Doctora *honoris causa*

Ingrid E. Lundberg

UAB

Universitat Autònoma de Barcelona

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Discurs llegit
a la cerimònia d'investidura
celebrada a l'Aula Magna d'UAB-Casa Convalescència
el dia 24 de març de 2025

UAB
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Índex

PRESENTACIÓ D'INGRID E. LUNDBERG PER ALBERT SELVA O'CALLAGHAN

Rector Magnífic de la UAB,
Degà de la Facultat de Medicina,
Presidenta del Consell Social,
Autoritats acadèmiques,
Membres de la Comunitat Universitària,
Senyores i senyors,

És per a mi un privilegi i un honor presentar, davant de la comunitat universitària, la professora Ingrid Lundberg, nominada com a doctora *honoris causa* per la nostra facultat a petició del Departament de Medicina. No faré aquí un recull exhaustiu dels seus mèrits o del seu *currículum vitae*, que, d'altra banda, ja estan recollits al llibret de l'acte d'investidura que trobareu al web de la UAB. Faré, en canvi, una *laudatio* dels aspectes més rellevants des del punt de vista acadèmic i humà d'aquesta professora.

Vaig conèixer Ingrid Lundberg durant una reunió científica que va tenir lloc a Estocolm, Suècia, concretament a l'Institut Karolinska, l'any 2004, fa més de vint anys. El lloc on se celebrava l'esdeveniment era la seu on es lliuren els premis Nobel, un marc, doncs, incomparable. Ja aleshores em va cridar l'atenció la manera d'expressar el seu coneixement sobre aquestes malalties tan complexes i rares, les miositis, i la facilitat de relacionar-se i comunicar-se amb altres metges clínics, però també amb científics d'altres especialitats i investigadors bàsics. El títol de la reunió, «Interaccions entre el sistema immunitari i el metabolisme muscular: per què es debiliten els músculs en pacients amb

malalties musculars inflamatòries cròniques?», ja era indicatiu de la seva curiositat i inquietud per la malaltia i del seu compromís per intentar ajudar els malalts que la pateixen.

Al llarg de tots aquests anys, la professora Lundberg ha estat clau en el lideratge de projectes rellevants que han canviat i modificat el coneixement mèdic i científic sobre aquestes malalties, entre els quals cal assenyalar la classificació de les miopaties inflamatòries de l'any 2017, actualment en revisió en un projecte liderat per ella mateixa, el registre europeu de miositis, o el recent projecte sobre la història natural de les malalties musculars inflamatòries. Ha publicat nombrosos estudis en revistes d'alt impacte i ha participat en conferències, simposis i congressos per tot el món.

Una de les capacitats de la professora Lundberg és la d'aglutinar metges clínics i investigadors d'arreu del món i engrescar-los en projectes que tenen repercussió en la pràctica clínica. L'any 2015, es va celebrar el primer congrés mundial dedicat a l'estudi de les miopaties inflamatòries (Global Conference on Myositis). Va ser precisament la professora Lundberg la impulsora d'aquestes reunions, que s'han anat celebrant cada dos anys i que únicament han estat interrompudes per la pandèmia de COVID-19, que tantes coses va interrompre. L'última reunió va tenir lloc l'any 2024 a Pittsburgh, Estats Units, i la professora Ingrid Lundberg hi va tenir també un paper molt rellevant.

El seu lideratge a l'Institut Karolinska, on desenvolupa la seva activitat assistencial, docent i investigadora, actua com un pol d'atracció per a metges i metgesses d'arreu d'Europa, Àsia o els Estats Units, que hi acudeixen no només per formar-se en l'estudi d'aquestes malalties, sinó també per participar en la recerca capdavantera que desenvolupa. Metges i metgesses formats a la Universitat Autònoma de Barcelona han tingut l'oportunitat de gaudir d'aquesta experiència. La doctora Ane Labirua (avui també present en aquest acte acadèmic), que va llegir la seva tesi doctoral sobre la síndrome antisintetasa, i de qui em

consta que la professora Lundberg guarda un record molt bo, va estar a l'Institut Karolinska l'any 2010. També els doctors Ernesto Trallero i Lluís Jubany van gaudir de l'experiència de participar, l'any 2011, a l'escola d'estiu sobre miositis que cada any organitzava la professora Lundberg. Més recentment, la doctora Irene Peralta, reumatòloga formada a la Unitat Docent Germans Trias i Pujol, uns mesos després d'una estada a l'Institut Karolinska ha passat a formar part de l'equip de la professora Lundberg com a responsable de l'estudi sobre la història natural d'aquestes malalties, les miositis. Tot això ha contribuït indubtablement a enfortir els llaços entre ambdues institucions. Aquesta generositat en acceptar metges i metgesses joves al seu centre és una característica que l'honora.

El compromís amb l'ensenyança i la formació ha estat palès durant tots aquests anys en incorporar l'estudi de les miositis a diversos grups d'associacions mèdiques, com ara l'Aliança Europea d'Associacions de Reumatologia (EULAR) o la Xarxa Europea de Malalties Rares i Complexes del Teixit Connectiu i Musculoesquelètic (ReCONNET), o en participar en grups d'estudi sobre la qualitat de vida relacionada amb la salut, com ara el Centre Neuromuscular Europeu (ENMC), entre altres temes d'interès. Hem de recordar que aquestes malalties, les miopaties inflamatòries idiopàtiques, han estat durant molts anys, degut a la seva raresa i dificultat en el tractament i la cura, malalties oblidades, la qual cosa ha comportat un important sofriment als malalts que les pateixen. Per tot això el paper de la professora Lundberg és tan important, ja que contribueix no solament al diagnòstic i tractament d'aquests trastorns, sinó també a visibilitzar-los.

N'hem ressaltat fins ara la capacitat de lideratge, el coneixement científic i la vàlua com a professional mèdica, però seria injust no ressaltar-ne aspectes més humans, com ara la seva relació amb els malalts i les associacions de malalts, la seva generositat i el respecte als seus col·legues i professionals de la salut i, per descomptat, també als malalts i metges joves en període de formació. El seu sentit de l'humor,

la seva bonhomia i una desconeguda per molts habilitat per al ball i la interacció social fora de les estructures acadèmiques són aspectes que també voldria destacar i que contribueixen a ressaltar encara més l'extraordinària persona que és la professora Lundberg.

És per tot això que tinc el plaer, l'honor i el privilegi de demanar al Rector Magnífic de la Universitat Autònoma de Barcelona que s'atorgui el grau de doctora *honoris causa* a la professora Ingrid Lundberg.

Moltes gràcies.

ENGLISH VERSION OF THE INTRODUCTION OF PROF. INGRID E. LUNDBERG BY PROF. ALBERT SELVA O'CALLAGHAN

It is a privilege and an honour to introduce to the university community Professor Ingrid Lundberg, nominated by our Faculty for an honorary doctorate upon the recommendation of the Department of Medicine. I will not provide you with an exhaustive list of the merits or CV of Professor Lundberg, which are already included in the booklet for this event which is available on the UAB website. Instead, I would like to offer you a laudation of Professor Lundberg's most significant achievements from both an academic and human perspective.

I met Professor Ingrid Lundberg during a scientific meeting held at the Karolinska Institute in Stockholm, Sweden, in 2004 – more than two decades ago. The event took place at the prestigious venue where the Nobel Prizes are awarded, providing an exceptional academic setting. Even then, I was deeply impressed by Professor Lundberg's ability to articulate her knowledge of the rare, challenging group of diseases known as myositis and her talent for engaging in meaningful dialogue with clinicians and professionals from other specialities, including basic research. The meeting's topic, "Interactions between the immune system and muscle metabolism – why do muscles weaken in patients with chronic inflammatory muscle diseases?", reflected her scientific curiosity and concern about these conditions and her commitment to helping the affected patients.

Over the years, Professor Lundberg has played a key role in leading projects to advance the medical and scientific knowledge related to these diseases. Among the most notable is the 2017 classification of inflammatory myopathies, which is currently undergoing an update in a project led by her, and the Euromyositis Registry. A more recent initiative is focused on the natural history of inflammatory muscle diseases. She has published numerous studies in high-impact journals and participated in conferences, symposia, and congresses worldwide.

One of Professor Lundberg's great abilities is bringing together clinicians and researchers from around the world, inspiring them to engage in projects that have a direct impact on clinical practice. In 2015, the first-ever Global Conference on Myositis was held, dedicated to the study of inflammatory myopathies. Professor Lundberg has spearheaded these meetings, which have since taken place every two years – interrupted only by the COVID-19 pandemic. At the most recent conference, held in 2024 in Pittsburgh, USA, Professor Lundberg once again played a highly significant role.

Professor Lundberg's leadership at the Karolinska Institute, where she carries out her clinical, teaching, and research activities, serves as a major attraction for physicians from Europe, Asia, and the United States. These professionals are drawn by the opportunity to receive specialised training in the study of these diseases, and to actively participate in the cutting-edge research conducted under her guidance.

Several physicians trained at our institution, the Universitat Autònoma de Barcelona, have benefitted from this experience. Dr. Ane Labirua, who is present at today's event, and held in fond regard by Professor Lundberg, defended her doctoral thesis on antisynthetase syndrome and spent time at the Karolinska Institute in 2010. Dr. Ernesto Trallero and Dr. Lluís Jubany participated in the Myositis Summer School in 2011,

an annual programme organised by Professor Lundberg. More recently, Dr. Irene Peralta, a rheumatologist trained at the Germans Tries i Pujol Teaching Unit, completed a research stay at the Karolinska Institute and has since joined Professor Lundberg's team, where she is now in charge of an international study on the natural history of myositis. This activity has undoubtedly strengthened the ties between our two institutions. Welcoming young doctors to her centre is a great mark of Professor Lundberg's generosity.

Her commitment to education and training has been increasingly evident over the years, through the integration of myositis research into various medical associations and networks, such as the European Alliance of Associations for Rheumatology (EULAR) and the European Reference Network on connective tissue disorders (ReCONNET), as well as participation in research groups, such as the European Neuromuscular Centre (ENMC), focusing on health-related quality of life among other issues. It is important to acknowledge that idiopathic inflammatory myopathies have historically been neglected due to their rarity and the challenges they present for treatment and management. This lack of attention has been detrimental for the individuals affected, and that is why Professor Lundberg's contributions are so important, as her work extends beyond the diagnosis and treatment of these diseases to include efforts aimed at increasing their visibility within the medical community and beyond.

So far, we have highlighted Professor Lundberg's leadership skills, her scientific knowledge, and her excellence as a medical professional. However, we cannot overlook her human qualities – her relationship with patients and patient associations, her generosity, and the respect she shows towards colleagues, healthcare professionals, and young doctors in training. Her sense of humour, warmth, her social interaction beyond academic settings, as well as a little-known talent for dancing, further highlight the extraordinary person she is.

For all these reasons it is my pleasure, honour and privilege to request of the Honourable Rector of the Universitat Autònoma de Barcelona that he confer the degree of Honorary Doctor on Professor Ingrid Lundberg.

Thank you very much.

DISCURS D'INVESTIDURA D'INGRID E. LUNDBERG

AUTOIMMUNE INFLAMMATORY MYOPATHY – A COMPLEX AUTOIMMUNE DISEASE WITH MANY FACES

First of all, I would express my sincere gratitude to Universitat Autònoma de Barcelona for selecting me for this prestigious award. I am really honoured! I would also like to acknowledge that the work behind this recognition was only possible thanks to a lot of work from many collaborators at Karolinska Institutet, as well as international collaborators over many years. The collaboration for more than 20 years with Professor Selva O'Callaghan has been extremely important and resulted in novel information that will come to benefit our own patients as well as for other patients world-wide.

Myositis

The autoimmune inflammatory myopathies, often named idiopathic inflammatory myopathies (IIM) as we do not know the cause of the disease, but in daily life just called myositis, are chronic disorders mainly affecting skeletal muscles leading to severe muscle weakness and low muscle endurance. Other organs are frequently involved such as skin, lungs, joints and heart contributing to varying degree of disability and low quality of life, and an increased risk of early death. This is a heterogenous group of diseases with various clinical manifestations and prognoses. Myositis is a rare rheumatic disorder and may not always be easy to diagnose.

My interest in myositis goes back to meetings with patients during my training to become a rheumatologist. One day I met a man in his fifties who was severely disabled with difficulties to move due to pronounced muscle weakness. He also had elevated muscle enzymes in serum. We performed the usual clinical examination which showed objective muscle weakness, an electromyogram showed signs of myopathy, and a muscle biopsy showed inflammation. At this time three subgroups of myositis had been identified based on differences in clinical and histopathological features in muscle biopsies: dermatomyositis with a skin rash, polymyositis, and inclusion body myositis – both with predominating muscle weakness, the latter with typical inclusions in muscle fibres. My patient had typical features of polymyositis. Treatment with high doses of glucocorticoids was started and the patient was told to rest as was the recommendation in the textbooks at that time. Patients with myositis often have a slow improvement of muscle strength and some hardly improved at all, despite the use of aggressive immunosuppressive treatment. This man came back to the clinic six weeks later, lay down on the floor in my office and performed an impressive set of push-ups to demonstrate how much he had improved after he started to do physical exercise with his elite swimming daughters.

This made me go back to the literature and question our recommendation of rest for individuals with myositis. To my surprise, I found that this recommendation was based on careful studies of muscle biopsies taken from runners before and after a marathon race. After a marathon race massive inflammation was often seen in the muscle tissue. This led to the interpretation that exercise could induce inflammation in the skeletal muscle and if you already had an inflammation this could possibly worsen. Hence the recommendation was to rest when you have signs of inflammation in your muscles.

Exercise in myositis

This was the start of our first research projects in patients with polymyositis and dermatomyositis where we hypothesised that exercise may not be harmful but possibly beneficial. Together with my colleague, physical therapist Helene Alexanderson, we designed an open-label study to test safety of exercise in patients with myositis. We introduced an exercise programme including moderate resistance exercise for 15 minutes followed by a 15-minute walk, 5 days per week, in patients with chronic myositis with low levels of disease activity (Alexanderson H et al. 1999). We performed careful monitoring with close clinical follow-up measuring muscle strength together with measurements of serum levels of enzymes, as well as magnetic resonance imaging (MRI) and muscle biopsy before and after the 12-week exercise programme. Our results were clear: patients improved in muscle strength and there were no signs of worsening muscle inflammation. This study has been followed by several other studies by our group and others with similar results. In another study we introduced exercise early in patients with short-term disease, as soon as possible after starting immunosuppressive treatment, also with no signs of worsening (Alexanderson H et al. 2000). These observations were followed by a randomised controlled trial to explore levels of inflammation and signs of hypoxia within muscle tissue before and after a period of exercise compared to control patients with myositis living their normal lives. In this study the patients in the group with active exercise improved their cycling capacity and achieved lower clinical disease activity (Fig. 1). In muscle tissue we found signs indicating decreased inflammation and improved metabolism (mitochondrial activity) after 12 weeks of supervised exercise performed by aerobic cycling (Alemo Munters L et al. 2013).

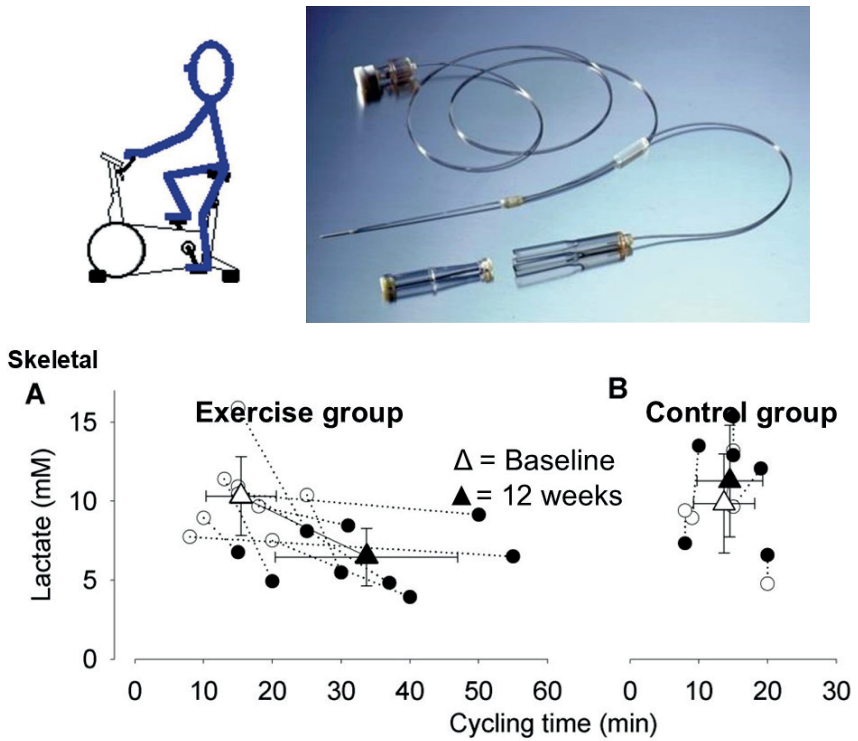

Figure 1. A 12-week randomised controlled trial with endurance exercise resulted in longer cycling time, and reduced lactate levels in the thigh muscle compartment after exhaustion measured by microdialysis in the muscle tissue. (Alemo Munters L et al. Arthritis Res Ther. 2013 Aug 13;15(4):R83. doi: 10.1186/ar4263)

Mitochondrial pathways including the oxidative phosphorylation metabolic pathway were most affected by the endurance exercise, as demonstrated by proteomics analysis. No such changes were seen in the control group. Therefore our data indicate that endurance exercise in patients with established polymyositis or dermatomyositis may activate an aerobic phenotype, promote muscle growth, and simultaneously suppress the inflammatory response in the muscles of patients subject to exercise as supported by a combination of data on gene expression, proteomics and capillary density in repeated muscle biopsies. Today, the textbooks have been revised and treatment recommendations now

include physical exercise in combination with immunosuppressive treatment for patients with myositis.

Classification criteria for myositis

During these studies we identified some clear differences in clinical presentation within the group of patients with myositis. It became clear that we needed new definitions of myositis and its subgroups to distinguish differences in the pathophysiology of these disorders in order to improve therapies. To develop new classification criteria a large study was needed involving many patients and, furthermore, a multidisciplinary effort was required as patients with myositis depending on their predomination clinical manifestations are seen by rheumatologists, dermatologists or neurologists. A huge project involving 47 centres, 976 patients with myositis and an almost equal number of comparator cases mimicking myositis was launched (Fig. 2). Based on data from these patients a proposal for the *2017 EULAR/ACR classification criteria for adult and juvenile myositis* was developed (Lundberg IE et al. 2017). In these criteria a new subgroup was identified: amyopathic dermatomyositis, that is patients with mild or no muscle weakness but with skin manifestations similar to dermatomyositis. These classification criteria have attracted a lot of attention and nowadays they are the most frequently used criteria in clinical trials.

1600 IIM and comparators
IIM 976 (74% adults; 26% children)
Comparators 624 (81% adults; 19% children)

IIM SUBGROUPS	n	%
Juvenile dermatomyositis	251	15.7
Polymyositis	241	15.1
Dermatomyositis	236	14.8
Inclusion body myositis	176	11.0
Amyopathic dermatomyositis	44	2.8
Hypomyopathic dermatomyositis	12	0.8
Immune-mediated necrotising myopathy	11	0.7
Juvenile polymyositis	5	0.3
Non-inflammatory myopathy	624	39.0

Figure 2. Red stars mark centres that contributed to the myositis classification criteria project

However, since the project started in 2004 some new myositis subgroups have been identified, defined by presence of myositis specific autoantibodies, and thus had not been included in the project. This shortcoming in the classification criteria has identified the need to revise the criteria, a project that is currently ongoing. This also reflects the rapid dynamics in the understanding of myositis much related to the identification of several new myositis-specific autoantibodies that are associated with distinct clinical phenotypes (Fig. 3).

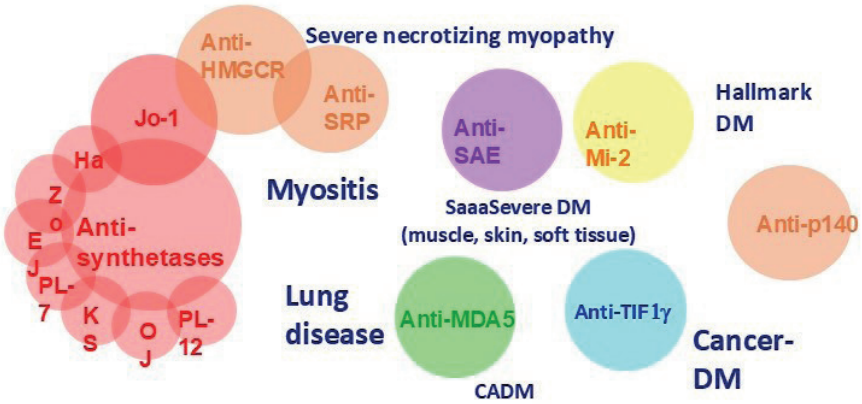

Figure 3. Myositis specific autoantibodies (MSA) and myositis associated autoantibodies (MAA). Courtesy: H. Gunawardena

Myositis registry, MYONET (former Euromyositis)

Encouraged by the international collaborative efforts in the criteria project, together with Professor H. Chinoy, in Manchester, and Professor J. Vencovsky, in Prague, and with the support of an EU-funded project, AUTOCURE, we established an international register, initially named Euromyositis, to follow patients with myositis. As it evolved into an international register it was renamed MYONET (https://euromyositis.eu/ and https://www.myonet.info/) (Fig. 4). The

intention of this collaborative effort was to collect clinical and laboratory data from a large number of patients with myositis and different clinical manifestations, to improve the understanding of the disease, its origin and its development, pathophysiology and treatment response. Another aim of the MYONET registry was to develop a tool to facilitate follow-up in the clinic, to use the register data in discussion with individual patients in making decisions on treatment.

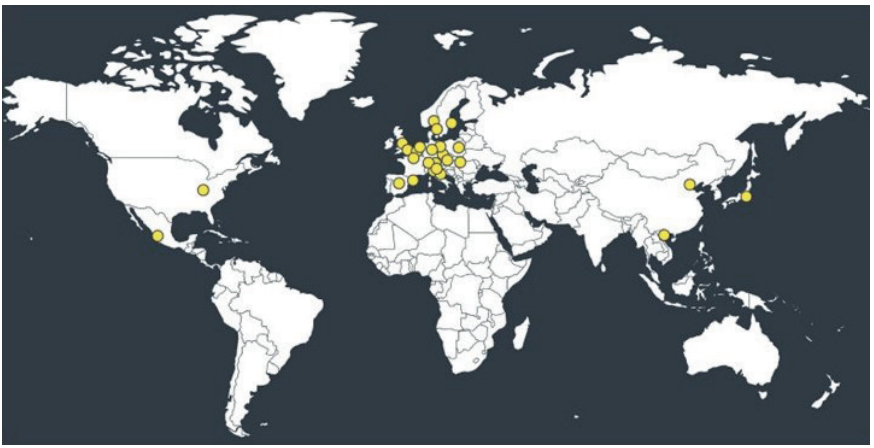

Figure 4. MYONET registry. Yellow dots are centres contributing to the MYONET collaboration

A primary objective of the MYONET registry was to collect basic clinical data together with DNA to identify genetic risk factors for myositis. The MYONET collaboration was part of the first genome-wide association studies (GWAS) and the first Immunochip study in Caucasian patients with polymyositis and dermatomyositis. From these studies it became clear that the strongest risk factor for myositis as a group was the human leukocyte antigen (HLA) 8.1 ancestral haplotype allele (Miller FW et al. 2015).

In follow-up studies of genetic risk factors, we applied subgrouping based on the identification of myositis specific autoantibodies together with clinical manifestations, such as antisynthetase syndrome (with a high frequency of interstitial lung disease, myositis and arthritis), dermatomyositis associated with anti-MDA5 autoantibodies and severe interstitial lung disease, immune mediating necrotising myopathies associated with anti-SRP, and anti-HMGCR autoantibodies (Fig. 3). One of the strongest HLA associations was with HLA-DRB1*03:01 and presence of anti-Jo1 autoantibodies, whereas anti-TIF1 gamma autoantibodies were associated HLADQB1*02:02 in adult-onset dermatomyositis and with HLADQB1*02:01 in juvenile-onset dermatomyositis. Interestingly, TIF1 gamma positive dermatomyositis in adults with dermatomyositis is strongly associated with cancer, which is not the case for juvenile-onset dermatomyositis. The differences in HLA association might indicate that the immune reaction in children and adults with anti-TIF1 gamma positive dermatomyositis target different epitopes of the TIF1gamma protein. Based on Scandinavian and Czech patients with myositis, we have identified additional HLA-alleles associated with other autoantibody defined subtypes, and importantly different autoantibodies or combinations of autoantibodies are strongly associated with different HLA-alleles (Fig. 5) (Leclair V et al. 2023).

Figure 5. Distribution of subgroups of myositis defined by autoantibody profiles, their HLA-associated alleles, amino acids, and clinical/pathological subsets.

Leclair et al. EBioMedicine. 2023 Oct;96:104804. doi: 10.1016/j.ebiom.2023.104804. Epub 2023 Sep 26

Together, the observed HLA-associations with distinct autoantibody-defined subgroups support the importance of the adaptive immune system in the pathophysiology of myositis as the major role of the HLA molecules is to present antigens to T cells to induce a specific immune activation which can lead to autoantibody production.

Registry data to predict outcome

In addition to the international MYONET we have also established a Swedish myositis registry, SweMyoNet, with longitudinal outcome data and treatment. These registry data were used to analyse the response to conventional immunosuppressive treatment in patients with myositis (IIM) applying the Total Improvement Score (TIS) (Aggarwal R et al. 2016). After one year, 62% of patients showed a

minimal response, 38% achieved a moderate response, but only 19% achieved a major response. When subgrouping patients based on autoantibodies we observed that patients with dermatomyositis-specific autoantibodies achieved better levels of response compared to other autoantibody-defined groups (Espinosa-Ortega F et al. 2022). In contrast, dysphagia, which was associated with a shorter timespan from symptom onset to diagnosis, and intensive initial immunosuppressive treatment was associated with a higher response rate after one year of pharmacologic treatment, regardless of autoantibody status.

The MYONET registry has made it possible to analyse whether certain autoantibodies could predict chronic disease. For this study we included 757 adult patients with myositis from 13 centres worldwide with available autoantibody profiles and clinical follow-up. The outcome was defined by the Myositis Damage Index (MDI) (Espinosa-Ortega F et al. 2024). Patients with anti-PM/Scl autoantibodies developed more damage per year of follow-up since diagnosis, independent of sex and age at diagnosis, compared to the seronegative group, whereas patients with dermatomyositis-specific autoantibodies developed less damage per year of follow-up since diagnosis. These results indicate that the autoantibody profile could serve as a predictor for chronic disease, but these data need to be confirmed in a prospective study.

Immune reactivity in myositis

An overarching question to understand the autoimmune reaction and the target of the immune reactions in order to develop new and more targeted therapies is to understand the triggering factors of the immune reactions and where they take place.

Based on epidemiology studies from the Swedish national registries we have identified infections as risk factors for myositis, in particular respiratory tract infections (Svensson J et al. 2017). But not only infections, also other lung diseases constituted a risk factor for myositis.

There was even a dose-response effect; the more visits due to pulmonary disease diagnoses the higher risk of developing myositis (Fig. 6). This observation was supported by data from a questionnaire sent to patients with myositis and to control individuals without myositis.

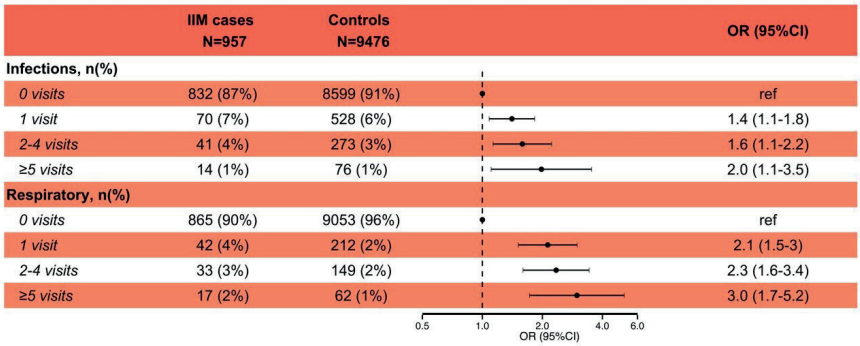

Figure 6. Effects of having multiple previous hospital visits indicating infections or respiratory tract disease on the future risk of developing idiopathic inflammatory myopathies. (OR, odds ratio; CI, confidence interval.)

Svensson J et al. Ann Rheum Dis. 2017 Nov;76(11):1803-1808.

Based on these observations, together with reports of the most common of the myositis specific autoantibodies, the anti-Jo1 autoantibody, being present in individuals before diagnosis of myositis (Miller FW et al. 1990) we combined our Swedish HLA data in patients with myositis from UK, Hungary and Czech Republic and tested the risk of a common environmental exposure for the lungs, namely smoking, and indeed found that smoking is a risk factor for anti-Jo1 positive myositis in patients with HLA-DRB1*03 allele (Chinoy H et al. 2012).

The high frequency of interstitial lung disease in patients with myositis, in particular patients with anti-Jo1 and anti-MDA5 autoantibodies, is intriguing. Furthermore, interstitial lung disease is a major factor contributing to morbidity and mortality in these subsets of myositis.

This has made us focus on these subgroups to understand more of the pathophysiology of this subgroup of myositis. Based on our longitudinally followed cohort of patients with myositis using the SweMyoNet and MYONET registries we identified patients with anti-Jo1 autoantibodies. Next we aimed to identify antigen-specific T cells that reacted with a peptide from the Histidyl-tRNA synthetase (HisRS). By stimulating cells from bronchoalveolar lavage (BAL) and peripheral blood, indeed we found that T cells from patients with HLA-DRB1*03 genotype had a high reactivity to the peptide we tested compared to controls (Galindo-Feria AS et al. 2020). Furthermore, the cells in BAL fluid had a significantly higher reactivity producing pro-inflammatory cytokines compared to cells derived from peripheral blood. We also detected anti-Jo1 autoantibodies in the BAL fluid and in bronchial biopsies we identified germinal centre-like structures. Together, these observations support the hypothesis that the lung may be a site of immune reaction and might contribute to autoantibody production. This work is continued in an ongoing project in which we have used tetramers with peptides from the HisRS protein and confirmed antigen-specific T cells in the peripheral blood of HLA-DRB1*03 anti-Jo1 positive patients.

Still, all the autoantibodies that have been identified do not explain the central involvement of the skeletal muscle in patients with myositis, as the target autoantigens of the autoantibodies found in patients with myositis are ubiquitously expressed in all cells. To challenge this we screened a cDNA library of muscle using patient sera and found some interesting hits. One muscle-specific protein that caught our attention was the Four and Half Limb 1 (FHL1) protein, as mutations in this protein are strongly associated with severe muscle dystrophies such as Emery Dreifuss. We developed assays to screen for anti-FHL1 autoantibodies and found these autoantibodies in up to 25% of patients with myositis but rarely in other autoimmune disorders (Albrecht I et al. 2015). In addition, we found that the anti-FHL1 autoantibodies were often associated with severe myopathy. In follow-up studies we

have observed that the anti-FHL1 autoantibodies are present at the time of diagnosis and often decrease after the start of immunosuppressive treatment (Galindo-Feria AS et al. 2024). They are not totally myositis-specific as they can also be detected at lower frequency and at lower levels in e.g. patients with systemic sclerosis. The anti-FHL1 autoantibody could potentially become a biomarker for prognosis but needs to be tested in other longitudinal cohorts with follow-up data.

Single cell T cell profile in myositis and anti-Jo1 follow-up

To get a better understanding of the immune reactivity in the muscle tissue we performed single cell RNA sequencing on T cells from muscle biopsies and paired circulating memory T cells from six patients with recently diagnosed myositis. A shared finding in all muscle biopsies was infiltration of tissue resident memory T cells (TRM) (Argyriou A et al. 2023). These cells, as well as effector memory cells, were found to be clonally expanded, suggesting that they contribute to the disease pathogenesis. Finally, we detected identical clonally expanded T cells persisting in the muscle tissue after nine months of conventional immunosuppressive treatment in two of these patients, both with clinical low disease activity. This observation might indicate persisting immune reactivity in muscle despite heavy immunosuppression for several months and these cells may contribute to the chronic disease.

Another autoantibody, anti-TIF1 gamma, is strongly associated with cancer in patients with the subgroup dermatomyositis. In a collaboration with Dr Selva O'Callaghan, we found that in anti-TIF1 gamma positive dermatomyositis a cancer diagnosis was confirmed in most patients within three years before or after dermatomyositis diagnosis (Fig. 7) (Dani L et al. 2020). We also detected anti-TIF1 gamma autoantibodies up to five years before cancer diagnosis and that the autoantibodies could disappear and the dermatomyositis could go into remission with successful treatment of the cancer, supporting

the idea that in these cases dermatomyositis could be a paramalignant phenomenon.

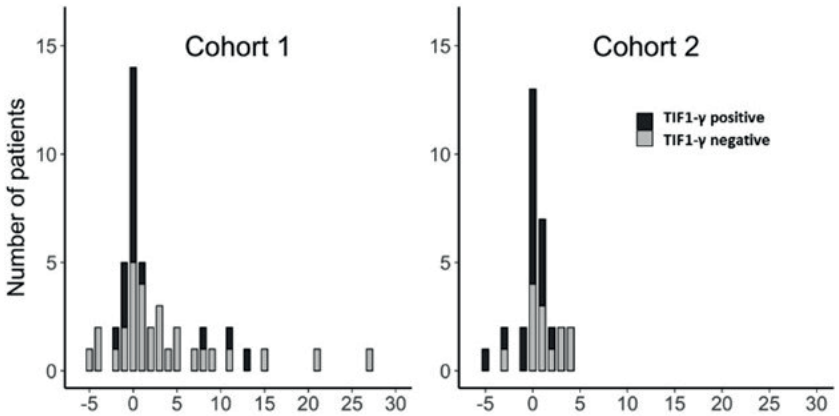

Figure 7. Time interval in years between myositis diagnosis (time 0) and cancer diagnoses in patients positive and negative for anti-TIF1-γ antibodies.
Adapted from Dani L et al. 2020

In summary, over the past decades we have seen an evolution of the myositis disease into several clinically different subgroups, each strongly associated with autoantibodies that are specific for myositis. These observations are clinically important to help in diagnosis but also to predict organ involvement and prognosis. We still need to learn more about the underlying molecular mechanisms in the respective subgroups to guide our treatment decisions in a more individualised fashion and to facilitate development of new and more targeted therapies to improve life for patients with myositis. As these are rare disorders we need to collaborate across the borders of countries and of disciplines, and with patients with myositis to accomplish our aim to improve their quality of life.

Acknowledgement

I want to thank all patients that have inspired me to carry out my research work. In addition, I want to thank my previous supervisors, the late Professor Eva Hedfors of the Karolinska Institutet, and the late Doctor Andrew Engel of the Mayo Clinic, USA, current and previous PhD students, postdocs and senior scientists, colleagues at Karolinska Institutet and collaborators and friends in SweMyoNet and MYONET, and Professor Albert Selva O'Callaghan for a longstanding fruitful collaboration and for nominating me for this prestigious award, and last but not least my family.

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CURRICULUM VITAE
D'INGRID E. LUNDBERG

Ingrid E. Lundberg (Estocolm, Suècia, 1950) és una de les científiques més destacades en l'estudi de les miopaties inflamatòries i les malalties autoimmunitàries sistèmiques.

Llicenciada en Medicina pel Karolinska Institutet d'Estocolm (Suècia), es va doctorar l'any 1991 a la mateixa institució amb una tesi sobre pacients amb anticossos anti-RNP, i posteriorment obtingué una beca per fer el postdoctorat a la Clínica Mayo (1992-1993). Ha estat científica visitant del Departament d'Epidemiologia i Reumatologia de la Harvard Medical School a Boston (els Estats Units). Actualment és catedràtica de Reumatologia del Departament de Medicina del Karolinska Institutet.

La seva recerca es centra en els mecanismes patògens en la inflamació muscular, o miositis, a partir d'estudis longitudinals que inclouen dades clíniques, biòpsies musculars i mostres de sang recollides en una cohort de més de 1.000 pacients amb miositis.

Ha dirigit més de trenta tesis doctorals, ha publicat més de tres-cents articles científics i ha organitzat la primera conferència mundial sobre l'estudi de les miositis (Global Conference on Myositis, GCOM 2015), que avui dia se celebra regularment.

Lundberg va liderar la creació de la Xarxa Europea de Miositis (EUMYONET) dins de l'EULAR, i de la Xarxa Sueca de Miosi-

tis (SweMyoNet), de les quals és investigadora principal. A més, és directora del projecte «Criteris internacionals de classificació per a la miositis».

Ha format part del Consell de la Societat Sueca de Reumatologia (1989-2000) i del Comitè Permanent per l'Educació i la Formació de l'Aliança Europea d'Associacions de Reumatologia (EULAR).

Durant la seva trajectòria ha rebut diferents premis i distincions, com la Medalla de la Universitat Carolina de Praga (2008), el Premi Wyeth de Reumatologia (2008), el Premi Karolina per la qualitat de l'atenció en miositis (2009), la designació de màster de l'American College of Rheumatology (2015), el Premi a l'Excel·lència en Mentoria Investigadora de l'American College of Rheumatology (2016), el Premi pels 25 anys de Serveis Destacats a The Myositis Association (2018), la Medalla Jan van Bremen de la Societat Neerlandesa de Reumatologia (2022), la Gran Medalla de Plata del Karolinska Institutet (2022) i el Premi Heroes in Health Care de The Myositis Association dels Estats Units (2024).

CURRICULUM VITAE

NAME: Ingrid E. Lundberg,

1. Medical Doctor (MD), 1977, Karolinska Institutet.

2. PhD in Rheumatology, 1991, Karolinska Institutet, "Clinical and immunological studies of patients with anti-RNP antibodies". Supervisor: Professor Eva Hedfors.

3. Post doc, 1992-1993, Department of Neurology, Neuromuscular Laboratory, Mayo Clinic, Rochester, USA.

4. Visiting scientist, Febr – May 2004, Department of Epidemiology and Rheumatology, Harvard Medical School, Boston, USA.

5. Docent, Associate professor in Rheumatology, 1998, Karolinska Institutet.

6. Specialized in Rheumatology, 1985, **Specialized in Internal Medicine**, 1987. Senior consultant in rheumatology at Karolinska University Hospital, Stockholm, 1996-2024

7. CURRENT POSITION Professor, Division of Rheumatology, Department of Medicine, Solna, Karolinska Institutet since 2003. Appointed as Senior Professor 2025-01-01

8. SUPERVISOR

Supervisor for 33 Graduate students (past), and 4 ongoing. Supervisor for 15 Post docs

Main supervisor for Graduate students (past): Gerdur Gröndal, 2001, Pernilla Englund (née Nyberg), 2002, Adla Hassan Bakri, 2002, Helene Alexanderson, 2003, Maryam Dastmalchi, 2007, Sevim Barbasso, 2007, Cecilia Wick (née Grundtman), 2008, Christina Dorph, 2009, Björn Löfström, 2009, Ingela Loell 2012, Mei Bruton (née Zong), 2014, Louise Ekholm, 2016, John Svensson, 2017, Quang Tan, 2018, Antonella Notarnicola, 2020, Angeles Galindo, 2022.

Co-supervisor for Graduate students (past): Iva Gunnarsson, 1999, Christer Malm, 2001, Snolaug Arnardottir, 2003, Maryam Fathi 2006, Susanne Pettersson, 2012, Li Alemo Munters, 2013, Malin Regardt, 2014, Jayesh Pandya, 2014, Birgitta Nordgren, 2014, Eva Melin, 2015, Joan Raouf, 2017, Lara Dani 2023, Fabricio Espinosa 2023, Valérie, Leclair, 2023, Weng Ian, 2024, Eveline van Gompel 2025, Kristofer Andreasson 2025

Co-supervisor for Graduate students (present)

Cecilia Leijding, Alexandra Argyriou, Alexander van Deventer, Marina Galesic

Supervisor for Post doc: Carina Boström, PhD, 2000- 2002, Mona Esbjörnsson Liljedahl, PhD, 2002- 2004, Stina Salomonsson, PhD, 2004- 2007, • Andreas Fasth, PhD, 2008- 01-01- 2016, Paulius Venalis, PhD, 2010-11-01- 2016, Karina Gheorghe, PhD, 2010-12 10- 1016, Inka Albrecht, PhD, 2011-03-01- 2016, Cecilia Wick, 2014-2017, Mei Bruton 2015-2019, Cátia Cerqueira, 2016- 2019. Begum Horuluoglu 2019- ongoing. Karin Lodin 2020- ongoing, Maho Nakazawa 2022- ongoing, Yue-Bei Luo 2023- ongoing and Fabricio Espinosa 2023-ongoing.

9. Other merits

EXTERNAL EXAMINER at PhD defense for 15 students in Scandinavia

DISTINCTIONS AND AWARDS (selected):

- Medal from Karl University, Prague, Czech Republic. 2008.
- Major Wyeth prize in Rheumatology, 2008.
- Karolina Prize for Quality care work in myositis, Karolinska University Hospital, 2009
- Affiliated Professor in Rheumatology, University of Southern Denmark, 2013-2018
- Master Award, American College of Rheumatology, 2015
- Excellence in Investigative Mentoring award, American College of Rheumatology, 2016
- Dr Feng Pao Hsii lecturer, Singapore, 2017
- The Myositis Association 25 year achievement award, 2018
- Van Bremen Medal, Dutch Society of Rheumatology, 2022
- Grand Silver Medal, Karolinska Institutet, 2022
- Heroes of Health Care Award 2024 from the The Myositis Association, US

COMMISSIONS OF TRUST (selected)

- Board member of the Swedish Society for Rheumatology 1989-2000, as Scientific Secretary 1993- 98 and as President 1998-2000.
- Chair of EULAR Standing Committee Education and Training, ESCET, 2013-15
- External reviewer for Adolescent Centre, University College London, 2016-2023

NETWORKS

- Project Director in an international project to develop classification criteria for myositis
- Principal investigator for a European Myositis Network, EuMyoNet
- Principal investigator of the Swedish Myositis Network, SweMyoNet

10. INVITED SPEAKER (selected)

- **EULAR, European Congress of Rheumatology** 2007, 2008, 2009, 2010, 2011, 2012, 2013, 2014, 2015, 2016, 2021, 2023
- **American College of Rheumatology,** 2010, 2012, 2013, 2014, 2016, 2017, 2018, 2021
- **APLAR,** Asian Pacific League Against Rheumatism 2010 (Hong Kong), 2019 (Australia)
- **PANLAR,** Pan American League Against Rheumatism (Ecuador), 2019
- **New Zealand Rheumatology Society,** 2019
- **Chile Rheumatology Society,** 2019
- **China Rheumatology Society,** 2020
- **Scandinavian Society of Rheumatology**, Keynote speaker, Norway, 2021, 2023
- **European Pediatric Rheumatology Society,** 2024

11. RESEARCH AREA: The overall aim of my research is to acquire improved understanding of pathogenic mechanisms in rheumatic muscle inflammation, myositis. This is undertaken by clinical and experimental research based on longitudinally followed patients, with collected muscle biopsies and blood samples in one international cohort with more than 6500 patients in the Euromyositis register and one national cohort with closely followed 1000 patients.

12. MAJOR GRANTS: Main applicant for the following grants in SEK

- Swedish Research Council since 2001; **2025-2027: SEK 3 m**
- Stockholm Region ALF**; 2025-27: SEK 2.2 m**
- Swedish Rheumatism Association, annually since 1992: **2023-24: SEK 700 000**
- King Gustaf V 80th Birthday Foundation, annually since 1991**: 2024: SEK 450 000**
- Swedish Heart Lung Foundation **2023-2025: SEK 3 m**
- Donations for myositis-lung research 2024-2025**: SEK 1.2 m**

13. PUBLICATIONS: **Original publications:** 266. **Reviews**: 57. **Book chapters**: 15.

PUBLICATIONS FROM THE LAST 10 YEARS

181 original publications, 30 review papers and 3 book chapters

Impact factor for Ann Rheum Dis IF 20.3 and Arthritis Rheum IF 11.4

1. Notarnicola A, Hellstrom C, Horuluoglu B, Pin E, Preger C, Bonomi F, De Paepe B, De Bleecker JL, Van der Kooi AJ, De Visser M, Sacconi S, Machado P, Badrising UA, Rietveld A, Pruijn G, Rothwell S, Lilleker JB, Chinoy H, Benveniste O, Svenungsson E, Idborg H, Jakobsson PJ, Nilsson P, **Lundberg IE**. Autoantibodies against a subunit of mitochondrial respiratory chain complex I in inclusion body myositis. *J Autoimmun*. 2024 Nov 18;149:103332. doi: 10.1016/j.jaut.2024.103332. Epub ahead of print. PMID: 39561568.
2. Reales G, Amos CI, Benveniste O, Chinoy H, De Bleecker J, De Paepe B, Doria A, Gregersen PK, Lamb JA, Limaye V, **Lundberg IE**, Machado PM, Maurer B, Miller FW, Molberg Ø, Pachman LM, Padyukov L, Radstake TR, Reed AM, Rider LG, Rothwell S, Selva-O'Callaghan A, Vencovský J, Wedderburn LR; Myositis Genetics Consortium; Wallace C. Discovery of new myositis genetic associations through leveraging other immune-mediated diseases. *HGG Adv*. 2024 Oct 10;5(4):100336. doi: 10.1016/j.xhgg.2024.100336. Epub 2024 Jul 22. PMID: 39044428.
3. Che WI, Kuja-Halkola R, Hellgren K, **Lundberg IE**, Westerlind H, Baecklund F, Holmqvist M. Impact of cancer on the mortality of patients with idiopathic inflammatory myopathies

by flexible parametric multistate modelling. *J Intern Med*. 2024 Oct;296(4):336-349. doi: 10.1111/joim.20003. Epub 2024 Aug 2. PMID: 39092528.

4. Espinosa-Ortega F, Lodin K, Dastmalchi M, Vencovsky J, Diederichsen LP, Shinjo SK, Danieli MG, Selva-O'Callaghan A, de Visser M, Griger Z, Ceribelli A, Gómez-Martin D, Andersson H, Vázquez-Del Mercado M, Chinoy H, Lilleker JB, New P, Krogh NS, **Lundberg IE**, Alexanderson H; MYONET Registry Study Group. Autoantibodies and damage in patients with idiopathic inflammatory myopathies: A longitudinal multicenter study from the MYONET international network. *Semin Arthritis Rheum*. 2024 Oct;68:152529. doi: 10.1016/j.semarthrit.2024.152529.
5. Faghihi-Kashani S, Yoshida A, Bozan F, Zanframundo G, Rozza D, Loganathan A, Dourado E, Sambataro G, Ventura IB, Bae SS, Lim D, Gallegos DR, Yamano Y, Selva-O'Callaghan A, Mammen AL, Scirè CA, Montecucco C, Oddis CV, Fiorentino D, Bonella F, Miller FW, **Lundberg IE**, Schmidt J, Rojas-Serrano J, Hudson M, Kuwana M, González-Gay MA, McHugh N, Corte TJ, Doyle TJ, Werth VP, Gupta L, Roman DIP, Bianchessi LM, Devarasetti PK, Shinjo SK, Luppi F, Cavazzana I, Moghadam-Kia S, Fornaro M, Volkmann ER, Piga M, Loarce-Martos J, De Luca G, Knitza J, Wolff-Cecchi V, Sebastiani M, Schiffenbauer A, Rider LG, Campanilho-Marques R, Marts L, Bravi E, Gunawardena H; CLASS project participating investigators; Aggarwal R, Cavagna L. Clinical Characteristics of Anti-Synthetase Syndrome: Analysis from the CLASS project. *Arthritis Rheumatol.* 2024 Oct 28. doi: 10.1002/art.43038. Epub ahead of print.
6. Christopher-Stine L, Ciesluk A, Chinoy H, Goyal NA, Gunter K, Isenberg D, Kielhorn A, **Lundberg IE**, Mozaffar T, Rakhade S, Vandenberg G, Aggarwal R. The Dermatomyositis Disease Symptom Questionnaire (DM-DSQ): A Measure to Assess the Patient Experience of Dermatomyositis Symptoms. *J Rheumatol*. 2024 Sep 1:jrheum.2023-1137. doi: 10.3899/jrheum.2023-1137. Epub ahead of print. PMID: 39089831.
7. Saygin D, Werth V, Paik JJ, Park JK, Needham M, **Lundberg IE**, Christopher-Stine L. Current myositis clinical trials and tribulations. *Ann Rheum Dis*. 2024 Jun 12;83(7):826-829. doi: 10.1136/ard-2023-224652. PMID: 38216318.

8. Hum RM, Lilleker JB, Lamb JA, Oldroyd AGS, Wang G, Wedderburn LR, Diederichsen LP, Schmidt J, Danieli MG, Oakley P, Griger Z, Nguyen Thi Phuong T, Kodishala C, Vázquez-Del Mercado M, Andersson H, De Paepe B, De Bleecker JL, Maurer B, McCann L, Pipitone N, McHugh N, New RP, Ollier WE, Krogh NS, Vencovsky J, **Lundberg IE**, Chinoy H; MYONET Registry. Comparison of clinical features between patients with anti-synthetase syndrome and dermatomyositis: results from the MYONET registry. *Rheumatology* (Oxford). 2024 Aug 1;63(8):2093-2100.
9. Leijding C, Kaewin S, Andreasson KM, Horuluoglu B, Galindo-Feria AS, Van Gompel E, Dastmalchi M, Gastaldello S, Alexanderson H, **Lundberg IE**, Andersson DC. Serum from patients with idiopathic inflammatory myopathy induces skeletal muscle weakness. *Ann Rheum Dis.* 2024 Aug 28:ard-2024-225912. doi: 10.1136/ard-2024-225912. Epub ahead of print. PMID: 39197873.
10. Loganathan A, Zanframundo G, Yoshida A, Faghihi-Kashani S, Bauer Ventura I, Dourado E, Bozan F, Sambataro G, Yamano Y, Bae SS, Lim D, Ceribelli A, Isailovic N, Selmi C, Fertig N, Bravi E, Kaneko Y, Saraiva AP, Jovani V, Bachiller-Corral J, Cifrian J, Mera-Varela A, Moghadam-Kia S, Wolff V, Campagne J, Meyer A, Giannini M, Triantafyllias K, Knitza J, Gupta L, Molad Y, Iannone F, Cavazzana I, Piga M, De Luca G, Tansley S, Bozzalla-Cassione E, Bonella F, Corte TJ, Doyle TJ, Fiorentino D, González-Gay MA, Hudson M, Kuwana M, **Lundberg IE**, Mammen AL, McHugh NJ, Miller FW, Montecucco C, Oddis CV, Rojas-Serrano J, Schmidt J, Scirè CA, Selva-O'Callaghan A, Werth VP, Alpini C, Bozzini S, Cavagna L, Aggarwal R; CLASS Project. Agreement between local and central anti-synthetase antibodies detection: results from the Classification Criteria of Anti-Synthetase Syndrome project biobank. *Clin Exp Rheumatol.* 2024 Feb;42(2):277-287. doi: 10.55563/clinexprheumatol/s14zq8. Epub 2024 Mar 14. PMID: 38488094.
11. Galindo-Feria AS, Lodin K, Horuluoglu B, Sarrafzadeh-Zargar S, Wigren E, Gräslund S, Danielsson O, Wahren-Herlenius M, Dastmalchi M, **Lundberg IE**; SweMyoNet consortium. Anti-FHL1 autoantibodies in adult patients with myositis: a longitudinal follow-up analysis. *Rheumatology* (Oxford). 2024 Jun 4:keae317. doi: 10.1093/rheumatology/keae317. Epub ahead of print. PMID: 38833674.

12. Bianchi M, Kozyrev SV, Notarnicola A, Sandling JK, Pettersson M, Leonard D, Sjöwall C, Gunnarsson I, Rantapää-Dahlqvist S, Bengtsson AA, Jönsen A, Svenungsson E, Enocsson H, Kvarnström M, Forsblad-d'Elia H, Magnusson Bucher S, Norheim KB, Baecklund E, Jonsson R, Hammenfors D, Eriksson P, Mandl T, Omdal R, Padyukov L, Andersson H, Molberg Ø, Pyndt Diederichsen L, Syvänen AC, Wahren-Herlenius M, Nordmark G, **Lundberg IE**, Rönnblom L, Lindblad-Toh K. Unraveling the Genetics of Shared Clinical and Serological Manifestations in Systemic Inflammatory Autoimmune Diseases. *Arthritis Rheumatol.* 2025 Feb;77(2):212-225.
13. Christopher-Stine L, Ciesluk A, Chinoy H, Goyal NA, Gunter K, Isenberg D, Kielhorn A, **Lundberg IE**, Mozaffar T, Rakhade S, Vandenberg G, Aggarwal R. The Dermatomyositis Disease Symptom Questionnaire (DM-DSQ): A Measure to Assess the Patient Experience of Dermatomyositis Symptoms. *J Rheumatol*. 2024 Sep 1:jrheum.2023-1137. doi: 10.3899/jrheum.2023-1137. Epub ahead of print. PMID: 39089831.
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Patent

Patent application PCT/SE2015/051189, “A Novel Autoantigen in idiopathic inflammatory myopathies” (filed 2015-11-10).

2024-02-26

Ingrid Lundberg

Acord 44/2024, de 24 d'abril, del Consell de Govern

Vista la petició formulada pel Deganat de la Facultat de Medicina i l'acord de la Junta de la Facultat de data 12 de desembre de 2023, pel qual se sol·licita al Consell de Govern el nomenament de la doctora Ingrid E. Lundberg com a doctora *honoris causa* de la Universitat Autònoma de Barcelona.

Atès que, tant del currículum de la candidata com de la documentació referent als seus mèrits i de les circumstàncies que concorren, queda acreditat que la seva activitat en el camp de la docència i de la recerca la fa mereixedora d'obtenir la distinció de doctora *honoris causa* de la Universitat Autònoma de Barcelona.

Atès que la normativa que regula el procediment per a l'atorgament del títol de doctor *honoris causa* aprovada pel Consell de Govern en data 26 de maig de 2004, en el seu article 5.2 estableix que el Consell de Govern podrà atorgar un nomenament cada dos anys a la Facultat de Ciències, a la Facultat de Filosofia i Lletres i a la Facultat de Medicina, i un nomenament cada quatre anys a cadascun dels centres restants.

Atès que la proposta de la Facultat de Medicina compleix els requisits exigits a la normativa abans esmentada.

S'ACORDA:

PRIMER. Nomenar la doctora Ingrid E. Lundberg doctora *honoris causa* de la UAB.

SEGON. Encarregar a la secretària general l'execució i el seguiment d'aquest acord.

TERCER. Comunicar el present acord al Deganat de la Facultat de Medicina.