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Cause-and-Effect Diagrams

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Abstract: Cause and effect is a basic knowledge driven by theoretical and empirical considerations. Several tools have been proposed to map cause-and-effect relationships, with some more heuristics and some highly quantitative. This article covers the Ishikawa fishbone diagram, structural equation models, Bayesian networks, and causal networks.

1 Introduction

Statisticians have always been careful not to confuse **correlation** with **causation**[1]. A famous example, derived from the statistics on the population of Oldenburg in Germany and the number of observed storks in the city in 1930–1936, demonstrates why correlation does not imply causation[2]. Figure 1 is a **scatter plot** of population size versus number of storks.

If we look at the data in Table 1, we quickly realize that time is a **lurking variable** and that both variables in the scatter plot increase with time, hence their correlation. Since storks need chimneys to make their nests, more buildings, due to an increase in population, might explain why more storks are nesting in Oldenburg.

Correlation is therefore not causation, and scatter plots are not sufficient to describe cause-and-effect relationships. **Causality** is, however, a basic element of the scientific method and management skills. Establishment of causality relies on a combination of axiomatic thought and empirical evidence derived from **observational studies** or **designed experiments**. Cause-and-effect diagrams are used to present such relationships. We will present three such diagrams: Ishikawa diagrams, **structural equation models**, and **Bayesian networks**. We will also discuss causal networks and the application of counterfactuals and the "do" calculus on such networks.

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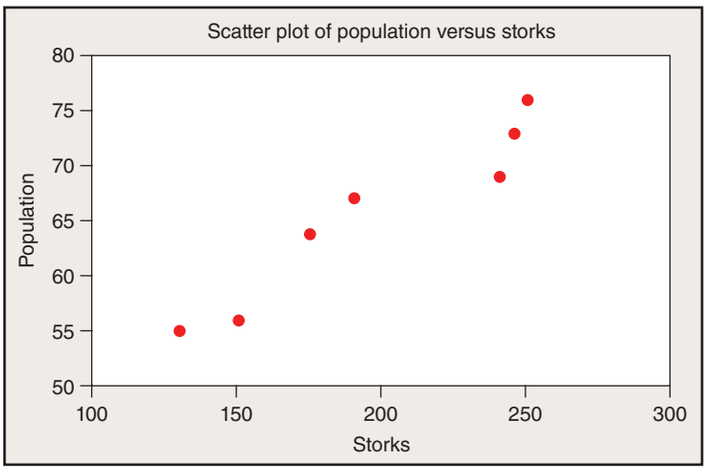

Figure 1. Scatter plot of number of storks versus population size.

Table 1. Population and number of storks, by year.

Year	1930	1931	1932	1933	1934	1935	1936
Population in thousands	50	52	64	67	69	73	76
Number of storks	130	150	175	190	240	245	250

2 Ishikawa Diagrams

In the summer of 1943, at the University of Tokyo, Dr Kaoru Ishikawa was explaining to the engineers from the Kawasaki Steel Works how various factors can be sorted out and related in specific ways. This was the origin of the cause-and-effect diagram, later called the *Ishikawa diagram*. The Ishikawa diagram, also called a *fishbone diagram*, came into wide use in the Japanese industry and became a critical tool for **quality control** and quality improvement all over the world. It is one of the seven essential tools for quality and process improvement, also called the *magnificent seven*[3]. The Ishikawa diagram is used to identify, explore, and display possible causes of a problem or event. A typical diagram will have 4–6 main branches for causes affecting one type of event. Traditional classifications of these branches are five Ms: Manpower, Machines, Materials, Measurements, and Methods or, in administrative areas, the four Ps: Policies, Procedures, People, and Plant. A modern variation on the five Ms uses six categories: People, Machines, Materials, Measurements, Methods, and Environment. These are only suggestions typically used to jump-start the brainstorming session in which the diagram is completed. Figure 2 is an example from an improvement project initiated by the dean of a school of management to improve the operation of his office, and, in particular, the scheduling of appointments[4]. The improvement team, consisting of the dean, two professors at the school, and the two secretaries at the dean's office, brainstormed to list causes for an unusually high number of "collisions" in the dean's schedule. Many meetings with the dean were delayed or even canceled at the last minute. This problem led to complaints by the faculty and staff about the poor service level of the dean's office. The possible causes that were listed included the open-door approach, lack of meeting agendas, interruptions of all sorts, and no scheduled end time to meetings. Following some more analysis, the dean's office instituted a procedure where all meetings were scheduled

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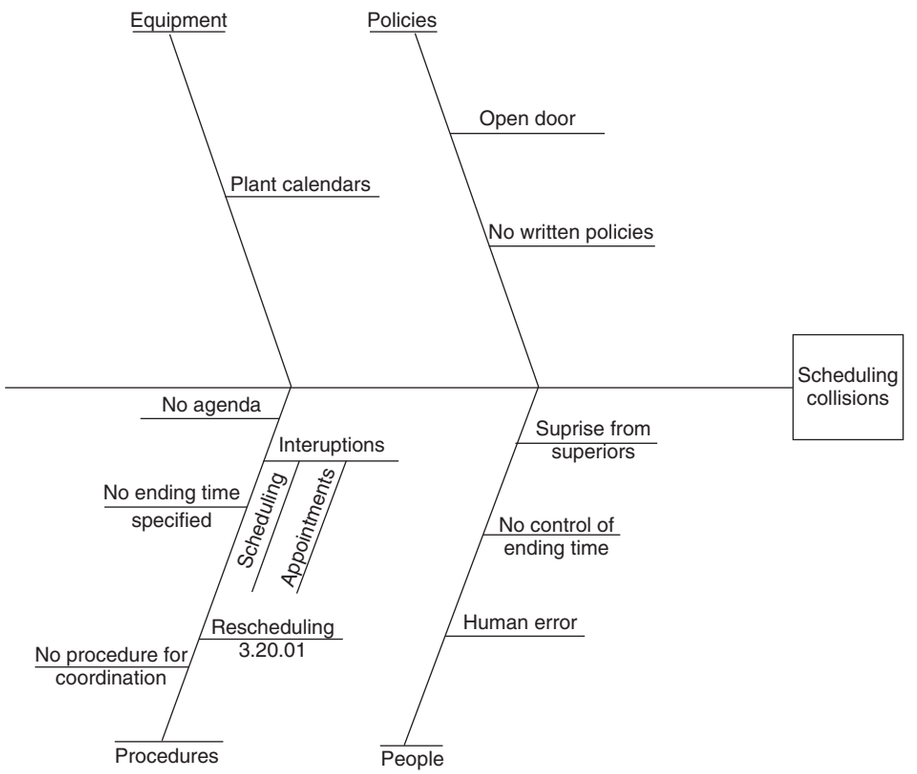

Figure 2. Ishikawa diagram of causes for scheduling collisions at a dean's office.

with an agenda and an end time. A confirmation note, with this information, was sent ahead of time to the meeting participants. "Collisions" dropped by 50%, and the dean's office became a model of service quality on campus. The secretaries even started planning teleconference meetings for specific topics at everyone's satisfaction. For more on Ishikawa diagrams, *see* Ishikawa[3], Brassard[5], and Kenett and Zacks[6].

3 Structural Equation Models

The geneticist Sewall Wright developed a "method of path coefficients," which is partly graphical and partly algebraic to explain genetic phenomena. **Path analysis** allows researchers to compute the magnitude of cause-and-effect relationships from correlation measurements, provided the path diagram represents correctly the causal process underlying the data[7, 8]. Later, economists developed a similar approach labeled structural equation modeling. We use here both terms interchangeably.

Structural equation modeling involves the specification of an underpinning **linear regression** model incorporating the structural relationships between unobserved and **latent variables**, together with a number of observed or measured indicator variables.

Structural equation models assume that there is a causal structure among a set of latent variables, and that the observed variables are indicators of the latent variables. The latent variables may appear as linear combinations of observed variables, or they may be intervening variables in a causal chain.

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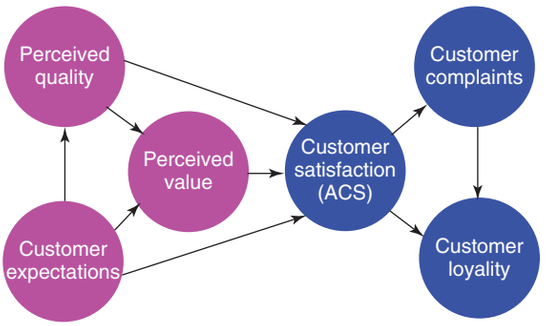

Figure 3. The American customer satisfaction survey index model.

As latent variables are, by definition, unobservable, their measurement must be obtained indirectly. This is done by linking one or more observed variables to each unobserved variable. A fully specified structural equation model is potentially a complex interplay between a large number of observed and unobserved variables, and residual and error terms. For more on structural equation models, *see* Bollen[9], Hoyle[10], and Kline[11].

The American customer satisfaction index (ACSI) is a prime application of a structural equation model. It uses telephone customer interviews, based on a structured questionnaire, as inputs. The latent variables in the ACSI model are presented in Figure 3 as a cause-and-effect model with indices for drivers of satisfaction on the left-hand side (customer expectations, perceived quality, and perceived value), satisfaction (ACS) in the center, and outcomes of satisfaction on the right-hand side (customer complaints and customer loyalty, including customer retention and price tolerance). The indices are multivariable components measured by several questions that are weighted within the model. The questions assess customer evaluations of the determinants of each index. Indices are reported on a 0–100 scale. The survey and modeling methodology quantifies the strength of the effect of the index on the left to the one to which the arrow points on the right. These arrows represent "impacts." The ACSI model is self-weighting, to maximize the explanation of customer satisfaction (ACS) on customer loyalty. Looking at the indices and impacts, users can determine which drivers of satisfaction, if improved, would have the strongest effect on customer loyalty. For more on ACSI, *see* Fornell *et al.*[12–15].

An example of such an integrated model was implemented by Sears, Roebuck and Company into what they called the *employee–customer-profit model*. The cause-and-effect chain links three strategic initiatives of Sears: (i) a compelling place to work, (ii) a compelling place to shop, and (iii) a compelling place to invest in. In order to promote these initiatives, Sears introduced an integrated structural equation model linking how employees felt about working at Sears, how their behavior affected customers' shopping experience, and how this experience affected profits. The model identifies the drivers to employee retention, customer retention, customer recommendation, and profits. Specifically, the model predicts that an increase in employee attitude by 5 units results in an increase in customer impression by 1.3 units, and this in turn adds 0.5% to the revenue[16]. For more on integrated management models, *see* Kenett[17] and Kenett and Lavi[18].

4 Bayesian Networks

Bayesian networks combine **Bayesian theory** with **graph theory** for inferring probabilistic relationships among variables. They are used to handle complex problems in which interactions of variables are too intricate for description by an analytic model. Bayesian networks represent the current knowledge by

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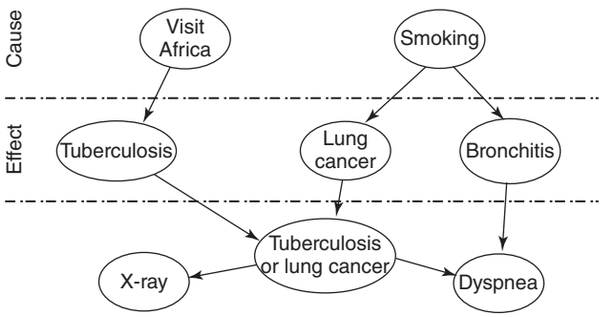

Figure 4. An example of causal network. *Source*: Adapted from Lauritzen and Spiegelhalter[27].

graphically linking associated variables. For example, consider a patient that visits a clinic with health-related problems. The physician examining her wants to identify the disease and prescribe the correct therapy. Figure 4 is a graph representing variables associated to tuberculosis and lung cancer. At the top are variables that describe the domain ("visit Africa," "smoking"). The domain knowledge experts have linked variables describing an effect (i.e., "tuberculosis") with possible causes (i.e., visiting Africa may cause tuberculosis).

Bayesian networks are directed acyclic graphs (DAGs) in which nodes represent **random variables**, and arcs represent direct probabilistic dependencies among them. The structure of the directed graph provides insights into the interactions among the variables and allows for prediction of effects of external manipulation. Prediction consists of conditioning a parent variable to determine its effect on variables depending on it on the graph, called its descendants. Diagnosis is obtained by conditioning on an effect, in order to ascertain the profile of the parent variables linked to that effect. Conditional dependence probabilities, estimated from data and/or elicitated from expert opinion, are used to generate a network representing learned associations. Bayesian networks represent the quantitative relationships among the modeled variables. Let $X_1, \ldots, X_n$ be a set of variables and $P(X_1, \ldots, X_n)$ be the joint probability defining the knowledge about the problem domain.

Numerically, a Bayesian network represents the joint probability distribution among the n variables. This distribution is described efficiently by exploring the probabilistic dependencies among the modeled variables, and the model size is reduced by **conditional independence**. Each node, X_i, is a random variable either with discrete or continuous states and is described by a probability distribution, $P(X_i|\ \text{parents}\ (X_i))$, conditional on its direct predecessors and quantifying parents' effects on the node. Nodes with no predecessors are described by prior probability distributions. Then, owing to the Markov property and conditional independence, the **joint distribution** is simplified through conditional distributions to

$$P(X_1, X_2, \ldots, X_n) = \prod_{i=1}^{n} P\ (X_i|\ \text{parents}\ (X_i)) \tag{1}$$

A set of oriented arcs links a pair of nodes and identifies probabilistic dependencies. For more on Bayesian networks, *see* Pearl[19, 20], Pearl and Mackenzie[21], Cowell[22], and Kenett[23].

5 Causal Networks

Causes, or interventions, have potential effects, different from what would have been otherwise observed under no intervention. To evaluate such effects, one needs to address potential outcomes. Consider people

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with headaches. If they take a pain relief tablet, we can observe its effect. We cannot observe, however, what would have happened, had they not taken the tablet. This corresponds to an unobserved potential outcome. Randomized control trials (RCTs) are considered the gold standard for assessing causality in interventions. A simple RCT has two arms, one with treated units, and the other with untreated control, placebo, units. When the allocation to arms is randomized, the difference in outcomes is attributed to the treatment. In practice, RCTs are affected by bias such as compliance to treatment, missing data, and nonignorable differences in treatment. Moreover, for ethical and technical reasons, RCTs are not always possible, and using observational data is necessary. In such studies, one collects covariate variables and outcome information. To study the effect of treatment with observational data (but not only), one needs to control for confounding covariates by regression or other statistical models. The difference between observed outcomes (treated patients) and potential outcomes (what would have happened had these patients not been treated) can be used to assess the effect of "counterfactual" conditions. Such counterfactuals are thought experiments, not physically carried out (such as applying treatment to an untreated group). By considering potential outcomes scenarios as missing data, Rosenbaum and Rubin[24] developed mathematical models for computing propensity scores (PSs). These PSs correspond to predicted probability of group membership, for example, treatment versus control group. PSs are based on observed predictors, usually obtained from logistic regression, to create a counterfactual group. Causal networks, or DAG causal graph models (Pearl[20, 21], offer a different approach based on graphical models. Causal networks require the researcher/analyst to state their causal assumptions explicitly, so that the study design and analysis that follows makes sense. In contrast to structural equation models, latent variables need not be explicitly listed, but the impact of unobserved variables can be accounted for. Causal networks, together with a causal calculus labeled "do" calculus, are used to predict the values of as-yet-unobserved variables from the values of observed ones. Since the arrows in a Bayesian network do not necessarily have a causal interpretation, Bayesian networks differ from causal networks. Specifically, the same joint distribution (X, Y) can be represented equally well by $X \to Y$ or $X \leftarrow Y$. However, if we accept that causes must be informative about their direct effects, then only nodes that are adjacent to a given node in a Bayesian network are candidates to be its direct causes or direct effects. Exogenously setting the values of one or more variables in a causal DAG model, to specified values, can change the probability distributions of the variables into which they point (their "children"), and hence the probability distributions of their more remote descendants. Exogenously specifying the values of some variables makes it unnecessary to infer their values, so that they can be disconnected from their predecessors in the DAG before their specified values are propagated through the rest of the model. As an example, consider the causal DAG model $X \to Y \to Z$ with CPTs $P(Y = y|X = x)$ representing the conditional probability that random variable Y has value y given that random variable X has value x; $P(z|y)$ for Z; and $P(x)$ for the marginal distribution of X. Then, setting X to a specific value x^*, an operation denoted by "do(x^*)," has the effect of changing the distribution of Y from its unconditional distribution $P(y) = \Sigma x P(y|x)P(x)$ to the new distribution $P(y|x^*)$. The interpretation is that setting X to x^* causes the value of Y to be drawn from distribution $P(y|x^*)$. Then Z, in turn, is drawn from the distribution $P(z|\text{do}(x^*)) = \Sigma y P(z|y)P(y|x^*)$ instead of $P(z) = \Sigma y P(z|y)P(y)$, and so the effect of setting X to x^* propagates through the distribution of Y to affect the distribution of Z. Likewise, the effect on Z of exogenously setting the value of Y to a specific value y^* is calculated by disconnecting X from Y in the DAG, and using the CPT for Z to determine $P(z|\text{do}(y^*)) = P(z|y^*)$. A causal or directed path from X to Z is a path that can be traversed along the direction of the arrows in the causal diagram.

In a chain junction, $X \to Y \to Z$, or a fork junction, $X \leftarrow Y \to Z$, controlling for Y prevents information about X from getting to Z. On the other hand, if X and Z are independent, in a collider chain, $X \to Y \leftarrow Z$, controlling for Y creates a dependence between them. To establish if we need to control for variables on a DAG, we apply the back-door path criterion[20]. A back-door path from X to Z is one that contains an arrow into X, and such that the other arrows in the path(s) from X to Z can be traversed in either direction along

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or against the direction of the arrows. In determining the causal effect of X on Z, one should disconnect all spurious (back-door) paths from X to Z. In each spurious path, one should condition on at least one variable that is not a collider (two arrows pointing to it) in each of the spurious paths. One should also leave all directed paths from cause to effect unperturbed and not condition on descendants of the cause. Pearl[20, 21] elaborates on this approach with numerous application examples.

An additional feature of causal networks is that they can be used to generalize findings from one study and make them relevant in another context. For more on this aspect, also called *transportability*, *see* Pearl[25]. Generalizability of findings is related to external validity of a study and is also the sixth dimension of the information quality (InfoQ) framework presented in Kenett and Shmueli[26].

Related Articles

Multivariate Directed Graphs; **Causality/Causation**; **Causal-Comparative Study**; **Causal Inference**; **Causal Direction, Determination**; **Causal Diagrams**; **Causality**; **Causation**; **Causal Inference**; **Causal Graph Models for Predictive and Prescriptive Analytics**; **Causal Prediction and Forecasting**; **Learning Causal Graph Models From Data**.

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Further Reading

Salini, S. and Kenett, R.S. (2009) Bayesian networks of customer satisfaction survey data. *J. Appl. Stat.*, **36** (11), 1177–1189.

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