

Incidence of Type 1 Diabetes in Philadelphia Is Higher in Black Than White Children From 1995 to 1999

Epidemic or misclassification?

TERRI H. LIPMAN, PHD, CRNP^{1,2}
 ABBAS F. JAWAD, PHD^{3,4}
 KATHRYN M. MURPHY, PHD, RN²
 ALAN TUTTLE, MSW, LCSW⁵

REBECCA L. THOMPSON, RN, BSN, CDE⁶
 SARAH J. RATCLIFFE, PHD⁵
 LORRAINE E. LEVITT KATZ, MD^{2,7}

OBJECTIVE — To determine the epidemiology of type 1 diabetes in children in Philadelphia, Pennsylvania, from 1995 through 1999 and compare these data with previous cohorts.

RESEARCH DESIGN AND METHODS — This is a report of a retrospective population-based registry maintained since 1985. Hospital records meeting the following criteria were reviewed: newly diagnosed type 1 diabetes, age 0–14 years, residing in Philadelphia at the time of diagnosis, and diagnosed from 1 January 1995 to 31 December 1999. The secondary source of validation was the School District of Philadelphia. Incidence rates by race and age were compared with 1985–1989 and 1990–1994 cohorts.

RESULTS — A total of 234 case subjects were identified, and the registry was determined to be 96% complete. The overall age-adjusted incidence rate in Philadelphia was 14.8 per 100,000/year. Incidence rates in Hispanic children (15.5 per 100,000/year) and white children (12.8 per 100,000/year) have been relatively stable over 15 years. The incidence in black children (15.2 per 100,000/year), however, has increased dramatically, rising 64% in children 5–9 years of age (14.9 per 100,000/year) and 37% in the 10- to 14-year age-group (26.9 per 100,000/year).

CONCLUSIONS — The overall incidence of type 1 diabetes in Philadelphia is increasing and is similar to other U.S. registries. These are the first data reporting a higher incidence in black children in a registry of children 0–14 years of age. The etiology of the marked increase in incidence in the black population is unknown and underscores the need to establish type 1 diabetes as a reportable disease, so that environmental risk factors may be thoroughly investigated.

Diabetes Care 29:2391–2395, 2006

Type 1 diabetes registries across the country and throughout the world have demonstrated that the incidence of the disease varies greatly. From 1990 to 1994, a >350-fold variation in incidence has been reported among 100

populations worldwide (1). There is also marked variability in the incidence of type 1 diabetes among racial and ethnic groups residing in the same area (2–4). These differences in diabetes incidence provide a basis to investigate the genetic and envi-

ronmental risk factors for the development of type 1 diabetes. The Philadelphia registry is unique in that it includes data from large populations of white, black, and Hispanic children of Puerto Rican origin. Philadelphia has a population of 320,061 children aged 0–14 years (5); 50% of the children are black, and 10% are Hispanic. The epidemiology of type 1 diabetes in children in Philadelphia has been reported for the years 1985–1989 (2) and 1990–1994 (6); those data demonstrated that the highest incidence by racial group was in the Hispanic children (15.2 and 15.5, respectively). A similarly high incidence in this population was also reported in the registries from Puerto Rico (7) and Chicago (3). The data from the 1990–1994 cohort demonstrated an epidemic of type 1 diabetes 2 years after a measles epidemic in Philadelphia and a rising incidence in black children (6).

The incidence of type 1 diabetes in children has been increasing in Europe (8–10). There has also been a rising incidence in the U.S., particularly evident in black children (3,4). The Philadelphia registry includes all children newly diagnosed since 1985, allowing for the examination of trends over time. The purpose of this study was to determine the epidemiology of type 1 diabetes in children in Philadelphia from 1995 to 1999 and compare these data with the previous 1985–1989 and 1990–1995 cohorts, particularly regarding racial differences and the increasing incidence in black children.

RESEARCH DESIGN AND METHODS

All Philadelphia hospitals that admit children were identified, and permission to review records was obtained. The charts of all children diagnosed with type 1 diabetes between 1 January 1995 and 31 December 1999 were reviewed using the World Health Organization (WHO) registry criteria. Applying the criteria from the WHO multinational study and the abstraction of

From the ¹University of Pennsylvania School of Nursing, Philadelphia, Pennsylvania; the ²Division of Endocrinology, Children's Hospital of Philadelphia, Philadelphia, Pennsylvania; the ³Division of Biostatistics and Epidemiology, Children's Hospital of Philadelphia, Philadelphia, Pennsylvania; the ⁴Department of Biostatistics and Epidemiology, University of Pennsylvania, Philadelphia, Pennsylvania; the ⁵Division of Neurology, Children's Hospital of Philadelphia, Philadelphia, Pennsylvania; the ⁶Section of Endocrinology, St. Christopher's Hospital for Children, Philadelphia, Pennsylvania; and the ⁷Department of Pediatrics, University of Pennsylvania, Philadelphia, Pennsylvania.

Address correspondence and reprint requests to Terri H. Lipman, PhD, CRNP, University of Pennsylvania School of Nursing, 420 Guardian Dr., Philadelphia, PA 19104. E-mail: lipman@nursing.upenn.edu.

Received for publication 7 March 2006 and accepted in revised form 4 August 2006.

Abbreviations: WHO, World Health Organization.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

DOI: 10.2337/dc06-0517

© 2006 by the American Diabetes Association.

The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

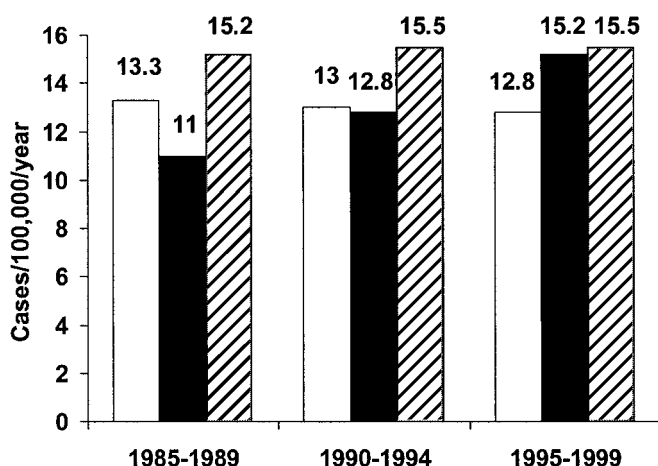


Figure 1—Incidence of type 1 diabetes by race in three cohorts, 1985–1999. □, white children; ■, black children ($P = 0.03$); ▨, Hispanic children.

standard registry data allows for worldwide registry comparison (11).

Data were collected for age at diagnosis, sex, race, and admitting institution. Hispanic case subjects were identified by a Hispanic surname as documented by the U.S. Census Bureau (5). The country of origin of the Hispanic case subjects was ascertained from the children's medical records or health care providers.

A secondary source of ascertainment was necessary to capture Philadelphia children with type 1 diabetes who were admitted to hospitals outside of Philadelphia or who were not admitted to the hospital at diagnosis. The standard of care in Philadelphia, however, remains admitting all children with newly diagnosed type 1 diabetes. Ascertainment of the completeness of the hospital registry was determined through a survey of nurses from the school district of Philadelphia who staff all public, private, and parochial schools. Surveys inquiring about students with diabetes were mailed to nurses of all 628 schools and were completed by nurses at 510 schools (81%), representing 252,896 children. Completeness of ascertainment was calculated through the capture-recapture method (12).

Age-adjusted incidence rates were calculated for the sample in general and for each subgroup (age, sex, and race) using 1990 Philadelphia census data. Age adjustment for the rates was done in 5-year intervals (0–4, 5–9, and 10–14 years) using the direct method with the 1990 U.S. population as the standard population. The 1990 census and population data were used since the 1995–1999 intercensal estimates for Philadelphia estimated an increasing pop-

ulation across the ages and races, while the results of the 2000 census showed that the population had actually been decreasing from 1990 to 2000. Using Poisson distribution, 95% CIs were computed and determined to be significant if the CIs did not overlap. The relationship between variables was analyzed using the χ^2 statistic. Incidence rates by year and cohort for each race were also analyzed over the entire period of 1985–1999. Time series methods were used to test for seasonality, linear trends, or spikes in these incidence rates. In addition, Poisson regression was used to test for differences between the races.

RESULTS— The hospital review identified 234 cases with newly diagnosed type 1 diabetes in Philadelphia in children 0–14 years of age from 1995 to 1999 from six hospitals in Philadelphia. White children accounted for 37%, black children 50%, and Hispanic children 9%; 59% were male. The distribution by age-group was similar to that of the previous data. The highest percentage of cases was in the 10- to 14-year age-group (45%), followed by 5–9 years (37%) and 0–4 years (18%), with a mean age of onset of 8.9 years. School records identified a total of 492 children diagnosed from 1989 to 2002. Because of missing data in schools related to registry variables, only 53 case subjects diagnosed from 1995 to 1999 were used for this analysis. Of the 53 eligible school children with diabetes, 50 were also identified in the hospital registry. The total number of estimated case subjects with the capture-recapture method was 245 (12). The ascertainment rate was calculated for the hospital regis-

try (94%), the secondary source (22%), and the combined registry of primary and secondary sources (96%).

The overall incidence rate was 14.6 per 100,000/year, and the age-adjusted incidence was 14.8 per 100,000/year. This incidence is higher than that reported in 1985–1989 (13.3) and 1990–1994 (13.1) (2,6). The ascertainment-corrected incidence rate was 15.3 per 100,000/year. The incidence in males and females was very similar in the previous cohorts (2,6). These data demonstrate a markedly higher incidence in males overall (15.7 vs. 10.7). The difference was evident in Hispanic and white children. The incidence by sex was equal in the black population. The overall incidence by age-group was 7.4, 16.7, and 20.7 per 100,000/year, respectively. The incidence over the 5 years of the study demonstrated a peak in 1996 (18.7) and the lowest rate in 1997 (11.6). The 1996 peak and 1997 nadir were evident in all races.

The racial data were analyzed across the three cohorts. The incidence of type 1 diabetes in Hispanic children in Philadelphia has been very stable (15.2, 15.5, and 15.5 per 100,000/year) and remains the highest of any racial group aged 0–14 years in the U.S. The incidence in white children has also remained stable (13.3, 13.0, and 12.8). There has been a steady rise in the incidence in black children (11, 12.8, and 15.2), an increase of 38%. Now, for the first time in Philadelphia, the incidence in black children has surpassed the incidence in white children (Fig. 1).

We examined the yearly racial incidence rates for the entire 1985–1999 period. Race itself was not significant in the Poisson regression model. No significant seasonal effects (all $P > 0.1$) or linear trends over time (white, $P = 0.84$; black, $P = 0.07$; Hispanic, $P = 0.41$) were found using time series methods. There was a spike in the incidence for Hispanic children in 1993, as found previously (2) (Fig. 2). However, these nonsignificant results could be due to the high year-to-year variability. While the year-to-year changes in incidence rates in black children were not quite statistically significant (at $\alpha = 0.05$), there was a rise in the majority of incidence rates from one year to the next. When the three cohort incidence rates were compared for each race, there was a significant increasing linear trend ($P = 0.03$) in black children but not in white or Hispanic children, thereby indicating an increasing incidence rate of type 1 diabetes in black children over

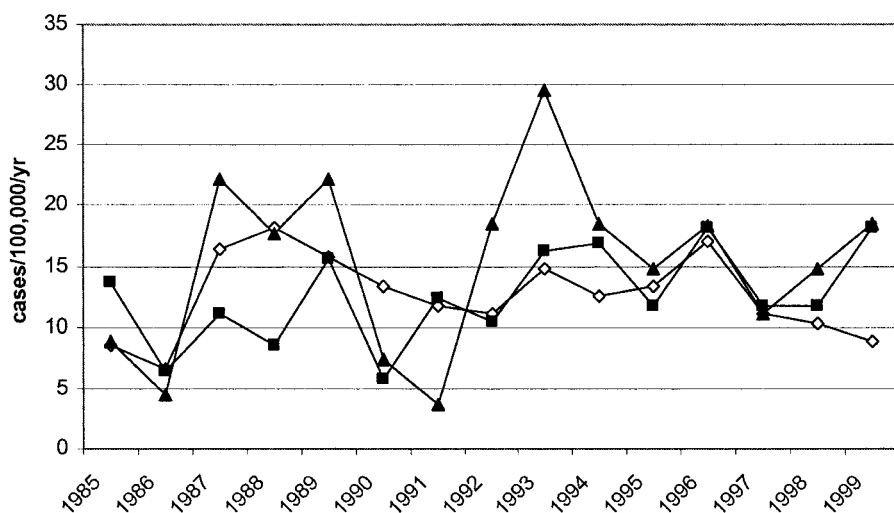


Figure 2—Incidence of type 1 diabetes by race and year, 1985–1999. \diamond , white children; \blacksquare , black children; \blacktriangle , Hispanic children.

time. The incidence rate in black children increased by 16–19% from one cohort to the next.

If age is considered in addition to race, different racial groups act significantly different according to age-group ($P = 0.001$) (Table 1). Analysis of the rise in incidence in black children by age-group demonstrates an increase in the 0- to 4-year age-group (5.0) and the highest rates ever reported in black children in the 5- to 9-year age-group (14.9) and the 10- to 14-year age-group (26.9) (Fig. 3).

The incidence in black children rises dramatically with age. The rate in children 10–14 years of age is significantly higher than in children 5–9 years of age ($P = 0.002$), which is significantly higher than children 0–4 years of age ($P < 0.001$). In white children, the peak incidence was in children 5–9 years of age, while the peak in black and Hispanic children was at 10–14 years of age. When comparing white with black children with diabetes, the risk of type 1 diabetes is 1.9 times greater in white children in the 0- to

4-year age-group ($P = 0.043$). The risk is significantly higher in black children in the 10- to 14-year age-group, 2.4 times that of white children ($P < 0.001$).

When race is combined with sex, the effect is significant ($P = 0.002$). In 1995–1999, the incidence was equal in black males and females. In white and Hispanic children, and in the total cohort, the incidence was higher in males (15.7 vs. 10.7 in the total population). In our previous data, the incidence in males and females was relatively equal (2,6).

CONCLUSIONS— In 1990, when the data from the first type 1 diabetes registry cohort in Philadelphia were collected, there were 155 registries in 70 countries. Of those registries, 12 were in the U.S. Currently, Philadelphia is one of only four U.S. ongoing population-based registries, and the data from Philadelphia remain integral in the identification of racial differences and temporal trends. The incidence of type 1 diabetes in children in Philadelphia was stable between the first and second cohorts but has increased from 1990–1994 to 1995–1999. The incidence in Hispanic children, who in Philadelphia are almost exclusively of Puerto Rican origin, remain the highest of any racial group of children 0–14 years of age in the U.S. Other registries have demonstrated a high incidence in Puerto Rican

Table 1—Incidence of type 1 diabetes in children in Philadelphia from 1995 to 1999 according to race and age

	Age (years)			Crude rate	Adjusted rate*
	0–4	5–9	10–14		
White					
<i>n</i>	22	41	24	87	
Population	48,736	44,577	42,352	135,665	
Rate/100,000	9.0	18.4	11.3	12.8	13.0
95% CI	5.66–13.68	13.20–24.95	7.26–16.86	10.27–15.82	10.2–15.6
Black					
<i>n</i>	14	36	67	117	
Population	55,637	48,205	49,843	153,685	
Rate/100,000	5.0	14.9	26.9	15.2	15.4
95% CI	2.75–8.44	10.46–20.68	20.83–34.13	12.59–18.25	12.6–18.1
Hispanic					
<i>n</i>	5	6	10	21	
Population	9,784	8,769	8,568	27,121	
Rate/100,000	10.2	13.7	23.3	15.5	15.6
95% CI	3.31–23.86	5.02–29.86	11.19–52.89	5.59–23.69	8.9–22.2
Total†					
<i>n</i>	43	86	105	234	
Population	115,678	102,785	101,688	320,151	
Rate/100,000	7.4	16.7	20.7	14.6	14.8
95% CI	5.38–10.02	13.38–20.67	16.89–25.00	12.81–16.61	12.9–16.7

*Direct age-adjusted method using the 1990 U.S. population; †totals are all races 0–14 years of age.

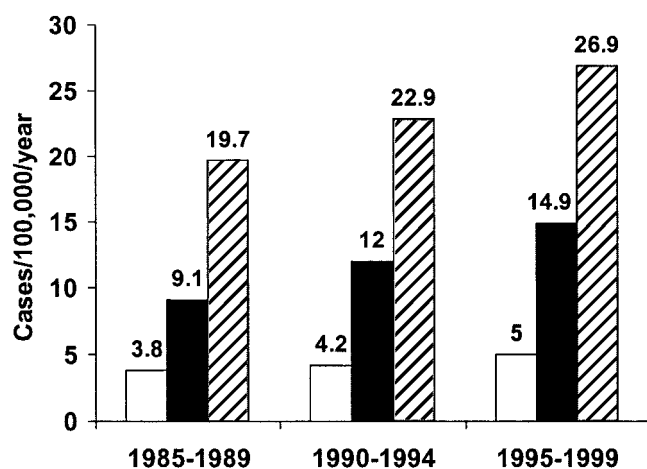


Figure 3—Incidence of type 1 diabetes in black children, 1985–1999. □, 0- to 4-year age-group; ■, 5- to 9-year age-group; ▨, 10- to 14-year age-group.

children (3,7). The etiology of this high incidence remains unclear, and the genetic and environmental factors need further exploration. The incidence of type 1 diabetes in Hispanic children of Puerto Rican origin is high both in Puerto Rico and in the U.S., unlike reports of other populations demonstrating significant changes in incidence rates after migration (13).

The rising incidence in Philadelphia is a reflection of the markedly increased incidence in black children evident in all age-groups. The incidence in black children has increased significantly with each cohort in the Philadelphia registry. Type 1 diabetes continues to be relatively rare in black children 0–4 years of age. White children 0–4 years of age are two times more likely to develop type 1 diabetes than black children. Some racial differences exist in the genetics of diabetes in children (14). Protective genes or environmental factors may be operating in very young black children.

The incidence of type 1 diabetes in black children 5–9 and 10–14 years of age is very high. The incidence in children 5–9 years of age has risen 64% over the three cohorts, from 9.1 to 12.0 per 100,000/year (6) to the current rate of 14.9 per 100,000/year. This is the highest incidence ever reported in black children in this age-group and is 1.6 times the rate we reported in the 1985–1989 cohort (2). The incidence of black children in the 10- to 14-year age-group has increased 37%, from 19.7 per 100,000/year in 1985–1989 to 26.9 per 100,000/year in the present data and is similar to the incidence of 23.6 per 100,000/year in black children 10–14 years of age in Allegheny

County in 1990–1994 (4). It is possible that diabetes in this age-group represents a range of typologies.

The incidence of type 1 diabetes reported by several registries from 1975 to 1984 was much lower in black children (15,16). The incidence in black children has now surpassed the incidence in white children in Philadelphia. The Allegheny County registry reported a higher incidence in black than white children because of a sharp rise in type 1 diabetes in black children 15–19 years of age from 1990 to 1994 (4). The Philadelphia registry does not include children older than 14 years; therefore, these are the first data demonstrating a higher incidence in black children based on a registry of children 0–14 years of age. The rise of type 1 diabetes in black children in Philadelphia was striking in both the 5- to 9-year and 10- to 14-year age-groups. Is this a true rise in type 1 diabetes or misclassification of type 2 diabetes? Type 2 diabetes in children is increasing and is more prevalent in black children (17).

There are methodological issues that could confound the data reporting the increase of type 1 diabetes in black children. WHO diabetes registry criteria requires “type of diabetes” be based on discharge diagnosis. It is possible that some of the cases are misclassified, since children who require insulin on discharge may have been identified as having type 1 diabetes in error. Some suggest that data related to diabetes antibodies and BMI should be collected to verify the type of diabetes. However, we and others have demonstrated that the characterization of diabetes in children is complex (18) and, particularly in black children, may have

features of both type 1 and type 2 diabetes and that evidence of obesity does not confirm the diagnosis (19,20).

Potential under-representation of minority populations by census data can falsely elevate incidence rates (3). This is an unlikely explanation for this epidemic. Census data from 1990 were used for the 1990–1994 cohort and the present cohort. Type 1 diabetes in Philadelphia has increased dramatically over that 10-year period, yet intercensal estimates indicate population changes have been similar for all races. Only an enormous undercount would account for the rise in incidence in black children.

Although some of our cases in Philadelphia may have been misclassified, it is likely that this cohort represents a true rise in the incidence of type 1 diabetes in black children because of the age and sex distribution. The incidence of type 1 diabetes in black children 5–9 years of age is the highest incidence ever reported in black children in this age-group. Type 2 diabetes is not prevalent in children 5–9 years of age, and the high incidence most likely represents actual cases of type 1 diabetes in black children. The sex breakdown in the Philadelphia registry also supports the diagnosis of type 1 diabetes.

The risk of type 1 diabetes in males was 1.5 times greater than in females. Allegheny County also demonstrated a higher incidence in males (4). The previous Philadelphia cohorts showed the incidence in males to be similar to the incidence in females, unlike type 2 diabetes, which is more prevalent in females (17).

The rise of type 1 diabetes in black children is a tremendous public health problem. Racial disparities exist in the treatment and outcomes of children with type 1 diabetes. It has been shown that black children with type 1 diabetes have poorer metabolic control (21–24). A ninefold increased risk of death was demonstrated for young African Americans with diabetes in Chicago, compared with non-Hispanic white patients with type 1 diabetes over an 8-year period (25). After 20 years of diabetes, the mortality rate was significantly higher in blacks than whites in Allegheny County (26).

With the rising incidence of type 1 diabetes in the black population, it is crucial that we develop culturally relevant interventions to minimize racial disparities in treatment and outcomes. We are currently expanding our research to identify goals and priorities of our patients/

families with diabetes with the purpose of informing clinicians of some of the differences between white and black families to get a better sense of how to most effectively serve these families (24).

The epidemiology of type 1 diabetes is known for ~1% of the world's children. Greater efforts in diabetes epidemiology, directed by ongoing population-based registries, are needed to clarify the causes of epidemics and temporal trends in populations across the world. These data underscore the need to establish type 1 diabetes as a reportable disease, so that genetic and environmental risk factors may be thoroughly investigated and defined.

Acknowledgments— This study was funded, in part, by a grant from the Pediatric Endocrinology Nursing Society.

The authors gratefully acknowledge the support of the nurses from the School District of Philadelphia and Drs. Stephen Dahlem, Iraj Rezvani, Alan Schindler, and Judy Ross. Data collection by Jesse Fuchs-Simon and technical assistance by Máire Abraham are appreciated.

References

1. Karvonen M, Viik-Kajander M, Moltchanova E, Libman I, LaPorte R, Tuomilehto J, the Diabetes Mondiale (DiaMond) Project Group: Incidence of childhood type 1 diabetes worldwide. *Diabetes Care* 23:1516–1526, 2000
2. Lipman TH: The epidemiology of type 1 diabetes in children 0–14 Yr of age in Philadelphia. *Diabetes Care* 16:922–925, 1993
3. Lipton RB, Fivecoate JA: High risk of IDDM in African-American and Hispanic children in Chicago, 1985–1990. *Diabetes Care* 18:476–482, 1995
4. Libman IM, LaPorte RE, Becker D, Dorman JS, Drash AL, Kuller L: Was there an epidemic of diabetes in nonwhite adolescents in Allegheny County, Pennsylvania? *Diabetes Care* 21:1278–1281, 1998
5. US Bureau of the Census: 1990 *Census of Population General Population Characteristics*. Summary file tape 3. Washington, DC, 1992
6. Lipman TH, Chang Y, Murphy KM: The epidemiology of type 1 diabetes in children in Philadelphia 1990–1994: evidence of an epidemic. *Diabetes Care* 25:1969–1975, 2002
7. Frazer de Llado T, Hawk B, Gonzalez de Pijem L, the Puerto Rican IDDM Coalition: Incidence of IDDM in children living in Puerto Rico. *Diabetes Care* 21:744–746, 1998
8. Feltbower RG, McKineey PA, Parslow RC, Stephenson CR, Bodansky HJ: Type 1 diabetes in Yorkshire UK: time trends in 0–14 and 15–29-year-olds, age at onset and age-period-cohort modeling. *Diabet Med* 20:437–441, 2003
9. Green A, Patterson CC, EURODIAB TIGER Study Group: Europe and diabetes: trends in the incidence of childhood-onset diabetes in Europe 1989–1998. *Diabetologia* 44 (Suppl. 3):B3–B8, 2001
10. Dziatkowiak H, Ciechanowska M, Wasikowa R, Symonides-lawecka A, Bieniasz J, Trippenbach-Dulska H, Korniszewski L, Szybinski Z: Increase in the incidence of type 1 diabetes mellitus in children in three cities in Poland, 1987–1999. *J Pediatr Endocrinol Metab* 15:1153–1160, 2002
11. World Health Organization DIAMOND Project Group: WHO multinational project for childhood diabetes. *Diabetes Care* 13:1062–1068, 1990
12. Bruno G, Biggeri A, LaPorte R, McCarty D, Merletti F, Pagano G: Application of capture-recapture to count diabetes. *Diabetes Care* 17:548–556, 1994
13. Siemiatycki J, Colle E, Campbell S, Dewar R, Aubert D, Belmonte MM: Incidence of IDDM in Montreal by ethnic group and by social class and comparisons with ethnic groups living elsewhere. *Diabetes* 37:1096–1102, 1988
14. McCarty BJ, Lipton R, Nichol L: HLA-DQA1 and -DQB1 alleles in Latino and African American Children with diabetes mellitus. *J Pediatr Endocrinol* 17:297–306, 2004
15. Lorenzi M, Lagliero E, Schmidt NJ: Racial differences in incidence of juvenile-onset type 1 diabetes: epidemiologic studies in southern California. *Diabetologia* 28:734–738, 1985
16. Wagenknecht LE, Roseman JM, Herman WH: Increased incidence of insulin-dependent diabetes mellitus following an epidemic of Coxsackievirus B5. *Am J Epidemiol* 133:1024–1031, 1991
17. Pinhas-Hamiel O, Dolan LM, Daniels SR, Standiford D, Khory PR, Zeitler P: Increased incidence of non-insulin-dependent diabetes mellitus among adolescents. *J Pediatr* 128:608–615, 1996
18. Katz LEL, Jawad AF, Ganesh J, Lipman TH, Hunter J, Weinzimer SA, Murphy KM: Parameters distinguishing childhood type 1 (T1DM) and type 2 (T2DM) diabetes at diagnosis (Abstract). *Diabetes* 50 (Suppl. 2):A55, 2001
19. Libman IM, Becker DJ: Coexistence of type 1 and type 2 diabetes mellitus: double diabetes? *Pediatr Diabetes* 4:110–113, 2003
20. Rapaport R, Wallach E, Greig F: Diabetes mellitus: type 1 or type 2? *J Pediatr* 138:612, 2001
21. Auslander WF, Thompson S, Dreitzer D, White NH, and Santiago JV: Disparity in glycemic control and adherence between African-American and Caucasian youths with diabetes. *Diabetes Care* 20:1569–1575, 1997
22. Chalew SA, Gomez R, Butler A, Hempe J, Compton T, Mercante D, Rao J, Vargas A: Predictors of glycemic control in children with type 1 diabetes: the importance of race. *J Diabetes Complications* 14:71–77, 2000
23. Delamater AM, Shaw KH, Applegate EB, Pratt IA, Eidson M, Lancelotta GX, Gonzalez-Mendoza L, Richton S: Risk for metabolic control problems in minority youth with diabetes. *Diabetes Care* 22:700–705, 1999
24. Ginsburg KR, Howe CJ, Jawad AF, Buzby M, Ayala JM, Tuttle A, Murphy K: Parents' perceptions of factors that affect successful diabetes management for their children. *Pediatrics* 116:1095–1104, 2005
25. Lipton R, Good G, Mikhailov T, Freels S, Donoghue E: Ethnic differences in mortality from insulin-dependent diabetes mellitus among people less than 25 years of age (Letter). *Pediatrics* 103:952, 1999
26. Nishimura R, LaPorte RE, Dorman JS, Tajima N, Becker D, Orchard TJ: Mortality trends in type 1 diabetes: the Allegheny county (Pennsylvania) registry 1965–1999. *Diabetes Care* 24:823–827, 2001