Causality on Longitudinal Data: Stable Specification Search in Constrained Structural Equation Modeling

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Abstract Developing causal models from observational longitudinal studies is an important, ubiquitous problem in many disciplines. A disadvantage of current causal discover algorithms, however, is the inherent instability in structure estimation. With finite data samples small changes in the data can lead to completely different optimal structures. The present work presents a new causal discovery algorithm for longitudinal data that is robust for finite data samples. We validate our approach on simulated data set and real-world data for Chronic Fatigue Syndrome.

Keywords: Causal modeling, Structural equation model, Stability selection, Multiobjective evolutionary algorithm.

1 Introduction

Developing causal models from observational longitudinal studies is an important, ubiquitous problem in many disciplines, which has led to the development of a variety of causal discovery algorithms in the literature [1–5]. A disadvantage of current causal discovery algorithms, however, is the inherent instability in structure learning. With finite data samples small changes in the data can lead to completely different optimal structures, since errors made by the discovery algorithm may be propagated and lead to further errors [6]. In [7] we developed a robust causal discovery algorithm for cross-sectional data. The method performs structure search over Structural Equation Models (SEMs) by maximizing model scores in terms of data fit and complexity. The present work extends our causal discovery algorithm to longitudinal data. We show how to modify the structure scoring rules to accommodate an arbitrary number of time slices. The algorithm produces accurate structure estimates and is shown to be robust for finite samples. We validate our approach on one simulated longitudinal data set and one real-world longitudinal data set for Chronic Fatigue Syndrome.

2 Proposed method

We use SEM for causal modeling. The general form of the equations is

$$x_i = f_i(\operatorname{pa}_i, \varepsilon_i), \quad i = 1, \dots, n.$$
 (1)

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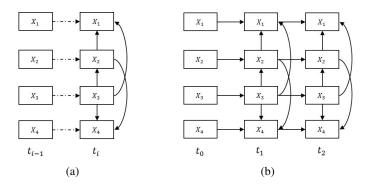


Figure 1: (a) The longitudinal causal model. (b) The "unrolled" causal graph used to generate longitudinal data. It contains four continuous variables (X_1, \ldots, X_4) in three different time slices t_0, \ldots, t_2 .

where pa_i denotes the *parents* which represent the set of variables considered to be direct causes of X_i and ε_i represents errors on account of omitted factors that are assumed to be mutually independent [8]. We score models using both the *chi-square* χ^2 (measuring the data fit) and the *model complexity* (measuring the number of parameters).

We use the method we developed in [7] to perform exploratory search over SEM models. Based on the idea of stability selection [9], the method subsamples the data and generates Pareto optimal models for each subset. The Pareto optimal models are then transformed into an edge and causal path stability graph, such as Figure 3a, by grouping the Pareto optimal models according to model complexity and properly counting the occurrences of edges and causal paths. Stability selection is then performed by specifying two thresholds, π_{occ} and π_{bic} , that determine the relevant part of the stability graphs (i.e., top-left corner) from which we derive the causal model. The method in [7] only handles cross-sectional data. Based on the idea of "unrolling" the network in Dynamic Bayesian Networks [4, 5], we extended the method to handle longitudinal data. We model longitudinal causal relationships with a SEM model consisting of two time slices (Figure 1a) that can be "unrolled" into a network with an arbitrary number of time slices (Figure 1b). Time slice t_{i-1} represents the prior relationships among variables. It does not contain any causal relationships since there is no data available for learning them. Time slice t_i represents relationships within a time slice (intra-slice causal relationships, solid arcs in Figure 1a). Causal relationships between time slices (inter-slice causal relationships, dashed arcs in Figure 1a) always go forward in time, i.e., from time slice t_{i-1} to time slice t_i .

3 Application to Simulated Data

For this experiment, we generated a longitudinal data set with 400 instances from a causal graph as depicted in Figure 1b. The data set consists of three time slices with

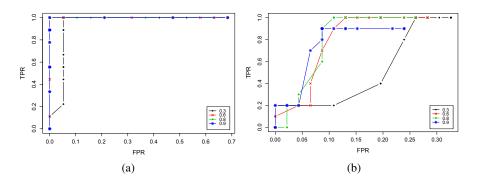


Figure 2: A plot of ROC curves for (a) the edge stability and (b) the causal path stability, for different values of π_{occ} .

four continuous variables for each time slice.⁴ When we searched over SEM models we added prior knowledge that variables X_1 and X_2 do not cause variable X_3 directly.

As the true model is known, we measure the performance of our method by means of the *Receiver Operating Characteristic* (ROC) [10] for both edges and causal paths. The threshold π_{occ} is fixed to a value ($\pi_{occ} \in \{0.3, 0.6, 0.8, 0.9\}$) while π_{bic} is varied. We compute the *True Positive Rate* (TPR) and the *False Positive Rate* from the CPDAG of the true model. As for an example, in the case of edge stability, a true positive means that an edge that appears within the top-left region bounded by π_{occ} and π_{bic} also exists in the CPDAG of the true model. Figure 2 portrays the ROC curves for both edge and causal path stability. Generally we can see that higher values of π_{occ} tend to give better ROC curves. This suggests that our approach is able to find the underlying structure with high reliability scores. A notable point is that the ROC curves stop at a TPR and/or FPR value lower than 1. Since some of the edges and paths are disallowed (i.e., no edges in time-slice t_{i-1} and no paths from t_i to t_{i-1}) some of the edges and causal paths in the stability graphs end up with a selection probability of 0 and the result is that the ROC curves cannot reach the upper right corner with TPR = FPR = 1.

4 Application to Real-world Data

For an application to real-world data, we consider a data set about *Chronic Fatigue Syndrome* (CFS) which consists of 183 subjects and five time slices with six discrete variables [11]. The variables are, *fatigue* severity, the sense of *control* over fatigue, *focusing* on the symptoms, the objective activity of the patient (*oActivity*), the subject's perceived activity (*pActivity*), and the physical *functioning*. We use *Expectation Maximization* implemented in SPSS [12] to impute the missing values. As all of the variables have large scales, e.g., in the range between 0 to 155, we treat them as continuous variables. We added prior knowledge that the variable fatigue does not cause any of the other variables directly.

⁴ Can be downloaded at http://cs.ru.nl/~rahmadi/data.html

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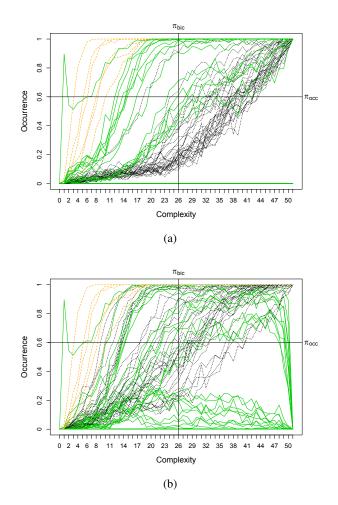


Figure 3: The top-left region contains the relevant causal relations. (a) The edge stability graph. (b) The causal path stability graph. Orange-dashed lines represent inter-slice relationships between the same variables, black-dotted lines represent inter-slice relationships between different variables, green-solid lines represent intra-slice relationships.

Figure 3a shows nineteen relevant edges were found, consisting of eleven intraslice and eight inter-slice relationships which among of these, six are between the same variables and two are between different variables. Figure 3b shows thirty-two relevant causal paths were found, consisting of twelve intra-slice and twenty inter-slice relationships which among of these, six are between the same variables and fourteen are between different variables. For a more intuitive representation, we combine the stability graphs into a model using the following procedure. First, the nodes are linked according to the nineteen relevant edges. Second, edges are oriented according to our background knowledge. Eight of the inter-slice relationships are oriented from time slice t_{i-1} to t_i and five of the intra-slice edges can be oriented since it is known that the variable fatigue does not directly cause any other variable. Third, the edges are oriented according to the relevant causal paths, which results in another twenty-eight directed edges. The inferred model is shown in Figure 4. Each edge is annotated with a reliability score which is the maximum score obtained in the top-left region of the edge stability graph.

From the stability graphs we can see that the most stable causal relations are the inter-slice relations between the same variables followed by some of the intra-slice causal relations. Almost all of the inter-slice relations between different variables are not considered relevant. A directed edge $X \rightarrow Y$ in Figure 4 indicates that a change in variable *X* causes a change in variable *Y*. In the intra-slice causal relationships, we found that all variables are direct causes for fatigue severity. We also found all variables, except *fatigue*, to be direct causes for the perceived activity. Furthermore, the variable *control* is a direct cause for both focusing on the symptoms and physical functioning. Generally the inter-slice relationships show direct causes between the same variables. In addition, the variables *pActivity* and *control* indicate a stronger direct cause for fatigue severity and focusing on symptoms, respectively, as they contribute a direct cause in both time slices. The inferred model is consistent with results reported in the medical literature [11, 13, 14].

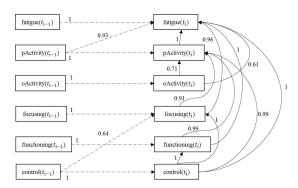


Figure 4: The inferred model of CFS by combining the edge stability and causal path stability graphs.

5 Conclusion

Causal discovery from longitudinal data is an important, ubiquitous problem in science. Current causal discovery algorithms, however, have difficulty dealing with the inherent instability in structure estimation. The present work introduces a new discovery algorithm for longitudinal data that is robust for finite samples. Experimental results on both artificial and real-world data sets show that the method results in reliable structure estimates. 6 Causality on Longitudinal Data

Acknowledgments

The research leading to these results has received funding from the DGHE of Indonesia and the European Community's Seventh Framework Programme (FP7/2007-2013) under grant agreement n° 305697.

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