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Statistical implications of endogeneity induced by residential segregation in small-area modelling of health inequities

Rachel C. Nethery^{1*}, Jarvis T. Chen², Nancy Krieger², Pamela D. Waterman², Emily Peterson³, Lance A. Waller³, Brent A. Coull¹

¹Department of Biostatistics, Harvard T.H. Chan School of Public Health, Boston, MA, USA

²Department of Social and Behavioral Sciences, Harvard T.H. Chan School of Public Health, Boston, MA, USA

³Department of Biostatistics and Bioinformatics, Emory Rollins School of Public Health, Atlanta, GA, USA

*Corresponding author 655 Huntington Ave, Building 2, 4th floor, Boston, MA 02215. **Email:** rnethery@hsph.harvard.edu

Health inequities are assessed by health departments to identify social groups disproportionately burdened by disease and by academic researchers to understand how social, economic, and environmental inequities manifest as health inequities. To characterize inequities, group-specific small-area health data are often modeled using log-linear generalized linear models (GLM) or generalized linear mixed models (GLMM) with a random intercept. These approaches estimate the same marginal rate ratio comparing disease rates across groups under standard assumptions. Here we explore how residential segregation combined with social group differences in disease risk can lead to contradictory findings from the GLM and GLMM. We show that this occurs because small-area disease rate data collected under these conditions induce endogeneity in the GLMM due to correlation between the model's offset and random effect. This results in GLMM estimates that represent conditional rather than marginal associations. We refer to endogeneity arising from the offset, which to our knowledge has not been noted previously, as "offset endogeneity". We illustrate this phenomenon in simulated data and real premature mortality data, and we propose alternative modeling approaches to address it. We also introduce to a statistical audience the social epidemiologic terminology for framing health inequities, which enables responsible interpretation of results.

Keywords: small-area analysis; generalized linear mixed models; disease mapping; offset endogeneity; interdisciplinary

1. Introduction

Modeling of small area disease/health event rates has long been critical to quantifying health inequities— that is, unjust, unnecessary, and in principle preventable differences in health status across social groups (Braveman and Gruskin 2003) – and to informing public health policies seeking to eliminate these inequities. Health departments employ such models to identify social groups disproportionately and unfairly burdened by disease and likely to benefit from public health resources (Association of State and Territorial Health Officials 2021; National Association of County Health Officials 2021). Academic researchers also model disease rates to investigate how social, economic, and

environmental inequities across social groups manifest as health inequities and how risk patterns vary over space and time (Beckfield 2018). Small-area disease rates for social groups, e.g. racialized or economic groups, are often utilized for this purpose, and either generalized linear models (GLM) or generalized linear mixed models (GLMM) for count/rate outcomes are employed to estimate inequities (Burton et al. 2010; Chen 2013; Massachusetts Department of Public Health 2010).

Under the usual modeling assumptions (described in Section 4), the standard log-linear GLM and GLMM used to model inequities estimate the same marginal rate ratio comparing health outcomes across social groups (Demidenko 2007; Young et al. 2007; Zhang et al. 2012). In academic research, log-linear GLMMs, also known as disease mapping models when applied to health data from small enumeration areas, are often preferred due to their ability to estimate inequity measures with a marginal interpretation while accounting for spatial correlation in small-area data. These models also yield smoothed rate estimates for small areas where observed rates may be noisy due to small local sample sizes. However, GLMMs may not estimate marginal parameters if the data violate key modeling assumptions. For instance, endogeneity in the form of correlation between covariates and areal-level random effects in a GLMM can compromise the marginal interpretations of parameters in the log-linear GLMM (Bates et al. 2014; Neuhaus and McCulloch 2006).

Although the impacts of endogeneity in multilevel model frameworks have been well-studied, little to no work has focused on the implications for disease mapping models in health inequity research. We find that residential segregation combined with social group differences in on-average individual risk give rise to small-area data that induce endogeneity due to correlation between *the offset* and random effects in disease mapping models. While others have examined endogeneity due to correlation between covariates and random effects (Bates et al. 2014; Neuhaus and McCulloch 2006), to our knowledge this specific form of endogeneity, which we call “offset endogeneity”, has not been documented previously. This study examines and demonstrates how offset endogeneity leads to model misspecification and contradictory findings from the GLM and GLMM approaches.

Specifically, using the example of health inequities across racialized social groups, we demonstrate how racialized residential segregation across small areas combined with baseline on-average racialized differences in health outcomes, conditions which frequently co-occur, leads to offset endogeneity and contradictory inequity estimates from the GLM and GLMM. We evaluate the magnitude and implications of these discrepancies in real data and in simulations, and we propose an adjusted model specification that we term the “individual and neighborhood inequities” model (INE). This alternative formulation addresses the endogeneity problem by explicitly conditioning the models on both individual-level racialized social group membership and neighborhood racialized social group composition, and it can provide greater insights into the factors driving health inequities.

As a secondary objective of this paper, responding to recent calls for more thoughtful and fair uses of social data in health-related analyses (Chen et al. 2021; Krieger 2021), we employ social epidemiologic terminology for framing health inequities, which is critical to proper interpretation of studies employing these concepts. In Section 2, we explain and justify this terminology. In Section 3, we motivate this work by describing census tract (CT) premature mortality rate data for two racialized social groups in Massachusetts (MA), as defined by the US census: the Black population and the white non-Hispanic population (WNH) (US Census Bureau 2017, 2020). In Section 4,

we formalize the GLM and GLMM approaches and examine how small-area disease/health event data stratified by racialized social group can induce offset endogeneity. In Sections 5 and 6, we evaluate the impact of this issue using simulated and real data, respectively. We conclude with a discussion in Section 7.

2. Language and terminology for health inequity studies

Because the aim of health inequity studies is to characterize unfair burdens of morbidity and mortality borne by social groups harmed by injustice (Braveman and Gruskin 2003; Krieger 2020), we utilize language that emphasizes the power dynamics and the agency (or denial thereof) of the groups that lead to inequities (Benjamin 2017; Krieger 2020, 2021). Recognizing that some of this terminology may be unfamiliar to a statistical audience, we explain and justify it here. We first note that, in statistics, much emphasis is placed on mathematical formalization of concepts, and, when working in interdisciplinary settings, formalizing the subject matter is equally important. This is perhaps uniquely critical when studying social constructs, as improper framing in such studies has historically been used to justify unjust beliefs and actions (Benjamin 2017; Krieger 2020, 2021). Because we examine a statistical problem brought about by complex social dynamics, formalizing and emphasizing groups' relative power and agency is essential for accurate interpretation.

First, we use the term health inequity, as opposed to health disparity, to make clear that we are focused on preventable health differences across groups that arise because of social and structural inequities rather than biological differences (Beckfield 2018; Braveman and Gruskin 2003; Krieger 2020). We consider health inequities between two social groups co-defined by their power relations, which we refer to abstractly as the “privileged” and “marginalized” groups. This language highlights the fact that the marginalized group is denied agency in determining its status. Note that the term marginalized when applied to social groups has no connection to and should not be confused with the statistical concept of marginalization. In our motivating example, we utilize the term racialized social group, instead of the more commonly used but problematic term “race” (Benjamin 2017; Krieger 2021), to more precisely describe the theoretical social construct we aim to study. This terminology clarifies that “race” is socially (rather than biologically) defined and is not an innate individual “property” or trait. In practice, we interpret the data on what is termed “race” and “ethnicity” in the administrative health and census data employed in our analyses as indicators of racialized social group. For easy reference, this terminology is summarized in Table 1. Additional references on this topic are provided in the Supplementary Materials.

3. Motivating data

To motivate this work, we introduce the data we use to investigate inequities in CT-level premature mortality (death before age 65) in the Black and WNH populations in MA. We analyze data for the Black population, as opposed to the Black non-Hispanic population, because CT-level age-stratified population count data are not available for the latter (9.8% of Black individuals in MA also identified as Hispanic in the 2010 census). CTs are US Census Bureau administrative units designed to include roughly 4,000 residents (US Census Bureau 2019). They are commonly used to investigate spatial patterns in health (Krieger et al. 2005). Premature mortality is a widely used population health indicator for characterizing health inequities (Chen et al. 2006).

We obtained records for all premature deaths in MA during 2008-2012, dates chosen to be centered around the 2010 decennial census, from the MA Department of Health (55,836 total premature deaths). We geocoded the residential address at death to the corresponding CT, with only 0.4% of deaths unable to be geocoded at that level of precision. We extracted the racial/ethnic-, age- and sex-stratified population counts, as categorized with US census terminology, for each CT from the 2010 decennial census

using the R package `tidycensus` (Walker 2020). We then multiplied each population count by five to obtain the at-risk person-years corresponding to our five years of mortality data. Our study includes all MA CTs with non-zero Black and WNH populations, totaling $N = 1,465$ CTs (99.1% of MA's 1,478 CTs in 2010).

Let $i = \{1, \dots, N\}$ index CT and $j = \{0, 1\}$ index racialized social group within CT so that Y_{ij} is the observed count of premature deaths in racialized group j within CT i . We constructed standardized mortality ratios (SMRs) using the indirect standardization approach (Chen 2013). CT and racialized social group-specific expected premature death counts are calculated to serve as a denominator (the log of the denominator often serves as the offset in statistical modeling) to account for differential at-risk person-time and age and sex compositions. See the Supplementary Materials for more detail. We denote the expected count for racialized social group j in CT i by P_{ij} .

The SMR is then given by $SMR_{ij} = Y_{ij}/P_{ij}$. Figure 1A shows the distributions of the CT racialized social group-specific 5-year expected counts and SMRs. To illustrate the racialized segregation and the concentration of the Black population in a small number of CTs, Figure 2 maps the percent Black for all CTs in MA, and a magnified view of the city of Boston. The average percent Black across all MA CTs in 2010 was 7.5%, and approximately 39% of MA CTs had populations composed of <2% Black individuals. Boston was more diverse, with an average of 23.7% Black across the 177 Boston CTs.

4. Methods

We focus on two traditional approaches to health inequity analysis using small-area data: GLMs, also called aggregated analyses, and GLMMs, also called disease mapping models, both of which we describe in detail below. For general exposition of the statistical concepts and methods, we abstract to two social groups: privileged and marginalized (in our motivating data, the WNH and Black populations). Analogous to the notation introduced above, let i index CT and j index social group.

A standard GLM specification for estimating health inequities across the study population's social groups is

$$\log(E(Y_{ij}|X_{ij})) = \alpha_0 + \alpha_1 X_{ij} + \log(P_{ij}), \quad (1)$$

where X_{ij} denotes the indicator of social group (marginalized = 1, privileged = 0). Y_{ij} and P_{ij} are the CT- and social group-specific observed event count and expected event count, described in Section 3. This model is often estimated assuming a Poisson likelihood, although more flexible approaches such as the quasi-Poisson or the negative binomial distribution are also common. The rate ratio (RR) for comparing the CT-level rate of the health event in the marginalized vs. privileged population is $\exp(\alpha_1)$.

The GLM approach assumes that the independent unit of analysis is the CT- and social group-specific rate; however, the independence assumption is likely to be violated, as the rates of a health outcome in marginalized and privileged populations in the same neighborhoods are almost certain to be correlated (Kiang et al. 2019). In spite of its oversimplified specification, the GLM approach retains relevance because the RR estimator from the GLM can be shown to be equivalent to that obtained from an aggregated social group inequity analysis. The aggregated approach, often used by state health departments

to monitor health inequities, aggregates incidence data from the entire state to generate a state-wide age-adjusted RR estimate comparing the marginalized and privileged groups (Massachusetts Department of Public Health 2010).

The GLMM extends the GLM approach by including a random intercept, often with a spatial correlation structure, along with the social group indicator fixed effect which is used to estimate the RR (Chen 2013; Kiang et al. 2019). In addition to estimating the overall RR, this model enables estimation of smoothed area-specific incidence/mortality rates, which can be used to further investigate spatial patterns in risk. A standard GLMM specification for estimating health inequities is

$$\log(E(Y_{ij}|X_{ij}, b_i)) = \beta_0 + \beta_1 X_{ij} + b_i + \log(P_{ij}), \quad (2)$$

where b_i is a CT-specific random intercept. In the standard mixed model framework, the b_i are assumed to be i.i.d. normal, i.e., $b_i \sim N(0, \sigma_b^2)$. In addition to accounting for correlation in social group-specific health event rates from the same CT, the random effect can also be conceived as capturing some unmeasured CT-level factors that drive baseline rates. The spatial version of this model for areal data generally specifies a conditionally auto-regressive (CAR) structure on b_i , which further allows for correlation in the event rates of spatially proximate CTs (Schmidt and Nobre 2018). Previous work has described conditions that can lead to discrepancies in fixed effect estimates from spatial and non-spatial mixed models (Hodges and Reich 2010). In this work, in order to isolate the impact of endogeneity resulting from residential segregation, we focus on the non-spatial GLMM, but the issues we illustrate here would extend to spatial analogues.

For many link functions, the fixed effects from GLMs and GLMMs are not directly comparable, as GLM parameters represent marginal associations while GLMM parameters represent conditional ones (given the CT-level random effects). Yet, under standard assumptions, a log-linear GLMM with normally distributed random intercepts is a special case in which the parameters in the two models coincide (Demidenko 2007; Young et al. 2007; Zeger, Liang, and Albert 1988; Zhang et al. 2012). This is apparent once one integrates over the random intercepts in the GLMM, yielding

$$E(Y_{ij}|X_{ij}) = E_b(E(Y_{ij}|X_{ij}, b_i)) = \exp\left(\left(\beta_0 + \frac{1}{2}\sigma_b^2\right) + \beta_1 X_{ij} + \log(P_{ij})\right).$$

Thus, the marginal mean structure for this GLMM is equivalent to the GLM mean structure, with the exception of the constant multiplicative term $\exp\left(\frac{1}{2}\sigma_b^2\right)$, a function of the variance of the random effects. While this term causes the intercepts from the two models to differ by $\frac{1}{2}\sigma^2$, the marginal RR for social group remains $\exp(\beta_1)$. This feature of a log-linear GLMM with a random intercept, namely its ability to estimate marginal fixed effect parameters while accounting for complex correlation structures and enabling estimation of smoothed rates, makes it a popular choice for disease mapping.

4.1 Endogeneity

As shown above, $\exp(\alpha_1)$ and $\exp(\beta_1)$ coincide when the true mean structure underlying the data is given by equation (1) or (2). However, we do not expect that these are the true, “causal” data generating mechanisms for complex health outcomes like premature mortality. In addition to describing patterns of unequal risk experienced by marginalized groups, researchers may also want to explore the mechanisms by which inequities arise,

e.g., by including additional variables such as economic indicators or environmental exposures in statistical models, without necessarily making causal claims about “the effect” of social group. Although building a causal model may not be the goal of inequity analyses, certain types of model misspecification and violations of assumptions can impact model performance and interpretability of the results.

In biostatistics, one often overlooked assumption of the GLMM is that the random effects b_i are uncorrelated with the fixed predictors on the right-hand side of the model (note that we use “fixed predictor/covariate/term” to refer to any covariates modeled with fixed rather than random effects). Most commonly discussed in the economics and education literature, correlation between random terms and fixed terms on the right-hand side of a regression model is referred to as the problem of *endogeneity* (Bates et al. 2014). GLMMs assuming i.i.d. normal random effects suffer from model misspecification in the context of endogeneity arising due to correlation between covariates and random effects, which has been noted in biostatistical contexts as well (Neuhaus and McCulloch 2006). Because endogeneity is a very general term, often referring to omitted variable bias in causal inference settings (where omitted variables are confounders of the “causal” effect of an exposure of interest), we clarify that our use of the term endogeneity throughout this paper refers to model misspecification arising from correlation between a fixed term and a random term in a non-causal setting.

Critically, when a fixed term is correlated with the random effect, fixed effect parameters in the properly-specified GLMM have a conditional, rather than marginal, interpretation, even in the special case of the log-linear model with a random intercept. Moreover, some literature on this topic suggests that, when a standard GLMM with i.i.d. random effects is erroneously specified and fit in the presence of this type of endogeneity, fixed effect parameter estimates are largely insensitive to the model misspecification (Neuhaus and McCulloch 2006), meaning that bias of the conditional effect estimator under endogeneity is typically small.

While the RR estimator from the GLMM estimates the conditional association parameter in the presence of endogeneity, the RR estimator from the GLM estimates the marginal association. Thus, endogeneity can lead to discrepancies between the GLM and GLMM inequity estimates. In the next section, we describe how endogeneity can arise in health inequity models due to correlation between the offset and the random intercept.

4.2 Example: Racialized residential segregation and baseline health differences

Here we demonstrate how the combination of racialized residential segregation and higher baseline rates of disease and mortality in neighborhoods with a high proportion of people of color— conditions that commonly co-occur in practice— induce a previously undocumented type of endogeneity, one relevant more broadly in the context of residential segregation and health inequities. In our setting, the history of racialized residential segregation at the neighborhood level results in within-CT homogeneity of residents’ racialized social group membership. As a result, CT expected event counts in Black and WNH groups, P_{i1} and P_{i0} respectively, will be negatively correlated in the presence of racialized residential segregation, with the magnitude of the correlation reflecting the degree of CT segregation. Moreover, if Black populations comprise a small portion of the study area’s total population and reside largely in a small number of neighborhoods, as in MA (Figure 2), P_{i1} may be very small in most neighborhoods, resulting in unstable, zero-heavy SMRs for Black populations.

In the US, adverse racialized residential segregation is often accompanied by the residents’ greater exposure to community disinvestment and environmental hazards, as well as

household- and individual-level exposures to racial discrimination and economic deprivation, together potentially increasing risk of poor health status among all persons living in this neighborhood (Duncan and Kawachi 2018; Roux and Mair 2010). The converse is that exclusionary racialized residential segregation, to protect privilege, adds to the health advantages of these areas' residents (Duncan and Kawachi 2018; Roux and Mair 2010). Higher baseline rates of the health outcome in neighborhoods whose residents are predominantly people of color— in our example, Black— can be considered in the context of a GLMM-type data generating mechanism (equation 2) as higher values of the random effect, b_i , occurring in CT with high Black population size/expected counts, P_{i1} . This leads to a setting in which b_i is positively correlated with P_{i1} and negatively correlated with P_{i0} —endogeneity arising from correlation between a random effect and the *offset*, which we refer to as “offset endogeneity”. Although the offset is in essence just a special type of covariate (with coefficient fixed to 1), to our knowledge it has never before been identified as a source of endogeneity. Note that, in this scenario, b_i has no association with X_{ij} , since each CT has exactly one count with $X_{ij} = 1$ and one with $X_{ij} = 0$ by design. Nonetheless, offset endogeneity introduced by the correlation between b_i and P_{i1} (and P_{i0}) creates a scenario in which the marginal and conditional associations between Y_{ij} and X_{ij} differ, and the RR parameters from a GLM and GLMM diverge, even when using a log link.

Further insight into this phenomenon can be obtained by deriving the closed form estimators of the coefficients in the Poisson GLM and a simplified analogue of the Poisson GLMM. First, note that the GLM we specified is a saturated model, so that the estimator of α_1 when using a Poisson distribution takes the form

$$\hat{\alpha}_1 = \log \left(\frac{(\sum_i Y_{i1})(\sum_i P_{i0})}{(\sum_i Y_{i0})(\sum_i P_{i1})} \right) = \log \left(\frac{Y_1 P_0}{Y_0 P_1} \right). \quad (3)$$

This confirms that the RR estimator from the GLM is equivalent to the RR estimator in an aggregated analysis, as stated in Section 1. Now, in order to develop closed-form expressions that highlight the impact of endogeneity, for the moment assume the random effects b_i in the GLMM model are *a priori* known constants. In this case the b_i can be absorbed into the offset term and the model is again a saturated model with closed form estimator of β_1

$$\hat{\beta}_1^* = \log \left(\frac{(\sum_i Y_{i1})(\sum_i P_{i0} \exp(b_i))}{(\sum_i Y_{i0})(\sum_i P_{i1} \exp(b_i))} \right) \quad (4)$$

Because this model explicitly conditions on b_i , $\hat{\beta}_1^*$ has a conditional interpretation. From these closed form estimators, we can see that if $P_{i1} = \pi P_{i0}$ for some constant $\pi \geq 0$, then $\hat{\beta}_1^*$ and $\hat{\alpha}_1$ reduce to the same expression. When P_{i1} is a constant proportion of P_{i0} across areas, this association indicates the absence of racialized residential segregation, and in this setting, the marginal and conditional inequity RR estimators will coincide.

However, when there is both racialized residential segregation, $P_{i1} \neq \pi P_{i0}$, and baseline heterogeneity in rates of the outcome across areas, $b_i \neq 0$, equations (3) and (4) are not equal. Thus, the marginal and conditional estimators will differ. To gain further insight, we now equivalently write equation (4) as

$$\hat{\beta}_1^* = \log \left(\frac{(\sum_i Y_{i1})(\sum_i P_{i0} \varphi_i)}{(\sum_i Y_{i0})(\sum_i P_{i1} \varphi_i)} \right) \text{ where } \varphi_i = \frac{\exp(b_i)}{\sum_i \exp(b_i)}$$

where the $\varphi_i \in [0,1]$ can be thought of as weights. This “weighted” expression makes clear that when the b_i are large for large P_{i1} (i.e., co-occurrence of racialized residential segregation and

higher baseline health risk in predominately Black communities), the conditional estimator $\hat{\beta}_1^*$ up-weights the Black expected counts in the denominator that are the largest, and down-weights the WNH counts in the numerator that are the largest. Comparing this to the estimator $\hat{\alpha}_1$ in equation (3), which takes the same form but with weights $\varphi_i = 1/N$, we see that when $\hat{\alpha}_1 > 0$, we also have $\hat{\alpha}_1 > \hat{\beta}_1^*$. Thus, the marginal estimate will be larger than the conditional estimate in the presence of racialized residential segregation and higher baseline risks in predominately Black communities. This suggests that, in the presence of offset endogeneity, the health inequity estimate from the GLMM will be an underestimate of the marginal association.

Our MA premature mortality data illustrate this phenomenon (Figure 1B). In the left panel, the CT observed vs expected counts are plotted for Black and WNH populations, and a steeper positive relationship on average for the Black population is evident by the corresponding regression lines. In the right panel, we multiply the expected counts for each racialized group by the total-CT baseline mortality rate, which we call the “adjusted expected premature mortality”. After this adjustment, a steeper relationship on average for the WNH population emerges. The left panel roughly corresponds to the Black vs WNH relationship detected by the GLM and the right panel to the relationship detected by the GLMM. We delve deeper into this issue below with pseudo-simulated data and provide results for the MA premature mortality data.

4.3 Separating neighborhood composition from individual group membership in health inequities modelling: the “individual and neighborhood inequities” model

The phenomenon described above leads to a GLM RR estimate that reflects the marginal comparison of Black vs. WNH premature mortality across all CTs, while

the GLMM RR estimate more closely approximates a conditional comparison of Black vs. WNH premature mortality within a given CT. Thus, the GLMM RR estimate is effectively comparing mortality rates for Black vs. WNH individuals living in the same neighborhood. Because the goal of small-area health inequity modeling is, in general, not to estimate the causal effect on the outcome of an individual belonging to a specified social group, but rather to understand how an accumulation of social factors results in social group inequities in morbidity or mortality, the GLMM may be “adjusting away” the association of interest in disease mapping studies of health inequities.

Because the GLMM may implicitly separate the neighborhood-level social group associations (random effect) from the individual-level social group associations (fixed effects), we suggest explicitly disentangling these factors in the models through the use of additional area-level fixed effects. In particular, we recommend the following modified GLMM specification:

$$\log\left(E(Y_{ij}|X_{ij}, b_i)\right) = \beta_0 + \beta_1 X_{ij} + \beta_2 W_i + b_i + \log(P_{ij}) \quad (5)$$

where W_i is the CT-level marginalized group composition (say, the percent Black in the CT). Here, $\exp(\beta_2)$ is the multiplicative change in premature mortality risk corresponding to a one-unit increase in a CT’s percent marginalized residents. The term $\exp(\beta_1)$ is the multiplicative association between premature mortality and individual-level marginalized group membership, conditional on the CT’s marginalized group composition. Analogously, W_i can be included as a covariate in the GLM.

This alternative modeling approach, which we call *the individual and neighborhood inequities (INE) model*, has several implications. First, including W_i in the GLMM

should reduce or eliminate the offset endogeneity issue. Thus, if W_i is included in both the GLM and GLMM, they should produce similar RR estimates. To explain heuristically why this addresses offset endogeneity using our real data example, think of the random intercept in equation (2) as capturing average residual structure across racialized groups with a CT, after adjusting for X_{ij} and P_{ij} . Even “adjustment” for the offset by including it in the fixed portion of the model does not account for disproportionately high premature mortality rates in CTs with larger Black populations, because there are separate racialized group-specific offsets for each CT (P_{i1} and P_{i0}), and the model does not allow for different effects of these two offset terms (in fact the coefficient of the offset is fixed to one across the board). Thus, the increased premature mortality risk in CTs with large Black populations remains in the residual structure after conditioning on X_{ij} and P_{ij} , and the random intercept captures it, leading to a correlation between b_i and P_{i1} and inducing offset endogeneity. However, including CT proportion Black, W_i —which is common for both racialized groups within a CT—as a covariate effectively adjusts for the increased baseline risk in CTs with large proportion Black and removes that structure from the residuals, so that the random intercept does not capture it and is not highly correlated with P_{i1} nor P_{i0} .

Including W_i explicitly separates the association between neighborhood social group composition and health from the association between individual membership in the social group and health, conditioning each association on the other. In many contexts, separating these associations may provide additional insights into the social factors that lead to inequities. Moreover, the modified GLMM can still provide smoothed small-area estimates of incidence rates or rate ratios, which are often a target of inference in the GLMM approach. As discussed by Chen et al. (2006), when including area-level covariates in health inequity models (W_i), consideration should be given to whether the smoothed quantities of interest are best represented by the b_i or by $\beta_2 W_i + b_i$. If, instead, interest lies in the RR estimate that compares overall disease incidence across social groups, the standard GLM specification in equation (1) or a generalized estimating equations (GEE) approach, which may produce more accurate uncertainty estimates, will be most appropriate. Although they are not commonly used in this context, in our simulations and data analysis below, we fit GEE models as a comparator for the GLMs and GLMMs.

5. Simulations

This simulation study was conducted in R (R Core Team 2020), and the code is provided in the online Supplementary Materials and on Github. To illustrate how racialized residential segregation and differing baseline health event rates impact common models for health inequity analyses, we simulate CT-level racialized social group-specific data reflecting various combinations of these features. Following the GLMM notation above, CT event counts are generated via independent Poisson draws, $Y_{ij} \sim \text{Poisson}(\lambda_{ij})$, with

$$\log(\lambda_{ij}) = \beta_0 + \beta_1 X_{ij} + b_i + \log(P_{ij}).$$

As detailed below, four different mean structures are created by altering the specification of the baseline rates, b_i , and the expected counts P_{ij} . All simulations have $N = 1,465$ CTs, corresponding to the number of MA CTs in our study.

Simulation 1: Racialized residential segregation, common baseline rate across CTs. The CT-specific Black and WNH expected counts are negatively correlated. They are

taken from the real MA CT data, to simulate a realistic degree of segregation and small Black populations in many CTs. A common baseline rate is achieved by setting $b_i = 0$.

Simulation 2: No racialized residential segregation, differing baseline rates across CTs. $b_i \sim N(0, \sigma_b^2)$ i.i.d., is randomly generated to create differing CT baseline event rates. Expected counts do not differ by racialized social group within a CT, i.e., $P_{i1} = P_{i0}$, to represent a setting with no racialized residential segregation. We generate $P_{i0} \sim Unif(0.2, 4)$ i.i.d.

Simulation 3: Racialized residential segregation, differing baseline rates across CTs. CT baseline rates independent of racialized group-specific expected counts. $b_i \sim N(0, \sigma_b^2)$ i.i.d., is randomly generated. CT-specific Black and WNH expected counts are negatively correlated. The expected counts are again taken from real MA CT data.

Simulation 4: Racialized residential segregation, differing baseline rates across CTs. CT baseline rates positively associated with Black expected counts and negatively associated with WNH expected counts. Expected counts P_{ij} are taken from real MA CT-level data. We generate the b_i independently from $b_i \sim N(\mu_i, \sigma_{b4}^2)$ where $\mu_i = -\tilde{P}_{i0}/5$ and \tilde{P}_{i0} is the centered and scaled WNH expected count. This leads to a correlation of -0.36 between b_i (the baseline rate) and the WNH expected count P_{i0} , and a correlation of 0.16 between b_i and the Black expected count P_{i1} . This simulation corresponds to the structure anticipated in real health data from segregated areas in MA.

In Simulations 1-3, where the coefficients have marginal interpretations, we fix $\beta_0 = 0.02$ and $\beta_1 = 0.32$. In Simulation 4, where the coefficients have a conditional interpretation, we let $\beta_0 = 0.03$ and $\beta_1 = -0.07$. Where applicable, $\sigma_b^2 = 0.19$ and $\sigma_{b4}^2 = 0.25$. We generate 200 datasets for each of the four simulation scenarios. To each dataset, we fit a Poisson and negative binomial GLM (equation 1), a Poisson and negative binomial GLMM (equation 2), and a GEE model with a Poisson-like quasi-likelihood and exchangeable working correlation structure within each CT. We report the bias of the Black vs. WNH RR estimate from each model (Figure 3), and the 95% confidence interval coverage probabilities for each RR estimate (Table S.1).

In Simulations 1-3, all models yield estimates of the RR with little empirical bias in settings with racialized residential segregation alone or differing CT baseline rates alone, or when both segregation and differing baseline rates are present but are uncorrelated. In Simulation 4, the scenario in which the baseline rates and racialized social group-specific expected counts are correlated, offset endogeneity yields discrepancies between the GLM and GLMM RR estimates. The GLMM estimates approximate the conditional association, while the Poisson GLM estimates the marginal association. The GEE estimates fall between the true marginal and conditional, although closer to the marginal.

In Simulation 4, we also fit INE models, including both an individual and neighborhood fixed effect for racialized social group as in equation (5), and assess the agreement between models (Figure 4). Note that because the INE model specification is not the true data generating model, we do not indicate true parameter values in the figure. The results show that the GLM, GLMM, and GEE estimates now largely agree.

6. Analysis of Massachusetts Premature Mortality Data

We now illustrate this phenomenon by investigating Black vs. WNH inequities in premature mortality in MA using the data described in Section 3. We also conduct

secondary analyses restricted to Boston, where a large portion of MA's Black population resides. The GLMs and GLMMs fit to these data are specified as in equations (1) and (2), respectively, using both Poisson and negative binomial likelihoods. The GEE models use the GLM mean structure with a Poisson-like quasi-likelihood and exchangeable working correlation structure within CTs. The INE models build on the standard models by adding CT proportion Black (centered and scaled) as a covariate. Each model is fit to the CT data for MA and, separately, to the Boston CTs only. All analyses were performed in R (R package citations in the Supplementary Materials).

The MA results mimic the findings from simulated data; i.e., the standard GLMs suggests that members of the Black compared to WNH population experience substantially higher rates of premature mortality, while the standard GLMMs find the opposite (Table 2). The GEE results fall between the GLM and GLMM results, though closer to those of the GLM. In contrast, the results of the INE GLMs, GLMMs, and GEE generally agree. Specifically, they suggest that CT proportion Black is associated with higher rates of premature mortality after conditioning on individual Black racialized social group membership, while individual Black racialized social group membership is associated with lower premature mortality risk conditional on CT proportion Black.

Although Boston is more diverse than MA as a whole, Boston's CTs are highly segregated in relation to the Black and WNH populations (Figure 2). In the analyses restricted to Boston (Table 2), inference from the standard GLMs and GLMMs agree that members of the Black population experience higher rates of premature mortality. However, the GLMM point estimates are substantially smaller than the GLM estimates, suggesting that endogeneity may still be impacting these analyses. Additionally, most INE models for Boston indicate that after conditioning on CT proportion Black, individuals categorized as Black have a higher risk of premature mortality, in contrast to the findings for MA. Hence, in Boston, both individual-level measures of membership in the Black population and neighborhood proportion Black contribute jointly to racialized inequities in premature mortality.

Beyond exemplifying offset endogeneity, our results, particularly from the INE models, offer novel substantive insights. First, they suggest that for MA in 2008-2012, the increased premature mortality risk for Black populations is explained mainly by area-level racialized social group composition, with individual-level Black race appearing to have a null or even protective association with mortality after adjusting for CT-level proportion Black. When analyzing Boston alone, these trends reverse, with individual-level membership in the Black racialized social group being equally or more important for explaining increased premature mortality risk as CT racialized social group composition. This geographic heterogeneity in associations further emphasizes the importance of accounting for space and context in social epidemiologic studies.

7. Discussion

In this paper, we report that the commonly co-occurring conditions of racialized residential segregation and increased baseline health risks in neighborhoods whose residents are predominately people of color can lead to divergent results in GLM and GLMM approaches often used to study racialized health inequities. This divergence occurs due to offset endogeneity, i.e., endogeneity induced by correlation between the offset (typically a measure of stratified population size) and the random intercept in the GLMM, and can occur even for models with a log link, for which the literature largely notes that marginal and conditional parameters coincide. In this case, regression coefficients in the log-linear GLM have conditional interpretations, while the GLM

coefficients represent marginal associations. We show that explicitly modeling separate, conditional associations between individual membership in racialized social groups and health outcomes, and between neighborhood racialized social group composition and health outcomes, may reduce offset endogeneity issues and may provide important insights into factors driving health inequities.

Critically, our findings suggest that the ability to estimate marginal association parameters, one of the most appealing features of log-linear GLMMs popular for disease mapping, may often be compromised in the presence of highly segregated study regions and heterogeneous area-level rates that are correlated with the area's social group composition. In practice, health inequity estimates from standard GLMMs stratified by racialized social group may reflect inequities after conditioning on neighborhood, effectively comparing mortality/morbidity across groups living in the same neighborhood. Moreover, we have shown that the combination of racialized residential segregation and increased baseline health risks in predominately Black neighborhoods results in GLMM effect estimates that are typically lower than their marginal counterparts. Thus, past analyses that estimated racialized health inequities using GLMMs may have underestimated the magnitude of marginal health inequities.

The epidemiologic literature is replete with cautions about the potential for ecologic bias when interpreting the effect of an area-level measure in the absence of control for its individual counterpart. Our results emphasize that the reverse is also true: estimates of associations with individual-level membership in social groups should also consider area-level social context to avoid the "individualistic fallacy." This observation, while previously noted in the social epidemiology literature, is relatively underappreciated (Diez-Roux 1998; Firebaugh 1978; Pearce 2000; Subramanian et al. 2009).

On the basis of this literature and our findings here, we recommend use of the INE model we have proposed in future small-area disease mapping for inequity studies, whether the social group under study are defined in relation to racialized groups, economic groups, or other social groups reflecting inequitable social relationships (Beckfield 2018; Krieger 2020). While the INE model is simple and straightforward, more complex GLMM specifications such as the shared component model, which allows for separate spatially correlated random effects across groups (Kiang et al. 2019), may also be suitable alternative approaches for dealing with offset endogeneity. We also note that studies of inequities in other fields—including sociology, economics, education, crime and criminal justice, policy, urban planning, and more—frequently utilize log-linear mixed model formulations similar to those presented here and include population denominators as offsets (see references to many such studies in the Supplementary Materials). This suggests that the offset endogeneity issue is likely to be relevant to the broader scientific community and that adoption of INE-type model specifications as standard practice may be advisable in inequity studies across fields.

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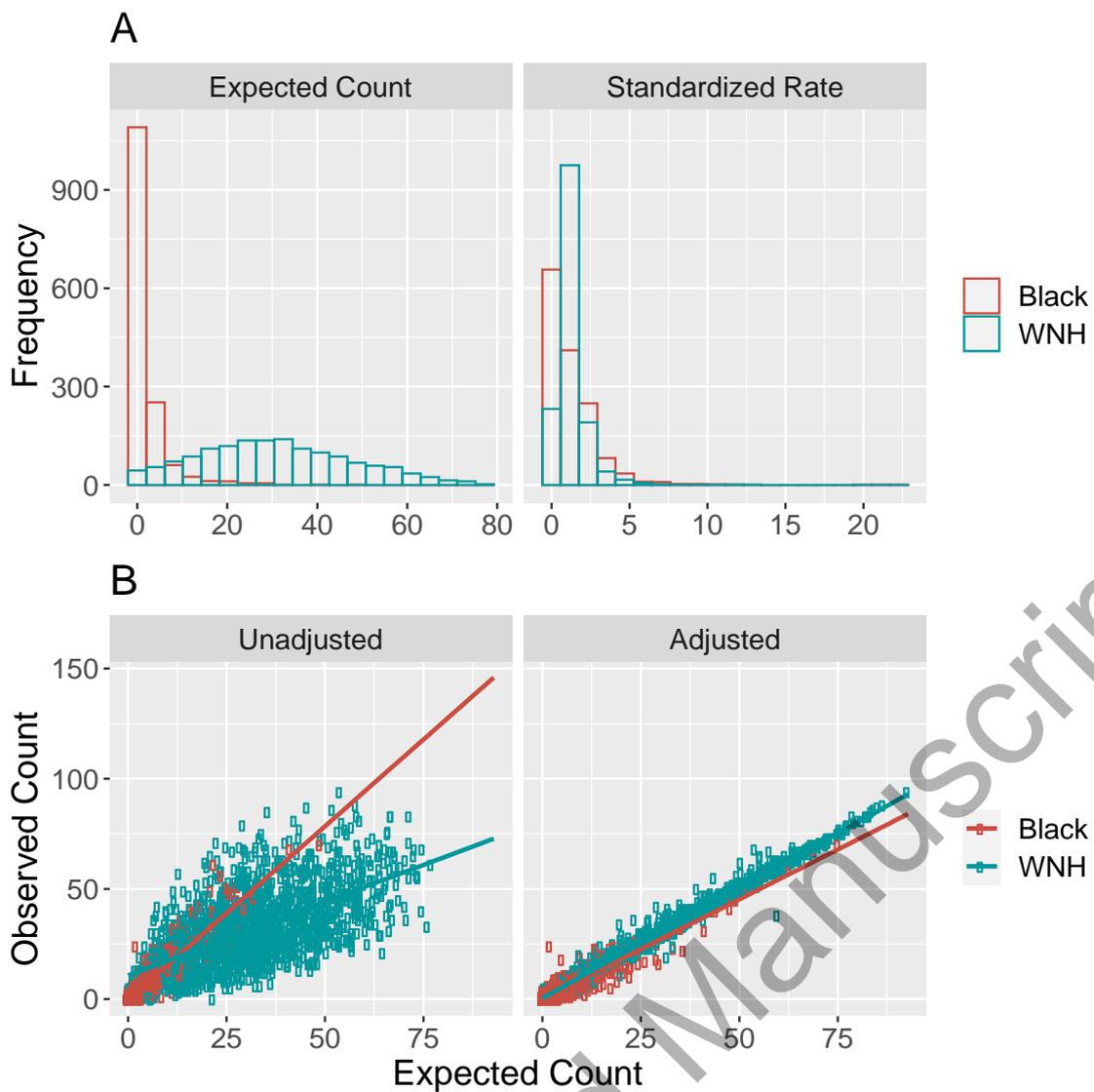
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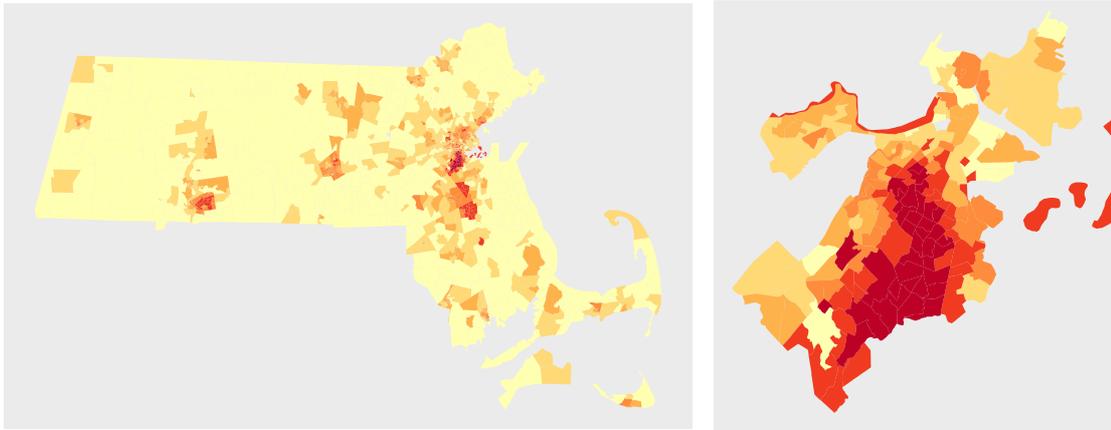
Table 1. Health inequities terminology

Term	Definition
Health inequity	Unjust, unnecessary, and in principle preventable differences in health status across social groups (Beckfield 2018; Braveman and Gruskin 2003; Krieger 2020).
Privileged vs. marginalized groups	Social group classifications for groups co-defined by unjust power relations, with these terms emphasizing the relative power and agency (or denial thereof) of the groups arising due to unfair social, economic, and political structures (Benjamin 2017; Krieger 2020, 2021).
Racialized social group	Precise terminology that clarifies the category of “race” is socially (rather than biologically) defined and is not an innate individual “property” or trait (Benjamin 2017; Krieger 2020, 2021).

Table 2. Fixed effect estimates (95% CI) from MA and Boston-only premature mortality models, years 2008-2012. Abbreviations: INE model= Individual and neighborhood inequities model; RR= rate ratio; CT= census tract; P-GLM= Poisson generalized linear model; P-GLMM= Poisson generalized linear mixed model; NB-GLM= negative binomial generalized linear model; NB-GLMM= negative binomial generalized linear mixed model; GEE= generalized estimating equations.

Massachusetts						
		P-GLM	P-GLMM	NB-GLM	NB-GLMM	GEE
Standard Model	Intercept	1.02 (1.01,1.03)	1.03 (1.00,1.06)	1.15 (1.12,1.18)	1.03 (1.01,1.06)	1.02 (0.99,1.04)
	Black RR	1.37 (1.33,1.42)	0.92 (0.88,0.95)	1.13 (1.07,1.19)	0.93 (0.89,0.96)	1.26 (1.19,1.33)
INE Model	Intercept	1.06 (1.05,1.07)	1.04 (1.02,1.07)	1.15 (1.12,1.18)	1.04 (1.02,1.07)	1.06 (1.03,1.08)
	Individual Black RR	0.89 (0.85,0.92)	0.85 (0.82,0.88)	0.98 (0.93,1.04)	0.86 (0.82,0.89)	0.82 (0.76,0.87)
	CT Proportion Black RR	1.20 (1.19,1.22)	1.26 (1.23,1.29)	1.22 (1.2,1.25)	1.25 (1.22,1.29)	1.22 (1.19,1.25)
Boston						
		P-GLM	P-GLMM	NB-GLM	NB-GLMM	GEE
Standard Model	Intercept	1.17 (1.12,1.21)	1.16 (1.07,1.26)	1.32 (1.21,1.45)	1.17 (1.08,1.26)	1.16 (1.06,1.28)
	Black RR	1.52 (1.43,1.61)	1.22 (1.12,1.32)	1.36 (1.19,1.56)	1.24 (1.14,1.35)	1.51 (1.35,1.69)
INE Model	Intercept	1.14 (1.09,1.18)	1.04 (0.95,1.13)	1.19 (1.09,1.3)	1.04 (0.96,1.13)	1.13 (1.03,1.24)
	Individual Black RR	1.21 (1.11,1.31)	1.11 (1.02,1.21)	1.22 (1.06,1.39)	1.13 (1.03,1.24)	1.20 (1.05,1.39)
	CT Proportion Black RR	1.07 (1.05,1.09)	1.12 (1.08,1.16)	1.11 (1.07,1.14)	1.12 (1.08,1.16)	1.07 (1.04,1.10)





Percent Black

■ <2	■ [5,10)	■ [20,50)
■ [2,5)	■ [10,20)	■ 50+

Figure 2. CT percent Black for MA (left) and Boston (right), 2008-2012.

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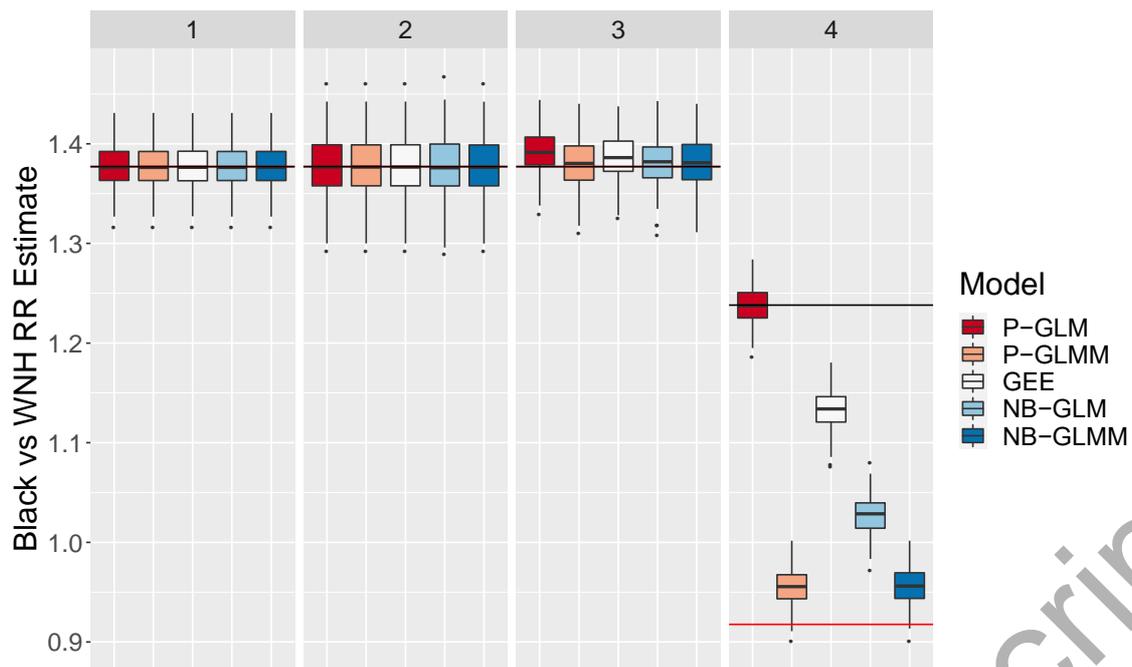


Figure 3. Simulation Results. Black vs. WNH RR estimates from the Poisson GLM and GLMM, the GEE, and the negative binomial GLM and GLMM fit to simulated data for each of the four simulation scenarios. True marginal RR used to generate the data is indicated by the black horizontal line and true conditional RR is indicated by the red horizontal line.

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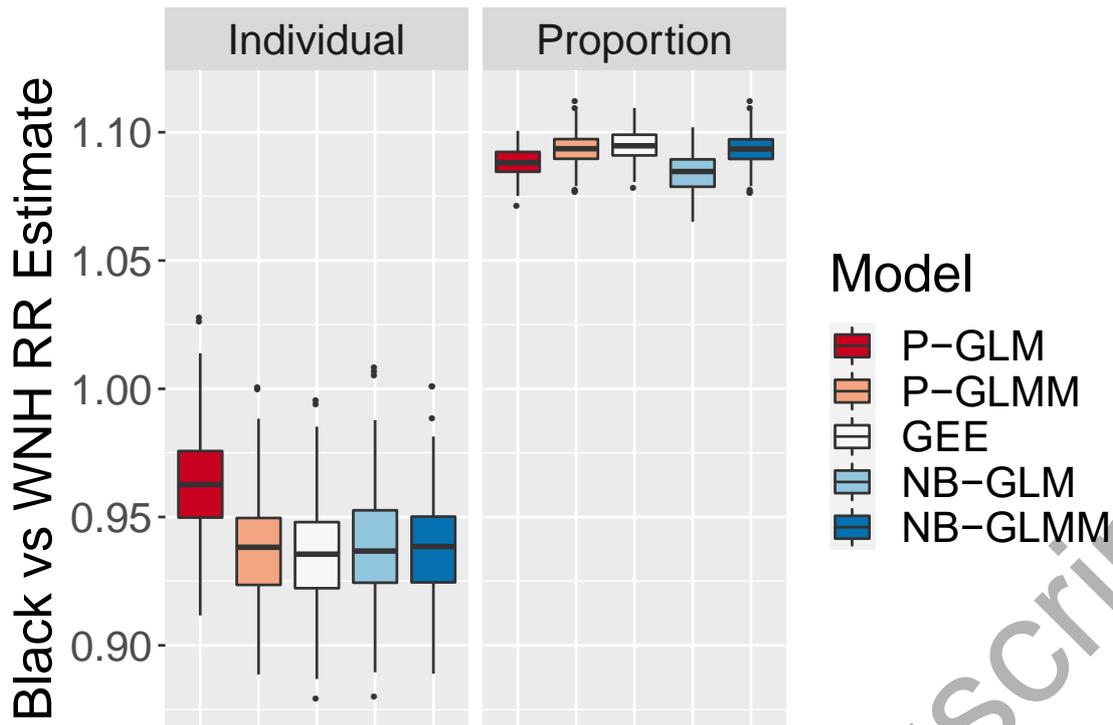


Figure 4. Simulation Results. RR estimates from the INE model specification of the Poisson GLM and GLMM, the GEE, and the negative binomial GLM and GLMM fit to synthetic data from Simulation 4. The left panel shows estimates of the RRs for the individual Black indicator and the right panel shows estimates of the RRs for CT proportion Black.

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