# Cause-and-Effect Interactions in Natural Sciences

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**Abstract**

This article aspires to offer more evidence for the existence, role and importance of cause-and-effect interactions in causal relations which govern some of the most important conceptions in the empirical comprehension of reality. The study takes up the idea of general causality in order to reveal more clearly the finite cause-and-effect interaction concept of progressing changes in nature. The changes in causal relations are mathematically formulated in the article as accumulations of effects induced by their interactions with causes. The interaction relates the elapsed effect to the available capacity of the forthcoming cause. The trans-temporal interaction concept links the past and future time separated by irreversible instant of reality observable only at the present moment. The analytical interaction model is applied to investigations of aging, vital capacity and fatigue. The study suggests that observable parameters for propensity and intensity of the interaction model describe in an appropriate way some natural phenomena.

**Keywords:** causality, interaction, aging, growth hormone decline, basal metabolic rate, forced vital capacity, fatigue

## 1. Introduction

A belief that the ways in which changes evolve in the reality must be understood at the moment of observation is not always sufficient for explanation of multifarious causal relations in which a cause induces an effect and the effect, in turn, may affect the cause. Changes have a connotation of uncertainty since their progressions beyond the instance of observation cannot be rationally predicted with certainty only by experience. The emerging causes and effects are characterized by subjective or objective instants of observation in which the changes are perceivable. The first perception of an ongoing causal relation may provide the primary information on the propensity for its continuation. The next and subsequent observations may indicate possible interactions between the cause and effect. In contrast to the theories on probabilistic causation, where the causes change the probabilities of their effects or in contrast to dualistic causal interactionism's doubts related to the possibility of interaction between material and immaterial, the article discusses the ways how the effects in the reality change their causes in an interactive trans-temporal manner. This study seeks a clue for the interaction phenomena in postulated cause-and-effect relations based on David Hume's causality concept and on Newtonian causation in natural sciences.

## 2. Cause and effect interaction

Hume's reading on causation in terms of invariable patterns of succession leads to regularity theories which imply that the cause $C$ and the effect $E$ are connected but different entities. The connection between a cause and effect is important in this concept but it doesn't imply interactions. That $C$ affects $E$ is the singular causal claim $C \Rightarrow E$ where $C$ and $E$ are relata of the claim. The knowledge of causal relations arises entirely from experience. The conclusions made from experience imply a belief that some observable courses of nature could be sufficiently continuous and regular so that the future would be conformable to the past. There is no assertion about directionality of causation, except for the common experience that an effect does not influence in reverse the cause $C \not\Leftarrow E$ (asymmetry). It is commonly agreed that the flow of causality proceeds from the present into the future, rather than into the past. In Newtonian causal determinism the world-at-a-time has an objective notion. Particular causes $C$ and effects $E(C)$ of $C$ are considered as empirical laws of nature. The claim, according to which every later effect is uniquely determined by its earlier cause, doesn't regard interactions of an effect $E$ and its cause $C$. The motivating belief of this study is that the concept of cause-and-effect interaction might be rooted in natural phenomena (Ziha, 2012). The study proceeds with an observable effect $E$ evolving uniformly under cause $C$ at a constant rate $\mathrm{d}E/\mathrm{d}C=E(C)/C=p$.

The linear accumulation of the effect is analytically presentable by the integral up to the ongoing cause $C$ (Fig. 1):

$$E(C) = \int_0^C pdC = pC \tag{1}$$

The cause $C$ in (1) stands for an efficient cause of the effect $E$. The linear CEI concept takes for granted that the primary effect is causally related to the cause that is presented by linear rule (1). The study is guided forward by an intuition that emerging Cause-and-Effect Relation (CER) as in (1) could in the reality ensue as a continuous Cause-and-Effect Interaction (CEI) of inseparable couple of finite interrelating cause and effect $C \Leftrightarrow E$, where $\Leftrightarrow$ does not stand for symmetry but for interaction. The CER represents the transfer of causal capacity $V$ between the cause $C$ and effect $E$. Over the time barrier defined by the moment of observation Individual CER is considered as an unexceptionally finite continuous and uniform process following some perceptible regularity, limited by ultimate causal capacity $V_U$. The CEI starts as CER at constant rate $E'_p = \mathrm{d}E_p(C)/\mathrm{d}C = p$ as in (1) that stands for the propensity to interaction. Simultaneously the remaining causal capacity ($V_u$-$V$) left behind the action of the cause $C$ is the reason for perpetuation of the causal relation beyond the instant of observation. For analytical simplicity the cause $C=V/V_U$ is applied in normalized form between zero and one. From the initial assumption of linearity between the primal cause and effect (1), it follows that the forthcoming unobservable cause $C_F = f\,(1\text{-}C)$ is also causally related to the remaining causal capacity at rate $f$. The variable interaction rate $E'_i = \mathrm{d}E_i(C)/\mathrm{d}C$ brings into relation the effect $E_p(C)=pC$ as it could be without interaction and the forthcoming residual cause $C_F = f\,(1\text{-}C)$ (Fig. 1) as shown:

$$I' = \frac{\mathrm{d}I}{\mathrm{d}C} = E'_p + E'_i = p + \frac{E_p = pC}{C_F = f(1-C)} = p + i\frac{V}{V_U - V} = p + i\frac{C}{1-C} \tag{2}$$

The integration of (2) until the cause $C$ yields to the CEI expression for the changes of the simple CER due to interaction between the elapsed effect $E$ and the forthcoming cause (1-$C$) at any instant in time, as it follows:

$$I = E_p + E_i = pC + \int_0^C i\frac{C}{1-C}dC = V_U\left\{pC - i\left[C + \ln(1-C)\right]\right\} \tag{3}$$

The constant $i=p/f$ is the interaction intensity parameter that defines the rate of change of the rate in (2) as:

$$I'' = \mathrm{d}^2 I / \mathrm{d}C^2 = -i\cdot(1-C)^{-2} \tag{4}$$

The CEI wastes the causal capacity $W(C)$ on the expense of the overall causal capacity in an amount that can be quantified by integration of (3) (Fig. 1), as it is shown below:

$$W = \int_0^C IdC = V_U^2\left\{p\cdot C^2/2 + i\cdot\left[-C^2/2 + C + (1-C)\cdot\ln(1-C)\right]\right\}, \tag{5}$$

The nonlinear part $E'_p$ of the CEI interaction rate (2) is at hand to the amplification or the gain factor in feedback theory of Wiener (1948). The gain factor represents the closed loop transfer function $A_f = x/(1+\beta x)$ where the feedback factor (FBF) is less than zero $\beta<0$. The separation of the cause $C$ from interaction term $I$ in (3) is possible only in the special case for $p=i$ when the inverse of (4) corresponds to the von Bertalanffy (1935) growth function (VBGF) in the form $C = C_U(1-e^{-I/i})$ where $C_U$ represents the ultimate cause. Thus, the interaction term (3) generalizes the von Bertalanffy exponential growth model as a combination of exponential and linear growth.

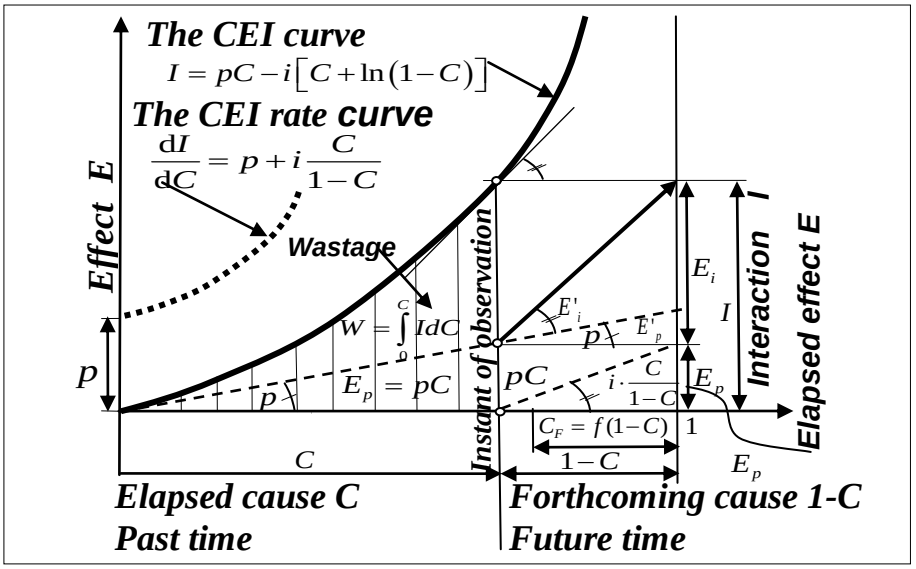


Figure 1. Cause-and-Effect Interaction (CEI)

The propensity rate $p$ characterizes the emergence of a CER. Its value is attainable at the very beginning of the CER process as the first derivative of the CEI function that is the slope of the curve at $C$=0 (Fig. 1).

Perpetuation of CER as a CEI occurs for interaction intensity rate $i \neq 0$, Fig. 2. The primitive meaning of the CEI intensity parameter $i$ is the negative second derivative (4) of the interaction term (3) for $C \to 0$. The interaction intensity parameter $i$ can be obtained from the observed waste $W$ (5) in the whole range of the elapsed cause 0-$C$ as:

$$i = \frac{W - p \cdot C^2 / 2}{-C^2 / 2 + C + (1-C) \cdot \ln(1-C)} \tag{6}$$

For $C \to 0$ in (6) follows the term of the interaction intensity parameter $i = 2W_U - p$.

The reversal of effect $E$ might occur at the stationary point of the interaction rate (2) for $C$ (Fig. 2) as it follows:

$$C = \frac{p}{p-i} \tag{7}$$

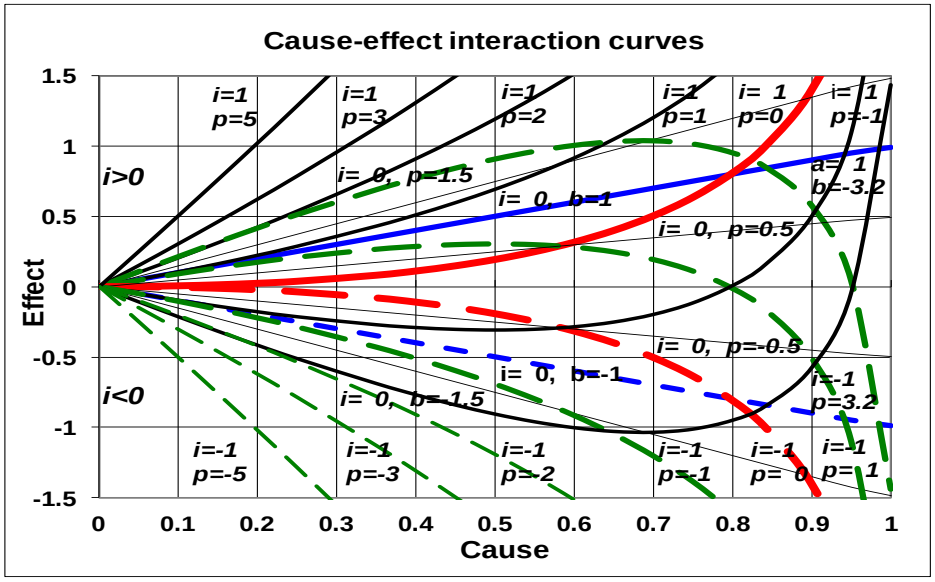


Figure 2. Effect of propensity $p$ to and intensity $i$ of interaction on CEI curves

**3. Aging-and-lifetime interaction**

Some body functions decline predictably with age. Evident aging (A) starts at same aging initiation time $L_o$ in the lifetime and continues throughout the rest of the life $L$. Aging is inseparably linked to irreplaceable losses of finite vital resources or capacities $V(L)$ during the aging period $L_o+L$. The study hypothesizes that the lifetime dependence of aging $L[A(V)]$ is influenced by the life shortening ($L_S$) that in turn interactively affects the aging $A[V(L)]$.

Aging-and-Lifetime Relation (ALR) is considered as unexceptionally finite process limited by given and available vital resources or capacities in lifetime. The finite ALR process results in life shortening $L_S$ until the available vital resources $V_A=V_o-V_U$ do not tire out from an aging initiation condition $V_o$ to ultimate condition $V_U$. The study defines aging $A(V)$ as a normalized cause $C$ between 0 and 1 in the interaction model (1-6). The relative decline of the vital resources or capacities $V(A)$ with respect to their anticipated ultimate capacities $V_U$ is then as shown:

$$A(V) = 1 - \frac{V(A) - V_U}{V_A} = \frac{V_o - V(A)}{V_A} \tag{8}$$

The linkage between the elapsed and forthcoming life perpetuates due to continuous trans-temporal sharing of the vital resource $V$ between the past aging $A(V)$ and remaining resource $V_U$-$V$ for the forthcoming aging period 1-$A$. The ultimate aging $A_U$=1 represents the threshold of resources $V=V_U$ below which the life is not really sustainable. Open-ended Aging-and-Lifetime (AL) phenomena make individual aging processes uncertain and the life beyond the present instant remains unpredictable due to unattainable information about forthcoming inescapable end, Fig. 1.

Mathematically the Aging-and-Lifetime Interaction (ALI) term $L_R/(1-A)$ represents the ratio between an ideal linear life $L_R=rA$ as it should be without interaction and the forthcoming aging 1-$A$ as in (2). The combined rate of the overall life shortening $L_S$ expresses the specific time loss due to decline in necessary vital resource availability. In the complete form the life shortening rate due to aging $A$ uses the CEI rate definition (2) when it reads:

$$\frac{dL_S}{dA} = p + i \cdot \frac{A}{1-A} \tag{9}$$

The life shortening $L_S$ is the consequence of the decline of the vital resource on the expense of the overall lifetime $L$. The overall life shortening $L_S$ at age $A$ due to the losses of vital resources is patterned after the CEI term in (3) as:

$$L_S = V_A \cdot \left[ (p-i) \cdot A - i \cdot \ln(1-A) \right] \tag{10}$$

At this point, the ALI is formulated as a CEI phenomenon since it fulfils the analytic conditions (1-6).

Even though that the ALI concept helps to estimate the life shortening (10) due to aging, the lifetime $L$ itself is not directly attainable solely from the observable deficiency of the vital resources. The lifetime $L(A)$ affected by ALI can be expressed as a result of the life shortening $L_S(A)$ (10) with respect to ALR primitive reference lifetime $L_R(A)=rA$:

$$L(A) = L_R(A) - L_S(A) \tag{11}$$

The relative efficiency $e(L)$ with respect to the full capacity $C_T(L) = V_U \cdot L'_S(A)$ of utilization of vital resources in the lifetime $L$ can be obtained by integration of the aging curve as $e(L) = 1 - d(L)$ (Fig. 2) where $d(L) = A - L_S / C_T$ is the relative deficiency of vital resources.

*3.1 Growth hormone decline by aging*

The Growth Hormone is a vital resource (V=GH) which is associated with symptoms of aging. The non-linear *NIH* diagram of GH Decline (GHD) (Fig. 3) stands for the amount of growth hormone secretion in the range $GH_o$=2 to $GH_E$=0 in Micrograms starting at age of $L_o$=11 years when the initiation of aging is observable (Journal of NIH Research, 1995). The threshold is the ultimate value of $GH_U$=0.16 below which the life is hardly sustainable.

*3.2 Basal metabolic rate decline by aging*

There seems to be a well-established relation of aging and Basal Metabolism (V=BM) that represents the body energy production for male and female (Mitchel, 1962). The diagram (Fig. 3) presents the rate of the vital capacity BM (BMR) in the range from $V_o$=$BMR_o$=2.1 to $V_E$=$BMR_E$=0.7 in Kcal/Kg hr starting at age $L_o$. The threshold is the ultimate $BMR_U$=0.784 below which the life is hardly sustainable. The numerical analysis using (8-11) provides interaction parameters *p* for propensity and *i* for intensity by numerical integrations of reported data using (10) and (11) (Fig. 3). The results suggest that the lifetime shortening due to GHD and the BMR follow the ALI pattern (8-11) based on the CEI concept (1-6). The reference lifetime) is estimated at $L_R(A)=100\cdot A$. The life shortening rates due to GHD and BMR decline in the normalized space 0-1 similarly. The GHD induces more life shortening with respect to the reference lifetime and lower efficiency due to higher propensity than the BMR (Fig. 3).

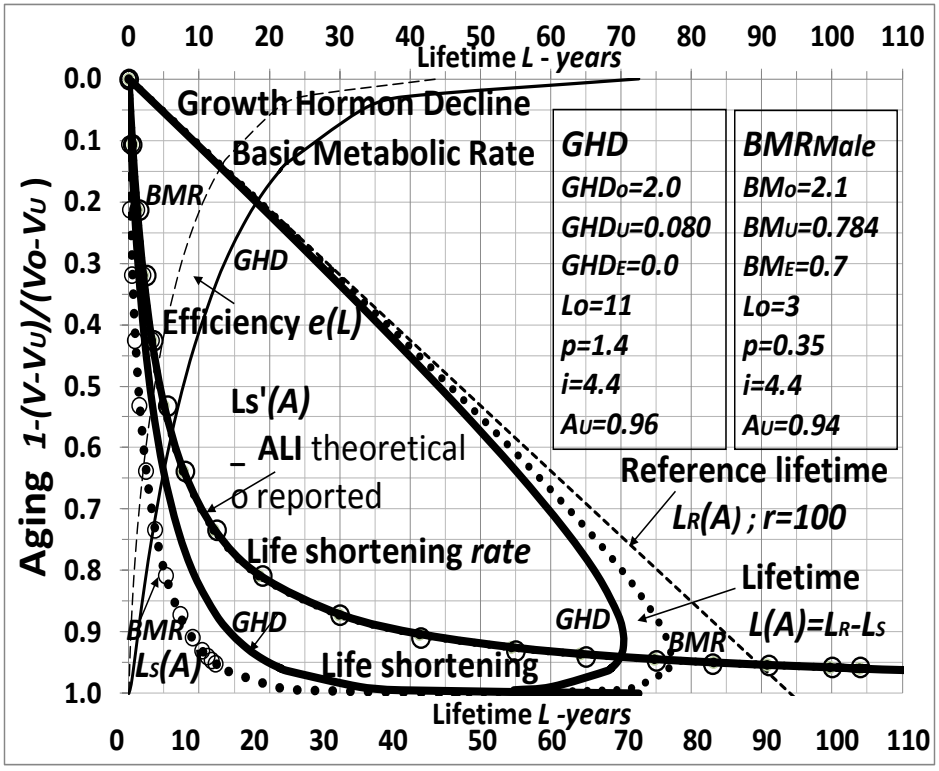


Figure 3. Interaction between basal metabolism and aging during the lifetime

**4. Pulmonary functions**

The study investigates the interaction of the air volume *V* forcibly exhaled (the cause) and the delivered volume (the effect) relative to the forced vital capacity *FVC* in time *T*. The interaction rate (3) and the interaction term (4) are interpreted directly in the *T*-*V* coordinates by recalculation of the propensity *p* and intensity *i* of interactions from reported spirograms for normal and Chronic Obstructive Pulmonary Disease **(**COPD**)** conditions (Fig. 4), as follows:

$$dT/dV = p + i\cdot V/(FVC - V) \quad (12)$$

$$T = (p-i)\cdot V + i\cdot FVC\cdot \ln\left[FVC/(FVC-V)\right] \quad (13)$$

The forced expiratory volume in one second, $FEV_1$ is then the solution of *V* in (13) for *T*=1.

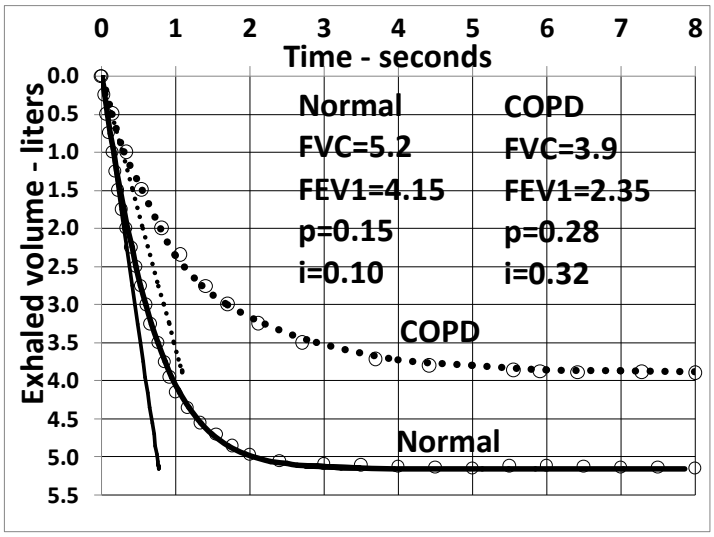


Figure 4. Spirogram (o-measured) represented by propensity and intensity of interaction

**5. Fatigue-and-endurance interaction**

Fatigue of materials and structures under lasting loads can cause yielding of durability (Ziha, 2009) and fractures. Body and mental fatigue are commonly considered as gradual wastage in time of some individual inherited or trained physiological or psychical limits. The study investigates the CEI concept (1-7) on an example of possible interaction between the crash risk due to driver's fatigue progression $F$ (the effect) and hours of driving $D$ (the cause) according to the Federal Motor Carrier Safety Administration (Fig. 5), as follows:

$$dF / dD = p + i \cdot d / (1 - d) \tag{12}$$

$$F(D, I) = (D_R - D_S) \cdot \left\{ p \cdot d - i \cdot \left[ d + \ln(1 - d) \right] \right\} \tag{13}$$

The fatigue related crash risk $F$ on daily basis $D_R$=24 hours in first $D_S$=6 hours slowly increase linearly to the driving hours at rate p=0.25 % per hour. The effect of driving hours intensifies after six hours due to interaction with progressing fatigue and limited endurance at additional rate of $i$=3.50 %/hour, where $d$=($D$-$D_S$)/($D_R$-$D_S$), Fig.5.

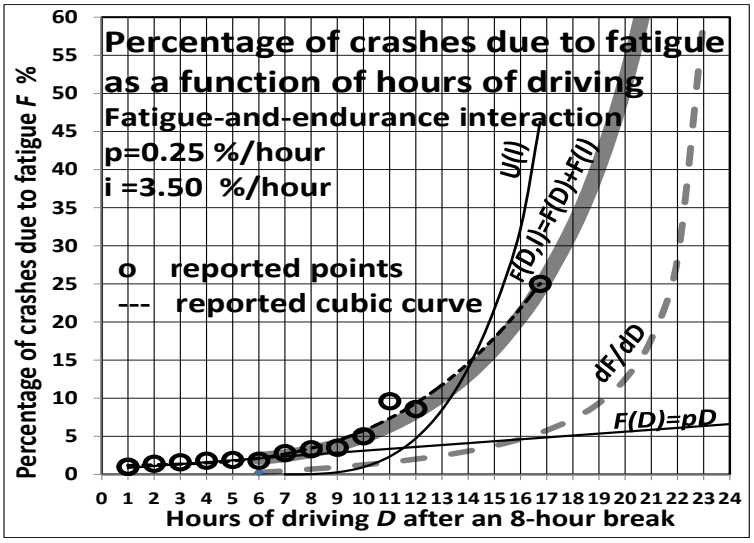


Figure 5. Fatigue related crash risk

## 5. Conclusion

The article has revealed the analytic formulation of the belief that some natural phenomena cannot be explained only as a simple causal relation of instantaneous or accumulated effects, but rather as cause-and-effect interaction.

The moment of observation, that is, the instantaneous present time, inevitably separates the past from the future time. The truth might be that the causality simultaneously depends on both past and future time over the moment of observation. With this respect, the observed elapsed effect $E(C)$ has been changing the upcoming cause $C$ on the expense of the remaining driving cause $C_R$-$C$ with respect to the finite causal capacity $C_R$. Therefore, the changes of the upcoming cause $C$ affected by the elapsed effect $E(C)$ may induce changes of the future effects in a trans-temporal interactive way. The experience of finiteness of changes in the world can identify the cause-and-effect interaction as the continuous natural sharing of limited causal capacities between the observed elapsed effect and the expected, but unobserved, forthcoming cause. The effect and its cause link the past and future at any time separated by the present instant of observation. Thus, the necessary cause of a perpetuating effect must exist in the future although it might be hidden beyond the moment of observation. The trans-temporal mental link between the past and future time is a belief founded on the former experience that the perceivable reality possesses conceivable natural properties of sufficient continuity and uniformity until it is not questioned by new observations. The conscious observers could try to predict what it will happen beyond the instant of observation as long as they could believe in some sort of empirical regularity of interaction. As it has been elaborated in the article, the mathematized linear cause-and-effect interaction concept, which follows from the general empirical causality notion, presents an analytic formulation of observable natural phenomena of trans-temporal continuous changes rather than an omnipresent natural law. The cause-and-effect interaction model is neither only an application of a straightforward fitting method to recorded data.

The character of changing phenomena can be estimated by two analytic parameters depending on the propensity to and the intensity of interactions which are then the subject of a post-observed verification. The analytical considerations based on the cause-and-effect interaction concept involve that the causal relations could be rather asymptotical logarithmic growth processes. The rapidly increasing sensitivities of the progressing effects to changes of elapsed and forthcoming causes typical for asymptotic behaviour may explain the great number of sudden ending or breaks of continuity in causal relations under uncertain and imperfect conditions.

The investigation of growth hormone decline and of basal metabolic rate demonstrates that the aging-and-lifetime interaction model can appropriately present some biological aging symptoms. The analysis of spirometric data shows that pulmonary physiology can be interpreted as inhalation and exhalation interactions. The paper reveals that the consequences of long exposures to loads and stresses can be represented as the fatigue–and-endurance interaction. The study also suggested that particular statements in the system feedback theory, as well as in the theory of growth, can be rational consequences of the cause-and-effect interaction concept. The article exposes that the interaction concept might be a rational approach to the understanding of some multifaceted causal relations of practical importance, sufficiently simple and accurate to tackle various realistic natural changing phenomena by analytical means. The future research should be focused on higher order interactions and probabilistic interpretations of possible randomness of multiple cause and effect interactions with respect to uncertainties in comprehension of causal resources and capacities in different natural phenomena.

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