

# Aggregation Trees

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**Summary** Uncovering the heterogeneous effects of particular policies or “treatments” is a key concern for researchers and policymakers. A common approach is to report average treatment effects across subgroups based on observable covariates. However, the choice of subgroups is crucial as it poses the risk of  $p$ -hacking and requires balancing interpretability with granularity. This paper proposes a nonparametric approach to construct heterogeneous subgroups. The approach enables a flexible exploration of the trade-off between interpretability and the discovery of more granular heterogeneity by constructing a sequence of nested groupings, each with an optimality property. By integrating our approach with “honesty” and debiased machine learning, we provide valid inference about the average treatment effect of each group. We validate the proposed methodology through an empirical Monte-Carlo study and apply it to revisit the impact of maternal smoking on birth weight, revealing systematic heterogeneity driven by parental and birth-related characteristics.

**Keywords:** *Causality, conditional average treatment effects, recursive partitioning, subgroup discovery, subgroup analysis.*

## 1. INTRODUCTION

Understanding the effects of a particular policy or “treatment” is a key concern for researchers and policymakers. Traditionally, the assessment of the policy’s actual effectiveness involves the identification and estimation of the Average Treatment Effect (ATE), a parameter that quantifies the average impact of the policy on the reference population (see, e.g., Angrist and Pischke, 2009; Imbens and Rubin, 2015). However, while the ATE is straightforward to interpret, it lacks information regarding effect heterogeneity and therefore does not allow us to explore the distributional impacts of the policy, which hold significant importance for decision-making when the social welfare criterion representing the preferences of the policymakers is not “utilitarian” (Kitagawa and Tetenov, 2021).

A common approach to tackle effect heterogeneity is to report the ATEs across different subgroups defined by observable covariates. These Group Average Treatment Effects (GATEs) enable us to explore heterogeneity while maintaining a certain level of interpretability and are widely employed in applied research.<sup>1</sup> GATEs are particularly valuable when decision rules need to be applied or interpreted by humans, such as treatment

<sup>1</sup> For instance, Chernozhukov et al. (2017) document that, among 189 randomized control trials published in top economic journals since 2006, 40% report at least one subgroup analysis.

guidelines for physicians (see, e.g., Athey and Imbens, 2016). However, there could be many ways to form subgroups, and iteratively searching for subgroups with significantly estimated GATEs raises the possibility of  $p$ -hacking (see, e.g., Imbens, 2021). Moreover, while increasing the number of subgroups can help uncover more granular heterogeneity, groupings that are too fine may hinder interpretability.

This paper introduces a methodology for constructing heterogeneous subgroups that enables a flexible and coherent exploration of the trade-off between interpretability and the discovery of more granular heterogeneity while avoiding complications associated with  $p$ -hacking. The approach offers an alternative to pre-analysis plans, which may be criticized for limiting the potential for uncovering unexpected heterogeneity.

This paper distinguishes itself from existing literature by specifically targeting the discovery of heterogeneous subgroups. While several studies have focused on methodologies for estimating GATEs (see, e.g., Abrevaya et al., 2015; Lee et al., 2017; Lechner, 2018; Zimmert and Lechner, 2019; Fan et al., 2022; Lechner and Mareckova, 2022), these methodologies require researchers to predefine groups before analysis.<sup>2</sup> In contrast, this paper introduces a data-driven methodology for constructing heterogeneous subgroups and shows how it can be combined with honesty (Athey and Imbens, 2016) and debiased machine learning (Semenova and Chernozhukov, 2021) to conduct valid inference about the GATEs.

The proposed methodology, hereafter referred to as *aggregation trees*, builds on standard decision trees (Breiman et al., 1984) to aggregate units with similar estimated responses to the treatment. The resulting tree is then pruned to generate a sequence of groupings, one for each level of granularity. We show that each grouping features an optimality property in that it ensures that the loss in explained heterogeneity resulting from aggregation is minimized. Moreover, the sequence is nested in the sense that subgroups formed at a particular level of granularity are never disrupted at coarser levels. This property guarantees the consistency of the results across the different granularity levels, which is a fundamental requirement for any classification system (see, e.g., Cotterman and Peracchi, 1992).

For a particular grouping, point estimates and standard errors for the GATEs are obtained by fitting an appropriate linear model. Under honesty (Athey and Imbens, 2016), we can use the estimated standard errors to conduct valid inference about the GATEs as usual, e.g., by constructing conventional confidence intervals. Honesty is a subsample-splitting technique that requires that different observations are used to form subgroups and estimate the GATEs. In analogy to classical econometrics, this is equivalent to using different subsamples to select and estimate a model. This way, the asymptotic properties of GATE estimates are the same as if the groupings had been exogenously given.

Our methodology is similar to the causal trees of Athey and Imbens (2016), but it differs in its direct applicability to observational studies and its focus on the trade-off between interpretability and the discovery of more granular heterogeneity. We compare the performance of aggregation and causal trees using both theoretical arguments and an empirical Monte-Carlo study (Huber et al., 2013; Lechner and Wunsch, 2013).<sup>3</sup> Our simulation shows that aggregation trees lead to lower root mean squared error in the

<sup>2</sup> Bearth and Lechner (2024) discuss how to analyze and interpret differences in GATEs across groups while accounting for variations in other covariates.

<sup>3</sup> The literature presents a wide array of causal machine learning methodologies for estimating dense heterogeneous treatment effects (see, e.g., Wager and Athey, 2018; Athey et al., 2019; Künzel et al., 2019). However, aggregation trees focus on the construction of heterogeneous subgroups and the subsequent

estimated treatment effects, with reductions of up to 121%. This improvement entirely stems from the lower variance of aggregation trees, resulting from a splitting strategy that is robust to covariates affecting the outcome levels but not the treatment effects.

We also investigate the benefits of honesty compared to more standard “adaptive” estimation that uses the same data for constructing the tree and GATE estimation. Honesty greatly benefits inference, ensuring approximately nominal coverage of confidence intervals. In contrast, adaptive estimation can result in coverage rates as low as 58%.

The proposed methodology is applied to revisit the impact of maternal smoking on birth weight (see, e.g., Almond et al., 2005; Cattaneo, 2010). The analysis finds evidence of systematic heterogeneity, as different subgroups react differently to the same treatment. Moreover, the analysis reveals that effect heterogeneity is driven by parental and birth-related characteristics. The results are consistent with previous research showing that the effects are stronger for children born to adult mothers (Abrevaya et al., 2015; Zimmert and Lechner, 2019). Furthermore, we provide evidence that the effects are more pronounced when prenatal care visits are more frequent and occur earlier.

The rest of the paper unfolds as follows. Section 2 discusses the estimands of interest and their identification. Section 3 introduces aggregation trees and compares them to causal trees. Section 4 shows the simulation results. Section 5 illustrates the empirical exercise. Section 6 concludes.

## 2. CAUSAL FRAMEWORK

### 2.1. Estimands

We define the estimands of interest using the potential outcomes model (Neyman, 1923; Rubin, 1974). Suppose to have access to a sample of  $n$  i.i.d. observations  $(Y_i, D_i, X_i)$ , where  $Y_i \in \mathcal{Y}$  is the outcome targeted by the treatment,  $D_i \in \{0, 1\}$  is the binary treatment indicator, and  $X_i = (X_{i1}, \dots, X_{ip})^\top \in \mathcal{X}$  is a  $p \times 1$  vector of pre-treatment covariates. We posit the existence of two potential outcomes  $Y_i(0)$  and  $Y_i(1)$ , representing the outcome that the  $i$ -th unit would experience under each treatment level. The observed outcome for unit  $i$  is then the potential outcome corresponding to the treatment received:<sup>4</sup>

$$Y_i = D_i Y_i(1) + (1 - D_i) Y_i(0). \quad (2.1)$$

To define the effect of the treatment, we can take the differences in the potential outcomes of each unit  $\xi_i := Y_i(1) - Y_i(0)$  and aggregate them at different levels of granularity. The coarsest estimand of interest is the Average Treatment Effect (ATE),

$$\tau := E[\xi_i]. \quad (2.2)$$

The ATE quantifies the average impact of the policy on the reference population and is straightforward to interpret. However, it lacks information regarding the distributional impacts of the policy.

To tackle effect heterogeneity, we can focus instead on the Conditional Average Treat-

estimation of GATEs. Given these distinct objectives, we do not include these methodologies in our simulations.

<sup>4</sup> The definition of potential outcomes and the observational rule linking them to the observed outcomes implicitly assume the absence of spillover effects among units, which is violated in settings where some units are connected through networks.

ment Effects (CATEs),

$$\tau(X_i) := E[\xi_i | X_i]. \quad (2.3)$$

The CATEs provide information at the finest level of granularity achievable with the information at hand and enable us to relate effect heterogeneity to the observable covariates. However, they are difficult to interpret.

The Group Average Treatment Effects (GATEs) provide a way to explore heterogeneity while maintaining a certain level of interpretability. The GATEs are defined as

$$\tau_g := E[\xi_i | X_i \in \mathcal{X}_g], \quad g = 1, \dots, G, \quad (2.4)$$

where the groups  $\mathcal{X}_1, \dots, \mathcal{X}_G$  represent a partition of  $\mathcal{X}$ .<sup>5</sup> GATEs are particularly useful for simplifying interpretation and communication of effect heterogeneity. However, forming GATEs requires selecting a value for  $G$ , which controls the level of granularity. While higher values of  $G$  can uncover more detailed heterogeneity, partitions that are too fine may not offer substantial advantages in terms of interpretability compared to CATEs. To address this, we introduce a methodology that constructs partitions of  $\mathcal{X}$  at various levels of granularity, enabling a flexible exploration of the trade-off between interpretability and the discovery of more granular heterogeneity.

## 2.2. Identification

All the estimands discussed in the previous section are defined in terms of potential outcomes. However, each unit is either treated or not treated. We thus observe only one potential outcome per unit, and further assumptions are needed for identification.

The following standard assumptions are sufficient to identify the CATEs (see, e.g., Imbens and Rubin, 2015):

ASSUMPTION 2.1. (*Unconfoundedness*):  $\{Y_i(0), Y_i(1)\} \perp\!\!\!\perp D_i | X_i$ .

ASSUMPTION 2.2. (*Common support*):  $0 < \pi(X_i) < 1$ , where  $\pi(X_i) := \mathbb{P}(D_i = 1 | X_i)$  is the conditional treatment probability (or propensity score).

The unconfoundedness assumption requires that  $X_i$  contains all “confounders” jointly affecting the treatment assignment and the outcome.<sup>6</sup> The common support assumption states that each unit must have a non-zero probability of belonging to the treatment and control groups.

Under Assumptions 2.1–2.2, the CATEs are identified from observable data:

$$\begin{aligned} \mu(1, X_i) - \mu(0, X_i) &= E[Y_i(1) | D_i = 1, X_i] - E[Y_i(0) | D_i = 0, X_i] \quad (\text{by equation 2.1}) \\ &= E[Y_i(1) | X_i] - E[Y_i(0) | X_i] \quad (\text{by Assumption 2.1}) \\ &= \tau(X_i), \end{aligned} \quad (2.5)$$

<sup>5</sup> If grouping is based on the levels of a single discrete variable  $Z_i \subset X_i$ , each GATE simplifies to  $\tau_g = E[Y_i(1) - Y_i(0) | Z_i = g]$ .

<sup>6</sup>  $X_i$  can also include additional “heterogeneity covariates” not necessary for identification but for which effect heterogeneity is of interest. The sets of confounders and heterogeneity covariates can overlap in any way or be disjoint.

where  $\mu(D_i, X_i) := E[Y_i|D_i, X_i]$  and Assumption 2.2 ensures that the conditional expectations are well-defined for all values within the support of  $X_i$ . The ATE and GATEs are expressed as expectations of the CATEs and are thus identified under the same assumptions.

### 3. AGGREGATION TREES

This section outlines the implementation of the methodology proposed in this paper, which involves three steps. First, an estimation step constructs an estimate  $\hat{\tau}(\cdot)$  of  $\tau(\cdot)$ . Second, a tree-growing step approximates  $\hat{\tau}(X_i)$  by a standard decision tree (Breiman et al., 1984) that constrains the set of admissible groupings. Third, a tree-pruning step generates a sequence of nested subtrees, one for each level of granularity. Each subtree provides an optimal grouping, where optimality means that, at each granularity level, the groupings minimize the loss in explained heterogeneity due to aggregation.

The next subsection describes the tree-growing step, detailing the splitting strategy used by aggregation trees and comparing it with that of causal trees (Athey and Imbens, 2016). Then, the tree-pruning step is discussed. Finally, we explain how to conduct valid inference about the GATEs using double machine learning (Semenova and Chernozhukov, 2021).

#### 3.1. Tree-growing step

Trees are typically constructed by greedily minimizing an assumed loss function based on the mean squared error criterion. This minimization follows the approach proposed by Breiman et al. (1984), which suggest recursively stratifying the covariate space using axis-aligned splits.

Starting with a region of the covariate space  $\mathcal{R}_m \subseteq \mathcal{X}$ , consider a candidate splitting variable  $X_{ij}$  and splitting point  $s$ . We define the corresponding subregions as:<sup>7</sup>

$$\mathcal{R}_{m+1}(j, s) = \{X_i | X_{ij} \leq s\}, \quad \mathcal{R}_{m+2}(j, s) = \{X_i | X_{ij} > s\}. \quad (3.1)$$

The split occurs on some pair  $(j, s)$ , and the population is stratified accordingly. The process is then repeated in the resulting subregions, thus obtaining increasingly finer partitions of  $\mathcal{X}$ . The whole procedure can be described by the shape of a decision tree: the “root” (i.e., the node with no “parent”) corresponds to  $\mathcal{X}$ , the  $m$ -th internal node represents subregion  $\mathcal{R}_m$  and has two “children” nodes representing subregions  $\mathcal{R}_{m+1}$  and  $\mathcal{R}_{m+2}$ , and the “leaves” (i.e., the collection of terminal nodes) correspond to a partition of  $\mathcal{X}$ .

Ideally, we would like to explore the space of all possible trees and pick the one whose associated partition minimizes the assumed loss function. However, it is generally infeasible to enumerate all the distinct binary decision trees.<sup>8</sup> To cope with this issue, Breiman

<sup>7</sup> In the case of categorical splitting variables,  $s$  corresponds to a subset of possible levels of  $X_{ij}$ , and the inequality signs are replaced by  $\in$  and  $\notin$ .

<sup>8</sup> Consider the situation where  $X_i$  is composed of  $p$  binary covariates, and let  $\mathcal{D}$  be the “depth” of a given tree (i.e., the number of nodes connecting the root to the furthest leaf). Online Supplement S3 shows that  $L_{\mathcal{D}} = \prod_{d=1}^{\mathcal{D}} (p - (d - 1))^{2^{d-1}}$  is a lower bound for the number of distinct binary decision trees grown by recursively partitioning  $\mathcal{X}$  and having a depth equal to or lower than  $\mathcal{D}$ .  $L_{\mathcal{D}}$  quickly diverges as  $p$  grows. For example, fixing  $\mathcal{D} = 3$  and letting  $p = 10$  yields a lower bound of 3,317,760, while letting  $p = 20$  leads to a bound of 757,926,720. Things only worsen with categorical covariates taking more than two values or continuous covariates discretized using a large number of bins.

et al. (1984) suggest a “greedy” approach that partitions each region  $\mathcal{R}_m \subseteq \mathcal{X}$  by choosing the split that minimizes the assumed loss function within the resulting subregions  $\mathcal{R}_{m+1}$  and  $\mathcal{R}_{m+2}$ . This process is then iterated until some particular “stopping criterion” is met, for instance the maximum depth of the tree. This approach is greedy in that it ignores that a suboptimal split could yield better results at later steps and is generally considered to be a reasonable way of circumventing the exhaustive search of the space of all possible trees.

Let  $\mathcal{T}$  be some tree constructed using a training sample  $\mathcal{S}^{tr}$ , and let  $\mathcal{S}^{te}$  be an independent test sample. Then, when heterogeneous treatment effects are the object of the analysis, one wants to build a tree that minimizes  $EMSE(\mathcal{T}) = E[MSE(\mathcal{S}^{te}, \mathcal{S}^{tr}, \mathcal{T})]$ , where the expectation is taken over the joint distribution of the training and test samples and:<sup>9</sup>

$$\begin{aligned} MSE(\mathcal{S}^{te}, \mathcal{S}^{tr}, \mathcal{T}) &= \frac{1}{|\mathcal{S}^{te}|} \sum_{i \in \mathcal{S}^{te}} \left\{ [\tau_i - \tilde{\tau}(X_i, \mathcal{S}^{tr}, \mathcal{T})]^2 - \tau_i^2 \right\} \\ &= \frac{1}{|\mathcal{S}^{te}|} \sum_{i \in \mathcal{S}^{te}} \tilde{\tau}^2(X_i, \mathcal{S}^{tr}, \mathcal{T}) - \frac{2}{|\mathcal{S}^{te}|} \sum_{i \in \mathcal{S}^{te}} \tau_i \tilde{\tau}(X_i, \mathcal{S}^{tr}, \mathcal{T}), \end{aligned} \quad (3.2)$$

with  $\tau_i \equiv \tau(X_i)$  and  $\tilde{\tau}(x, \mathcal{S}, \mathcal{T})$  some estimate of  $\tau(\cdot)$  within the leaf  $\ell(x, \mathcal{T})$  of  $\mathcal{T}$  where  $x$  falls obtained using observations in the sample  $\mathcal{S}$ . In practice, trees are constructed by greedily minimizing an in-sample loss function  $MSE(\mathcal{S}^{tr}, \mathcal{S}^{tr}, \mathcal{T})$ .<sup>10</sup>

The key challenge in a causal inference framework is that we do not observe  $\tau_i$ . Thus,  $MSE(\cdot, \cdot, \cdot)$  is an infeasible criterion and needs to be estimated. We propose an estimator of  $MSE(\cdot, \cdot, \cdot)$  obtained by plugging an estimate  $\hat{\tau}(\cdot)$  of  $\tau(\cdot)$  constructed from the training sample in a preliminary estimation step:

$$\widehat{MSE}_{AT}(\mathcal{S}^{te}, \mathcal{S}^{tr}, \mathcal{T}) = \frac{1}{|\mathcal{S}^{te}|} \sum_{i \in \mathcal{S}^{te}} \tilde{\tau}_{AT}^2(X_i, \mathcal{S}^{tr}, \mathcal{T}) - \frac{2}{|\mathcal{S}^{te}|} \sum_{i \in \mathcal{S}^{te}} \hat{\tau}_i \tilde{\tau}_{AT}(X_i, \mathcal{S}^{tr}, \mathcal{T}), \quad (3.3)$$

with:

$$\tilde{\tau}_{AT}(x, \mathcal{S}, \mathcal{T}) = \frac{1}{|i \in \mathcal{S} : X_i \in \ell(x, \mathcal{T})|} \sum_{i \in \mathcal{S} : X_i \in \ell(x, \mathcal{T})} \hat{\tau}_i. \quad (3.4)$$

If we use a consistent estimator of  $\tau_i$ , then  $\widehat{MSE}_{AT}(\mathcal{S}^{te}, \mathcal{S}^{tr}, \mathcal{T})$  is an approximately unbiased estimator of  $MSE(\mathcal{S}^{te}, \mathcal{S}^{tr}, \mathcal{T})$  if the assignment to treatment is random conditional on  $X_i$ . In practice, we construct aggregation trees by greedily minimizing the in-sample loss function  $\widehat{MSE}_{AT}(\mathcal{S}^{tr}, \mathcal{S}^{tr}, \mathcal{T})$ . This is equivalent to selecting splits that minimize the conditional variance of  $\hat{\tau}_i$  in the resulting nodes. Consequently, the greedy approach partitions each region  $\mathcal{R}_m \subseteq \mathcal{X}$  in a way that maximizes systematic heterogeneity between the resulting subgroups, thereby constructing a set of admissible groupings represented by the resulting tree  $\mathcal{T}_0$ .

<sup>9</sup> We are departing from the standard criterion  $E[\{\tau_i - \tilde{\tau}(X_i, \mathcal{S}^{tr}, \mathcal{T})\}^2]$  by subtracting  $E[\tau_i^2]$ . Because this term does not depend on an estimator, the tree that minimizes the standard criterion also minimizes  $EMSE(\cdot)$ .

<sup>10</sup> This is what Athey and Imbens (2016) denote as the “adaptive” case, where the same sample is used to both construct and estimate the tree. Athey and Imbens (2016) also consider an alternative “honest” criterion  $MSE(\mathcal{S}^{te}, \mathcal{S}^{hon}, \mathcal{T})$  that uses different samples for construction of the tree ( $\mathcal{S}^{tr}$ ) and treatment effect estimation ( $\mathcal{S}^{hon}$ ). For simplicity, we focus on the adaptive case here and postpone the discussion of honesty to a later section.

Athey and Imbens (2016) propose instead the following estimator of  $MSE(\cdot, \cdot, \cdot)$ :

$$\widehat{MSE}_{CT}(\mathcal{S}^{te}, \mathcal{S}^{tr}, \mathcal{T}) = \frac{1}{|\mathcal{S}^{te}|} \sum_{i \in \mathcal{S}^{te}} \tilde{\tau}_{CT}^2(X_i, \mathcal{S}^{tr}, \mathcal{T}) - \frac{2}{|\mathcal{S}^{te}|} \sum_{i \in \mathcal{S}^{te}} \tilde{\tau}_{CT}(X_i, \mathcal{S}^{te}, \mathcal{T}) \tilde{\tau}_{CT}(X_i, \mathcal{S}^{tr}, \mathcal{T}), \quad (3.5)$$

with:

$$\tilde{\tau}_{CT}(x, \mathcal{S}, \mathcal{T}) = \hat{\mu}(1, x, \mathcal{S}, \mathcal{T}) - \hat{\mu}(0, x, \mathcal{S}, \mathcal{T}), \quad (3.6)$$

and

$$\hat{\mu}(d, x, \mathcal{S}, \mathcal{T}) = \frac{1}{|i \in \mathcal{S} : X_i \in \ell(x, \mathcal{T}), D_i = d|} \sum_{i \in \mathcal{S} : X_i \in \ell(x, \mathcal{T}), D_i = d} Y_i. \quad (3.7)$$

In randomized experiments,  $\widehat{MSE}_{CT}(\mathcal{S}^{te}, \mathcal{S}^{tr}, \mathcal{T})$  is an approximately unbiased estimator of  $MSE(\mathcal{S}^{te}, \mathcal{S}^{tr}, \mathcal{T})$ , as  $E[\tau_i | i \in \mathcal{S}^{te} : i \in \ell(x, \mathcal{T})] = E[\tilde{\tau}_{CT}(x, \mathcal{S}^{te}, \mathcal{T})]$ , with the expectations taken over the distribution of the test samples. As before, we construct causal trees by greedily minimizing the in-sample loss function  $\widehat{MSE}_{CT}(\mathcal{S}^{tr}, \mathcal{S}^{tr}, \mathcal{T})$ .

We expect the splitting strategy of aggregation trees to result in a lower sampling variance, especially when dealing with covariates that influence outcome levels but not treatment effects. For example, consider potential outcomes expressed as

$$Y_i(d) = \phi(X_i) + \frac{1}{2}(2d - 1)\tau(X_i) + \epsilon_i, \quad (3.8)$$

with  $\phi(X_i) = \frac{1}{2}X_{i1} + X_{i2}$  a model for the mean effect and  $\tau(X_i) = \frac{1}{2}X_{i1}$ . When exploring covariate values as potential splitting points, we shift one observation at a time from one region of the covariate space to its complement. Since each observation belongs to either the treatment or control group, this alters the sample average of the observed outcomes of only one group, thus affecting  $\tilde{\tau}_{CT}(\cdot, \cdot, \cdot)$ . Due to the substantial influence of  $X_{i2}$  on mean outcomes, moving a single observation between child nodes based on this covariate can substantially alter the sample average of the observed outcomes of one group. Consequently, we expect  $\tilde{\tau}_{CT}(\cdot, \cdot, \cdot)$  to exhibit considerable variability with the choice of splitting point, even though  $X_{i2}$  does not enter the model for  $\tau(\cdot)$ . This variability may also cause the estimator to identify spurious splits involving  $X_{i2}$ . In contrast, for accurately estimated CATEs,  $\tilde{\tau}_{AT}(\cdot, \cdot, \cdot)$  is expected to remain stable when shifting a single observation between child nodes based on  $X_{i2}$ . Consequently, we expect that  $\tilde{\tau}_{AT}(\cdot, \cdot, \cdot)$  will vary less with the choice of splitting point, resulting in a lower sampling variance.

### 3.2. Tree-pruning step

After a deep tree has been constructed, the standard practice is to prune it according to an assumed cost-complexity criterion. Aggregation and causal trees rely on the same criterion, which is composed of two terms:

$$C_\alpha(\mathcal{T}) = MSE(\mathcal{S}^{tr}, \mathcal{S}^{tr}, \mathcal{T}) + \alpha|\mathcal{T}|. \quad (3.9)$$

The first term corresponds to the loss function used for constructing the tree and measures the in-sample goodness-of-fit of the model. The second term is a regularization component that penalizes the model's complexity - defined as the number of leaves  $|\mathcal{T}|$  - according to the cost-complexity parameter  $\alpha \in [0, \infty)$ . Regularization is needed to prevent overfitting: the in-sample loss function  $MSE(\mathcal{S}^{tr}, \mathcal{S}^{tr}, \mathcal{T})$  always decreases with additional splits, even in the cases where the out-of-sample  $MSE(\mathcal{S}^{te}, \mathcal{S}^{tr}, \mathcal{T})$  actually increases.

The parameter  $\alpha$  controls the relative weight of the two components and thus the balance between the accuracy and the interpretability of the model. Define a subtree  $\mathcal{T} \subset \mathcal{T}_0$  as any tree that can be obtained by collapsing any number of internal nodes of  $\mathcal{T}_0$  and let  $\mathcal{T}_\alpha \subseteq \mathcal{T}_0$  be the smallest subtree for which (3.9) is minimized. For each  $\alpha$ , a unique  $\mathcal{T}_\alpha$  exists, which can be identified by “weakest link pruning”: starting from  $\mathcal{T}_0$ , we iteratively collapse the internal node that gives the slightest increase in the accuracy of the approximation.

Following this procedure, we can generate a sequence of nested subtrees  $\mathcal{T}_{\alpha_0}, \mathcal{T}_{\alpha_1}, \dots, \mathcal{T}_{\alpha_{\max}}$ , where  $0 = \alpha_0 < \alpha_1 < \dots < \alpha_{\max} < \infty$  are threshold values such that all  $\alpha$  in a given interval lead to the same subtree and  $\mathcal{T}_{\alpha_{\max}}$  corresponds to the tree’s root. Each subtree in this sequence is associated with a partition of the covariate space. Therefore, the tree-pruning step generates a sequence of groupings, one for each threshold value  $\alpha_0 < \alpha_1 < \dots < \alpha_{\max}$ .

Cross-validation procedures are commonly used to determine the optimal cost-complexity parameter and thus select a single partition (see, e.g., Hastie et al., 2009).<sup>11</sup> However, the whole sequence of groupings generated by the pruning process is also of significant interest. Because each tree  $\mathcal{T}_{\alpha_k}$  is obtained by collapsing the weakest node of  $\mathcal{T}_{\alpha_{k-1}}$ , the tree-pruning step constructs optimal groupings by aggregating the two subgroups for which the loss in explained heterogeneity resulting from aggregation is minimized. Moreover, because the sequence is nested, subgroups formed at a particular level of granularity are never disrupted at coarser levels. This property guarantees the consistency of the results across the different granularity levels (see, e.g., Cotterman and Peracchi, 1992). Consequently, the sequence allows for a flexible and coherent exploration of the trade-off between interpretability and the discovery of more granular heterogeneity.<sup>12</sup>

### 3.3. Estimation and inference

For a particular grouping  $\mathcal{T}_\alpha$ , we can estimate the GATEs in several ways. In randomized experiments, taking the difference between the mean outcomes of treated and control units in each group is an unbiased estimator of the GATEs. Equivalently, we can obtain the same point estimates in addition to their standard errors by estimating via OLS the following linear model:

$$Y_i = \sum_{l=1}^{|\mathcal{T}_\alpha|} L_{i,l} \gamma_l + \sum_{l=1}^{|\mathcal{T}_\alpha|} L_{i,l} D_i \beta_l + \epsilon_i \quad (3.10)$$

with  $L_{i,l}$  a dummy variable equal to one if the  $i$ -th unit falls in the  $l$ -th leaf of  $\mathcal{T}_\alpha$ . Exploiting the random assignment to treatment, we can show that each  $\beta_l$  identifies the GATE in the  $l$ -th leaf.

In observational studies, estimating model (3.10) would yield biased GATE estimates due to the selection into treatment. To get unbiased estimates, we can use the orthogonal estimator of Semenova and Chernozhukov (2021) to estimate the best linear predictor of  $\tau(\cdot)$  given a set of dummies denoting leaf membership. The key idea is to construct a random variable  $\psi_i$ , generally called score, such that  $\tau(X_i) = E[\psi_i | X_i]$ , and project it onto  $L_{i,1}, \dots, L_{i,|\mathcal{T}_\alpha|}$ .

<sup>11</sup> Athey and Imbens (2016) also utilize cross-validation, adapting the standard criterion for the honest case.

<sup>12</sup> A single partition can still be selected for practical purposes using standard cross-validation methods.



Consider the doubly-robust scores of Robins and Rotnitzky (1995):

$$\psi_i^{DR} = \mu(1, X_i) - \mu(0, X_i) + \frac{D_i [Y_i - \mu(1, X_i)]}{\pi(X_i)} - \frac{(1 - D_i) [Y_i - \mu(0, X_i)]}{1 - \pi(X_i)} \quad (3.11)$$

Because  $E[\psi_i^{DR}|X_i] = \tau(X_i)$ , this score is a natural candidate. We recognize that it depends on unknown functions  $\eta(X_i) := \{\mu(1, X_i), \mu(0, X_i), \pi(X_i)\}$  and make this explicit by writing  $\psi_i^{DR} := \psi_i^{DR}(\eta)$ . We refer to  $\eta$  as nuisance functions, as they are not of direct interest but necessary to construct a plug-in estimate  $\psi_i^{DR}(\hat{\eta})$  of  $\psi_i^{DR}(\eta)$  that we aim to regress on  $L_{i,1}, \dots, L_{i,|\mathcal{T}_\alpha|}$ .

Semenova and Chernozhukov (2021) show that  $\psi_i^{DR}(\eta)$  is a Neyman-orthogonal score (Chernozhukov et al., 2018), that is, its plug-in estimate  $\psi_i^{DR}(\hat{\eta})$  is insensitive to bias in the estimation of  $\hat{\eta}$ . They then suggest the following two-stage procedure. First, construct an estimate  $\hat{\eta}$  of the nuisance functions  $\eta$  using  $K$ -fold cross-fitting: split the sample into  $K$  folds of similar sizes and, for each  $k = 1, \dots, K$ , estimate  $\hat{\eta}_k$  using all but the  $k$ -th folds. Second, construct  $\hat{\psi}_i^{DR} := \psi_i^{DR}(\hat{\eta}_k)$ , where the observation  $i$  belongs to the  $k$ -th fold, and estimate via OLS the following linear model:

$$\hat{\psi}_i^{DR} = \sum_{l=1}^{|\mathcal{T}_\alpha|} L_{i,l} \beta_l + \epsilon_i \quad (3.12)$$

As before, each  $\beta_l$  identifies the GATE in the  $l$ -th leaf. Moreover, Semenova and Chernozhukov (2021) show that thanks to the Neyman-orthogonality of  $\psi_i^{DR}$ , the OLS estimator  $\hat{\beta}_l$  of  $\beta_l$  is root- $n$  consistent and asymptotically normal, provided that the product of the convergence rates of the estimators of the nuisance functions  $\mu(\cdot, \cdot)$  and  $\pi(\cdot)$  is faster than  $n^{1/2}$ . This allows using machine learning estimators such as random forests and LASSO to estimate the nuisance functions, as they are shown to achieve an  $n^{1/4}$  convergence rate and faster under particular conditions.

However, GATE estimates may show some bias if we use the same data to construct the tree and to estimate (3.10)–(3.12), leading to invalid inference. One way out is to grow “honest” trees (Athey and Imbens, 2016). Honesty is a subsample-splitting technique that requires that different observations are used to form the subgroups and estimate the GATEs. For this purpose, we split the observed sample into a training sample  $\mathcal{S}^{tr}$  and an honest sample  $\mathcal{S}^{hon}$  of arbitrary sizes. We use  $\mathcal{S}^{tr}$  to estimate  $\tau(\cdot)$  and construct the sequence of groupings and, for a particular grouping  $\mathcal{T}_\alpha$ , we use  $\mathcal{S}^{hon}$  to estimate (3.10)–(3.12). This way, the asymptotic properties of GATE estimates are the same as if the groupings had been exogenously given. Therefore, we can use the estimated standard errors to conduct valid inference as usual, e.g., by constructing conventional confidence intervals. However, honesty generally comes at the expense of a larger mean squared error, as fewer observations are used to estimate  $\hat{\tau}(\cdot)$ , construct the tree, and compute GATE estimates.

#### 4. EMPIRICAL MONTE-CARLO

We conduct an empirical Monte-Carlo study (Huber et al., 2013; Lechner and Wunsch, 2013) to investigate the performance of aggregation and causal trees in a context that closely approximates a real application.<sup>13</sup> Empirical Monte-Carlo studies aim to base the

<sup>13</sup> See also Lechner (2018), Knaus et al. (2021), and Lechner and Mareckova (2022) for more recent implementations of empirical Monte-Carlo studies.

data-generating processes (DGPs) on real data as much as possible, thereby evaluating the estimators in a realistically representative context. The idea is to use a sufficiently large data set as the population of interest, from which we can draw random samples for estimation.

In the following subsection, we describe the data set forming our population. Then, we present the results. Online Supplement S2 illustrates the DGPs, the implementation of estimators, and the performance measures we employ and presents additional results.

#### 4.1. Population

We base our simulations on a data set previously used to evaluate the impact of maternal smoking on birth weight (see, e.g., Almond et al., 2005; Cattaneo, 2010; Heiler and Knaus, 2021; Bodory et al., 2022). Since we will use the same data set in our empirical illustration in the next section, we postpone the discussion of previous findings to that section and focus here on the details of the data.

The clean data set consists of 435,124 observations measured in Pennsylvania between 1989 and 1991. The outcome of interest is the infant’s weight at birth in grams. The treatment indicator equals one if the mother smoked during pregnancy and zero otherwise. The pre-treatment covariate vector contains 39 confounders and heterogeneity variables, providing information on the mother’s and father’s background characteristics (age, ethnicity, whether the mother was married or foreign-born), mother’s behavior possibly associated with smoking (whether she drank alcohol during pregnancy, how many drinks per week), maternal medical risk factors not affected by smoking during pregnancy, and birth characteristics (e.g., whether the infant is first born, number and quality of prenatal care visits).<sup>14</sup>

To avoid common support issues, we drop children whose parents were particularly young or old at birth, or who attended more than thirty prenatal care visits. Moreover, we drop children whose mothers used to consume more than ten alcoholic drinks per week during pregnancy. A total of 596 observations are removed from the original data set.

Table S.2 in Online Supplement S1 presents the summary statistics for the treated and control groups in the final sample. The table displays sample averages and standard deviations for each covariate, along with two measures of difference in the distribution across treatment arms: the normalized difference, measuring the difference between the locations of the distributions, and the logarithm of the ratio of standard deviations, measuring the difference in the dispersion of the distributions. Overall, the sample appears to be sufficiently balanced, with only five relatively unbalanced covariates: `meduc`, `unmarried` and `feduc` exhibit strong differences in locations, while `alcohol` and `n_drink` exhibit strong differences in dispersion. These results are robust to the inclusion of the excluded observations.

#### 4.2. Results

Table 1 presents the results obtained from  $R = 1,000$  replications across three sample sizes and two levels of heterogeneity. Overall, aggregation trees outperform causal trees in terms of prediction accuracy, with both  $AT_{XL}$  and  $AT_{CF}$  showing lower RMSE than causal

<sup>14</sup> Table S.1 in Online Supplement S1 provides a description of all the variables.

trees. This effect is particularly strong when treatment is randomly assigned, where the RMSE of causal trees is between 28% and 121% larger than that of aggregation trees. When units self-select into treatment, the RMSE of causal trees is still larger by 17% to 66%, except for the smallest sample size, where causal trees marginally outperform  $AT_{CF}$ .  $AT_{XL}$  and  $AT_{CF}$  consistently show similar performances.

The second and third panels of Table 1 offer additional insights into the superior prediction performance of aggregation trees by examining the absolute bias and standard deviation of the estimators. As expected, the advantage of aggregation trees is entirely driven by their lower sampling variance, resulting from the more stable splitting strategy employed. All estimators exhibit some bias, which increases as heterogeneity strengthens. When treatment is randomly assigned, the bias across all estimators is approximately the same. When treatment is instead assigned based on observable characteristics, causal trees exhibit a slightly lower bias than both  $AT_{XL}$  and  $AT_{CF}$ . However, aggregation trees show substantially lower sampling variance than causal trees, with approximately the same magnitudes and exceptions noted for RMSE. For all estimators, sampling variance remains consistent across different heterogeneity levels.

The fourth panel of Table 1 presents the coverage rates for 95% confidence intervals. Both  $AT_{XL}$  and  $AT_{CF}$  achieve coverage rates close to the nominal level, while causal trees consistently show lower coverage. The fifth panel of Table 1 reveals that causal trees generate more complex models with a larger number of leaves compared to aggregation trees. The complexity of causal trees increases significantly with larger sample sizes, while that of  $AT_{XL}$  and  $AT_{CF}$  grows more conservatively. This could lead to overfitting and raise concerns about multiple hypothesis testing in causal trees, potentially explaining their lower coverage rates. For all estimators, model complexity does not vary across

**Table 1:** Simulation results for honest trees.

	Randomized experiment						Observational study					
	Low heterogeneity			High heterogeneity			Low heterogeneity			High heterogeneity		
	500	1,000	2,000	500	1,000	2,000	500	1,000	2,000	500	1,000	2,000
<b>Panel 1: <math>RMSE</math></b>												
$AT_{XL}$	238.54	181.90	136.67	239.59	188.98	144.79	303.39	228.18	173.94	307.69	237.66	186.39
$AT_{CF}$	224.59	175.43	132.89	231.20	183.63	140.72	284.92	208.18	160.66	290.06	220.61	172.21
$CT$	306.66	304.49	294.14	318.38	307.98	302.31	280.94	273.06	267.65	283.52	280.03	270.30
<b>Panel 2: <math> Bias </math></b>												
$AT_{XL}$	17.07	16.55	16.26	40.20	40.00	39.89	22.17	23.46	21.68	44.85	46.99	43.22
$AT_{CF}$	16.82	16.53	16.24	40.19	40.07	39.85	23.10	23.11	21.43	45.55	47.80	42.98
$CT$	17.59	17.63	17.48	40.76	40.56	40.47	18.81	18.21	18.45	40.45	41.49	40.49
<b>Panel 3: <math>SD</math></b>												
$AT_{XL}$	237.56	180.68	135.07	234.21	182.17	135.89	302.08	226.25	171.78	302.38	230.22	178.25
$AT_{CF}$	223.59	174.19	131.30	225.71	176.74	131.85	283.42	206.15	158.38	284.34	212.40	163.56
$CT$	305.86	303.67	293.31	314.28	303.74	298.01	279.94	272.10	266.63	278.99	275.22	265.52
<b>Panel 4: Coverage of 95% CI</b>												
$AT_{XL}$	0.92	0.94	0.94	0.93	0.93	0.94	0.93	0.94	0.93	0.92	0.92	0.92
$AT_{CF}$	0.93	0.94	0.94	0.94	0.94	0.94	0.94	0.94	0.94	0.93	0.92	0.92
$CT$	0.89	0.89	0.89	0.89	0.89	0.89	0.85	0.85	0.85	0.84	0.85	0.85
<b>Panel 5: <math> \mathcal{T} </math></b>												
$AT_{XL}$	9.58	11.75	13.36	9.46	11.93	13.43	8.38	10.39	11.96	8.32	10.37	12.07
$AT_{CF}$	8.83	11.26	12.84	9.04	11.37	12.96	7.40	8.96	10.80	7.51	8.86	10.68
$CT$	15.29	29.81	56.95	15.51	29.96	57.77	6.89	13.29	25.33	6.78	13.37	24.69

**Note:** The first three panels report the average over the validation sample of the root mean squared error ( $RMSE$ ), absolute bias ( $|Bias|$ ), and standard deviation ( $SD$ ) of each estimator. The fourth panel reports coverage rates for 95% confidence intervals. The last panel reports the average number of leaves.

heterogeneity levels and is larger when treatment is randomly assigned, particularly for causal trees.

Comparing these results with Table S.4 in Online Supplement S2 allows us to assess the benefits of honesty. Using different data for constructing the trees and treatment effect estimation greatly benefits inference. The coverage rates of adaptive trees are considerably below the nominal rate, particularly those of causal trees that can be as low as 58%.

While honesty is expected to come at the expense of a larger mean squared error, our simulations show that the RMSE is actually higher for adaptive trees than for honest trees. For aggregation trees, this discrepancy may be due to the slightly more complex models generated by adaptive estimation, resulting in higher sampling variance while maintaining the same bias. In the case of causal trees, the discrepancy may also be due to the greater bias induced by adaptive estimation when the assignment is based on observable characteristics. An exception arises for causal trees under randomized treatment assignment, where adaptivity produces extremely shallow trees, which results in lower RMSE compared to honest estimation.

## 5. EMPIRICAL ILLUSTRATION

In this section, we apply the methodology developed in this paper to revisit the impact of maternal smoking on birth weight using the same data set as in the previous section.<sup>15</sup> First, we review previous findings from the literature. Then, we construct the sequence of optimal groupings. Finally, we discuss the results.

### 5.1. Previous findings

As documented in Almond et al. (2005), infants born at low birth weight (LBW) can impose substantial costs on society, with estimated expected costs of delivery and initial care exceeding 100,000\$ (at prices of year 2000) for babies weighing 1,000 grams at birth.<sup>16</sup> Moreover, LBW is associated with a higher risk of death within one year of birth. For these reasons, birth weight is considered the primary measure of a baby's health and is often the direct target of health policies. Thus, understanding what causes LBW is crucial.

The impact of maternal smoking on LBW has received considerable attention in the literature, for it is regarded as one of the most significant and modifiable risk factors. Several studies consistently find that smoking during pregnancy causes lower average birth weight, with estimated ATEs ranging between -600 and -100 grams (see, e.g., Almond et al., 2005; Abrevaya, 2006). As for effect heterogeneity, it is now well understood that the effects are increasingly negative with the mother's age (Abrevaya et al., 2015; Lee et al., 2017; Zimmert and Lechner, 2019; Fan et al., 2022). Finally, treatment heterogeneity has also been investigated. Cattaneo (2010) and Bodory et al. (2022) consider different smoking intensities as different treatments and show that higher smoking intensities lead to more negative effects. Heiler and Knaus (2021) find that heterogeneous effects can be partly explained by different smoking behaviors of ethnic and age groups.

<sup>15</sup> We provide the *aggTrees* R package to facilitate the implementation of the methodology developed in this paper. The package is freely available on CRAN at <https://CRAN.R-project.org/package=aggTrees>. The associated vignette is at <https://riccardo-df.github.io/aggTrees/>.

<sup>16</sup> An infant is considered born at LBW if she weighs less than 2,500 grams at birth.

### 5.2. Constructing the sequence of groupings

We split the sample into a training sample and an honest sample of equal sizes. We estimate the CATEs by fitting an honest causal forest (Athey et al., 2019).<sup>17</sup> We then construct the set of admissible groupings by approximating the estimated CATEs with a decision tree. We perform these operations utilizing only observations from the training sample.

Once the tree is constructed, we use observations from the honest sample to estimate the GATEs by constructing and averaging the doubly-robust scores in (3.11). Honest regression forests and 5-fold cross-fitting are employed to estimate the necessary nuisance functions.

Figure 1 displays the resulting tree. At the root, we see the estimated ATE of  $-211$  grams. The observations are initially split into two groups: non-first-born children (root's left child) and first-born children (root's right child). This division, among all possible two-group splits, maximizes heterogeneity in treatment effects. The first group, which includes 58% of the units, has an estimated GATE of approximately  $-236$  grams, while the second group, representing 42% of the units, has an estimated GATE of about  $-176$  grams.

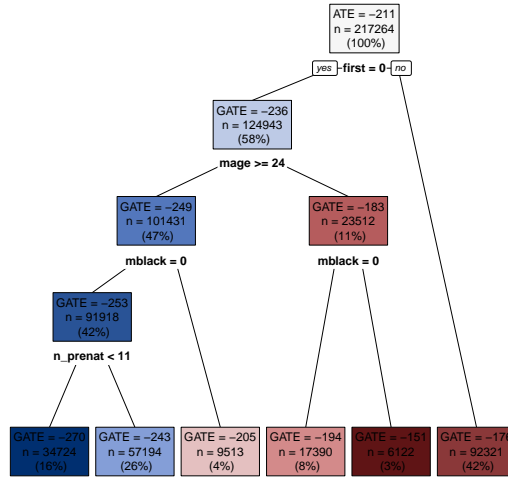


Figure 1: Aggregation tree.

The group of non-first-born children is further divided based on whether the child's mother is young or older, with additional splits into smaller subgroups depending on whether the mother is black. The process is repeated until a stopping criterion is met.

The sequence of optimal groupings is then constructed by progressively aggregating the two subgroups that yield the smallest loss in explained heterogeneity. Figure 2 visually illustrates this procedure. Reading the figure from left to right and top to bottom, each panel corresponds to a grouping in the sequence, derived by collapsing the node that minimizes the loss in explained heterogeneity. The figure clearly demonstrates the

<sup>17</sup> Figure S.1 in Online Supplement ?? displays the estimated CATEs sorted by magnitude, along with their corresponding 95% confidence intervals. Almost all predicted effects are negative and statistically different from zero at the 5% significance level.

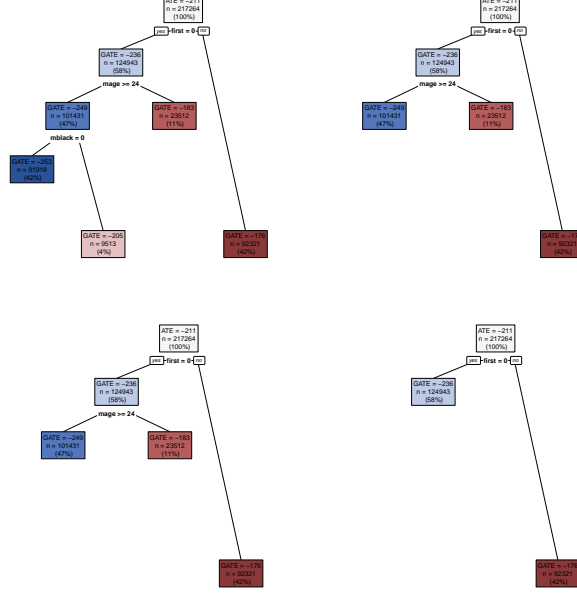


Figure 2: Sequence of optimal groupings.

consistency of the results across the different granularity levels, highlighting how the generated sequence enables a coherent exploration of the trade-off between interpretability and the discovery of more granular heterogeneity.

### 5.3. Discussion

We investigate whether systematic effect heterogeneity is present by examining whether distinct subgroups exhibit different reactions to the treatment.<sup>18</sup> For this purpose, we select the optimal grouping composed of five groups and estimate model (3.12) using only observations from the honest sample. Table 2 reports point estimates and 95% confidence intervals. The estimated GATEs exhibit substantial differences, ranging from  $-252$  grams for the most affected group (*Leaf 1*) to  $-151$  grams for the least affected group (*Leaf 5*). All estimates are negative and statistically different from zero at the 5% significance level.

Table 2 also reports the GATE differences across all pairs of groups, along with  $p$ -values testing the null hypothesis that each difference equals zero. To account for multiple hypothesis testing, we adjust the  $p$ -values using the procedure of Holm (1979). Many groups exhibit significant differences. For example, the GATE differences between *Leaf 1* and all other groups are both large and statistically significant (at the 10% confidence level for *Leaf 2* and 5% for the other groups). Additionally, the  $p$ -values for the differences between *Leaf 5* and *Leaf 2* or *Leaf 3* are just above the 10% significance level. For

<sup>18</sup> Looking at the distribution of the estimated CATEs is not an effective strategy for this task, as high variation in predictions due to estimation noise does not necessarily imply heterogeneous effects.

**Table 2:** GATE estimation and inference.

	<i>Leaf 1</i>	<i>Leaf 2</i>	<i>Leaf 3</i>	<i>Leaf 4</i>	<i>Leaf 5</i>
	-252.941	-205.349	-193.974	-176.181	-151.200
GATEs	[-265.761, -240.121]	[-237.959, -172.739]	[-212.363, -175.585]	[-189.003, -163.359]	[-182.001, -120.399]
<i>Leaf 1</i>	-	-	-	-	-
	(-)	(-)	(-)	(-)	(-)
<i>Leaf 2</i>	47.59	-	-	-	-
	(0.054)	(-)	(-)	(-)	(-)
<i>Leaf 3</i>	58.97	11.38	-	-	-
	(0.000)	(0.551)	(-)	(-)	(-)
<i>Leaf 4</i>	76.76	29.17	17.79	-	-
	(0.000)	(0.411)	(0.411)	(-)	(-)
<i>Leaf 5</i>	101.74	54.15	42.77	24.98	-
	(0.000)	(0.108)	(0.108)	(0.411)	(-)

**Note:** The first part of the table reports point estimates and 95% confidence intervals for the GATEs. The second part of the table displays the GATE differences across all pairs of leaves.  $p$ -values testing the null hypothesis that a single difference is zero are adjusted using Holm's procedure and reported in parenthesis under each point estimate.

**Table 3:** Heterogeneity across observable covariates.

	<i>Leaf 1</i>		<i>Leaf 2</i>		<i>Leaf 3</i>		<i>Leaf 4</i>		<i>Leaf 5</i>	
	Mean	(S.E.)	Mean	(S.E.)	Mean	(S.E.)	Mean	(S.E.)	Mean	(S.E.)
<b>Parental characteristics</b>										
mage	30.296	(0.013)	29.275	(0.043)	21.185	(0.013)	25.009	(0.018)	20.408	(0.025)
meduc	13.171	(0.008)	12.442	(0.024)	11.229	(0.013)	12.972	(0.008)	11.188	(0.027)
fage	32.334	(0.017)	31.914	(0.069)	24.780	(0.033)	27.512	(0.021)	23.546	(0.058)
feduc	13.294	(0.009)	11.884	(0.037)	11.338	(0.018)	12.886	(0.009)	11.003	(0.043)
<b>Birth characteristics</b>										
n_prenatal	11.257	(0.010)	8.844	(0.046)	9.997	(0.029)	11.314	(0.011)	6.916	(0.057)
prenatal0	0.004	(0.000)	0.062	(0.002)	0.015	(0.001)	0.006	(0.000)	0.098	(0.004)
prenatal1	0.870	(0.001)	0.614	(0.005)	0.677	(0.004)	0.836	(0.001)	0.405	(0.006)
prenatal2	0.103	(0.001)	0.243	(0.004)	0.243	(0.003)	0.131	(0.001)	0.337	(0.006)
prenatal3	0.019	(0.000)	0.071	(0.003)	0.060	(0.002)	0.023	(0.000)	0.147	(0.005)

**Note:** Average characteristics are obtained by regressing each covariate on a set of dummies denoting leaf membership using only the honest sample. Standard errors are estimated via the Eicker-Huber-White estimator.

other pairs, GATE differences are small, and we fail to reject the null hypothesis at any conventional confidence level. Overall, these findings provide evidence of systematic heterogeneity in treatment effects.

To understand the factors driving this heterogeneity, we examine how treatment effects relate to observable covariates by analyzing how the average characteristics of units vary across subgroups (see, e.g., Chernozhukov et al., 2017).<sup>19</sup> Table 3 reports the average values of selected covariates across *Leaves 1–5* (see Table S.3 in Online Supplement S1 for the remaining covariates). The least affected group comprises children born to younger parents, suggesting more negative effects at higher parental ages. This finding is consistent with previous research (see, e.g., Abrevaya et al., 2015; Zimmert and Lechner, 2019). In contrast, the most affected group has higher parental educational attainment,

<sup>19</sup> Another approach is to assess which variables the tree-growing process used to construct groups and measure their relative importance. However, we should not conclude that covariates not used for splitting are unrelated to heterogeneity, because if two covariates are highly correlated, trees generally split on only one of them.

likely due to older parents having had more time to study. This group also includes parents who attended more prenatal visits and were more likely to have their first visit in the first trimester of pregnancy. This may reflect mothers with problematic pregnancies self-selecting into more frequent and earlier prenatal visits.

## 6. CONCLUSION

This paper introduces a methodology for constructing heterogeneous subgroups that enables a flexible and coherent exploration of the trade-off between interpretability and the discovery of more granular heterogeneity. The proposed methodology constructs a sequence of groupings, one for each level of granularity. We show that each grouping features an optimality property and that the sequence is nested. We also show how the proposed methodology can be combined with honesty (Athey and Imbens, 2016) and debiased machine learning (Semenova and Chernozhukov, 2021) to conduct valid inference about the GATEs.

We compare the performance of aggregation and causal trees (Athey and Imbens, 2016) using both theoretical arguments and an empirical Monte-Carlo study (Lechner and Wunsch, 2013; Huber et al., 2013). Our simulation shows that aggregation trees substantially improve the root mean squared error of treatment effects due to lower variance resulting from a more robust splitting strategy.

We apply the proposed methodology to revisit the impact of maternal smoking on birth weight (see, e.g., Almond et al., 2005; Cattaneo, 2010). The analysis finds evidence of systematic heterogeneity driven by parental and birth-related characteristics.

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## ONLINE SUPPLEMENT

## S1. DATA

Table S.1: Description of variables in the data set.

Label	Description
<b>OUTCOME.</b>	
bweight	Infant birth weight (in grams)
<b>TREATMENT.</b>	
smoke	=1 if mother smoked during pregnancy
<b>COVARIATES.</b>	
<b>Mother's characteristics.</b>	
mage	Mother's age
meduc	Mother's educational attainment
mwhite	=1 if mother is white
mblack	=1 if mother is black
mhispan	=1 if mother is hispanic
foreign_born	=1 if mother is foreign born
unmarried	=1 if mother is unmarried
alcohol	=1 if mother drank alcohol during pregnancy
n_drink	Number of drinks per week during pregnancy
<b>Father's characteristics.</b>	
fage	Father's age
feduc	Father's educational attainment
fwhite	=1 if father is white
fblack	=1 if father is black
fhispan	=1 if father is hispanic
<b>Birth characteristics.</b>	
birthmonth1	=1 if birth in January
birthmonth2	=1 if birth in February
birthmonth3	=1 if birth in March
birthmonth4	=1 if birth in April
birthmonth5	=1 if birth in May
birthmonth6	=1 if birth in June
birthmonth7	=1 if birth in July
birthmonth8	=1 if birth in August
birthmonth9	=1 if birth in September
birthmonth10	=1 if birth in October
birthmonth11	=1 if birth in November
birthmonth12	=1 if birth in December
first	=1 if the infant is first born
plural	=1 if twins or greater birth
n_prenatal	Number of prenatal care visits
prenatal0	=1 if no prenatal visit
prenatal1	=1 if first prenatal visit in first trimester of pregnancy
prenatal2	=1 if first prenatal visit in second trimester of pregnancy
prenatal3	=1 if first prenatal visit in third trimester of pregnancy
adequacy1	=1 if adequacy of care is adequate (Kessner Index)
adequacy2	=1 if adequacy of care is intermediate (Kessner Index)
adequacy3	=1 if adequacy of care is inadequate (Kessner Index)
<b>Maternal medical risk factors.</b>	
diabete	=1 if mother is diabetic
anemia	=1 if mother is anemic
hyper	=1 if mother had pregnancy-associated hypertension

**Table S.2:** Balance between treatment and control groups.

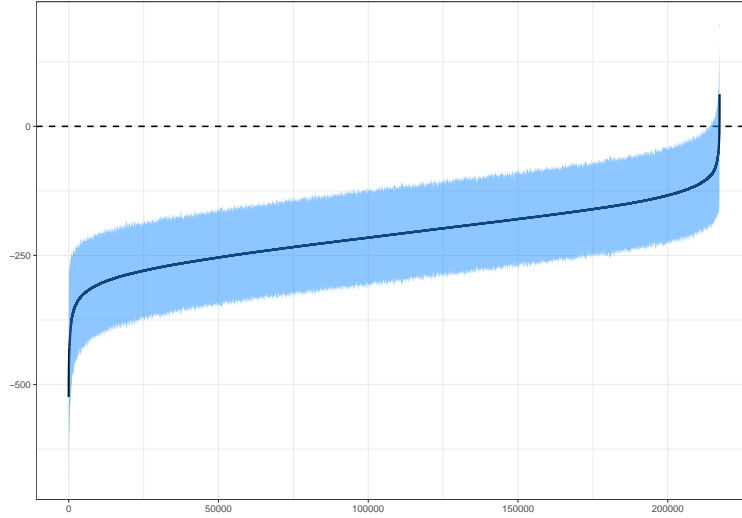
	Treated		Controls		Overlap measures	
	$(n_t = 81,388)$		$(n_c = 353,140)$		$\hat{\Delta}_j$	$\hat{\Gamma}_j$
	Mean	(S.D.)	Mean	(S.D.)		
mage	25.503	(5.372)	27.340	(5.553)	-0.336	-0.033
meduc	11.783	(1.883)	13.088	(2.430)	-0.600	-0.255
mwhite	0.850	(0.357)	0.865	(0.342)	-0.043	0.044
mblack	0.147	(0.354)	0.116	(0.321)	0.090	0.099
mhispan	0.020	(0.140)	0.031	(0.173)	-0.068	-0.209
foreign_born	0.019	(0.138)	0.056	(0.229)	-0.190	-0.504
unmarried	0.444	(0.497)	0.196	(0.397)	0.552	0.225
alcohol	0.045	(0.207)	0.007	(0.080)	0.243	0.943
n_drink	0.123	(0.729)	0.013	(0.212)	0.205	1.237
fage	28.451	(6.556)	29.640	(6.264)	-0.185	0.046
feduc	11.686	(2.628)	13.102	(2.800)	-0.522	-0.064
fwhite	0.831	(0.375)	0.857	(0.350)	-0.072	0.068
fblack	0.162	(0.369)	0.123	(0.329)	0.112	0.115
fhispan	0.028	(0.166)	0.033	(0.178)	-0.025	-0.068
birthmonth1	0.081	(0.273)	0.078	(0.268)	0.011	0.017
birthmonth2	0.074	(0.262)	0.075	(0.264)	-0.003	-0.004
birthmonth3	0.082	(0.274)	0.086	(0.280)	-0.015	-0.023
birthmonth4	0.076	(0.265)	0.083	(0.277)	-0.027	-0.043
birthmonth5	0.081	(0.273)	0.087	(0.282)	-0.022	-0.032
birthmonth6	0.083	(0.277)	0.086	(0.280)	-0.009	-0.013
birthmonth7	0.092	(0.289)	0.089	(0.284)	0.011	0.016
birthmonth8	0.094	(0.291)	0.089	(0.285)	0.017	0.024
birthmonth9	0.090	(0.286)	0.087	(0.282)	0.011	0.016
birthmonth10	0.086	(0.281)	0.084	(0.277)	0.009	0.013
birthmonth11	0.078	(0.269)	0.077	(0.267)	0.003	0.005
birthmonth12	0.082	(0.275)	0.079	(0.270)	0.012	0.019
first	0.367	(0.482)	0.438	(0.496)	-0.146	-0.029
plural	0.015	(0.120)	0.016	(0.127)	-0.015	-0.060
n_prenatal	10.210	(3.989)	11.125	(3.395)	-0.247	0.161
prenatal0	0.025	(0.156)	0.007	(0.086)	0.141	0.603
prenatal1	0.718	(0.450)	0.838	(0.368)	-0.292	0.200
prenatal2	0.204	(0.403)	0.124	(0.330)	0.216	0.200
prenatal3	0.047	(0.212)	0.026	(0.159)	0.114	0.289
adequacy1	0.631	(0.483)	0.762	(0.426)	-0.287	0.125
adequacy2	0.258	(0.437)	0.184	(0.388)	0.178	0.121
adequacy3	0.105	(0.306)	0.049	(0.216)	0.210	0.347
diabete	0.018	(0.132)	0.018	(0.135)	-0.006	-0.022
anemia	0.014	(0.119)	0.008	(0.092)	0.057	0.266
hyper	0.019	(0.138)	0.029	(0.168)	-0.065	-0.202

**Note:** Sample averages and standard deviations for each covariate are displayed, along with two measures of difference in the distribution across treatment arms: the normalized differences  $\hat{\Delta}_j$ , measuring the difference between the locations of the distributions, and the logarithm of the ratio of standard deviations  $\hat{\Gamma}_j$ , measuring the difference in the dispersion of the distributions.

**Table S.3:** Heterogeneity across observable covariates.

	<i>Leaf 1</i>		<i>Leaf 2</i>		<i>Leaf 3</i>		<i>Leaf 4</i>		<i>Leaf 5</i>	
	Mean	(S.E.)	Mean	(S.E.)	Mean	(S.E.)	Mean	(S.E.)	Mean	(S.E.)
mwhite	0.983	(0.000)	0.000	(-)	0.988	(0.001)	0.863	(0.001)	0.000	(-)
mblack	0.000	(-)	1.000	(-)	0.000	(-)	0.118	(0.001)	1.000	(-)
mhispan	0.022	(0.000)	0.010	(0.001)	0.093	(0.002)	0.026	(0.001)	0.013	(0.001)
foreign_born	0.051	(0.001)	0.050	(0.002)	0.059	(0.002)	0.048	(0.001)	0.014	(0.002)
unmarried	0.086	(0.001)	0.599	(0.005)	0.356	(0.004)	0.299	(0.002)	0.867	(0.004)
alcohol	0.013	(0.000)	0.049	(0.002)	0.011	(0.001)	0.010	(0.000)	0.034	(0.002)
n_drink	0.029	(0.001)	0.168	(0.010)	0.026	(0.002)	0.022	(0.001)	0.106	(0.009)
fwhite	0.972	(0.001)	0.028	(0.002)	0.948	(0.002)	0.853	(0.001)	0.029	(0.002)
fblack	0.010	(0.000)	0.962	(0.002)	0.037	(0.001)	0.128	(0.001)	0.963	(0.002)
fhispan	0.024	(0.001)	0.020	(0.001)	0.097	(0.002)	0.029	(0.001)	0.028	(0.002)
birthmonth1	0.078	(0.001)	0.081	(0.003)	0.079	(0.002)	0.078	(0.001)	0.085	(0.004)
birthmonth2	0.075	(0.001)	0.074	(0.003)	0.074	(0.002)	0.075	(0.001)	0.080	(0.003)
birthmonth3	0.086	(0.001)	0.082	(0.003)	0.081	(0.002)	0.085	(0.001)	0.084	(0.004)
birthmonth4	0.083	(0.001)	0.079	(0.003)	0.078	(0.002)	0.083	(0.001)	0.076	(0.003)
birthmonth5	0.090	(0.001)	0.077	(0.003)	0.084	(0.002)	0.084	(0.001)	0.081	(0.003)
birthmonth6	0.087	(0.001)	0.082	(0.003)	0.088	(0.002)	0.085	(0.001)	0.090	(0.004)
birthmonth7	0.087	(0.001)	0.096	(0.003)	0.095	(0.002)	0.088	(0.001)	0.091	(0.004)
birthmonth8	0.089	(0.001)	0.091	(0.003)	0.094	(0.002)	0.089	(0.001)	0.088	(0.004)
birthmonth9	0.086	(0.001)	0.087	(0.003)	0.086	(0.002)	0.089	(0.001)	0.079	(0.003)
birthmonth10	0.085	(0.001)	0.084	(0.003)	0.082	(0.002)	0.085	(0.001)	0.078	(0.003)
birthmonth11	0.077	(0.001)	0.083	(0.003)	0.079	(0.002)	0.079	(0.001)	0.082	(0.004)
birthmonth12	0.078	(0.001)	0.085	(0.003)	0.080	(0.002)	0.081	(0.001)	0.086	(0.004)
first	0.000	(-)	0.000	(-)	0.000	(-)	1.000	(-)	0.000	(-)
plural	0.016	(0.000)	0.021	(0.001)	0.012	(0.001)	0.016	(0.000)	0.017	(0.002)
adequacy1	0.794	(0.001)	0.500	(0.005)	0.578	(0.004)	0.766	(0.001)	0.280	(0.006)
adequacy2	0.165	(0.001)	0.295	(0.005)	0.306	(0.003)	0.189	(0.001)	0.356	(0.006)
adequacy3	0.037	(0.001)	0.194	(0.004)	0.110	(0.002)	0.040	(0.001)	0.349	(0.006)
diabete	0.021	(0.000)	0.026	(0.002)	0.009	(0.001)	0.017	(0.000)	0.007	(0.001)
anemia	0.007	(0.000)	0.019	(0.001)	0.017	(0.001)	0.008	(0.000)	0.036	(0.002)
hyper	0.016	(0.000)	0.024	(0.002)	0.011	(0.001)	0.043	(0.001)	0.013	(0.001)

**Note:** Average characteristics are obtained by regressing each covariate on a set of dummies denoting leaf membership using only the honest sample. Standard errors are estimated via the Eicker-Huber-White estimator. Leaves are sorted in increasing order of the GATEs.



**Figure S.1:** Sorted CATEs and 95% confidence intervals. Predictions on the honest sample are shown. Standard errors are smoothed by a Nadaraya-Watson regression.

## S2. EMPIRICAL MONTE-CARLO

We conduct an empirical Monte-Carlo study (Huber et al., 2013; Lechner and Wunsch, 2013) to investigate the performance of aggregation and causal trees (Athey and Imbens, 2016) in a context that closely approximates a real application. We base our simulations on a data set previously used to evaluate the impact of maternal smoking on birth weight (see, e.g., Almond et al., 2005; Cattaneo, 2010; Heiler and Knaus, 2021; Bodory et al., 2022).

Section 4.1 of the paper describes the data set forming our population. In the next subsection, we illustrate the DGPs, the implementation of estimators, and the performance measures we employ. Then, we present additional results not shown in the paper.

### S2.1. Simulation details

We estimate the propensity score via a logistic regression using the full sample and all baseline covariates. The resulting estimate  $\hat{\pi}(\cdot)$  is later used as the true selection model in the simulation to ensure that the selection behavior closely mimics that observed in the real data. We then remove all treated units from the sample. This implies that we observe  $Y_i(0)$  for all units.

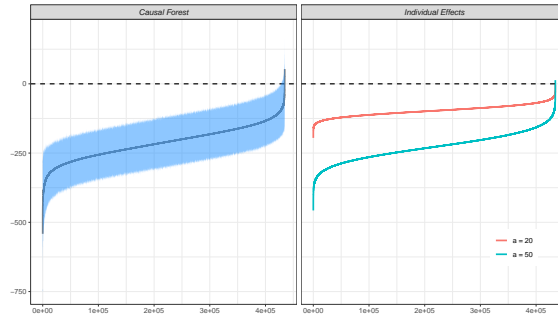
We now need to specify a model for the individual effects. Since model specification can be somewhat arbitrary, we aim to reduce this arbitrariness and better approximate a realistic scenario by fitting an honest causal forest (Athey et al., 2019) using the full sample and choosing a model that mimics the resulting predictions.<sup>20</sup> Figure S.2 presents the estimated CATEs sorted by magnitude, along with their 95% confidence intervals.

<sup>20</sup> While it would be possible to directly use the estimated CATEs in the DGPs, this approach might bias our results towards estimators similar to those used for CATE estimation (Knaus et al., 2021).

We find that all predicted CATEs are negative and statistically different from zero at the 5% significance level. Therefore, we specify the following model for the individual effects:

$$\xi(X_i) = -a\phi(\tilde{\pi}(X_i)) - \max_i \{-a\phi(\tilde{\pi}(X_i))\} \quad (\text{S.1})$$

with  $\tilde{\pi}(X_i) = \frac{\hat{\pi}(X_i)}{\max_i \hat{\pi}(X_i)}$  a normalized version of the estimated propensity score  $\hat{\pi}(X_i)$ , and  $\phi(\cdot)$  the standard normal probability distribution function. The parameter  $a$  controls the degree of heterogeneity. If  $a = 0$ , all individual effects are zero. As  $a$  increases, the degree of heterogeneity increases as well. Figure S.2 displays the individual effects generated by model (S.1) for two different values of  $a$ .



**Figure S.2:** Sorted CATEs and 95% confidence intervals (left panel) and individual effects generated by model (S.1) (right panel).

We use model (S.1) to compute  $\hat{Y}_i(1) = Y_i(0) + \xi(X_i)$  for all units. We then set aside a random validation sample of 10,000 units to evaluate our performance measures detailed below. This approach allows us to assess the out-of-sample predictive power of estimators under investigation (Knaus et al., 2021). We treat the remaining 343,140 units as our population from which we draw random samples for estimation.

After drawing a sample of size  $n$ , we assign the treatment using a Bernoulli process. We consider two scenarios: one with random assignment ( $D_i \sim \text{Bernoulli}(0.5)$ ) and another with assignment based on the “true” propensity score ( $D_i \sim \text{Bernoulli}(\hat{\pi}(X_i))$ ). Notice that in the latter case  $\hat{\pi}(\cdot)$  is used in both the model for individual effects and for treatment assignment. This complicates the task for estimators to separate selection bias from effect heterogeneity. Finally, we construct the observed outcomes for units in the drawn sample as in (2.1).

We split each drawn sample into a training sample  $\mathcal{S}^{tr}$  and an honest sample  $\mathcal{S}^{hon}$  of equal sizes. We then construct a causal tree and two aggregation trees. To build the aggregation trees, we estimate  $\tau(\cdot)$  using the X-learner (Künzel et al., 2019) and the causal forest (Athey et al., 2019) estimators. We use standard cross-validation procedures to select a single partition from the resulting causal and aggregation trees. All these operations are performed utilizing only observations from  $\mathcal{S}^{tr}$ .

To obtain point estimates and standard errors for the GATEs, causal trees follow the approach of Athey and Imbens (2016) and estimate model (3.10). On the other hand, aggregation trees estimate model (3.12). Honest regression forests and 5-fold cross-fitting are employed to estimate the nuisance functions necessary for constructing the doubly-robust scores  $\psi_i^{DR}$ . All these operations are performed using only observations from  $\mathcal{S}^{hon}$ .

We use the external validation sample to assess the quality of estimation. Three performance measures are computed: the root mean squared error, the absolute bias, and the standard deviation of the predictions for each observation in the validation sample:<sup>21</sup>

$$\begin{aligned}
 RMSE(x) &= \sqrt{\frac{1}{R} \sum_{r=1}^R [\hat{\tau}_r(x) - \xi(x)]^2}, \\
 |Bias(x)| &= \left| \frac{1}{R} \sum_{r=1}^R \hat{\tau}_r(x) - \xi(x) \right|, \quad SD(x) = \sqrt{\frac{1}{R} \sum_{r=1}^R \left[ \hat{\tau}_r(x) - \frac{1}{R} \sum_{r=1}^R \hat{\tau}_r(x) \right]^2},
 \end{aligned} \tag{S.2}$$

with  $x$  a generic point in the validation sample,  $R$  the number of replications, and  $\hat{\tau}_r(\cdot)$  the CATE estimated by the tree at the  $r$ -th replication. We summarize these performance measures by averaging over the validation sample. Additionally, we evaluate the actual coverage rates of conventional 95% confidence intervals for the GATEs constructed using the estimated standard errors.

## S2.2. Additional results

Table 1 in the paper presents the results obtained from  $R = 1,000$  replications across three sample sizes and two levels of heterogeneity for honest trees. Table S.4 presents additional results for adaptive trees. Relevant findings are discussed in the paper.

<sup>21</sup> Note that we are evaluating which estimator best approximates the individual effects  $\xi(\cdot)$ . However, the relative performance of the estimators in approximating the unknown CATEs is the same, as the estimator that minimizes the mean squared error for  $\xi(x)$  also minimizes the mean squared error for  $\tau(x)$  (Künzel et al., 2019).



Table S.4: Simulation results for adaptive trees.

	Randomized experiment						Observational study					
	Low heterogeneity			High heterogeneity			Low heterogeneity			High heterogeneity		
	500	1,000	2,000	500	1,000	2,000	500	1,000	2,000	500	1,000	2,000
<b>Panel 1: <math>RMSE</math></b>												
$AT_{XL}$	280.84	223.02	167.98	286.88	227.09	176.50	331.85	264.74	205.49	336.30	267.95	212.67
$AT_{CF}$	256.06	207.57	163.08	258.09	213.20	169.13	268.20	224.43	184.71	275.93	235.53	192.87
$CT$	137.92	94.53	57.36	163.90	111.22	75.63	341.11	334.26	325.07	429.13	450.82	469.35
<b>Panel 2: <math> Bias </math></b>												
$AT_{XL}$	17.49	16.92	16.56	40.38	40.31	39.92	23.80	21.41	21.16	44.17	45.66	43.21
$AT_{CF}$	16.94	16.78	16.44	40.31	40.35	39.83	24.21	21.99	20.75	45.73	46.2	43.13
$CT$	16.39	16.14	15.96	40.15	39.95	39.75	49.96	45.32	43.40	84.52	78.65	87.62
<b>Panel 3: <math>\overline{SD}</math></b>												
$AT_{XL}$	279.96	221.97	166.63	282.30	221.28	169.08	330.51	263.38	203.79	331.57	261.73	205.61
$AT_{CF}$	255.17	206.49	161.75	253.09	207.12	161.63	266.46	222.71	182.83	269.84	228.29	185.15
$CT$	136.29	92.01	53.17	155.88	99.37	57.42	336.22	330.12	321.08	417.16	440.76	457.51
<b>Panel 4: Coverage of 95% CI</b>												
$AT_{XL}$	0.74	0.72	0.70	0.74	0.72	0.70	0.82	0.79	0.76	0.80	0.78	0.76
$AT_{CF}$	0.79	0.75	0.72	0.79	0.75	0.71	0.86	0.83	0.79	0.85	0.82	0.78
$CT$	0.69	0.71	0.76	0.66	0.68	0.75	0.60	0.58	0.58	0.61	0.61	0.62
<b>Panel 5: <math> \overline{T} </math></b>												
$AT_{XL}$	11.79	13.59	14.32	12.00	13.47	14.47	10.40	12.02	13.37	10.38	11.84	13.30
$AT_{CF}$	11.20	12.87	14.02	11.10	12.95	13.91	8.87	10.70	12.52	8.91	10.80	12.37
$CT$	2.02	1.75	1.46	2.50	1.99	1.55	6.35	11.40	20.59	11.68	25.79	58.77

**Note:** The first three panels report the average over the validation sample of the root mean squared error ( $RMSE$ ), absolute bias ( $|Bias|$ ), and standard deviation ( $\overline{SD}$ ) of each estimator. The fourth panel reports coverage rates for 95% confidence intervals. The last panel reports the average number of leaves.

### S3. BOUNDING THE NUMBER OF TREES

**THEOREM S1.** Define the “depth”  $\mathcal{D}$  of a binary decision tree as the number of nodes connecting the root to the furthest leaf. Let  $X \in \mathcal{X}$  be a  $p$ -vector of binary covariates. The number of distinct decision trees constructed by recursively partitioning  $\mathcal{X}$  and having a depth equal to or lower than  $\mathcal{D}$  is bounded from below by  $L_{\mathcal{D}} = \prod_{d=1}^{\mathcal{D}} (p - (d - 1))^{2^{d-1}}$ .

**Proof of Theorem S1:** The proof involves careful counting and is based on the fundamental theorem of counting. We define a *symmetric  $\mathcal{D}$ -depth tree* as any binary decision tree in which the number of nodes connecting the root to each leaf equals  $\mathcal{D}$ , considering the root as a 0-depth tree.

Starting from the entire covariate space  $\mathcal{X}$ , i.e., from the unique 0-depth tree, we observe that since all  $p$  covariates are binary, there is a unique candidate splitting point  $s$  for each covariate. Consequently, there are  $p$  distinct candidate pairs  $(j, s)$  for the first split, allowing the construction of  $p$  distinct symmetric 1-depth trees.

Next, fix a symmetric 1-depth tree, assuming without loss of generality that the split occurred on the first covariate. A symmetric 2-depth tree is generated by splitting both leaves of the nested symmetric 1-depth tree. As a split has already occurred on the first covariate, there are  $p - 1$  distinct candidate pairs  $(j, s)$  available for splitting each terminal node. Thus, from a given symmetric 1-depth tree, it is possible to construct  $(p - 1)^2$  distinct symmetric 2-depth trees. By the fundamental theorem of counting, the total number of distinct symmetric 2-depth trees is  $p(p - 1)^2$ .

A similar argument shows that the number of distinct symmetric 3-depth trees that can be constructed from any symmetric 2-depth tree is  $(p - 2)^4$ . Therefore, the total number of distinct symmetric 3-depth trees is  $p(p - 1)^2(p - 2)^4$ .

Continuing this reasoning, we can derive a closed-form expression for the number of symmetric  $\mathcal{D}$ -depth trees constructed using  $p$  binary covariates:

$$L_{\mathcal{D}} = \prod_{d=1}^{\mathcal{D}} (p - (d - 1))^{2^{d-1}}. \quad (\text{S.3})$$

Note that any binary decision tree with a depth equal to or lower than  $\mathcal{D}$  can be regarded as a subtree of a given symmetric  $\mathcal{D}$ -depth tree, obtainable by collapsing a certain number of internal nodes from the latter. Hence, the set of symmetric  $\mathcal{D}$ -depth trees is a subset of all possible distinct binary decision trees that can be constructed by recursively partitioning  $\mathcal{X}$  with a depth of at most  $\mathcal{D}$ . Therefore,  $L_{\mathcal{D}}$  serves as a lower bound for the number of such trees. □

REMARK S1. Equation (S.3) has an intuitive interpretation. A symmetric  $\mathcal{D}$ -depth tree comprises  $2^{\mathcal{D}}$  terminal nodes. The formula illustrates that, starting from any symmetric  $(d - 1)$ -depth tree,  $2^{d-1}$  leaves must be split to form a symmetric  $d$ -depth tree, with  $p - (d - 1)$  candidate pairs  $(j, s)$  available for each of these splits.

REMARK S2. Symmetric trees with a depth of  $\mathcal{D} > p$  cannot be grown; in such cases,  $L_{\mathcal{D}} = 0$ . Moreover,  $L_{p-1} = L_p$ : starting from any symmetric  $(p - 1)$ -depth tree, each leaf can be split by choosing only one unique candidate pair  $(j, s)$ , resulting in a unique symmetric  $p$ -depth tree for each distinct symmetric  $(p - 1)$ -depth trees.

REMARK S3. For  $p$  categorical covariates, each with  $k$  categories, Theorem S1 holds when substituting  $p(k - 1)$  for  $p$ .