Name, Vorname: Klöting, Nora

Geburtsdatum: 23.03.1977

Geburtsort: Greifswald

Familienstand: ledig

Wohnhaft in: 17495 Lühmannsdorf, Am Sportplatz 14

Dienst-Adresse Medizinische Klinik III
Universitätsklinikum Leipzig
Ph.-Rosenthal-Str. 27
04103 Leipzig

Telefon: +49 341 97 13 306

FAX: +49 341 97 13 239

e-mail: nora.kloeting@medizin.uni-leipzig.de

Tabellarischer Lebenslauf

09/1983-09/1987	Grundschule in Karlsburg
09/1987-09/1992	Realschule in Karlsburg
09/1992-09/1993	Schloß-Gymnasium in Gützkow
08/1993-07/1994	Auslandsaufenthalt USA, Highschool, Waco, Texas
09/1994-06/1996	Schloß-Gymnasium in Gützkow
06/1996	Abitur
10/1996-11/1996	BWL-Studium an der Ernst-Moritz-Arndt-Universität, Greifswald
12/1996-03/1996	Versuchstierkundliches Praktikum an der Ernst-Moritz-Arndt-Universität, Greifswald
04/1997-08/2001	Humanbiologiestudium an der Ernst-Moritz-Arndt-Universität, Greifswald
08/2001	Diplom Thema: HLA-DQB1 Genotypen bei Typ-1-Diabetikern mit Manifestation nach dem 30. Lebensjahr in Abhängigkeit von der Dauer der Latenz bis zur Insulinbedürftigkeit und zur Höhe der Antikörper
09/2001-12/2004	Promotionsstudent an der Medizinischen Fakultät, Abteilung für Versuchstierkunde, Ernst-Moritz-Arndt-Universität Greifswald
01/2005	Kumulative Promotion Thema: Phenotypic and genetic analysis in animal models and humans with type 1 diabetes or metabolic syndrome: unraveling complex mammalian diseases
03/2005 -	Wissenschaftliche Mitarbeiterin, Medizinische Klinik III, Universität Leipzig

Phenotypic and genetic analysis in animal models and humans with type 1 diabetes or metabolic syndrome: unraveling complex mammalian diseases

The analysis of complex human diseases is complicated both by genetic heterogeneity and by environmental factors. One way to overcome the problem of genetic heterogeneity in humans may be to cluster diabetic patients by kinship. Here, it was shown by analysis of maternal lines of type 1 diabetics using mitochondrial DNA that a clustering is possible, which should be supplemented by analyzing paternal lines using microsatellite markers on the Y chromosome.
However, an alternative to the genetic differential analysis of complex mammalian diseases is the use of animal models. The availability of inbred animal models closely resembling the human disease are an essential component of genetic investigations in this field, as shown in the results of this work. These findings do not only underscore the utility of the congenic approach in differentially analyzing complex traits, but also show that candidate genes can be identified and that chromosomal exchange can variously influence the phenotype, leading to sub-phenotypes, which may be representative for human beings. Moreover, with the aid of differentially analyzed phenotypes in subcongenic vs. congenic strains, it will also be possible to locate the syntenic region in the human genome. Furthermore, these congenic and subcongenic strains can also be used to study interactions between chromosomal regions and various selected environmental conditions. In this way, it may be possible to learn which region can be influenced by environmental factors and to which extent, an undertaking which will require prospective projects.