Causality in complex systems

Abstract
Purpose The concept we call "naive" causality can be stated more generally as the belief (or knowledge) that results follow actions, and that these results are not random, but are consistently linked with causes. We have thus formed a very general and precarious concept of causality, but one that appropriately reflects the meaning of causality at the level of common sense.
Design/methodology/approach Mathematical and logical development of the causality in complex systems.
Findings There are three aspects of rationality that give the human mind a unique vision of Reality: a) Quantification: reduction of phenomena to quantitative terms. b) Cause and effect: causal relationship, which allows predicting. c) The necessary and valid use of (deterministic) mechanical models. This work is dedicated to the second aspect, that of causality, but at present leaves aside the discussion of possibility-necessity, propose a modification to philosophical synthesis of causality specified by Bunge (1959), with contributions made by Patten et al. (1976) and LeShan and Margenau (1982).
Originality/ Value Causality is an epistemological category, because it concerns the experience and knowledge of the human subject, without being necessarily a property of reality.

Keywords: causality, complexity, causal chain, causal link, determination

1. INTRODUCTION: THE PRINCIPLE OF CAUSALITY

In Western culture an early treatment of the causal principle appears in the philosophy of Democritus: *By necessity are foreordained all things that were, are and will be*. Aristotle (1947) distinguishes four types of causes or explanations:

1) *Causa formalis* is the essence or essential nature of a thing, what defines it.

2) *Causa materialis*, is the material from which a thing is made, its properties.

3) Causa efficiens, is the outer compulsion, the cause of a change.

4) Causa finalis, explains how the causa efficiens works: the purpose or goal; for this reason it is the summing up of every generative or motivated process. It is the principle of finality.

The causal theory of Aristotle involves understanding the production of effects. This knowledge chooses the means, i.e.. it governs the occurrence of efficient causes directing the process to produce and end. All this is very important to us, since *it was precisely the superseding of previous knowledge that concealed details of causal mechanisms, which is clear in Aristotelian causality, which characterizes modernity and postmodernity. As for the precise nature of the causes, Aristotle's view was broad. He established three distinctions*

1) Things: the seed is the cause of the plant.

2) Events: an accident is the cause of death.

3) States: the current position and velocity of a body determines its future position and speed.

A common element in experimental science and in much of philosophy in modern times is the objectification of the causal link, one consequence of which "de-spiritualises" reality. Aristotle's analysis of causality separates knowledge and ethics from the world itself. So modernity can be understood in part as the beginning of a culture, which is characterized by *the separation of wisdom and reality, in which reality is considered as a pure object*. After the separation knowledge/reality, the separation of subject and object follows as an immediate consequence. The division of knower and known, is thought to be a real and primary division, rather than just a conventional philosophical position as it really is. Since then causality is understood as belonging to one of these two poles, the subject or the object. So, one possibility is that causality is objective as a scheme of actual effectuation, where the relationship between cause and effect is purely external, and so is blind and oblivious to the will and human knowledge (is the kind of causality that is modeled on Newtonian physics). Or alternatively, causality is reduced to a purely subjective character given only in the mind of the observer but with no connection with reality itself (the empiricist conception). Philosophers who consider causation as "real", conceived the relationship between cause and effect as a *productive relationship* (ontological effectuation) but face difficulties to account for that productivity. Therefore in fact, there is something in the causal link that one cannot observe or fully rationalize. The concomitance of cause and effect is observed, and in many cases the proof of the causal relationship is demonstrated, but the effectuation or production itself remains a conjecture of the researcher. *Causation as such is not evident, and does not show itself in its phenomenal externality.* The attribution of cause always involves the intervention of a mind that cuts the real process and interprets it. This difficulty seems irreducible to such an extent that the German philosopher N. Hartman, who wanted to found a materialist ontology, had to recognize the existing "irrational" component in causality. Hartman tried to circumvent that obstacle in the materialist theory of causation, stating that such irrationality is not a "real" character but is a methodological limitation that probably someday may be remedied (Väyrynen, 2016).

Consider any object. Among its many observable attributes in Euclidean three dimensional space, we have its mass, size, shape, color, position and speed. Suppose that the object is moving as the result of an impact. Important observables are: mass, position and current speed and the force acting on the object. The law for these observables is Newton's second law, which states that force equals mass multiplied by acceleration of the object. Mathematically, this law is a differential equation d^2x/dt^2, the solution always requires knowledge of two constants concerning the current movement. One is the current position that can be measured directly. The other is its current speed. But the current speed is determined by estimating the current position and determining its previous position, that is, by the operation of dividing the position change by the time interval. We could say that to determine the present speed, we need to take a little jump back in the past. If we are to find the current acceleration we should know the current speed and its speed a little earlier. But the latter requires knowledge of the position at a specific time, a little before that time which gave us the present speed. We will have to take two steps back in the past. The resolution of the equation gives the position and velocity of the object at any future time. This will be a causal prediction because it gives us the knowledge of the future position and speed of present or past. In general, a *causal prediction* involves a law, and a limited and specific set of observables, called *causal observables* (LeShan and Margenau, 1982). So with regard to the movement, only the mass, force, acceleration, velocity and position are significant. The other observable also comes from valid combinations in other processes that are not

moving. In Ecology biomass and population are observable. This allows us to develop models of differential equations over time, by a procedure similar to that discussed above for the moving object and thus make predictions.
What we want to establish here it is that the principle of causality allows us to predict the future in response to observables relative to the past. All theories that describe or reveal physical reality are guided by the principle of causality and, therefore, satisfy it. This is not intrinsically necessary and it always has to be that way. It was a question debated by philosophers like Kant and Hume. Kant (1950) considers this principle as something a priori necessary for thought, while Hume (1784) considered it as a useful instrument and incidental.
Direct and indirect causality in complex systems have been dealt with extensively by authors such as Higashi and Patten (1989), Patten et al. (1976), Higashi and Nakajima (1995). Lloret-Climent (2002) studied direct and indirect causality in living systems by studying cells and analysing relationships such as cellular meiosis and mutation. Recently, Fisher (2015), presented a discussion about the construction of mental models of causal relationships.

2. BUNGE PRINCIPLE OF CAUSALITY

Galileo (1623 [1953]) defined the efficient cause as a necessary and sufficient condition for the emergence of something: *that, and nothing else will be called cause, the presence of which is always followed by an effect and when it disappears, so does the effect.* Symbolically $(C \to E) \wedge (\neg C \to \neg E)$. Bunge (1959) criticizes this concept of Galileo's as:

1) *Inclusive*, admitting everything as cause, which could possibly influence the effect.

2) *General* given the set of conditions for the occurrence of an event of any kind caused by any kind of particular process.

To specify more precisely the causal connection $C \to E$, Bunge (1959) developed a logical series of progressively more precise formulations:

1) *Constant conjunction*: exceptions to the formulation of Galileo are established when C and E are recognized as sets. Then a constant conjunction of establishing the cause is "*If C happens, then E always happens."* Symbolically we can expressed this as $(\forall C) \wedge (\forall E), C \to E$. The cause as a set is necessary but not sufficient for the effect, which is also a set. This formulation of causality is *legal* (If C declares conditions), *asymmetrical* (C is essentially priority to E, however, not necessarily prior in time), and *constant* (no exceptions, as is set by the operator $\forall$). But it lacks singularity and productivity.

2) *Constant production*: The genetic link is outside the logical domain and must be entered factually: If C happens then E will always be produced by it. symbolically $(\forall C) \wedge (\forall E), C \to E.$

3) *Necessary conjunction:* uniqueness can be introduced in the constant conjunction to incorporate the need: *If C happens, then and only then E always*

happens. If is the logical symbol of the need, then $(\forall C) \wedge (\forall E), C \rightarrow$ E. The cause is now necessary, and only constant and sufficient for the effect.

4) *Necessary production*: The uniqueness and genetic principle combine to: If C happens, then and only then E is always produced by it. That is to say $(\forall C) \wedge (\forall E), C \leftrightarrow$ E.

This Principle of Causality determined by Bunge (1959) will be adopted in the theory presented in this paper, and we use it as an expression of the causal link $C \rightarrow E$.

3. CHARACTERISTICS OF CAUSALITY

Some of the issues we consider most important for causality are the following (Patten, Bosserman, Finn and Cale, 1976):

1) *Spatial contiguity*: Production of an effect by contact seems necessary for causality. However, spatial contiguity and causality are logically different categories, and when cause and effect are remote and cannot be empirically tested, causality remains as a hypothesis. For our purposes we will say that *cause is consistent with contiguity, but it is not necessary*.

2) *Temporal antecedence*: If C and E are spacially separated; they cannot occur simultaneously; moreover, they can. Therefore, generally temporal priority is not required on the effect, and causality is compatible with the instantaneous link $C \rightarrow E$. Antecedece and cause are logically independent but consistent categories. The temporal precedence of C is not necessary; however, it is a necessary existential priority. The cause must exist for the effect to occur. This follows from the nature of the conditional *"If C ...."* in each causal formula.

3) *Causality and succession*: The cause has been considered a reducible temporal succession, or equal to uniform. Comte (1830) observed that human thought does not seek to know the root causes of phenomena, but only their effective laws, that is, their invariable relations of succession and likeness. Mill (2002) noted that the main pillar of inductive science the Law of Causality, corresponded to the thought that invariance of succession based on the observation of any act of nature, and some other phenomena that preceded it. A causal principle based on temporal antecedence and succession has been formulated by Bunge (1959). The state of a closed system unfolds in time in a unique and continuous manner and the same initial state is always followed by the same final state. Many ecological models represented by homogeneous differential equations, including the Lotka-Volterra system $\frac{dx_i}{dt} = \left(a_i - \sum_{j=1}^{n} b_{ij} x_i \right) x_i \quad i = 1,2,...,n$ tacitly assume successional definition of the cause. The above statements are considered sufficient to know any system changing over time. The causes are necessary and sufficient to produce the effects and states are only necessary. States have a productive nature in themselves, but are also determined from internal and external processes. They are the agents of the cause, the cause carriers in effect but not the cause-effect itself.

4) *Externality of the cause*: Internal causes and centripetal concepts of causality were widely used in earlier times. Bruno (2014) distinguished between the causal principle

"that which intrinsically occurs in the constitution of the thing and remains in effect," cause or *"that which extrinsically occurs in the production of the thing."* Self causality, *causa sui*, now occupies a role more and more reduced in modern science, however, it appears again as an implicit assumption in homogeneous differential equations, which are used to model causal phenomena, such as ecological models. Generally, in modern science, intrinsic determinants have been relegated to other categories of determination, which are different from causality, or associated with formal and material causes in which case they can indicate some state relations. Innatism is not accepted by science, and vitalism considered as an inherent life force has been rejected for more than a century. Causal determination ensures that the universal operation of the efficient cause is taken from the extrinsic. Sustaining a cause to effect link must be maintained, and the disappearance of the cause is necessary and sufficient condition for the disappearance of the effect. $(\forall C)\wedge(\forall E),\neg C\leftrightarrow\neg E$. *Causa cesante cessat effectus* (Aquinas, 1947).

5) *Causation and functionalism*: The functional point of view of causality leads to the idea that the causal link can be replaced by mathematical relationships that express functional dependence. Problems may be presented as the functions are syntax, and therefore are legitimate; also functions have semantic and therefore genetic quality. Functional dependencies are not a priori causal, but may represent causality due to the fact that relations expressed are productive.

6) *Causal links of a system*: The causal link $C\rightarrow E$ is the only elementary unit of propagation of influence on a system, abstracted from the causal chains (serial sequences), and these in turn link to causal networks with convergence, branches and feedback.

7) *Multiple Causality*: If C and E are finite sets of causes and effects, then the necessary production of causality, logically $(\forall C)\wedge(\forall E), C\leftrightarrow E$ accompanies plurality of causes and effects diversity (Figure 1).

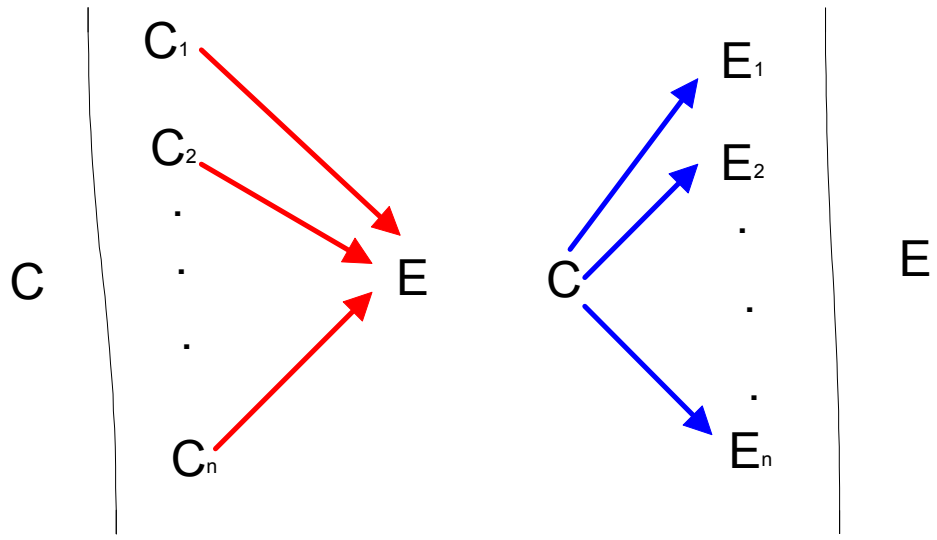

Figure 1: Multiple causality.

This is the reality of the causal link in a particular system. From the standpoint of Bunge (1959), multiple causality is not strictly causal in its uniqueness $(\forall C)\wedge(\forall E)$and applied to sets cannot be analyzed beyond the elements of the set. *Same cause, same effect* says the old dictum and not *same causes, same effects*.

4. CAUSAL LINK AND CAUSAL CHAIN

"Whatever it is, it has become, and is based on what has been." The state of the system is the link between its past and future existence.
In this section we will outline causal link theory following Lloret-Climent, Villacampa-Esteve and Usó-Domènech (1998); Lloret-Climent, Usó-Domènech, Patten and Vives-Maciá (2002); Patten, Bosserman, Finn and Cale (1976); Usó-Domènech, Mateu and Patten (2002); Usó-Domènech, Lloret-Climent, Vives-Maciá, Patten and Sastre-Vazquez (2002) and Usó-Doménech, Nescolarde-Selva and Lloret- Climent (2014, 2016).
Let H be a Holon, defined by m variables of the system $x_1, x_2, ..., x_n$.. These variables can be distributed in stimuli, state variables, and responses, based on their contribution to relations within the system (Figure 2).

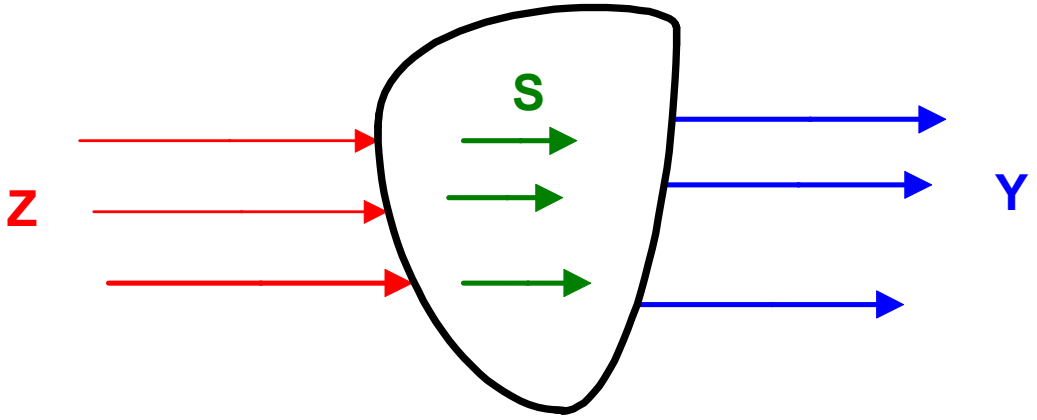

Figure 2: The Holon.

The cause is transmitted into the system through dependency paths, which are the causal structure.
Let $P_i(x_1, x_2, ..., x_n)$, $i = 1,2,...,n$ be a set of functions that expresses how each undifferentiated system variable is produced by others in accordance with the Genetic Principle.

Definition 1: *A system variable* x_i *is said to be in direct dependence with another system variable* x_j, *and denoted by* $x_j \rightarrow x_i$, *and we will read "*x_j *produces* x_i*" or "*x_i *is directly dependent of* x_j *", iff* $\frac{\partial P_i}{\partial x_j} \neq 0$.

Direct dependence is the mathematical expression of the philosophical Genetic Principle. The different categories of system variables can be differentiated according to the above Definition 1.

Definition 2: *A variable z of the system is a stimulus iff* $\left(\sum_{j=1}^{n}\left|\frac{\partial P_z}{\partial x_j}\right|=0\right)\wedge\left(\sum_{j=1}^{n}\left|\frac{\partial P_j}{\partial z}\right|\right)\neq 0.$

That is, *z* is a stimulus, if not directly dependent on any other system variable, and then there is at least one state variable that depends directly on it.

Definition 3: *A variable s is a state variable iff* $\left(\sum_{j=1}^{n}\left|\frac{\partial P_s}{\partial x_j}\right|\neq 0\right)\wedge\left(\sum_{j=1}^{n}\left|\frac{\partial P_j}{\partial s}\neq 0\right|\right).$

The state variable *s* depends on at least one system variable. And at least one variable of the system is directly dependent on the state variable.

Definition 4: *A variable y a response iff* $\left(\sum_{j=1}^{n}\left|\frac{\partial P_y}{\partial x_j}\right|\neq 0\right)\wedge\left(\sum_{j=1}^{n}\left|\frac{\partial P_j}{\partial y}=0\right|\right).$

That is, the response *y* is directly dependent on at least a system variable, and there is no system variable produced by *y*. In addition to *direct dependence*, system variables are also indirectly dependent on other variables, and the cause and effect, are propagated along sequences of serial dependence. Such sequences are known as *causal chains*.

Definition 5: *A system variable* x_i *is sequentially dependent on another system variable* x_j *iff there is a finite sequence of system variables between* x_i, x_j *and such that* $x_j \to x_{j+1} \to x_{j+2} \to \ldots \to x_i$.

Definition 6: *Each pair* $x_k \to x_{k+1},\ k = j,\ldots,i-1,$ *denotes a direct link of dependency.*

Definition 7: *Sequence* $x_j \to x_{j+1} \to x_{j+2} \to \ldots \to x_i$ *defines a sequential path of dependence from* x_j *to* x_i *symbolized by* $x_j \dashrightarrow x_i$.

Definition 8: *We define the length of a route as the number of links directly dependent on that route.*

Associations of dependence on a system variable x_i can be represented by the following groups:

1) *The set of direct dependence* D_i on the system variable x_i is the set of variables x_j for which there is a direct dependency link from x_j *to* x_i. $D_i = \{x_j ; x_j \to x_i\}\ \ i = 1,2,\ldots,n\ \ \ j = 1,2,\ldots,n$

2) *The set of sequential dependence* S_i of the system variable x_i is the set of variables x_j for which there is a sequential path of dependence from x_j *to* x_i. $S_i = \{x_j; x_j \dashrightarrow x_i\}$ $i = 1,2,...,n$ $j = 1,2,...,n$

THEOREM 1: *Given a set of undifferentiated variables* x_i $i = 1,2,...,n$, *of the system the following statements are true:*

1) *Each sequential path of dependence* $x_j \dashrightarrow x_i$ *contains an element of* D_i.

2) $D_i \subset S_i$.

Proof:

1) Each route of sequential dependence $x_j \dashrightarrow x_i$ has the form $x_j \to x_{j+1} \to ... \to x_{i-1} \to x_i, \forall x_j \in S_i$. The penultimate variable of the system x_{i-1} is obviously a member of the set of direct dependence on x_i.

2) Let $x_{i-1} \in D_i$ and $x_j \in S_i$ be. Each path of sequential dependence originating from the set of variables x_j ends in one and only one x_{i-1}. That is, in the sequence of dependence

$$x_j \to x_{j+1} \to x_{j+2} \to ... \to x_{i-1} \to x_i, \quad x_j, x_{j+1}, ..., x_{i-1} \in S_i,$$

but only $x_{i-1} \in D_i$. In general, then, $\forall x_{i-1} \in D_i, x_{i-1} \in S_i$; but $\forall x_j \in S_i, x_j \notin D_i$. Thus $D_i \subset S_i$.

Corollary 1 *The set of sequential dependence* S_z, *of the stimulus z is the empty set.*

Proof:

Consider $S_Z \neq \varnothing$. Theorem 1 ensures that each route of sequential dependency from z must contain an element of D_z. However, from the definition of stimulus z, we know that it has no dependence on any system variable. Thus $D_z = \varnothing$, which contradicts Theorem 1. Thus $S_z = \varnothing$.

Let $x_j \dashrightarrow x_i$ be a route of sequential dependency from x_j to x_i.

Definition 9: *We will call the variable* x_j *the origin variable, and call the variable* x_i *the terminal variable.*

Definition 10: *We define the route of complete dependence the one whose origin variable is a stimulus z and whose terminal variable is a response y.*

Definition 11: *We define a route of incomplete or partial dependence one whose origin variable and/or terminal variable is a state variable s.*

THEOREM 2: *Given a set of undifferentiated system variables* $x_i \quad i = 1,2,...,n,$ *the following statements are true:*

1) *Each stimulus z has caused at least one path of sequential dependence.*

2) *Each response y is terminal for at least one path of sequential dependence.*

3) *Each variable system, which exists between the origin and terminal of one path variables of sequential dependence, is a state variable s.*

Proof:

1) Each stimulus *z* has at least one system variable that is directly dependent on it. Therefore, there is at least one variable x_j such that $\frac{\partial P_j}{\partial z} \neq 0,$ and therefore x_j. It is directly dependent on *z* which causes at least one direct dependency link. Following Theorem 1 *z* is causing at least one path of sequential dependence.

2) Each response *y* is directly dependent on at least one other system variable. That is, at least there is a system variable x_j such that $\frac{\partial P_y}{\partial x_j} \neq 0,$ and then there is a dependency link $x_j \rightarrow y$ from x_j to *y*. Following Theorem 1 there is at least one path of sequential dependence $x_j \dashrightarrow y$ from x_j to *y*. Therefore, *y* is a terminal variable with at least one path of sequential dependence.

3) Each system variable x_j between the original variable and terminal variable of a sequential path dependence should be directly dependent on the preceding variable x_{j-1}. Similarly, the system variable x_{j+1} which is directly successor of x_j should be directly dependent on the latter. Therefore $\left(\frac{\partial P_j}{\partial x_{j-1}} \neq 0\right) \wedge \left(\frac{\partial P_{j+1}}{\partial x_j} \neq 0\right)$. x_j will be a state variable *s*. Therefore, any existing variable between the origin and terminal variables of a path of sequential dependence will be a state variable.

LEMMA 1: *The sequential dependency is transitive:* $x_j \dashrightarrow x_k$ *and* $x_k \dashrightarrow x_i \Rightarrow x_j \dashrightarrow x_i.$

Proof:

Subsequently $x_j \dashrightarrow x_k, \exists x_{j+1},...,x_{k+1}$ such that $x_j \to x_{j+1} \to ... \to x_{k-1} \to x_k$. Also, subsequently $x_k \dashrightarrow x_i, \exists x_{k+1},...,x_{i-1}$ such that $x_k \to x_{k+1} \to ... \to x_{i-1} \to x_i$. Therefore $x_j \to x_{j+1} \to ... \to x_k \to x_{k+1} \to ... \to x_{i-1} \to x_i$, $x_j \dashrightarrow x_i$.

THEOREM 3: *Given a set of undifferentiated system variables* $x_i \quad i = 1,2,...,n$, *the following statements are true:*

1) *Each state variable s is sequentially dependent of a stimulus z. That is to say* $\forall s, \exists z \in S_s$, S_s *being the set of causal dependence of s.*

2) *Each state variable s is an element of the set of causal dependence* S_y *on a response y.*

3) *Each state variable s is contained in at least one full path of sequential dependence. That is to say,* $\forall s, \exists z, y$ *such that* $z \dashrightarrow s \dashrightarrow y$.

4) *Each response y is sequentially dependent of a z stimulus. That is to say,* $\forall y, \exists z$ *such that* $z \dashrightarrow y$. *Each system response is terminal variable terminal with at least one full path of sequential dependence.*

5) *Each stimulus z of the system is within the set of sequential dependency* S_y *of a system response y. That is to say,* $\forall z, \exists y$ *such that* $z \in S_y$.. *Each system stimulus is causing at least one full path of sequential dependence.*

Proof:

1) As stated above, the main role of the state in the causal determination is to assign stimuli to the responses. A state variable s of a system will not be superfluous. Therefore $\forall s, \exists z$ such that $z \in S_s$. Each state variable has at least one stimulus in the set of sequential dependence.

2) For the same reason, if a state variable s not used to assign a *z* stimulus to some response *y*, it will be superfluous in a causally determined system. Therefore, $\forall s, \exists y$ such that $s \in S_y$. Each state variable is in the set of sequential dependence on at least one system response.

3) Let *s* be a state variable. By Theorem 3, s is sequentially dependent on at least one stimulus *z,* that is to say, $z \dashrightarrow s$. By Theorem 3, *s* is an element of the set of sequential dependence on any response *y*, that is to say $s \dashrightarrow y$. Therefore, by Lemma 1, there is a full path of sequential dependence containing *s*, originating from *z*, and ending at *y*, and such that $z \dashrightarrow s \dashrightarrow y$.

4) Each response *y* is directly dependent on at least one state variable *s*. Therefore, there is a state variable s such that $s \to y$. By Theorem 1, $z \dashrightarrow y$, and by Theorem 3, *s* is sequentially dependent on a *z* stimulus. Therefore,

$z - - - \rightarrow s$, and Lemma 1 states that the transitivity of sequential dependence gives $z - - - \rightarrow y$. The latter is a full path of sequential dependency, then y is the variable terminal at least one full sequential dependency.

5) Each stimulus z is an element of the set of direct dependence on a state variable s, or a response y. We consider z is an element of the set of direct dependence on y. Then there will be a full path of sequential dependence from z to y such that $z \rightarrow y$, which by Theorem 1 implies that $z - - - \rightarrow y$. On the other hand, suppose that z is an element of the set of direct dependence on the state variable s. Then $z \rightarrow s$, which, by Theorem 1 implies that $z - - - \rightarrow s$. By Theorem 3, s is an element of the set of sequential dependence on y, then $s - - - \rightarrow y$. Applying Lemma 1 $z - - - \rightarrow y$. This is a full path of sequential dependence, and therefore z causes at least one full sequential dependence

Consequence 1: The Theorem 3 states that:

1) There are no superfluous state variables in a system.

2) It ensures that each state variable is clearly related to some stimulus, illustrating the role of stimulus in determining the state space of the system.

3) It provides that each state variable is related to a response, and this result corresponds in the context of sequential dependence to the notion that states take stimuli and convert them into sequences.

4) Each state variable becomes relevant in the propagation of the cause.

Definition 12: *Two undifferentiated system variables* x_i, x_j *are said to be mutually independent iff* $(x_i \notin S_j) \wedge (x_j \notin S_i)$.

Definition 13: *Two undifferentiated system variables* x_i, x_j *are said to be mutually dependent iff* $(x_i \in S_j) \wedge (x_j \in S_i)$.

THEOREM 4: *Given a set of undifferentiated system variables* $x_i \;\; i = 1,2,...,n$, *the following statements are true:*

1) Each set of mutually dependent variables of a system, contains as elements only state variables.

2) If two variables of a system are mutually dependent, then their dependence sequential sets are identical.

Proof:

1) The mutual dependence of two undifferentiated variables x_i, x_j means that $(x_i \in S_j) \wedge (x_j \in S_i)$. Then, $(x_i \dashrightarrow x_j) \wedge (x_j \dashrightarrow x_i)$. Applying Lemma 1 entails the existence of a path of sequential dependence $x_i \dashrightarrow x_j \dashrightarrow x_i$ in which the same variable is both the origin and terminal. By Theorem 2, the variable x_j within the sequence must be a state variable. For the same reason, $(x_i \dashrightarrow x_j) \wedge (x_j \dashrightarrow x_i) \Rightarrow (x_j \dashrightarrow x_i \dashrightarrow x_j)$, and x_i is therefore a state variable as a member of a sequence of dependency that originates and terminates at x_j. In addition, neither a stimulus nor a response can be mutually dependent.

2) Suppose two variables of the system x_i, x_j, are mutually dependent, and such that $(x_i \in S_j) \wedge (x_j \in S_i)$. Then, $x_i \dashrightarrow x_j \dashrightarrow x_i$. Let x_k be a system variable such that $x_k \in S_i$, That is to say $x_k \dashrightarrow x_i$. When $(x_k \dashrightarrow x_i) \wedge (x_i \dashrightarrow x_j)$, and as a consequence of Lemma 1 then $x_k \dashrightarrow x_j$. Therefore, $x_k \in S_j, S_i \subset S_j$. By the same reasoning an element of S_j is also an element of S_i such that $S_j \subset S_i$. Therefore $S_i = S_j$, and sets of sequential dependence of mutually dependent variables of the system will be identical.

5. MATRIX OF CAUSAL DEPENDENCY

Definition 14: *We define a direct dependency matrix as a binary matrix, a nxn dimensional square with the i-th row and i-th column corresponding to the system variables,* $x_i, i = 1,...,n.$

The elements of this matrix are binary, with 0 in the *ij-th* position if $\frac{\partial P_i}{\partial x_j} = 0,$ and 1 if $\frac{\partial P_i}{\partial x_j} \neq 0.$ Each non-zero element indicates the presence of a direct link of dependence between $x_j, x_i, x_j \rightarrow x_i.$ Such a matrix is called an incidence matrix in Graph Theory (Bondy and Murty, 2008; Busaker and Saaty, 1965)[1].

Definition 15: *We define an adjacency matrix as the result of replacing each non-zero element in the matrix of direct dependence with the number of directly dependent links between corresponding system variables.*

[1] An incidence matrix is a matrix that shows the relationship between two classes of objects. If the first class is *X* and the second is *Y*, the matrix has one row for each element of *X* and one column for each element of *Y*. The entry in row *x* and column *y* is 1 if *x* and *y* are related (called incident in this context) and 0 if they are not. It can generate a family of matrices from the matrix by direct dependence replacing various amounts by binary 1.

The adjacency matrix has meaning in other contexts where the number of links of dependency has a relevant consideration[2].

Definition 16: *We define as a logical matrix, binary matrix, relation matrix or Boolean matrix any adjacency matrix where each direct link of dependence is indicated by the Boolean 1, in the appropriate position.*

Definition 17: *A Jacobian matrix results when a binary 1 of the matrix of direct dependence, is replaced by non-zero values of the partial derivatives* $\frac{\partial P_i}{\partial x_j}$.

The multiplication of matrices of direct dependence can be used to build routes of sequential dependence through interactive networks of complex systems Σ.
Let $A=(a_{ij})$ be a *nxn* adjacency matrix whose elements denote the position and number of links of direct dependence from the system variable x_j to the variable x_i. Elements of A^2 represent the number of 2-links, in routes of sequential dependence from the system variable x_j to the variable x_i. In general, A^l indicates the number of routes of sequential dependence of length l, where l links direct dependence from x_j to x_i. The following property is well known for Linear Algebra and the Theory of Networks:

Property 1: *Let A be one adjacency matrix, which describes the position and the number of directly dependent links between system variables, then the* $\sum_{k=1}^{l} A^k$ *series converges to the matrix* $A^{(l)}$ *whose elements describe the position and the number of all routes of sequential dependency, of length* $\leq l$ *between system variables* $x_j, x_i, i=1,2,...,n.$

Only if the adjacency matrix is Boolean, are the positions B* of routes of sequential dependency indicated. Let $B=(b_{ij})$ be a Boolean adjacency matrix *nxn*, whose zero and non-zero elements and are subject to Boolean operations. Then, B^2 describes positions of 2-links routes of sequential dependence and B^l describe the positions of l-link routes, and $B^{(l)}=\sum_{k=1}^{l} B^k$ describes the positions of the sequences of dependence of all lengths $\leq l$ (Ponstein, 1966).

Definition 18: *The infinite series* $\lim_{l\to\infty}\sum_{k=1}^{l} B^k = B^*$ *represents the positions of all possible routes of sequential dependence of any length and B* It will be referred to as transitive closure matrix (Lidl and Pilz., 1998*; *Ore, 1962).*

[2] In graph theory and computer science, an adjacency matrix is a square matrix used to represent a finite graph. The elements of the matrix indicate whether pairs of vertices are adjacent or not in the graph.

THEOREM 5: *The infinite series* $\lim_{l\to\infty}\sum_{k=1}^{l} B^k = B*$ *converges to the transitive closure matrix B * when it has n + 1 terms, where n is the order or number of state variables of the system. That is to say* $B^* = \sum_{k=1}^{n+1} B^k$, *and with* $b_{ij}^* \in B^*$, *(1)* $b_{ij}^* = 1 \Rightarrow s_j \dashrightarrow s_i$, *considering that (2)* $b_{ij}^* = 0 \Rightarrow s_j \dashrightarrow\!\!\!\!\!\!\downarrow\ s_i$, $i = 1,2,...,n.$

Proof:

Consider the path of sequential dependency $s_j \dashrightarrow s_i$ from the state variable s_j to the state variable s_i. By Theorem 2 we know that the variables that propagate the cause between s_j and s_i are state variables. The shortest route $min\left[s_j \dashrightarrow s_i\right]$ between s_j and s_i contain certain intermediate state variables, on the other hand if a loop or closed path $\left(s_j \dashrightarrow s_k \dashrightarrow s_i\right)$ is indicated, the path will not be shorter. Considering all possible routes of the state variables in the system, and the longest possible and shortest possible route between them, that is to say $max\left\{min\left[s_j \dashrightarrow s_i\right]\right\}, \forall s_j, \forall s_i,$ may contain, therefore, at most *n* state variables, where n is the order of the system. The length of this route would be *n-1* links of direct dependence. By Theorem 2, each stimulus z of system has a unique link of direct dependence, with the closest state variable, that is to say s_j. Therefore, the longest possible and the shortest possible route between the stimulus of origin, is the farthest terminal state s_i, that is to say, $max\left\{min\left[z \dashrightarrow s_i\right]\right\}, \forall z, \forall s_i,$ and it shall consist of no more than *n* links of direct dependency. For the same reason, by Theorem 2, each response of the system y has a single link of direct dependence from the nearest state variable s_i. The longest possible and the shortest possible route between the farthest stimulus and response, that is to say $\max\{\min[z \dashrightarrow y]\}, \forall z, \forall y,$ shall be, at most of *n + 1* links of direct dependence.

Therefore, the truncated series $\sum_{k=1}^{n+1} B^k$ is sufficient to identify the positions of all routes of sequential dependency of all possible lengths in the system, and therefore $\lim_{l\to\infty}\sum_{k=1}^{l} B^k = \sum_{k=1}^{n+1} B^k$.

The b_{ij}^* elements of the above matrix, that is to say B*, have a value of 1 when a path of sequential dependence of any length exists between s_j and s_i, and 0 otherwise.

COROLLARY 2: *The set of sequential dependence* S_i *of a system variable* x_i *consists of variables in row i of the transitive closure matrix B* whose elements are nonzero.*

Proof:

It follows from the definitions of S_i and B*.

COROLLARY 3: *The system variables x_i, which are sequentially dependent of s_j, that is to say, those whose set of sequential dependence S_i contains x_j, consists of the variables in column j of B* whose elements are non-zero.*

Proof:

This follows directly from the definition of B*.

6. CONCLUSION AND REFLECTIONS

Causality is a category, a principle and a doctrine. Today the conception of causality that seems to dominate as a mathematical function only expresses the correlation between variables, and says nothing about the "effective action" of the causes in reality. The *category causality* refers to the *causal link* cause $\rightarrow$ effect $C \rightarrow E$. The principle basically says the *same cause, same effect*. Causality is an epistemological category because it concerns the experience and knowledge of the human subject, without being necessarily a property of reality.
Looking at its ontological status, Bunge (1959) argued that the cause is not only a category of relationship between ideas, but a category of connection and determination corresponding to a current feature of the factual world (external and internal). Therefore, the causal link may take the appearance of an ontological category. The question we ask is this, why are there precisely the four causes described by Aristotle? This question follows the analysis of our representation of causes and raises the search for the origin of the idea of a *first cause*, and leads to the contradiction of the idea of original causes. Have not we continually expected that there is always one cause superior to the other cause, and that is enshrined as the links of a causal chain? Or do we have to wait for a final link, one ultimate cause on which all others depend?
Three aspects of rationality give to the human mind a unique vision of Reality:

1) *Quantification*: reduction of phenomena to quantitative terms.

2) *Cause and effect*: causal relationship that allows prediction.

3) *The necessary and valid use of deterministic mathematical models.*

We have dedicated this work to the second aspect, causality, but we will not enter into the possibility-necessity debate, which we will leave for later.
We have dedicated this work to making, with modifications, a philosophical and mathematical synthesis of causality according to Bunge (1959), with the contributions made by Patten et al. (1976) and LeShan and Margenau (1982). We have laid the theoretical basis of deterministic mathematical models, so necessary for the understanding and prediction of complex dynamic systems such as ecological, social and economic.

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