Health & Place 28 (2014) 194–204

Contents lists available at ScienceDirect

Health & Place

journal homepage: www.elsevier.com/locate/healthplace

Spatial and temporal variation in type 1 diabetes incidence in Western Australia from 1991 to 2010: Increased risk at higher latitudes and over time

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ARTICLE INFO

Article history:
Received 9 October 2013
Received in revised form
14 February 2014
Accepted 14 May 2014

Keywords:
type 1 diabetes mellitus
Latitude
Remoteness
Socioeconomic status
Population density

ABSTRACT

This study analysed spatial and temporal variation in childhood incidence of type 1 diabetes mellitus (T1DM) among Western Australia's 36 Health Districts from 1991 to 2010. There was a strong latitudinal gradient of 3.5% (95% CI, 0.2–7.2) increased risk of T1DM per degree south of the Equator, as averaged across the range 15–35° south. This pattern is consistent with the hypothesis of vitamin D deficiency at higher latitudes. In addition there was a 2.4% (95% CI, 1.3–3.6) average increase in T1DM incidence per year. These effects could not be explained by population density, socioeconomic status, remoteness or ethnicity.

1. Introduction

The incidence of type 1 diabetes mellitus (T1DM) among children is a growing health problem worldwide. While there have been significant gains in the management and prognosis of T1DM cases (Nathan et al., 2009), there has been a sustained increase of 2–5% per year in the incidence of T1DM throughout the world (Diamond Project Group, 2006; Onkamo et al., 1999). In addition, geographic variation in T1DM is among the largest observed for any non-communicable disease (Liese et al., 2010).

The aetiology of T1DM is complex and not yet well-understood (Atkinson and Eisenbarth, 2001). The established view is that multiple genetic and environmental factors are required to initiate the onset of T1DM (Akerblom and Knip, 1998; Atkinson and Eisenbarth, 2001; Gan et al., 2012), with environmental factors thought to include diet, vitamin D deficiency and exposure to infections.

Epidemiological studies have helped to identify potential environmental risk factors of T1DM (Akerblom et al., 2002). For example, spatiotemporal clusters of T1DM incidence might be explained by episodic infectious agents triggering disease onset (McNally et al., 2009; Samuelsson and Carstensen, 2003). Patterns of increased risk of T1DM at higher latitude have been interpreted in terms of reduced exposure to ultraviolet radiation leading to vitamin D deficiency (Mohr et al., 2008; Staples et al., 2003). Higher rates of T1DM in sparsely-populated areas could be explained by fewer opportunities for early childhood exposure to communicable infections which would otherwise promote the development of a more regulated immune system (Cardwell et al., 2006; du Prel et al., 2007; Patterson et al., 1996; Waldhoer et al., 2008). Geographic associations between socioeconomic status and T1DM incidence may be attributable to spatial patterns in population composition, leading to differences in lifestyle, maternal age, diet and exposure to infections (du Prel et al., 2007; Patterson et al., 2001; Patterson and Waugh, 1992).

Efforts to identify plausible and predictable mechanisms for geographic risk factors of T1DM have been challenged by inconsistent results among studies. Whereas some studies show higher rates of T1DM in sparsely-populated rural areas than in urban areas

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http://dx.doi.org/10.1016/j.healthplace.2014.05.004
1353-8292/

(Cardwell et al., 2006; Holmqvist et al., 2008; Patterson and Waugh, 1992), other studies show the reverse pattern (Cherubini et al., 1999; Haynes et al., 2006) or no difference (Schober et al., 2003; Schoenle et al., 2001). Results for socioeconomic status are similarly diverse: high risk of T1DM has been variously associated with high socioeconomic status (Haynes et al., 2006; Patterson and Waugh, 1992; Siemiatycki et al., 1988; Tarn et al., 1983; Torres-Aviles et al., 2010), low socioeconomic status (Blom et al., 1989; Christau et al., 1977; Crow et al., 1991; Hungarian Childhood Diabetes Epidemiology Study Group, 1994; Waugh, 1986), or not at all (Bruno et al., 2000; Evans et al., 2000; Harron et al., 2011; Laporte et al., 1981).

Modelling multiple factors within individual studies is an important tool in resolving apparently inconsistent effects of risk factors. For example, urban-versus-rural differences in T1DM risk may be strongly correlated with socioeconomic patterns (Holmqvist et al., 2008; Patterson and Waugh, 1992). Thus, the multi-factorial nature of environmental contributors to T1DM requires a multi-factorial approach to epidemiological studies; otherwise ongoing inconsistencies can be expected from the effects of unmeasured but correlated environmental or sociodemographic factors.

Beyond hidden effects within studies, the meaning of key variables is highly context-dependent. For example the meaning of remoteness as applied to Northern Ireland (Cardwell et al., 2006) with 1.7 million people within 14,135 km^2 in 2001 (Cardwell et al., 2007) is very different to the meaning of remoteness in Western Australia (Haynes et al., 2006) with 1.8 million people within 2,529,875 km^2 in 2001 (Edwards, 2003). The effect of socioeconomic status may also be highly context-dependent given that it probably acts as a proxy for a diverse set of more directly-acting mechanisms (Patterson and Waugh, 1992). Therefore, to better understand the epidemiology of T1DM, there is a need for more multi-factorial studies across a variety of locations.

Here, we simultaneously model socioeconomic status, population density, latitude, remoteness and ethnicity as risk factors for childhood T1DM incidence using a complete register of all childhood T1DM cases in Western Australia. Previous research in Western Australia showed that a higher incidence of T1DM was associated with areas of high socioeconomic status, and urban areas rather than rural/remote areas, and that these associations were independent of each other (Haynes et al., 2006). A motivation for the current study was to examine spatial differences in T1DM in more detail, and to test additionally for the effects of latitude, population density and ethnicity. Western Australia has a highly centralised population, with three-quarters of the State's population within the Perth metropolitan area (Edwards, 2003). While the remaining population is sparse (total=508,157 outside Perth at an average of 0.2 people per km^2 in 2001 (Edwards, 2003)), its broad latitudinal distribution (15–35° south) provides a valuable opportunity for examining an effect of latitude within a single diabetes data register. Given previous evidence of a strong temporal trend in the incidence of T1DM worldwide (Onkamo et al., 1999), we structure the analysis as a spatiotemporal model of T1DM incidence, using 20 years of data (1991–2010) to allow for temporal as well as spatial differences in socioeconomic status, population density, ethnicity and where children live in relation to latitude and remoteness. Finally, to measure spatiotemporal variation in T1DM incidence that is unexplained by these covariates, we add terms for spatial random effects and a temporal trend.

2. Methods

2.1. Setting

This study analysed spatial and temporal variation in the incidence of T1DM diagnosed in children aged less than 15 years among Western Australia's 36 Health Districts, from 1991 to 2010.

2.2. Data source and data structure

Cases of T1DM were sourced from the Western Australian Children's Diabetes Database. This is a diabetes register, maintained at Princess Margaret Hospital, of all known cases of T1DM diagnosed in Western Australia since 1985, with case ascertainment estimated at more than 99% from capture-recapture methods (Kelly et al., 1994). We identified cases as those patients diagnosed with T1DM who commenced insulin therapy before 15 years of age. Patients with type 2 diabetes or diabetes secondary to other causes such as cystic fibrosis and mature-onset diabetes of the young were excluded. The data were based on children diagnosed with T1DM between January 1, 1991 and December 31, 2010.

The data were aggregated within Western Australia's 36 Health Districts (Fig. 1). We stratified the data by year of diagnosis, sex, and age group at diagnosis (0–4 years; 5–9 years; 10–14 years). The numbers of children at risk by Health District, sex and age group were obtained from the Australian Bureau of Statistics' national census data, based on five-yearly censuses: 1991, 1996, 2001, 2006 and 2011. Population sizes for the years between censuses were estimated by adjusting for annual births and deaths data (Rates Calculator software – Epidemiology Branch, Department of Health Western Australia, 2013). Denominators were based on non-Indigenous children, as T1DM is very rare among Indigenous children in Western Australia (Glatthaar et al., 1988). Throughout this paper we use the term Indigenous to refer collectively to people of Aboriginal and Torres Strait Islander descent. Only one of the 1571 cases of T1DM in the study was an Indigenous child. The identity of this record was not supplied for this study; therefore the record remained in the analysis. We assume this had a negligible impact on the results.

2.3. Covariates

We modelled socioeconomic status, population density, latitude, remoteness and ethnicity as covariates. Remoteness was assigned as urban, rural or remote as defined in 2005 by the Western Australian Department of Health (Fig. 1). We committed to a static classification of remoteness over the 20-year study period, for consistency with previous research (Haynes et al., 2006) which found differences in the incidence rate of T1DM between these same remoteness categories. The effects of any changes in remoteness that are not captured by this static classification may be partially captured by our inclusion of a variable for population density, and the inclusion of random effects (see below).

Socioeconomic status (SES) was based on the Australian Bureau of Statistics' Index of Socioeconomic Relative Disadvantage (IRSD) (Pink, 2008). This index, which is generated for each five-yearly national census, summarises a range of information relating to the economic and social conditions of people and households within areas (e.g. income, educational attainment and unemployment). The set of variables differs somewhat between censuses. In deriving the Index for each census, the Australian Bureau of Statistics use a Principal Components Analysis to assign each variable a weighting (Pink, 2008). Individual census areas are then given a score based on the sum of their variable weightings. For censuses prior to 2011 we assigned each Health District a population-weighted mean IRSD value based on component Census Collection Districts (assigned by their centroid locations). In urban areas, Census Collection Districts each contain approximately 250 households (Pink, 2008). There were 4370 Census Collection Districts in Western Australia in 2006 (Trewin, 2006). For the 2011 census we used the Australian Bureau of Statistics' new small-area category of Statistical Area 1 (n=5512 in Western