

# The Mathematical Formula of the Causal Relationship $k$

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**Abstract:** The deterministic relationship between cause and effect is deeply connected with our understanding of the physical sciences and their explanatory ambitions. Though progress is being made, the lack of theoretical predictions and experiments in quantum gravity makes it difficult to use empirical evidence to justify a theory of causality at quantum level in normal circumstances, i. e. by predicting the value of a well-confirmed experimental result. For a variety of reasons, the problem of the deterministic relationship between cause and effect is related to basic problems of physics as such. Despite the common belief, it is a remarkable fact that a theory of causality should be consistent with a theory of everything and is because of this linked to problems of a theory of everything. Thus far, solving the problem of causality will help us to solve the problems of the theory of everything (at quantum level).

**Key words:** Quantum theory, relativity theory, unified field theory, causality.

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## 1. Introduction

On the one hand, as already mentioned above, and this may not come as a surprise, it is highly desirable to formulate a quantum mechanical version of the relationship between cause and effect. But at least one of the difficult questions that chaos theory raises for the epistemology of determinism of the relationship between cause and effect, can there exist a deterministic relationship between a cause and an effect at all. In other words, what is necessity, what is randomness? Quantum gravity for instance, can provide us a completely new view concerning the most fundamental of all relationships, the deterministic relationship between the cause and the effect. Although numerous attempts have been made in this topic, there is no commonly accepted solution of quantum gravity up to the present day. Research in quantum gravity, extremely difficult due to the missing close relationship between theory and experiment, is owing both, a technical and a conceptual difficulty too. A non-negligible minority of the physicist focus their attention on what is now called loop quantum gravity while the majority of the physicists is working in the field called string theory. Thus far, there is no single, generally agreed theory in quantum gravity. However, it is still quite unclear, in principle and even in practice, how to make any concrete predictions in these theories.

Under these conditions, quantum gravity and the deterministic relationship between a cause and an effect appear to be intimately connected with one another. The solution of the problems of causation can help to solve the problems of quantum gravity too.

## 2. Definitions

### 2.1. Definition. The Expectation Value and the Variance of a Random Variable $\mu X_t$

Let  ${}_R X_t$  denote a random variable which is determined by the random variable  ${}_0 X_t$  with the probability  $p({}_0 X_t)$ , the random variable  ${}_1 X_t$  with the probability  $p({}_1 X_t)$  and so on up to the random variable  ${}_N X_t$  with the probability  $p({}_N X_t)$ . The expectation of a single random variable  ${}_i X_t$  is defined as

$$E({}_i X_t) \equiv p({}_i X_t) \times {}_i X_t \tag{1}$$

while the expectation value of  $E({}_R X_t)$  at one Bernoulli trial  $t$  is defined as

$$E({}_R X_t) \equiv E({}_0 X_t) + E({}_1 X_t) + \dots + E({}_N X_t) \tag{2}$$

More important, all probabilities  $p_i$  add up to one ( $p({}_0 X_t) + p({}_1 X_t) + \dots + p({}_N X_t) = 1$ ). Quite naturally, the expected value can be viewed something like the weighted average, with  $p_i$ 's being the weights.

$$E({}_R X_t) \equiv \frac{p({}_0 X_t) \times {}_0 X_t + p({}_1 X_t) \times {}_1 X_t + \dots + p({}_N X_t) \times {}_N X_t}{p({}_0 X_t) + p({}_1 X_t) + \dots + p({}_N X_t)} \tag{3}$$

Under conditions where all outcomes  ${}_i X_t$  are equally likely (that is,  $p({}_0 X_t) = p({}_1 X_t) = \dots = p({}_N X_t)$ ), the weighted average turns finally into a simple average. In general, it is known, that  $E({}_i X_t^2) = p({}_i X_t) \times {}_i X_t^2 = {}_i X_t \times E({}_i X_t)$ .

**2.2. Definition. The Complex Conjugate  $\Psi^*({}_i X_t)$  a Random Variable  ${}_i X_t$**

In general, let  $\Psi({}_i X_t)$  denote the probability current/amplitude of the (complex) random variable (a complex number)  ${}_i X_t$ , such that  $\Psi({}_i X_t) = A({}_i X_t) + j^* B({}_i X_t)$ , where 'j' is the square root of -1 or  $j^2 = -1$ . In the same context, let  $\Psi^*({}_i X_t) = A({}_i X_t) - j^* B({}_i X_t)$  denote the complex conjugate of the (complex) random variable  ${}_i X_t$ . Generalizing Born's rule [1], we obtain

$$p({}_i X_t) = \Psi({}_i X_t) \times \Psi^*({}_i X_t) \equiv \frac{E({}_i X_t)}{{}_i X_t} \equiv \frac{E({}_i X_t)^2}{E({}_i X_t^2)} = \frac{p({}_i X_t)^2 \times X_t^2}{p({}_i X_t) \times X_t^2} \tag{4}$$

Under conditions, where  $\Psi({}_i X_t)$  is real, it is  $\Psi^*({}_i X_t) = \Psi({}_i X_t)$  and  $p({}_i X_t) = \Psi({}_i X_t) \times \Psi({}_i X_t)$ . In general, the complex conjugate  $\Psi^*({}_i X_t)$  of a (complex) random variable  $\Psi({}_i X_t)$  can be calculated as

$$\Psi^*({}_i X_t) \equiv \frac{p({}_i X_t)}{\Psi({}_i X_t)} \equiv \frac{E({}_i X_t)}{\Psi({}_i X_t) \times {}_i X_t} \tag{5}$$

**2.3. Definition. The Variance  $\sigma({}_i X_t)^2$  of the Random Variable  ${}_i X_t$**

Let  $\sigma({}_i X_t)^2$  denote the variance of the random variable  ${}_i X_t$  at the Bernoulli trial  $t$ . The variance of the random variable  ${}_i X_t$  at a single Bernoulli trial  $t$  is defined as

$$\sigma({}_i X_t)^2 \equiv E({}_i X_t^2) - E({}_i X_t)^2 \equiv ({}_i X_t^2) \times p({}_i X_t) - ({}_i X_t)^2 \times p({}_i X_t) \tag{6}$$

or as

$$\sigma({}_i X_t)^2 \equiv E({}_i X_t^2) - E({}_i X_t)^2 \equiv {}_i X_t^2 \times p({}_i X_t) \times (1 - p({}_i X_t)) \tag{7}$$

or as

$$\sigma({}_i X_t)^2 \equiv {}_i X_t \times E({}_i X_t) - E({}_i X_t)^2 \equiv E({}_i X_t) \times ({}_i X_t - E({}_i X_t)) = E({}_i X_t) \times E({}_i \underline{X}_t) \quad (8)$$

where  $E({}_i \underline{X}_t) = ({}_i X_t - E({}_i X_t)) = ({}_i X_t) \times (1 - p({}_i X_t))$  denotes something like an expectation value of anti  ${}_i X_t$ , a kind of a “hidden” random variable. Let  $\sigma({}_i X_t)$  denote the standard deviation of the random variable  ${}_i X_t$ . The standard deviation of the random variable  ${}_i X_t$  is defined as

$$\sigma({}_i X_t) \equiv \sqrt[2]{E({}_i X_t^2) - E({}_i X_t)^2} \equiv \sqrt[2]{({}_i X_t^2) \times p({}_i X_t) \times (1 - p({}_i X_t))} \quad (9)$$

#### 2.4. Definition. The Logical Contradiction $\Delta({}_i X_t)^2$ of the Random Variable ${}_i X_t$

Let  $\Delta({}_i X_t)^2$  denote the logical contradiction. We define the logical contradiction as

$$\Delta({}_i X_t)^2 \equiv \frac{\sigma({}_i X_t)^2}{({}_i X_t^2)} \equiv \frac{E({}_i X_t^2) - E({}_i X_t)^2}{({}_i X_t^2)} \equiv p({}_i X_t) \times (1 - p({}_i X_t)) \equiv p({}_i X_t) \times p({}_i \underline{X}_t) \quad (10)$$

Let  $\Delta({}_i X_t)$  denote the inner contradiction. We define

$$\Delta({}_i X_t) \equiv \frac{\sigma({}_i X_t)}{({}_i X_t)} \equiv \sqrt[2]{\frac{E({}_i X_t^2) - E({}_i X_t)^2}{({}_i X_t^2)}} \equiv \sqrt[2]{p({}_i X_t) \times (1 - p({}_i X_t))} \quad (11)$$

#### Remark.

Under conditions of special theory of relativity,  ${}_R X_t$  can denote the expectation value as determined by the stationary observer  $R$  while  ${}_0 X_t$  can denote the value (i. e. after the collapse of the wave function) as determined by the moving observer  $O$ .

#### The Cause

#### 2.5. Definition. The Expectation Value of the Cause $E({}_R U_t)$ at a Bernoulli Trial $t$

In general, we define the expectation value of the cause  ${}_R U_t$  at one single Bernoulli trial  $t$  (i. e. at a certain point in space-time  $t$  et cetera) as

$$E\left({}_R U_t\right) \equiv p\left({}_R U_t\right) \times {}_R U_t \quad (12)$$

where  $p\left({}_R U_t\right)$  denotes the probability at one single Bernoulli trial  $t$  that the random variable  ${}_R U_t$  is

the cause of an effect (denoted by the random variable  ${}_R W_t$ ).

#### 2.6. Definition. The Expectation Value of the Cause Squared Denoted As $E({}_R U_t^2)$ at a Bernoulli Trial $t$

In general, we define the expectation value of the cause squared at one single Bernoulli trial  $t$  (i. e. at a

certain point in space-time t et cetera) as

$$E\left({}_R U_t^2\right) \equiv p\left({}_R U_t\right) \times {}_R U_t^2 \quad (13)$$

where  $p\left({}_R U_t\right)$  denotes the probability at one single Bernoulli trial  $t$  that the random variable  ${}_R U_t$  is the cause of the effect (denoted by the random variable  ${}_R W_t$ ).

**2.7. Definition. The Variance  $\sigma({}_R U_t)^2$  of the Cause at a Bernoulli Trial  $t$**

In general, we define the variance  $\sigma({}_R U_t)^2$  of the cause at one single Bernoulli trial  $t$  (i. e. at a certain point in space-time et cetera) as

$$\sigma\left({}_R U_t\right)^2 \equiv E\left({}_R U_t^2\right) - \left(E\left({}_R U_t\right)\right)^2 = \left({}_R U_t\right) \times p\left({}_R U_t\right) \times \left(1 - p\left({}_R U_t\right)\right) \quad (14)$$

The standard deviation  $\sigma({}_R U_t)$  of the cause  ${}_R U_t$  at one single Bernoulli trial  $t$  follows as

$$\sigma\left({}_R U_t\right) \equiv \sqrt[2]{\sigma\left({}_R U_t\right)^2} \equiv \sqrt[2]{E\left({}_R U_t^2\right) - \left(E\left({}_R U_t\right)\right)^2} = \left({}_R U_t\right) \times \sqrt[2]{p\left({}_R U_t\right) \times \left(1 - p\left({}_R U_t\right)\right)} \quad (15)$$

**The Effect**

**2.8. Definition. The Expectation Value of the Effect  $E({}_0 W_t)$  at a Bernoulli Trial  $t$**

In general, we define the expectation value of the effect as  $E({}_0 W_t)$  at one single Bernoulli trial  $t$  (i.e. at a certain point in space-time t et cetera) as

$$E\left({}_0 W_t\right) \equiv p\left({}_0 W_t\right) \times {}_0 W_t \quad (16)$$

where  $p\left({}_0 W_t\right)$  denotes the probability at one single Bernoulli trial  $t$  of the random variable  ${}_0 W_t$ , which is an effect.

**2.9. Definition. The Expectation Value of the Effect Squared  $E({}_0 W_t^2)$  at a Bernoulli Trial  $t$**

In general, we define the expectation value of the effect squared denoted as  $E({}_0 W_t^2)$  at one single Bernoulli trial  $t$  (i. e. at a certain point in space-time et cetera) as

$$E\left({}_0 W_t^2\right) \equiv p\left({}_0 W_t\right) \times {}_0 W_t \times {}_0 W_t \quad (17)$$

where  $p\left({}_0 W_t\right)$  denotes the probability at one single Bernoulli trial  $t$  of the random variable  ${}_0 W_t$

which is an effect.

**2.10. Definition. The Variance  $\sigma({}_0W_t)^2$  of the Effect  ${}_0W_t$  at a Bernoulli Trial  $t$**

In general, we define the variance  $\sigma({}_0W_t)^2$  of the effect  $E({}_0W_t)$  at one single Bernoulli trial  $t$  (i. e. at a certain point in space-time et cetera) as

$$\sigma({}_0W_t)^2 \equiv E({}_0W_t^2) - (E({}_0W_t))^2 = ({}_0W_t) \times p({}_0W_t) \times (1 - p({}_0W_t)) \tag{18}$$

The standard deviation  $\sigma({}_0W_t)$  of the effect  ${}_0W_t$  at one single Bernoulli trial  $t$  follows as

$$\sigma({}_0W_t) \equiv \sqrt{\sigma({}_0W_t)^2} \equiv \sqrt{E({}_0W_t^2) - (E({}_0W_t))^2} = ({}_0W_t) \times \sqrt{p({}_0W_t) \times (1 - p({}_0W_t))} \tag{19}$$

**The Cause and The Effect**

**2.11. Definition. The Expectation Value of the Cause  ${}_R U_t$  and Effect  ${}_0W_t$  at a Bernoulli Trial  $t$**

In general, we define the expectation value of the cause  ${}_R U_t$  and the effect  ${}_0W_t$  at one single Bernoulli trial  $t$  (i. e. at a certain point in space-time et cetera) as

$$E({}_R U_t, {}_0W_t) \equiv p({}_R U_t \cap {}_0W_t) \times {}_R U_t \times {}_0W_t \tag{20}$$

where  $p({}_R U_t \cap {}_0W_t)$  denotes the joint probability distribution of the cause  ${}_R U_t$  and the effect  ${}_0W_t$  at one single Bernoulli trial  $t$ .

**2.12. Definition. The Co-variance of the Cause  ${}_R U_t$  and the Effect  ${}_0W_t$  at a Bernoulli Trial  $t$**

In general, we define the co-variance of the cause  ${}_R U_t$  and the effect  ${}_0W_t$  denoted as  $\sigma({}_R U_t, {}_0W_t)$  at one single Bernoulli trial  $t$  (i. e. at a certain point in space-time t et cetera) as

$$\sigma({}_R U_t, {}_0W_t) \equiv E({}_R U_t, {}_0W_t) - (E({}_R U_t) \times E({}_0W_t)) = ({}_R U_t \times {}_0W_t) \times (p({}_R U_t \cap {}_0W_t) - p({}_R U_t) \times p({}_0W_t)) \tag{21}$$

**2.13. Definition. The Mathematical Formula of the Causal Relationship  $k$  at a Bernoulli Trial  $t$**

In general, we define the mathematical formula of the causal relationship  $k$  as

$$k({}_R U_t, {}_0W_t) \equiv \frac{\sigma({}_R U_t, {}_0W_t)}{\sigma({}_R U_t) \times \sigma({}_0W_t)} \equiv \frac{E({}_R U_t, {}_0W_t) - (E({}_R U_t) \times E({}_0W_t))}{\sqrt{E({}_R U_t) \times ({}_R U_t - E({}_R C_t)) \times E({}_0W_t) \times ({}_0W_t - E({}_0W_t))}} \tag{22}$$

or something as

$$k\left({}_R U_t, {}_0 W_t\right) \equiv \frac{\sigma\left({}_R U_t, {}_0 W_t\right)}{\sigma\left({}_R U_t\right) \times \sigma\left({}_0 W_t\right)} \equiv \frac{\left({}_R U_t \times {}_0 W_t\right) \times \left(p\left({}_R U_t \cap {}_0 W_t\right) - p\left({}_R U_t\right) \times p\left({}_0 W_t\right)\right)}{\sqrt{\left({}_R U_t \times {}_R U_t\right) \times \left(p\left({}_R U_t\right) - p\left({}_R U_t\right)^2\right) \times \left({}_0 W_t \times {}_0 W_t\right) \times \left(p\left({}_0 W_t\right) - p\left({}_0 W_t\right)^2\right)}} \quad (23)$$

or as

$$k\left({}_R U_t, {}_0 W_t\right) \equiv \frac{\sigma\left({}_R U_t, {}_0 W_t\right)}{\sigma\left({}_R U_t\right) \times \sigma\left({}_0 W_t\right)} \equiv \frac{\left(p\left({}_R U_t \cap {}_0 W_t\right) - p\left({}_R U_t\right) \times p\left({}_0 W_t\right)\right)}{\sqrt{p\left({}_R U_t\right) \times \left(1 - p\left({}_R U_t\right)\right) \times p\left({}_0 W_t\right) \times \left(1 - p\left({}_0 W_t\right)\right)}} \quad (24)$$

**Remark.**

The range of k is  $-1 \leq k \leq +1$ . Thus far, it is important to note, that the mathematical formula of the causal relationship k is not identical with Pearson’s coefficient of correlation. While causation is not identical with correlation, it is not the purpose of this publication to provide a sharp distinction between causation and correlation. A very precise distinction between causation and correlation can be found in literature.

**2.14. Axioms.**

The following theory is based on the following axioms.

**Axiom I.**

$$+1 = +1. \quad (25)$$

**Axiom II.**

$$\frac{+0}{+0} \equiv +1 \quad (26)$$

**Axiom III.**

$$\frac{+1}{+0} \equiv +\infty \quad (27)$$

**3. Theorems**

**3.1. Theorem. The Cause  ${}_R U_t$ .**

**Claim.**

In general, the cause  ${}_R U_t$  is determined as

$${}_R U_t = \frac{\sigma\left({}_R U_t\right)}{\sqrt{p\left({}_R U_t\right) \times \left(1 - p\left({}_R U_t\right)\right)}}. \quad (28)$$

**Proof.**

Starting with Axiom I it is

$$+1 = +1 \tag{29}$$

Multiplying this equation by standard deviation  $\sigma({}_R U_t)$  of the cause  ${}_R U_t$  it follows that

$$\sigma\left({}_R U_t\right) = \sigma\left({}_R U_t\right). \tag{30}$$

Due to the definition of the standard deviation  $\sigma({}_R U_t)$  of the cause  ${}_R U_t$  at a certain Bernoulli trial  $t$  as

$$\sigma\left({}_R U_t\right) \equiv \sqrt{\sigma\left({}_R U_t\right)^2} \equiv \sqrt{E\left({}_R U_t^2\right) - E\left({}_R U_t\right)^2} = \left({}_R U_t\right) \times \sqrt{p\left({}_R U_t\right) \times \left(1 - p\left({}_R U_t\right)\right)} \text{ we obtain}$$

$$\sigma\left({}_R U_t\right) = \left({}_R U_t\right) \times \sqrt{p\left({}_R U_t\right) \times \left(1 - p\left({}_R U_t\right)\right)}. \tag{31}$$

After division it follows that

$${}_R U_t = \frac{\sigma\left({}_R U_t\right)}{\sqrt{p\left({}_R U_t\right) \times \left(1 - p\left({}_R U_t\right)\right)}}. \tag{32}$$

**Quod erat demonstrandum.**

*Remark.*

The cause  ${}_R U_t$  at a certain Bernoulli trial  $t$  is determined by the standard deviation  $\sigma({}_R U_t)$  and the probability  $p({}_R U_t)$  associated with the cause  ${}_R U_t$ .

**3.2. Theorem. The Effect  ${}_0 W_t$ .**

**Claim.**

In general, the effect  ${}_0 W_t$  is determined as

$${}_0 W_t = \frac{\sigma\left({}_0 W_t\right)}{\sqrt{p\left({}_0 W_t\right) \times \left(1 - p\left({}_0 W_t\right)\right)}} \tag{33}$$

**Proof.**

Starting with Axiom I it is

$$+1 = +1 \tag{34}$$

Multiplying this equation by standard deviation  $\sigma({}_0 W_t)$  it follows that

$$\sigma\left({}_0 W_t\right) = \sigma\left({}_0 W_t\right) \tag{35}$$

Due to the definition of the standard deviation  $\sigma({}_0W_t)^2$  of the effect  ${}_0W_t$  at a certain Bernoulli trial  $t$  as  $\sigma({}_0W_t) \equiv \sqrt{\sigma({}_0W_t)^2} \equiv \sqrt{E({}_0W_t^2) - (E({}_0W_t))^2} = ({}_0W_t) \times \sqrt{p({}_0W_t) \times (1 - p({}_0W_t))}$  we obtain

$$\sigma({}_0W_t) = ({}_0W_t) \times \sqrt{p({}_0W_t) \times (1 - p({}_0W_t))} \tag{36}$$

After division it follows that

$${}_0W_t = \frac{\sigma({}_0W_t)}{\sqrt{p({}_0W_t) \times (1 - p({}_0W_t))}} \tag{37}$$

**Quod erat demonstrandum.**

*Remark.*

The effect  ${}_0W_t$  at a certain Bernoulli trial  $t$  itself is determined by the standard deviation  $\sigma({}_0W_t)$  and the probability  $p({}_0W_t)$  as associated with the effect  ${}_0W_t$ .

**3.3. Theorem. The Cause  ${}_R U_t$  and the Effect  ${}_0 W_t$ .**

**Claim.**

In general, cause  ${}_R U_t$  and effect  ${}_0 W_t$  are determined too as

$${}_R U_t \times {}_0 W_t = \frac{\sigma({}_R U_t, {}_0 W_t)}{p({}_R U_t \cap {}_0 W_t) - p({}_R U_t) \times p({}_0 W_t)}. \tag{38}$$

**Proof.**

Starting with Axiom I it is

$$+1 = +1 \tag{39}$$

Multiplying this equation by the co-variance  $\sigma({}_R U_t, {}_0 W_t)$  of cause  ${}_R U_t$  and effect  ${}_0 W_t$  it follows that

$$\sigma({}_R U_t, {}_0 W_t) = \sigma({}_R U_t, {}_0 W_t) \tag{40}$$

Due to the definition of the co-variance of the cause  ${}_R U_t$  and the effect  ${}_0 W_t$  at a certain Bernoulli trial  $t$  as  $\sigma({}_R U_t, {}_0 W_t) \equiv E({}_R U_t, {}_0 W_t) - (E({}_R U_t) \times E({}_0 W_t)) = ({}_R U_t \times {}_0 W_t) \times (p({}_R U_t \cap {}_0 W_t) - p({}_R U_t) \times p({}_0 W_t))$  we obtain

$$\sigma({}_R U_t, {}_0 W_t) = ({}_R U_t \times {}_0 W_t) \times (p({}_R U_t \cap {}_0 W_t) - p({}_R U_t) \times p({}_0 W_t)) \tag{41}$$

After Division, it follows that



$${}_R U_t \times {}_0 W_t = \frac{\sigma({}_R U_t, {}_0 W_t)}{p({}_R U_t \cap {}_0 W_t) - p({}_R U_t) \times p({}_0 W_t)} \quad (42)$$

**Quod erat demonstrandum.**

*Scholium.*

It is necessary to make a difference between one single Bernoulli trial  $t$  and the whole population (i. e. sample) of the size  $N$ .

**3.4. Theorem. The Mathematical Formula of the Causal Relationship  $k$**

**Claim.**

In general, the mathematical formula of the causal relationship  $k$  is determined as

$$k({}_R U_t, {}_0 W_t) = \frac{\sigma({}_R U_t, {}_0 W_t)}{\sigma({}_R U_t) \times \sigma({}_0 W_t)} = \frac{p({}_R U_t \cap {}_0 W_t) - p({}_R U_t) \times p({}_0 W_t)}{\sqrt[2]{p({}_R U_t) \times (1 - p({}_R U_t))} \times p({}_0 W_t) \times (1 - p({}_0 W_t))}} \quad (43)$$

**Proof.**

Starting with Axiom I it is

$$+1 = +1 \quad (44)$$

Multiplying this equation by the cause  ${}_R U_t$  it is

$${}_R U_t = {}_R U_t. \quad (45)$$

The multiplication by the effect  ${}_0 W_t$  yields

$${}_R U_t \times {}_0 W_t = {}_R U_t \times {}_0 W_t. \quad (46)$$

or, under *the assumption of commutativity*, it is  ${}_R U_t \times {}_0 W_t = {}_0 W_t \times {}_R U_t$ . Due to the theorem above, it is

Thus far, the relationship  ${}_R U_t \times {}_0 W_t = {}_R U_t \times {}_0 W_t$  changes to

$${}_R U_t = \frac{\sigma({}_R U_t)}{\sqrt[2]{p({}_R U_t) \times (1 - p({}_R U_t))}}.$$

$${}_R U_t \times {}_0 W_t = \frac{\sigma({}_R U_t)}{\sqrt[2]{p({}_R U_t) \times (1 - p({}_R U_t))}} \times {}_0 W_t. \quad (47)$$

Due to the other theorem above, it is

$${}_0 W_t = \frac{\sigma({}_0 W_t)}{\sqrt[2]{p({}_0 W_t) \times (1 - p({}_0 W_t))}}.$$

Consequently, the equation before is equivalent with

$${}_R U_t \times {}_0 W_t = \frac{\sigma({}_R U_t)}{\sqrt[2]{p({}_R U_t) \times (1 - p({}_R U_t))}} \times \frac{\sigma({}_0 W_t)}{\sqrt[2]{p({}_0 W_t) \times (1 - p({}_0 W_t))}}. \tag{48}$$

The theorem about the co-variance of cause and effect yields  ${}_{R U_t \times 0 W_t} = \frac{\sigma({}_R U_t, {}_0 W_t)}{p({}_R U_t \cap {}_0 W_t) - p({}_R U_t) \times p({}_0 W_t)}$ .

Thus far, we obtain the next relationship as

$$\frac{\sigma({}_R U_t, {}_0 W_t)}{p({}_R U_t \cap {}_0 W_t) - p({}_R U_t) \times p({}_0 W_t)} = \frac{\sigma({}_R U_t)}{\sqrt[2]{p({}_R U_t) \times (1 - p({}_R U_t))}} \times \frac{\sigma({}_0 W_t)}{\sqrt[2]{p({}_0 W_t) \times (1 - p({}_0 W_t))}}. \tag{49}$$

Rearranging this equation yields

$$\frac{\sigma({}_R U_t, {}_0 W_t)}{\sigma({}_R U_t) \times \sigma({}_0 W_t)} = \frac{p({}_R U_t \cap {}_0 W_t) - p({}_R U_t) \times p({}_0 W_t)}{\sqrt[2]{p({}_R U_t) \times (1 - p({}_R U_t)) \times p({}_0 W_t) \times (1 - p({}_0 W_t))}} \tag{50}$$

which is equivalent to the definition of the causal relationship k at each Bernoulli trial t as

$$k({}_R U_t, {}_0 W_t) = \frac{\sigma({}_R U_t, {}_0 W_t)}{\sigma({}_R U_t) \times \sigma({}_0 W_t)} = \frac{p({}_R U_t \cap {}_0 W_t) - p({}_R U_t) \times p({}_0 W_t)}{\sqrt[2]{(p({}_R U_t) - p({}_R U_t)^2) \times (p({}_0 W_t) - p({}_0 W_t)^2)}} \tag{51}$$

**Quod erat demonstrandum.**

*Remark.*

The following Table 1 may illustrate the definitions and relationships above in more detail.

Table 1. The Definitions and Relationships Above in More Detail

Fig.		Effect		
		Yes	No	
Cause	Yes	${}_0 U$	${}_{\Delta} U$	${}_R U$
	No	${}_0 \underline{U}$	${}_{\Delta} \underline{U}$	${}_R \underline{U}$
		${}_0 W$	${}_{\Delta} W$	${}_R W$

The above formula of the causal relationship k is ensuring the deterministic relationship between cause and effect at **every single Bernoulli trial t**. Under the assumption that the probabilities from trial to trial t are constant and not changing (i. e. conditions of special theory of relativity, v=constant), we obtain

$\sum_{t=1}^N k \left( {}_R U_t, {}_0 W_t \right) = N \times k \left( {}_R U_t, {}_0 W_t \right)$ , while N is the population size or the number of Bernoulli trials.

**3.5. Theorem. Quantum Theory and Causality**

The influence of the Copenhagen dominated interpretation of quantum mechanics has caused deep doubts about the unrestricted validity of the principle of causality at quantum level and in general as such. Thus far, it is useful to express the mathematical formula of the causal relationship k using the mathematical framework of quantum theory.

**Claim.**

Under conditions of quantum theory, the mathematical formula of the causal relationship k is determined as

$$k \left( {}_R U_t, {}_0 W_t \right) = \frac{\Psi \left( {}_R U_t \cap {}_0 W_t \right) \times \Psi^* \left( {}_R U_t \cap {}_0 W_t \right) - \left( \Psi \left( {}_R U_t \right) \times \Psi^* \left( {}_R U_t \right) \right) \times \left( \Psi \left( {}_0 W_t \right) \times \Psi^* \left( {}_0 W_t \right) \right)}{\sqrt[2]{\left( \Psi \left( {}_R U_t \right) \times \Psi^* \left( {}_R U_t \right) \right) \times \left( 1 - \left( \Psi \left( {}_R U_t \right) \times \Psi^* \left( {}_R U_t \right) \right) \right) \times \left( \Psi \left( {}_0 W_t \right) \times \Psi^* \left( {}_0 W_t \right) \right) \times \left( 1 - \left( \Psi \left( {}_0 W_t \right) \times \Psi^* \left( {}_0 W_t \right) \right) \right)}}$$

(52)

**Proof.**

Starting with Axiom I it is

$$+1 = +1$$

(53)

Multiplying this equation by the causal relationship  $k \left( {}_R U_t, {}_0 W_t \right)$  it is

$$k \left( {}_R U_t, {}_0 W_t \right) = k \left( {}_R U_t, {}_0 W_t \right)$$

(54)

which is equivalent with

$$k \left( {}_R U_t, {}_0 W_t \right) = \frac{\sigma \left( {}_R U_t, {}_0 W_t \right)}{\sigma \left( {}_R U_t \right) \times \sigma \left( {}_0 W_t \right)} = \frac{p \left( {}_R U_t \cap {}_0 W_t \right) - p \left( {}_R U_t \right) \times p \left( {}_0 W_t \right)}{\sqrt[2]{\left( p \left( {}_R U_t \right) - p \left( {}_R U_t \right)^2 \right) \times \left( p \left( {}_0 W_t \right) - p \left( {}_0 W_t \right)^2 \right)}}$$

(55)

Due to Born’s rule, the joint probability distribution function of cause and effect is determined by the function  $p \left( {}_R U_t \cap {}_0 W_t \right) \equiv \Psi \left( {}_R U_t \cap {}_0 W_t \right) \times \Psi^* \left( {}_R U_t \cap {}_0 W_t \right)$ , the probability as associated with the cause is determined by  $p \left( {}_R U_t \right) \equiv \Psi \left( {}_R U_t \right) \times \Psi^* \left( {}_R U_t \right)$ , while the probability as associated with the effect is determined as  $p \left( {}_0 W_t \right) \equiv \Psi \left( {}_0 W_t \right) \times \Psi^* \left( {}_0 W_t \right)$ . The mathematical formula of the causal relationship k, using the mathematical framework of quantum theory, follows straightforward as

$$k\left({}_R U_t, {}_0 W_t\right) = \frac{\Psi\left({}_R U_t \cap {}_0 W_t\right) \times \Psi^*\left({}_R U_t \cap {}_0 W_t\right) - \left(\Psi\left({}_R U_t\right) \times \Psi^*\left({}_R U_t\right)\right) \times \left(\Psi\left({}_0 W_t\right) \times \Psi^*\left({}_0 W_t\right)\right)}{\sqrt[2]{\left(\Psi\left({}_R U_t\right) \times \Psi^*\left({}_R U_t\right)\right) \times \left(1 - \left(\Psi\left({}_R U_t\right) \times \Psi^*\left({}_R U_t\right)\right)\right) \times \left(\Psi\left({}_0 W_t\right) \times \Psi^*\left({}_0 W_t\right)\right) \times \left(1 - \left(\Psi\left({}_0 W_t\right) \times \Psi^*\left({}_0 W_t\right)\right)\right)}} \quad (56)$$

**Quod erat demonstrandum.**

*Remark.*

There may exist circumstances where  $p\left({}_R U_t \cap {}_0 W_t\right) \equiv \Psi\left({}_R U_t \cap {}_0 W_t\right) \times \Psi^*\left({}_R U_t \cap {}_0 W_t\right) = 0$ .

### 3.6. Theorem. The Formula of the Causal Relationship k of a Binomial Random Variable

**Claim.**

Thus far, we define  $\sum\left({}_R U \cap {}_0 W\right) \equiv E\left({}_R U \cap {}_0 W\right) \equiv \sum_{t=1}^N {}_R U_t \times {}_0 W_t \times p\left({}_R U_t \cap {}_0 W_t\right) = N \times p\left({}_R U_t \cap {}_0 W_t\right)$  and  $\sum\left({}_R U\right) \equiv E\left({}_R U\right) \equiv N \times p\left({}_R U_t\right)$  and  $\sum\left({}_0 W\right) \equiv E\left({}_0 W\right) \equiv N \times p\left({}_0 W_t\right)$ . In general, under conditions where **the probability of an event p and the causal relationship k are constant from trial to trial**, the mathematical formula of the causal relationship k can be simplified as

$$k\left({}_R U_t, {}_0 W_t\right) \equiv \frac{N \times \sum\left({}_R U \cap {}_0 W\right) - \left(\sum {}_R U \times \sum {}_0 W\right)}{\sqrt[2]{\left(N \times \sum {}_R U - \sum {}_R U \times \sum {}_R U\right) \times \left(N \times \sum {}_0 W - \sum {}_0 W \times \sum {}_0 W\right)}} \quad (57)$$

**Proof.**

Starting with Axiom I it is

$$+1 = +1 \quad (58)$$

Multiplying this equation by the causal relationship  $k\left({}_R U_t, {}_0 W_t\right)$  it is

$$k\left({}_R U_t, {}_0 W_t\right) = k\left({}_R U_t, {}_0 W_t\right) \quad (59)$$

which is equivalent to

$$k\left({}_R U_t, {}_0 W_t\right) = 1 \times k\left({}_R U_t, {}_0 W_t\right) \quad (60)$$

or to

$$k\left({}_R U_t, {}_0 W_t\right) = \frac{N \times N}{N \times N} \times k\left({}_R U_t, {}_0 W_t\right) \quad (61)$$

where  $N$  denotes the total number of Bernoulli trials  $t$ , the number of experiments, the sample size et cetera.

Due to the theorem above, this equation is equivalent with

$$k\left({}_R U_t, {}_0 W_t\right) \equiv \frac{N \times N \times \left( p\left({}_R U_t \cap {}_0 W_t\right) - p\left({}_R U_t\right) \times p\left({}_0 W_t\right) \right)}{N \times N \times \sqrt{2} \sqrt{p\left({}_R U_t\right) \times \left(1 - p\left({}_R U_t\right)\right) \times p\left({}_0 W_t\right) \times \left(1 - p\left({}_0 W_t\right)\right)}} \quad (62)$$

or with

$$k\left({}_R U_t, {}_0 W_t\right) \equiv \frac{N \times N \times p\left({}_R U_t \cap {}_0 W_t\right) - \left( N \times N \times p\left({}_R U_t\right) \times p\left({}_0 W_t\right) \right)}{\sqrt{2} \sqrt{N \times N \times \left( p\left({}_R U_t\right) - p\left({}_R U_t\right)^2 \right) \times N \times N \times \left( p\left({}_0 W_t\right) - p\left({}_0 W_t\right)^2 \right)}} \quad (63)$$

Due to our definitions  $\sum({}_R U \cap {}_0 W) \equiv E({}_R U \cap {}_0 W) \equiv \sum_{t=1}^N {}_R U_t \times {}_0 W_t \times p({}_R U_t \cap {}_0 W_t) = N \times p({}_R U_t \cap {}_0 W_t)$ ,  $\sum({}_R U) \equiv E({}_R U) \equiv N \times p({}_R U_t)$  and  $\sum({}_0 W) \equiv E({}_0 W) \equiv N \times p({}_0 W_t)$ , the formula above can be simplified as

$$k\left({}_R U_t, {}_0 W_t\right) \equiv \frac{N \times \sum\left({}_R U \cap {}_0 W\right) - \left( \sum {}_R U \times \sum {}_0 W \right)}{\sqrt{2} \sqrt{\left( N \times \sum {}_R U - \sum {}_R U \times \sum {}_R U \right) \times \left( N \times \sum {}_0 W - \sum {}_0 W \times \sum {}_0 W \right)}} \quad (64)$$

**Quod erat demonstrandum.**

*Remark.*

Under the conditions above, the significance of the causal relationship  $k$  can be tested using the Chi-Square distribution with one degree of freedom. The following Table 2 may provide an overview.

Table 2. An Overview of the Significance of the Causal Relationship  $k$  Can Be Tested Using the Chi-Square Distribution with One Degree of Freedom

Fig.		Effect		
		Yes	No	
Cause	Yes	$E({}_R U \cap {}_0 W) \equiv N \times p({}_R U_t \cap {}_0 W_t)$	${}_{\Delta} U$	$E({}_R U) \equiv N \times p({}_R U_t)$
	No	${}_{0} \underline{U}$	${}_{\Delta} \underline{U}$	$E({}_R \underline{U}) \equiv N \times (1 - p({}_R U_t))$
		$E({}_0 W) \equiv N \times p({}_0 W_t)$	${}_{\Delta} W$	$E({}_R W) \equiv N \times (1 - p({}_R U_t) + p({}_R U_t)) \equiv N$

In statistics, the *phi coefficient* as a measure of strength of association, pioneered especially by Karl Pearson (1857 - 1936), is one of the known measures of association between two binomial random variables. While there may exist situations where Pearson's phi coefficient is numerically identical with the mathematical formula of the causal relationship  $k$ , both are not identical as such.

**3.7. Theorem. The Standard Normal Distribution and the Formula of the Causal Relationship  $k$ .**

**Claim.**

Under some assumptions, the mathematical formula of the causal relationship  $k$  is determined by the standard normal distribution (i. e. by chi-square distribution) as

$$k\left({}_R U_t, {}_0 W_t\right) = \sqrt{\frac{Z^2}{N}} \quad (65)$$

**Proof.**

Starting with Axiom I it is

$$+1 = +1 \quad (66)$$

Multiplying this equation by the causal relationship  $k\left({}_R U_t, {}_0 W_t\right)$  it is

$$k\left({}_R U_t, {}_0 W_t\right) = k\left({}_R U_t, {}_0 W_t\right) \quad (67)$$

which is equivalent with

$$\frac{k\left({}_R U_t, {}_0 W_t\right) - E\left(k\left({}_R U_t, {}_0 W_t\right)\right)}{\sigma\left(k\left({}_R U_t, {}_0 W_t\right)\right)} = \frac{k\left({}_R U_t, {}_0 W_t\right) - E\left(k\left({}_R U_t, {}_0 W_t\right)\right)}{\sigma\left(k\left({}_R U_t, {}_0 W_t\right)\right)} \quad (68)$$

where  $E\left(k\left({}_R U_t, {}_0 W_t\right)\right)$  denotes the expectation value of the causal relationship  $k$  at each single

Bernoulli trial  $t$ ,  $\sigma\left(k\left({}_R U_t, {}_0 W_t\right)\right)$  denotes the standard deviation of the causal relationship  $k$  at each

Bernoulli trial  $t$ . In general, the normal random variable  $Z$  of a standard normal distribution at each single Bernoulli trial  $t$  (called a standard score or a z-score) is determined

as  $Z\left(k\left({}_R U_t, {}_0 W_t\right)\right) = \frac{k\left({}_R U_t, {}_0 W_t\right) - E\left(k\left({}_R U_t, {}_0 W_t\right)\right)}{\sigma\left(k\left({}_R U_t, {}_0 W_t\right)\right)}$ . Thus far, we obtain

$$Z\left(k\left({}_R U_t, {}_0 W_t\right)\right) = \frac{k\left({}_R U_t, {}_0 W_t\right) - E\left(k\left({}_R U_t, {}_0 W_t\right)\right)}{\sigma\left(k\left({}_R U_t, {}_0 W_t\right)\right)} \quad (69)$$

Under conditions where  $E\left(k\left({}_R U_t, {}_0 W_t\right)\right) = 0$  and  $\sigma\left(k\left({}_R U_t, {}_0 W_t\right)\right) = 1$  it follows that

$$Z\left(k\left({}_R U_t, {}_0 W_t\right)\right) = \frac{k\left({}_R U_t, {}_0 W_t\right) - 0}{1} \quad (70)$$

or that

$$Z\left(k\left({}_R U_t, {}_0 W_t\right)\right) = k\left({}_R U_t, {}_0 W_t\right) \quad (71)$$

After the square root operation we obtain

$$Z\left(k\left({}_R U_t, {}_0 W_t\right)\right)^2 = k\left({}_R U_t, {}_0 W_t\right)^2 \quad (72)$$

Summarizing yields

$$\sum_{t=1}^N Z\left(k\left({}_R U_t, {}_0 W_t\right)\right)^2 = \sum_{t=1}^N k\left({}_R U_t, {}_0 W_t\right)^2 \quad (73)$$

which is equivalent with

$$Z^2 = \sum_{t=1}^N k\left({}_R U_t, {}_0 W_t\right)^2 \quad (74)$$

In other words, under conditions, where the causal relationship  $k$  is **constant** from trial to trial, it is equally  $k\left({}_R U_1, {}_0 W_1\right)^2 = k\left({}_R U_2, {}_0 W_2\right)^2 = \dots = k\left({}_R U_N, {}_0 W_N\right)^2 = N \times k\left({}_R U_t, {}_0 W_t\right)^2$ . We obtain

$$Z^2 = \sum_{t=1}^N k\left({}_R U_t, {}_0 W_t\right)^2 = k\left({}_R U_1, {}_0 W_1\right)^2 + k\left({}_R U_2, {}_0 W_2\right)^2 + \dots + k\left({}_R U_N, {}_0 W_N\right)^2 = N \times k\left({}_R U_t, {}_0 W_t\right)^2 \quad (75)$$

The chi-square distribution as such is connected to a number of other special distributions. Under conditions where  $Z_1, Z_2, \dots, Z_N$  is a sequence of independent standard normal variables then the sum of the squares has the chi-square distribution with  $N$  degrees of freedom. We obtain

$$Z^2 = X^2_N = \sum_{t=1}^N k\left({}_R U_t, {}_0 W_t\right)^2 \equiv N \times \overline{k\left({}_R U_t, {}_0 W_t\right)^2} \quad (76)$$

where  $\overline{k\left({}_R U_t, {}_0 W_t\right)^2}$  denotes the average value of the causal relationships  $k$  squared after  $N$  Bernoulli trials. We re-write the equation above as

$$Z^2 = X^2_N = N \times \overline{k\left({}_R U_t, {}_0 W_t\right)^2} \quad (77)$$

where  $X^2_N$  denotes the chi-squared distribution (also chi-square distribution) with  $N$  degrees of freedom. At the end, it follows that

$$k\left(\overline{R U_t}, \overline{0 W_t}\right) = \sqrt[2]{\frac{X^2}{N}} = \sqrt[2]{\frac{Z^2}{N}} \quad (78)$$

**Quod erat demonstrandum.**

#### 4. Discussion

There is a long tradition of dualism between causality and statistics. For a long time, statistics seemed to exclude causality and vice versa. Especially, due to some quantum mechanical positions (Heisenberg's uncertainty, Bell's theorem, CHSH-Inequality) the deterministic relationship between a cause and its own effect became an impossibility. Hans Reichenbach (1891-1953) comes to a similar conclusion in his own words: "Quantenmechanik zu Zweifeln an der unumschränkten Gültigkeit des Kausalprinzips geführt" [2].

Heisenberg himself states this straightforward. "Weil alle Experimente den Gesetzen der Quantenmechanik und damit der Gleichung (1) unterworfen sind, so wird durch die Quantenmechanik die Ungültigkeit des Kausalgesetzes definitiv festgestellt." [3]. Bohr elaborates on this matter too. Niels Bohr is of the position that "... **physics ... forces us to replace ... causality by ... complementarity**" [4]. The deep conflict between the Copenhagen dominated interpretation of quantum mechanics and the principle of causality might have been one of the major reasons for the sponsors of the Second International Congress for the Unity of Science to organize the Second International Congress for the Unity of Science in Copenhagen (June 21-26, 1936). Indeed, the unity of science is based on the unity of nature and at the end consequently on causality. Causality is the common logical and mathematical foundation for relativity and for quantum theory. In so far, it was necessary and correct that "The Second International Congress for the Unity of Science was to deal primarily with the problem of causality" [5]. But the problem of causality was not solved by the Second International Congress for the Unity of Science at all. The solution of the problem of causality was endangered [6] especially by the contributions of Niels Bohr and his pseudo-scientific dogma of the dualism between quantum mechanics and causality. "Niels Bohr and Philipp Frank were to discuss the problem of causality in physics and biology." [7]. Meanwhile, the quantum mechanical no-go-theorems which excluded the deterministic [8], [9] relationship between cause and effect at quantum level are already refuted [10]-[12]. While the mathematical methodology to extract cause and effect relationship out of (non-) experimental data is already published [13]-[19] and presented to the scientific community [20], this highly original approach gives a new, unknown and exact mathematical derivation of the mathematical formula of the causal relationship  $k$  from a purely mathematical starting point. The mathematical formula of the causal relationship  $k$  is not only of theoretical importance, the same is useful in every day research too.

#### **Inferential statistics and hypothesis (significance) testing**

In hypothesis testing data gathered through an observational study, through an experiment et cetera can show whether the value stated in a null hypothesis ( $H_0$ ) is likely to be true. Similar to a proof by contradiction, we are testing a **null hypothesis ( $H_0$ )** (the 'thesis') because we are of the opinion that the same is wrong. We state what we think is wrong about the null hypothesis in an alternative hypothesis. An **alternative hypothesis ( $H_1$ )** (the 'anti thesis') directly contradicts the null hypothesis ( $H_0$ ) by stating what is wrong about the null hypothesis ( $H_0$ ).

#### **Two sided hypotheses test.**

##### **Conditions.**

Set alpha level = 5%.

Determine the critical value of the causal relationship  $k$  for alpha level 5% =  $k_{\text{critical}}$ .

##### **Claims.**



Null hypothesis (H0):  $k=0$ . (No causal relationship).

Alternative hypothesis (H1):  $k \neq 0$ . (Causal relationship).

**Proof by contradiction. Decision.**

Compute the test statistic i. e.  $k_{\text{Obtained}}$ .

If  $k_{\text{Obtained}}$  (the value of the test statistic) exceeds  $k_{\text{Critical}}$  (the critical value), then **reject** the null hypothesis.

If  $k_{\text{Obtained}}$  (the value of the test statistic) does not exceed  $k_{\text{Critical}}$  (the critical value), then retain the null hypothesis.

Reject the alternative hypothesis (H1).

**Quod erat demonstrandum.**

The *alpha level* (the rejection region of a hypothesis test or the criteria for a decision), the probability of a Type I error (**the level of significance of the test**) is typically set at 5%. The *alpha level* (Type I error) is the probability to reject the null hypothesis (H0) in favor of the alternative hypothesis HA when the null hypothesis (H0) is true. In a non-directional **two-tailed test**, divide the alpha value in half thus that an equal proportion of area is placed in the lower and upper tail. In a directional or **one-tailed test** either the alternative hypothesis is stated as greater than (>) the null hypothesis or the alternative hypothesis is stated as less than (<) the null hypothesis. In general, a one-tailed test makes it easier to reject the null hypothesis while a two-tailed test is more conservative on this account and makes it more difficult to reject a null hypothesis. For this reason, in causal research two-tailed test should be preferred. Now, determine the cutoff value (**critical value**) which defines the boundaries beyond which less than 5% (the alpha value) can be obtained if the null hypothesis is true. In other words, the region beyond the critical value in a hypothesis test is called rejection region. Afterwards, we compute a **test statistic** to determine how likely the causal relationship  $k$  of the sample is, if the causal relationship of the population as stated by the null hypothesis is true. In other words, we select a random sample of a certain size from a population and calculate or measure the causal relationship  $k$  of the sample (test statistic). The mathematical formula of the causal relationship  $k$  of the sample can be used as a test of significance to support or to reject hypotheses/claims based on data gathered. We expect the causal relationship  $k$  of the sample to be equal to the causal relationship  $k$  of the population. The larger the discrepancy or the difference between the causal relationship  $k$  of the sample and the causal relationship of the population, the less likely it is that we could have found that causal relationship  $k$ , if the value of the causal relationship  $k$  of the population is correct.

Especially, there are circumstances where the generally known Pearson's chi-square statistic, uncorrected for continuity (i. e. one degree of freedom) can be used as a statistical test of independence of observations on two binomial variables. The Pearson chi-square statistic, uncorrected for continuity, is calculated as follows:

$$X^2_{\text{Obtained}} \equiv \frac{N \times (a \times d - b \times c) \times (a \times d - b \times c)}{(a + b) \times (c + d) \times (a + c) \times (b + d)} \tag{79}$$

The relationship between cause and effect and Pearson's chi-square statistic is tabularized by the following contingency Table 3.

**Compare** the value of the test statistic, called the obtained value, to the critical value. Under circumstances where the value of a test statistic obtained is in the rejection region (the obtained value exceeds a critical value), we decide to reject the null hypothesis (H0) otherwise, we retain the null hypothesis (H0).

A hypothesis test or an experiment should ensure that it is a test or an experiment of high quality. One possibility to quantify the quality of a hypothesis test or an experiment is the calculation of the power of a

test or the power of an experiment. The probability that a hypothesis test correctly rejects the null hypothesis (H0) when the alternative hypothesis (H1) is true, is called the power of a test. The statistical power, i. e. the probability of rejecting a false null hypothesis, may depend on a number of factors (the sample size (used to detect the effect), the statistical significance (used in the test), the magnitude of the effect et cetera). Increasing sample size, the alpha level and the effect size will increase power. Researchers try to make sure that the power of their study is at least 0.80. The beta ( $\beta$ ) error (Type II error) denotes the probability of retaining a null hypothesis which is actually false. A Type II error is the probability to fail to reject the null hypothesis H0 when the alternative hypothesis HA is true. The statistical power of a test (denoted by  $\underline{\beta}$ ) is equal to  $\underline{\beta} = 1 - \beta$ , thus far decreasing beta error ( $\beta$ ) increases power.

Table 3. The Relationship between Cause and Effect and Pearson's Chi-Square Statistic

Fig.		Effect		
		Yes	No	
Cause	Yes	$a \equiv E({}_R U \cap {}_0 W)$	$b \equiv E({}_\Delta W)$	$a + b \equiv E({}_R U)$
	No	$c \equiv E({}_0 \underline{U})$	$d \equiv E({}_\Delta \underline{U})$	$c + d \equiv E({}_R \underline{U})$
		$a + c \equiv E({}_0 W)$	$b + d \equiv E({}_\Delta W)$	$a + b + c + d \equiv N \equiv E({}_R W)$

Under some assumptions, the theorems above enable us to use the mathematical formula of the causal relationship k for hypothesis testing with the possibility to calculate the p-values, the  $\beta$ -value et cetera even under conditions where the k is not constant from trial to trial. Under these conditions, please recall the relationship  $t_N = \frac{Z}{\sqrt{\frac{X^2_N}{N}}}$ , where  $t_N$  denotes the t-distribution with N degrees of freedom and Z denotes the

Z value.

**Example.**

Helicobacter pylori has been discussed [21] for a long time as being associated with human gastric cancer.

"There is sufficient evidence in humans for the carcinogenicity of infection with Helicobacter pylori...Infection with Helicobacter pylori is carcinogenic to humans (Group 1)." [22].

In several, previous (epidemiologic) studies and meta-analysis it has been reported that there is a close relation between a H. pylori infection of human stomach and human gastric cancer. Still, the cause of human gastric cancer is not identified. Naomi Uemura et al. [23] conducted a long-term, prospective study of N=1526 Japanese patients, 1246 had H. pylori infection and 280 did not (mean follow up 7.8 years, endoscopy at enrollment and then between one and three years after enrollment). None of the uninfected patients developed gastric cancer. Let us show this data in the following Table 4.

Table 4. None of the Uninfected Patients Developed Gastric Cancer

Fig.		Human gastric cancer		
		Yes	No	
Helicobacter pylori infection of human stomach	Yes	36	1210	1246
	No	0	280	280
		36	1490	1526

**Two sided hypotheses test.**

**Conditions.**

Alpha level is 5%.

The two tailed critical Chi square value for alpha level 5% is 5.02388619.

**Claims.**

Null hypothesis (H0):  $k=0$ .

No significant causal relationship between Helicobacter pylori infection of human stomach and human gastric cancer.

Alpha = 5 %.

Alternative hypothesis (H1):  $k \neq 0$ .

Significant causal relationship between Helicobacter pylori infection of human stomach and human gastric cancer.

**Proof by contradiction. Decision.**

Perform an experiment. Gather data et cetera. Compute the test statistic. Based on the study above, the test statistics follows as

$$k \left( \begin{matrix} U_t \\ R \end{matrix} , \begin{matrix} W_t \\ 0 \end{matrix} \right)_{Obtained} = \sqrt[2]{\frac{X^2_N}{N}} \equiv \frac{(1526 \times 36) - (36 \times 1246)}{\sqrt[2]{(1246 \times 280) \times (36 \times 1490)}} = +0,07368483 \quad (80)$$

Or

$$X^2_{Obtained} \equiv \frac{N \times (a \times d - b \times c) \times (a \times d - b \times c)}{(a + b) \times (c + d) \times (a + c) \times (b + d)} = 8,28534801 \quad (81)$$

The value of the test statistic is 8.28534801 and exceeds the critical value 5.02388619. Pearson's chi-square statistic, uncorrected for continuity, is 8.28534801 and equivalent to a P value of 0.00399664. Consequently, we **reject** the null hypothesis and **accept** the alternative hypotheses.

**Helicobacter pylori is the cause of human gastric cancer ( $k = +0,074$ ,  $p$  Value = 0.00399664).**

**The result is significant at  $p < 0.05$ .**

**Quod erat demonstrandum.**

**5. Conclusion**

While there are historically, epistemologically and many other approaches to the problem of the relationship between cause and effect, this publication provides an exact mathematical derivation of the relationship between a cause and its own effect. A new mathematical methodology for making causal inferences on the basis of (non-) experimental data for evaluating causal relationships from (non-) experimental data is presented in the simplest and most intelligible form. Anyone who wishes to elucidate cause effect relationships from (non-) experimental data will find this publication useful. Finally, a unified mathematical and statistical model of the relationship between the cause and the effect is available. As a side effect of this publication, the cause of human gastric cancer is identified.

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