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Neighborhood context and incidence of type 1 diabetes: The SEARCH for Diabetes in Youth Study

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Abstract

Findings regarding type 1 diabetes mellitus (T1DM) and neighborhood-level characteristics are mixed, with few US studies examining the influence of race/ethnicity. We conducted an ecologic study using SEARCH for Diabetes in Youth Study data to explore the association of neighborhood characteristics and T1DM incidence. 2002–2003 incident cases among youth at four SEARCH centers were included. Residential addresses were geocoded to US Census Tract. Standardized incidence ratios tended to increase with increasing education and median household income. Results from Poisson regression mixed models were similar and stable across race/ethnic groups and population density. Our study suggests a relationship of T1DM incidence with neighborhood-level socioeconomic status, independent of individual-level race/ethnic differences.

Keywords

Type 1 Diabetes; Neighborhood; Socioeconomic status

1. Introduction

Type 1 Diabetes Mellitus (T1DM) is one of the leading chronic diseases in childhood, and worldwide rates are increasing (Onkamo et al., 1999). According to data from the SEARCH for Diabetes in Youth study, the largest surveillance effort of diabetes among youth in the United States (US), approximately 15,000 cases are newly diagnosed each year (Writing Group for the SEARCH for Diabetes in Youth Study Group, 2007). In addition, Colorado data suggest that between 1978 and 2004, the incidence of T1DM has increased by 2.7 percent annually in non-Hispanic whites and 1.6 percent in Hispanic youth (Vehik et al., 2007). Findings from EURODIAB, which includes data from 20 population based registries

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in 17 countries, indicate an annual overall increase of 3.9% in Europe and project a doubling of cases among young children from 2005 to 2020 (Patterson et al., 2009).

Despite a large and growing body of research, the understanding of T1DM etiology is still unfolding. T1DM clusters in families, with offspring of parents with T1DM showing increased incidence (The TEDDY Study Group, 2007). Certain genotypes, particularly the human leukocyte antigen, have also been associated with increased risk of developing T1DM (Risch, 1989). However, family history and genetics only explain a portion of cases. Based on migrant studies, twin studies, and biological plausibility, Knip and colleagues suggest that environmental factors may be involved in triggering T1DM (Knip et al., 2005). Additional evidence comes from studies that have shown geographic variation of T1DM, including spatial clustering (McNally et al., 2006; Rytkonen et al., 2001; Samuelsson and Lofman, 2004).

The hygiene hypothesis is one of several potential causal explanations for T1DM development. It posits that the immune system needs stimulation by the environment early in life in order for it to fully develop and mature. A reduction of exposure to infections and other antigenic exposures in this critical phase may result in an inappropriate immune response and increased autoimmune disorders (Gale, 2002; Kolb and Elliott, 1994). Environments that are sparsely populated or subject to excessive hygiene would likely result in lack of exposure to infections, and would therefore be thought to be associated with increased risk of T1DM. In fact, a study in Poland reported an increase in T1DM incidence corresponding to social, environmental and economic improvements (Jarosz-Chobot et al., 2008). Conversely, increased population density may lead to higher population mixing, crowded living situations, and generally poor socioeconomic settings, which in turn may lead to more antigenic exposures. Following the hygiene hypothesis, high population density, crowding and low socioeconomic status should be associated with lower risk of T1DM. To date, studies of population density and T1DM incidence have been inconsistent, with some studies reporting increased incidence with urbanicity or higher population density, and others finding opposing or no effects (du Prel et al., 2007; Feltbower et al., 2005; Gopinath et al., 2008; Thomas et al., 2008). Similarly, there is some lack of agreement with respect to the impact of neighborhood socioeconomic context (Cardwell et al., 2007; Patterson and Waugh, 1992) However, a variety of markers of lower individual socioeconomic status, such as having Medicaid insurance, an unmarried mother or inadequate prenatal care, were associated with a lower risk of T1DM (D'Angeli et al., 2010).

In addition, income level, education and unemployment may play a role in dietary intakes (e.g. introduction to cow's milk or wheat) which have been associated, though somewhat inconsistently with T1DM incidence (Norris, 2010; Norris et al., 2003, Norris and Scott, 1996, The TEDDY Study Group, 2007). Given that family history and environmental factors separately contribute to increased risk of T1DM, but do not explain a substantial portion of cases, gene-environment interactions appear to be a promising area for etiologic research. However, questions remain regarding which environmental factors may be involved in these interactions and the direction of their influence.

It is important to recognize that much of the research regarding the environmental context has been conducted in Europe where several registries have documented T1DM incidence over time (Cardwell et al., 2007; du Prel et al., 2007; Parslow et al., 2001; Rytkonen et al., 2001). In the US, the additional influence of race/ethnicity on T1DM incidence has received limited attention. The few studies available have been limited with respect to race/ethnic groups and/or geographic diversity including a Chicago study of African-American and Latino children (Lipton et al., 1999) and a Colorado study (Hamman et al., 1990; Lipton et

al., 1999). To enhance our understanding of neighborhood contextual characteristics, we conducted an ecologic study using data from the SEARCH for Diabetes in Youth study to explore whether neighborhood contextual factors are associated with T1DM incidence among populations in the US. Based on previous research, we hypothesized that lower population density and various indicators of more affluent neighborhood socioeconomic status would be associated with T1DM incidence and that population density may modify relationships of T1DM incidence with socioeconomic status. We also explored the contribution of neighborhood level and individual level race/ethnicity.

2. Study design

Details of the SEARCH study design (SEARCH Study Group, 2004) and the ancillary spatial epidemiology study (Liese et al., 2010) have been published elsewhere. Briefly, SEARCH is an observational study ascertaining non-gestational cases of diabetes among youth under age 20. Prevalent case data collection began in 2001, and incident case data collection began in 2002, continuing through the present. Cases are ascertained at six centers, of which, two serve health plan memberships and four serve geographical areas. Eligible cases are identified through networks of pediatric and adult endocrinologists, existing pediatric diabetes databases, hospitals, the databases of health plans, and other health care providers. Physician reports and medical record reviews are primarily used for case validation, with the few remaining cases validated by self-reports of a physician's diagnosis. The Coordinating Center at Wake Forest University in North Carolina uses Health Insurance Portability and Accountability Act (HIPAA) compliant procedures to register all validated cases. Each center's institutional review board approved the study protocol which complies with the privacy rules of the HIPAA.

3. Study population

For the current study, we included all cases of type 1 diabetes mellitus (T1DM) occurring in 2002 and 2003 at four of the SEARCH centers: (1) the state of Colorado, (2) eight counties around Cincinnati, Ohio including Butler, Clermont, Hamilton, Warren counties in Ohio, Boone, Campbell, and Kenton counties in Kentucky, and Dearborn, Indiana (hereafter referred to as the Ohio center), (3) the state of South Carolina, and (4) five counties around Seattle, Washington, including King, Kitsap, Pierce, Snohomish, and Thurston counties (hereafter referred to as the Washington center).

4. Exposure assessment

The geocoding process has been described in detail elsewhere (Hibbert et al., 2009; Liese et al., 2010). Briefly, a single staff person traveled to each of the four centers in order to geocode each residential address. Software and data used for geocoding included ArcGIS 9.3 software (ESRI, Redlands, CA), Topographically Integrated Geographic Encoding and Referencing (TIGER) 2000 Road Network data (US Census, 2008), and Zip Code Tabulation Areas (ZCTA) data (US Census Bureau, 2009). TIGER 2006 Road Network Files were used to supplement 2000 files in South Carolina due to recent land development. Residential addresses were successfully matched to Census tracts for 79% (1,617) of T1DM cases overall, with specific percentages for each center as follows: Colorado: 86.4% (867), Ohio: 91.3% (252), South Carolina: 73.6% (337), and Washington: 60.9% (259) (60.9%). The 15% (300) of addresses that were geocodable to zip code level only were allocated to Census tract by a random assignment based imputation method and process which was weighted by demographic information (Henry & Boscoe, 2008; Hibbert et al., 2009).

5. Summary

File 1 (SF1) and Summary File 3 (SF3) data from the Census Bureau (US Census, 2000) were used to obtain socioeconomic and demographic variables for each Census tract including: total population, tract area, population by race/ethnicity, population with high school and above education, population living below poverty, median household income, and median value of housing. Population density was determined as population per square mile. The predominant racial group of each Census tract was determined as the racial group composing 60% or more of the total population (Moore et al., 2006). Census tracts with 20 percent or more of the population living below the federally defined poverty level are hereafter identified as “poverty area”(Krieger et al., 2003). For the purposes of this analysis, socioeconomic status and population density variables were categorized.

6. Statistical analyses

To calculate the standardized incidence ratios (SIRs), incident cases observed in each Census tract center were summed for 2002 and 2003. The annual expected number of cases was determined using US Census population estimates for 2000 (multiplied by two years to reflect the time period used in this study) and corresponding SEARCH published pooled (age group, gender, and race-specific) annual incidence rates (Dabelea et al., 2007). The expected counts for each Census tract were calculated by summing stratum-specific expected counts. The total observed cases were then divided by the total expected cases for each Census tract to estimate the SIR. In addition, SIRs specific to each of the following characteristics were calculated: gender, age (four five-year age groups: 0 to 4, 5 to 9, 10 to 14 and 15 to 19) and race (six groups: Hispanic, Non-Hispanic white, African American, Asian/Pacific Islander, and American Indian/Native American, Multiple and other). SIRs were also estimated for each level of the socioeconomic variables and for the predominant race/ethnicity of each Census tract (African-American, Hispanic, Mixed or White). These and all other statistical analyses were performed using SAS 9.2 (Cary, NC).

Generalized linear mixed models with a Poisson distributed error and a random intercept term for the tract were used to predict the number of observed cases for each tract. The log of the expected counts (calculated from gender, age and race specific incidence rates) was used as an offset variable and results from these models were used to estimate relative risks. Models were fitted with the GLIMMIX procedure in SAS 9.2. The means and standard deviations for each of the socioeconomic variables, race/ethnicity, and population density were estimated to show the distribution of each of these variables in the pooled data as well as for each of the four centers. Correlations among each of the socioeconomic variables were examined. Poisson models with pooled data and an interaction term for each socioeconomic status and population density covariate by center were used to assess the possibility of analyzing pooled data. If center interactions were not significant, Poisson mixed models were used to analyze pooled data examining the association between T1DM incidence and each of the socioeconomic status variables and population density in separate models. In an exploratory analysis, we also examined the relationship of T1DM incidence with neighborhood level race/ethnicity and whether stratification by individual level race/ethnicity influenced univariate associations of T1DM with socioeconomic status and population density. Finally, we explored whether stratification by population density modified any relationships between T1DM incidence and neighborhood socioeconomic status factors.

7. Results

A total of 1502 T1DM cases developed in the two-year period (2002–2003) among youth aged 0–19 years across the four SEARCH centers with 516 in Colorado, 269 in Ohio, 362 in South Carolina, and 355 in Washington. 3132 total Census tracts were included in the study (Table 1). The South Carolina center had the greatest percentage of participants living in tracts that are more than 60% African American (15.6%) or of mixed race/ethnicity (23.3%), while Colorado had the greatest percentage of residents in predominantly Hispanic Census tracts (4.1%). Washington had the highest population density, while South Carolina exhibited the lowest population density. South Carolina also had the worst neighborhood socioeconomic status indicators. Compared to the other SEARCH centers, it had the highest percent poverty areas, least percent high school-educated, and the lowest median household income and median housing value. Washington tended to have the best socioeconomic status indicators, with the least poverty, most high school graduates, and highest median housing value and household income. Socioeconomic status variables were highly correlated; therefore we did not include multiple socioeconomic status variables within the same predictive model.

Pooled SIRs for the association of neighborhood contextual factors with T1DM incidence are shown in Table 2. No significant difference in T1DM incidence was observed across any of the population density levels examined, however significant differences were associated with certain levels of socioeconomic status indicators. SIRs increased with increasing education, median housing value and median household income. Lower SIRs were evident in tracts that were below the federally defined poverty level. Living in a Census tract designated as predominantly racially mixed was protective, and site-specific results showed predominantly white tracts with a significantly elevated SIR in Ohio and a significantly decreased SIR in South Carolina (data not shown).

Findings from the Poisson mixed models are also shown in Table 2. Results from these models, adjusting for center, gender, age and race/ethnicity, are highly consistent with the SIR results. Given that interactions by center were not significant for any of the socioeconomic status or population density factors examined, pooled data are presented. Living in a Census tract of medium population density, or a suburban type area, was associated with significantly increased risk of incident T1DM. As Census tract level of education, median household income, and median housing value increased, risk of T1DM incidence also increased, with the lowest categories of neighborhood socioeconomic status showing significantly decreased incidence. Rates were lower, though not a statistically significant difference, in tracts where 20% of residents or more were living below the federally defined poverty level.

In models stratified by individual level race/ethnicity and adjusted for center, age, and gender; consistently higher risks of T1DM incidence were found in tracts with higher median income, higher housing value and no poverty designation (Table 2). These results did not differ among race/ethnicities. The association of incident T1DM with percent of high school educated adults differed somewhat by race/ethnicity, with significantly lower risks among African-Americans and Hispanics living in areas with the least educational level, and among Whites at the middle educational level. Both Whites and African-Americans living in suburban areas (e.g. with 500 to less than 1000 people per square mile) showed greatest risks of incident T1DM; whereas the highest risk, though not significantly increased, for Hispanic youth was in areas with lowest population density.

In order to explore whether the relationship of T1DM and socioeconomic status indicators was influenced by population density, Table 3 shows results of Poisson mixed models with

pooled data stratified by population density and adjusting for center, age and race/ethnicity. In areas with the least and greatest population densities, risk of T1DM incidence increased with increasing income. In both types of areas, the lowest level of household income was significantly associated with lower risk of incident T1DM. A similar relationship was found with the least level of high school education in the most populated areas. No other strong differences were evident across levels of population density. Overall, T1DM incidence appeared to increase with increasing affluence regardless of population density.

8. Discussion

In this ecologic study of T1DM among diverse youth in Colorado; eight counties around Cincinnati, Ohio; South Carolina; and five counties around Seattle, Washington, we found overall that risk of T1DM incidence tended to increase for children living in increasingly affluent Census tracts, regardless of population density and individual-level race/ethnicity. This relationship was strongest with median household income and median housing value in the Census tract of residence. Though living in a tract with a lower percent of high school graduates or in a tract with at least 20% of residents living below the federally defined poverty level showed some evidence of decreased T1DM incidence, strong differences in T1DM incidence were not apparent across individual-level races/ethnicities or with varying levels of population density. Analyses adjusting for center showed the middle or suburban category of population density had the greatest risk of T1DM.

Findings from our study agree with many previous studies of the relationship between T1DM incidence and neighborhood-level factors conducted in Europe and Australia that have reported increasing incidence with affluence (Cardwell et al., 2007; Feltbower et al., 2005; Gopinath et al., 2008; Haynes et al., 2006; Holmqvist et al., 2008; Patterson and Waugh, 1992). A study among children in the Northern Ireland T1DM register showed lower T1DM incidence associated with income deprivation, as measured by the proportion of individuals in families receiving unemployment or working but receiving additional financial support (Cardwell et al., 2007). A similar pattern of increasing incidence rate ratios with increasing average family income and education level was observed in southeastern Sweden (Holmqvist et al. 2008). No clear trend in incidence was observed across categories of per capita average income in Stockholm County, Sweden, however the smallest SIR was observed in the lowest income category (Gopinath et al., 2008). In contrast to these findings, a study of cases registered in North Rhineland-Westphalia, Germany, found increased household income associated with decreased risk (RR: 0.89; 95%CI: 0.84,0.94), however this study also showed an inverse relationship with unemployment rate and percentage of welfare recipients (du Prel et al., 2007). In addition, several studies have combined various socioeconomic status variables into indices: the Index of Relative Socioeconomic Disadvantage in Australia combines income, Indigenous descent and unemployment or employment in unskilled occupations (Haynes et al., 2006); the Townsend score in the United Kingdom combines unemployment, overcrowding, car ownership and housing tenure (Feltbower et al., 2005); and finally the Carstairs Index in Scotland combines unemployment, employment in manual occupations, overcrowding and car ownership (Patterson and Waugh 1992). These studies have found increased T1DM with increasing area-level affluence as measured by their respective SES index. The variety of socioeconomic status indicators used suggests that socioeconomic status may not translate similarly across countries or different indicators may have unique relationships with T1DM (e.g. income versus unemployment) (du Prel et al., 2007). In addition, further research is needed regarding differences between individual-level and area-level socioeconomic status influences on T1DM, as important heterogeneity between these two levels has been found in other studies (Diez-Roux et al., 2001).

Though our study did not find strong differences across differing levels of population density, this factor figures prominently in the environmental factors influencing incidence among studies in Europe, Australia and the US. Several studies have found increasing incidence in areas with less dense population (Cardwell et al., 2007; Gopinath et al., 2008; Patterson and Waugh, 1992). For example, children living in less population dense areas of Northern Ireland (Cardwell et al., 2007), North Rhineland Westphalia (du Prel et al., 2007) and Stockholm County, Sweden (Gopinath et al., 2008) showed higher T1DM incidence. However, a study of Italian children (Cherubini et al., 1999) and a study of children in Western Australia (Haynes et al., 2006) found the highest incidence among more populous urban areas. Findings from a study of children in southeastern Sweden (Holmqvist et al., 2008) were more similar to our study, where areas of medium population density fared worse. According to the United Nations, Australia and New Zealand have the lowest population density (2.9 persons per square kilometer), followed by North America (16.9), while northern Europe (57.2) and western Europe are more densely populated (168.5) (United Nations Department of Economic and Social Affairs Population Division (2004)). Differences in background population density by country may partially explain inconsistencies in the relationship of population density and T1DM, as a study in sparsely populated Australia (Haynes et al., 2006) found an association in contrast to those reported by some studies in more densely populated Europe (Cardwell et al., 2007, Gopinath et al., 2008).

Some studies, such as ours, have also examined simultaneously the effects of socioeconomic status and population density. Similar to our results, studies in Germany and Scotland found that the association between incidence and income was stable when adjusting for population density/urbanicity (du Prel et al., 2007; Patterson and Waugh, 1992). Among children in Canterbury, New Zealand, Miller and colleagues found the highest T1DM incidence was in areas designated as satellite urban communities, rather than urban or rural centers, while adjusting for such factors as deprivation and population density (Miller et al., 2010). Lastly, an Australian study reported independent effects of urbanicity/population density and affluence (Haynes et al., 2006).

An important contribution of this study is the evaluation of environmental contextual factors with respect to race/ethnicity. In the US, racial/ethnic health disparities as well as socioeconomic health disparities are widespread and a significant public health problem. Differences in T1DM incidence rates among race/ethnic groups in the US have been documented (Mayer-Davis et al., 2009), but extremely few studies have examined environmental context in conjunction with race/ethnicity. A study of mostly non-Hispanic White and Hispanic children in Colorado, USA (Hamman et al., 1990) reported no rural–urban gradient in incidence rates and no difference in the season of T1DM onset due to ethnicity. The smaller number of Hispanic cases in this study, particularly with respect to those living in urban areas, may have precluded further analyses of environmental factors. However, this research group showed in another study that T1DM incidence among non-Hispanic Whites in Colorado is about twice that of Hispanics (Gay et al., 1989). Another study of differences between African-American and Latino children in Chicago found increased risk with increasing socioeconomic status among African-Americans but not among Latinos (Lipton et al., 1999). Though we found more statistically significant associations with socioeconomic indicators among Whites, the trends were similar for African-Americans and Hispanics. Our power to detect an effect may have been limited by smaller numbers of Hispanic children.

Though our study provides a unique and valuable contribution to the literature with four different geographic areas in the US, this study is ecologic and includes the limitations inherent to this type of study design. We conducted analyses with individual level race/

ethnicity, and many of our environmental factors of interest bear their influence at the area level rather than or in addition to the individual level. Further study is necessary to distinguish which level is more influential for T1DM incidence. Though our sample size was considerable, some categories of certain variables were smaller in size and many variables were correlated; thus limiting our ability to examine many variables in combination. Other modeling techniques (i.e. structural equation modeling) could be used to examine these more complex questions regarding pathways.

In summary, this study of a diverse racial/ethnic US population examined the contribution of a number of environmental contextual variables and found that socioeconomic status, regardless of race/ethnicity and population density, plays an important role in T1DM incidence. Findings from the current study lend support to the hygiene hypothesis and socioeconomic status-related lifestyle differences (i.e. dietary intakes) as potential etiologic influences for T1DM. Finally, potential interactions between these environmental factors and genetic predisposition (e.g. high risk HLA genotype) with T1DM incidence should be explored.

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Table 1

Pooled and center-specific descriptive tract statistics for each neighborhood characteristics.

Characteristic	All Mean (SD)	CO Mean (SD)	OH Mean (SD)	SC Mean (SD)	WA Mean (SD)
Tracts (N)	3132	1056	460	867	749
Race					
Predominantly African-American %	5.9	0.4	10.0	15.6	0
Predominantly Hispanic %	1.4	4.1	0	0	0
Predominantly mixed %	15.2	14.1	8.0	23.3	11.7
Predominantly white %	77.5	81.5	82.0	61.1	88.2
Population density (1000 s)	2.8(3.3)	3.3(3.2)	3.4(3.2)	1.1(1.6)	3.6(4.2)
Percent live in poverty	11.6(10.3)	9.7(8.6)	12.3(13.9)	15.8(10.6)	8.8(7.3)
Percent high school education or above	82.9(12.5)	86.3(11.9)	80.1(12.3)	74.9(12.4)	89.1(7.2)
Median household income ($1000+)	46.3(19.5)	49.9(21.6)	44.6(19.0)	36.7(14.2)	53.6(17.5)
Median housing value ($10,000 s)	14.7(9.2)	17.0(9.2)	11.3(5.6)	9.0(6.3)	20.2(0.2)

Table 2

Standardized incidence ratios and univariate model relative risks for the association of T1DM incidence with neighborhood characteristics, pooled and stratified by individual level race/ethnicity [a].

Model	Number of census tracts	All Standardized incidence ratio (95% CI)	All Relative risk (95% CI)	African-American Relative risk (95% CI)	Hispanic Relative risk (95% CI)	White Relative risk (95% CI)
Population density						
< 500	937	0.93 (0.85, 1.02)	1.04(0.93,1.18)	1.26(0.81,1.95)	1.30(0.84,2.01)	0.99(0.87,1.13)
500 to < 1000	299	1.15 (0.99, 1.33)	1.26(1.07,1.48)[b]	1.44(0.79,2.63)	0.87(0.37,2.00)	1.26(1.06,1.50)[b]
≥ 1000	1896	0.98 (0.91, 1.04)	1.00	1.00	1.00	1.00
Median household income						
< $35,000	951	0.80 (0.71, 0.90)[b]	0.77(0.66,0.88)[b]	0.70(0.43,1.15)	0.51(0.31,0.81)[b]	0.81(0.68,0.96)[b]
$35,000 to < $50,000	1039	0.95 (0.86, 1.04)	0.89(0.79,1.00)	0.88(0.54,1.46)	0.80(0.52,1.22)	0.87(0.76,0.99)[b]
> $50,000	1142	1.10 (1.02, 1.18)[b]	1.00	1.00	1.00	1.00
Median housing value						
< $50,000	160	0.61 (0.42, 0.86)[b]	0.60(0.42,0.86)[b]	0.49(0.27,0.90)[b]	0.58(0.31,1.10)	0.56(0.35,0.91)[b]
$50,000 to < $100,000	930	0.87 (0.78, 0.96)[b]	0.84(0.72,0.97)[b]	0.57(0.39,0.84)[b]	0.77(0.46,1.29)	0.86(0.75,0.99)[b]
$100,000 to < $125,000	373	0.94 (0.81, 1.09)	0.86(0.72,1.01)	0.64(0.34,1.19)	0.92(0.57,1.49)	0.88(0.74,1.05)
> $125,000	1669	1.07 (1.00, 1.14)	1.00	1.00	1.00	1.00
Education						
< 70% High School or Above	546	0.81 (0.69, 0.93)[b]	0.82(0.69,0.97)[b]	0.63(0.42,0.93)[b]	0.59(0.35,0.98)[b]	0.98(0.79,1.21)
70–80% High School or Above	533	0.86 (0.75, 0.98)[b]	0.86(0.73,1.00)	0.67(0.44,1.03)	1.35(0.88,2.07)	0.81(0.68,0.97)[b]
≥ 80% High School or Above	2053	1.04 (0.98, 1.10)	1.00	1.00	1.00	1.00
Poverty areas [c]						
No	2657	0.99 (0.94, 1.05)	1.14(0.95,1.36)	1.18(0.83,1.68)	1.19(0.74,1.91)	1.06(0.82,1.38)
Yes	475	0.83 (0.70, 0.99) [b]	1.00	1.00	1.00	1.00

[a] All models adjusted by center, age, sex, race/ethnicity.

[b] P value < 0.05.

[c] Poverty Areas–Areas with ≥ 20% of the population living below federally defined poverty line.

Table 3

Relative risks and 95% confidence intervals for the association of T1DM incidence with neighborhood characteristics stratified by population density [a].

	Population density		
	< 500	**500 to < 1000**	**≥ 1000**
Median household income			
< $35,000	0.69 (0.52,0.91) [b]	0.94 (0.60,1.47)	0.78 (0.65,0.94) [b]
$35,000 to < 50,000	0.82(0.65,1.03)	1.16 (0.81,1.65)	0.89 (0.76,1.03)
> $50,000	1.00	1.00	1.00
Median housing value			
< $50,000	0.53 (0.32,0.90) [b]	0.87 (0.21,3.56)	0.65 (0.37,1.13)
$50,000 to < 100,000	0.75 (0.57,1.00)	0.84 (0.55,1.27)	0.88 (0.72,1.08)
$100,000 to < 125,000	0.90 (0.65,1.26)	0.96 (0.60,1.53)	0.82 (0.66,1.01)
> $125,000	1.00	1.00	1.00
Education			
< 70% High school or above	0.84 (0.62,1.12)	1.21 (0.72,2.05)	0.75 (0.58,0.96) [b]
70 – < 80% High school or above	0.84 (0.64,1.09)	0.83 (0.50,1.38)	0.87 (0.70,1.08)
≥ 80% High school or above	1.00	1.00	1.00
Poverty areas			
No	0.89 (0.66,1.21)	1.02 (0.45,2.32)	1.26 (0.99,1.60)
Yes	1.00	1.00	1.00

[a] All models adjusted by center, age, gender, race/ethnicity.

[b] P value < 0.05.