Error and Bias in Geocoding School and Students' Home Addresses

Zandbergen and Green (2007) recently described the effect of positional error on the distance between geocoded addresses and major roads, an often-used proxy for traffic-related exposures. They found a 200–500 m range of mean positional errors in their study of 126 Orange County, Florida, public school addresses, a somewhat higher range than that associated with geocodes assigned by four commercial vendors to a larger variety and number of street addresses in the 48 contiguous U.S. states (Whitsel et al. 2006). In both studies, however, the ranges exceeded commonly used thresholds for identifying those at greatest potential risk of traffic-related exposures, raising due cause for concern.

Zandbergen (2007) found that the use of such low thresholds to define traffic-related exposure surrogates leads to the consistent overestimation of the number of Orange County school children at risk. In this recent study (Zandbergen and Green 2007), the finding has been extended to the schools the children attend. To explain the overestimates, Zandbergen and Green illustrated the idiosyncratic positioning of schools and homes—both within land parcels and along street segments—and the uniformly higher percentage of false positive versus negative determinations of whether the geocoded locations were inside or outside the 50–1,000-m buffer radii examined in their studies.

The collective findings of Zandbergen and Green (2007) nonetheless differ from those based on a previously described 5% random sample of 2,608 street addresses from the Environmental Epidemiology of Arrhythmogenesis in WHI (EEAWHI) (Whitsel et al. 2006). In that study, we found that the fraction of participants' addresses determined to be < 100 m from the nearest highway was relatively constant across mean positional errors of 150–600 m, a finding driven by the counterbalance of approximately equal false positive and negative rates over the same range. The sensitivity and specificity of the 100-m threshold tested in EEAWHI—one-fifth the minimum distance to schools deemed acceptable by Zandbergen and Green— were also around 90% at positional errors of 250–300 m. Moreover, even when the

sensitivity and specificity of the 100-m threshold exceeded 90%, its strength of association with coronary heart disease was still underestimated, albeit in the absence of confounding and under the assumption of nondifferential misclassification.

It is tempting to generalize about the magnitude of error and direction of bias observed by Zanbergen and Green (2007)— to students' school and home addresses outside Orange County, or more generally to epidemiologic measures of environmental exposure–health outcome association—but the most prudent course of action may be to wait until the external validity of their potentially important findings is established.

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Eric A. Whitsel
Department of Epidemiology
School of Public Health
University of North Carolina, Chapel Hill
Chapel Hill, North Carolina
E-mail: ewhitsel@email.unc.edu

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Geocoding School and Student's Home Addresses: Zandbergen Responds

Our research (Zandbergen and Green 2007) strongly suggests that the positional error in street geocoding is not random in direction and that the displacement along the street segment often occurs toward one side of the street because of incorrect address ranges in the street reference data. This "squeeze" effect is a common observation in geocoding using many different street data sets. The extent to which this occurs will vary among locales due to the varying quality of street reference data. The extent to which this introduces any bias into exposure assessments will vary with the specific pollution source being considered. Proximity to major roads with high traffic

counts represents a particular case that is very much influenced by this effect, because many residential streets are perpendicular to major roads and address ranges often start at major roads. For other exposure scenarios, such as air pollution from industrial facilities, the "squeeze" effect will contribute to the overall positional error in geocoding and therefore to any misclassifications, but much less likely to any bias.

Whitsel et al. (2006) determined positional accuracy of geocoding by four commercial vendors through an empirical comparison of criterion locations and vendor-assigned coordinates. In the analysis of the effects of positional error on exposure classification, however, Whitsel et al. (2006) displaced address locations at random over a uniform distribution of the angle of displacement. This assumes there is no direction in the positional error and ignores the "squeeze" effect. Our studies show that the displacement of a street-geocoded location relative to the actual location of the residence is frequently along the street segment, and definitely not random in direction. For a large sample, the distribution of the direction of positional error may appear to be uniform because the directions of street segments often approximate a uniform distribution, unless the street segments follow a very strong grid pattern (e.g., Zimmerman et al. 2007). I therefore argue that the error propagation modeling used by Whitsel et al. (2006) substantially underestimates the effects of positional errors in geocoding on exposure classification for the particular scenario where exposure potential is determined on the basis of distance to major roads. Given the relatively complex nature of the spatial pattern in geocoding errors, we feel that determining misclassification based on actual geocoded locations is more reliable than employing simulated displacements.

I agree, however, that care should be taken in generalizing the results from our studies, and we do not think the 250–500-m range is the lower limit of spatial epidemiologic analysis in general. However, I challenge the commonly held assumption that positional errors in geocoding are relatively small, random in terms of their direction, and without positional bias.

Contrary to other forms of digital spatial data (e.g., land use, roads, census boundaries), geocoding results do not have an implicit scale, and hence the spatial resolution is not known without testing. Certainly, the scale of geocoded locations is not the same as the scale of the street reference data employed. The studies by Whitsel et al. (2006) and my own research represent the few attempts at determining the effective

resolution of geocoding; that is, how reliable is spatial analysis of geocoding results at small distances? This effective resolution will depend on several factors, not the least of which is the variation across urban–rural gradients. For Orange County, Florida (Zandbergen 2007), I found that street geocoding of residential addresses using local street centerlines (1:5,000) resulted in a 90th percentile of the error distribution of 100 m. This corresponds very closely to the results of Cayo and Talbot (2003), who found a value of 96 m for urban areas and much larger values for suburban and rural areas. Based on this 90th percentile, typical street geocoding of residential addresses does not meet the positional accuracy standards for a 1:100,000 scale map based on the National Map Accuracy Standards (U.S. Bureau of the Budget 1947).

Higher-quality street reference data is expected to improve the positional accuracy of geocoding results, primarily through improved address ranges. However, I argue that the linear interpolation algorithm used in street geocoding presents inherent limitations, resulting in data that are insufficient for many large-scale applications. Higher-accuracy alternatives will need to be considered, including address points. In the address-point data model, residences and other buildings are represented as single points, with a much greater positional accuracy than is achievable using street geocoding. For a review and comparison of methods, see Zandbergen (2008). Several other jurisdictions, including Australia, Canada, and the United Kingdom, have already developed national address-point databases. In the United States, address-point databases are currently limited to selected areas, but this is expected to change. Epidemiologic researchers that employ geocoding would greatly benefit from being aware of alternatives to traditional street geocoding, in particular when analysis at fine spatial scales is required.

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Paul A Zandbergen
Department of Geography
University of New Mexico
Albuquerque, New Mexico
E-mail: zandberg@unm.edu

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Defective Spermatogenesis in Cryptorchid Testes: Cause or Effect?

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Martin et al. (2008) recently published their quantitative meta-analysis focusing on the estrogen hypothesis of testicular dysgenesis syndrome. I congratulate the authors on their thorough review and excellent summary of the existing literature. The study findings are in line with other articles; however, there are several concerns that need further attention.

Martin et al. (2008) pointed out that a common etiology underlies impaired spermatogenesis, male reproductive tract abnormalities such as hypospadias and cryptorchidism, and testicular cancer. I am especially interested in exploring the relationship between defective spermatogenesis and cryptorchidism.

Maldescended testes is commonly cited as an important cause for defective spermatogenesis (Tomomasa et al. 2002). In contrast, testicular ascent (acquired cryptorchidism) could also be a risk factor for spermatogenesis in infertile men without any history of maldescended testes (Mieusset et al. 1997). However, it remains controversial whether impaired testicular function and spermatogenesis imparts an increased risk—and therefore represents a common pathogenetic mechanism of both congenital and acquired cryptorchidism—or is merely associated with disease. Recently, a potential link was proposed relating spermatogenesis and testicular descent (Skandhan and Rajahariprasad 2007). Observational studies of many lower animals (rodents, bats, and insectivores) have revealed that testicular position is dependent on its functional status: It is scrotal during breeding seasons and inguinal or abdominal at other times (Bannister and Dayson 1995). Therefore, it is possible that maldescended testes or acquired testicular ascent simply report a state of defective testicular function and spermatogenesis. In animal studies, estrogen has been shown to increase the number of type A spermatogonia, together with inhibition of their differentiation into further steps (Kula et al. 1997). Furthermore, supportive evidence suggests that undifferentiated type A spermatogonia are the only germ cells present in cryptorchid testes (Nishimune et al. 1978). I believe that the results of Martin et al. (2008) would have been more convincing if the authors could have shown that high levels of estrogens suppress spermatogenesis.

The data of Martin et al. (2008) do not allow us to extrapolate whether exposure to environmental chemicals and pollutants with estrogenic or antiandrogenic effects can cause testicular "ascent" (Barthold and González 2003). There is strong experimental evidence that prenatal exposure to environmental chemicals, including phthalate esters, is associated with an increased risk of postnatal cryptorchidism (Imajima et al. 1997). The similarity in the histopathology of the ascending testis and the testis undescended since birth suggests that ascending testes are not retractile testes trapped in scar tissue (Rusnack et al. 2002). Furthermore, this finding also suggests that, as in primary undescended testes, estrogen/antiandrogen hypotheses could explain the cause of ascending testes, because a thermal effect cannot be blamed for the decreased germ cell count in the descended testis.

Overall, the systematic review and meta-analysis by Martin et al. (2008) is the most extensive attempt to date to investigate the link between estrogenic agents and testicular dysgenesis syndrome. Although some of the data from the cited studies are of limited quality, the fact that nearly all of the included studies identified an increase in the risk of hypospadias, cryptorchidism, and testicular cancer in the groups prenatally exposed to diethylstilbestrol provides strong support for that association being genuine. However, from the data of Martin et al. (2008), we cannot conclude whether exposure to environmental chemicals with estrogenic effects significantly increases the risk of developing acquired cryptorchidism. Further research to evaluate the effects of endocrine-disrupting chemicals (EDCs)—particularly those with estrogen-like effects on reproductive health—is justified and should continue.

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Hari Prasad
School of Medical Science and Technology
Indian Institute of Technology
Kharagpur, India
E-mail: haribhata@yahoo.com

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Defective Spermatogenesis: Martin et al. Respond

In response to Prasad's constructive comments on our quantitative meta-analysis of the estrogen hypothesis and testicular dysgenesis syndrome (Martin et al. 2008), we offer the following observations regarding the scope of our study and limitations of the methodology applied.

The primary objective of a quantitative meta-analysis is to combine the results of previous studies examining a specific research question to arrive at a summary conclusion about a body of research. This statistical pooling of several studies, taking into account the size of individual studies, confers more power to detect a potential association, and quantitative meta-analyses are often put at the top of evidence hierarchies. It cannot, however, correct for potential bias and confounding of the studies included; we addressed this issue in our review (Martin et al. 2008) by rating the quality of individual studies and carrying out a sensitivity analysis by excluding studies for which the quality score was below a chosen value. The method also requires that included studies report a measure of association such as a risk ratio or odds ratio. For this reason— although we did mention impaired spermatogenesis as one of the end points encompassed by the testicular dysgenesis syndrome—it was necessary to exclude this end point from our analysis.

In previous work and a scoping study, we found that most of the research carried out in relation to impaired spermatogenesis had investigated time trends rather than association with specific risk factors (Martin et al. 2007). Further, our analysis was limited to prenatal exposure to estrogenic agents. A number of studies have found associations between sperm motility or sperm DNA damage with levels of estrogenic chemicals measured either in urine or serum (Duty et al. 2003; Spanò et al. 2005). It would not be possible however to relate such levels to prenatal exposure. This also illustrates the difficulty of selecting a suitable marker of impaired spermatotogenesis.

Our study was implicitly limited to congenital cryptorchidism because the literature search did not yield any case–control or cohort studies that addressed the question of prenatal exposure to estrogenic compounds and acquired cryptorchidism in humans. In retrospect, this should have been explicitly stated in our article (Martin et al. 2008).

We concluded that the significant association between prenatal diethylstilbestrol exposure and all three end points considered conferred weight to the hypothesis of a common etiology for these disorders, and therefore to the existence of a testicular dysgenesis syndrome (Martin et al. 2008). Separate analyses were carried out for the three end points but the methodology applied did not allow us to explore the specific nature of causal relationships between congenital cryptorchidism, hypospadias, and testicular cancer. We are therefore grateful for Prasad's insights.

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Olwenn V. Martin
Tassos Shialis
Alan R. Boobis
Nikolaos Voulvoulis
Imperial College London
London, United Kingdom
E-mail: n.voulvoulis@imperial.ac.uk

John N. Lester
Centre for Water Sciences
Cranfield University
Cranfield, United Kingdom

Mark D. Scrimshaw
Institute for the Environment
Brunel University
Uxbridge, United Kingdom

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