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Diabetes och celiaki  
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## Forskning

Immunogenetiken vid autoimmun (typ 1) diabetes och relaterade organ specifika autoimmuna sjukdomar har inte helt klarlagts. Vår forskning är fokuserad på betydelsen av HLA och icke-HLA genetiska faktorer när autoimmunitet utvecklas mot betacell specifika autoantigen inklusive insulin, GAD65, IA-2, ZnT8, INS-IGF2 och tetraspanin7. Cellulära och autoantikroppsmedierade funktioner studeras hos barn med förhöjd ärftlig risk för att utveckla en första autoantikropp, som visar sig vara antingen mot insulin i DR4-DQ8 barn eller mot GAD65 i DR3-DQ2 barn. Avsikten är att förstå sjukdomens etiologi genom att ta reda på om en omgivningsfaktor kan göras ansvarig för att trigga den autoimmuna reaktionen. Dessa mekanismer studeras i TEDDY (The Environmental Determinants of Diabetes in the Young) studien. Under 2004-2010 screenades nära 60 000 nyfödda barn för ökad ärftlig risk för autoimmun (typ 1) diabetes och gluten intolerans, och 2525 barn följdes från 3 månaders ålder fyra gånger om året fram till fyra års ålder, och därefter två gånger om året till 15 års ålder. Barn (10%) som utvecklade autoantikroppar mot betaceller fortsatte att följas fyra gånger om året. TEDDY studien bekostas av National Institute of Health (NIH) i USA och omfattar tre kliniker i USA (Georgia/Florida, Colorado och Washington State), Finland, Tyskland och Sverige. De svenska barnen står för 30% av deltagarna. Barn födda 2000-2004 i Diabetes Prediktion i Skåne (DiPiS) studien studeras på liknande sätt.

Vår forskning innefattar också att identifiera autoantigen, och utveckla metoder att bestämma autoantikroppar och cellulära metoder att bestämma hur autoantigen kan trigga autoimmunitet. Studier omfattar också undersökningar hur Pandemrix vaccinet som användes att vaccinera en stor del av befolkningen kunde inducera narkolepsi bara hos personer med HLA DQB1\*06:02. Studier av primär prevention kommer att genomföras i ett internationellt samarbete med Global Platform for the Prevention of Autoimmun Diabetes (GPPAD). Sekundär prevention av autoimmun diabetes genomföres i samarbete med TrialNet, en NIH sammanslutning där vi är ett TrialNet Center för Sverige. Preventionsstudier genomföres i TEDDY Family study (TEFA med stöd av Juvenile Diabetes Research Foundation) där vi tar reda på om glutenfri kost kan förbättra betacellernas funktion hos personer med flera betacellsautoantikroppar.

Experimentellt genomför vi genetiska och funktionella studier på den spontandibetiska BB råtten för att förklara hur monogen diabetes kan uppstå när Gimap5, ett anti-apoptopiskt protein inte längre uttrycks.

Sammantaget går vår forskning ut på att förutsäga och förebygga autoimmun (typ 1) diabetes genom att kombinera screening för HLA högrisk gener och icke-HLA gener för att identifiera personer med hög risk att utveckla en första autoantikropp efter en ännu okänd triggnande faktor i miljön. Studier av primär och sekundär prevention genomföres för att till sist kunna förebygga och bota autoimmun (typ 1) diabetes.

## Anställning

### Principal Investigator

Diabetes och celiaki  
Lunds universitet  
Malmö, Sverige  
2015 sep 21 → present

### Principal Investigator

EXODIAB: Excellence of Diabetes Research in Sweden  
Lunds universitet  
Lund, Sverige  
2010 jan 1 → present

### Emeritus Professor in Medicine

University of Washington, Seattle  
Seattle, USA  
2009 jan 1 → present

### Adjunct Professor

Lund University  
Lund, Sverige

2006 jan 1 → 2008 jan 1

**Adjunct Professor**

Lund University  
Lund, Sverige  
1998 jan 1 → 2005 jan 1

**Member of the Graduate Faculty**

University of Washington, Seattle  
Seattle, USA  
1997 jan 1 → 2008 jan 1

**Robert H. Williams Professor i Medicin**

University of Washington, Seattle  
Seattle, USA  
1995 jan 1 → 2008 dec 31

**Adjunct Professor**

Karolinska Institute  
Stockholm, Sverige  
1995 jan 1 → 2001 jan 1

**Adjunct Professor**

University of Washington, Seattle  
Seattle, USA  
1995 jan 1 → 2007 jan 1

**Professor i experimentell endokrinologi**

Karolinska Institute  
Stockholm, Sverige  
1993 jan 1 → 1994 dec 31

**Affiliate Professor**

University of Washington, Seattle  
Seattle, USA  
1993 jan 1 → 1994 jan 1

**Adjunct Professor**

University of Washington, Seattle  
Seattle, USA  
1991 jan 1 → 1993 jan 1

**Visiting Professor**

Free University of Brussels  
Brussels, Belgien  
1990 jan 1 → 2000 jan 1

**Professor in Diabetes Research**

Lund University  
Lund, Sverige  
1989 jan 1 → 1991 jan 1

**Robert H. Williams Professor i Medicin**

University of Washington, Seattle

Seattle, USA  
1988 jan 1 → 1992 dec 31

### **Adjunct Professor of Medical Cell Biology**

Lund University  
Lund, Sverige  
1984 jan 1 → 1989 jan 1

### **Forskningschef**

Hagedorn Research Institute  
Gentofte, Danmark  
1979 jan 1 → 1987 dec 31

### **Assistant professor**

University of Chicago  
Chicago, USA  
1977 jan 1 → 1978 dec 31

### **Docent (avlönad)**

Umeå University  
Umeå, Sverige  
1973 jan 1 → 1979 dec 31

### **Forskarassistent**

Umeå University  
Umeå, Sverige  
1970 jan 1 → 1973 dec 31

### **Amanuens**

Umeå University  
Umeå, Sverige  
1968 aug 1 → 1970 dec 31

## **Forskningsoutput**

### **Heterogeneity of beta-cell function in subjects with multiple islet autoantibodies in the TEDDY family prevention study - TEFA**

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### **An Age-Related Exponential Decline in the Risk of Multiple Islet Autoantibody Seroconversion During Childhood**

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### **Associations of breastfeeding with childhood autoimmunity, allergies, and overweight: The Environmental Determinants of Diabetes in the Young (TEDDY) study**

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### **First-appearing islet autoantibodies for type 1 diabetes in young children: maternal life events during pregnancy and the child's genetic risk**

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**The KAG motif of HLA-DRB1 ( $\beta$ 71,  $\beta$ 74,  $\beta$ 86) predicts seroconversion and development of type 1 diabetes**

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**Decreased HLA-DQ expression on peripheral blood cells in children with varying number of beta cell autoantibodies**

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### **Hierarchical Order of Distinct Autoantibody Spreading and Progression to Type 1 Diabetes in the TEDDY Study**

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### **Next Generation HLA Sequence Analysis Uncovers Seven HLA-DQ Amino Acid Residues and Six Motifs Resistant to Childhood Type 1 Diabetes**

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### **Prospective virome analyses in young children at increased genetic risk for type 1 diabetes**

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### **Reduced display of conformational epitopes in the N-terminal truncated GAD65 isoform: relevance for people with stiff person syndrome or DQ8/8-positive Type 1 diabetes mellitus**

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### **Predicting Islet Cell Autoimmunity and Type 1 Diabetes: An 8-Year TEDDY Study Progress Report**

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### **Maternal dietary supplement use and development of islet autoimmunity in the offspring: TEDDY study**

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### **Autoantibodies in Pandemrix®-induced narcolepsy: Nine candidate autoantigens fail the conformational autoantibody test**

Wallenius, M., Lind, A., Akel, O., Karlsson, E., Svensson, M., Arvidsson, E., Ramelius, A., Törn, C., Palm, L., Lernmark, Å. & Elding Larsson, H., 2019, I: *Autoimmunity*. 52, 4, s. 185-191 7 s.

### **Eleven Amino Acids of HLA-DRB1 and Fifteen Amino Acids of HLA-DRB3, 4 and 5 Include Potentially "Causal Residues" Responsible for the Risk of Childhood Type 1 Diabetes**

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