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Impaired metabolic control and socio-demographic status in immigrant children at onset of type 1 diabetes

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Abstract

The aim of the present study was to compare clinical and socio-demographic conditions at the onset of type 1 diabetes in children born to immigrant families and children born to Swedish families, and to assess whether those conditions had an impact on metabolic status.

Design: Observational nationwide population-based matched cohort-study on prospectively recorded registry data.

Setting: All children with diabetes in Sweden and their families during 2000 – 2010.

879 children born to immigrant parents out of a total of 13 415 children from the Swediabkids registry were collected. To these we added 2 627 children with Swedish-born parents, matched for gender, age and year of onset.

Results

The proportion of low capillary pH (< 7.30) at onset was higher in the immigrant cohort, 25.8 %, vs 16.4 % in the Swedish cohort (p < 0.001). HbA_{1c} was also higher, 95 mmol/mol (10.8 %) vs 88 (10.2 %), respectively (p < 0.001). In a logistic regression model with low pH as the

dependent variable we were unable to reveal any significant association to socio-demographic factors, but the OR for HbA_{1c} was 0.983 (95 % CI 0.976 – 0.991) and the OR for plasma glucose was 0.953 (95 % CI 0.933 – 0.973).

Conclusion

Children born to immigrant parents have lower capillary pH and higher HbA_{1c} at diabetes onset. Immigrant families harbour lower socio-demographic living conditions, but this fact does not seem to influence the inferior metabolic condition at diabetes onset.

Key words: diabetes type 1, HbA_{1c}, children, adolescents, ethnology, immigration

Introduction

The annual incidence of diabetes mellitus type 1 (T1D) in children varies considerably among different populations in the world [1]. Finland and Sweden have very high incidence rates. Incidence is considerably lower in southern Europe, and is lowest in Asia and Latin America. T1D is a fairly rare disease in East Asia. A secular trend towards increasing incidences has been reported from many high and middle income countries [2-4]. This has been accompanied by a shift to a younger age at onset in Sweden, now also seen in other countries [2, 5]. The aetiology of T1D is probably due to a complex interaction between genetics, environment and lifestyle, mediated by an autoimmune process leading to destruction of the insulin-producing beta cells in the endocrine pancreas and ultimately to a complete lack of endogenous insulin [6, 7].

Migration as a natural experiment

During recent decades, significant numbers of immigrants have moved to Sweden from regions where the incidence of T1D is considerably lower. In a previous study we showed that children born in Sweden into these families have a much lower risk of T1D compared to their indigenous Swedish peers [8]. We have also seen that the impact of being born in Sweden into

these families increases the risk of T1D in offspring [9]. This same finding of increased risk for immigrant children to incur diabetes, if being born in a western country, was confirmed by another Swedish study focusing on genetics and an Italian study focusing on age at onset [10, 11].

The aim of the present study was to compare clinical and socio-demographic conditions at T1D onset of children born to immigrant families and children born to Swedish families, and to assess whether those conditions had an impact on metabolic status.

The principal, but complex, exposure in this study differentiating the two groups of children is migration of the parents.

Methods

Design: Observational nationwide population-based matched cohort-study on prospectively collected registry data.

Setting: According to Swedish national guidelines for childhood diabetes, every child with symptoms of diabetes is referred to a paediatric clinic for hospital treatment for approximately one week. In Sweden, paediatric departments treat all children and adolescents aged 0 to 18 years with diabetes within their catchment areas. Outpatient attendance data from all Swedish paediatric diabetes centres (n=43) are registered in the National Quality Registry for Paediatric Diabetes in Sweden – *Swediabkids*, which was established in 2000 [12, 13]. The completeness of centres reporting data increased from 32% to 100% from 2000 to 2007. Thus the registry includes data on almost all (around 99%) of the children and adolescents with diabetes in Sweden. Up to the end of 2011, the registry includes data from more than 315 000 outpatient visits. In addition, in 2008 we found complete concordance with reports to the Swedish Diabetes Registry. This register is estimated to have completeness close to 100%. In all 13 415 children and adolescents were registered in *Swediabkids* from 2000 to 2010. From this registry we collected every patient with non-Swedish background, i.e. both parents are

born outside Sweden. We found 879 (6.6 %) children with diabetes to immigrant families (Immigrant cohort). The overall proportion of people in Sweden with this background is 20 % of whom 380 000 are children (0 -17 years). To this cohort of 879 children we added 2 627 Swedish children from the same registry, for whom both parents are born in Sweden, three for each immigrant child (Swedish cohort). The individuals from the two cohorts were matched according to gender, age and year of diabetes onset. Socio-demographic data were collected from Swedish national registers held by Statistics Sweden. All Swedish residents are assigned a unique 10-digit ID number at birth or immigration, personal identification number (PIN) [14]. This PIN was used to link information from different registry sources. The PINs were replaced by consecutive numbers, thereby concealing the identity of the patients to all investigators. All collected data relate to the date of T1D onset of each child. Height and weight were measured, and BMI was calculated and expressed according to Swedish national reference data [15]. Glucose was measured in plasma; pH, standard bicarbonate and HbA_{1c} were measured in capillary blood. All blood tests were done at arrival to the hospital before start of insulin treatment. HbA_{1c} values are presented in IFCC units (mmol/mol), followed by NGSP units (%), in parentheses: 58 mmol/mol (IFCC) corresponds to 7.5% (NGSP) [16]. All paediatric diabetes centres in Sweden participate in Equalis, External Quality Assurance in Laboratory Medicine in Sweden, for external quality assessment of clinical laboratory investigations [17, 18].

Ethics

This study was approved by the Regional Ethics Committee in Stockholm.

Statistics

Clinical characteristics of children with type 1 diabetes at onset are described with median and inter-quartile range, while the variables are non-normally distributed (Table 1). Maternal socio-demographic status is described with percentages (Table 2). Ordinal and non-normally

distributed continuous data were analyzed using Pearson Chi-square test or Mann Whitney U-test.

Multiple logistic regression models were used to estimate associations between biological and socio-demographic data as independent variables, and pH < 7.30 at onset as the dependent variable. Statistical significance was defined by $p < 0.05$ (2-sided).

Data were analysed by using IBM SPSS Statistics 20.0, Armonk; NY; USA.

Results

Clinical and socio-demographic data from diabetes onset were collected and compared between the two cohorts. The proportion of girls among the children with diabetes was higher in the immigrants (49.1%) in relation to the whole diabetes population (45.7%), $p = 0.049$. Paternal age was higher in the immigrant group, but no obvious difference was observed for maternal age. Median BMI-sds was lower in the Swedish cohort ($p = 0.027$) and there was a tendency towards a greater proportion of weight loss ($p = 0.054$). There was no difference in plasma glucose at onset. The proportion of low capillary pH (< 7.30) was higher in the immigrant cohort, 26 % vs. 16 % ($p < 0.001$). The immigrant children had lower median capillary pH of 7.35 vs. 7.37 ($p < 0.001$) and a corresponding difference was seen for bicarbonate. HbA_{1c} was higher in the immigrant group, 94 mmol/mol (10.8 %) vs. 88 (10.2 %) ($p = 0.005$) (Table 1).

There were considerable inequalities for socio-demographic conditions in almost every aspect. The parents of the immigrant children comprised more single parents, were less well educated, needing more social welfare, were often unemployed and lived in renting accommodations. The number of children differed between the two cohorts as did the birth order (Table 2).

The numbers and the region/country of origin of the immigrant parents and their children are shown in table (Table 3). The majority, 641 (72.9 %), of the children born into the immigrant

families, were born in Sweden. Besides the Nordic countries Iraq, former Yugoslavia and Somalia have a considerable number of immigrants in Sweden according to national demographic data from Statistics Sweden.

When comparing girls to boys within the cohorts, we were unable to discern any disparities according to low capillary pH. However, for HbA_{1c}, there was a gender difference among the Swedish children, girls having higher HbA_{1c}, 90 vs. 87 for boys ($p = 0.032$).

In a multiple logistic regression model using $pH < 7.30$ in capillary blood as the dependent variable, testing for socio-demographic factors, type (Immigrants/Swedes) was the only significant outcome, OR 1.776 (95 % CI 1.339 – 2.357). In a similar model with the same dependent variable, tested for both social and clinical variables, the outcomes were OR 1.797 (95% CI 1.196 – 2.700) for type, OR 0.953 (95% CI 0.933 – 0.973) for plasma glucose and OR 0.983 (95% CI 0.976 – 0.991) for HbA_{1c}.

Discussion

According to annual reports from the Swediabkids registry more than 97 % of all diagnosed diabetes among children and adolescents in Sweden is type 1, confirmed by immunological and genetic studies [19, 20]. Immigrants change environment, and perhaps lifestyle, influencing an altered risk of developing diseases for themselves and their offspring. The present study indicates that children born to immigrant families in Sweden have worse metabolic status at diabetes onset compared to children of Swedish descent according to capillary pH, bicarbonate and HbA_{1c}, but not plasma glucose and BMI- SDS. A high HbA_{1c} – value from start or during the first year of treatment predisposes future poor control and thereby increases the risk for late complications [21-24]. The figures found for low pH in the Swedish children were equivalent to those in an earlier Swedish study [25]. The gender ratio was more equal in the immigrant group. There was a slight gender difference for HbA_{1c} in the Swedish children, girls having higher HbA_{1c}. This finding may seem surprising as we could

not see the same in immigrant girls. The lower BMI-SDS and the tendency to a higher proportion of weight loss in the Swedish cohort were somewhat surprising as we had expected the opposite, the immigrant children coming later for diagnosis. On the other hand, it is possible that the immigrant children being a complex group of diverse ethnicity had higher BMI before diabetes onset than the Swedish children and therefor still presenting a higher BMI after onset.

Immigrant families possess poorer social living conditions, but socio-demographic conditions do not seem to explain the inferior metabolic situation at diabetes onset. One can assume that shortcomings in understanding Swedish, the immigrant parents caring for more children and the fact that they come from a different cultural background, might explain why their offspring has inferior metabolic status at T1D onset. We have not been able to investigate those issues in our study, but this has been addressed in other studies [26, 27]. Immigrant families often originate from countries where T1D occurs more seldom and are therefore less likely to be familiar with early symptoms of this condition. The Parma campaign successfully completed in Italy to diminish the incidence of diabetes keto-acidosis is an admirable example of informing the public about severe disease symptoms, although it does not address immigrant families specifically [28]. The problem of handling patients and families with different cultural background and language is a demanding task with the aim of equalising the metabolic control of T1D between ethnic groups and Swedes [29].

Finland, the only country with a higher incidence than Sweden, is included in the group of immigrants, this may have an impact on the results, the differences seen being less obvious. However, when we performed a separate calculation excluding the Nordic countries the results became the same, figures not shown. Genetic explanations for the worse metabolic start could not be completely ruled out, but seem unlikely. The factor of being born in Sweden, was not addressed in this study, but in previous studies this was presented as a

reason for increased risk of incurring T1D among immigrant children but for the conditions at onset this factor seems irrelevant [9, 30]. Epigenetic factors are probably active in the pathogenesis of T1D but for the metabolic status at diabetes onset this is more elusive [7]. In fact the majority (72.9 %) of the immigrant children were born in Sweden. The immigrant cohort comprises children from very diverse ethnic and geographic regions, so their cultural and living conditions are most disparate and this fact delimits etiological associations related to separate regions or countries.

The major strength of this study is the large, representative and ethnically diverse study population made possible by the high quality and wide coverage of the Swedish national registers and the comparably high immigration rate into the Swedish society in recent decades. The completeness, covering all children with diabetes in an entire country of more than 9.5 million people for more than 10 years is also a considerable strength. The unique personal identification number (PIN) for every resident in Sweden makes it possible to link socio-demographic data from national registers held by Statistics Sweden not only for the patient but for all family members [14]. There may be a few immigrant families that have moved abroad and thus are missing in the national registries, but this hardly affects the results. The immigrant populations living in Sweden are not necessarily equivalent to the ones in the country of origin. The fact that families from foreign countries with children suffering from a chronic disease tend to refrain from emigration, the healthy migrant effect, could have an impact on the results. This study addresses however the conditions at diabetes onset and not the risk of developing T1D and the comparison with indigenous Swedish peers living in the country of origin is perhaps not crucial in that point.

The present observational study using the influence of migration as a very broad exposure involving cultural, lifestyle, genetic and epigenetic factors delimits the chances of finding specific associations and rules out causality. There is reason to believe that the modern

affluent society, nowadays more prevalent worldwide, increases beta cell stress, modulated by factors above including nutrition and immunity [7]. However, the disparities seen in metabolic conditions at diabetes onset in the present study are perhaps only partly explained by social and cultural stress and may rather be due to linguistic and other cultural differences. Further studies are needed. It would, if possible, be interesting to see the effect of “reverse” migration from high-incidence to low-incidence countries, compare the results and eventually discover keys to protective factors. For the near future, it would be interesting to see whether the differences in metabolic conditions at T1D onset found in this study, will prevail after some years of treatment.

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The authors declare no conflict of interest.

Contribution statement:

US designed the study, collected the data, made statistical analyses, interpreted the results and wrote the manuscript.

JÅ contributed to the design of the study, the interpretation of the results and participated in writing the manuscript.

USa contributed to the design of the study, collecting the data, the interpretation of the results and participated in writing the manuscript.

LS contributed to the collecting of data, made the statistical analyses and participated in writing the manuscript.

All four authors consent to the aproval of the manuscript.

Tables

Table 1. Clinical characteristics of children with type 1 diabetes at onset; the first three on top are matched

	Immigrants n = 879		Swedes n = 2627		p
	Median	I-q range ^a	Median	I-q range ^a	
Female sex (%)	49.1		49.1		1.000
Year of onset	2004	8	2004	8	0.986
Age, yrs	10.2	6.7	10.5	6.9	0.432
Maternal age, yrs	39.0	9.3	39.0	9.0	0.448
Paternal age, yrs	44.0	11.0	42.0	9.0	< 0,001
Height, cm	145.6	38	145.8	40.1	0.501
Weight, kg	34.0	27.9	34.3	26.0	0.769
Weight loss (%) ^b	25.8		30.2		0.054
BMI, SDS	-0.18	2.21	-0.52	1.97	0.027
P-glucose, mmol/L	24.0	10.5	25.0	9.7	0.20
HbA1c, mmol/mol	94	36	88	35	0.005
pH (capillary blood)	7.35	0.1	7.37	0.07	< 0.001
pH < 7.30 (%) ^b	25.8		16.4		< 0.001
Bicarbonate, mmol/L	22.0	7.0	23.0	5.3	0.001
Diastolic blood-pressure, mm Hg	66.0	12	65	12	0.789
Systolic blood-pressure, mm Hg	109	20	110	17	0.563

^a Inter-quartile range (75th – 25th percentile)

Group comparisons by Mann-Whitney or ^bChi-square test

Table 2. Maternal socio-demographic status at child's diabetes onset

	Immigrants N = 879	Swedes N = 2627	p
<i>Marital status, %</i>			
married/ attached	72.5	79.8	
single	27.5	20.2	< 0.001
<i>Education, %</i>			
pre-college (< 9 years)	34.8	9.7	
college	41.1	53.9	
university	24.1	36.4	< 0.001
<i>Social welfare, %</i>			
	30.8	4.3	< 0.001
<i>Employment, %</i>			
	45.9	84.6	< 0.001
<i>Housing, %</i>			
renting apartment	63.3	19.0	
own house	36.7	81.0	< 0.001
<i>Number of children, %</i>			
1	7.8	7.6	
2 - 3	55.2	78.9	
> 3	37.0	13.5	< 0.001
<i>Birth order, %</i>			
1st without siblings	7.8	7.6	
1st with siblings	26.8	35.2	
≥ 2nd	65.3	57.3	< 0.001

Group comparisons by Chi-square test

Table 3. Region/country of origin of the immigrant parents and country of birth of their children

<i>Country of origin</i>	<i>mother</i>	<i>father</i>	<i>child^a</i>
	N (%)	N (%)	N (%)
Finland	123 (14.0)	112 (12.7)	10 (1.1)
Other Nordic	37 (4.2)	33 (3.8)	14 (1.6)
Western Europe/USA	18 (2.0)	22 (2.5)	16 (1.8)
Former Yugoslavia	108 (12.3)	111 (12.6)	41 (4.7)
East/South Europe	72 (8.2)	47 (5.3)	18 (2.0)
Iraq	147 (16.7)	160 (18.2)	73 (8.3)
East Asia	42 (4.8)	38 (4.3)	15 (1.7)
South Asia	137 (4.8)	140 (15.9)	23 (2.6)
Somalia	67 (7.6)	66 (7.5)	11 (1.3)
Other Africa	98 (11.1)	109 (12.4)	8 (0.9)
Latin America	25 (2.8)	28 (3.2)	5 (0.6)
Other countries	5 (0.6)	13 (1.5)	4 (0.4)
All	879 (100.0)	879 (100.0)	

^a641 (72.9%) were born in Sweden

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