Leader in Precision Medicine for Autoimmune Diabetes
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Targeting Autoimmune and Insulin deficient Diabetes

Leading clinical stage pipeline
• First-in-class disease modifying therapies Diamyd® and Remygen®
• Pivotal program with a precision medicine approach

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®), pivotal program (Diamyd®), evaluation of Accelerated Approval pathways (Diamyd®) and establishing GMP vaccine manufacturing facility
• Cash-position of MSEK 228 (February 28, 2021). Market Cap ~MSEK 1,298 (March 31, 2022)

Experienced team
• Significant operational experience in clinical development within diabetes
• Access to world leading scientists and clinical experts
## Leading pipeline in autoimmune diabetes

<table>
<thead>
<tr>
<th>Product</th>
<th>Indication</th>
<th>Trial</th>
<th>Participants</th>
<th>Sponsor</th>
<th>Development Phase</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diamyd®</td>
<td>T1D, intervention</td>
<td>DIAGNODE-3</td>
<td>~330</td>
<td>Diamyd Medical</td>
<td>Preclinical, I, Ia, Ib, III</td>
<td>Screening in Europe ongoing</td>
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<td>Diamyd®</td>
<td>T1D, intervention</td>
<td>DIAGNODE-2</td>
<td>109</td>
<td>Diamyd Medical</td>
<td>Preclinical, I, Ia, Ib, III</td>
<td>Finalized. Results published</td>
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<td>Diamyd®</td>
<td>T1D, intervention</td>
<td>DIAGNODE-1</td>
<td>12</td>
<td>Linköping University</td>
<td>Stage 1 results published</td>
<td>Finalized. Results published</td>
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<tr>
<td>Diamyd®</td>
<td>LADA, intervention</td>
<td>GADinLADA</td>
<td>15</td>
<td>NTNU, Trondheim</td>
<td>Preclinical, I, Ia</td>
<td>Fully recruited / 6 month results released</td>
</tr>
<tr>
<td>Remygen®</td>
<td>T1D, T2D</td>
<td>ReGenerate-1</td>
<td>36</td>
<td>Uppsala University</td>
<td>Stage 1 results published</td>
<td></td>
</tr>
</tbody>
</table>
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
- Pivotal precision medicine program
- Strong support for clinical response and excellent safety profile

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

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

Primary indication and market
> 1.5 million incident cases

Label expansion
Estimated 2.3 million at-risk individuals

Regeneration Autoimmune- and insulin-deficient Type 2 Diabetes

Intervention Autoimmune diabetes

Prevention Autoimmune diabetes

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> 1.5 million incident cases

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Primary indication and market
> 1.5 million incident cases

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 Ahlvist 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.

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* 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 Pozilli & Pieralice, Latent Autoimmune Diabetes in Adults: Current Status and New Horizons, Endocrinol Metab, 2018
Significant annual economic burden of Type 1 Diabetes (T1D)

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.
A disease modifying therapy for T1D

Reduced risk of
- Low blood glucose episodes (hypoglycaemia)
- Life-threatening acidification of the blood (ketoacidosis)
- Diabetic eye disease (retinopathy) and blindness
- Nerve damage (neuropathy) and possible limb amputation
- Diabetes kidney disease (nephropathy) and possible kidney failure
- Cardiovascular complications such as a myocardial infarction and stroke
- Negative social aspects
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

Licensing Status
Global rights available
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.
Genetic variants influence the appearance of autoimmunity

T1D is a collection of distinct endotypes

GAD-specific immunotherapy is influenced by the same genetic variants that influence risk and autoimmunity

Introducing the Endotype Concept to Address the Challenge of Disease Heterogeneity in Type 1 Diabetes

Efficacy of GAD-alum immunotherapy associated with HLA-DR3-DQ2 in recently diagnosed type 1 diabetes

Intramylophatic Glutamic Acid Decarboxylase With Vitamin D Supplementation in Recent-Onset Type 1 Diabetes: A Double-Blind, Randomized, Placebo-Controlled Phase Ib Trial

Genetic variants influence the appearance of autoimmunity

T1D is a collection of distinct endotypes

GAD-specific immunotherapy is influenced by the same genetic variants that influence risk and autoimmunity
Acknowledged Precision Medicine approach

<table>
<thead>
<tr>
<th>Highlights</th>
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<tbody>
<tr>
<td>• New medical consensus regarding genetically defined groups of T1D</td>
</tr>
<tr>
<td>• Strong case for the emerging precision medicine – in line with Diamyd Medical’s approach</td>
</tr>
</tbody>
</table>

Battaglia et al, Introducing the endotype concept to address the challenge of disease heterogeneity in type 1 diabetes, Diabetes Care, 2020
Meta-analysis based on data from more than 500 individuals identifies responders to Diamyd® treatment

Efficacy of GAD-alum immunotherapy associated with HLA-DR3-DQ2 in recently diagnosed type 1 diabetes

Meta-analysis based on 521 individuals from three placebo-controlled randomized trials (Phase III Europe, Phase II Sweden, Phase II US). Low dose = 2 injections; High dose = 3 och 4 injections

Effect on preserving endogenous insulin production in genetically predefined group of T1D

Hannelius et al, Efficacy of GAD-alum immunotherapy associated with HLA-DR3-DQ2 in recently diagnosed type 1 diabetes, Diabetologia 2020.
Phase IIb results prospectively support clinical effect in genetically defined patient population
Updated Meta-analysis based on 600+ individuals strongly supports positive disease-modifying effect in HLA DR3-DQ2 positive individuals

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Estimated treatment effect ratio (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>All individuals (n=627)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>1.176 (1.065, 1.298)</td>
<td>0.0013</td>
</tr>
<tr>
<td>2 injections</td>
<td>1.188 (1.049, 1.344)</td>
<td>0.0066</td>
</tr>
<tr>
<td>3-4 injections</td>
<td>1.166 (1.041, 1.308)</td>
<td>0.0083</td>
</tr>
<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>2 injections</td>
<td>1.208 (1.017, 1.434)</td>
<td>0.0316</td>
</tr>
<tr>
<td>3-4 injections</td>
<td>1.484 (1.264, 1.741)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Presence of HLA DR3-DQ2/X, X not DR4-DQ8 (n=149)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>1.567 (1.299, 1.890)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>2 injections</td>
<td>1.326 (1.052, 1.671)</td>
<td>0.017</td>
</tr>
<tr>
<td>3-4 injections</td>
<td>1.771 (1.431, 2.191)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Meta-analysis based on 627 individuals from four placebo-controlled randomized trials (Phase III Europe, Phase II Sweden, Phase II US, Phase IIb Europe). Low dose = 2 injections; High dose = 3 och 4 injections

Note: Preliminary unpublished results
Meta-analysis supports significant treatment effect on HbA1c (mmol/mol) in HLA DR3-DQ2 positive individuals

<table>
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<tr>
<th>Subgroup</th>
<th>Estimated treatment effect (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presence of HLA DR3-DQ2 (n = 313)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>-3.120 (-5.970, -0.269)</td>
<td>0.032</td>
</tr>
<tr>
<td>2 injections</td>
<td>-1.112 (-4.560, 2.336)</td>
<td>0.5268</td>
</tr>
<tr>
<td>3-4 injections</td>
<td>-4.789 (-8.078, -1.500)</td>
<td>0.0044</td>
</tr>
</tbody>
</table>

Meta-analysis based on 627 individuals from four placebo-controlled randomized trials (Phase III Europe, Phase II Sweden, Phase II US, Phase IIb Europe).

Note: Preliminary unpublished results
Diamyd® responders targeted in precision medicine Phase III trial

Non-responders
HLA DR3-DQ2(-)

Super responders
HLA DR3-DQ2(+), HLA DR4-DQ8(-)

Responders
HLA DR3-DQ2(+), HLA DR4-DQ8(+)
Group 1: 3 injections of 4 µg of Diamyd®

Group 2: 3 injections of Placebo

Study Drug Administration
Vitamin D supplementation
Mixed Meal Tolerance Test

DIAGNODE-3 design
Press Release, April 15, 2021

Diamyd Medical contracts global CRO for Phase III trial with the diabetes vaccine Diamyd®

Diamyd Medical has contracted the global contract research organization (CRO) ICON plc for DIAGNODE-3, a placebo-controlled Phase III precision medicine trial with the diabetes vaccine Diamyd®. The trial is designed to confirm the efficacy and safety of Diamyd® in individuals recently diagnosed with type 1 diabetes who carry the genetically defined haplotype HLA DR3-DQ2. The trial is expected to begin recruiting patients later this year.
Intralymphatic administration route enhances effect of Diamyd® antigen-specific immunotherapy 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.
Convenient 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
Superior safety profile

Patient drop-out rate in clinical trials

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

Total patient exposure

- Diamyd: 513
- Placebo: 990

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

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 T-regls 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.
GADA response and GAD stimulated proliferation and cytokine secretion further support HLA specificity of Diamyd® treatment with stronger response in HLA DR3-DQ2 positive individuals.

Median change from baseline of anti-GAD65 antibodies (GADA) and Proliferation of PMBC (Stimulation Index, SI) (A), and GAD-stimulated secretion by PBMC of IL-10 and IL-13 levels (B) for GAD-alum treated subjects with and without the DR3-DQ2 haplotype Placebo treatment subjects.

P values, Wilcoxon test, are indicated.

Data from the phase IIb trial DIAGNODE-2
Vaccine manufacturing – control and predictability

• 10,000 square feet site comprising clean rooms, laboratory facilities and office space
• Taking over manufacturing of recombinant GAD65 (active pharmaceutical ingredient in the diabetes vaccine Diamyd®)
• The manufacturing facility property acquired in September 2021
• Making Diamyd Medical independent of third parties
Diamyd® (rhGAD65/alum) manufacturing

Upstream process:

Baculovirus expression system & Insect cells

Downstream process:

Clarification Capture Polish Nanofiltration

DP formulation
Diamyd® market exclusivity and manufacturing

Core Intellectual Property
- **Substance of matter** in the US until 2032
- Intralymphatic administration of Diamyd® in Europe, Japan, China, Australia and Russia, additional countries pending, expiry 2035. HLA subgroups and biomarkers in national phase with expiry 2035 and later pending. Intralymphatic administration of additional betacell antigens (proinsulin, preproinsulin etc) approved in Australia, additional countries 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
- Accelerated approval pathways are being evaluated

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
Presence of HLA DR3-DQ2 increases effect of GAD-alum

Additional subcutaneous injections further increases effect of GAD-alum

Superior safety profile and convenience

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
Milestones

Seek Regulatory position on Accelerated Approval pathway for Diamyd®

Full control and predictability over recombinant GAD, a key asset

Regulatory View | Initiate Phase III | GMP facility | Commercial readiness
---|---|---|---
Diamyd® trial to target the genetically predefined group with intralymphatic treatment | | | Building commercial organization and establishing global partnerships
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/Ila 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 induces a hormonal counter-regulatory response in subjects with long-standing type 1 diabetes

Daniel Espes, Hanna Liljebäck, Henrik Hill, Andris Elksnis, José Caballero-Corbalan, Per-Ola Carlsson
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.
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

*Clinically applicable GABA receptor positive allosteric modulators promote \(\beta\)-cell replication. Sci Rep. 2017 Mar 23
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
Erik Nerpin
Holdings in Diamyd Medical as of August 31, 2021: 41 065 B-shares.

Anders Essen-Möller
Holdings in Diamyd Medical as of August 31, 2021: 2 556 223 A-shares and 5 313 040 B-shares. Essen-Möller also holds 1 545 000 B-shares via an endowment insurance.

Maria-Teresa Essen-Möller
Born in 1970. M.Sc. in Business Administration. Independent to the Company, not independent to its principal owners. Chief Commercial Officer at ScientificMed AB. Previous experience include CEO of Health Solutions AB, Digital Marketing Manager at Sanofi and Account Director at Creuna. Board member since 2009.
Holdings in Diamyd Medical as of August 31, 2021: 463 998 B-shares.

Torbjörn Bäckström
Born in 1948. MD, PhD. 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.
Holdings in Diamyd Medical as of August 31, 2021: 1 000 B-shares via company.

Mark A. Atkinson
Born in 1961. PhD. 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, 2021: 16 750 B-shares.

Karin Hehenberger
Born in 1972. M.D., Ph.D, Karolinska Institute, Post-doc at the Joslin center, Harvard Medical School. Founder and CEO of Lyfebulb, Member of the 3B Future Health Ventures Advisory board, Board observer AADI pharmaceuticals, Board member Rolf Luft Foundation for Diabetes research, Board member American Diabetes Association NY/NJ Community Board. Affiliated Board member since April 2021.
Holdings in Diamyd Medical as of August 31, 2021: 10 000 B-shares.
Management

Ulf Hannelius
Chief Executive Officer
Born in 1975. 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, CEO since 2016.
Holdings in Diamyd Medical as of August 31, 2021:
155 000 B shares.

Martina Widman
Director Clinical Development
Holdings in Diamyd Medical as of August 31, 2021:
10 000 B shares.

Anna Styrd
Chief Financial Officer
Born in 1961. 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, 2021:
105 000 B-shares.

Anton Lindqvist
Chief Scientific Officer
Born in 1980. 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, 2021:

Maja Johansson
Head of Production Facility
Holdings in Diamyd Medical as of August 31, 2021:

Eva Karlström
Regulatory Affairs Officer
Holdings in Diamyd Medical as of August 31, 2021:

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. Åke 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 Mar 31, 2022 ~ MSEK 1,298
- Cash Feb 28, 2021: MSEK 228

INDICATIONS

- Diabetes
- Autoimmunity

PRODUCT CANDIDATES

- Diamyd® (Phase III)
- Remygen® (Phase Ib/Ila)

INVESTMENTS

- NextCell Pharma (Stockholm, Sweden)
- MainlyAI (Stockholm, Sweden)