BUILDING PROVEN CAUSAL MODEL BASES FOR STRATEGIC DECISION SUPPORT

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Abstract: Since many Decision Support Systems (DSS) in the area of causal strategy planning methods incorporate techniques to draw conclusions from an underlying model but fail to prove the implicitly assumed hypotheses within the latter, this paper focuses on the improvement of the model base quality. Therefore, this approach employs Artificial Neural Networks (ANNs) to infer the underlying causal functions from empirical time series. As a prerequisite for this, an automated proof of causality for nomothetic cause-and-effect hypotheses has to be developed.

1 INTRODUCTION

The main task of corporate strategy planning is the construction of a variety of decisions which are highly interrelated and characterized by a rather complex informational background. In order to reduce this complexity, the raw data originating from operational databases or Management Information Systems has to be arranged within decision models which becomes the principal issue of Decision Support Systems (DSS). Hence it can be observed that the architecture of any DSS necessarily incorporates the notion of a mental model underlying the respective decision theory as well as appropriate decision techniques.

A considerable number of recent approaches within the domain of strategic decision making propose to organize business indicators in the form of causal models. The main task of these models is to visualize the cause-and-effect hypotheses between given variables (Hillbrand and Karagiannis, 2002). Well-known examples for this type of strategic decision methodologies are the Balanced Scorecard technique (Kaplan and Norton, 2004), the tableau de board methodology (Mendoza et al., 2002), as well as cybernetic concepts like VESTER's Biocybernetic Approach (Vester, 1988) or the St. Gallen Management Model (Schwaninger, 2001).

Although these managerial approaches for strategic decision support provide some practical aspects for the reduction of complexity, the implementations of these ideas in the form of DSS are rather weak (Hillbrand and Karagiannis, 2002, p. 368): The model base, however, usually remains unproven with respect to the empirical evidence of the hypothetic cause-and-effect relations. Moreover these techniques are not able to provide quantitative forecasts for future impacts of an analyzed strategic scenario.

The discussion in this area restricts to the concept of correlation to prove causal relations. As this technique fails to sufficiently explain the phenomenon of causality, the relevant literture predominantly shares a rather dogmatic conception that it is not admissible at all to assess relations of this type (Hillbrand, 2003a, pp. 6ff.).

However, if we abandon the restriction to correlation as a concept for the proof of causality and a measure of association, it seems to be possible to infer further causal knowledge from empirical data and therefore improve the quality of the model base significantly. Hence this paper proposes an approach to automatically prove managerial cause-and-effect relations and to approximate the unknown causal function underlying these associations.

Based on a brief overview of causality concepts in section 2 this paper proposes a methodology to prove the causality of nomothetic cause-and-hypotheses in section 3 as well as to approximate the underlying functions (section 4) based on empirical evidence. The paper is concluded by the presentation of experimental results in section 5.

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2 CAUSALITY CONDITIONS

As it has been outlined in the introduction, the mere correlation of two variables seems to be insufficient for the causal validation of associations between them. Moreover, causality per se cannot be observed or tested by objective means. According to KANT it is a synthetic judgment a priori (Schnell et al., 1999, p. 56). Causality must therefore be regarded as an assumption about the connection between cause and effect made by the human mind and based on a variety of experiences rather than some kind of natural phenomenon which can be observed in an objective manner. Therefore some notions of causality incorporate the interventionistic idea which suggests the admissibility of experimental results as the only proof for causality. In an enterprise, however, there is hardly any situation where an experiment-like situation can be created because the trial-and-error manner of these settings usually would inflict losses for the business and the response time of basic cause-and-effect relations can be rather long.

Applied to managerial cause-and-effect relations, an appropriate concept of causality must restrict itself to observational studies in terms of empirical data as a consequence. Therefore a first necessary but not sufficient criterion (conditio sine qua non) for causality is that a cause provides information which can be used to (partly) explain its effects. In case of linear cause-and-effect relations this property of informational redundancy is also known as correlation or covariance. However, as it has been shown in the introduction, these concepts fail to fully explain causality. According to HUME, there has to be a temporal precedence relation between a cause and its effects additionally to informational redundancy. Regardless of the ability of these two necessary properties to fully explain many causal relations, there still remains the problem of an exogenous common cause to induce spurious associations between presumably causal variables. Therefore the definition of causality has to be enhanced by the postulate to control for this type of association.

Based on an extensive research of causality concepts within the relevant literature (Hillbrand, 2003a, pp. 152 – 170) the following definition of an appropriate concept of causality to analyze associations between managerial variables can be derived:

Theorem 1 (managerial causal relation): *A causal relation between variables of a managerial system exists if and only if there exist appropriate nomothetic (i.e. unproven) cause-and-effect hypotheses based on causal a priori knowledge where the following conditions are fulfilled:*

- *The empirical observations of a potential cause provide informational redundancy regarding its potential effect.*
- *The variation within the time series of the potential cause must always precede the response of this variation within the time series of the potential effect.*
- *The three causality properties as defined above (causal a priori knowledge, informational redundancy and temporal precedence) must not originate from the influence of a known or unknown cause, common to the potential cause and the potential effect.* □

As it is obvious from the above theorem, the underlying notion of causality follows the ideas of logical empirism which regards a hypothesis as true as long as it cannot be falsified. Therefore it is the task of a causality proof to rule out non-causal associations according to the above criteria from a given strategy model consisting of nomothetic cause-and-effect hypotheses. The next section develops an appropriate approach for the automated proof of causality.

3 PROOF OF CAUSALITY BETWEEN BUSINESS VARIABLES

The analysis and definition of a homogeneous notion of causality in the preceding section of this paper represents the conceptual basis for the construction of proven causal models for strategic decision support. A set of nomothetic cause-and-effect hypotheses contained in a rudimentary cause-and-effect model has to be given by strategic decision makers and represents the first necessary causal property of a priori knowledge.

As HILLBRAND proposes in his meta-model (Hillbrand, 2003b), the key modeling element of this model-type is an indicator which can be of crisp or fuzzy type. These indicators are linked by either undefined or defined influence relations, where the latter is described by axiomatically determined rules (e.g.: ROI, ROCE, etc.).

Consequently it is the focus of this section to provide appropriate methods in order to analyze the hypothetically established undefined influences within a strategic DSS with respect to their causal validity. This task of the proposed approach is to detect so-called α-errors of undefined nomothetic cause-and-effect hypotheses between variables. Therefore the starting point for the reconstruction of a proven causal model is a rather overdefined rudimentary model as described above.

In order to analyze the causality criterion of informational redundancy it seems to be suitable to restrict to the linear case as this proof per se does not build the model base but is used to select variables for

the following approximation of arbitrary causal functions. The admissibility of this theory for different types of causal functions has been shown by HILLBRAND (Hillbrand, 2003a, pp. 299ff.).

When considering the third necessary condition for causality of temporal precedence between cause and effect, the inadequacy of the concept of correlation alone to prove cause-and-effect relations becomes obvious: The correlation of two time series would show that the variations of an independent and a dependent variable are similar and that they take place contemporaneously. As this is mutually contradictory to the notion of causality as defined in the previous section, the concept of correlation has to be adopted to measure temporally lagged responses of the variation of an independent factor within the time series of the dependent variable. Therefore the cross-correlation $\rho_{X,Y}(\Delta t)$ implies a time lag Δt between a cause X and an effect Y in the following form:

$$\rho_{X,Y}(\Delta t) = \frac{\frac{\sum_{t=1}^{T}(y_t-\bar{Y})(x_{t-\Delta t}-\bar{X})}{T}}{\sigma_X \cdot \sigma_Y} \quad (1)$$

Where y_t and x_t stand for the values of the variables Y and X at time t, $\bar{X}$ and $\bar{Y}$ for the average values and σ_X as well as σ_Y for the standard deviation of the respective time series.

By calculating the cross correlations for varying time lags it is possible to identify a window of impact between an independent and a dependent variable (characterized by a minimum time lag and a number of subsequent effects). For this purpose it is necessary to determine the statistical significance of a cross correlation at a given time lag. Therefore this approach uses BARTLETT's significance test (Bartlett, 1955) following the suggestions of the appropriate literature in this area (Levich and Rizzo, 1997, p. 6): The null hypothesis that two given time series at a certain time lag are independent has to be rejected if the following constraint is satisfied:

$$|\rho_{X,Y}(\Delta t)| > \frac{1}{\sqrt{n-|\Delta t|}} \quad (2)$$

Where n stands for the number of samples in the time series of X and Y, respectively.

By increasing the time lag Δt by discrete steps beginning at a lag of zero time periods, it is possible to identify the minimum time lag by recording the first significant cross correlation between the time series.

This approach is illustrated in figure 1 for the following synthetically generated time series:

$$x_t = \varepsilon_{U(0;1)} \quad (3)$$

$$y_t = 0.2y_{t-1} + 0.5x_{t-2} + 0.2x_{t-3} + 0.1\varepsilon_{U(0;1)} \quad (4)$$

Where $\varepsilon_{U(0;1)}$ is a random variable uniformely distributed between zero and one.

As it is obvious from the above equations, the time series y_t of the independent variable Y incorporates past values of the time series x_t with a time lag of two and three time periods, respectively. Therefore the correct window of impact is $[2, 3]$.

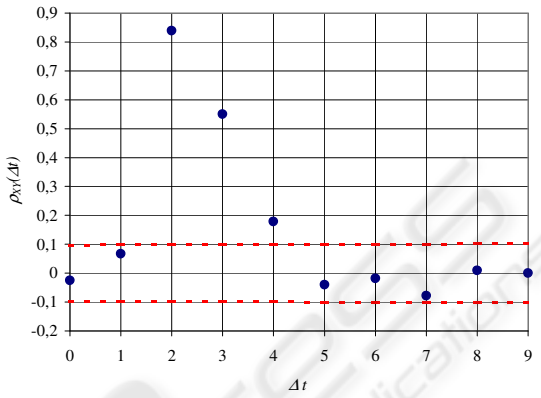

Figure 1: Correlogram between two timeseries

Figure 1 shows the cross correlations computed from the artificial time series x_t and y_t for the time lags $\Delta t = 0, \ldots, 9$, as well as the bandwidth of their standard deviation which lies between the dotted lines. The clear consequence which can be drawn from this correlogram is that the first significant correlation starting from zero occurs at a time lag of $\Delta t = 2$ which corresponds exactly to the generating function of y_t as stated above.

The first attempt to determine the appropriate length of the window of impact using the correlogram in figure 1 yields time lags between $\Delta t = 2$ and $\Delta t = 4$. Hence it can be shown that significant autocorrelation of the independent time series leads to the so-called echo effect (Hillbrand, 2003a, pp. 178f.) which describes the indirect effects of independent values prior to the window of impact through an autocorrelated dependent time series: In the example of figure 1 the term "$\ldots 0.2y_{t-1} \ldots$" leads to a reflection of x_{t-3} and x_{t-4} within the time series of y_t.

Therefore it is crucial for a correct identification of the impact window to eliminate any autocorrelation within the time series to be analyzed. Since a disquisition of these so-called prewhitening approaches exceeds the limits of this paper, it remains to refer to the appropriate literature (Hillbrand, 2003a, pp. 181ff.).

Potentially causal relations which show a significant window of temporally lagged impact are subject to a further analysis of common causation by third variables. For this purpose PEARL and VERMA propose an approach to identify spurious associations induced by a common cause (Pearl and Verma, 1991):

Theorem 2 (Controlling for third variables): *One can assume a relation $X \rightarrow Y$ to be causal if*

and only if the time series of the potential effect Y incorporates not only patterns of its direct potential cause X but also those of the predecessors P of X in the cause-and-effect model. If X and Y as well as P and X are informationally redundant but P and Y are not, an unknown third variable U rather than a causal relation must be assumed to induce informational redundancy between X and Y. □

As a consequence the patterns of P are reflected within the time series of X but they are not inherited on to Y due to the absence of a genuine cause-and-effect relation $X \rightarrow Y$.

A basic tool for the analysis of these assumptions within causal graphs is the concept of conditional independence: Two variables A and B are conditionally independent given a set of variables S_{AB} - written as $(A \perp B \mid S_{AB})$ - if A and B are informationally redundant. However, if the impacts of S_{AB} on B are eliminated, this property vanishes. Therefore S_{AB} is said to "block" the causal path between A and B.

The theory to detect third variable effects as outlined in this paper is implemented by the IC[1]-Algorithm. For reasons of lucidity, this paper dispenses with a detailed discussion of these procedures but refers to the appropriate literature (Pearl and Verma, 1991) or (Hillbrand, 2003a, p. 198).

Summarizing this approach, the modeling of nomothetic cause-and-effect hypotheses by decision makers represents a prerequisite for their proof as well as the first causality criterion. The second and third condition for causality - informational redundancy and temporal sequence - are tested by analyzing the cross correlations between the prewhitened time series of the respective variables connected by a cause-and-effect hypothesis. To rule out a third variable inducing informational redundancy between two lagged variables, this analysis is completed by the application of the IC-Algorithm as outlined above. Only relations which pass all these tests satisfy the necessary causality conditions and are therefore said to be genuinely causal.

4 APPROXIMATION OF UNKNOWN CAUSAL FUNCTIONS

The proof of causality as proposed in the previous section is the main prerequisite for the approximation of the unknown causal function affecting the values of any arbitrary business variable within a cause-and-effect structure. This provides the necessary numeric properties for the causal model base of a DSS to run numeric analyses (e.g.: simulation, how-to-achieve or what-if-analyses).

Many function approximation techniques either require a priori knowledge about basic functional dependencies (e.g.: regression analysis) or their approximation results are only valid within rather tight local boundaries (e.g.: FOURIER or TAYLOR series expansion). Since the form of these cause-and-effect-functions cannot be assessed a priori it is necessary to employ so-called universal function approximators for the purpose as described above (Tikk et al., 2001). Hence these techniques are able to learn any arbitrary function from mere empirical observations without the need to narrow down some base function. As it can be shown, microeconomic functions which usually underly strategic reasoning are almost never of linear type (Hillbrand, 2003a, pp. 201ff.). The reasons for this observation are manifold: Saturation as well as scale effects or resource limitations are only a few issues. One well-known example is the association between the market price and the customer demand for a certain product: Lowering prices will not linearly result in an increasing demand. Rather this association is expected to follow some S-shaped - also known as sigmoidal or logistic - pattern (Allen, 1964).

For these reasons it is essential to abandon all restrictions regarding a priori assumptions about the unknown function underlying a cause-and-effect relation. Therefore this approach studies the potential and limitations of Artificial Neural Networks (ANNs) for universal causal function approximation. The central theory in this area has been proposed by KOLMOGOROV who proved that any arbitrary unknown function f can be approximated by two nested known functions (Kolmogorov, 1957). Further enhancements of KOLMOGOROV's superposition theory have been developed by several authors which lead to the notion of ANNs as universal function approximators (Hillbrand, 2003a, pp. 210 – 215).

Since this universal approximation property has been proved for numerous of types of ANNs, this approach focuses on the construction of MLPs out of empirically proven cause-and-effect hypotheses. Therefore the causal strategy model has to be separated into causal function kernels (CFK). The latter describe a set of variables and interjacent cause-and-effect relations, each of which consists of a dependent element and its direct predecessors. Following the theory underlying this approach, the totality of all cause-and-effect relations within a causal function kernel represent the unknown causal function determining the values of the dependent variable.

Due to the possible existence of indirect associations between independent and dependent variables within a CFK - also known as multicollinearity - it is likely that the overall effect between two such elements has to be separated in order to obtain the direct

[1] IC = Inductive Causation

share of influence. Hence it is necessary to extend the causal function kernels for cause-and-effect relations which directly and/or indirectly link two independent variables $X_i \rightarrow X_j$. These auxiliary cause-and-effect relations accounting for multicollinearity can be discovered by analyzing the transitive closure of each independent variable within the global causal system: For every pair of independent variables X_i and X_j within a causal function kernel $\mathcal{K}_Y$ there exists an auxiliary cause-and-effect relation $X_i \rightsquigarrow X_j$ if and only if X_j is contained in the transitive closure of X_i according to the global model $\mathcal{G}$ as shown in figure 2.

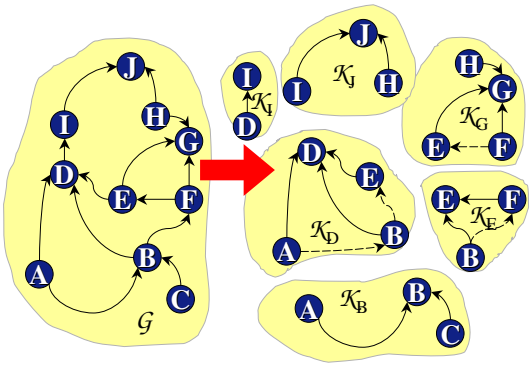

Figure 2: Separation of (extended) causal function kernels

Since an unknown causal function to be approximated does not exist between variables but rather between their lagged time series, the (e)CFKs have to be temporally disaggregated. While the dependent variable Y is represented by its instantaneous time series y_t as an output node, each independent variable leads to a number of input nodes corresponding to the length of the appropriate window of impact (see section 3). As far as eCFKs are concerned, it is necessary to introduce a second input layer which accounts for auxiliary cause-and-effect relations: The time series of the second layer are derived by the same procedure as described above taking the influenced indirect time series of the auxiliary association as output node and the influencing element as input node. Second input layer elements which affect first layer time series and the output node directly are of specific interest since they combine direct and indirect influence as figure 3 shows for the extended causal function kernel $\mathcal{K}_D$.

As temporal disaggregation delivers the appropriate input and output nodes for a neural function approximator in the form of temporally lagged time series there remains the issue to complete the model selection of the ANN. This includes an adequate dimensioning of the hidden layer(s) as well as the selection of input and transfer functions for all ANN-nodes.

Since the universal approximation property postulates a limitation of the inner function of KOLMOGOROV's theorem, it is necessary to use so-called squashing functions which encompass sigmoid as well as logistic, sine or heaviside functions (Hillbrand, 2003a, p. 214). For practical reasons the use of an additive input function for hidden and output neurons is recommended.

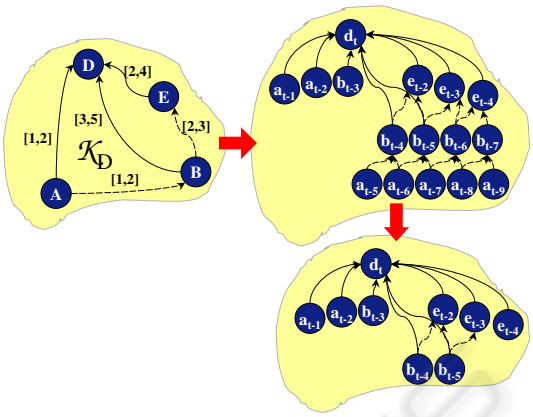

Figure 3: Temporal disaggregation of (e)CFKs

The selection of an appropriate number of hidden neurons is directly related to the generalization ability of the ANN (i.e. to learn a certain function instead of memorizing input-output mappings). As this specific model selection task depends on a variety of issues which cannot be analytically determined a priori, it is necessary to rely on heuristics (for details see (Hillbrand, 2003a, pp. 226 – 230)) and evaluate the prediction accuracy of the trained ANN by using a validation data set.

As it follows from the temporal disaggregation of eCFKs as discussed above, the resulting auxiliary cause-and-effect relations between second-level and first-level time series have to be incorporated into the neural function approximator in order to account for indirect effects. Therefore auxiliary sub-MLPs are introduced as symbolized by dotted connectors in the example of figure 4.

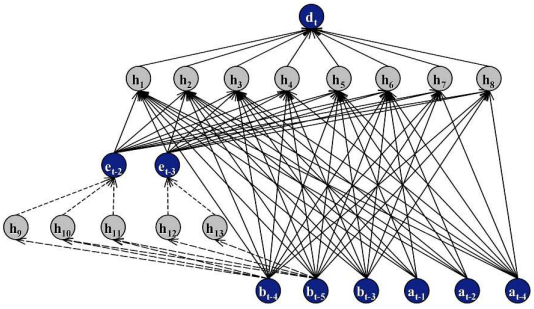

Figure 4: Neural function approximator for (e)CFKs

Before the training of the overall causal function

approximator it is necessary to learn these correcting functions, each of which has one first-level time series (e.g.: e_{t-2} and e_{t-3}) as output node and one or more second-level time series as input nodes. After the training of all correcting ANNs, their weights are kept fixed and included in the main neuron model. For the overall training of the causal function approximator it is necessary to equip first-level nodes with a specific input function since they are input and hidden nodes in the same way. Consequently the input function of a first level node calculates the weighted output sum of all preceding nodes plus the respective input value of the node itself. The ratio between these two shares of cumulative input is needed for training purposes when employing an error backpropagating algorithm: The same portion by which the overall input for a first-level node consists of values from a lower network layer is used to distribute the output error - backpropagated from higher network levels - among lower level neurons.

Since all further characteristics regarding layout and training of neural causal function approximators correspond with those of MLPs, they are not discussed in further detail.

Having determined the appropriate connection weights for these neural function approximators reconstructing a causal function , they can be used to explain the associations between business variables and goals as well as for the prediction of future values for dependent variables in a numeric way.

5 CONCLUSIONS

Experimental results with synthetically generated time series of causally dependent business variables have yielded the admissibility of the theoretic foundations for this approach (Hillbrand, 2003a, pp. 288 – 319): All cause-and-effect relations implicitly contained in the generating processes for five time series of an experimental case study could be recovered from a fully interlinked causal system (i.e.: Every variable is linked to all other elements) by analyzing the four causality criteria and the falsification of all spurious associations. Studying the relevance of anomalies for the results of this causality proof shows its robustness against nonlinearity, multicollinearity as well as autocorrelation within the causal function kernels. The exposure to highly noisy causal associations is the only issue which remains for future research in this context as this seems to affect the results of this causality validation approach negatively. The neural approximation of the causal functions underlying these proven cause-and-effect relations results in a significantly higher ex post prediction quality for the validation set than various regression techniques.

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