G-Computation for Decomposing Population Health Disparities: 3-way Effect Decomposition with A New R Package *multmed*[*]

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Abstract

A central aim in demographic analysis is explaining the source of population disparities in health and mortality. However, conventional decomposition techniques are limited in handling high dimensional data from survey samples, accounting for time-varying confounding, and lack a clear counterfactual interpretation. Recent studies have shown how population decomposition can be more flexibly constructed as a causal mediation analysis. This paper extends previous work by demonstrating a flexible framework for G-computation that can handle 1) survey weights, 2) time-varying confounders, 3) multiple mediators, 4) repeated observations, and 5) VanderWeele's three-way effect decomposition given treatment-mediator interactions (CDE, PAI, PIE). We discuss how each decomposed effect relates to a distinct and theoretically important counterfactual by using a worked decomposition example of a Black-white disparity in cardio-metabolic risk. Last, we provide a new R package, *multmed*, to easily specify DAGs and efficiently estimate total and decomposed effects (rNDE, rNIE, CDE, PAI, PIE) using this g-computation framework.

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1 Introduction

Formal demography offers a rigorous mathematical framework for modelling the dynamic ways in which populations shift over time, which more recently has included advanced stochastic and Bayesian projection techniques (Raftery et al. 2012; Schmertmann and Gonzaga 2018). However, many research and policy questions relate to *why* disparities exist across populations, and particularly the social and economic explanatory drivers. Common mathematical methods in demography and econometrics include Kitagawa (1955) decomposition, Das Gupta (1993) decomposition, and Oaxaca-Blinder (1973) decomposition. These methods are widely used, but limited when applied to survey samples, high dimensional data (i.e., continuous variables or multiple groups), and settings where components are reciprocally intertwined (time-varying confounding). There have been recent developments in simulation and multistate life table frameworks, but decomposition is logistically difficult and, perhaps most importantly, these decompositions still do not have a clear counterfactual interpretation (Jackson & VanderWeele 2018, Sudharsanan & Bijlsma 2020).

As demonstrated by Jackson & VanderWeele (2018) and extended by Lundberg (2019) and Sudharsanan & Bijlsma (2020), demographic decompositions of a population disparity can be framed as a causal mediation analysis. In discussing applications to questions of intersectional health disparities, Bauer & Scheim (2019) and Jackson & VanderWeele (2019) discuss how these techniques can account for the complex patterns of time-varying confounding and interaction necessary for examining a dynamic social *process* over time, such as the ways in which complex, reciprocal social exposures influence individuals within the life course and across generations.

There are several difficulties and limitations that result in less uptake in these methods across demography for decomposing population disparities:

1. **Estimation limitations in mediation methods:** Only until recently have studies started to address problems about multiple mediators and repeated observations (Bijlsma et al. 2019, Zhou & Yamamoto 2020). Yet, most of these work has not incorporated survey design weights or flexible 2-way and 3-way effect decompositions when there are treatment-mediator interactions in the outcome model (Wang & Arah 2015, Zhou & Wodtke 2019, Jackson & VanderWeele 2019).

2. **Difficulties with interpretation:** How to interpret each effect estimates in 2-

way and 3-way effect decomposition? How can we translate methodological terminology into substantive research questions in demography and sociology, such as in explaining population disparities over time?

3. **Implementation limitations:** It is not easy to implement causal decomposition methods with existing R software. The package *paths* allows for multiple mediators, but requires no time-varying confounders, all time-varying mediators to be measured in all waves, and no survey weights (Zhou & Yamamoto 2020). The package *rwrmed* allows for time-varying confounding, weights and 2-way/4-way decomposition, but not multiple mediators or repeated observations (Zhou & Wodtke 2019).

This paper demonstrates a flexible framework for g-computation that can handle 1) survey weights, 2) time-varying confounders, 3) multiple, especially high-dimensional, mediators, 4) repeated observations or measurements, and 5) effect decomposition (VanderWeele's 2-way and 3-way) given treatment-mediator interactions. We describe how each decomposed effect relates to a distinct and theoretically useful counterfactual with a worked example of a Black-white disparity in cardio-metabolic risk from the The National Longitudinal Study of Adolescent to Adult Health (Add Health). Last, we describe a new R package to easily specify DAGs and efficiently estimate total and decomposed effects using this g-computation framework.

2 Common decomposition settings

There are three settings related to decomposing a population disparity for which g-computation is particularly well-suited, and all three are extremely common in demography and sociology. We illustrate them in Figures 1–3.

2.1 Unordered Multiple Mediators

The first is Kitagawa-Oaxaca-Blinder decomposition (Figure 1), where we are interested in decomposing differences in Y between levels of X that are attributable to 1) differences in composition across independent mediators and 2) differences in their "returns" (or effects) by X. However, these terms are often interpreted causally, assuming no confounding and independent mediators over time.

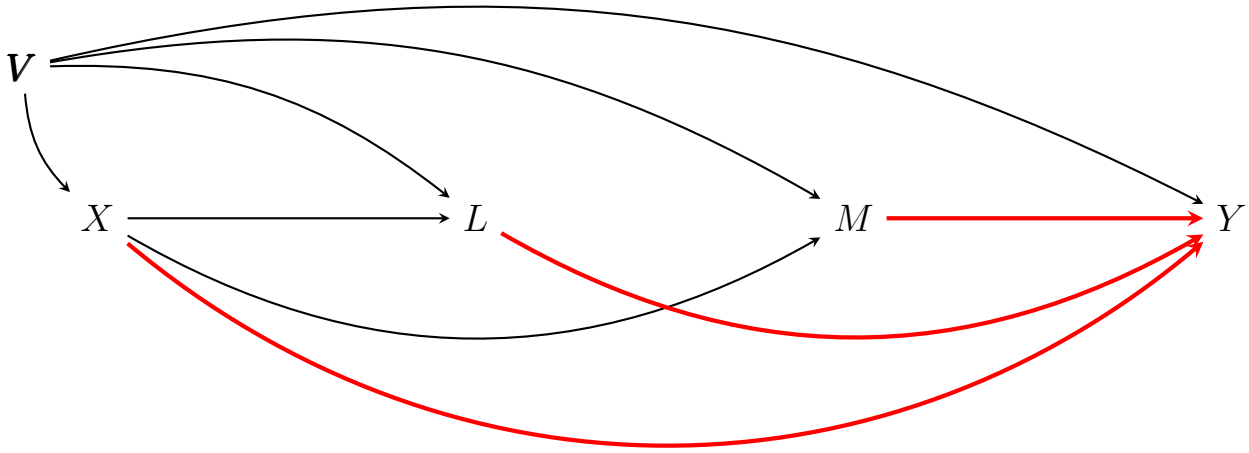


Figure 1: DAG representing connection between X and Y with any number of independent mediators (L, M, etc.). Effects of mediators on the outcome may include treatment interactions (Kitagawa-Oaxaca-Blinder decomposition).

2.2 Causally Ordered Multiple Mediators

In Figure 2, we consider cases where we have multiple mediators (M, L) that may explain differences in Y across groups X, and these mediators are causally ordered. In causal mediation settings in terms of a single mediator, M, L is considered a "treatment-induced mediator-outcome confounder." But L itself also serves as a mediator in decomposing the total effect of any change in X on Y.

If we consider that there is an interaction between X and M in affecting Y, we can consider three distinct pathways through which the total effect, $E[Y|X = x]$ - $E[Y|X = x^*]$ operates. First is unobserved mediation, or the proportion of the total disparity that is not explained by observed mediators (i.e. the controlled direct effect, or CDE, $X \rightarrow Y$). Second is the interactive effect via each mediator (i.e. the proportion attributable to interaction, or PAI, via $X \rightarrow M|X = x \rightarrow Y|X = x$ and $X \rightarrow L|X = x \rightarrow Y|X = x$). Last is the main effect via each mediator (e.g. the pure indirect effect, or PIE, via $X \rightarrow M|X = x \rightarrow Y|X = x^*$ and $X \rightarrow L|X = x \rightarrow Y|X = x^*$) (VanderWeele 2014, Naimi et al. 2016). In others words, there are two ways through which M might "explain" some portion of the total disparity in Y across levels of X, which is the

intuition behind examining differences in compositions vs. effects in Oaxaca-Blinder decomposition.

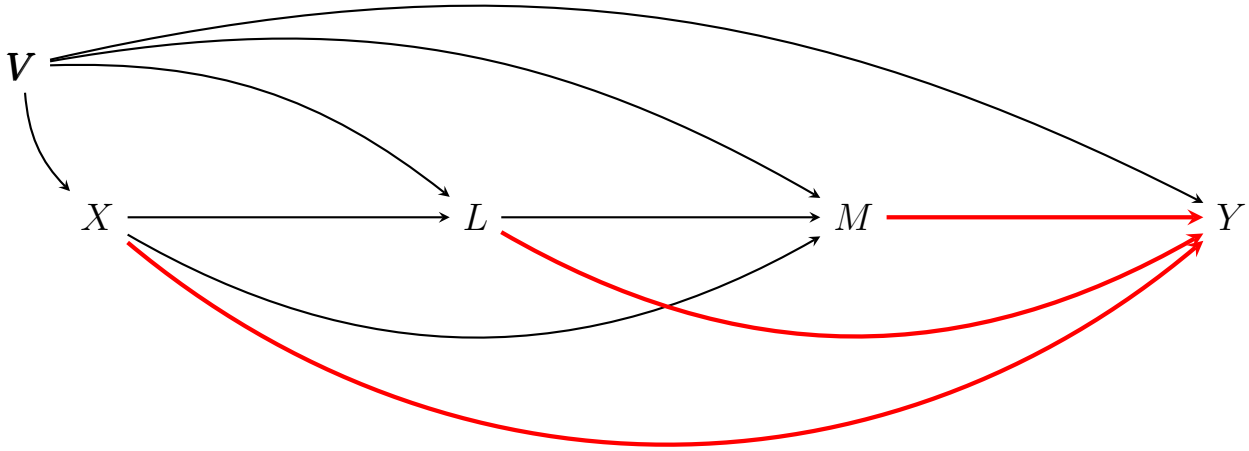


Figure 2: DAG representing connection between X and Y with any number of causally ordered dependent mediators (L, M, etc.). Effects of mediators on the outcome may include treatment interactions.

2.3 Time-Varying Multiple Mediators

In Figure 2, we are considering two dependent mediators (L, M), but we could incorporate any number of mediators to this DAG and consider a PAI or PIE for each, accounting for confounding via previous variables. But another very common situation in explaining population disparities is having repeated measures over the same treatment, mediators, and outcome over time. This situation could be constructed in a DAG as in Figure 2 by considering each ordered mediator over time (L_1, M_1, M_2, L_2, ...) but can more compactly be described by the DAG in Figure 3 describing data collected over many ages or time periods, t. For example, this DAG may be a relationship between X and Y over time in longitudinal data such as the Panel Study of Income Dynamics (PSID), The National Longitudinal Study of Adolescent to Adult Health (Add Health), or the Fragile Families & Child Wellbeing Study (FFCWS) cohorts.

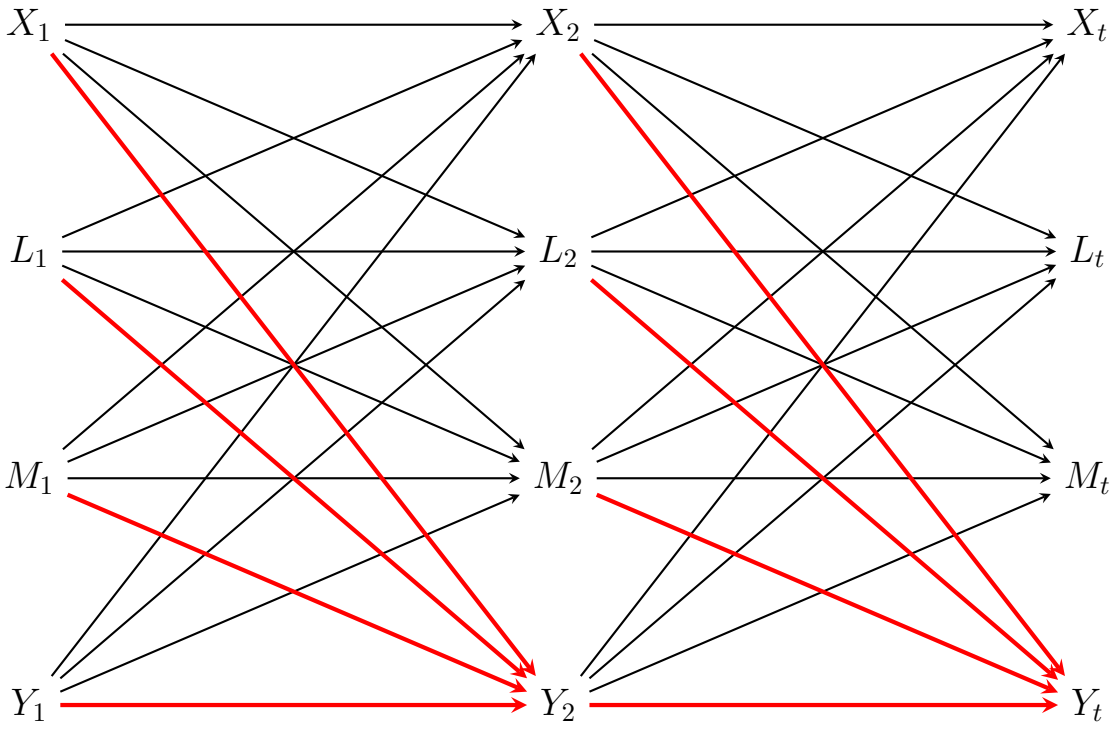


Figure 3: DAG representing connection between X and Y with any number of causally ordered dependent mediators (L, M, etc.) over t repeated observations. Models for the outcome and all mediators can be panel models, such as fixed or mixed effects models. Effects of mediators on the outcome may include treatment interactions. Time-invariant or intermediate confounders $\boldsymbol{V}$ suppressed for visual clarity.

The three settings in Figures 1-3 describe many of the empirical settings in which we would like to decompose a population disparity across levels of X via some explanatory components, or mediators. As discussed above, nonparametric demographic decomposition methods have difficulty adjusting for confounding over time, especially with high dimensional data from survey samples, and thus can provide very biased estimates of the "proportion explained" by a given component in a causal, counterfactual sense (Jackson & VanderWeele 2018, Sudharsanan & Bijlsma 2020). In addition, linear decomposition methods based on regression techniques do not account for time-varying confounding (Robins & Hernan 2009, Naimi et al. 2016, Jackson & VanderWeele 2018). Methods to address these issues in the causal inference literature include marginal structural models and G-computation (Naimi et al. 2016), but in their application to causal mediation all suffer from some key limitations discussed below.

3 G-computation for 2-way and 3-way Decomposition

Building on the methods of Jackson & VanderWeele (2018), Lundberg (2019), Bijlsma et al. (2019), and Sudharsanan & Bijlsma (2020), we provide a flexible parametric method based on g-computation to estimate rNIE, rNDE, rPSE, CDE, PAI, and PIE with any number of dependent or repeated mediators in the presence of treatment-induced confounders. We use X as shorthand for $X = x$ for all variables below.

- Y = outcome
- X = treatment (x = index value, x^* = reference value)
- M = mediator 2 (m = index value, m^* = reference value)
- L = mediator 1 (l = index value, l^* = reference value)
- $\boldsymbol{V}$ = pre-treatment confounders (or post-treatment confounders not affected by treatment)

We can define the expectation of Y using the g-formula:

$$
\begin{aligned}
E[Y] = \sum_{y}\sum_{x}\sum_{m}\sum_{l}\sum_{v}\Big\{ & y{\cdot}P(Y|\boldsymbol{V}, X\cdot M, X\cdot L){\cdot} \\
& P(M|\boldsymbol{V}, X\cdot L){\cdot} \\
& P(L|\boldsymbol{V}, X){\cdot} \\
& P(X|\boldsymbol{V}){\cdot} \\
& P(\boldsymbol{V})\Big\}
\end{aligned}
$$

When considering mediating effects via M, L is a treatment-induced mediator-outcome confounder. But L also serves as a mediator in decomposing the total effect of any change in X. It is straightforward in the g-formula above to insert any number of k additional ordered mediators, which introduces more effect pathways and more instances

of treatment-induced mediator-outcome confounding in estimating each of those effect pathways. For this reason, we consider all post-treatment variables influenced in any way by the treatment to be *mediators*, recognizing that each of these also *confounds* the estimation of effect pathways via *other downstream mediators*. We may also observe subsequent values of X, which are affected by previous values of X and other mediators or treatment-induced confounders.

We provide an additive decomposition of the total effect, $E[Y|X = x]-E[Y|X = x^*]$, into either 1) one rNDE and one rNIE for each mediator or 2) one CDE (relative to the reference value of all mediators that have an interactive effect on the outcome depending on treatment), one PAI for each mediator, and one PIE for each mediator. Wodtke & Zhou (2020) provide the same decomposition, but for only a single mediator. Our decomposition is an extension of Wang & Arah (2015) to incorporate multiple mediators. Daniel et al. (2015) describe a similar decomposition for multiple mediators, but do not include a component attributable to interaction in their decomposition.

$$\text{rNDE} = E[Y_{xM_{x^*}}] - E[Y_{x^*M_{x^*}}]$$

$$\text{rNIE} = E[Y_{xM_x}] - E[Y_{xM_{x^*}}]$$

$$\text{CDE}_{M=m^*,L=l^*} = E[Y_{xm^*l^*}] - E[Y_{x^*m^*l^*}]$$

$$\text{PAI}^{(M)} = E[(Y_{xml^*} - Y_{x^*ml^*} - Y_{xm^*l^*} + Y_{x^*m^*l^*})(M_x)]$$

$$\text{PIE}^{(M)} = E[Y_{x^*M_xl^*}] - E[Y_{x^*M_{x^*}l^*}]$$

Estimation is achieved by calculating all conditional probabilities in the g-formula above, and then simulating from these probabilities and calculating probabilistic draws of all desired effects. The simplest method is fitting a GLM for the outcome and each post-treatment variable in the g-formula above. For example, we specify the following parametric models to represent the data-generating process:

$$Y = \beta_0 + \beta_1 M + \beta_2 L + \beta_3 V + \beta_4 X + (M \cdot X) + \beta_5 (L \cdot X) + \epsilon_Y$$

$$M = \beta_0 + \beta_1 L + \beta_2 V + \beta_3 X + \beta_4 (L \cdot X) + \epsilon_M$$

$$L = \beta_0 + \beta_1 V + \beta_2 X + \epsilon_L$$

In low-dimensional settings, such as a single binary mediator and binary outcome, all necessary conditional probabilities can be derived from empirical population proportions. However, this quickly becomes difficult in settings using data from samples rather than full populations or with many mediators or confounders, especially if any are continuous. The conditional expectations of M and L can be estimated using any parametric or non-parametric approach (e.g. BART). The same is true of the conditional expectation of Y when calculating rNIE, rNDE, or rPSE. But for calculating the CDE, PAI, and PIE, the conditional expectation of Y must come from a collapsible model (VanderWeele 2014, Wang & Arah 2015).

4 Implementation

We implement the aforementioned decomposition methods using the following algorithms:

1. If data comes from a complex survey, create a replicate dataset (*survey* R package)
2. Fill in missing values due to item non-response using chained equations (*mice* R package); e.g., 30 imputed datasets.
3. Create a probabilistic draw of the parameters defining the conditional probabilities by either 1) fitting models using a single bootstrap of the data or 2) sampling from the models fitted to the entire data, such as drawing a single random multivariate-normal sample of parameters from all GLMs above using the fitted coefficients and variance-covariance matrices (e.g. "parametric bootstrap").

4. Create n replicates of the data (e.g., $n = 30$) to remove Monte Carlo errors arising from stochastic individual-level response prediction (e.g. for binomial or categorical variables) from Step 2.

5. Simulate the necessary control and treatment regimes by predicting data forward through the g-formula using the parameter samples obtained above. This step gives us a draw from the full probability density of Y, M, and L under each treatment regime.

6. Use simulated data values from Step 5 to calculate all the conditional expectations necessary to estimate the effect of interest above (e.g. rNDE). The result is one draw from the full probability density of that effect.

7. Repeat Steps 1–6 as many times as necessary (e.g., 1000 times) to produce the full probability density of the desired effect. We can then summarize this distribution using different statistics measures (e.g. mean and 95% intervals for each effect).

5 Example: Decomposing Black-white Disparity in Cardio-Metabolic Risk

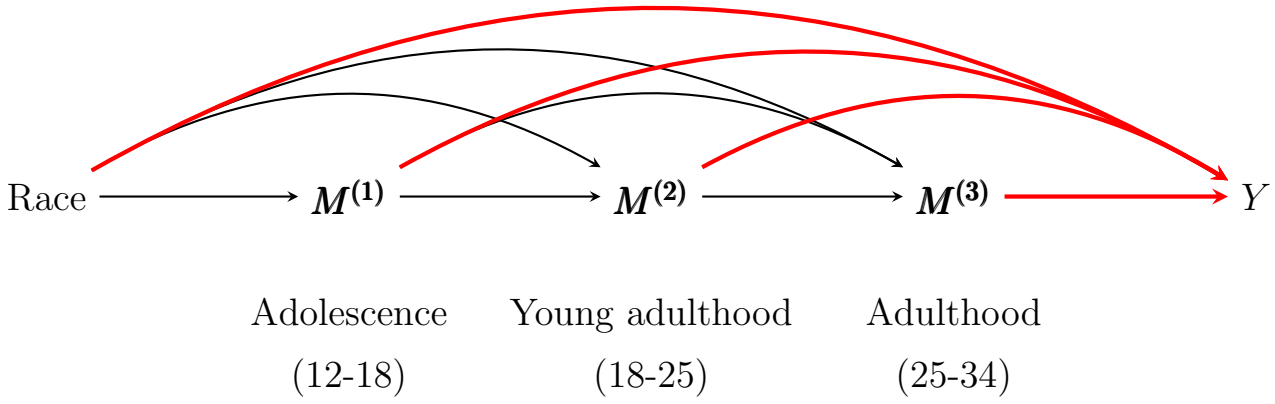
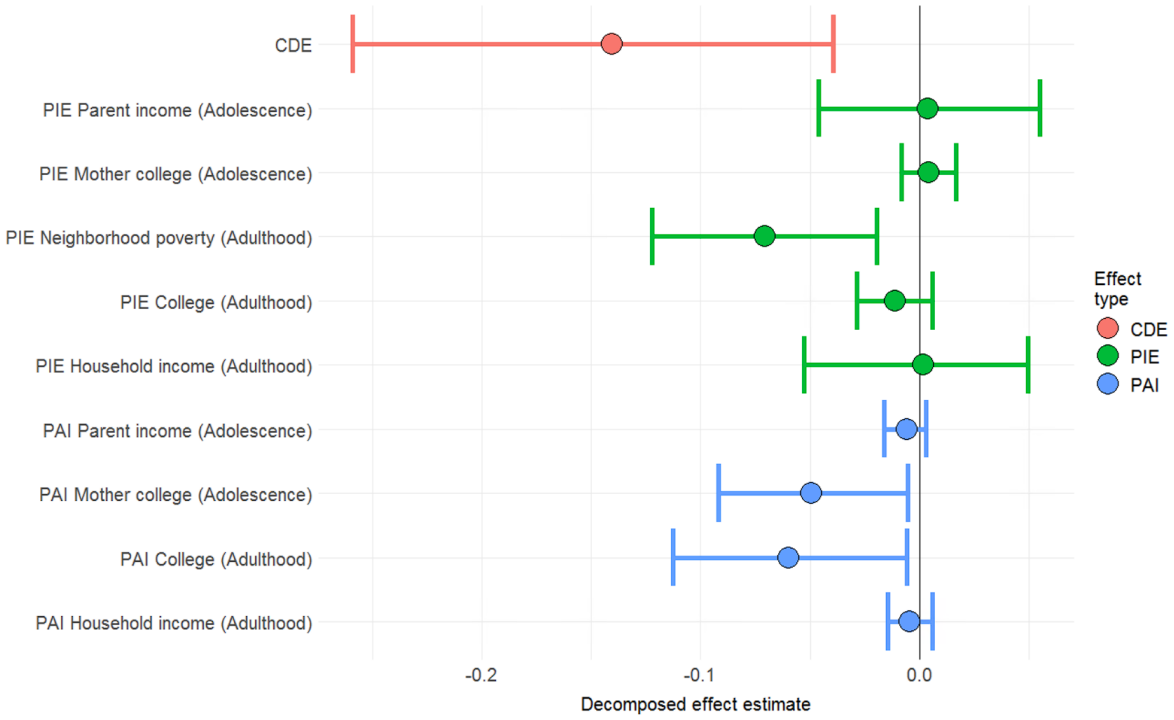


Figure 4: Directed acyclic graph describing the theorized causal model with the data observed in a prospective cohort study.



6 R package for Multiple Mediation Analyses: *multmed*

The following section describes the inputs necessary to estimate 2-way and 3-way effect decomposition using the R package *multmed*. The user inputs required from the user include

1. **data** = A data.table with required columns "id" and "pweight" (which can just all be 1 is data are unweighted). This dataset will be wide on pre-treatment confounders, treatment, mediators, and outcome. If a treatment or mediator variable is observed more than once over time, use separate columns (i.e., x_1, m_2, x_2, m_3, x_3, etc.). Using models pooled over time (e.g. a single model for m at all steps using lagged predictors) requires that m and predictors at each step are named _(t). More on this below. Otherwise, there are no naming requirements.

2. **path_cb** = A data.table specifying the complete DAG (see examples).

3. **intervention_rules** = A named list of functions that apply the desired intervention(s) to the data.

4. **decomp_compare_df** = A string indicating the name of the control simulation

course to which every other intervention simulation will be compared. This must be one of the names in intervention_rules.

5. **decomp_paths** = A named list of effect pathways. Names can include any simulations from intervention_rules that you want to compare to **decomp_compare_df** to calculate a total effect and all decomposed effects of the intervention. Each named item in this list is itself a list of length=2, with items named `paths` and `outcomes`. Each of these items is itself a list, where `paths` is a list of string vectors with the names of mediators in that pathway. All string vectors taken together in `paths` must be mutually exclusive and exhaustive of all mediators in the specified DAG.

This package relies on three primary functions, and *g*-formulas can be estimated in parallel:

- `multmed_glms(path_cb, data)`
- `multmed_gformula(path_cb, data, models, intervention_rules, decomp_paths, ...)`
- `multmed_decomp(decomp_paths, decomp_type, ...)`

7 References

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