirm commitments for the Company on balance sheet date are considered and shown in the balance sheet.

Notes

NOTE 1 SEGMENT RESULTS (GROUP)

During the year, Diamyd Medical has changed its view of grouping the segments due to the Company's acquisition of the development project, NTDDS, in the previous year. Therefore only one comparative year is disclosed. The grouping has its basis in risk and opportunities in the different projects.

Segment result of the financial year

	2006/2007			2005/2006		
	GAD	NTDDS	Diamyd Group	GAD	NTDDS	Diamyd Group
Total segment revenue	531	–	531	4,309	90	4,399
Other revenue	117	423	540	50	–	50
Total revenue	648	423	1,071	4,359	90	4,449
Segment results	-42,859	-12,089	-54,948	-36,930	-2,752	-39,682
Financial revenue			2,574			1,808
Financial costs			-1,447			-56
Total financial revenue and costs			1,127			1,752
Dividends from holdings			350			250
Loss before income tax			-53,471			-37,680
Income tax expense			266			–
Net loss for the year			-53,205			-37,680
<i>Amortization</i>	<i>-521</i>	<i>-28</i>	<i>-549</i>	<i>-762</i>	<i>-9</i>	<i>-771</i>

Assets and liabilities for the segments

	2006/2007			2005/2006		
	GAD	NTDDS	Diamyd Group	GAD	NTDDS	Diamyd Group
Assets	80,914	33,307	114,221	69,452	35,637	105,089
Liabilities	7,088	1,999	9,087	7,400	2,087	9,487
Purchases	31	396	427	28	17,062	17,090



Sales per Geographic Regions

kSEK	Nordic countries	Europe (excluding the Nordic countries)	North America	Rest of the World	Total
2006/2007					
Revenue	188	171	712	–	1,071
Assets	111,855	–	2,366	–	114,221
Intangible purchases	–	–	–	–	–
Tangible purchases	31	–	396	–	427
2005/2006					
Revenue	142	94	4,196	17	4,449
Assets	100,840	–	4,249	–	105,089
Intangible purchases	–	–	17,062	–	17,062
Tangible purchases	28	–	–	–	28
2004/2005					
Revenue	469	76	350	36	931
Assets	125,473	–	–	–	125,473
Intangible purchases	–	–	–	–	–
Tangible purchases	33	–	–	–	33

Geographic Regions has been divided after main revenues and results.

NOTE 2 SALES (GROUP)

kSEK	2006/2007	2005/2006	2004/2005
Sales of GAD-protein and diagnostic products	531	721	883
Out-licensing of GAD technology	–	3,602	–
<i>Net sale</i>	<i>531</i>	<i>4,323</i>	<i>883</i>
<i>Other operating revenue</i>	<i>540*</i>	<i>126</i>	<i>48</i>
Total	1,071	4,449	931

Parent Company's net sale amounted to SEK 0 (0). All sales are made by the subsidiaries Diamyd Diagnostics and Diamyd Inc.

* The increase of other operating revenue relates to a co-operation agreement with Sangamo BioSciences, Inc related to NTDDS.

NOTE 3 EARNINGS PER SHARE

The calculation of earnings per share before dilution is based on net profit for the year for the operations divided by average number of shares for the year.

	2006/2007	2005/2006	2004/2005
Earnings that is related to parent	-53,205	-37,680	-36,632
Average number of outstanding shares	9,660	8,583	8,411
Earnings per share before dilution	-5.5	-4.4	-4.4

For calculation of earnings per share after dilution, adjustments are made in average number of shares for dilution effects of potential ordinary shares. The Company has only outstanding warrants. The calculation of warrants is based on possible number of shares that is buyable to an average market price. The earning per share improves as the total earning is negative which results in the same earning before and after dilution.

	2006/2007	2005/2006	2004/2005
Earnings that is related to parent	-53,205	-37,680	-36,632
Number of shares after dilution	9,751	9,544	8,443
Earnings per share after dilution	-5.5	-4.4	-4.4

NOTE 4 LIQUID ASSETS

Other liquid assets apart from cash and cash equivalents are short-termed (placements with duration under 3 months) liquid placements that can easily convert into a known amount in cash and cash equivalents and that is exposed to a minimum risk of change in value.

kSEK (Group)	2006/2007	2005/2006	2004/2005
Short-term placements	—	—	—
Cash and bank balance	68,803	13,190	24,161
Total liquid assets	68,803	13,190	24,161

kSEK (Parent)	2006/2007	2005/2006	2004/2005
Short-term placements	—	—	—
Cash and bank balance	59,631	4,915	21,022
Total liquid assets	59,631	4,915	21,022



NOTE 5 PERSONNEL AND REMUNERATION (GROUP)

Average Number of Employees	2006/2007	2005/2006	2004/2005
Women	5	3	2
Men	6	6	4
Total	11	9	6

Distribution in terms of gender

	August 31, 2007		August 31, 2006		August 31, 2005	
	Women	Men	Women	Men	Women	Men
Board members	0%	100%	0%	100%	0%	100%
Employees	40%	60%	17%	83%	33%	67%
Foreign subsidiaries	0%	100%	0%	100%	0%	100%

Sick Leave*

	2006/2007	2005/2006	2004/2005
Women	0.00%	3.61%	1.57%
Men	6.50%	2.01%	0.76%
Total	3.50%	2.70%	1.11%

* Definition: Sick leave is defined as the absence from work divided by the available working time adjusted for leave of absence.

Remunerations to the CEO

The CEO was paid a salary of kSEK 1,454 (1,361) including a remuneration of kSEK 82,5 (81) in connection with membership of the Board of Directors. The Company pays two occupational pension insurances for the CEO. For occupational pension insurance, the Company pays a premium of 35% of the annual salary. Commencement of the policy was on 1 March, 2002. Both insurances mature when the insured reaches 65 years of age. The total pension expenses for the CEO during the fiscal year were kSEK 509 (526). Pension expenses refer to the expenses that are charged until closing date of the fiscal year. Share-related remunerations were made to the CEO during the fiscal year and are disclosed under Note 30. The CEO's holdings in employee options are 10,000. No other benefits are being paid out.

Remuneration to management team

Salaries paid to other key executives during the fiscal year were kSEK 3,600 (4,800). Occupational pension premiums for other key executives amounted to kSEK 690 (695). The Company offers Pension Schemes to all key executives equal to 20–35% of the pension-entitled salary. The pension schemes mature when the insured key executive reaches 65 years of age. All the pension benefits are transferable, i.e., not conditional on future employment. Pension expenses refer to the expenses that are charged until closing date of the fiscal year. No other benefits are being paid out.

Termination Payments for the CEO and other key executives

The contract between the Company and the CEO is subject to twelve months notice from the Company and six months notice from the CEO. The CEO's employment agreement does not include any provisions for severance pay. The contracts between the Company and key executives are subject to three months' notice by either party. Employment agreements currently make no provisions for severance pay.

kSEK	2006/2007	2005/2006	2004/2005
CEO's salary	1,454	1,361	1,315
Salaries other key executives (5)	3,600	4,800	3,800
Other employees' salaries	3,593	480	161
Total salaries	8,647	6,641	5,276
Social security costs, CEO	148	330	426
Social security costs, other key executives	1,167	1,450	1,210
Social security costs, other employees	1,104	155	52
Pension costs, CEO	509	526	482
Pension costs, other key executives	690	695	765
Pension costs, other employees	172	174	191
Other personnel costs	1,340	255	371
Total personnel costs*	13,777	10,226	8,773

* Total personnel costs includes salaries of kSEK 161 (265) and social security costs of kSEK 62 (85), classified as Research and Development costs in the Income Statement, 2006/2007.

All employees in the group is employed in subsidiaries.

Remuneration to the members of the Board of Directors

The Annual General Meeting of Shareholders on 11 December 2006 approved a salary of kSEK 82.5 to the members of the Board of Directors and kSEK 165 to the Chairman of the Board of Directors for the period ending with the Annual General Meeting of Shareholders for 2007. In addition to this, the Chairman of the Board of Directors invoiced consulting fees amounting to kSEK 268 during the fiscal year. The fees regarded legal services provided outside the normal duties of the Chairman of the Board of Directors. This compensation has been decided by the board. No other remuneration was granted.

kSEK	Salary	Other Remunerations	Total
Chairman of the board**	165	268	433
Board members	330	–	330
Total	495	268	763

** Excluding social security expenses.

Share-related remunerations are disclosed under Note 30.



NOTE 6 RELATED-PARTY TRANSACTIONS (GROUP)

During the fiscal year kSEK 696 (581) were paid to companies represented by immediate family members of the CEO for providing IT-services to the Company excluding value added tax. Pricing has been set by arm's length principle. Salary of kSEK 824 (468) were paid to immediate family members of the CEO. No other immediate family member of the members of the Board of Directors or the Employees have been directly or indirectly involved in any business transaction with the Company that was unusual in its character or terms and conditions and took place during the current fiscal year. Neither has the Company given any loans, provided any guarantees or surety for the benefit of any member of the Board of Directors, employees or accountants in the Company. For further information on transactions to key-persons, see Note 5.

(kSEK)	2006/2007	2005/2006	2004/2005
Purchase of services (inter company)	11,334	–	–
Salaries	824	468	168
Consultant fees	696	581	433
Services by the Chairman	268	358	–

NOTE 7 OTHER EXTERNAL EXPENSES

During the fiscal year 2006/2007, auditing expenses for the Diamyd Group amounted to kSEK 385 (314) and consultancy fees amounted to kSEK 177 (200). Auditing costs are part of the total external costs.

kSEK	2006/2007	Group		2006/2007	Parent	
		2005/2006	2004/2005		2005/2006	2004/2005
Öhrlings PricewaterhouseCoopers AB						
Audit fees	315	–	–	220	–	–
Other services	140	–	–	140	–	–
Total	455	–	–	360	–	–
Focus revision AB						
Audit fees	–	199	257	–	113	220
Other services	–	46	31	–	41	28
Total	–	245	288	–	154	248
Ernst & Young AB						
Audit fees	–	100	103	–	100	103
Other services	–	154	34	–	154	34
Total	–	254	137	–	254	137
Lally & Lally & Co.						
Audit fees	70	15	–	–	–	–
Other services	37	–	–	–	–	–
Total	107	15	–	–	–	–

Audit Engagement refers to audit of the annual report, the accounts and the administration of the board of directors and the president and other assignments for the Company's auditor and advice or other assistance or other guidance caused by observations at the audit or during other assignments. Everything else is other services.

NOTE 8 EXPENSES FOR THE COMPANY'S WEB SITE (GROUP)

The Company's web site is intended to provide information about the Company in accordance with the information requirements of the Company's Listing Agreement with the Nordic Stock Exchange. Thus, expenses for the Company's web site are currently not classified as an intangible asset, as neither income nor cost savings are expected as a result of the web site. The Expenses associated with the Company's web site are allocated as presented in the table below.

kSEK	2006/2007	2005/2006	2004/2005
Maintenance	360	259	182
Translations	–	–	30
Other	–	6	3
Total	360	265	215

NOTE 9 LEASE CONTRACT

The Company's premises leasing agreement runs until August 31, 2010. Termination must take place at least 9 months before the contractual end date otherwise the contract is extended by 3 years. The Company has paid rent in an amount of kSEK 512 (424) during the fiscal year 2006/2007. For the upcoming fiscal year 2007/2008 the rent will amount to kSEK 544 according to the contract. For the rest of the period with maturity date between 1 and 5 years is the cost kSEK 1,088. The premises leasing agreement of Diamyd Inc. has a notice period of one month.

NOTE 10 ADJUSTED FINANCIALS

During 2006, the Company acquired the NTDDS research and development project. Last year, the Company amortized the project. As an acquired research and development project which has not yet been utilized should not be amortized in accordance with IAS 38, we have adjusted the financial statement for 2005/2006. The effect of the adjustments on last year financials is summarized below. There is no effect in the year end numbers for FY 2006/2007. The effect on this year has been kSEK 415 each quarter from the first to the third quarter 2006/2007 and will be adjusted for the comparative figures.

kSEK	2005/2006	Adjustment	Adjusted
Decrease in depreciation,	-1,626	970	-656
Decrease of net loss	-38,650	970	-37,680
Increase of equity	94,632	970	95,602
Increase of intangible assets	15,656	970	16,626



NOTE 11 PROPERTY, PLANT AND EQUIPMENT AND DEPRECIATION (GROUP)

Equipments are depreciated over 5 years and computers over 3 years.

kSEK (Group)	2006/2007	2005/2006	2004/2005
Opening net book amount	1,507	1,479	1,446
Additions	427	28	33
Disposals	-1,121	–	–
Closing net book amount	813	1,507	1,479
Opening depreciation	-1,374	-1,259	-1,109
Disposals	1,121	–	–
Depreciation for the year	-146	-115	-150
Closing depreciation	-399	-1,374	-1,259
Net book amount, 31 August	414	133	220

The Parent Company has no tangible assets.

NOTE 12 FINANCIAL INCOME AND COSTS

kSEK	Group			Parent		
	2006/2007	2005/2006	2004/2005	2006/2007	2005/2006	2004/2005
Interest, bank	1,480	267	600	1,365	167	519
Interest, bonds and commercial papers	1,094	1,541	2,595	1,094	1,541	2,595
Financial income	2,574	1,808	3,195	2,459	1,708	3,114
Interest expenses	-26	-56	-26	-5	-1	-13
Foreign currency fluctuations cost	-1,421	–	–	-1,421	–	–
Financial costs	-1,447	-56	-26	-1,426	-1	-13

NOTE 13 TAX

The Company does not have any other temporary differences other than tax loss carry-forwards. Deferred income tax assets are recognized for tax loss carry-forward to the extent that the realization of the related tax benefit through the future taxable profits is probable. Diamyd Inc accounts for a deferred tax income of kSEK 266 due to the utilization of previously not recognized loss carry-forward in the Company. The group does not account for deferred tax assets amounting to 70,519 (56,021 respectively 45,377) in respect to losses amounting to MSEK 251,853 (200,075 respectively 162,061) that can be carried forward against future taxable income. The parent company does not account for deferred tax assets amounting to 15,715 (11,237 respectively 9,942) in respect to losses amounting to MSEK 56,124 (40,132 respectively 35,506) that can be carried forward against future taxable income. For Swedish limited companies there is no expiration date regarding the possibility of utilizing loss carry-forward.

Weighted average tax rate for the Group was 28% (28%).

	2006/2007	2005/2006	2004/2005
Current tax	–	–	–
Deferred tax	266	–	-63
Total	266	–	-63

Reconciliation between actual and nominal tax

	Group			Parent		
	2006/2007	2005/2006	2004/2005	2006/2007	2005/2006	2004/2005
Reported loss before tax	-53,471	-37,680	-36,569	-47,641	-41,327	-34,950
Tax at nominal tax rate 28%	14,972	10,550	10,236	13,339	11,572	9,786
Tax effect of free revenue	100	71	1	100	71	1
Tax effect of non-deductible items	-1	-58	-24	-8,963	-10,499	-10,441
Tax effect on loss carry-forward	–	–	-63	–	-1,094	654
Tax effect of deficits for which tax assets are not considered	-14,805	-10,563	-10,213	-4,476	–	–
Tax on reported loss	266	–	-63	–	50	–



NOTE 14 INTANGIBLE ASSETS AND DEPRECIATIONS

Intangible assets are licensed patent rights regarding GAD from University of Florida, University of Washington and University of California, these are utilized in research and are amortized over five years. The remaining acquisition value amounts to kSEK 10,208. The acquired research and development project regarding NTDDS is not amortized as they are not yet utilized. The acquisition value amounts to kSEK 16,627. The impairment test did not give any adjustments to the book value this year on the NTDDS project.

kSEK	Group			Parent		
	2006/2007	2005/2006	2004/2005	2006/2007	2005/2006	2004/2005
Cost, opening net book amount	27,262	10,200	10,200	16,627	–	–
Additions	–	17,062	–	–	16,627	–
Disposals	-427	–	–	–	–	–
Cost, closing net book amount	26,835	27,262	10,200	16,627	16,627	–
Amortization, opening net book amount	-9,547	-8,891	-8,140	–	–	–
Disposals	–	–	–	–	–	–
Correction	–	970	–	–	970	–
Amortization charge	-403	-1,626	-751	–	-970	–
Accumulated amortization	-9,950	-9,547	-8,891	–	–	–
Net book amount, 31 August	16,885	17,715	1,309	16,627	16,627	–

NOTE 15 FINANCIAL FIXED ASSETS / OTHER LONG TERM BOND HOLDINGS

Financial fixed assets consists of a holding of shares in Mercodia AB, Uppsala, Sweden, Corporate Identity Number 556157-5100. The Group's holding is 19% of the capital or 1,000 shares with a net book value of kSEK 800, in addition to holding of shares in Protein Science Corporation, Meriden, CT, U.S.A., Corporate Identity Number 2008700. The Group's holding is 6.7% after full dilution of the capital or 3,943,151 shares. Net Book Value is kSEK 20,618.

kSEK (Group/Parent)	2006/2007	2005/2006	2004/2005
Cost, opening balance	800	800	800
Investments during the year	20,618	–	–
Cost, closing balance	21,418	800	800
Net book amount, 31 August	21,418	800	800

NOTE 16 INVENTORY

The inventory consists of GAD-related products for sale.

NOTE 17 PREPAID EXPENSES AND ACCRUED INCOME

kSEK	Group			Parent		
	2006/2007	2005/2006	2004/2005	2006/2007	2005/2006	2004/2005
Prepaid patent fees	875	600	510	–	–	–
Prepaid insurance fees	136	100	82	–	–	–
Accrued interest income	1,095	1,540	4,037	1,017	1,478	3,989
Other prepaid expenses	603	360	818	407	158	126
Total	2,709	2,600	5,447	1,424	1,636	4,115

NOTE 18 CURRENT INVESTMENTS

Current investments are valued at cost in accordance with IAS 39, from 1 September, 2005. All current investments are in interest bearing bonds in the securities market. The value of the Company's current investments can temporarily vary due to changes in market interest rates. All current investments are subject to trade and, thus, are classified as current, even if expiry date is longer than one year.

The Parent Company applies the Swedish Financial Accounting Standards Council recommendation RR 32:05, which means that current investments for the Parent Company are also valued at market value.

kSEK (Group & Parent)	2006/2007	2005/2006	Fair value	Book value
			2004/2005	2004/2005
Swedish mortgage bonds	–	15,157	15,552	15,674
Banks	–	15,346	14,892	14,432
Other Swedish issuers	–	15,048	61,809	61,268
Total	–	45,551	92,253	91,374

Listed current investments

kSEK	2006/2007	2005/2006	2004/2005
Remaining premium	–	628	1,942
Remaining discount	–	–	–

Durations

kSEK	2006/2007	2005/2006	2004/2005
0–1 years	–	45,551	44,544
1–2 years	–	–	46,830
Total	–	45,551	91,374

NOTE 19 FINANCIAL RISK MANAGEMENT

The purpose of the Group's investments is to maximize yield with the lowest possible risk by placing the Company's liquid assets with reputable market participants such as banks. Diamyd classifies its investments as "possessed for sale" they are valued to fair value through equity until they are sold and then the effect will affect the income statement.

Interest rate risk

Interest rate risk arises when the term of a financial instrument deviates from the investment horizon. By means of an investment portfolio, with terms distributed over time, cash is invested, with consideration of the expected timing of operational cash requirements, in order to reduce the risk of discrepancies in the timing between redemptions and operational cash requirements. Thereby the interest rate risk is minimized.

Foreign exchange risk

The Group is active on an international arena in which both income and costs arise in foreign currencies. It is uncertain how exchange rates for SEK will change in the future. The Company does not hedge itself against currency fluctuations and there is a possibility that such fluctuations may affect the Company's ability to develop its operations according to plan. The growing operations of Diamyd Inc. may involve an increased exposure to currency fluctuations between USD and SEK, with a corresponding impact on the Group's financial result. If the SE/USD exchange rate had been 5% higher the Groups loss would have increased by 1% or kSEK 535.

Translation Risks

The net result is affected when assets and liabilities are denominated in different currencies. This influence is currently marginal as most assets and liabilities are denominated in SEK. The net result and equity are affected when Diamyd Inc.'s income statement and balance sheet are translated into SEK. With the current scale of the operations in the U.S.A., the impact of the translation is marginal.

Liquidity risk

There is a risk that the Group will not have sufficient funds to pay anticipated or unanticipated current expenses as they become due. This risk is associated with current investments that cannot be disposed of without delay at the Company's demand. Liquidity risk in the Group is managed through consideration of the expected timing of operational cash requirements in order to reduce the risk of discrepancies in the timing between redemptions and operational cash requirements. Presently, the liquidity risk is low.

Credit risk

The Company's current investment policy allows discount securities, deposits with Swedish banks, fixed coupon bonds, floating rate notes, zero coupon bonds and other capital guaranteed products. Additionally, bonds issued by large capital companies listed on the Nordic Stock Exchange are permitted.

Valuation

Current investments are valued at fair value. All current investments are interest-bearing instruments with registered securities institutions. There is a risk that the value of the Company's financial instruments will temporarily fluctuate due to changes in market interest rates. However, the Company judges that the investments will be held until expiry and, hence, will be redeemed at book value plus accrued interest.

The carrying value less impairment provision on trade receivables and payables are assumed to approximate their fair values due to the short-term nature of trade receivables and payables.

Cash Flow risk

This year's operating cash flow amounted to kSEK -51,555 (-32,669). The total cash flow amounted to kSEK 55,655 (-10,766). Liquid assets amounted to kSEK 68,803 as of 31 August 2007. It is the Company's judgment that the liquid assets will last until December 2008 without additional financing.

NOTE 20 CASH FLOW STATEMENT

The Parent Company manages the majority of the Group's funds. Liquid assets intend cash and bank and short-term investments with a maximum maturity of three months. Transfer of funds is made to subsidiaries throughout the year when needed and is accounted for through Group's contributions or shareholders' contributions at the end of the fiscal year. As of 31 August 2007, cash and cash equivalents amounted to kSEK 68,803 (13,190).

NOTE 21 THE COMPANY'S SURVIVAL

The Parent Company's and the Group's current operations principally consist of research and development. Thus, the operations do not generate positive cash flow. It is the Board's and the management's assessment that further financing will be required to finalize the Diamyd® project and other activities. The Parent Company's and the Group's survival prospects are continuously analyzed at a 12 month horizon by the Board of Directors.

NOTE 22 LIABILITIES

All of the Company's liabilities are non-interest bearing.

NOTE 23 ACCRUED EXPENSES AND PREPAID INCOME

kSEK	Group			Parent		
	2006/2007	2005/2006	2004/2005	2006/2007	2005/2006	2004/2005
Accrued vacation pay	–	11	59	–	–	–
Accrued social security expenses	225	133	170	–	–	–
Accrued expenses, clinical trials	1,962	3,303	3,571	–	–	–
Other expenses	2,664	2,302	1,952	719	767	499
Total	4,851	5,749	5,752	719	767	499

NOTE 24 AMORTIZATION OF SHARES IN THE GROUP COMPANIES/SHAREHOLDERS' CONTRIBUTIONS

Amortization of shares in the Group companies/shareholders' contributions refers to the amortization of shares and is equivalent to supplied shareholders' contributions. These contributions amount to kSEK 38,000 (31,500) to Diamyd Therapeutics. Amortization is made after calculation of recovery value or the net realizable value. A reverse has been made of the write-down made last year of kSEK 5,995, on the basis that Diamyd Inc. had a profit this year and is expected to have positive results in the coming years.

NOTE 25 LONG-TERM LIABILITIES TO SUBSIDIARIES

kSEK	2006/2007	2005/2006	2004/2005
Liabilities, Diamyd Therapeutics AB	–	29	3,950
Liabilities, Diamyd Inc., NJ	181	188	199
Liabilities, Diamyd Diagnostics AB	–	14	–
Total	181	231	4,149



NOTE 26 SHARES IN SUBSIDIARIES

Subsidiary	Corporate Identity Number	Registered office	Equity (kSEK)	Earnings (kSEK)	Ownership share	Number of shares	Book value (SEK)
Diamyd Therapeutics AB	556242-3797	Stockholm	754	-38,838	100%	1,000,000	1,100,000
Diamyd Diagnostics AB	556552-2280	Stockholm	345	213	100%	1000	100,000
Diamyd Inc.	3695413	Pittsburgh, PA	-607	1,424	100%	1000	7
Diamyd Inc.	1487145	Tom's River, NJ	-406	–	100%	1000	9,000

The subsidiary with its domicile in New Jersey is under dissolution and will be dissolved during the next fiscal year.

NOTE 27 LONG-TERM RECEIVABLES FROM SUBSIDIARIES

kSEK	2006/2007	2005/2006	2004/2005
Long-term receivables from Diamyd Therapeutics AB	4,074	–	–
Long-term receivables from Diamyd Diagnostics AB	127	178	1,563
Long-term receivables from Diamyd Inc	2,583	–	–
Total	6,784	178	1,563

NOTE 28 SHARE CAPITAL

Specification on the changes in shareholder's equity can be found in the Group changes in shareholder's equity in the report. On August 31, 2007 the assets of Diamyd Medical were divided among 9,210,807 (8,264,016) shares of serie B (1/10 vote) and 561,671 (471,200) shares of serie A. At the end of the fiscal year the share capital in Diamyd Medical was SEK 9,772,478 (8,735,216). The quotient value is SEK 1(1). All issues are fully paid.

Year	Transaction	A-shares (increase, number)	B-shares (increase, number)	Number of shares (accumulated, number)
As of September 1, 2004		471,200	7,874,280	8,345,480
2004/2005	Exercise of warrants	–	72,563	8,418,043
2005/2006	Non-cash issue	–	317,173	8,735,216
2006/2007	Warranter	90,471	821,791	9,647,478
2006/2007	Exercise of warrants	–	30,000	9,677,478
2006/2007	Exercise of warrants	–	25,000	9,702,478
2006/2007	Directed issue	–	70,000	9,772,478
As of August 31, 2007		561,671	9,210,807	9,772,478

NOTE 29 SHARE OPTION PLAN 2004/2007.

A share option plan was approved by the shareholders at the Company's Annual General Meeting of Shareholders in 2004 (DIAM TO 2004/2007). The DIAM TO 2004/2007 Share Option Plan comprises 200,000 subscription options, all of which had been signed by 31 August 2006. The premium per option was SEK 3 in December 2004. The subscription price is SEK 50 and transactions are made at market value, based on Black & Scholes' equation. As the share option plan is classified under marketable securities and the options have been bought from the Company at market price, they are not considered share-related remuneration (IFRS 2).

At valuation of the warrants value Balck & Scholes valuation method has been applied. The following assumptions has been used for the calculation as of December 2004: share value SEK 38, exercise price SEK 50, volatility 30%, duration 1,095 days, no expected dividends and a risk free interest of 2% and a liquidity discount due to the fact that no organized trade will occur in this securities. The value was determined to SEK 3.

At valuation of the warrants value Balck & Scholes valuation method has been applied. The following assumptions has been used for the calculation as of June 2006: share value SEK 39, exercise price SEK 50, volatility 53%, duration 557 days, no expected dividends and a risk free interest of 2% and a liquidity discount due to the fact that no organized trade will occur in this securities. The value was determined to SEK 6.

The share options expire December 31, 2007.

Warrants 2004/2007	2006/2007	2005/2006	2004/2005
As of September 1	200,000	120,000	–
Allocated	–	80,000	120,000
Exercised	55,000	–	–
As of August 31	145,000	200,000	120,000

NOTE 30 EMPLOYEE OPTION PROGRAM 2007/2010

Diamyd has introduced one stock option program in accordance with a resolution of the Annual Shareholders' Meeting in May 2007. The program is a stock option program settled with shares in Diamyd Medical AB and covers permanent employees of Diamyd Group.

The financial exposure from the stock option program is hedged by warrants issued to a wholly-owned subsidiary. A specified portion of the warrants is reserved to cover payroll taxes and other related costs. The terms of the stock option program provide for adjustments of the exercise price and number of shares for each stock option if new shares are issued with preferential rights to shareholders. The figures below are adjusted accordingly, unless otherwise indicated.

The program involves 250,000 stock options, representing 250,000 shares. Warrants are reserved to cover payroll taxes. The maximum allocation of stock options represents 10,000 shares maximum to all employees.

The valuation was made in accordance with IFRS 2 Share-Based Payments. The Black-Scholes model for option pricing has been used for the valuation. The following assumptions has been used for the calculation as of May 2007: share value SEK 133, exercise price SEK 146, volatility 51%, duration 838, 1,010 and 1,190 days, no expected dividends and a risk free interest of 3.9%, 4.0% and

4.1% and a liquidity discount due to the fact that no organized trade will occur in this securities. The value was determined to SEK 40, SEK 44 and SEK 49. Volatility used was average volatility for the previous 6 months before the valuation date, At each quarterly report a revaluation is made to account for the social security expenses for the program according to URA 46.

Stock options vest annually over one, two and three years and are exercisable on June 1 2008, May 22 2009, May 22 2010 and until December 31 2010. The stock options have an exercise price of SEK 146.

EFFECT ON FINANCIAL STATEMENTS

The accounting policies for stock option programs are described above. The cost charged to the income statement in 2006/2007 amounts to kSEK 492, with a corresponding entry to equity and a cost regarding social security amounting to kSEK 98.

The future exercise of stock options will have a positive effect on the Company's financial position, as plan participants will pay monies to the Company to exercise options in accordance with the exercise price. Additional costs occurring as an effect of the program, consisting primarily of payroll taxes levied upon exercise,


NOTE 30 EMPLOYEE OPTION PROGRAM 2007/2010 (Continued from previous page)

will be covered by the exercise of the additional warrants held by an external party. There will be no adverse effect on the Company's financial position from the program, provided that the percentage at which payroll taxes are levied does not change significantly during the remainder of the exercise period.

INCREASE IN NUMBER OF SHARES

Exercise of all options outstanding under the two plans would lead to an increase of the number of shares by 2.5%, including warrants required to cover payroll taxes. The issued warrants would not imply dilution of earnings per share in 2006/2007, as a conversion to shares would lead to a decrease in the reported loss per share.

Allocation of stock options (corresponding number of shares)	2006/2007
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Number of stock options decided at May 22, 2007	250,000
Allocated	100,000
Forfeited	-20 000
Outstanding at August 31, 2007	80 000
Vested	–

NOTE 31 DEFICITS IN SUBSIDIARIES

The Parent Company warrants capital cover annually for the Swedish subsidiaries as they are running deficits. For 2006/2007 the outstanding capital cover amounts kSEK 38,000 (31,500) as was adjusted at the end of 2007.

NOTE 32 OTHER INVESTMENTS

The Company have under the fiscal year 2005/2006 invested in a convertible debt in Protein Sciences Corporation, Meriden, CT, U.S.A. The currency on the convertible debt was until December 31, 2006 and converted into 3,943,151 shares, see also Note 15.

NOTE 33 ASSETS PLEDGED AND CONTINGENT LIABILITIES

kSEK	2006/2007	2005/2006	2004/2005
Diamyd Medical AB (leasing warranty)	157	–	–
Diamyd Therapeutics AB (leasing warranty)	–	157	157
Total	157	157	157

The Group has no contingent liabilities.

NOTE 34 EVENTS AFTER THE FINANCIAL YEAR-END

Diamyd Medical increased the investment in Protein Sciences Corporation, Meriden, CT, U.S.A., with another one million US dollars. The investment is made in form of a convertible debt. Diamyd Medical has prior made investments amount to 3 million US dollars, that now days is converted in to a holding of 6.7% of the shares after full dilution in Protein Sciences. The latest investment makes Diamyd Medical the next largest shareholder in Protein Sciences.

The Board of Directors and the President assure that the consolidated financial statement as well as the annual report has been prepared according to the international accounting standards as intended in the European Parliament and the Councils regulation (EC No 1606/2002) as of the 19th of July 2002 about application of international accounting standard and generally accepted accounting principles and gives a fair overview of the Group and the Parents position and result.

The administration report for the Group and the Parent Company gives a fair overview of the gives a fair overview of the Company's and the Group's operations, position and result and describes significant risks and uncertainty which the Company and companies in the Group are facing.

The Balance Sheet and Income Statement published in the Annual Report for 2007 will be presented at the Annual General Meeting on December 11, 2007.

Stockholm, November 20, 2007

Joseph Janes
Chairman

Anders Essen-Möller
President

Björn O. Nilsson

Peter Rothschild

Hans Wigzell

Our auditor's report has been submitted on November 22, 2007.
Öhrlings PricewaterhouseCoopers AB

Eva Riben
Authorized public accountant
Auditor in charge

Liselott Stenudd
Authorized public accountant

Audit report

To the annual meeting of the shareholders of Diamyd Medical AB (publ)

Corporate identity number 556530-1420

We have audited the annual accounts, the consolidated accounts, the accounting records and the administration of the board of directors and the managing director of Diamyd Medical AB for the year 2006-09-01 to 2007-08-31. The company's annual accounts and the consolidated accounts are included in the printed version on pages 27–62. The board of directors and the managing director are responsible for these accounts and the administration of the Company as well as for the application of the Annual Accounts Act when preparing the annual accounts and the application of international financial reporting standards IFRSs as adopted by the EU and the Annual Accounts Act when preparing the consolidated accounts. Our responsibility is to express an opinion on the annual accounts, the consolidated accounts and the administration based on our audit.

We conducted our audit in accordance with generally accepted auditing standards in Sweden. Those standards require that we plan and perform the audit to obtain reasonable assurance that the annual accounts and the consolidated accounts are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the accounts. An audit also includes assessing the accounting principles used and their application by the board of directors and the managing director and significant estimates made by the board of directors and the managing director when preparing the annual accounts and consolidated accounts as well as evaluating the overall presentation of information in the annual accounts and the consolidated accounts. As a basis for our opinion concerning discharge from liability, we examined significant decisions, actions taken and circumstances of the Company in order to be able to determine the liability, if any, to the Company of any board member or the managing director. We also examined

whether any board member or the managing director has, in any other way, acted in contravention of the Companies Act, the Annual Accounts Act or the Articles of Association. We believe that our audit provides a reasonable basis for our opinion set out below.

The annual accounts have been prepared in accordance with the Annual Accounts Act and give a true and fair view of the Company's financial position and results of operations in accordance with generally accepted accounting principles in Sweden. The consolidated accounts have been prepared in accordance with international financial reporting standards IFRSs as adopted by the EU and the Annual Accounts Act and give a true and fair view of the group's financial position and results of operations. The statutory administration report is consistent with the other parts of the annual accounts and the consolidated accounts.

We recommend to the annual meeting of shareholders that the income statements and balance sheets of the parent company and the group be adopted, that the loss of the parent company be dealt with in accordance with the proposal in the administration report and that the members of the board of directors and the managing director be discharged from liability for the financial year.

Stockholm, November 22, 2007

Öhrlings PricewaterhouseCoopers AB

Eva Riben.
Authorized Public Accountant
Auditor in charge

Liselott Stenudd
Authorized Public Accountant

Key Ratios

Group	2006/2007	2005/2006	2004/2005
Earnings per Share, USD	-5.5	-4.5	-4.4
Earnings per Share, Diluted, USD	-5.5	-4.5	-4.4
Shareholders' Equity per Share, USD	10.8	10.9	13.7
Shareholders' Equity per Share, Diluted, USD	10.8	10.0	13.7
Cash Flow per Share, USD	-1.0	-11.9	-4.3
Dividends	—	—	—
Closing Share Price, USD	78.25	97.00	53.50
Closing Share Price/Equity per Share, USD	7.3	9.0	3.9
P/E-ratio	Neg	Neg	Neg
Operating Margin, %	Neg	Neg	Neg
Return on Equity, %	-53	-36.8	-27.4
Return on Capital Employed, %	-51.8	-36.7	-27.3
Return on Assets, %	-47.4	-33.6	-25.8
Risk-Bearing Capital, %	92.0	90.9	92.0
Debt/Equity Ratio	—	0.1	0.1
Interest Coverage Ratio	-36	-672	-1,400
Solidity, %	92.0	91.0	92.0
Average number of Employees	8.25	7.34	6.25
Research and Development Costs, kUSD	29,049	23,167	24,676
Investment in Fixed Assets, kUSD	21,045	17,090	33
Number of Shares	9,772,478	8,735,216	8,418,043
Numbers of Shares, Average	9,772,478	8,582,797	8,410,787
Numbers of Shares, Diluted	9,831,104	9,544,076	8,442,800

KEY RATIO DEFINITIONS (ALPHABETICAL ORDER)

CASH FLOW PER SHARE
The Cash Flow divided by the average Number of Shares.

CLOSING SHARE PRICE
The Closing Share Price as of the Fiscal Year-End on August 31st.

CLOSING SHARE PRICE/SHARE-HOLDER'S EQUITY PER SHARE
The Closing Share Price divided by the Shareholder's Equity per Share.

DEBT-EQUITY RATIO
Interest-bearing liabilities on average divided by the Shareholder's Equity.

EARNINGS PER SHARE
The Net Result divided by the average Number of Shares.

EARNINGS PER SHARE, DILUTED
The Net Result divided by the diluted Number of Shares. As the Company does not make profit, the Earnings per Share, Diluted must not be larger than Earnings per Share.

INTEREST COVERAGE RATIO
The Net Result before tax divided by the financial expenses.

NUMBER OF SHARES, AVERAGE
The weighted Number of Shares during the year, taking into account new share issues during the period.

NUMBER OF SHARES, DILUTED
The number of outstanding shares for the period, taking into account outstanding share warrants, option programs etc., provided that the issue price is higher than the Closing Share Price.

OPERATING MARGIN
The Operating Result after depreciations and amortization as a percentage of the earnings.

P/E RATIO
The Closing Share Price divided by the Earning per Share.

RETURN ON ASSETS
The Net Result divided by the average Balance Sheet Total, expressed in percent.

RETURN ON CAPITAL EMPLOYED
The Net Result divided by the average Balance Sheet Total with allowance for the average liabilities not charging interest, expressed in percent.

RETURN ON EQUITY
The Net Result divided by the average Shareholder's Equity, expressed in percent.

RISK-BEARING CAPITAL
The sum of the Shareholder's Equity and latent tax liabilities divided by the Balance Sheet Total, expressed in percent.

SHAREHOLDER'S EQUITY PER SHARE
The Shareholder's Equity divided by the Number of Shares.

SHAREHOLDER'S EQUITY PER SHARE, DILUTED
The Shareholder's Equity divided by the Number of Shares, Diluted.

SOLIDITY
The Shareholder's Equity divided by the Balance Sheet Total.

Annual general meeting

The Annual General Meeting of Diamyd Medical AB will be held on December 11th, 2007 at 1,30 pm. Location: Armémuseum. Riddargatan 13, Stockholm. Shareholders who wish to attend the Annual General Meeting must be recorded in the Company's register of shareholders, held by the VPC (the Swedish Securities Register Center) by December 5th, 2007, and must notify the Company of their intention to attend no later than 4:00 pm December 5th, 2007.

Shareholders who want to participate in the Annual General Meeting and whose shares are registered in custodial accounts through the trust department of a bank or a stockbroker must re-register the shares temporarily in the shareholder's own name by December 5th, 2007 at the latest.

The shareholder's rights at the Annual General Meeting can be exercised by an agent. If a legal entity is represented by an agent the power of attorney should be signed by the person authorized to sign for the entity and a copy of the current certificate of incorporation shall be enclosed.

REGISTRATION TO ATTEND CAN BE MADE:

on the website www.diamyd.com

by e-mail to investor.relations@diamyd.com

by mail to Diamyd Medical AB, Linnégatan 89B, SE-115 23 Stockholm

by fax +46 8 661 63 68

by phone +46 8 661 00 26

WHEN REGISTERING THE SHAREHOLDER SHOULD STATE:

His/her name

Social security number / national tax ID number

Address and phone number

Number of shares



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