

## Causal Discovery Using A Bayesian Local Causal Discovery Algorithm

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### Abstract

This study focused on the development and application of an efficient algorithm to induce causal relationships from observational data. The algorithm, called BLCD, is based on a causal Bayesian network framework. BLCD initially uses heuristic greedy search to derive the Markov Blanket (MB) of a node that serves as the “locality” for the identification of pair-wise causal relationships. BLCD takes as input a dataset and outputs potential causes of the form variable  $X$  causally influences variable  $Y$ . Identification of the causal factors of diseases and outcomes, can help formulate better management, prevention and control strategies for the improvement of health care. In this study we focused on investigating factors that may contribute causally to infant mortality in the United States. We used the U.S. Linked Birth/Infant Death dataset for 1991 with more than four million records and about 200 variables for each record. Our sample consisted of 41,155 records randomly selected from the whole dataset. Each record had maternal, paternal and child factors and the outcome at the end of the first year—whether the infant survived or not. Using the infant birth and death dataset as input, BLCD output six purported causal relationships. Three out of the six relationships seem plausible. Even though we have not yet discovered a clinically novel causal link, we plan to look for novel causal pathways using the full sample.

### Keywords:

Causal Discovery, Infant Mortality, Bayesian Networks

### Introduction

Causal discovery is a challenging and important task. Causal knowledge aids planning and decision making in almost all fields. For example, in the domain of medicine, determining the cause of a disease helps in prevention and treatment.

Well-designed experimental studies, such as randomized controlled trials, are typically employed in assessing causal relationships. Here the value of the variable postulated to be causal is set randomly and its effects measured. These studies are appropriate in certain situations, for example, animal studies and studies involving human subjects that have undergone a thorough procedural and ethical review. Experimental studies may not, however, be feasible in many contexts due to ethical, logistical, or cost considerations. These practical limitations of experimental studies heighten the importance of exploring, evaluating and

refining techniques to learn more about causal relationships from observational data, as for example data routinely collected in astronomy, earth sciences and healthcare. The aim is not to replace experimental studies, which are extremely valuable in science, but to complement experimental studies when feasible.

This paper introduces a new Bayesian local causal discovery algorithm called BLCD that is designed for efficient discovery of possible causal relationships from large observational databases. The time complexity of BLCD makes it appropriate for exploring possible causal relationships in databases that contain a very large number of records (on the order of hundreds of thousands) and a moderately large number of measured variables per record (on the order of hundreds).

In our study using BLCD we focus on investigating factors that may contribute causally to infant mortality in the United States. Infant mortality is one of the most important public health problems in the U.S. [1]. International comparisons based on data from the United Nations statistical office for the year 1991 show that there are 21 countries in the world with lower infant mortality rates than the United States. Japan had the lowest rate of 4.4, while the US rate was 8.9 [2]. The present study is a prelude to further work using larger real-world medical and biological datasets.

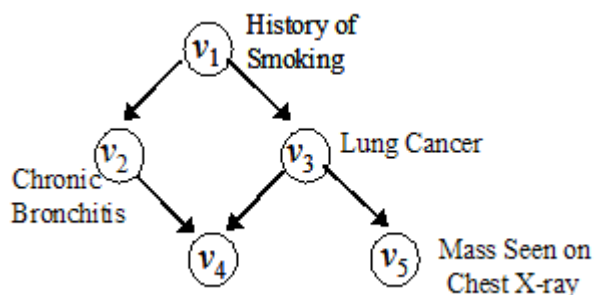


Figure 1 - A hypothetical causal Bayesian network structure

### Materials and Methods

#### Assumptions for Causal Discovery

In the research reported here, we use causal Bayesian networks to represent causal relationships among model variables. This section provides a brief introduction to causal Bayesian net-

works, as well as a description of the assumptions we used to apply these networks for causal discovery.

A causal Bayesian network (CBN) is a Bayesian network in which each arc is interpreted as a direct causal influence between a parent node (variable) and a child node, relative to the other nodes in the network [3]. Figure illustrates the structure of a hypothetical causal Bayesian network structure, which contains five nodes. The causal network structure in Figure indicates, for example, that a *History of Smoking* can causally influence whether *Lung Cancer* is present, which in turn can causally influence whether a patient experiences *Fatigue* or presents with a *Mass Seen on Chest X-ray*.

The **causal Markov condition** gives the independence relationships that are specified by a causal Bayesian network:

A variable is independent of its non-descendants (i.e., non-effects) given just its parents (i.e., its direct causes).

According to the Markov condition, the causal network is representing that the chance of a *Mass Seen on Chest X-ray* will be independent of a *History of Smoking*, given that we know whether *Lung Cancer* is present or not. While the causal Markov condition specifies independence relationships among variables, the **causal faithfulness condition** specifies *dependence* relationships:

Variables are independent only if their independence is implied by the causal Markov condition.

For the causal network structure in Figure three examples of the causal faithfulness condition are (1) *History of Smoking* and *Lung Cancer* are probabilistically dependent, (2) *History of Smoking* and *Mass Seen on Chest X-ray* are dependent, and (3) *Mass Seen on Chest X-ray* and *Fatigue* are dependent. The intuition behind that last example is as follows: a *Mass Seen on Chest X-ray* increases the chance of *Lung Cancer* which in turn increases the chance of *Fatigue*; thus, the variables *Mass Seen on Chest X-ray* and *Fatigue* are expected to be probabilistically dependent. In other words, the two variables are dependent because of a common cause (i.e., a confounder). The causal Markov and faithfulness conditions describe *probabilistic* independence and dependence relationships, respectively, that are represented by a CBN.

Before we move on to the next property of a CBN, we introduce our notational convention. Sets of variables are represented in bold and upper case, random variables by upper case letters italicized and lower case letters will be used to represent the value of a variable or sets of variables. When we say  $\mathbf{X} = \mathbf{x}$ , we mean an instantiation of all the variables in  $\mathbf{X}$ , while  $X=x$  denotes that the variable  $X$  is assigned the value  $x$ . Graphs are denoted by a bold italicized  $\mathbf{G}$ .

### BLCD: A Bayesian Local Causal Discovery Algorithm

We introduce a Bayesian local causal discovery algorithm (BLCD) that conjectures causal relationships between pairs of

variables that have no common causes (confounders). Instead of using constraint-based independence and dependence tests, we score the models by a Bayesian method. This confers the following advantages:

1. Allows informative causal priors to be incorporated.
2. Provides a quantitative posterior assessment of causality, based on prior belief and data.

BLCD assumes the following:

**Assumption 1:** The causal Markov condition

**Assumption 2:** The causal faithfulness condition

$$X \rightarrow Y \quad (1) \qquad X \leftarrow Y \quad (2)$$

Figure 2 - Two independence-equivalent BNs.

We now introduce the concept of *independence equivalence*. Consider two Bayesian network structures  $Bs1$  and  $Bs2$ .  $Bs1$  and  $Bs2$  are said to be independence equivalent if they represent the same conditional independence assertions for  $\mathbf{V}$ , where  $\mathbf{V}$  is the set of variables in  $Bs1$  and  $Bs2$  [4]. The two network structures in Figure 4 are independence equivalent. In particular they represent no conditional independence assertion. Likewise, all the three network structures in Figure 5 are also independence equivalent asserting that  $X$  and  $Z$  are conditionally independent given  $Y$ . Two network structures are independence equivalent if and only if they have the same “V” structures and also share other edges (ignoring directions) that are not part of a “V” [5]. A “V” structure involving variables  $W1$ ,  $W2$  and  $X$  is shown in Figure 4. There is a directed edge from  $W1$  to  $X$  and another directed edge from  $W2$  to  $X$ . There is no edge between  $W1$  and  $W2$ . A “V” structure contains a “collider”; the node  $X$  is a collider. Since  $W1$  and  $W2$  are marginally independent,  $X$  is also termed an *unshielded* collider. Figure shows a model in which  $W1$  and  $W2$  are dependent and where  $X$  is a *shielded* collider. BLCD requires that a node  $X$  be an unshielded collider in order to discover the causal effects of  $X$ .

Using the BLCD search strategy, for each pair of nodes  $X$  and  $Y$  where  $X$  is a collider, the probability of  $XY$  will be derived under assumptions. We illustrate this first using a hypothetical domain with four discrete random observed variables —  $W1$ ,  $W2$ ,  $X$ , and  $Y$  (see Figure 6). The model  $\mathbf{G1}$  has the “Y” structure format. A “Y” structure is required in BLCD to infer pairwise causal relationships in an unconfounded way from observational data making just the two basic assumptions (causal Markov and causal faithfulness) for causal discovery.

$$X \rightarrow Y \rightarrow Z \quad (1)$$

$$X \leftarrow Y \leftarrow Z \quad (2)$$

$$X \leftarrow Y \rightarrow Z \quad (3)$$

Figure 3 - Three independence-equivalent BNs

Figure 6 also shows models  $\mathbf{G2}$  and  $\mathbf{G3}$ , from our four variable domain. The 3 models shown are a small subset of the 543 potential CBN models for a four variable domain.

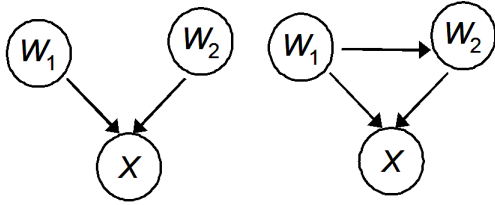


Figure 4- A «V» structure Figure 5- A «Shielded» collider

The following equation is used in BLCD to approximate the probability of an unconfounded causal relationship  $X \rightarrow Y$ :

$$P(X \rightarrow Y | D) = \frac{\text{Score}(G_1 | D)}{\sum_{i=1}^{543} \text{Score}(G_i | D)} \quad (1)$$

The above scoring function assigns relative scores to the models that represent the probability of the model given data and prior knowledge. The scoring can be done, for example, using a Bayesian metric such as the K2 metric [6] or the BDe metric [7]. Our implementation of the scoring function uses the BDe metric with uniform parameter and structure priors.

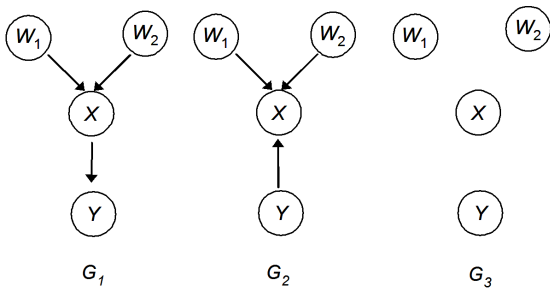


Figure 4 - Three causal models that contain four nodes out of the possible 543 models.  $G_1$  has a “Y” pattern.

The 543 structures cover the space of CBN models exhaustively and no other structure has the same dependence/independence properties as  $G_1$ , i.e., there is no other CBN in the four node domain in the independence equivalent class of  $G_1$ . In the large sample limit the posterior probability of  $G_1$  will approach 1.0 relative to the other 542 models if indeed (1) X causally influences Y in an unconfounded manner, (2) X is a collider of W1 and W2 in the distribution of the causal process generating the data, and (3) the Markov and Faithfulness conditions hold. Equation 1 serves as a lower bound on  $X \rightarrow Y$ , relative to considering only the 543 CBN structures on four variables. Equation 1 does not, however, model latent variables, and thus, it is only a heuristic Bayesian score. We are giving up a full Bayesian treatment of the problem in order to gain computational efficiency.

The heuristic methodology to select the tetrads ( $W_1, W_2, X$  and  $Y$ ) is given below. The method involves identification of the *Markov Blanket* of each random variable. The Markov Blanket (MB) of a node  $X$  in a CBN  $G$  is the union of the set of parents of  $X$ , the children of  $X$ , and the parents of the children of  $X$  [4]. Note that it is the *minimal* set of nodes when conditioned on (in-

stantiated) that ensures that a node  $X$  will be independent of all the other nodes in the CBN. Let  $V$  be the set of all variables in  $G$ ,  $B$  the MB of  $X$ , and  $A$  be  $V \setminus (B \cup X)$ . Conditioning on  $B$  renders  $X$  independent of  $A$ . For example, in the hypothetical CBN shown in Figure , the MB of node  $v_4$  is composed of  $v_2$  and  $v_3$ ; the MB of node  $v_2$  is  $v_1, v_3$ , and  $v_4$ .

We derive the MB of a node (designated as  $B$ ) by a greedy heuristic search and refer to it as the *Procedure MB*. The set  $B \subseteq (V \setminus X)$  that maximizes the score for the structure  $BX$  based on a greedy forward search as described in [6] is initially identified. This is followed by a one step backward greedy search that prunes  $B$  to yield set  $B \subseteq B$  that maximizes the score for the structure  $BX$ . This set  $B$  is updated using the following rule: If  $X$  is in the MB of  $Y$ , but  $Y$  is not in the MB of  $X$ , add  $Y$  to the MB of  $X$ . We refer to this rule as the *MB update rule*.

**BLCD steps**

1. **Derive the Markov Blanket:** For each node  $X \in X$ , heuristically derive the Markov Blanket of  $X$  using the *Procedure MB*.  $X$  denotes the set of all random observed variables in the dataset. Let  $B$  denote the MB of  $X$ .
2. **Update B:** Apply the *MB update rule*.
3. **Pick  $W_1, W_2, X$  and  $Y$ :** Select sets of four variables from the set obtained by the union of  $B$  and  $X$  as follows. We refer to each set of four variables as a tetrasets  $T$ . Since we are focusing on the MB of  $X$ ,  $X$  is an essential element of  $T$ . Note that each tetrasets can give rise to 3 “Y” patterns where the  $X$  variable is a cause and each of the other three are potential effects.
4. **Derive  $P(X \rightarrow Y | D)$ :** For each of the 3 “Y” patterns, the probability of  $XY$  is derived using Equation 1.
5. **Generate output:** If  $P(X \rightarrow Y | D) > t$ , where  $t$  is a user-set threshold, then output  $X \rightarrow Y$ .

By setting  $t$  close to 1.0, we avoid generating many false positives, which is one of our key goals. In causal datamining we do not want to overwhelm the user with many false positives. We would like to trade off recall (number of true causal relationships output over the total number of true causal relationships) for precision or positive predictive value (number of true causal relationships over the total number of relationships output). In other words we want to improve the signal to noise ratio in the output and the goal is to pick just the signals (true causal relationships).

BLCD can be implemented in an *anytime* framework, to output the purported causes as they are found. To discover the effects of a node  $X$ , we require only the MB of  $X$  and data on  $X$  and the variables in the MB of  $X$ .

**Related work**

A review of the philosophical literature on causality is beyond the scope of this paper. For a detailed discussion of the relationship between statistical association and causation, including philosophical issues, see for example [8] and [9].

Constraint-based approaches to causal discovery were put forward by Pearl and Verma [10] and by Spirtes, Glymour, and Scheines [11]. The PC and FCI algorithms, for instance, take a global approach to causal discovery and output a graph with different types of edges between all the variables to represent for

example that  $X$  causes  $Y$ ,  $X$  does not cause  $Y$ , or the causal direction is undetermined [12]. The FCI algorithm can also model latent variable patterns.

Earlier research on learning Bayesian networks from data using a Bayesian approach [6,7] has simultaneously modeled all the causal relationships among the model variables. These global approaches can require long search times when the number of variables is large. When they are used to model latent variables, these approaches can be extremely slow, even when modeling only a few variables.

LCD [13] and its variants [14] output causes of the form  $X$  causes  $Y$  and take a local and constraint-based approach to causal discovery (evaluate only triplets of the form  $WXY$ ). By searching only for pairwise causal relationships, they trade off completeness for efficiency. They also need background knowledge in the form of instrumental variable(s). Silverstein and others have used a variant of LCD to perform *market basket analysis* to discover causal association rules [15]. Their algorithm uses patterns such as ACB to infer that  $A$  and  $B$  cause  $C$ , assuming no hidden variables and confounding.

Researchers have also proposed a constraint-based algorithm for induction of Bayesian networks by first identifying the *neighborhood* (Markov blanket) of each node [16]. They refer to this as the *GS Markov blanket algorithm*. The Grow-Shrink (GS) Markov blanket algorithm attempts to address the two main limitations of the PC and FCI algorithms: 1. possible exponential time complexity and 2. unstable higher order conditional independence tests [16]. However, GS is still exponential in the size of the Markov blanket. The GS algorithm does not make explicit claims about causal discovery. Tsamardinos et al. have proposed a feature subset selection method for supervised learning using the Markov blanket approach [17]. Aliferis et al. have proposed HITON, an algorithm to determine the MB of an outcome variable [18]; it would be interesting to use HITON in step 1 of BLCD. Tsamardinos et al. discuss the possibility of interpreting the MB as direct causal relationships but the algorithm in that paper does not specifically distinguish between causes and effects of a node [19].

To the best of our knowledge there is no Bayesian local causal discovery algorithm described in the literature.

## Experimental Methods

In this study we apply BLCD to the Infant Birth and Death dataset that is described below.

### Infant Birth and Death Dataset

We used the U.S. Linked Birth/Infant Death dataset for 1991 [20]. This dataset consists of information on all the live births in the United States for the year 1991. It also has linked data for infants who died within one year of birth. More than two hundred variables containing various maternal, paternal, fetal and infant parameters are available. For the infants who died within the first year, additional data on mortality, including cause of death, is reported. The records total more than four million and the infant death record number is 35,496. We selected a random subset of 41,155 cases for use in the current study. We did so in order to limit the computational time complexity of searching for caus-

al patterns in the data. A total of 87 variables were selected after eliminating redundant variables and variables not of clinical interest, such as ID number.

### Applying BLCD to the Infant Mortality Database

We implemented BLCD in the C programming language. It takes as input the infant birth and death dataset  $D$  and outputs pairwise causal relationships of the form “variable  $X$  causally influences variable  $Y$ ”. It took 2 hours to examine all 87 variables in  $D$ , evaluate 3741 potential pairwise causal relationships, and output six purported causal relationships. The program was run on a PC with a 3 GHz Intel processor, 2 Gigabytes of RAM, and running the GNU/Linux operating system.

## Results

When applied to the infant birth and death dataset, BLCD output six purported causal relationships. Table 1 contains the relationships.

Table 1: The output of BLCD

Cause	Effect
1. Heart malformations	Infant outcome
2. Hydrocephalus	Infant outcome
3. Weight gain during pregnancy	Infant circulatory/respiratory anomalies
4. Microcephalus	Ultrasound
5. Diaphragmatic hernia	Plurality of birth
6. Five minute Apgar score	Infant heart malformations

The probability distributions associated with relationships 1 and 2 in Table 1 are as follows:

$$P(\text{infant alive at one year} \mid \text{heart malformations}) = 0.797$$

$$P(\text{infant alive at one year} \mid \text{no heart malformations}) = 0.992$$

$$P(\text{infant alive at one year} \mid \text{hydrocephalus}) = 0.5$$

$$P(\text{infant alive at one year} \mid \text{no hydrocephalus}) = 0.992$$

## Discussion

In this section we discuss the biological plausibility [21] of the BLCD output. We realize that additional evaluation is needed, and as stated in the next section, we intend to pursue it. Out of the six relationships in Table 1, three appear plausible. Causal relationships #3 linking “weight gain during pregnancy” to “infant circulatory/respiratory anomalies”, #5 linking “diaphragmatic hernia” to “plurality of birth”, and #6 linking “five minute apgar score” to “heart malformations” can be interpreted with confidence only as an association.

Causal relationship #1 postulates that “heart malformations” is a cause of “infant mortality”. Likewise, causal relationship #2 proposes “hydrocephalus” as a cause for “infant mortality”. Both these congenital anomalies are known to adversely affect infant outcomes. The conditional probability distributions mortality”.

Relationship #4 proposes “microcephalus” as a causal influence for “ultrasound”. If microcephalus is suspected it is likely that the frequency of ultrasound investigations will increase as a part of follow up.

## Conclusions and Future Work

BLCD is an efficient algorithm that uses the local causal discovery framework and a Bayesian approach. By making use of the “Y” pattern for identifying unconfounded pairwise causal relationships and the Markov Blanket for defining the “locality” of a node, BLCD is able to output direct causal relationships, while keeping the number of false positives low. The results in this paper provide preliminary support for BLCD serving as a useful tool in generating causal hypotheses from observational datasets.

In a separate paper, we plan to publish our results of applying BLCD to data that was generated from well known causal Bayesian networks that have been described in literature.

In future research, we plan to explore new search techniques in an attempt to increase the number of causal relationships output by BLCD while still retaining computational efficiency and a high positive predictive value. We also plan to use additional datasets and domain experts to evaluate the output of the algorithm.

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