Uncertainty in the Design Stage of Two-Stage Bayesian Propensity Score Analysis

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The two-stage process of propensity score analysis (PSA) includes a design stage where propensity scores are estimated and implemented to approximate a randomized experiment and an analysis stage where treatment effects are estimated conditional upon the design. This paper considers how uncertainty associated with the design stage impacts estimation of causal effects in the analysis stage. Such design uncertainty can derive from the fact that the propensity score itself is an estimated quantity, but also from other features of the design stage tied to choice of propensity score implementation. This paper formalizes a Bayesian framework for obtaining the posterior distribution of causal effects after marginalizing over a distribution of design-stage outputs, lending underlying formality to Bayesian methods for PSA (BPSA) that have gained attention in recent literature. Formulation of a probability distribution for the design-stage output depends on how the propensity score is implemented in the design stage, and propagation of uncertainty into causal estimates depends on how the treatment effect is estimated in the analysis stage. We explore these differences within a sample of commonly-used propensity score implementations (quantile stratification, nearest-neighbor matching, caliper matching, inverse probability of treatment...
weighting, and doubly robust estimation) and compare operating characteristics with standard Frequentist PSA in a simulation study. The methods are then deployed in an investigation of the association between levels of fine particulate air pollution and elevated exposure to emissions from coal-fired power plants.

**KEYWORDS**
bayesian, propensity score, observational study

## 1 | INTRODUCTION

Propensity score (PS) analysis refers to a wide range of strategies for estimating causal treatment effects with observational data. Rubin and others [1, 2] motivate this process by conceptualizing an observational study as having arisen from a randomized clinical trial where the rules of assignment have been lost and must be estimated. This perspective draws focus to the “design” stage of a propensity score analysis (PSA), in which PS are estimated then implemented to create a sub- or pseudo-population of the data representative of a hypothesized randomized trial [1, 3]. Following the design stage, observed outcome information is used to estimate a treatment effect in the “analysis” stage.

A well-conducted design stage is "absolutely essential for drawing objective inferences for causal effects," and is typically conducted without access to outcome data [1]. Many analytic decisions are required to create a successful "design" which implements the estimated propensity score (e.g., via matching, weighting, or subclassification) to achieve treated and untreated groups that are "balanced" with respect to observed covariates [4, 5, 6, 7, 8]. In traditional PSAs, once the researcher has satisfactorily approximated the design of the hypothesized randomized study, the sub- or pseudo-population of observations created in the design stage is treated as fixed or known and estimation of causal effects in the analysis stage is conducted conditional on the design. In light of the importance of the design stage and the multi-faceted decisions made towards estimating and implementing the propensity score, it stands to reason that uncertainty associated with the design stage of PSA should be propagated into the estimation of causal effects in the analysis stage in order to more fully acknowledge all potential sources of PSA uncertainty.

Consideration of design uncertainty is often framed solely in terms of whether and how to incorporate uncertainty from the estimation of the PS into variance estimation of causal effects [9, 10, 11, 12, 13, 14, 15]. However, more sources of uncertainty may exist in the design stage. Dehejia and Wahba [16] illustrated that when implementing the PS with nearest neighbor matching without replacement, the ordering of observations in the matching procedure resulted in different estimates of treatment effect, even when using the same set of estimated PS [16]. Abadie and Imbens derive a variance estimator for causal effects that accounts for both PS estimation uncertainty and uncertainty in the construction of matches [17]. Further expansion of the idea of design uncertainty has appeared in Zigler and Dominici, who regard the variables to include in the propensity score model as uncertain [18], Spertus and Normand [19] who construe probabilistic design weights, and Zigler and Cefalu, who regard the subset of observations pruned (or truncated) from the design to be unknown [6].

This paper identifies the components of “design uncertainty,” and offers a Bayesian perspective on propagating such uncertainty into inference for causal effects conducted in the analysis stage. While many of the issues contained herein have appeared sporadically within related work [20, 21, 22, 23, 24, 19], the common themes have not been brought together towards a formalization of the role of Bayesian methods in accounting for design stage uncertainty...
in propensity score analysis. The theoretical framework of this paper is supported by and gives conceptual structure to recently developed methodology variously described with terms such as two-step Bayesian, quasi-Bayesian, or approximately-Bayesian, which leverage the mechanics of Bayesian inference in both the propensity score “design” and “analysis” stages, but without full joint Bayesian updating of both stages simultaneously. We offer the formality of a probability distribution for the output of the PS design stage, the structure of which is determined by a) the PS estimation model and b) the specific type of implementation used in the design.

We consider a setting where the the following PSA decisions are made *a priori*: the causal estimand of interest, the form of the PS model, the type of PS implementation (e.g., matching, weighting, etc.), and the form of the analysis stage. The goal is then to marginalize estimation of causal effects in the analysis stage over the posterior distribution of “designs” estimated in the design stage, while maintaining sufficient separation so that information in the outcome analysis does not contribute to estimation of the design. The mechanics of marginalizing over design uncertainty depend on the type of PS implementation utilized and the subsequent formulation of the analysis stage. Implications of this marginalization for operating characteristics of causal estimates also varies by type of implementation.

Developing a “distribution” for the design stage clarifies the distinction between quantities we define as 1) “design estimation uncertainty” (DEU) corresponding to familiar notions of estimation uncertainty in the PS, and 2) “design decision uncertainty,” (DDU) corresponding to the possibility of additional uncertainty due to stochasticity inherent in the PS implementations, even for a fixed value of the PS and a chosen form of implementation. Uncertainty arising from treatment effect estimation in the design stage is defined as analysis estimation uncertainty (AEU).

We examine this framework within the context of several commonly-used PS implementations and corresponding analysis stages: quantile stratification, nearest-neighbor matching with replacement, caliper matching with replacement, inverse probability of treatment weighting (IPTW), and a common doubly-robust estimator, although the formalization applies to a broader range of PSAs. We consider both settings where the analysis stage consists of a parametric “outcome model” (conditional on the PS), and also settings where no outcome-model likelihood exists, as would be the case with many common non- or semi-parametric (e.g., matching or weighted) estimators with known asymptotic properties. The latter case is particularly complicated for Bayesian inference, in which case we propose an approximation to the asymptotic posterior distribution of a causal effect. The collection of PS analyses considered here is not designed to be exhaustive and we make no argument for superiority of any given approach. Rather, these specific implementations and analysis stages are used to illustrate the different mechanistic sources of uncertainty that can arise when using the PS. Similarly, this work makes no attempt to establish superiority of a Bayesian vs. non-Bayesian approaches, but rather aims to elaborate on considerations and complications inherent to Bayesian PSA in order to bolster a foundation from which to advance Bayesian methods in this realm.

After distinguishing between different sources of design uncertainty and offering a formalization of integrating design uncertainty into the analysis stage, we outline a corresponding computational algorithm and illustrate the approach in a simple simulation study meant to compare performance against standard estimation procedures that regard the design as fixed. We then illustrate the method in an analysis of emissions from coal-fired power plants and ambient particulate air pollution collected over 22,723 zip codes in the Northeast, Southeast and Industrial Midwest regions of the United States. The paper concludes with a discussion of possible future directions.
2 | NOTATION, ESTIMANDS, AND OVERVIEW OF MARGINALIZING OVER DESIGN UNCERTAINTY

2.1 | Notation and Estimand

Let $Y_i$, $X_i$ and $T_i$ represent the observed outcome, covariate vector (of length $p$) and treatment indicator for unit $i$, where $i = 1, 2, \ldots, n$. $T_i \in [0, 1]$ is dichotomous and $Y_i$ is a continuous random variable. Let $Y$ and $T$ be vectorized representations of the data and $X$ a matrix of observed covariates. We state without comment the following assumptions common to causal inference literature and defer readers to other work for details [1]: positivity (each subject has a non-zero probability of receiving either treatment) and SUTVA (units do not interfere with each other’s outcomes and potential outcomes are well-defined).

The additional key assumption of strongly ignorable treatment assignment states that potential outcomes which exist under either treatment are independent of observed treatment assignment after adjusting for observed covariates in $X$, which must include all confounders. Under ignorability, the average treatment effect (ATE) may be estimated from observed outcomes among units that exhibit the same distribution in background covariates:

$$\Delta_{ATE} = E[E(Y_i | T_i = 1, X_i) - E(Y_i | T_i = 0, X_i)] \quad (1)$$

All relevant covariate information may be summarized in a "coarsened" manner with the propensity score, defined as the probability to receive the treatment rather than the control conditional on pre-treatment covariates of that individual [25]. Let $e_i$ represent the propensity score for individual $i$.

$$e_i = P(T_i = 1 | X_i) \quad (2)$$

Units which are homogeneous with respect to $e_i$ are said to be “balanced” with respect to $X_i$, and under the assumption of strong ignorability, units with the same value of propensity score but assigned to different treatments have an expected difference in responses equal to the average treatment effect [25].

While $\Delta_{ATE}$ is defined above as the marginal average treatment effect in the population, $\Delta$ (without a subscript) will be used from this point to generically represent a causal estimand such as the ATE or the ATT(C) (Average Treatment Effect on the Treated (Control)). The choice of estimand considered is implied by the PS implementation, which is assumed to be chosen a priori. Differences between estimands are not the focus of this paper, rather, we will consider estimation of each in the context of a corresponding implementation stage (e.g., matching will be implicitly taken to be estimating the ATT, whereas IPW estimates the ATE).

2.2 | Overview of Marginalizing over Design Uncertainty

We begin with a heuristic description of the proposed framework for Bayesian PSA, deferring details to subsequent sections. Without the use of the propensity score, traditional Bayesian inference for $\Delta$ would follow from specification of a likelihood for $(T, X, Y)$ conditional on unknown parameters, $\theta$, a prior distribution for $\theta$, and some function relating the data and $\theta$ to the quantity $\Delta$. This standard procedure for Bayesian inference is incompatible with use of the propensity score as the PS is not intended to be part of the data-generating likelihood for $Y$ [24, 26, 27]. This
fundamental feature of using the PS in Bayesian inference motivates careful delineation of unknown quantities in the design and analysis stages.

Let \( \nu \) be a parameter summarizing the output of a PS implementation, that is, representing one propensity score "design" implied by implementing a set of estimated propensity scores. For example, \( \nu \) may be defined as a partition of observations into strata based on their estimated propensity scores (detailed definition of \( \nu \) is developed in the context of several PS implementations in Section 3.2). Under the assumptions outlined in Section 2.1 and a design stage that is successful in the sense that it appropriately balances observed covariates between treated and untreated units, inference (Bayesian or otherwise) for \( \Delta \) may be carried out with standard methodology conditional on a single value of \( \nu \), possibly with variance estimators adapted to account for uncertainty in \( \nu \) [28, 17, 10]. Instead of conditioning on a single realization of \( \nu \), the goal of this paper is to marginalize over a probability distribution for all possible values of \( \nu \). This is represented heuristically as:

\[
f(\Delta|T, X, Y) = \int f(\Delta|T, X, Y, \nu) f(\nu|T, X) d\nu
\]

The distribution \( f(\nu|T, X) \) is written to explicitly acknowledge that the design stage is conducted without outcome information \( (Y) \). Integrating the posterior distribution of \( \Delta \) over the marginal distribution of \( \nu \) is a mathematical representation of propagating design uncertainty in to the analysis stage by marginalizing over a "distribution of designs". The concept is similar to that of Bayesian model averaging, with different values of \( \nu \) representing different models, each receiving different weights in the final posterior distribution.

While the details of Bayesian inference in either the "design" or "analysis" stage of PSA is straightforward, combining them the manner implied by expression (3) requires careful consideration. Sections 3 and 4 provide details of the specification of the components of expression (3) under a variety of propensity score implementations and analyses while Section 4.3 describes a corresponding two-stage computational procedure.

### 3 | BAYESIAN PROPENSITY SCORE DESIGN STAGE

#### 3.1 | Bayesian Propensity Score Estimation and Design Estimation Uncertainty

The parameter \( \nu \) represents the result of propensity score estimation and implementation for which ignorable treatment assignment is assumed, thus its probability distribution \( f(\nu|X, T) \) (representing a probability distribution of "designs") must be anchored to the model for estimating the PS. We expand the posterior distribution of \( \nu \) to reflect its dependence on parameters from the PS model:

\[
f(\nu|X, T) = \int f(\nu|\alpha, X, T) f(\alpha|X, T) d\alpha = \int f(\nu|\alpha, X, T) L(T|X, \alpha) \pi(\alpha) d\alpha,
\]

where \( L(T|X, \alpha) \) represents a likelihood function for the treatment assignment mechanism (i.e., a propensity score model) and \( \pi(\alpha) \) represents a prior distribution for the unknown parameter \( \alpha \).

For illustration throughout this paper, we consider a simplistic propensity score model based on a generalized linear model \( L(T|X, \alpha) = \prod_{i=1}^{n} g(\alpha X_i)^{T_i} (1 - g(\alpha X_i))^{1-T_i} \), with a logit link function \( g(\alpha X_i) \), although the ensuing discussion will be relevant to any parametric propensity score model specification with unknown parameter \( \alpha \). Note that the propensity score for each observation, \( e_i \), is a deterministic function of \( (\alpha, X, T) \). In the case of a logistic propensity
score model, \( e_i = g(\alpha X_i) = \frac{e^{\alpha X_i}}{1 + e^{\alpha X_i}} \). A traditional Frequentist estimate of the propensity score may be obtained by replacing the unknown parameter \( \alpha \) with its maximum likelihood estimate. Instead, integrating with respect to \( \alpha \) in expression (4) may be thought of as simulating from the posterior “predictive” distribution of propensity scores for each individual [25]. From this point forward, we refer to \( \alpha \) and the PS interchangeably with regards to describing a posterior distribution of quantities derived from the treatment assignment model.

Variability in the predictive distribution of propensity scores captures uncertainty in the estimation of the propensity scores, which we refer to as “design estimation uncertainty” (DEU). Propagation of DEU into causal effect estimates has been previously considered in Bayesian and non-Bayesian contexts [20, 5, 21].

### 3.2 Propensity Score Implementation and Design Decision Uncertainty

To lend formality to the heuristic represented with expression (3), we define \( \nu \) as a vector of length \( n \), where \( \nu = [\nu_1, \nu_2, \ldots, \nu_n] \) and \( \nu_i \) encodes the output of the PS implementation for individual \( i \). The parameter space for each \( \nu_i \) depends upon the choice of PS implementation procedure and the space of the entire vector \( \nu \) may receive additional restrictions based on the details of the implementation.

Delineation of the components of \( f(\nu \mid T, X) \) as expressed in expression (4) draws a distinction between uncertainty derived from estimation of the propensity score (DEU) and any additional source of uncertainty owing to the stochasticity inherent to the implementation, to which we refer as “design decision uncertainty” (DDU). The possibility of DDU is denoted by the expression \( f(\nu \mid \alpha, X, T) \), which can be a non-degenerate probability distribution, even for a fixed \( \alpha \) (and PS).

A PS implementation may or may not generate DDU. We refer to implementations that do not generate DDU as deterministic implementations, which produce only one possible value of \( \nu \) for each value of \( \alpha \). In these implementations, \( f(\nu \mid T, X, \alpha) \) is a point-mass function and all design uncertainty derives from DEU. In contrast, probabilistic implementations can produce multiple values of \( \nu \) for a single \( \alpha \), as dictated by the non-degenerate distribution \( f(\nu \mid T, X, \alpha) \). In the case of probabilistic implementations, both DDU and DEU contribute to design uncertainty.

This section specifies \( \nu \) and \( f(\nu \mid T, X, \alpha) \) in the context of five propensity score implementations: quintile stratification, nearest neighbor matching with replacement, caliper matching with replacement, and weighting for normalized inverse-probability-weighted (IPW) and doubly-robust (DR) estimators.

#### 3.2.1 Stratification

Stratification is a type of sub-classification performed on quantiles of the estimated propensity score distribution. For this implementation, \( \nu_i \) may be specified as a categorical variable, with \( \nu_i = q \) if individual \( i \) is assigned to strata \( q \), when the sample is stratified into one of \( q = 1, 2, \ldots, Q \) quantiles. We consider quintile stratification such that \( Q = 5 \), \( \nu_i \) takes on a value in \( \{1, 2, 3, 4, 5\} \) and \( \nu \) is defined on the space of possible allocations of \( n \) units into quintiles.

Stratification is a deterministic PS implementation method in that only one set of \( \nu \) is possible when conditioning on a fixed \( \alpha \), making \( f(\nu \mid T, X, \alpha) \) a point-mass function.

#### 3.2.2 Nearest Neighbor Matching With Replacement, implemented with a caliper

Nearest neighbor (NN) matching with replacement considers treated observations one at a time and matches each to the control observation(s) (ratio of matching decided \textit{a priori}) with the closest propensity score. Controls are allowed to be matched to multiple treated observations, and thus may appear multiple times in the matched set. By implementing NN
matching with a caliper, the pool of possible matches is limited by the caliper width, preventing unsuitable matches and instead pruning treated observations with propensity scores too removed from the distribution of control propensity scores.

In this implementation (and all matching with-replacement implementations), $v_i$ may be defined as the frequency weight for observation $i$, which is calculated by standardizing the frequency of inclusion of each observation to sum to the number of unique observations within each treatment arm in the matched set. Thus $v_i$ is defined on the space of real numbers bounded above by the maximum (across treatments) number of observations within each treatment arm.

As the case with stratification, NN matching with replacement is a deterministic implementation yielding one matched set for each set of propensity scores and a point-mass function $f(v|T, X, \alpha)$.

### 3.2.3 Caliper Matching With Replacement

In addition to NN matching, we also consider matching with replacement using a caliper, which we refer to as “caliper matching.” While this implementation is not widely used in propensity score literature, we incorporate it specifically as a stochastic counterpart to the NN algorithm of Section 3.2.2. The difference is that the NN algorithm chooses matches deterministically (closest based on PS distance) whereas the caliper matching algorithm chooses matches randomly among the candidates contained within the caliper.

Within the context of caliper matching with replacement, $v$ is defined in the same manner as NN matching with replacement. However, the random choice of control matches within the caliper renders this a probabilistic implementation; even for a fixed $\alpha$, there may still be variability in $v$ owing to the random selection of matches. $f(v|T, X, \alpha)$ is a probability distribution without an easily obtainable closed-form solution, but draws may be taken via iteratively performing the caliper matching algorithm multiple times conditional on the same $\alpha$.

### 3.2.4 Weighting for IPW and DR estimators

In contrast to matching and subclassification, weighting implementations utilize the propensity score to create a “pseudo-population” of observations in the treatment group, control group, or both in order to balance covariate distributions between the two groups. Inverse probability weighting (IPW) assigns weights based the following transformation of the propensity score:

$$w_i = \frac{T_i}{\hat{e}_i} + \frac{1 - T_i}{1 - \hat{e}_i}$$

For IPW, $v_i = w_i$, living on the space of positive real numbers. Since weights are created from a one-to-one transformation of a given a set of PS, the implementation is deterministic and $f(v|T, X, \alpha)$ is point-mass. This applies whether the weights are deployed in a standard IPW analysis or in tandem with an outcome model specification towards construction of a doubly-robust estimator.

### 4 BAYESIAN ANALYSIS STAGE INTEGRATING OVER DESIGN UNCERTAINTY

This section discusses marginalization of design uncertainty formulated in Section 3.2 into the posterior distribution of causal effects obtained in the analysis stage. Using $v$ to generically represent the output of any of the design stages from
Section 3, we expand expressions (3) and (4) as:

\[
f(\Delta | T, X, Y) = \int f(\Delta | T, X, Y, \nu) f(\nu | T, X) d\nu \tag{6}
\]

\[
\propto \int f(\Delta | T, X, Y, \nu) \int f(\nu | \alpha, X, T) f(\alpha | X, T) d\alpha d\nu \tag{7}
\]

\[
\propto \int_{\nu} f(\Delta | T, X, Y, \nu) \int_{\alpha} f(\nu | \alpha, X, T) \pi(\alpha) \prod_{i=1}^{n} L(T_i | X_i, \alpha) \ d\alpha d\nu \tag{8}
\]

The posterior distribution of the treatment effect conditional on the design is represented with \( f(\Delta | T, X, Y, \nu) \), and variability in this distribution is defined as "Analysis Estimation Uncertainty" (AEU). The functional form of \( f(\Delta | T, X, Y, \nu) \) is chosen a priori, depending upon the form of \( \nu \) chosen in the design stage as well as choice of analysis procedure. Some implementations permit analysis with use of parametric models defined conditional on \( \nu \). Other implementations, in particular those based on weighting, dictate a specific form of the analysis stage by using weighted estimators for \( \Delta \) which are not generally expressed with a parametric model. This distinction has important implications for conducting Bayesian inference, and the following sections describe each setting as it relates to developing an expression for \( f(\Delta | T, X, Y, \nu) \).

4.1 Analysis stages where \( \Delta \) is estimated with a parametric model

We first consider settings where the analysis stage consists of estimating a parametric outcome model, conditioned on \( \nu \). Following propensity score stratification, for example, the analysis stage may be performed with a parametric outcome model having the likelihood function \( L(Y | T, X, Y, \nu, \theta) \), where \( \Delta \) is defined as a function of the data and unknown parameters \( \theta \). Allowing definition of the conditional posterior distribution of \( \Delta \) from expression (6) as:

\[
f(\Delta | T, X, Y, \nu) = \int f(\Delta | T, X, Y, \nu, \theta) f(\theta | T, X, Y, \nu) d\theta \tag{9}
\]

where \( f(\theta | T, X, Y, \nu) = L(Y | T, X, Y, \nu, \theta) \pi(\theta) \) is the posterior distribution of the outcome model parameters under prior distribution \( \pi(\theta) \). For illustration, we consider \( f(\cdot | \cdot) \) to be a linear regression model with \( \theta = (\beta, \sigma^2) \) representing, respectively, a vector of regression coefficients and a conditional variance parameter. In this paper, we only consider a parametric outcome model in the case of stratification, where \( \nu \), a factor variable indicating stratum membership, is included as a covariate in the outcome model, along with an interaction with the treatment. In this case, the causal effect \( \Delta \) is a function of \( \theta \) and observed data.

4.2 Analysis stages where \( \Delta \) is estimated with an estimator with known asymptotic properties

Some PS implementations, in particular those based on weighting, do not lend themselves to estimation of \( \Delta \) with a parametric outcome model. The form of the estimator depends on the definition of the weights, which we illustrate with implementations of caliper matching with replacement, NN matching with replacement, IPW, and a simple DR estimator (see Table 1 for weights and weighted estimators of these implementations). The lack of a parametric model
for $\mathbf{Y}$ and corresponding likelihood expression requires a different approach for specifying $f(\Delta|\mathbf{T}, \mathbf{X}, \mathbf{Y}, \nu)$ in (6) and propagating design uncertainty into estimation of causal effects.

One option utilized in past literature [21] is specification of $f(\Delta|\mathbf{T}, \mathbf{X}, \mathbf{Y}, \nu)$ in expression (10) with the known asymptotic distribution of the estimator for $\Delta$, conditional on one realization of $\nu$, which typically have the form:

$$
f(\Delta|\mathbf{T}, \mathbf{X}, \mathbf{Y}, \nu) \sim N(E(\Delta^*(\nu)), Var(\Delta^*(\nu)))
$$

(10)

$E(\Delta^*(\nu))$ and $Var(\Delta^*(\nu))$ represent the asymptotic mean and variance of $\Delta$, conditional on a single design. The exact form of $E(\Delta^*(\nu))$ and $Var(\Delta^*(\nu))$ will depend on the implementation. Table 1 lists the specific expressions for asymptotic mean and variance estimators used to approximate $f(\Delta|\mathbf{T}, \mathbf{X}, \mathbf{Y}, \nu)$ for BPSA performed with caliper matching, NN matching, IPW and DR estimation.

When using asymptotic distributions of the form in (10) to approximate $f(\Delta|\mathbf{T}, \mathbf{X}, \mathbf{Y}, \nu)$, evaluation of the posterior in (6) can be construed as evaluation of the asymptotic posterior distribution of $\Delta$, marginalized over design uncertainty. Incorporating asymptotic approximations in the BPSA framework has drawbacks depending on asymptotic estimators available to describe the conditional distribution. Asymptotic estimators with poor finite-sample performance might propagate similar properties into posterior estimates of $\Delta$.

### 4.3 Outline of Computational Procedure for Marginalizing over Design Uncertainty

Here we outline a Markov-chain Monte Carlo (MCMC) procedure for evaluating the posterior distribution of causal effects, marginalized over design uncertainty as defined in expression (10), specific instances of which can be found in previous literature [21, 5, 6, 19].

#### 4.3.1 Drawing from the posterior predictive distribution of $\nu$

In the first stage of BPSA, multiple draws are taken from $f(\nu|\mathbf{T}, \mathbf{X})$ using the following procedure:

1. Obtain a sample of $K$ draws from the posterior distribution of the parameters of the propensity score model, $\alpha$. This can be accomplished with standard MCMC routines, for example, with the R package MCMCpack.
2. For each of the $K$ draws from posterior distribution of $\alpha$, obtain a set of $n$ propensity scores with the use of covariate information $\mathbf{X}$ and an expit transform. Steps 1 and 2 in combination may be conceived as taking $K$ samples from the posterior predictive distribution of propensity scores.
3. For deterministic PS implementations, use the $K$ draws from the posterior predictive distribution of propensity scores to perform $K$ propensity score implementations, each representing a design stage $\nu_k$, indexed by $k = 1, 2, ..., K$. For stochastic PS implementations, perform the implementation $R$ times conditional on each set of PS for all $K$ draws, resulting in $R \times K$ values of $\nu$.

The output of the first stage, which may be performed in its entirety independent of outcome information, produces either $K$ or $R \times K$ draws of $\nu$ from its marginal distribution, $f(\nu|\mathbf{T}, \mathbf{X})$, each representing a different "design." Going into the analysis stage, we utilize the notation $\nu_k$ to describe the output of a design stage conditional on a single set of PS, reflecting a deterministic implementation. Computationally, if the PS distribution is explored well enough ($K$ is large), $R$ does not need to be too large (and may even be set to 1) as the MCMC algorithm re-visits the same sets of PS multiple
times and the variability of \( f(\nu | T, X, \alpha) \) is explored in that way.

### 4.3.2 Drawing from the conditional posterior distribution of \( \Delta \)

Estimation in the analysis stage is conducted conditional on each of the simulated values of \( \nu_k \) from the design stage. Specifically, for each value of \( \nu_k \), \( k = 1, 2, \ldots, K \):

4. Draw \( S \) samples from the conditional posterior distribution \( f(\Delta | T, X, Y, \nu_k) \)
   
   **When the analysis stage entails a parametric distribution for \( f(\theta | T, X, Y, \nu_k) \)**

4.1a. Take \( S \) draws from the posterior distribution of \( \theta = [\beta, \sigma^2] \), a vector of parameters with the posterior distribution \( f(\theta | T, X, Y, \nu) = L(Y | T, X, \theta, \nu) \pi(\theta) \). Let \( \theta_{sk} \) represent one such draw, with \( s = 1 \ldots S \). Again, this may be accomplished with standard MCMC procedure and a packages such as MCMCpack.

4.2a. Each draw of \( \theta_{sk} \) may be transformed into \( \Delta_{sk} \) (ex: for stratification, \( \Delta \) is a linear combination of coefficients of a regression fit where treatment assignment is interacted with strata membership).

   **When an asymptotic approximation is used for the distribution of \( f(\Delta | T, X, Y, \nu_k) \)**

4.1b. Calculate \( E(\Delta^*(\nu_k)) \) and \( Var(\Delta^*(\nu_k)) \). 

4.2b. Draw \( S \) samples from \( N(E(\Delta^*(\nu_k)), Var(\Delta^*(\nu_k))) \) of \( \Delta_{sk} \), \( s = 1 \ldots S \).

With either step 4a or 4b of the analysis, the end result will be \( K \times S \) draws from the posterior distribution \( f(\Delta | T, X, Y) \), which is marginalized over design uncertainty.

If interest lies primarily in estimating the posterior mean and variance of \( \Delta \), instead of retaining \( S \) draws from \( f(\Delta | T, X, Y, \nu_k) \) for each \( k \), it is only necessary to save \( \Delta_k = E(\Delta | T, X, Y, \nu_k) \) and \( \sigma^2_k = Var(\Delta | T, X, Y, \nu_k) \). Then \( E(\Delta | T, X, Y) \) may be calculated by averaging over all \( \Delta_k \) and \( Var(\Delta | T, X, Y) \) may be calculated using Rubin’s combining rules [29] which considers both variability in \( \Delta_k \) as well as averaging over \( \sigma^2_k \).

### 5 SIMULATION STUDY AND COMPARISON WITH TRADITIONAL PSA

We conduct a simple simulation study to compare the BPSA approach of Section 4 against traditional PSA and investigate the performance of each approach amid varying degrees of design uncertainty. 200 data sets are simulated, each having \( n = 1000 \) observations of 15 uncorrelated normally-distributed covariates with mean 0 and variance 1. Treatment assignment \( T \) is simulated from a Bernoulli distribution with probability of treatment specified by the logistic regression described in Section 3.1, with intercept set to zero and coefficients \( \alpha \) set to 0.75 for all covariates. Outcomes, \( Y \), are simulated from a linear regression model of the form \( Y = 0.1 + \Delta T + \beta X + \epsilon \), with \( \Delta \) set to 1.5, and \( \beta \) set to [0.2, 0.3, 0.4] for the first three covariates and 0 for the remaining 12 covariates. \( \epsilon \) is a normally-distributed random error with mean 0 and variance 1. Thus simulated datasets were constructed to contain, on average, equal proportions of treated and control observations where only three of the 15 covariates were true confounders leaving the remaining 12 as “instrumental variables” in the sense that they are related to treatment assignment but not the outcome.

#### 5.1 BPSA and PSA procedures

We analyzed simulated datasets with BPSA (procedure described in Section 4.3 with \( K = 1000 \), \( S = 200 \) and \( R = 1 \) for caliper matching) and standard PSA (which conditions analysis on the MLE of the PS) in the context of two different PS
model specifications and six PS implementations. Both PS model specifications correctly specify the functional form of the treatment assignment mechanism (i.e., logistic regression models with linear covariate adjustment) and include all three true confounders. They differ in terms of the number of instrumental variables included; the "0-instrument model" includes only the 3 true confounders and represents a setting with low DEU, and the "5-instrument model" adds 5 instrumental variables, representing a setting with comparatively higher DEU. The performance contrast between PSA and BPSA in the environments of low and high design uncertainty is of particular interest.

With each of the two PS model specifications, we employ the following PS implementations: 1-1 caliper matching with a caliper of 0.5, 1-5 caliper matching with a caliper of 0.5, 1-1 NN matching with a caliper of 0.5, quintile stratification, and weighting with IPW and DR estimators. Different ratios used for caliper matching were designed to influence the amount of DDU in the implementation; increasing the number of control matches decreases the amount of DDU.

Under PSA, the analysis stage following stratification is performed with parametric modeling of the conditional distribution of $\Delta$ (see Section 4.1) while the analysis stage following all other implementations involve asymptotic approximation of the conditional distribution of $\Delta$ (see Section 4.2).

Under PSA, the analysis stage after all implementations utilizes asymptotic estimators for both the treatment effect and variance, where "robust" variance estimators designed to account for some design uncertainty are employed after caliper matching (with both ratios), NN matching and IPW estimation and a standard OLS regression is performed for stratification utilizing the same linear model as BPSA (which interacts treatment and strata membership). Details of all treatment effect and variance estimators may be found on Table 1.

### 5.2 | BPSA Accounting for Design Uncertainty vs. Traditional PSA

Table 3 compares the performance of BPSA and PSA under all six implementations and two PS model specifications based on the following metrics: bias of point estimates (posterior mean for BPSA, MLE estimation for PSA), empirical variance of point estimates across replications, mean squared error (MSE) of point estimates, coverage of 95% uncertainty intervals (posterior credible intervals for BPSA, confidence intervals for PSA), and the average estimated variance of point estimates (posterior variance of $\Delta$ for BPSA, asymptotic variance for PSA).

Contrasts between PSA and BPSA vary across choice of PS implementation, but note that for all implementations, analysis under the 5-instrument model resulted in higher uncertainty (quantified by empirical variance as well as average estimated variance) than under the 0-instrument model, owing to the increase in DEU. We provide a more detailed discussion of these contrasts within matching implementations, followed by stratification and finally IPW and DR.

#### 5.2.1 | Matching

BPSA produces similar average bias but lower empirical variance compared to PSA for all matching implementations under both PS models. As a result, BPSA estimates exhibit lower MSE than PSA for all matching implementations. The decrease in variability of point estimates may be attributed to the manner in which BPSA averages conditional point estimates for $E(\Delta|X, T, Y, \nu)$ over multiple matched sets, resulting in more stable marginal point estimates for $E(\Delta|X, T, Y)$.

For 1-1 caliper matching and 1-1 NN matching, BPSA estimates similar average variance to PSA under the 0-instrument PS model, and smaller average variance than PSA under the 5-instrument model. For 1-5 caliper matching, BPSA estimates smaller average variances than PSA under both PS models. As explored further in Section A.2, performing caliper matching with a 1-5 ratio decreases the randomness in the probabilistic algorithm which generates DDU. BPSA responds to the decrease in DDU by estimating a lower treatment effect variance, while PSA estimates the same
variance for both 1-1 caliper matching and 1-5 caliper matching under either PS model.

For NN matching, analysis under the 5-instrument model creates more dramatic differences between BPSA and PSA than analysis under the 0-instrument model, while this is not the case with either caliper matching implementation. We explore the additional sensitivity to design uncertainty for 1-1 NN matching in Section A.1.

Despite for the most part estimating smaller variances than PSA, BPSA-calculated uncertainty intervals result in higher coverage. This is due to the lower empirical variability displayed by treatment effects estimated under BPSA for reasons discussed previously. BPSA-constructed intervals also result in over-coverage, suggesting the possibility of more appropriate variance estimators than the asymptotic ones employed here.

5.2.2 | Stratification

Stratification exhibits little contrast between BPSA and PSA with regard to any of the performance metrics under either PS model. Marginalizing over all possible stratification designs with BPSA does not produce treatment effect or variance estimates which substantially differ from conditioning on a single design with PSA. This is due to quintile stratification exhibiting little sensitivity to design uncertainty, which we explore further in Section A.1.

5.2.3 | Weighting with IPW or DR estimation

As with stratification, the weighting implementation utilized with IPW results in minimal contrast between BPSA and PSA with regard to bias, empirical variance or MSE. Average estimated variance only displays a contrast between BPSA and PSA under the 5-instrument model, where the BPSA-estimated marginal variance is lower than the PSA-estimated robust sandwich variance, leading to evidence of under-coverage.

The DR estimator does not display much contrast between PSA and BPSA under the 0-instrument PS model, but BPSA displays more erratic performance under the 5-instrument PS model, producing higher bias, empirical variance, MSE and average estimated variance compared to PSA. This is due to the construction of the DR estimator, which not only utilizes the same weights as the IPW estimator, but augments them with PS-weighted predictions from a potential outcome model (which includes 5 instrumental variables and is thus misspecified when the DR estimator is calculated under the 5-instrument PS model). Since the DR estimator incorporates propensity scores in multiple ways, it is much more sensitive to perturbations in the estimated PS. BPSA averages over possibly extreme conditional treatment effect estimates by marginalizing over the distribution of PS, creating more erratic point estimates than PSA.

Figure 1 provides a visual representation of the spread of estimated treatment effect bias across replications when various implementations are performed with PSA or BPSA, echoing the trends discussed above.

6 | INVESTIGATING THE EFFECT OF HIGH POWER PLANT EMISSIONS ON AMBIENT POLLUTION

Cummisky et. al. [30], evaluated the association between long-term exposure to emissions from coal-fired power plants and Ischemic Heart Disease (IHD) hospitalizations among Medicare beneficiaries. One important feature of this analysis was the way in which the analysis incorporated ambient fine particulate matter (particles less than 2.5 micrometers in diameter, denoted PM). Ambient PM concentrations are expected to be derived in part from coal-fired power plant emissions in some regions, and PM is known to be associated with a variety of adverse health outcomes [31]. While the primary analysis in Cummisky et. al.[30] investigated the link between coal emissions and IHD without adjusting for PM,
a secondary analysis regarded PM as an adjustment covariate and briefly evaluated whether PM could be ruled out as a possible mediator of the relationship between coal emissions and IHD hospitalizations. Here we revisit the secondary analysis of Cummiskey et. al. [30], deploying the methods described in Section 5.2 to investigate the extent to which a binary metric of high/low coal emissions exposure causally impacts annual average PM. Details in Cummiskey et. al. [30] describe the creation of a binary metric of coal power plant exposure derived using a reduced-complexity chemical transport model (InMap) [32], as well as the data-fusion derived estimates of PM [33].

We examine the effect of elevated coal emissions on ambient particle concentrations measured at 22,723 zip codes in the Northeast, Southwest and Industrial Midwest regions of the United States. The analysis adjusts for 17 possible confounders, including location of zip code (latitude and longitude), climate (temperature and humidity), population density, demographics (e.g. racial makeup, education, household income, gender) as well as the makeup of the residential areas in each zip code (e.g. urban/rural, real estate value). A full list of covariates considered can be found in Table 4. Further details on consolidating the zip-code-level covariates (retrieved from 2000 US Census data) may be found in Cummiskey et. al [30].

Among the 22,723 observations in this dataset, 7,211 are classified as “exposed” (to high coal emissions) and 15,512 are control observations. Figure 2 visualizes the overlap of estimated propensity scores among treated and control observations using maximum likelihood estimates of the PS, and Table 4 contains average values of included confounders compared across exposure status. On average, control zip codes are located further West, have lower population densities, are less urban and more rural, and exhibit a lower median household income and higher percent poverty.

Analysis was performed with the PSA and BPSA procedures described in Section 5.2, compared within the contexts of quintile stratification, caliper matching with replacement, NN matching with replacement (both with caliper = 0.5 and a 1-1 ratio), and weighting with IPW and DR estimators. In this analysis, we focus on estimating the ATE, but employ caliper and NN matching methods which measure the ATT for purposes of illustration.

Estimated treatment effects, 95% confidence intervals and analysis details may be found in Table 5, and are depicted graphically in Figure 3. Presented on the same scale for comparison are also estimates and intervals constructed via fully adjusted and unadjusted linear regressions.

While point estimates of treatment effects are nearly identical between PSA and BPSA procedures in the context of DR, IPW and stratification, BPSA point estimates are lower than PSA point estimates in the context of NN matching and caliper matching. This may be attributed to the large pool of control observations available compared to treated observations. Both matching algorithms on average use only 50% of the available observations in their treatment effect estimation, and selection of controls into the matched set is sensitive to design uncertainty arising from both DEU (the PS model includes many possible confounders, some of which may be instrumental variables) as well as DDU (in the case of caliper matching. By averaging over multiple matched sets, BPSA was shown to produce more stable estimates of the treatment effect in Section 5 and it is reflected in this analysis via the contrast between PSA and BPSA-estimated treatment effects.

IPW and stratification produced similar point estimates for the average treatment effect ($\approx 1.75$) while both matching implementations display a lower estimation of the ATT and the DR estimator falls in the middle. Both the fully adjusted and unadjusted linear regressions estimate lower treatment effects than any of the propensity score analysis methods. This may explain in part the DR estimate falling below the IPW estimate.

As witnessed in the simulation study, BPSA produces intervals similar in width (due to estimating similar SEs) to PSA when performed with stratification, and larger intervals when performed with DR. Only under IPW does BPSA create narrower intervals than PSA, a result which occurred in the simulation study only when instruments were included in the PS model. The most unexpected result occurs under 1-1 NN and caliper matching, where BPSA produces slightly wider
intervals (larger estimated SEs) than PSA, a result not observed in the simulation study. This is due to the difference in proportion of treated observations, which was on average equal to the proportion of control observations in the simulated data but much lower than the proportion of control observations in the power plant data. The high number of control observations makes construction of the matched set more sensitive to perturbations in the PS, and the marginalization procedure of BPSA accounts for this in its SE estimate.

Overall, the results of the analysis provide the consistent message that elevated exposure to coal-fired power plant emissions causally increases the overall annual concentration of ambient fine particulate matter, underscoring the care with which the secondary analysis in Cummiskey et al. [30] should be interpreted.

7 | DISCUSSION

The framework developed by this paper creates a foundation for understanding propensity score analysis within the context of Bayesian inference, underpinning previous work on this topic [20, 5, 21]. The development of a parameter linked to the output of the design stage allows definition of a distribution of “designs”, even when non- or semi-parametric PS implementations are utilized. Marginalization over this distribution ensures propagation of full design uncertainty - not just PS estimation uncertainty - to the posterior distribution of treatment effects.

Our work shares conceptual similarities to that of Branson [34], who compares the analysis stage of a PSA to that of a RCT with randomization restricted to pre-determined bounds on covariate balance. However, while Branson describes a conditional distribution of treatment effects which accounts for certain limitations placed on PS implementations, the paper does not address uncertainty arising from models or implementations utilized in the design stage nor how to propagate it into estimation of the treatment effect. Furthermore, Branson primarily focuses on PS matching while this paper extends to any PS implementation.

The simulation studies herein showed that BPSA performance is highly variable across choice of implementation. When used with matching implementations, BPSA calculated more stable treatment effect estimates and more efficient variances than PSA but constructed intervals resulted in over-coverage. BPSA performed with stratification or IPW resulted in similar treatment effect and variance estimates to PSA with slight under-coverage issues while BPSA performed with the DR estimator actually created higher bias and MSE of treatment effect estimates compared to PSA.

One explanation for BPSA’s poor performance with IPW and DR is that the role of weighted estimators in two-stage BPSA is still not well understood and should be an area of future study. Since no alternatives were available, an ad hoc asymptotic distribution was used to approximate $f(\Delta | T, X, Y, \nu)$ for the IPW estimator where the variance estimator was developed for a weighted least squares regression and requires the assumption that inputted weights are “known” to be the inverse of heteroskedastic variability in outcome.

One direction forward is further exploration of sub-setting implementation equivalents to weighting, such as a probabilistic pruning method [6] or the incorporation of weights as parameters in a prior for the distribution of treatment effects [19]. Both implementations include fully-specified outcome distributions, and have been demonstrated to achieve more stable treatment effect estimates compared to standard weighting procedures performed with PSA [19, 6]. Other extensions could incorporate non-parametric, machine-learning [35] or flexible [18] PS estimation methods which have become recently popular.

Though propensity score analysis is widely used in various fields including epidemiology and econometrics, notions of design uncertainty are rarely considered explicitly. Bayesian propensity score analysis provides a versatile computational procedure which marginalizes over all components of design uncertainty in a clearly defined manner. Though the performance of BPSA varies across implementations, it has been regarded as an attractive alternative to conditioning
inference on a single estimate of the propensity score [5]. Explication and exploration of the ideas presented here can hopefully ground future work investigating how Bayesian methods can add to the existing literature on propensity score analysis.

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Code to implement the simulation study and power plant analysis are available at: https://github.com/shirleyxiao/Uncertainty-in-the-Design-Stage-of-Two-Stage-Bayesian-Propensity-Score-Analysis

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A CLOSER LOOK AT COMPONENTS OF DESIGN UNCERTAINTY

A.1 Sensitivity to Design Uncertainty under Different Implementations

Simulations in Section 5.2 noted how implementation algorithms display varying degrees of sensitivity to design uncertainty, leading to some implementations displaying a larger contrast between BPSA and PSA under a PS model which generated more DEU. This section explores this phenomenon in more detail, utilizing the same 200 datasets and 0-instrument and 5-instrument PS models described in Section 5.2 and adding a new model with 12 instrumental variables to yield even more DEU. Table 6 shows how on average PS distributions for each observation becomes more variable (higher $\text{Var}(\Delta|T, X, Y)$) as more instrumental variables are added to the PS model. Conditional upon draws from the three PS models, five PS implementations are explored: 1-1 caliper matching with a caliper of 0.5, 1-1 NN matching with a caliper of 0.5, quintile stratification and weighting with IPW and DR estimators.

When an implementation is sensitive to design uncertainty, higher variability in the distribution of PS (DEU) creates higher variability in the distribution of $\nu$ and, ultimately, in the estimation of $\Delta$. Implementations which are less sensitive to design uncertainty calculate $\nu$ in a manner that exhibits very little in response to perturbations of the PS.

To investigate in detail, we decompose the marginal variance of $\Delta$ according to the law of total variance:

$$\text{Var}(\Delta|T, X, Y) = E(\text{Var}(\Delta|T, X, Y, \nu)) + \text{Var}(E(\Delta|T, X, Y, \nu))$$ (11)

While $E(\text{Var}(\Delta|T, X, Y, \nu))$ averages over the conditional variances of $\Delta$ which account for uncertainty in the analysis stage (AEU), $\text{Var}(E(\Delta|T, X, Y, \nu))$ describes the variability of conditional treatment effect estimates across the distribution of $\nu$. $\text{PROP}_{DU} = \frac{\text{Var}(E(\Delta|T, X, Y, \nu))}{\text{Var}(\Delta|T, X, Y)}$ describes the percentage of variation in $\Delta$ attributable to design uncertainty.

Table 6 shows that $\text{PROP}_{DU}$ increases with the inclusion of more instrumental variables in the PS model under all implementations. Stratification displays the lowest $\text{PROP}_{DU}$ of any implementation for any PS model, making it the least sensitive implementation to design uncertainty. NN matching, on the other hand, may be considered very sensitive to design uncertainty as its $\text{PROP}_{DU}$ is higher than that of all other implementations under the 0- and 5-instrument models, and is only lower than IPW under the 12-instrument model.
Under the 0- and 5-instrument models, IPW and 1-1 caliper matching display similarly moderate \( \text{PROP}_{DU} \), both lower than that of NN matching with replacement but higher than stratification. Under the 12-instrument model, however, \( \text{PROP}_{DU} \) for IPW becomes very high while \( \text{PROP}_{DU} \) for caliper matching remains at a low level. This would indicate that IPW is much more sensitive to high design uncertainty (12-instrument model) than low or moderate design uncertainty. Caliper matching, as a probabilistic algorithm, generates design uncertainty from both DEU and DDU. Since the caliper is set at a generous 0.5, perturbations in the PS do not cause dramatic changes to the pool of control matches assigned to each treated observation. Randomness of the matching algorithm (DDU) actually plays a larger role in design uncertainty for caliper matching, which explains why \( \text{PROP}_{DU} \) for caliper matching is the second-highest of all implementations under the 0-instrument model, yet increases only 11% (the lowest increase of all implementations) in response to higher DEU from the 12-instrument PS model.

The DR estimator is a special case. While its \( \text{Var}(\Delta | T, X, Y) \) is much higher than any other implementation under the 12-instrument model, the \( \text{PROP}_{DU} \) remains relatively moderate. This is due to the proportional increase in analysis uncertainty (captured by \( E(\text{Var}(\Delta | T, X, Y, \nu)) \)) from potential outcome models which are increasingly mis-specified since they include the same number of instrumental variables as the PS model.

A.2 | DDU in Caliper matching

This section examines how the quantity of DDU generated by the probabilistic implementation of caliper matching (as described in Section 3.2.3) varies in response to adjusting the ratio of controls matched to each treated observation as well as downstream consequences of this for the marginal distribution of \( \Delta \).

Caliper matching with a caliper of 0.5 was performed with treatment-to-control ratios of 1-1, 1-2 and 1-5 conditional on the same set of PS drawn from the 0-instrument PS model described in Section 5.1. BPSA was performed with \( K = 250 \) and \( R = 15 \), in order to explicitly calculate the variability of \( f(\nu | T, X, \alpha) \). Results of these three implementations may be found on Table 7, which contains the same metrics described for Table 6 as well as two additional: \( \text{Var}(\nu | T, X, \alpha) \), defined as the variance of within-observation frequency weights \( \nu \) calculated by caliper matching over 15 iterations performed on the same set of propensity scores, and the same quantity after marginalization over the posterior distribution of \( \alpha \), \( \text{Var}(\nu | T, X) \).

Thus \( \text{Var}(\nu | T, X, \alpha) \) may be thought of as a measure of DDU, and displays an inverse relationship with the number of control observations in the ratio as it describes how the randomness of matches decreases when we get closer to matching the entire pool of possible controls. \( \text{Var}(\nu | T, X) \), meanwhile, is a measure of design uncertainty which draws from DEU and DDU. Since DEU is “held constant” by performing all three implementations on the same set of propensity scores, the difference between \( \text{Var}(\nu | T, X) \) and \( \text{Var}(\nu | T, X, \alpha) \) is constant across the changing ratios.

\( \text{Var}(\Delta | T, X, Y) \) and \( \text{PROP}_{DU} \) both decrease as the ratio increases from 1-1 to 1-5, indicating how decreasing DDU not only decreases the variability of the marginal posterior distribution of \( \Delta \), it decreases the proportion that design uncertainty contributes to that variability.

B | TABLES AND FIGURES
Mean + 95% density of estimated ATE bias over replications

**FIGURE 1** Mean estimated ATE and density of 95% estimates across replications
**Figure 2** Histogram of estimated propensity scores under treated and control observations
**FIGURE 3** Estimated ATEs and constructed 95% confidence/credible intervals created via PSA and BPSA
| Table 1 | BPSA and PSA estimators for the conditional distribution of $\Delta$ |
|---------|-------------------------------------------------------------|
| **BPSA:** $E(\Delta|T, X, Y, \nu)$ | Stratification | DR | IPW/NN matching/Caliper matching |
| **PSA:** $\hat{\Delta}$ | Linear combination of parameters $\beta$ of the linear outcome model | See Table 1 in Robins et. al. [36] | $\frac{1}{n} \sum_{i=1}^{n} Y_i T_i \nu_i - \frac{1}{n} \sum_{i=1}^{n} Y_i (1 - T_i) \nu_i$ |
| **BPSA:** $\mathbb{V}ar(\Delta|T, X, Y, \nu)$ | Empirical conditional posterior variance of $\Delta$ | See expression (21) in Lunceford and Davidian [37] | Variance estimator for weighted regression with known weights |
| **PSA:** $\hat{\mathbb{V}}ar(\hat{\Delta})$ | Transformation of variance-covariance matrix for $\beta$ | Huber-White sandwich estimator | |

NN matching = Nearest neighbor matching
DR = Doubly robust estimator
W = matrix of weights
|                         | 0 instrumental variables | 5 instrumental variables |
|-------------------------|--------------------------|--------------------------|
|                         | PSA          | BPSA         | PSA          | BPSA         |
| **Bias (empirical variance)** |             |              |              |              |
| 1-1 Caliper matching    | 0.03 (0.007) | 0.04 (0.004) | 0.03 (0.011) | 0.04 (0.008) |
| 1-5 Caliper matching    | 0.03 (0.007) | 0.04 (0.004) | 0.03 (0.009) | 0.04 (0.008) |
| 1-1 NN matching         | 0.00 (0.008) | 0.01 (0.005) | 0.00 (0.016) | 0.01 (0.011) |
| Stratification          | 0.03 (0.005) | 0.04 (0.004) | 0.04 (0.007) | 0.05 (0.007) |
| IPW                     | 0.01 (0.005) | 0.00 (0.005) | 0.01 (0.008) | 0.00 (0.008) |
| DR                      | 0.00 (0.005) | 0.02 (0.005) | -0.01 (0.009) | 0.04 (0.010) |
| **MSE**                 |             |              |              |              |
| 1-1 Caliper matching    | 0.008       | 0.006       | 0.011       | 0.009       |
| 1-5 Caliper matching    | 0.008       | 0.006       | 0.010       | 0.009       |
| 1-1 NN matching         | 0.008       | 0.005       | 0.016       | 0.011       |
| Stratification          | 0.006       | 0.006       | 0.008       | 0.009       |
| IPW                     | 0.005       | 0.005       | 0.008       | 0.008       |
| DR                      | 0.005       | 0.005       | 0.009       | 0.011       |
| **Average estimated variance** |             |              |              |              |
| 1-1 Caliper matching    | 0.009       | 0.009       | 0.012       | 0.011       |
| 1-5 Caliper matching    | 0.009       | 0.007       | 0.012       | 0.010       |
| 1-1 NN matching         | 0.011       | 0.011       | 0.018       | 0.015       |
| Stratification          | 0.005       | 0.005       | 0.007       | 0.008       |
| IPW                     | 0.006       | 0.006       | 0.009       | 0.007       |
| DR                      | 0.006       | 0.007       | 0.013       | 0.023       |
| **Coverage**            |             |              |              |              |
| 1-1 Caliper matching    | 0.94        | 1.00        | 0.95        | 0.97        |
| 1-5 Caliper matching    | 0.96        | 1.00        | 0.94        | 0.98        |
| 1-1 NN matching         | 0.98        | 1.00        | 0.95        | 0.99        |
| Stratification          | 0.94        | 0.93        | 0.94        | 0.93        |
| IPW                     | 0.98        | 0.98        | 0.95        | 0.93        |
| DR                      | 0.98        | 0.99        | 0.97        | 1.00        |

PSA, propensity score analysis. BPSA, Bayesian propensity score analysis. SE, standard error. MSE, mean squared error. NN, nearest neighbor.
### Table 4
Application data: table of unadjusted averages of covariates across treatment

|                                | Unexposed | Exposed |
|--------------------------------|-----------|---------|
| Number of observations         | 15,512    | 7,211   |
| Avg. estimated PS              | 0.25      | 0.46    |
| Latitude                       | 38.30     | 38.37   |
| Longitude                      | -82.84    | -79.46  |
| County smoking rate            | 0.27      | 0.26    |
| Total population               | 10,791    | 13,231  |
| Percent residing in rural area | 0.58      | 0.49    |
| Percent of white residents     | 0.85      | 0.82    |
| Percent of African-American residents | 0.11   | 0.13    |
| PctHighSchool                  | 0.35      | 0.35    |
| Median household income        | 39,385    | 43,594  |
| Percent living below poverty threshold | 0.13   | 0.12    |
| Percent female                 | 0.51      | 0.51    |
| Percent of housing units occupied | 0.87   | 0.90    |
| Percent who have moved in past 5 years | 0.42   | 0.42    |
| Median house value             | 102,070   | 121,912 |
| Population per Sq. Mile        | 1,167     | 2,455   |
| Avg. temperature (2005)        | 286       | 286     |
| Avg. relative humidity (2005)  | 0.0086    | 0.0085  |

### Table 5
Estimated treatment effects of high power plant emissions on ambient pollution

|                                | ATE [95% CI] |
|--------------------------------|--------------|
| UNADJ regression               | 1.41 [1.35,1.47] |
| ADJ regression                 | 1.46 [1.41,1.51] |

|                                | BPSA        | PSA         |
|--------------------------------|-------------|-------------|
| NN matching                    | 1.63 [1.52,1.74] | 1.65 [1.56,1.74] |
| Caliper matching               | 1.59 [1.49,1.68] | 1.63 [1.55,1.70] |
| Stratification                 | 1.75 [1.67,1.84] | 1.76 [1.68,1.83] |
| IPW                            | 1.74 [1.64,1.85] | 1.74 [1.65,1.83] |
| DR                             | 1.68 [1.55,1.81] | 1.67 [1.57,1.77] |
TABLE 6  Marginal variances of \( \Delta \) and percentage of such attributable to design uncertainty calculated under five implementations and 3 PS models with varying instrumental variables

| Instrumental variables in the PS model | 0   | 5   | 12  |
|---------------------------------------|-----|-----|-----|
| \( \text{Var}(e|T, X) \)              | 0.0008 | 0.0016 | 0.0018 |
| **IPW**                               |     |     |     |
| \( \text{Var}(\Delta|T, X, Y, \nu) \) | 0.0067 | 0.0073 | 0.0132 |
| \( \text{PROP}_{DU} \)               | 23.9% | 30.1% | 72.0% |
| **DR**                                |     |     |     |
| \( \text{Var}(\Delta|T, X, Y, \nu) \) | 0.0074 | 0.0229 | 1.265 |
| \( \text{PROP}_{DU} \)               | 18.9% | 33.6% | 47.1% |
| **Stratification**                    |     |     |     |
| \( \text{Var}(\Delta|T, X, Y, \nu) \) | 0.0046 | 0.0078 | 0.0328 |
| \( \text{PROP}_{DU} \)               | 4.3% | 14.1% | 22.3% |
| **NN matching with replacement**      |     |     |     |
| \( \text{Var}(\Delta|T, X, Y, \nu) \) | 0.0110 | 0.0154 | 0.0315 |
| \( \text{PROP}_{DU} \)               | 31.8% | 43.5% | 64.8% |
| **Caliper matching with replacement** |     |     |     |
| \( \text{Var}(\Delta|T, X, Y, \nu) \) | 0.0091 | 0.0113 | 0.0159 |
| \( \text{PROP}_{DU} \)               | 24.2% | 30.1% | 35.3% |

\( \text{PROP}_{DU} \) = Proportion of \( \text{Var}(\Delta|T, X, Y) \) arising from design uncertainty

TABLE 7  Conditional and marginal variances of \( \nu \) and \( \Delta \) for caliper matching under a variety of ratios

| Ratio (treatment-control) | 1-1 | 1-2 | 1-5 |
|---------------------------|-----|-----|-----|
| \( \text{Var}(\nu|T, X, \alpha) \) | 0.27 | 0.26 | 0.19 |
| \( \text{Var}(\nu|T, X) \)       | 0.28 | 0.27 | 0.21 |
| \( \text{Var}(\Delta|T, X, Y) \) | 0.096 | 0.084 | 0.076 |
| \( \text{PROP}_{DU} \)           | 24.0% | 14.3% | 6.6% |

\( \text{PROP}_{DU} \) = Proportion of \( \text{Var}(\Delta|T, X, Y) \) arising from design uncertainty