Leading Developer of Disease Modifying Therapies for Autoimmune Diabetes
Disclaimer

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Targeting Autoimmune and Insulin deficient Diabetes

**Leading clinical stage pipeline**
- First-in-class *disease modifying therapies* Diamyd® and Remygen®
- Upcoming *pivotal program with a precision medicine approach*
- Addressable market encompassing *up to 25%* of all diabetes patients

**De-risked development program**
- *Responder patients* identified for Diamyd®, significantly *increasing likelihood for success* in pivotal program with a precision medicine approach
- *Excellent safety* profile and *simple procedure* support successful commercialization

**Strong growth opportunity**
- *Phase Ila results* (Remygen®) and *start of pivotal program* (Diamyd®) during 2021

**Experienced team**
- Significant *operational experience* in *clinical development* within diabetes
- Access to *world leading* scientists and clinical experts
Clinical stage disease modifying therapies
Inducing immunological tolerance to preserve endogenous insulin production. Stimulating regeneration of insulin producing beta cells and alleviating beta cell stress.

**Diamyd® (rhGAD65/alum)**
- First-in-class
- Antigen-specific immunotherapy
- Upcoming pivotal program
- Strong clinical support for response and excellent safety profile

**Remygen® (GABA)**
- First-in-class
- Regeneration and immunomodulation
- Phase IIa clinical development
- Promising clinical results

**The opportunity for regeneration**
Approx. 25% of all diabetes patients

**Primary indication and market**
> 1.5 million incident cases

**Regeneration**
Autoimmune- and insulin-deficient Type 2 Diabetes

**Intervention**
Autoimmune diabetes

**Prevention**
Autoimmune diabetes

**Label expansion**
Estimated 2.3 million at-risk individuals
Prioritizing unmet medical need

25% of the patients, autoimmune and severely insulin deficient diabetes, lack treatment options that address their underlying disease pathology

**Autoimmune diabetes**
Approximately 10% of all diabetes patients have severe autoimmune diabetes (SAID)

> **Therapeutic development**: Induce immunological tolerance to preserve endogenous insulin production. Stimulate regeneration of insulin producing beta cells and alleviate beta cell stress

**Insulin deficient non-autoimmune diabetes**
Approximately 15% of all diabetes patients have severe insulin deficiency (SIDD) in the absence of autoimmunity

> **Therapeutic development**: Stimulate regeneration of insulin producing beta cells and alleviate beta cell stress

**Insulin resistant, age-onset and obesity-onset diabetes**
Approximately 75% of diabetes patients are severely insulin resistant or have a disease related to either age or obesity

> **Therapeutic development**: Better patient profiling to align existing therapies with treatment guidelines

*IDF 2019 Atlas
** Subgroups based on Ahlqvist et al. Novel subgroups of adult-onset diabetes and their association with outcomes: a data-driven cluster analysis of six variables. Lancet, 2018
Type 1 Diabetes
~ 300,000 new cases every year*
132,000 children and adolescents (0-20 years of age) and equally many adults are diagnosed with type 1 diabetes every year. It is more prevalent in western countries with the highest incidence in the Nordic countries.

The disease is characterized by life-long dependence on exogenous insulin therapy and blood glucose monitoring. No disease modifying therapies are available and the disease is associated with severe short and long-term complications that lead to shorter life-expectancy, decreased quality of life and significant health economic costs.

LADA
>1.2 million new cases every year**
It is estimated that up to 10% of all type 2 diabetes patients have autoimmune diabetes characterized by autoantibodies against GAD and faster progression to insulin dependence. While type 1 diabetes is rare in many non-western countries, LADA is a prevalent form not only in western countries but also in India, China and Japan.

The disease is today (mis)treated as type 2 diabetes and no disease modifying therapies are available. It is associated with severe short and long-term complications that lead to shorter life-expectancy, decreased quality of life and significant health economic costs.

* Incidence for children and adolescents from IDF 2019 Atlas
* Incidence for adult type 1 diabetes estimated from Thomas et al. Frequency and phenotype of type 1 diabetes in the first six decades of life: a cross-sectional, genetically stratified survival analysis from UK Biobank, The Lancet 2018
** 10% Prevalence of LADA based on Pozzilli & Pieralice, Latent Autoimmune Diabetes in Adults: Current Status and New Horizons, Endocrinol Metab, 2018
Disease modifying therapies for T1D are predicted to have a multibillion-dollar economic impact in the US alone

1) Modelling the total economic value of novel T1D therapeutic concepts, January 2020, Health Advances.
## Leading pipeline in autoimmune diabetes

<table>
<thead>
<tr>
<th>Product</th>
<th>Indication</th>
<th>Trial</th>
<th>Participants</th>
<th>Sponsor</th>
<th>Clinical Trials</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diamyd*</td>
<td>T1D, intervention</td>
<td>DIAGNODE-2</td>
<td>109</td>
<td>Diamyd Medical</td>
<td>Preclinical I Ia Iib III</td>
<td>Results available</td>
</tr>
<tr>
<td>Diamyd*</td>
<td>T1D, intervention</td>
<td>DIAGNODE-1</td>
<td>12</td>
<td>Linköping University</td>
<td>Preclinical I Ia Iib III</td>
<td>Results available</td>
</tr>
<tr>
<td>Diamyd*</td>
<td>LADA, intervention</td>
<td>GADinLADA</td>
<td>15</td>
<td>NTNU, Trondheim</td>
<td>Preclinical I Ia Iib III</td>
<td>Recruiting</td>
</tr>
<tr>
<td>Remygen*</td>
<td>T1D, T2D</td>
<td>ReGenerate-1</td>
<td>36</td>
<td>Uppsala University</td>
<td>Preclinical I Ia Iib III</td>
<td>Recruiting / Stage 1 results available</td>
</tr>
</tbody>
</table>

- **Diamyd**: Product name
- **T1D**: Type 1 Diabetes
- **LADA**: Latent Autoimmune Diabetes
- **T2D**: Type 2 Diabetes
- **Preclinical**: Early stage of clinical trials
- **I**: Phase I clinical trials
- **IIa**: Phase IIa clinical trials
- **IIb**: Phase IIb clinical trials
- **III**: Phase III clinical trials
- **Completed**: Clinical trials completed
- **Ongoing**: Clinical trials ongoing
- **Results available**: Clinical trial results are available
- **Recruiting**: Clinical trials are currently recruiting participants
Diamyd®

Recombinant GAD65 Formulated in Alum (rhGAD65/alum)

Primary Indication
New-onset type 1 Diabetes with HLA type DR3-DQ2

Label expansion
Type 1 Diabetes prevention, LADA

Mechanism of Action
Induce immunological tolerance against GAD65

Clinical Effect and Benefit
Preserve the endogenous insulin production, reduce short- and long-term complications

Mode of Administration
Three intranodal injections one month apart

Development Status
Phase III planning

Licensing Status
Global rights available
Acknowledged Precision Medicine approach

**Highlights**

- New medical consensus regarding genetically defined groups of T1D
- Strong case for the emerging precision medicine – in line with Diamyd Medical’s approach

Battaglia et al, Introducing the endotype concept to address the challenge of disease heterogeneity in type 1 diabetes, Diabetes Care, 2020
Responders to Diamyd® treatment identified
Phase IIb topline results demonstrate significant treatment effect of Diamyd® in genetically predefined group of individuals recently diagnosed with T1D.
Topline results – significant treatment effect of Diamyd® in genetically predefined group

• Phase IIb verified meta-analysis – effect of Diamyd® was seen in HLA DR3-DQ2 positive individuals. No observed effect in individuals negative for HLA DR3-DQ2

• More than **50% greater preservation** (p < 0.01) of endogenous insulin production compared to placebo in genetically predefined group

• **Positive trends in important secondary endpoints** (change in HbA1c, insulin dose and insulin-adjusted HbA1c) in genetically predefined group

• **No safety concerns**

→ Fewer short and long-term complications, easier disease management, improved quality of life

→ **Potential to prevent**, pathway to a cure
### DIAGNODE-2 results support large-scale meta-analysis

<table>
<thead>
<tr>
<th></th>
<th>Diamyd®</th>
<th>Placebo</th>
<th>Effect estimate (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HLA DR3-DQ2 absent</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low dose (sc)</td>
<td>96</td>
<td>89</td>
<td></td>
</tr>
<tr>
<td>High dose (sc)</td>
<td>70</td>
<td>89</td>
<td></td>
</tr>
<tr>
<td>DIAGNODE-2 (il)</td>
<td>26</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td><strong>HLA DR3-DQ2 present</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low dose (sc)</td>
<td>90</td>
<td>97</td>
<td></td>
</tr>
<tr>
<td>High dose (sc)</td>
<td>79</td>
<td>97</td>
<td></td>
</tr>
<tr>
<td>DIAGNODE-2 (il)</td>
<td>29</td>
<td>17</td>
<td></td>
</tr>
</tbody>
</table>

- **sc** = 20 µg subcutaneous*
- **il** = 4 µg intralymphatic

*evaluated in trials part of published meta-analysis

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*DISCLAIMER: The information provided is for educational purposes only and should not be considered as medical advice. Please consult with a healthcare professional for any concerns related to your health.*

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*Favors placebo*  
*Favors Diamyd®*
Combined analysis including DIAGNODE-2 further supports precision medicine approach

Meta-analysis based on more than 600 individuals from four placebo-controlled randomized intervention trials (Phase III Europe, Phase II Sweden, Phase II US, Phase IIb Europe)

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Estimated treatment effect (adjusted 95% CI)</th>
<th>p value (adjusted)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presence of HLA DR3-DQ2 (n=313)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>1.361 (1.183, 1.566)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Low Dose</td>
<td>1.208 (0.979, 1.489)</td>
<td>0.0917</td>
</tr>
<tr>
<td>High Dose</td>
<td>1.484 (1.221, 1.803)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Absence of HLA DR3-DQ2 (n=314)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>1.019 (0.888, 1.171)</td>
<td>0.7848</td>
</tr>
<tr>
<td>Low Dose</td>
<td>1.158 (0.942, 1.424)</td>
<td>0.2437</td>
</tr>
<tr>
<td>High Dose</td>
<td>0.908 (0.747, 1.103)</td>
<td>0.557</td>
</tr>
</tbody>
</table>

Low dose = 2 injections; High dose = 3 or 4 injections
Treatment effect estimate at 15 months from baseline based on MMRM
Note: Preliminary unpublished results
## Secondary endpoints in meta-analysis support effect on primary endpoint in DR3-DQ2 individuals

### HbA1c (mmol/mol)

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Estimated treatment effect (adjusted 95% CI)</th>
<th>p value (adjusted)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presence of HLA DR3-DQ2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>-3.229 (-6.170, -0.289)</td>
<td>0.032</td>
</tr>
<tr>
<td>Low Dose</td>
<td>-1.509 (-5.763, 2.746)</td>
<td>0.5268</td>
</tr>
<tr>
<td>High Dose</td>
<td>-4.743 (-8.951, -0.534)</td>
<td>0.0044</td>
</tr>
<tr>
<td>Absence of HLA DR3-DQ2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>1.164 (-1.739, 4.067)</td>
<td>0.2130</td>
</tr>
<tr>
<td>Low Dose</td>
<td>2.219 (-1.881, 6.321)</td>
<td>0.0684</td>
</tr>
<tr>
<td>High Dose</td>
<td>-0.018 (-4.227, 4.192)</td>
<td>0.7157</td>
</tr>
</tbody>
</table>

### Insulin dose (IU/kg/24h)

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Estimated treatment effect (adjusted % CI)</th>
<th>p value (adjusted)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presence of HLA DR3-DQ2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>-0.062 (-0.119, -0.006)</td>
<td>0.03</td>
</tr>
<tr>
<td>Low Dose</td>
<td>-0.071 (-0.153, -0.010)</td>
<td>0.07</td>
</tr>
<tr>
<td>High Dose</td>
<td>-0.054 (-0.134, -0.026)</td>
<td>0.2858</td>
</tr>
<tr>
<td>Absence of HLA DR3-DQ2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>0.049 (-0.004, 0.104)</td>
<td>0.0705</td>
</tr>
<tr>
<td>Low Dose</td>
<td>0.023 (-0.055, 0.101)</td>
<td>0.8571</td>
</tr>
<tr>
<td>High Dose</td>
<td>0.077 (-0.002, 0.156)</td>
<td>0.0576</td>
</tr>
</tbody>
</table>

### IDAAC1

Note: Preliminary unpublished results
The current standard treatment for type 1 diabetes is life-sustaining, subcutaneous deliveries of insulin by injection or pump therapy, combined with continuous glucose monitoring (CGM). In addition to non-insulin anti-diabetic drugs and aids, such as artificial pancreases and smart insulin pens to help patients manage their condition, therapies targeting the underlying causes of the disease are also being developed.

Antigen-specific immunotherapy with Diamyd® targets the body’s immune system by reprogramming it to stop attacking the insulin-producing cells. This treatment has the potential for long-term efficacy. Compared with other technologies under development often requiring hospitalization, the diabetes vaccine Diamyd® displays an excellent safety profile and is a fast and easy treatment.
Intralymphatic administration route enhances effect of antigen-specific immunotherapy Diamyd® in type 1 diabetes
More efficient uptake in and drainage to lymph nodes following intralymphatic compared to subcutaneous administration
Lessons Learned from Allergy Immunotherapy Trials

Subcutaneously (S.C.) injected large molecules including proteins do not effectively spread to the draining lymphnodes. Intranodal (I.L.) injections lead to immediate spreading to deeper lymphnodes. The image depicts radio tracing of labeled IgG at 20 minutes and 25 hours after subcutaneous and intranodal injection in a healthy human volunteer.

Adapted from: Intralymphatic immunotherapy, Senti & Kündig, World Allergy Organization Journal 2015
Simple outpatient procedure enhances value proposition for Diamyd®

Potential to reach patients outside specialized clinics and avoiding costs related to hospitalization

The procedure is performed by a radiologist by way of ultrasound guided injections that are given three times, one month apart. Clinical results and safety support the addition of annual booster injections in the pivotal trial.

Confirmed* by interviews and questionnaires involving radiologists and study nurses taking part in the ongoing Phase IIb program, the procedure is simple and convenient, and can be performed using hand-held ultrasound devices. Non-radiologists could be educated to perform the procedure.

* Evaluation of the Feasibility of Intralymphatic Injection of Diamyd®, Selam Fessehaye 2019, Master Thesis, Uppsala University
Dose-response relationship
Intralymphatic injections are superior to subcutaneous injections

Comparison of three SC injections vs three IL (Bayesian analysis)
→ 99% probability that IL is superior to SC regarding C-peptide retention
→ 98% probability that IL is superior to SC regarding reduction of HbA1c
→ 77% probability that IL is superior to SC regarding reduction of insulin dose
→ 97% probability that IL is superior to SC regarding reduction of insulin dose adjusted HbA1c

Note: Preliminary unpublished results
Clinical and immunological results from Diamyd® trials support the mechanistic rational for antigen-specific reprogramming of the immune response

1. GAD65 is taken up, processed and presented by APCs to T-cells by MHC Class II protein complexes.

Clinical and immunological results show that the dose and dosage of the antigen as well as the route of administration influence the tolerogenic effect.

2. MHC proteins (coded by HLA genes) bind and present protein peptides to T cells.

A comprehensive meta-analysis shows that individuals that have a certain HLA protein (Hannelius et al, Diabetologia 2020) receive the strongest response to GAD65 therapy.

Different MHC proteins present peptides differently and recent research highlighting the rational of focusing on HLA defined endotypes of type 1 diabetes.

3. T-cell deactivation

Immunological results from clinical trials show that malignant GAD-reactive T-cells are deactivated following GAD therapy.

These cells are responsible for tissue damage.

4. Induction of regulatory T cells

Immunological results from clinical trials indicate that GAD-reactive Tregs are formed/activated following GAD therapy.

These cells deactivate tissue-damaging immune processes in the islets in response to GAD65.

5. Modulation of T cell function

Immunological results from clinical trials show an increase in non-Th1 T-cells (elevated regulatory molecules e.g. IL-10; reduced effector cytokines INFγ) following GAD therapy.

These cells help redirect the balance of immune reaction in response to GAD65 in the islets and decrease tissue damage cellular homing.
**Superior safety profile**

Patient drop-out rate in clinical trials

- Diamyd®: 0.8%
- Vaccines: 12%
- Metabolic/CV: 7%

Total patient exposure

- Diamyd: 376
- Placebo: 844

Most commonly reported adverse events:

- tenderness, injection site edema, injection site pain and injection site reaction.
- no difference in the rate of occurrence of the adverse events between active Diamyd® treatment and placebo

Vaccine Manufacturing – Control and Predictability

• 10,000 square feet site comprising clean rooms, laboratory facilities and office space
• Transferring manufacturing of recombinant GAD65 (active pharmaceutical ingredient in the diabetes vaccine Diamyd®) to own facility
• Making Diamyd Medical independent of third parties
Diamyd® market exclusivity and manufacturing

Core Intellectual Property
- **Substance of matter** in the US until **2032**
- Intralymphatic administration of Diamyd® in Europe, Japan, Australia and Russia, additional countries pending, expiry **2035**. HLA subgroups and biomarkers in national phase with expiry **2035** and later pending.

Regulatory exclusivity
- US BLA approval provides **12 years exclusivity**
- US orphan designation provides **7 years exclusivity** from approval
- European approval provides **10 years of exclusivity**

Manufacturing
- Formulated drug product (Diamyd®) in place for ongoing trials and phase III
- Transfer of drug substance (GAD) manufacturing process to own manufacturing facility to secure core asset and prepare for commercial readiness
Comprehensive knowledge ahead of Phase III

1. Presence of HLA DR3-DQ2 increases effect of GAD-alum
2. Additional subcutaneous injections further increases effect of GAD-alum
3. Superior safety profile and convenience
4. Intralymphatic injections of GAD-alum superior to subcutaneous injections

Diamyd® has been evaluated without safety concerns in clinical trials encompassing more than 1,000 individuals
Modified Release GABA

Primary Indication
Type 1 diabetes

Label expansion
LADA, Insulin-deficient type 2 diabetes

Mechanism of Action
Activate GABA-receptors in the pancreas

Clinical Effect
• Regenerate endogenous insulin production, reduce short- and long-term complications
• Prevention of hypoglycemia

Mode of Administration
Oral

Development status
Phase Ib/IIa ongoing

Licensing Status
Global rights available
Clinical results with attractive path to market for Remygen®

- Ongoing clinical Phase IIa trial*
  - ReGenerate-1 at the University of Uppsala where Remygen® (proprietary formulation of GABA) alone and in combination with low-dose alprazolam (GABA receptor modulator to enhance effect, see next slide) are being evaluated in long-standing type 1 diabetes patients

- Clinical effects (dose-escalation) from ReGenerate-1 shown on preventing hypoglycemia by correcting the counter regulatory hormone response and increasing time-in-range in long-term type 1 diabetes*
  - Clinical effects of GABA shown on decreasing glucagon secretion in recent-onset type 1 diabetes**
  - Preclinical effects on insulin secretion, glucagon secretion and beta cell regeneration
  - Endogenous substance with very good safety profile***

*Favorable clinical effects following dose-escalation communicated in November 2019
**Preliminary results presented at EASD 2019 by Professor Kenneth McCormick, University of Alabama at Birmingham
***Favorable safety review following dose-escalation in November 2019 and combination with Alprazolam in January 2021
GABA treatment improves the hormonal response to hypoglycemia

Comparisons between noro- and hypoglycemia for the respective group using a multiple T-test with p-values corrected for multiple testing using the Holm-Sidak method. * denotes p<0.05, ** <0.01

Values are given as mean±SEM

Data based on ReGenerate-1 clinical trial. Presented by professor Per-Ola Carlsson om the Word Diabetes Day 2020
Positive allosteric modulators enhance GABA:s beta cell regenerative effects*

Effect of PAMs on INS-1 cell proliferation. INS-1 cells were cultured with the indicated PAM at a dose range of $10^{-9}$ to $10^{-6}$ M and assessed for their proliferation. Data shown are the average rate of proliferation relative to that of cultures with media alone (designated as 1).

Alprazolam enhances GABA's ability to promote human islet cell replication. Human islets were incubated with a dose range of GABA together with alprazolam (100 ng/ml) for 4 days in the presence of $^{3}$H thymidine. Data shown are the average rate of proliferation relative to that of cultures with medium alone (designated as 1) in a representative study. N = two independent studies with triplicate cultures. The results were very similar in both studies. **$p < 0.01$ and ***$p < 0.001$ for GABA, or GABA + alprazolam vs. control medium alone; *$p < 0.05$ and ***$p < 0.01$ for GABA + alprazolam vs. GABA alone, determined by Student T-test.

→ Potential to safely enhance GABA:s regenerative effects on beta cells by using a small (sub-CNS) dose of benzodiazepines

Remygen® market exclusivity and manufacturing

Core Intellectual Property
- Exclusive license from UCLA on treating diabetes and other inflammatory diseases with GABA
- Formulation patent application (Remygen®). Application in national phase.
- Exclusive license from UCLA on GABA in combination with GABA receptor modulators to enhance the regenerative and immunomodulatory effect. Application in national phase.

Regulatory exclusivity
- 505(b)(2) regulatory pathway in the US provides potentially faster time to market at reduced cost

Manufacturing
- GMP drug substance (GABA) and drug product (Remygen®) manufacturing in place
Board, Management and Scientific Advisors
Board of Directors

Erik Nerpin
Independent of the Company and its principal owners. Chairman since March 2015, Board member since 2012. Other assignments include Chairman of Kancera AB and Blasieholmen Investment Group AB and Board member in among others Effnetplattformen AB.

Anders Essen-Möller

Maria-Teresa Essen-Möller
CEO of Health Solutions AB. Previous experience include Digital Marketing Manager at Sanofi and Account Director at Creuna. Board member since 2009.

Torbjörn Bäckström
CEO of Umecrine AB. Independent of the Company and its principal owners. Board member since 2017. Head of Neurosteroid Research Centre in Umeå and Senior Professor in the Department of Clinical Science, Obstetrics and Gynecology at Umeå University.

Mark A. Atkinson
Professor of Diabetes Research, Department of Pathology, Immunology and Laboratory Medicine, University of Florida, USA. American Diabetes Association Eminent Scholar for Diabetes Research. Director, UF Diabetes Institute, University of Florida. Independent of the Company and its principal owners. Board member since 2018.

Holdings in Diamyd Medical as of August 31, 2020: 41 065 B-shares.
Management

Ulf Hannelius
Chief Executive Officer
PhD in Molecular Biology from Karolinska Institutet in Stockholm and Executive MBA from Stockholm School of Economics. Prior experience from business development in the biotech and medtech industries as well as from academic research in the fields of genetics and molecular biology. Joined Diamyd Medical in 2015.
Holdings in Diamyd Medical as of August 31, 2020: 147 666 B shares.

Martina Widman
Director Clinical Development
M.Sc. in Mechanical Engineering from the Royal Institute of Technology in Stockholm, with a specialization in Biomedical Engineering. Prior experience of clinical operation from the pharmaceutical industry. Joined Diamyd Medical in 2008.
Holdings in Diamyd Medical as of August 31, 2019: 20 000 B shares.

Anna Styrud
Chief Financial Officer
B.Sc. in Business Administration from Uppsala University. Prior experience include Treasurer of Vasakronan AB and various positions in finance and control within real estate and engineering industry. Joined Diamyd Medical in 2010.
Holdings in Diamyd Medical as of August 31, 2020: 100 000 B shares.

Anton Lindqvist
Chief Scientific Officer
M.Sc in Molecular Biotechnology Engineering from Uppsala University. Research experience from University of Pittsburgh, Uppsala University, the Royal Institute of Technology and Karolinska Institutet. Prior experience in managing technical development at several bio-tech companies. Joined Diamyd Medical in 2013.
Holdings in Diamyd Medical as of August 31, 2020: -

Maja Johansson
Facility Manager
Holdings in Diamyd Medical as of August 31, 2020: -

Eva Karfström
Regulatory Manager
Holdings in Diamyd Medical as of August 31, 2020: -
Top Worldwide Experts
Covering the areas of clinical practice and scientific excellence in Type 1 Diabetes and LADA

Prof. Johnny Ludvigsson
Professor of Pediatrics. First in the world to use immune intervention in children and teenagers with newly diagnosed T1D, and in collaboration with others 64kD was found. An alum-formulation of GAD was developed (Diamyd®), used as a treatment in an effort to deviate the immune system and create tolerance.

Prof. David Leslie
Professor of Diabetes and Autoimmunity. Professor Leslie has been Director of the British Diabetic Twin Study since 1982, the world’s largest twin study of its type and Principal 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.

Prof. Ake Lernmark
Professor in Experimental Diabetes Research, Professor Lernmark has 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 using biochemical methods and was thus the first to define autoantibodies against GAD65 in patients with type 1 diabetes.

Prof. Daniel Kaufman
Professor Kaufman's research is focused on studies in the field of autoimmunity, particularly type 1 diabetes (T1D) and understanding the disease mechanisms in order to develop novel therapeutics in mouse models that could potentially be translated to clinical use. Using preclinical models, Dr. Kaufman’s lab helped to develop some of the GAD and GABA-based diagnostics and therapeutics for T1D that are in clinical use or are being tested in clinical trials.

Prof. Mark A. Atkinson
Professor of Diabetes Research, Department of Pathology, Immunology and Laboratory Medicine, University of Florida, USA. American Diabetes Association Eminent Scholar for Diabetes Research. Director, UF Diabetes Institute, University of Florida. Independent of the Company and its principal owners.

Diamyd Medical Board member.
DIAMYD MEDICAL

• Swedish clinical phase pharmaceutical company, founded 1994
• NASDAQ First North Growth Market, ticker DMYD B

FINANCES

• Market Cap Feb 8, 2021 ~ MSEK 1 800
• Cash Nov 30, 2020: MSEK 173

INDICATIONS

• Diabetes
• Autoimmunity

PRODUCT CANDIDATES

• Diamyd® (Phase IIb)
• Remygen® (Phase Ib/IIa)

INVESTMENT

• Next Cell Pharma (Stockholm, Sweden)