Abstract. A central problem for correct development of statistical methodology in observational studies with network interference is extending the propensity score definition to include network information. There are five approaches to member-level propensity score definition with corresponding estimation procedures of varying causal complexity. The general network approach quantifies relevant causality in the member-level propensity score using full member information and the entire network. The specific neighborhood approaches quantify relevant causality in the member-level propensity score using member, neighbor (alter), and neighborhood (alter-set) information. There are four types of neighborhood approaches of generally increasing causal complexity: neighborhood as cluster, summary function of member and alter covariate values, variation in binary neighborhood treatment vector, variation in continuous neighborhood treatment. If any nonrandomized propensity score definition must consider either network-based or neighborhood-based information as relevant causality then it is possible to derive a methodological organization.

Key words and phrases: unconfoundedness, propensity score, network interference, observational studies.

1. INTRODUCTION

The theory of nonrandomized inference [49, 44, 10, 48] with network interference [3, 53, 24, 41, 35, 46, 14, 29, 62, 57, 56, 61] has a useful methodological organization [34, 58, 42, 18, 19]. The central object quantifying relevant causal information in nonrandomized studies is the member-level propensity score [47, Section 2.3], so the problem arises of extending the member-level propensity score to nonrandomized studies with network interference. The emphasis on quantifying relevant causality in member-level propensity is supported by emphasis on member-level treatment in randomized causal theory [28, Sections 1.10, 3.1].

The core nonrandomized causal notion in the propensity score is presence (absence) of causal connection [51, Sections 2-3]. Perhaps the presence (absence) potential outcomes are the simplest and most appropriate consideration when conceptual emphasis is on causal connection, which seems a central consideration in aspects of basic causal inquiries [22, Section 5]. There cannot be explicit description of the notion of causal presence (absence) if the potential outcomes are considered from actual treatments of two different types (for instance, as in [20, Section 2]), or from multi-valued [26] or continuous [21] possible treatments. Explicit description of the propensity score as probability of causal presence (absence) seems required to discuss basic aspects of statistical causal connection. It seems useful to distinguish binary- and multi-valued treatments from continuous treatments since presence (absence) reasoning is essentially discrete.

2. ORGANIZATION

The general organization of observational studies with network interference consists of:

1. a *network approach* where the member-level propensity score quantifies relevant causality by conditioning on full member information and the entire network [34],
2. a *neighborhood approach* where the entire network is not needed for quantifying relevant causality in the member-level propensity score [58, 42, 18, 19].

The only valid simplification of relevant causality from the entire network setting of [34] is emphasis on causally-relevant neighborhoods (alter-sets) since subgraph considerations beyond neighborhoods require the network unconfoundedness theory, propensity score definition, and estimation arguments of [34]. Theoretical network observational studies begins with an "unconfoundedness condition" that "considers units observationally equivalent if they occupy identical positions in the network, meaning that they match on all observed neighborhood and higher-order neighborhood dimensions" [34, Section 1]. Any simplification from entire network causality requires the stronger causal assumption of ‘all relevant causality in member-level neighborhoods’ since considering causality from "higher-order neighborhood dimensions" requires the network approach. This stronger assumption delineates four approaches of generally increasing causal complexity (increasing strength of causal assumptions). Each neighborhood approach quantifies relevant neighborhood causality in the member-level propensity score:

1. neighborhood as cluster [58, Section 5],
2. summary function of member and alter covariate values [42, Section 3.2],
3. variation in binary neighborhood treatment vector [18, Section 4],
4. variation in continuous neighborhood treatment [19, Section 2.6].

The notion of causal complexity is relevant in both theory and practice of observational studies. The member-level propensity score is the central object of observational studies since it quantifies relevant causal information [47, Theorems 2, 3]. The propensity score must be used and discussed carefully [30, 13], but it is considered the correct theoretical ground from which to compare causal complexity of observational studies methodologies. There can also be relevant causal information in counterfactual model [2, 6] or sample covariate balance [5, 25] (perhaps in both cases from causality implicit in the population overlap assumption [12] since "more extreme weights" result "when overlap is poor" [5, Section 4]); however, the propensity score is usually considered the central statistical object describing relevant causality in theoretical observational studies. It is unclear whether the network approach should be considered more causally complex than the neighborhood approaches since considering relevant causality from full member information and the entire network seems to require simpler assumptions but more complicated estimation [34]. There is a tradeoff in network observational studies between complexity (or depth) of assumed causal understanding in the propensity score and complexity (or caution) of corresponding estimation procedure; increasing complexity of causal assumptions should increase caution of corresponding causal estimation and decision [40].

2.1 Relevant Causality From Network

[34] build on [52, 9, 32, 55, 33] to study "the use of 'network controls' that depend more generally on $\mathbf{X}$ and $\mathbf{A}$", where $\mathbf{X}$ is the dataset of full member information describing all relevant member causality and $\mathbf{A}$ is the entire adjacency matrix describing all relevant network causality. [34] suggest an estimand, outcome and treatment model, unconfoundedness condition, graph neural network estimation procedure, and propensity score definition motivated by "economically interesting forms of interference such as endogenous peer effects" and "observational data on economic agents that choose to select into treatment" [Section 2.1]. The outcome model and unconfoundedness condition are "considerably weaker" than in settings [38, 1] where interference considerations "require correct specification of a low-dimensional function ... of $(\mathbf{X}, \mathbf{A})$ to summarize confounding" [34, Section 2.1]. The unconfoundedness condition [34, Assumption 1] describes most abstractly the notion of relevant causality in observational studies with network interference: member-level conditional assignment ignorability with well-collected full member information and entire network $(\mathbf{X}, \mathbf{A})$.

[34] articulate the most general propensity score definition in network observational studies with a corresponding nonparametric estimation procedure. [34, Section 2.3] define the probability of member-level causal presence (absence) as $P(Z_i = 1|\mathbf{X}, \mathbf{A})$. Full member information and the entire network describe member-level causal presence (absence) with the simplest assumptions. The entire network is required for general theory since "the presence of complex interference in both the outcome and treatment stages" makes it "generally insufficient to control only for a simple summary statistic" [34, Section 2.1]. The propensity definition of [34] uses the weakest unconfoundedness assumption about the observed dataset's relevant causality. This weakest unconfoundedness assumption [34, Assumption 1] is shared among network observational studies theorists and practitioners in the sense that "normal science, the activity in which most scientists inevitably spend almost all their time, is predicated on the assumption that the scientific community knows what the world is like" [31, p. 5].

[34] also articulate the main distinguishing feature of relevant causality in the network approach from the neighborhood approach:

> unit i's 'covariates' correspond to its network position $(i, \mathbf{X}, \mathbf{A})$ ... Nothing in the standard toolbox for high-dimensional estimation suggests that it is possible to nonparametrically adjust for graph-structured confounders, which is presumably why this has been explicitly avoided in the literature [34, Section 1].

> the literature on neighborhood interference ... impos[es] the additional restriction that ... units are observationally equivalent if they have identical controls W_i, and observationally equivalent units have identical probabilities of being assigned to an exposure mapping ... In our setting, W_i is insufficient to account for confounding. The natural generalization is to require assignment probabilities to be equivalent for units that have *isomorphic network positions* ... Invariance is substantially weaker than the restriction employed under neighborhood interference [Emphasis in original] [34, Section 3.3].

Isomorphic network position considerations imply that "graph neural networks can be used to construct a flexible basis for graph-structured confounders" [34, Section 1.1].

2.2 Relevant Causality From Neighborhood

If the entire network is not needed to quantify relevant network causality in the member-level propensity score then each causal population member must have a causally-relevant neighborhood (or alter-set) since relevant causality beyond neighborhoods requires considering isomorphic network position [34, Section 3.3]. If isomorphic network position considerations–which follow from the simplest assumptions describing relevant network causality [34, Section 3.3]–are not suitable then the only remaining approach is to consider causal neighborhoods. "Higher-order neighborhood dimensions" [34, Section 1] require consideration of isomorphic network position, and the approximate neighborhood interference setting of [32] "creates significant new complications in the form of high-dimensional network confounding" [34, Section 1.1]. Isomorphic network position-based propensity definition and corresponding estimation procedure seems required for observational studies with causal considerations beyond causally-relevant neighborhoods. If isomorphic network position considerations cannot quantify each member's probability of causal presence (absence) then network-related causal information must be through neighborhoods (alter-sets) collected in $\mathbf{A}$. Each neighborhood is interpreted as a causally-relevant (or possibly causally-relevant) set of members. Causally-relevant members are often described as "alters" [42], while possibly causally-relevant members can be described more generally as neighbors.

Neighborhood adaptive estimation–when interference for node i in a randomized experiment depends on an "m-hop neighborhood of node i" which "includes the induced subgraph of all nodes for whom the shortest path to i is less than or equal to m edges " [4, Section 1.2]–can perhaps inform development of the notion of isomorphic network position in observational studies, but cannot inform development of relevant causality from neighborhoods in observational studies since m-hop neighborhoods require considerations of high-dimensional network confounding. Therefore "features of further degree neighbors" [15, Section 2.1] also require consideration of high-dimensional network confounding. Low-order neighborhood interactions [11] extend neighborhood modelling capabilities for randomized experiments and could suggest new estimators [Section 7.2] for the nonrandomized neighborhood approach.

2.2.1 Partial Interference A causal population with valid population overlap contains member outcome values considered causally comparable [12]. Increasing population overlap increases causal comparability as quantified by the strict overlap constant [12, Assumption 3]. Analogously, increasing neighborhood overlap among members of a causal population increases causal comparability through relevant neighborhood causality. Estimation is reduction of member-level variation [16, Section 1.1], so increasing overlap of relevant neighborhood causality simplifies estimation since there is less member-level causal variation to reduce. The cluster-propensity methodology of [58, Section 5] results when neighborhoods overlap completely as clusters.

[58] build on [53, 24, 23, 60] to develop inverse-propensity methodology [63] in network observational studies. Inverse-propensity theory suggests that member outcomes can be correctly weighted to provide unbiased causal estimates from causal quantification in the propensity score [45]. [36] develop the cluster-level inverse-propensity theory of [58] into member-level inverse-propensity theory where each member has a separate causally-relevant neighborhood; however, "a clear use of Horvitz-Thompson inverse probability-weighted estimator in observational network data is still lacking" [18, Section 7]. The cluster-level inverse-propensity estimator of [58, Theorem 6] is a more valid nonrandomized estimator than the inverse-propensity estimator of [36] since relevant causality can be far simpler to quantify in the propensity score when neighborhoods overlap as clusters [58, 37]. If each member has a separate neighborhood of relevant causality it seems too strong to assume relevant causality is quantified in the propensity score to the extent that inverse-propensity estimation is licensed for observational network dataset analysis [15, Section 2.2]. Cautionary considerations when transitioning from relevant network causality to relevant neighborhood causality can invalidate inverse-propensity methodology in studies where each member maps surjectively to all (possibly) causally-relevant neighborhoods.

2.2.2 Neighborhood Summary Function [42] develop [59] by emphasizing latent variable dependence and sparse asymptotics [8, 7] where number of alters grows sparsely with sample size. Unconfoundedness is alter-level [42, Section 3.2], implying that a summary function of member and alter covariate values can simply quantify all relevant alter-set causality in the member-level propensity score [42, Sections 3.1-3.2]. In a dataset reanalysis [42] state "we do not purport to be estimating a true, unconfounded causal effect", which should be read as cautious causal practice rather than clarification of methodological assumptions. The summary functions in [15, Section 2.2] seem inapplicable in the neighborhood approach since causal consideration of "further degree neighbors" implies graph-structured confounders and weaker unconfoundedness assumption [34]. [42] do not require jointly defining and modelling variation in neighborhood-level treatment [18], [19] since every alter in the well-collected network dataset is assumed causally-relevant.

Of the four neighborhood approaches [58, 42, 18, 19] only [42] emphasize causal diagrams. [42] develop new methodology, which present simply in causal diagram language, that critiques previous dataset analysis. Perhaps an emphasis on potential outcomes over causal diagrams in econometrics pedagogy [27] prioritizes empirical practice over methodological development instead of correctly prioritizing methodological development as grounding critique of empirical practice. Methodologies for reducing nonrandomized datasets to causal estimates use causal assumptions as interpretation of the nonrandomized dataset since causal assumptions fit applied problem to methodology. Therefore causal methodological development can always criticize previous dataset analysis on grounds of incorrect methodology as incorrect interpretation: "that the *interpretation* of the experiment is faulty ... such criticisms of interpretation are usually treated as falling within the domain of *statistics*" [Emphasis in original] [17, Section 1.1]. Every nonrandomized dataset requires correct causal assumptions interpreting the natural "experiment". Perhaps potential outcomes and causal diagrams are parts of an incomplete articulation of statistical causal processes [43, p. 249], with causal processes considered as a tentative generalization of causal connections [51].

2.2.3 Binary Neighborhood Causal Variation Adding causal complexity to the methodology of [42] requires jointly considering binary neighborhood treatment vector variation in member-level propensity score definition [18, Section 4]. [42] use summary functions of member and alter covariate values while [18] use summary functions (including the identity function [Section 2.2]) of neighborhood treatment vectors. In not jointly considering neighborhood treatment vector variation in the propensity score definition [42] assume the network dataset includes only causally-relevant alters, whereas [18] require jointly considering possibilities where neighbors can have no causal connection or relevance. Jointly considering neighborhood treatment vector variation increases causal complexity and caution of approach. [42] seem to assume a better-collected causal network dataset than [18] which results in a simpler propensity score definition. Perhaps subclassification-based estimation procedures [18] are generally more causally-cautious than TMLE procedures [42] which are perhaps generally more causally-cautious than cluster-level inverse-propensity procedures [58].

2.2.4 Continuous Neighborhood Causal Variation The methodology of [19, Section 2.6] extends the methodology of [18]–to what seems the limit of causal complexity and caution–by defining continuous neighborhood treatment as weighted average of neighbor treatment. Causal complexity stems from assumed causal understanding so increasing causal complexity requires increasingly rigorous critique of causal assumptions and resulting methodology. Causal understanding of continuous neighbor-level weights requires stronger assumptions than causal understanding of binary neighborhood treatment vector variation since neighbor-level weights seem capable of describing the subtlest quantification of relevant neighbor-member causality.

Summary functions of neighborhood treatment vector can relate jointly to binary member treatment [18], but continuous neighborhood treatment as weighted average of neighbor treatment may jointly require continuous member treatment [19]. Adding causal complexity to the joint propensity score with binary member treatment seems to require complete extension to continuity [19]. Perhaps an observational studies adaptation of low-order neighborhood interactions [11], or of other developments in causal neighborhood theory, can apply to any of [42, 18, 19] (perhaps also [58]) to provide new approaches which clarify a statistical notion of methodological causal complexity. The propensity scores in [18, 19] are member-level propensity scores if the factorization of joint propensity distribution into member distribution and neighborhood distribution is considered methodologically prior to the joint propensity distribution.

3. CONCLUSION

Organization of statistical methodology can help dataset analysis by supporting comparative methodological choice. Methodological choice in network observational studies is necessary since each member-level propensity definition can have varying interpretations of causal complexity (or caution) in various applied problems. For instance, [42] use a propensity definition based in covariate value summary function and emphasize latent variable dependence and sparse asymptotics while [34] use the most general propensity definition and emphasize a weaker unconfoundedness assumption and graph-structured confounders; it is unclear in which types of applied problems the weaker unconfoundedness assumption should be considered as crucial when selecting which methodology provides more scientifically principled propensity definition and estimation procedure. Each new network observational studies dataset requires principled [39] justification of relevant causal assumptions [40] since the new network dataset raises a question of methodological choice answerable only with basic causal considerations. All methodological causal assumptions in network observational studies relate to the propensity definition, so a methodological choice theory for observational studies with network interference could help statisticians discuss causal complexity of applied problems by clarifying the mapping from propensity definitions to causal estimation procedures.

Perhaps a methodological choice theory is not needed in physics since a single experiment can be sufficient. Newton suggests:

> instead of a multitude of things try only the *Experimentum Crucis*. For it is not number of experiments, but weight to be regarded; where one will do, what need of many? ... For if it has already cost us so much trouble to agree upon the matter of fact in the first and plainest experiment, and yet we are not fully agreed: what an endless trouble might it create us, if we should give ourselves up to dispute upon every argument that occurs, and what would become of truth in such a tedious dispute [54].

Statistical causal inference, in contrast, requires "elaborate" investigations "envisag[ing]" as "many different consequences" of the "truth" of "a causal hypothesis" about member outcomes "as possible" [10, Section 5]. Member outcomes are not directly related to relevant causality since design is distinct from analysis [50, 2]. Instead, relevant causality is directly described with unconfoundedness and propensity [47, Theorem 1]. Different approaches to unconfoundedness and propensity can envisage different types of causal consequences for the same applied problem. Basic causal inquiry can help design cautious and elaborate nonrandomized inference.

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