Operationalizing Complex Causes

A Pragmatic View of Mediation

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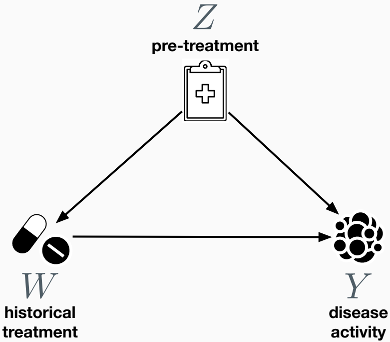
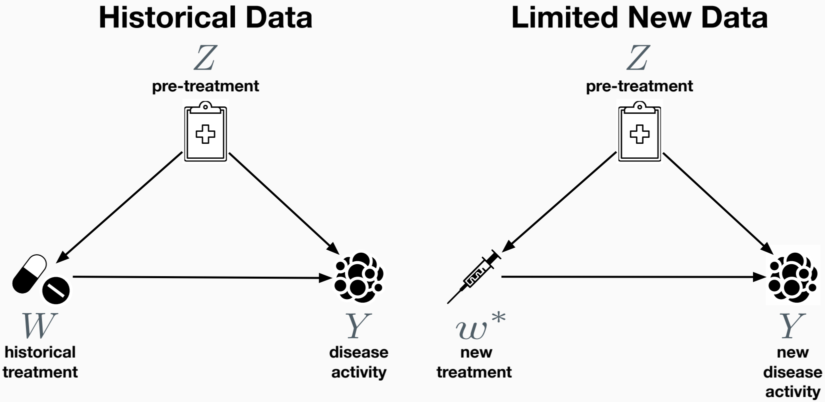
UCL

The Alan Turing Institute

Complex Causes Motivation




Historical Data
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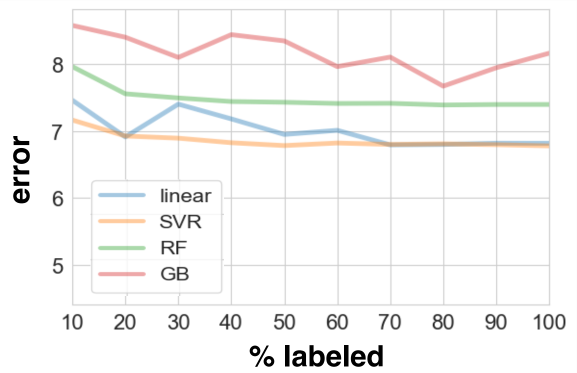
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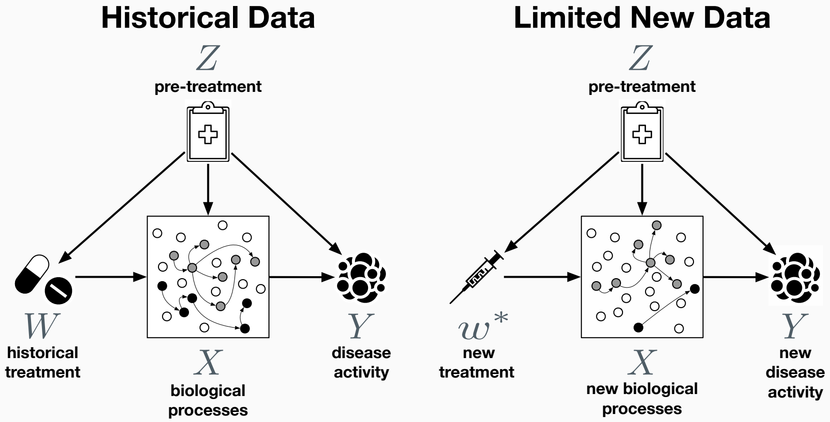
Historical Data
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disease activity
Limited New Data
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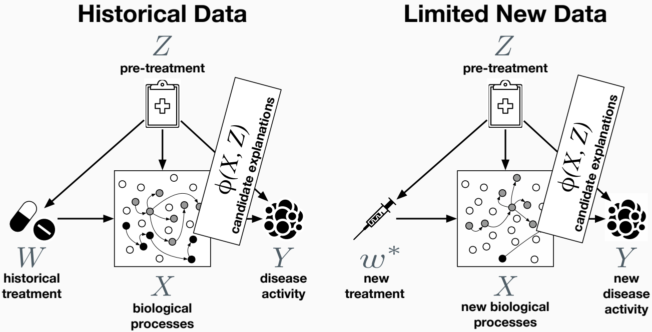
new disease activity



$$Y = \theta_0 + \sum_{i=1}^{d} \theta_i \phi_i(X, Z) + \epsilon$$

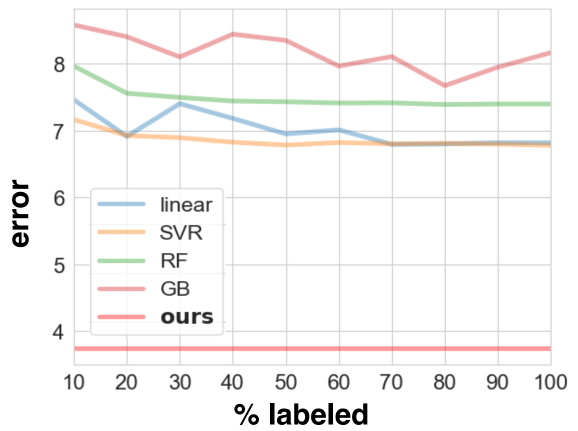
$$\mathbb{E}[Y \mid do(w), z] = \theta_0 + \sum_{i=1}^{d} \theta_i \mathbb{E}[\phi_i(X, Z) \mid w, z]$$

Stage 1

Learn $g_i(W, Z) \equiv \mathbb{E}[\phi_i(X, Z) \mid W, Z]$ for all abstract features via black-box

Stage 2

Learn $\hat{\theta} = \arg\min_{\theta} \mathbb{E}[(Y - \theta^{\top}\hat{\mathbf{g}})^2]$ via regularized regression, to provide sparsity



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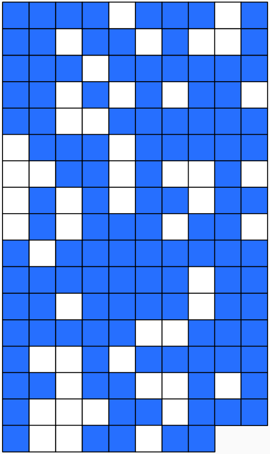
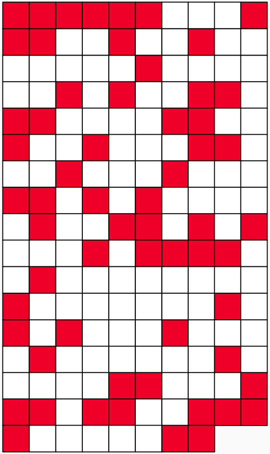
Complex Causes Identifying Pragmatic Mediators

2 Step procedure

Sparse regression, step 2	Cond. independence test, step 1
$\phi \rightarrow Y$	$W \rightarrow \phi$

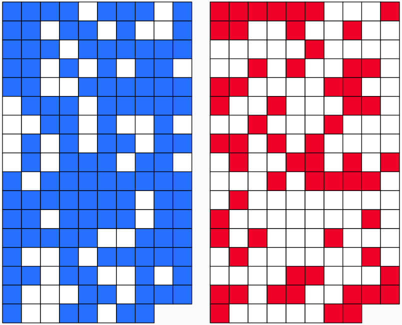




2 Step procedure

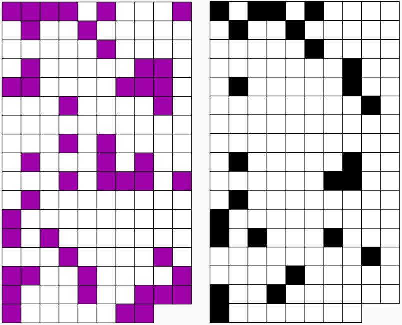
Sparse regression, step 2 — Cond. independence test, step 1

$\phi \rightarrow Y$ — $W \rightarrow \phi$



Intersection identifies pragmatic mediators

$\{W \rightarrow \phi\} \cap \{\phi \rightarrow Y\}$ — ground truth



Conclusion

Common Problem Setup

Interventions applied to complex objects are often **crude**, and we can leverage invariances between **pragmatic mediators** and outcomes to make quality predictions for new interventions where labels are unavailable.

Transfer Learning of Causal Estimations

We propose a simple **2-Stage approach for estimation** of causal effects in novel domains, based on historical data.

Identifying Mechanisms

We show how our 2-Stage approach can help **discover pragmatic mediators that explain outcomes**, and provide insight for new experiments.

Conclusion

Please check out our full paper at
https://arxiv.org/abs/2106.05074

Operationalizing Complex Causes: A Pragmatic View of Mediation

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Abstract

We examine the problem of causal response estimation for complex objects (e.g., text, images, genomics). In this setting, classical *atomic* interventions are often not available (e.g., changes to characters, pixels, DNA base-pairs). Instead, we only have access to indirect or *crude* interventions (e.g., enrolling in a writing program, modifying a scene, applying a gene therapy). In this work, we formalize this problem and provide an initial solution. Given a collection of candidate mediators, we propose (a) a two-step method for predicting the causal responses of crude interventions; and (b) a testing procedure to identify mediators of crude interventions. We demonstrate, on a range of simulated and real-world-inspired examples, that our approach allows us to efficiently estimate the effect of crude interventions with limited data from new treatment regimes.

1. Introduction

Understanding causal mechanisms is a primary goal of scientific inquiry and a crucial prerequisite for planning ef-

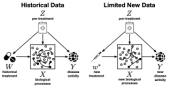

Figure 1. The complex cause problem setting. See text for details.

impact of drug therapies on disease activity. However, careful analysis is required to detect and operationalize these sparse signals, as causal effects are not defined in terms of direct interventions on, say, individual genes, but are instead propagated from a crude treatment (drug administration) on a complex object (the human transcriptome), which affects outcomes (disease activity) through several mediating pathways. Similar complexity arises in other fields, for instance when purported causes are social constructs like "gross domestic product" (Arnold et al., 2020) or large-scale natural phenomena like "El Niño" (Chalupka et al., 2016).

Despite a substantial and growing literature on causal in-

Or play with the code at
https://github.com/limorigu/ComplexCauses

Hoping to see you soon at the poster session!