



The diffusion of autism spectrum disorder in Costa Rica: Evidence of information spread or environmental effects?



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ABSTRACT

In the U.S., children with autism spectrum disorder (ASD) have been found to live in spatial clusters. Studies have suggested that the clustering is caused by social or environmental factors, but determining the cause of the clustering is difficult in the U.S. setting because of unmeasured variation in healthcare access and diagnostic practices. The present study explores the diffusion of ASD in a small setting in which the diagnosis is not widely publicised and there is no variation in healthcare access or diagnostic practices. Costa Rica provides universal healthcare and only has one diagnosing clinic for young children, and the diagnosis is relatively new and little known among clinicians and parents. In addition, the potential for mercury exposure from the source that has been associated with ASD is absent, and areas with high levels of air pollution are spatially concentrated. Focusing on all young children who underwent an ASD assessment from 2010 to 2013, we identify spatial clusters that suggest a mechanism that does not depend on information about ASD, healthcare access, diagnostic practices, or environmental toxicants. These findings provide details of the “contextual drivers” of the increasing worldwide prevalence of ASD.

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1. Introduction

In the U.S., children with autism spectrum disorder (ASD) have been found to live in spatial clusters (Mazumdar et al., 2010; Hoffman et al., 2012). From a public health perspective, these clusters mean that children in certain neighbourhoods—often overrepresented by Latinos, African Americans, and low socioeconomic status (SES) families (Durkin et al., 2010)—are less likely than others to receive important services that can improve life outcomes (Liptak et al., 2008). Identifying the causes of the clusters will inform public health officials on how to remediate these inequities and inform us on “contextual drivers” of the increasing prevalence of ASD (Mazumdar et al., 2013).

Theories about the causes of the ASD clustering are diverse. For example, sociologists have found strong positive correlations between the diagnosis and both SES and parental education (Van Meter et al., 2010; Hoffman et al., 2012), and others have found anecdotal evidence of a social network effect on the diagnosis (Liu et al., 2010). In California, ASD prevalence in low SES neighbourhoods nearly caught up to the rate in high SES neighbourhoods, and this happened as information about ASD was becoming

widespread (King and Bearman, 2011). In other studies, biologists have identified associations between ASD clusters and congenital malformations of the reproductive system, which can be caused by exposure to toxicants during pregnancy (Rzhetsky et al., 2014). Others have linked the spatial patterns to exposure to heavy metals (Windham et al., 2006), air pollution from traffic (Becerra et al., 2013), and mercury from coal-fired power plants (Palmer et al., 2006; Palmer et al., 2009).

Determining the cause of the clustering is difficult in the U.S. setting for several reasons. Specifically, there is substantial variation in access to healthcare (Kirby and Kaneda, 2006; Wang and Luo, 2005; Williams, 2012; related to ASD specifically, see Liptak et al., 2008; Fountain and Bearman, 2011) and the distribution of diagnosing clinics (e.g., Mazumdar et al., 2013), and the clinics engage in highly variable diagnostic practices (Bresnahan et al., 2009; Charman et al., 2009); there are even insurance incentives that variously promote the diagnosis of ASD (Grinker, 2007). Thus, diagnostic variation across clinics that are unequally distributed across space and populations could in itself lead to spatial patterns in the diagnosis, and the clustering patterns could be indistinguishable from clustering related to information spread or exposure to environmental toxicants. All of these factors would likely create spurious correlations with neighbourhood and demographic characteristics (e.g., Kirby et al., 2006).

Another difficulty in the U.S. setting, particularly for hypotheses

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suggesting that information diffusion through parental social networks leads some parents and not others to seek a diagnosis for their children (e.g., Weintraub, 2011; Keyes et al., 2012), is that information about ASD is readily available from many sources (Eyal et al., 2010). For this reason, it is difficult to imagine that parents of symptomatic children require information about ASD specifically from other parents. While there is evidence that parents rely on other parents for information *after* their children are diagnosed (Mackintosh et al., 2005), it is unclear whether these parents make contact *before* the diagnosis. Other sources of information about autism, such as resources on the internet, would not directly lead to spatial clustering.

The objective of the present study is to identify and explain spatial patterns of ASD diagnosis in young children—a group that has seen dramatic increases in prevalence in the U.S. (Keyes et al., 2012)—in a setting in which the diagnosis has not been widely publicised, there is no variation in healthcare access or diagnostic practices, and there are no insurance incentives for ASD (see McInnes et al., 2005). These factors allow us to effectively control for several potentially misleading, cluster-producing variables.

In our study site, Costa Rica, the diagnosis of ASD was first used in 1999, and fewer than 1000 children have been diagnosed. Information about ASD is not easily available on Costa Rican websites, and there is very limited information about ASD in media outlets. In fact, according to our unpublished surveys, most parents have not heard of the diagnosis, and many clinicians are not aware of ASD symptoms. However, children in Costa Rica have universal healthcare that is relatively accessible and equitable (Rosero-Bixby, 2004; Savedoff, 2009), and for diagnostic services, all children under six years old are referred to one centralized clinic, the Hospital Nacional de Niños (HNN). Thus, any clustering in Costa Rica will implicate factors other than variation in healthcare access or diagnostic services.

The first such factor is an information effect. While people tend to know little about autism, an information effect is still likely in Costa Rica, as the period of focus in this study is five years after a small-scale, targeted information campaign was employed to locate cases for a genetic study on ASD (McInnes et al., 2005). The study targeted the founder population that is limited to the Central Valley of Costa Rica, of which the capital, San Jose, is on the eastern edge. The campaign targeted clinicians in close proximity to the HNN by organising seminars about the diagnosis, and teachers in special education schools were also contacted to identify potential cases. Clustering around these clinics could indicate an information effect, but clustering outside the Central Valley, where the special education schools were not contacted by the campaign, would likely indicate a different clustering mechanism; any clustering outside the Central Valley would likely not be a result of information spread. Alternatively, if the information campaign caused clustering, then there should be an increased risk of ASD near the HNN and potentially near daycares, preschools, and special education schools where teachers have learned to identify symptoms.

Also related to an information effect, because early symptom recognition is difficult, information spread would lead to earlier diagnosis and the identification of less severe symptoms (Liu et al., 2010; King and Bearman, 2011). If information spread through professional or parental networks is the cause of clustering, then children living inside a geographic cluster will have received an earlier diagnosis and will exhibit less severe symptoms than children living outside the cluster.

The second factor that could be implicated in the case of clustering is an environmental mechanism that led to more cases in exposed areas. Importantly, there are no coal-fired power plants in Costa Rica, which are significant emitters of mercury and have been linked to spatial patterns of ASD diagnosis (Palmer et al.,

2009). In terms of previously identified environmental factors, the most plausible mechanism for an environmental agent in Costa Rica is air pollution (e.g., Becerra et al., 2013), and because of the mountains that surround the densely populated Central Valley, the highest levels of air pollution are spatially concentrated near the HNN (Barrientos, 2010). Therefore, any clustering patterns that exclude the HNN (and west of the hospital) are likely indicative of non-environmental factors. Clustering patterns that include the HNN could still be explained by travelling distance or urbanicity, which has been associated with the diagnosis (Mandell et al., 2005).

The final factor that could be implicated in the case of clustering is population density. If population density were to explain all the variation in diagnosis, there would be no evidence of social or environmental causes of the clustering.

This study begins on an exploratory note by searching for spatial clusters of ASD diagnosis among children. Then, we use census data and hospital records of the diagnosed children to identify and rule out potential causes of the clustering patterns, first by predicting cases at the smallest census scale and then by focusing on smaller scales.

2. Materials and methods

This project was approved by the bioethics committee at the HNN. The data for the study originate from two sources: (1) district-level census data from the Costa Rican National Institute of Statistics and Census (Instituto Nacional de Estadística y Censos, INEC); and (2) four years of hospital records (2010–2013) of children who were assessed with the Autism Diagnostic Observation Schedule (ADOS) at the HNN. The hospital records included date of birth, sex, age at the time of first words, household district at the time of diagnosis, date of diagnosis, Modified Checklist for Autism in Toddlers (M-CHAT) and Vineland Adaptive Behaviour Scale (VBAS) scores for ASD screening, developmental age, and diagnostic outcome.

We excluded children who were older than five years at the time of screening, as a subgroup of older children is referred to a separate hospital. Also, because the diagnosis and severity of ASD are determined at the time of assessment, we chose not to exclude cases based on the assessment outcome. Thus, cases were defined as all individuals who were screened for ASD at the HNN before the age of six and who were later assessed using the ADOS between 2010 and 2013. Using the M-CHAT and VBAS, the screening process was completed in the developmental unit by a nurse who was trained by the primary diagnosing physician. For the years of focus, there were 127 1–5-year-old patients who underwent an ADOS assessment; nine cases were excluded because of missing address data, so the final sample was 118.

Before identifying district-level clustering, we tested the covariates that could predict the presence of cases in a district. Out of 468 districts, 90 had at least one case, and the maximum number of cases was 6. Thus, the distribution was heavily weighted to zero. We determined that there were not excessive zeros (which would occur if older children were included; see Cox et al., 2009), and the dataset was not overdispersed, so Poisson regression was used to predict district-level cases.

The covariates included at-risk population, percentage of households below poverty, percentage of the population over 15 years old with at least a secondary (middle school) education, percentage of the population in urban areas, distance to the HNN, percentage of children under five years old attending daycare or preschool, and population density. With the exception of distance to the HNN, all values were obtained from the INEC website (<http://www.inec.go.cr>; accessed on May 15, 2014) from the 2011 census.

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