A Causality-based Graphical Test to obtain an Optimal Blocking Set for Randomized Experiments

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Abstract

Randomized experiments are often performed to study the causal effects of interest. Blocking is a technique to precisely estimate the causal effects when the experimental material is not homogeneous. We formalize the problem of obtaining a statistically optimal set of covariates to be used to create blocks while performing a randomized experiment. We provide a graphical test to obtain such a set for a general semi-Markovian causal model. We also propose and provide ideas towards solving a more general problem of obtaining an optimal blocking set that considers both the statistical and economic costs of blocking.

1 Introduction

Studying the causal effect of some variable(s) on the other variable(s) is of common interest in social sciences, computer science, and statistics. However, a mistake that people usually make is, confusing the causal effect with an associational effect. For instance, if high levels of bad cholesterol and presence of a heart disease are observed at the same time, it doesn’t mean that the heart disease is caused by the high levels of bad cholesterol. The question is then how do we get to know if at all a variable causes the other? If the answer is yes, then what is the direction (positive or negative) and what is the magnitude, of the causal effect?

Fisher (1992) provided the framework of randomized experiments to study the causal effect, where the variable whose causal effect is to be studied also known as treatment or cause, is randomized over the available experimental material like humans, rats, agricultural plots etc. and changes in the variable on which the causal effect is to be studied also known as response or effect, are recorded. A statistical comparison of values of the response with or without the treatment can therefore be done to study the existence, direction and magnitude of the cause-effect relationship of interest.

Randomized experiments work on three basic principles viz. randomization, replication, and local control. Randomization states that the assignment of the treatment has to be random, replication states the treatment should be given to multiple but homogeneous units, i.e. there are multiple observations of the effect variable for both with and without the treatment. Hence, as long as the entire experimental material is homogeneous for instance, the fertility of all the agricultural plots is same, the responsiveness of all the humans is same for the drug, etc. then a ‘good’ randomized experiment can be carried out using the first two principles viz. randomization and replication, which gives rise to something called completely randomized design (CRD).

But the cases when the entire experimental material is not homogeneous, i.e. some attributes of experimental units also known as covariates differ from each other, then the causal effect may get influenced by the covariates causing non-homogeneity like fertility, responsiveness etc. The remedy

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to this problem is the third principle of randomized experiments, i.e. local control or also known as blocking, which states to stratify the entire experimental material based on that covariate(s) causing non-homogeneity, and then randomize the treatment within those strata to eliminate the effect of the covariates. These strata are called blocks, and this third principle along with the first two give rise to something called randomized block design (RBD).

Note that blocking tries to control/eliminate the variability in the response attributed through the covariates. This reduces sources of variability and thus leads to greater 'precision'. Precision is defined as inverse of the variability which measures how much the response would have varied if the sample would have changed. In the context of estimating the causal effects, precision is nothing but the inverse of the variance of the estimate of the causal effect.

Statistics literature (Yates (1935), Kempthorne (1952), Kempthorne (1955), Cochran and Cox (1948)) presents profound discussions on relative efficiency (ratio of variances) of RBD over CRD. It can be seen that in general, RBD is always more or at least as efficient as CRD. Hence, the natural question is how do we do blocking in an intelligent manner such that maximum gain in precision can be attained, i.e. variability in the response can be controlled to the maximum possible extent? More specifically, we are interested in knowing which all covariates should be used to create blocks? Intuitively, we can say that we need not take the ones into consideration which remain constant in the population. But, among the ones which vary, can we choose some over the other, using some criterion? Most of the blocking schemes discussed in the literature discuss about creating blocks by grouping the experimental units which are close to each other based on some attributes. For instance, threshold blocking (Higgins et al. (2016)).

The question that we are interested in answering is, can we decide which covariates to use for creating blocks using the causal structure (diagram) of the all the variables viz. treatment, response and covariates (observed and unobserved), provided the causal structure is given?

The main contributions of this paper can be summarized as follows.

1. We formalize the problem of obtaining a statistically optimal set of covariates to be used to create blocks in randomized experiments.
2. We provide a graphical test to obtain such a set for a general semi-Markovian causal model.
3. We also propose and provide ideas towards solving a more general problem of obtaining an optimal blocking set that considers both the statistical and economic costs of blocking.

2 Definitions and Problem Formulation

2.1 Causal Effect Definitions and Estimation

For all the discussions, we consider a structural causal model \( M = \{U, V, F\} \), where \( U \) is the set of exogenous variables, \( V \) is the set of endogenous variables and \( F \) is the set of functions controlling the causal structure.

We are interested in studying the causal effect of a treatment \( (X) \) on some response \( (Y) \) by performing a randomized experiments over \( n \) experimental units. We define the following.

\[
X_i = \begin{cases} 
1, & \text{if i}^\text{th} \text{ unit receives the treatment,} \\
0, & \text{if i}^\text{th} \text{ unit is under control, i.e. doesn’t receive the treatment.}
\end{cases}
\]

\[
Y_{i1} = Y_i \text{ if } X_i = 1, \text{ i.e. response for the i}^\text{th} \text{ unit when it receives the treatment},
\]

\[
Y_{i0} = Y_i \text{ if } X_i = 0, \text{ i.e. response for the i}^\text{th} \text{ unit when it is under control.}
\]

Rosenbaum and Rubin (1983) has termed \( Y_{i1} \) and \( Y_{i0} \) as potential outcomes for the \( i^\text{th} \) unit when it receives the treatment and when it is under control, respectively. For the \( i^\text{th} \) unit we can define the causal effect of the treatment as the difference between the potential outcomes under treatment and control, i.e. \( Y_{i1} - Y_{i0} \). Note that individual treatment effects cannot be calculated as we only observe \( X_i \) and \( Y_i = X_i Y_{i1} + (1 - X_i) Y_{i0} \) (Holland (1986)), which is due to the fact that we don’t assign treatment to all the units rather we randomize. Because of this missing data problem, attention has been focused on some parameters which can summarize the causal effect, usually in
terms of expectations. Following [Heckman 1992], [Clements et al. 1994], and [Heckman and Smith 1995], we define the effect of treatment (ET) in the population as follows.

$$\beta = E[Y_1 - Y_0]$$

The natural non-parametric estimate of the above expectation is the corresponding sample average, i.e.

$$\hat{\beta} = \hat{E}[Y_1 - Y_0] = \frac{1}{n} \sum_{i=1}^{n} [Y_{1i} - Y_{0i}] = \frac{1}{n} \sum_{i=1}^{n} Y_{1i} - \frac{1}{n} \sum_{i=1}^{n} Y_{0i} = \bar{Y}_1 - \bar{Y}_0, \text{ say.} \quad (1)$$

$$E(\hat{\beta}) = \beta$$, i.e. $$\hat{\beta}$$ is unbiased for $$\beta$$. The variance of $$\hat{\beta}$$ (normalized for sample size) is given as follows.

$$V(\hat{\beta}) = \begin{cases} E_Z \left[ \frac{V[Y_1|Z]}{P[X=1|Z]} + \frac{V[Y_0|Z]}{P[X=0|Z]} + [\beta(Z) - \beta]^2 \right], & \text{under non-homogeneity,} \\ \frac{V[\hat{Y}_1]}{n} + \frac{V[\hat{Y}_0]}{n}, & \text{under homogeneity.} \end{cases} \quad (2)$$

Literature suggests to do blocking in the situation when the experimental units are not homogeneous. Let us see how does the estimator $$\hat{\beta}$$ defined in Equation (1) should be updated, under blocking using covariates in $$Z$$. Note the following due to [Petersen et al. 2006].

$$\beta = E[Y_1 - Y_0] = E_Z [E[(Y_1 - Y_0)|Z]],$$

$$= E_Z [E[Y_1|Z] - E[Y_0|Z]] = \sum_{Z=z} [E[Y_1|Z = z] - E[Y_0|Z = z]] P(Z = z).$$

Hence, we can define an estimator of $$\beta$$ under blocking using covariates in $$Z$$, as follows.

$$\hat{\beta}_Z = \sum_{Z=z} [\hat{E}[Y_1|Z = z] - \hat{E}[Y_0|Z = z]] \hat{P}(Z = z),$$

$$= \sum_{Z=z} (E[Y_1|Z = z] - E[Y_0|Z = z]) \frac{1}{n} \sum_{i=1}^{n} I(Z_i = z), \quad (3)$$

where $$\frac{1}{n} \sum_{i=1}^{n} I(Z_i = z)$$ is the proportion of units in the block formed with characteristics $$Z = z$$.

Suppose there are no units in one or more blocks, i.e. $$\frac{1}{n} \sum_{i=1}^{n} I(Z_i = z) = 0$$ for some $$z$$, then the overall average treatment effect can be estimated but the $$z$$-specific causal effect cannot be estimated. Hence, it is desirable to have units in every block. This also rules out the argument against randomized block experiments that matching of units can be done post a completely randomized experiment based on the covariates, as it is possible that there are no units corresponding to a particular covariate combination.

$$E(\hat{\beta}_Z) = \beta$$, i.e. $$\hat{\beta}_Z$$ is unbiased for $$\beta$$. The variance of $$\hat{\beta}_Z$$ (normalized for sample size) is given as follows.

$$V(\hat{\beta}_Z) = \begin{cases} E_Z \left[ \frac{V[Y_1|Z]}{P[X=1|Z]} + \frac{V[Y_0|Z]}{P[X=0|Z]} + \sum_{Z} \beta(Z) \hat{P}(Z) - \beta \right]^2, & \text{non-homogeneity,} \\ \frac{V[\hat{Y}_1]}{n} + \frac{V[\hat{Y}_0]}{n}, & \text{homogeneity.} \end{cases} \quad (4)$$

### 2.2 Gain in Precision under Blocking and Challenges with Blocking

Comparing (2) and (4) it can be seen that the variance of the estimate of causal effect under blocking is less than that of under no blocking. Hence blocking improves the precision of the causal effects. Also, note that under homogeneity of the experimental units both the variances are same, i.e. blocking doesn’t bring any improvement. In fact $$\hat{\beta}_Z = \beta$$ under homogeneity of the experimental units. Another motivating example can be seen in the context of a linear model. Suppose there are $$k$$ treatments, and $$b$$ blocks then the response can be defined as a linear equation: $$y_{ij} = \mu + \tau_i + \beta_j + \epsilon_{ij}$$, where $$\mu$$ is the general mean effect, $$\tau_i$$ is the effect due to the the $$i$$th treatment, $$\beta_j$$ is the effect due to the $$j$$th block and $$\epsilon_{ij}$$ is the noise or the experimental error. Clearly, in the absence of $$\beta_j$$ error is
In the experimental studies under non-homogeneity of the experimental units, blocking provides a method for more precise estimation of $\beta$ as compared to the case when no blocking is done. But, the practical difficulty with blocking is that depending upon the no. of covariates and the dimension (no. of distinct values, denoted as $\dim(.)$) of the different covariates, the no. of blocks can be very large. For covariates in the set $Z$ we need to form $\prod_{Z_j \in Z} \dim(Z_j)$ different blocks. Refer to Appendix C.

2.3 Problem Formulation - Optimal Blocking

Now we formalize the problem of interest in this paper, i.e. instead of using all the covariates to form the blocks, can we use a subset only of the covariates, in some optimal manner? In general there can be several considerations for optimality but we restrict ourselves to the following question.

What is the smallest subset of the set of observed covariates (excluding some forbidden variables if there are any) which will account (through blocking) for ‘maximum possible’ elimination of variability in the response, and hence in the estimation of the causal effects, based on the causal structure (diagram) of the all the variables viz. treatment, response and covariates (observed and unobserved)?

The phrase maximum possible in the above question emphasizes that there is always some uncontrollable variability in the response due to two reasons viz. its own exogenous variable, and some unobserved (latent) confounders. Hence hereafter the phrase controlling the variability in response will always mean controlling the variability in response due to the observed variables only, unless specified otherwise.

Definition 1. Any smallest subset of the set of observed covariates (excluding some forbidden variables if there are any) which will account (through blocking) for maximum possible elimination of variability in the response is called a statistically optimal blocking set.

3 Methodology

For all the discussions hereafter, let $G$ be the causal diagram representing the causal structure of the variables in $V$ viz. treatment ($X$), response ($Y$), observed covariates ($Z_1, ..., Z_n$), and the latent or unobserved variables in $U$. We are interested in finding a statistically optimal blocking set (a subset of $\{Z_1, ..., Z_n\}$) to study the causal effect of interest $X \rightarrow Y$.

As discussed earlier, we are interested performing a randomized experiment to study the causal effect of interest $X \rightarrow Y$ where we are randomizing the treatment ($X$). In terms of the causal graph, randomizing $X$ is same as making interventions to set levels/values of $X$. This means that $X$ is not affected by any variable in the graph instead it’s value is decided/set during the experiment. Hence, we can ignore all the arrows coming into $X$ and work with the subgraph of $G$ where all arrows coming into $X$ are ignored. We denote such subgraph as $G_X$. Hence, for the purpose of examples, we work with causal graphs with no arrows coming to $X$.

3.1 Motivating Examples and Key Observations

Example 1. Consider the causal graphs in Figure 1 and Figure 2.
In Figure 1 and Figure 2, the parent set of $Y$, i.e. $\{Z_1, Z_2\}$ blocks all causal paths (except the one from the treatment $X$) going to $Y$. Hence, blocking for the parent set of $Y$ will control for all the variability in $Y$ (except the causal effect of interest).

**Example 2.** Consider the causal graphs in Figure 3.

![Figure 3: Example semi-Markovian models with latent variables involving $Y$.](image)

In Figure 5a, there is a latent structure involving $Y$ but still the parent set of $Y$ works well for controlling the variability in $Y$ because no parent of $Y$ is a collider on a path from ancestors of $Y$ to $Y$ due to the presence of the latent structure. In Figure 5b, there is a latent structure involving $Y$ but in this case the parent set of $Y$ will not be sufficient for controlling the variability in $Y$ as $Z_2$ (a parent of $Y$) is a collider on a path from $Z_4$ (an ancestor of $Y$) to $Y$. Hence, to control the variability in $Y$ we need to control for all the ancestors of $Y$ along with its parents which can influence the response through the latent structure, i.e. we need $\{Z_1, Z_2, Z_4\}$ for controlling the variability in $Y$.

**Example 3.** In the examples so far, there were no descendants of $Y$. In fact, we need not block the descendants of $Y$ because the variations in $Y$ can be at the maximum through its ancestors. Consider the causal graphs in Figure 4.

![Figure 4: Example Semi-Markovian Models with $Y$ having descendants.](image)

In Figure 4a, clearly, $Z_5$ (a descendant of $Y$) can’t cause any variability in $Y$ through the edge $Y \rightarrow Z_5$. Also, $Z_2$ a parent of $Y$ can’t affect $Y$ through $Z_5$ as $Z_2$ is a collider on the path $Z_2 \rightarrow Z_5 \leftarrow Y$. Moreover, even when there is a latent structure between $Z_2$ (ancestor of $Y$) and $Z_5$ (descendant of $Y$) as seen in Figure 4b, $Z_2$ can’t affect $Y$ through $Z_5$ as $Z_5$ is still a collider on the same path. Also, we don’t need to consider the parents of the descendants of $Y$ which are not the ancestors of $Y$ due to the same reason as seen in Figure 4c.

**Observation 1.** We need to consider all the variables that can potentially cause variability in $Y$ in the graph $G_X$. Clearly, for any variable in an SCM, the source of variability can be at most the set of its ancestors. Hence it is sufficient to work with the ancestral graph of $Y$ (Y and its ancestors) in the graph $G_X$, denoted as $G_{An_X}(Y)$.

**Example 4.** In the examples so far, there were no ancestors of the response ($Y$) which were descendants of treatment ($X$). Actually, we should not block any such covariates as doing so will block the causal effect of interest. Consider the causal graphs in Figure 5.

In Figure 6a, $Z_5$ and $Z_6$ are the post-treatment ancestors of $Y$ which we should not block as blocking them will block the causal effect $X \rightarrow Y$. As observed earlier $\{Z_1, Z_2\}$ works well for controlling the variability in $Y$ caused due to just pre-treatment covariates. But note that, we can’t simply drop these post-treatment variables from the discussion as they are ancestors of $Y$ and are valid sources of variability in $Y$. For instance, in Figure 6b, $\{Z_1, Z_2\}$ doesn’t work as the blocking set to control the variability in $Y$ caused due to just pre-treatment covariates, as the presence of a latent structure between $Z_1$ and $Z_5$ opens the paths from $Z_3$ to $Y$ as $Z_1$ is a collider on those paths.

**Definition 2.** (Shpitser et al 2012). Let $X \rightarrow Y$ be the causal effect of interest. A causal path from a variable $X \in X$ to $Y \in Y$ is called proper if it doesn’t intersect $X$ except the endpoints. When $X$ is a single variable then all causal paths are proper.
We formalize the steps for obtaining a statistical optimal blocking set in Algorithm 1, because controlling for them would block the causal effect of interest ($X \rightarrow Y$). Hence we shouldn’t include them in the blocking set, but the subtlety is that we can’t simply drop them and the corresponding edges (more importantly the bi-directed ones) from $G_{An_X(Y)}$.

Hence, we ignore all the variables falling on a proper causal path from $X$ to $Y$ in the graph $G_{An_X(Y)}$. Following Van der Zander et al. (2014), the set of all such variables is $Dpcp(X, Y) = De(\overline{De_X(X)} \setminus An_X(Y))$ where $De(\cdot)$ represents the set of descendants excluding the variable itself and $An_X(Y)$ is the ancestral graph of $Y$ where all edges leaving $X$ are removed.

Note that by definition $Dpcp(X, Y)$ may include non-descendants of $Y$ which we have already argued to not take into consideration in Observation 2. Hence, we can say that specifically we should not block the ancestors of $Y, G$, or a bi-directed path. Assume that $\bar{X}$ is the ancestral graph of $Y, G$.

For the proof of Theorem 1, refer to Appendix D. Note that in a Markovian model there are no bi-directed paths then the elementary partition of $\overline{An_X(Y)}$ is nothing but the $c$-component of $G_{An_X(Y)}$. Hence, $C_Y = \{Y\}$, and $Pa_{c_Y,G_{An_X(Y)}} = Pa_{Y,G_{An_X(Y)}}$. In fact in Markovian case,

**Observation 2.** We should not block the ancestors of $Y$ which are on a proper causal path from $X$ to $Y$ in the graph $G_{An_X(Y)}$. For the usual graph $G$, the set of variables $V$ can be partitioned into disjoint groups by assigning two variables to the same group if and only if they are connected by a bi-directed path. Assume that $V$ is thus partitioned into $k$ groups $\{S_1, \ldots, S_k\}$. We will call each $S_j, j = 1, \ldots , k$, a $c$-component (abbreviating confounded component) of $V$ in $G$ or a $c$-component of $G$.

**Definition 3.** Tian and Pearl (2002). Let a path composed entirely of bi-directed edges be called a bi-directed path. For the usual graph $G$, the set of variables $V$ can be partitioned into disjoint groups by assigning two variables to the same group if and only if they are connected by a bi-directed path. Assume that $V$ is thus partitioned into $k$ groups $\{S_1, \ldots, S_k\}$. We will call each $S_j, j = 1, \ldots , k$, a $c$-component (abbreviating confounded component) of $V$ in $G$ or a $c$-component of $G$.

**Definition 4.** The $S_j$ such that $Y \in S_j$ is called the $c$-component of a variable $Y$ in $G$ and is denoted as $c_Y \setminus G$. As $\{S_1, \ldots, S_k\}$ is a partition of $V$, $c$-component of a variable always exists and is unique.

**Theorem 1.** The smallest $Z$ such that $Y \perp An_X(Y) \setminus Z|Z$, is nothing but the set of parents of $c$-component of $Y$ in $G_{An_X(Y)}$, i.e. $Z = Pa_{c_Y,G_{An_X(Y)}}$.

For the proof of Theorem 1 refer to Appendix D. Note that in a Markovian model there are no bi-directed paths then the elementary partition of $\overline{An_X(Y)}$ is nothing but the $c$-component of $G_{An_X(Y)}$. Hence, $C_Y = \{Y\}$, and $Pa_{c_Y,G_{An_X(Y)}} = Pa_{Y,G_{An_X(Y)}}$. In fact in Markovian case,
$Pa_{Y,G_{\Lambda G_{X}(Y)}} = Pa_{Y}$, as in this case a variable is always independent of its non-descendants excluding its parents given its parents. Application of Algorithm 1 to an example Semi-Markovian model is discussed in Appendix E.

4 Conclusion and Future Work

We have obtained a statistically optimal blocking set ($Z^*$) such that it is a smallest subset of the observed covariates ($Z_1, ..., Z_n$) (excluding the forbidden ones) which will account (through blocking) for maximum possible elimination of variability in response ($Y$). Further perspective on optimality is to prune a statistically optimal blocking set for economic reasons. For instance, suppose that we are restricted to create blocks using at most $k$ covariates and a statistically optimal blocking set is of cardinality $> k$. One possible way to optimally prune a statistically optimal blocking set is by dropping the covariates which are least significant in controlling the variability in response. Or the question can be what is the minimum number of covariates required to eliminate as much as $(1 - \alpha) \times 100\%$ of the variability eliminated by a statistically optimal blocking set, for some user-defined $\alpha \geq 0$. Refer to Appendix E for a continued discussion on future work.
References

Clements, N., Heckman, J. J., and Smith, J. A. (1994). Making the most out of social experiments: Reducing the intrinsic uncertainty in evidence from randomized trials with an application to the jtpa exp.

Cochran, W. G. and Cox, G. M. (1948). Experimental designs. Technical report, North Carolina State University. Dept. of Statistics.

Fisher, R. A. (1992). Statistical methods for research workers. In Breakthroughs in statistics, pages 66–70. Springer.

Heckman, J. J. (1992). Randomization and social policy evaluation. Evaluating welfare and training programs, 1:201–30.

Heckman, J. J. and Smith, J. A. (1995). Assessing the case for social experiments. Journal of economic perspectives, 9(2):85–110.

Higgins, M. J., Sävje, F., and Sekhon, J. S. (2016). Improving massive experiments with threshold blocking. Proceedings of the National Academy of Sciences, 113(27):7369–7376.

Holland, P. W. (1986). Statistics and causal inference. Journal of the American statistical Association, 81(396):945–960.

Kempthorne, O. (1952). The design and analysis of experiments.

Kempthorne, O. (1955). The randomization theory of experimental inference. Journal of the American Statistical Association, 50(271):946–967.

Pearl, J. (2009). Causality. Cambridge university press.

Petersen, M. L., Sinisi, S. E., and van der Laan, M. J. (2006). Estimation of direct causal effects. Epidemiology, pages 276–284.

Rosenbaum, P. R. and Rubin, D. B. (1983). The central role of the propensity score in observational studies for causal effects. Biometrika, 70(1):41–55.

Shpitser, I., VanderWeele, T., and Robins, J. M. (2012). On the validity of covariate adjustment for estimating causal effects. arXiv preprint arXiv:1203.3515.

Tian, J. (2002). Studies in causal reasoning and learning. University of California, Los Angeles.

Tian, J. and Pearl, J. (2002). A general identification condition for causal effects. In Aaai/iaai, pages 567–573.

Van der Zander, B., Liskiewicz, M., and Textor, J. (2014). Constructing separators and adjustment sets in ancestral graphs. In CI@ UAI, pages 11–24.

Yates, F. (1935). Complex experiments. Supplement to the Journal of the Royal Statistical Society, 2(2):181–247.
A Appendix 1 - Proofs of the Intermediate Results

A.1 Unbiasedness and Variance of the Estimate of Causal Effect without Blocking

A.1.1 Unbiasedness

\( \hat{\beta} \) defined in (1) is unbiased for \( \beta \), which can be seen as follows.

\[
E[\hat{\beta}] = E[\bar{Y}_1 - \bar{Y}_0].
\]

In general, experimental units may not be homogeneous, hence we can’t say that \( E(\bar{Y}_1) = E(Y_1) \), and \( E(\bar{Y}_0) = E(Y_0) \). But, suppose \( Z \) is the set of covariates, then we can always do the following.

\[
E[\hat{\beta}] = E[\bar{Y}_1 - \bar{Y}_0] = E_Z[E[(\bar{Y}_1 - \bar{Y}_0)|Z]],
\]

\[
= E_Z[E[\bar{Y}_1|Z] - E[\bar{Y}_0|Z]].
\]

As given the covariates the experimental units are identical, we can always write that \( E[\bar{Y}_1|Z] = E[Y_1|Z] \) and \( E[\bar{Y}_0|Z] = E[Y_0|Z] \). Hence, we have the following.

\[
E[\hat{\beta}] = E_Z[E[\bar{Y}_1|Z] - E[\bar{Y}_0|Z]] = E_Z[E[Y_1|Z] - E[Y_0|Z]],
\]

\[
= E_Z[E[(Y_1 - Y_0)|Z]] = E[Y_1 - Y_0] = \beta.
\]

Hence, \( \hat{\beta} \) is always unbiased for \( \beta \), including under non-homogeneity of the experimental units.

A.1.2 Variance

We calculate the variability (and hence the precision) of this estimator under non-homogeneity of the experimental material as follows.

\[
V[\hat{\beta}] = V[Y_1 - Y_0],
\]

\[
= V_Z[E[(\bar{Y}_1 - \bar{Y}_0)|Z]] + V_Z[E[(\bar{Y}_1 - \bar{Y}_0)|Z]],
\]

\[
= V_Z[E[\bar{Y}_1|Z] - E[\bar{Y}_0|Z]] + V_Z[V[\bar{Y}_1|Z] + V[\bar{Y}_0|Z]].
\]

As given the covariates, the experimental units are identical, we can always write that \( E[\bar{Y}_1|Z] = E[Y_1|Z] \), \( E[\bar{Y}_0|Z] = E[Y_0|Z] \), \( V[\bar{Y}_1|Z] = \frac{V[Y_1|Z]}{\#(X = 1|Z)} \), and \( V[\bar{Y}_0|Z] = \frac{V[Y_0|Z]}{\#(X = 0|Z)} \). Hence, we have the following:

\[
V[\hat{\beta}] = V_Z[E[Y_1|Z] - E[Y_0|Z]] + E_Z\left[\frac{V[Y_1|Z]}{\#(X = 1|Z)} + \frac{V[Y_0|Z]}{\#(X = 0|Z)}\right],
\]

\[
= V_Z[E[(Y_1 - Y_0)|Z]] + E_Z\left[\frac{V[Y_1|Z]}{\#(X = 1|Z)} + \frac{V[Y_0|Z]}{\#(X = 0|Z)}\right],
\]

\[
= V_Z[\beta(Z)] + E_Z\left[\frac{V[Y_1|Z]}{\#(X = 1|Z)} + \frac{V[Y_0|Z]}{\#(X = 0|Z)}\right],
\]

where \( \beta(Z) := E[(Y_1 - Y_0)|Z] \) is defined as the \( Z \)-specific causal effect of \( X \) on \( Y \).

\[
V[\hat{\beta}] = E_Z[\beta(Z) - E_Z[\beta(Z)]]^2 + E_Z[V[Y_1|Z] + V[Y_0|Z]],
\]

Using the fact that \( E_Z[\beta(Z)] = E_Z[E(Y_1 - Y_0)|Z] = E[Y_1 - Y_0] = \beta \), we have the following.

\[
E_Z[\beta(Z) - \beta]^2 + E_Z\left[\frac{V[Y_1|Z]}{\#(X = 1|Z)} + \frac{V[Y_0|Z]}{\#(X = 0|Z)}\right],
\]

\[
= E_Z\left[\frac{V[Y_1|Z]}{\#(X = 1|Z)} + \frac{V[Y_0|Z]}{\#(X = 0|Z)} + (\beta - \beta)^2\right].
\]

In the literature the expressions \( \#(X = 1|Z) \) and \( \#(X = 0|Z) \) in the above formula are usually replaced by the sample proportions, i.e. \( P[X = 1|Z] = \frac{\#(X = 1|Z)}{\#(Z)} \) and \( P[X = 0|Z] = \frac{\#(X = 0|Z)}{\#(Z)} \).
respectively, and termed as variance normalized for sample size. Hence the variance of \( \hat{\beta} \) normalized for sample size is given as follows.

\[
V[\hat{\beta}] = E_Z \left[ \frac{V[Y_1|Z]}{P[X = 1|Z]} + \frac{V[Y_0|Z]}{P[X = 0|Z]} + [\beta(Z) - \beta]^2 \right].
\]

It is easy to see that when the experimental units are homogeneous, i.e. \( Z \) is deterministic then \( \beta(Z) = \beta \), \( \frac{V[Y_1|Z]}{P[X = 1|Z]} = \frac{V[Y_1]}{P[X = 1]} \), and \( \frac{V[Y_0|Z]}{P[X = 0|Z]} = \frac{V[Y_0]}{P[X = 0]} \). Hence, \( V(\hat{\beta}) = E_Z \left[ \frac{V[Y_1]}{P[X = 1]} + \frac{V[Y_0]}{P[X = 0]} \right] + [\beta - \beta]^2 \).

A.2 Unbiasedness and Variance of the Estimate of Causal Effect under Blocking

A.2.1 Unbiasedness

When the experimental units are homogeneous, i.e. \( Z \) is deterministic then \( \hat{\beta}_Z \) defined in (3) is same as \( \beta \). Hence, \( \hat{\beta}_Z \) is unbiased for \( \beta \) under the homogeneity of the experimental material. \( \hat{\beta}_Z \) is also unbiased for \( \beta \) under non-homogeneity of the experimental material which can be seen as follows.

\[
E[\hat{\beta}_Z] = E \left[ \sum_{Z=z} \left( [Y_1|Z = z] - [\bar{Y}_0|Z = z] \right) \frac{1}{n} \sum_{i=1}^{n} I(Z_i = z) \right],
\]

\[
E[\hat{\beta}_Z] = E \left[ \sum_{Z=z} \left( [Y_1|Z = z] - [\bar{Y}_0|Z = z] \right) \right] E \left[ \frac{1}{n} \sum_{i=1}^{n} I(Z_i = z) \right],
\]

\[
= \sum_{Z=z} \left( E[Y_1|Z = z] - E[\bar{Y}_0|Z = z] \right) P(Z = z),
\]

\[
= E_Z \left[ E[Y_1|Z] - E[\bar{Y}_0|Z] \right] = E_Z \left[ E[(Y_1 - Y_0)|Z] \right] = E_Z(\beta(Z)) = \beta.
\]

Hence, \( \hat{\beta}_Z \) is always unbiased for \( \beta \), including under non-homogeneity of the experimental units.

A.2.2 Variance

We calculate the variability (and hence the precision) of this estimator under non-homogeneity of the experimental material as follows.

\[
V[\hat{\beta}_Z] = V_Z \left[ E \left[ \sum_{Z} \bar{Y}_1[Z - \bar{Y}_0[Z] \hat{P}(Z) \right] \right] + E_Z \left[ V \left[ \sum_{Z} \bar{Y}_1[Z - \bar{Y}_0[Z \hat{P}(Z) \right] \right],
\]

\[
= V_Z \left[ \sum_{Z} E[\bar{Y}_1[Z - \bar{Y}_0[Z \hat{P}(Z) \right] + E_Z \left[ \sum_{Z} V[\bar{Y}_1[Z - \bar{Y}_0[Z \hat{P}(Z) \right] \right],
\]

\[
= V_Z \left[ \sum_{Z} E[(Y_1 - Y_0)|Z] \hat{P}(Z) \right] + E_Z \left[ \sum_{Z} \left( V[\bar{Y}_1[Z] + V[\bar{Y}_0[Z] \right] \hat{P}(Z) \right].
\]

As given the covariates, the experimental units are identical, we can always write that \( E[\bar{Y}_1[Z] = E[Y_1[Z], E[\bar{Y}_0[Z] = E[Y_0[Z], V[\bar{Y}_1[Z] = \frac{V[Y_1]}{\#[X = 1]}, \) and \( V[\bar{Y}_0[Z] = \frac{V[Y_0]}{\#[X = 0]} \). Hence, we have the following.

\[
= V_Z \left[ \sum_{Z} \beta(Z) \hat{P}(Z) \right] + E_Z \left[ \sum_{Z} \left( \frac{V[Y_1]}{\#[X = 1]} + \frac{V[Y_0]}{\#[X = 0]} \right) \hat{P}(Z) \right],
\]

\[
= E_Z \left[ \sum_{Z} \beta(Z) \hat{P}(Z) - E_Z \left[ \sum_{Z} \beta(Z) \hat{P}(Z) \right] \right]^2
\]

\[
+ \sum_{Z} \frac{V[Y_1]}{\#[X = 1]} E_Z[\hat{P}(Z)] + \sum_{Z} \frac{V[Y_0]}{\#[X = 0]} E_Z[\hat{P}(Z)].
\]
Note that \( \hat{P}(Z = z) = \frac{1}{n} \sum_{i=1}^{n} I(Z_i = z) \Rightarrow E[\frac{1}{n} \sum_{i=1}^{n} I(Z_i = z)] = P(Z = z) \), i.e. unbiased estimation using sample proportion. Hence, we have the following.

\[
E_Z \left[ \sum_Z \beta(Z) \hat{P}(Z) - \beta \right]^2 + \sum_Z \frac{V[Y_1|Z]}{P[X=1|Z]} P(Z) + \sum_Z \frac{V[Y_0|Z]}{P[X=0|Z]} P(Z),
\]

\[
E_Z \left[ \sum_Z \beta(Z) \hat{P}(Z) - \beta \right]^2 + E_Z \left[ \frac{V[Y_1|Z]}{\#|X=1|Z} \right] + E_Z \left[ \frac{V[Y_0|Z]}{\#|X=0|Z} \right],
\]

\[
E_Z \left[ \frac{V[Y_1|Z]}{\#|X=1|Z} + \frac{V[Y_0|Z]}{\#|X=0|Z} + \left[ \sum_Z \beta(Z) \hat{P}(Z) - \beta \right]^2 \right].
\]

In the literature the expressions \#|X=1|Z\] and \#|X=0|Z\] in the above formula are usually replaced by the sample proportions, i.e. \( P[X=1|Z] = \frac{\#|X=1|Z}{\#|Z|} \) and \( P[X=0|Z] = \frac{\#|X=0|Z}{\#|Z|} \) respectively, and termed as variance normalized for sample size. Hence the variance of \( \hat{\beta} \) normalized for sample size is given as follows:

\[
V[\hat{\beta}_Z] = E_Z \left[ \frac{V[Y_1|Z]}{P[X=1|Z]} + \frac{V[Y_0|Z]}{P[X=0|Z]} + \left[ \sum_Z \beta(Z) \hat{P}(Z) - \beta \right]^2 \right].
\]

It is easy to see that when the experimental units are homogeneous, i.e. \( Z \) is deterministic then \( \hat{P}(Z) = 1 \), \( \beta(Z) = \beta \), \( \frac{V[Y_1|Z]}{P[X=1|Z]} = \frac{V[Y_1]}{P[X=1]} \), and \( \frac{V[Y_0|Z]}{P[X=0|Z]} = \frac{V[Y_0]}{P[X=0]} \). Hence, \( V(\hat{\beta}_Z) = E_Z \left[ \frac{V[Y_1]}{P[X=1]} + \frac{V[Y_0]}{P[X=0]} + [\beta - \beta]^2 \right] = \frac{V[Y_1]}{P[X=1]} + \frac{V[Y_0]}{P[X=0]} \).

**B  A Note on Gain in Precision under Blocking**

We have seen that blocking is useful to study the \( z \)-specific causal effects and to improve the precision of the estimates. Sometimes, the interest is to study the causal effects from the observational data, possibility because carrying out a randomized experiment is either economically or ethically infeasible. An approach similar to blocking (in randomized experiments) called *matching* is used to study the causal effects from the observational data, where grouping of units is done on covariates after getting the observational data. There covariate adjustment is done to reduce the confounding bias. For instance, Rosenbaum and Rubin (1983) provided a way to create blocks by matching the observation on propensity score, which is a famously termed as *propensity score matching* (PSM). General concerns with matching have been raised by Pearl (2009), who has argued that hidden bias may actually increase because matching on observed variables may unleash bias due to dormant unobserved confounders. Similarly, Pearl has argued that bias reduction can only be assured (asymptotically) by modeling the qualitative causal relationships between treatment, outcome, observed and unobserved covariates. Confounding occurs when the experimenter is unable to control for alternative, non-causal explanations for an observed relationship between independent and dependent variables. Such control should satisfy the *back-door criterion* (Pearl (2009)).

**C  An Example to illustrate the Challenges with Blocking**

If we want to study the effect of a drug \( (X) \) on some heart disease \( (Y) \) where the subjects under consideration have the following attributes which can potentially affect the effect of drug.

1. Gender: Male and Female, i.e. \( \dim(\text{Gender}) = 2 \),
2. Age: <25, 25-45, 45-65, >65, i.e. \( \dim(\text{Age}) = 4 \),
3. Weight: Underweight, Normal, Overweight, Obese I, Obese II, i.e. \( \dim(\text{Weight}) = 5 \),
4. Blood Pressure: Normal, Prehypertension, Hypertension I, Hypertension II, Hypertensive Crisis, i.e. \( \dim(\text{Blood Pressure}) = 5 \), and
5. Bad Cholesterol: Optimal, Above Optimal, Borderline High, High, Very High, i.e. \( \dim(\text{Bad Cholesterol}) = 5 \).
Hence, we need to form \(2 \times 4 \times 5 \times 5 = 1000\) blocks. Performing a randomized experiment with a large no. of blocks can be very costly. Sometimes, it may be not at all be possible as the no. of blocks can be larger than the subjects or the experimental units, which would cause some of the blocks to be empty. For instance, there may not be any male subjects under the age of 25 who are Obese II with a Hypertensive Crisis and Optimal Cholesterol level.

D Proof of Theorem 1

Proof. The proof follows from Tian (2002), which in Chapter 4 Testable Implications of Causal Models, states that a node is independent of its non-descendants excluding the parents of its \(c\)-component given the parents of its \(c\)-component.

E Applying the Proposed Algorithm to a General Semi-Markovian Model

We apply Algorithm 1 to obtain an optimal blocking set for a general semi-Markovian causal graph given in Figure 6.

![Figure 6: The Original Graph G.](image)

Obtain the graph \(G_{\bar{X}}\) as displayed in Figure 7 and further it to \(G_{An\bar{X}}(Y)\) as displayed in Figure 8.

![Figure 7: The Graph G_{\bar{X}}.](image)  
![Figure 8: The Graph G_{An\bar{X}}(Y).](image)

Following Definition 3, the \(c\)-components of \(G_{An\bar{X}}(Y)\) are \(\{X\}, \{Z_1, Z_5, Y, Z_2\}, \{Z_3, Z_4\}\). Following Definition 4, \(c_{Y,G_{An\bar{X}}(Y)} = \{Z_1, Z_5, Y, Z_2\}\).

Following Theorem 2, \(Z = Pa_{c_{Y,G_{An\bar{X}}(Y)}} = Pa(Z_1, Z_5, Y, Z_2, Z_3, Z_4, Z_5)\).

Following Observation 2, \(Dpcp(X, Y)_{G_{An\bar{X}}(Y)} = \{Z_5, Z_6\}\). Following Step 4 and 5, a statistically optimal blocking set is given as follows.

\[Z^* = Z \setminus Dpcp(X, Y)_{G_{An\bar{X}}(Y)} \setminus X = \{Z_1, Z_2, Z_3, Z_4, Z_5\} \setminus \{Z_5, Z_6\} \setminus X = \{Z_1, Z_2, Z_3, Z_4\} \]

F Future Work (continued)

We want to have a way to decompose the total variability controlled by a statistically optimal blocking set in terms of its individual elements, i.e. to quantify the contribution of each individual covari-
ate in the optimal blocking set controlling the variability in the response. For the example discussed in Section 2.2, we have seen that a statistically optimal blocking set is $Z^* = \{Z_1, Z_2, Z_3, Z_4\}$. We are interested in further pruning this set due to economic reasons. Note that, $Z_1$ and $Z_2$ are the parents of $Y$ and have direct effect on $Y$ through $Z_1 \rightarrow Y$ and $Z_2 \rightarrow Y$, respectively. $Z_4$ affects $Y$ through either of the routes viz. through $Z_4 \rightarrow Z_2 \rightarrow Y$ when $Z_2$ is unblocked, and through $Z_4 \rightarrow Z_2 \rightarrow Y$ when $Z_2$ is blocked. Similarly, $Z_3$ also affects $Y$ one of the two routes. Suppose it is possible to quantify all the causal effects through the observed variables then we can take the following subgraph of Figure 8 the with varying thickness of the edges between observed variables, where the thickness represents strength of the link.

Figure 9: Subgraphs of Figure 8 involving $\{Z_4, Z_2, Y\}$ with varying link strengths.

In Figure both the links are weak, hence we can discard both. In Figure the link $Z_4 \rightarrow Z_2$ is strong but the strength of the effect of $Z_4$ on $Y$ through the path $Z_4 \rightarrow Z_2 \rightarrow Y$ depends on how strong the link $Z_2 \rightarrow Y$ is? It can be seen that in Figure 4(b) the link $Z_2 \rightarrow Y$ is weak which suggest that we can discard $Z_2$ and as long as we don’t block $Z_2$ we never open the path $Z_4 \rightarrow Z_2 \rightarrow Y$, and hence we can discard $Z_4$ also. In Figure the link $Z_2 \rightarrow Y$ is strong which suggest that we should block $Z_2$. Blocking $Z_2$ will open the path the $Z_4 \rightarrow Z_2 \rightarrow Y$ but as the link $Z_3 \rightarrow Z_4$ is weak we can discard $Z_3$. In Figure the link $Z_3 \rightarrow Y$ is strong which suggest that we should block $Z_3$. Blocking $Z_2$ will open the path the $Z_4 \rightarrow Z_2 \rightarrow Y$ and as the link $Z_3 \rightarrow Z_4$ is strong we should block $Z_4$ also.

The discussion can be used to motivate towards how and when further pruning of a statistically optimal blocking set can be done due to economic reasons. But there are several issues as follows.

1. Quantification of causal effects will require some observational data which may be difficult specifically for the treatment and response variables. Though all the covariates that we need to worry about are all pre-treatment and pre-response which are usually observed.

2. Even with the availability of some observational data for some or all variables, it may be possible that the causal effect is not identifiable.

In case of some specific cases of linear models simple linear regression and Wright’s rule can be of some help. But making some general statements is very difficult. For instance, the strength of the links $Z_4 \rightarrow Z_2$ and $Z_3 \rightarrow Z_4$ can be assessed by regressing $Z_4$ on $Z_2$, and $Z_3$ on $Z_1$.

In general, economic restriction may not just be on the number of covariates to block using. As seen in the example in Section 2.2 economic cost of blocking depends also on the dimensionality of the covariates. Hence, one possible way to think about economic cost of a blocking set is the resulting no. of blocks. But, the cost of a blocking set may not just be the no. of blocks. It can also involve how costly each covariate is otherwise. For instance, blocking using gender may be costlier than using age, though gender results in 2 blocks and age results in 4 blocks. Hence, the general definition of economic cost of a blocking set should account for the resulting no. of blocks and the cost of forming those blocks for different covariates in that blocking set.

**Definition 5.** The economic cost of a blocking set is defined as a weighted sum of the dimensions of the covariates in it, where weights are their individual costs of forming blocks.

Note that, when all the covariates are equally costly and are of same dimension, the cost of blocking is nothing but its cardinality, the case which we have considered in the beginning of this section.

**Definition 6.** The statistical cost of a blocking set is defined as the inverse of the amount of variability in the response that it controls for.

Note that, we haven’t completely addressed how to quantify the statistical cost of blocking.
Definition 7. A smallest subset of the set of observed covariates (excluding some forbidden variables if there are any) is said to be an optimal blocking set if it minimizes the statistical cost of blocking among all the subsets of the set of observed covariates (excluding some forbidden variables if there are any), having an economic cost less than or equal to the some user-defined threshold.

Developing an algorithm to obtain an optimal blocking set defined in Definition 7 is an open problem which we aim to address in our future work.