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Without high blood pressure, no coronary artery disease

Research article

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Abstract

Background:

The relation between and high blood pressure and coronary artery disease has been re-investigated by a thought experiment.

Methods:

New statistical methods were used.

Results:

High blood pressure and coronary artery disease are related in a certain manner.

Conclusion:

Without high blood pressure, **no** coronary artery disease.

Keywords: Hypertension; Coronary artery disease; Conditio sine qua on; Cause; Effect; Causal relationship k; Causality; Causation

1. Introduction

Cardiovascular disease (CVD) is a major cause of premature mortality worldwide [1], [2], [3], [4] and includes stroke, heart failure, hypertensive heart disease, rheumatic heart disease, cardiomyopathy, abnormal heart rhythms, congenital heart disease, valvular heart disease, carditis, aortic aneurysms,

[1] Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, Barengo NC, Beaton AZ, Benjamin EJ, Benziger CP, Bonny A, Brauer M, Brodmann M, Cahill TJ, Carapetis J, Catapano AL, Chugh SS, Cooper LT, Coresh J, Criqui M, DeCleene N, Eagle KA, Emmons-Bell S, Feigin VL, Fernández-Solà J, Fowkes G, Gakidou E, Grundy SM, He FJ, Howard G, Hu F, Inker L, Karthikeyan G, Kassebaum N, Koroshetz W, Lavie C, Lloyd-Jones D, Lu HS, Mirijello A, Temesgen AM, Mokdad A, Moran AE, Muntner P, Narula J, Neal B, Ntsekhe M, Moraes de Oliveira G, Otto C, Owolabi M, Pratt M, Rajagopalan S, Reitsma M, Ribeiro ALP, Rigotti N, Rodgers A, Sable C, Shakil S, Sliwa-Hahnle K, Stark B, Sundström J, Timpel P, Tleyjeh IM, Valgimigli M, Vos T, Whelton PK, Yacoub M, Zuhlke L, Murray C, Fuster V; GBD-NHLBI-JACC Global Burden of Cardiovascular Diseases Writing Group. Global Burden of Cardiovascular Diseases and Risk Factors, 1990-2019: Update From the GBD 2019 Study. J Am Coll Cardiol. 2020 Dec 22;76(25):2982-3021. doi: 10.1016/j.jacc.2020.11.010. Erratum in: J Am Coll Cardiol. 2021 Apr 20;77(15):1958-1959. PMID: 33309175; PMCID: PMC7755038.

[2] GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020 Oct 17;396(10258):1204-1222. doi: 10.1016/S0140-6736(20)30925-9. Erratum in: Lancet. 2020 Nov 14;396(10262):1562. PMID: 33069326; PMCID: PMC7567026.

[3] Mensah GA, Roth GA, Fuster V. The Global Burden of Cardiovascular Diseases and Risk Factors: 2020 and Beyond. J Am Coll Cardiol. 2019 Nov 19;74(20):2529-2532. doi: 10.1016/j.jacc.2019.10.009. PMID: 31727292.

[4] GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020 Oct 17;396(10258):1204-1222. doi: 10.1016/S0140-6736(20)30925-9. Erratum in: Lancet. 2020 Nov 14;396(10262):1562. PMID: 33069326; PMCID: PMC7567026.

peripheral artery disease, thromboembolic disease, venous thrombosis et cetera and coronary artery disease (CAD). Coronary artery disease, whether clinically symptomatic or asymptomatic, is a chronic, most often progressive pathological process of epicardial arteries. An acute atherothrombotic event caused by plaque erosion or plaque rupture might result in various clinical presentations of CAD, such as either acute coronary syndromes (ACS) or chronic coronary syndromes (CCS). Today, among the many key cardiovascular risk factors for coronary artery disease are dyslipidaemia or high-fat (LDL cholesterol) dairy foods [5], [6], [7], type 2 diabetes mellitus [8], smoking, unhealthy alcohol intake and several other (modifiable lifestyle) factors, family history of CVD, and high blood pressure (BP) [9], [10]. Hypertension has been defined in the year 1999 by WHO/ISH Hypertension Guidelines (see Who, 1999) as systolic blood pressure (SBP) $\geq$ 140 mmHg (18.7 kPa) and/or diastolic blood pressure (DBP) $\geq$ 90 mmHg (12.0 kPa). To date, hypertension is understood as a disease which is caused by a combination of various environmental and genetic factors. As an example, Jason DeGuire et al. [11] published data of high blood pressure prevalence among adults in Canada (see table 1). Sungwa et

Table 1. High blood pressure in Canada.

Average systolic and diastolic blood pressure (mm Hg) by age group, Canada, 2012-2015

Age group	n	Systolic	Std. Dev.	Diastolic	Std. Dev.
20 to 39 years	2098	106	11	70	9
40 to 59 years	2141	114	15	74	10
50 to 69 years	1344	120	16	73	9
70 to 79 years	711	126	18	70	10

al. [12] conducted a cross sectional study involving 742 children aged 6 to 16 years in Mwanza region

[5] O'Sullivan TA, Hafekost K, Mitrou F, Lawrence D. Food sources of saturated fat and the association with mortality: a meta-analysis. Am J Public Health. 2013 Sep;103(9):e31-42. doi: 10.2105/AJPH.2013.301492. Epub 2013 Jul 18. PMID: 23865702; PMCID: PMC3966685.

[6] Chowdhury R, Warnakula S, Kunutsor S, Crowe F, Ward HA, Johnson L, Franco OH, Butterworth AS, Forouhi NG, Thompson SG, Khaw KT, Mozaffarian D, Danesh J, Di Angelantonio E. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. Ann Intern Med. 2014 Mar 18;160(6):398-406. doi: 10.7326/M13-1788. Erratum in: Ann Intern Med. 2014 May 6;160(9):658. PMID: 24723079.

[7] de Souza RJ, Mente A, Maroleanu A, Cozma AI, Ha V, Kishibe T, Uleryk E, Budylowski P, Schünemann H, Beyene J, Anand SS. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. BMJ. 2015 Aug 11;351:h3978. doi: 10.1136/bmj.h3978. PMID: 26268692; PMCID: PMC4532752.

[8] Gao D, Ning N, Wang C, Wang Y, Li Q, Meng Z, Liu Y, Li Q. Dairy products consumption and risk of type 2 diabetes: systematic review and dose-response meta-analysis. PLoS One. 2013 Sep 27;8(9):e73965. doi: 10.1371/journal.pone.0073965. PMID: 24086304; PMCID: PMC3785489.

[9] He J, Ogden LG, Bazzano LA, Vupputuri S, Loria C, Whelton PK. Risk factors for congestive heart failure in US men and women: NHANES I epidemiologic follow-up study. Arch Intern Med. 2001 Apr 9;161(7):996-1002. doi: 10.1001/archinte.161.7.996. PMID: 11295963.

[10] Weber T, Lang I, Zweiker R, Horn S, Wenzel RR, Watschinger B, Slany J, Eber B, Roithinger FX, Metzler B. Hypertension and coronary artery disease: epidemiology, physiology, effects of treatment, and recommendations : A joint scientific statement from the Austrian Society of Cardiology and the Austrian Society of Hypertension. Wien Klin Wochenschr. 2016 Jul;128(13-14):467-79. doi: 10.1007/s00508-016-0998-5. Epub 2016 Jun 9. PMID: 27278135.

[11] DeGuire J, Clarke J, Rouleau K, Roy J, Bushnik T. Blood pressure and hypertension. Health Rep. 2019 Feb 20;30(2):14-21. doi: 10.25318/82-003-x201900200002. PMID: 30785635.

[12] Sungwa EE, Kibona SE, Dika HI, Laisser RM, Gemuhay HM, Kabalimu TK, Kidenya BR. Prevalence and factors that are associated with elevated blood pressure among primary school children in Mwanza Region, Tanzania. Pan Afr Med J. 2020 Nov 30;37:283. doi: 10.11604/pamj.2020.37.283.21119. PMID: 33654510; PMCID: PMC7896535.

(Tanzania) from June to August 2019. The distribution of blood pressure for boys and girls at different age groups is illustrated by table 2.

Table 2. High blood pressure in Children, Tanzania.

Age	Blood pressure (mmHg)			
(Years)	Boys Systolic	Diastolic	Girls Systolic	Diastolic
6	103	61	112	64
7	104	61	107	66
8	110	63	107	63
9	109	63	112	68
10	111	65	114	69
11	113	66	111	64
12	109	66	116	64
13	113	65	114	61
14	109	63	117	64
15	113	63	118	73
16	116	67	114	66

These data will are only of exemplary use for our further investigations. More and more, evidence is increasing that high blood pressure is the strongest or one of the strongest [13] of all modifiable risk factor for all clinical manifestations of coronary artery disease. [14] However, the major efforts undertaken over the years have not been able to achieve a sustainable answer to the question whether arterial hypertension is a cause or the cause of coronary artery disease. [15]

2. Material and methods

Scientific knowledge and objective reality are more than only interrelated. It cannot be repeated often enough that objective reality or processes of objective reality is the foundation of any scientific knowledge. Our human experience teaches us however that seen by light, grey is never merely simply grey, and looked at from different angles, many paths may lead to climb up a certain mountain. In general, it is appropriate to ensure as much as possible a broader consideration of a research question and to take into account the different facets and viewpoints of an issue investigated in order to reach a goal.

[13] Kjeldsen SE. Hypertension and cardiovascular risk: General aspects. Pharmacol Res. 2018 Mar;129:95-99. doi: 10.1016/j.phrs.2017.11.003. Epub 2017 Nov 7. PMID: 29127059.

[14] McInnes GT. Hypertension and coronary artery disease: cause and effect. J Hypertens Suppl. 1995 Aug;13(2):S49-56. doi: 10.1097/00004872-199508001-00008. PMID: 8576788.

[15] Fuchs FD, Whelton PK. High Blood Pressure and Cardiovascular Disease. Hypertension. 2020 Feb;75(2):285-292. doi: 10.1161/HYPERTENSIONAHA.119.14240. Epub 2019 Dec 23. PMID: 31865786.

2.1. Material

2.1.1. Study design and bias

Systematic observation and experimentation, inductive and deductive reasoning are essential for any formation and testing of hypotheses and theories about the natural world. In one way or another, logically and mathematically sound scientific methods and concepts are crucial constituents of any scientific progress. When all goes well, different scientists at different times and places using the same scientific methodology should be able to generate the same scientific knowledge. However, more than half (52%) of scientists surveyed believe that studies do not successfully reproduce sufficiently similar or the same results as the original studies (Baker, 2016). In a very large study on publication bias in meta-analyses, Kicinski et al. (Kicinski et al., 2015) found evidence of publication bias even in systematic reviews. Therefore, a careful re-evaluation of the study/experimental design, the statistical methods and other scientific means which underpin scientific inquiry and research goals appears to be necessary once and again. While it is important to recognise the shortcoming of today's science, one issue which has shaped debates over studies published is the question: **has a study really measured what it set out to**? Even if studies carried out can vary greatly in detail, the data from the studies itself provide information about the credibility of the data.

2.1.1.1. Index of unfairness (IOU)

Definition 2.1 (Index of unfairness).

The index of unfairness (Barukčić, 2019e) (IOU) is defined as

$$p\left(IOU\left(A,B\right)\right) \equiv Absolute\left(\left(\frac{A+B}{N}\right)-1\right) \tag{1}$$

Under ideal conditions, it is desirable that an appropriate study design is able to assure as much as possible an index of unfairness (see Barukčić, 2019e) of p(IOU) = 0. In point of fact, against the background of lacking enough experience with the use of p(IOU), a p(IOU) up to 0.25 could be of use too. Especially under conditions where **a necessary condition relationship or a sufficient condition relationship** is tested, an index of unfairness is of use to prove whether sample data obtained are biased and to what extent.

Table 3. The quality of data (see Barukčić, 2019e, p. 25)

p(IOU)	Quality of study design
$0 < p(IOU) \leq 0,25$	Unfair study design
$0,25 < p(IOU) \leq 0,5$	Very unfair study design
$0,5 < p(IOU) \leq 0,75$	Highly unfair study design
$0,75 < p(IOU) \leq 1,0$	Extremely unfair study design

2.1.1.2. Index of independence (IOI)

Definition 2.2 (**Index of independence**).

The index of independence(Barukčić, 2019d) (IOI) is defined as

$$p\left(IOI\left(A,\underline{B}\right)\right) \equiv Absolute\left(\left(\frac{A+\underline{B}}{N}\right)-1\right) \tag{2}$$

The index of independence(see Barukčić, 2019d) has the potential to indicate the extent to which the study design of a study could be biased.

Table 4. The quality of data (see Barukčić, 2019e, p. 25)

p(IOI)	Quality of study design
$0 < p(IOI) \leq 0,25$	Unfair study design
$0,25 < p(IOI) \leq 0,5$	Very unfair study design
$0,5 < p(IOI) \leq 0,75$	Highly unfair study design
$0,75 < p(IOI) \leq 1,0$	Extremely unfair study design

Under ideal conditions, a study design which aims to prove **an exclusion relationship or a causal relationship** should assure as much as possible a p(IOI) = 0. However, once again, against the background of lacking enough experience with the use of p(IOI), sample data with a p(IOI) up to 0.25 are of use too. Today, most double-blind placebo-controlled studies are based on the demand that **p(IOU) = p(IOI)** while the value of p(IOU) of has been widely neglected. Such an approach leads to unnecessary big sample sizes, the increase of cost, the waste of time and, most importantly of all, to epistemological systematically biased sample data and conclusions drawn. A change appears to be necessary.

2.1.2. Experimental methods

In view of the many and sometimes each other excluding results of scientific investigations, a theoretical appreciation of scientific statistical, experimental, study design and other proof methods et cetera becomes pressing. In short, once again, let us highlight Albert Einstein's position. In a letter to the student J. S. Switzer on April 23rd, 1953, **Albert Einstein** gets right to the point.

> "Development of Western **science** is based on two great achievements: the invention of the **formal logic**al system (in Euclidean geometry) by the Greek philosophers, and the discovery of the possibility to find out **causal relationships** by systematic **experiment** (during the Renaissance). "
>
> (Hu, 2005)

In other words, according to Einstein experiments and other generally accepted, scientific proof methods which are logically consistent constitutes our ground of scientific evidence which itself might help us to refute or to confirm scientific theories even if authors respond many times by different and sometimes inappropriate counter-measures when some theorems or theories are falsified by a formal proof or by observations et cetera. Even though the pressure by which we are sometimes forced to believe in different scientific publications or theories, although there are already predictively superior rivals to turn to may be very high, a reliable and clear scientific methodology should be able to help us to decide what is true and what is false and to assure a kind of **a demarcation line between science and non-science** (see also Popper, Karl Raimund, 2002, p. 429). For these reasons and others, scientific proof methods are equally necessary for scientific knowledge and the demarcation line between non-science i.e. ((justified) personal) belief and exceedingly clear and well-verified scientific knowledge and at the end between ideology and science. We are allowed to consider that it is more than unsatisfactory if principles of scientific methodology and inquiry (Barukčić, 2019c) are equally a source of justification of wishful thinking, of scientific mysteries and of other errors of (human) reasoning in science, upon which sometimes whole theories rest. In this context it is incomprehensible, nay irritating and aggravating in the extreme, when scientific mistakes are created unconsciously and unintentionally i.e. by carelessness, superficiality, lack of methodological skill or other factors not caused by some inappropriate (ideological and other) motives of an author himself. However, in view of single publications it cannot be excluded that the primary motivation of an author while presenting some arguments in his own, unique and many times very complicated way is more to trick the reader into agreement and less to provide a long-lasting and reliable contribution to scientific progress. In point of fact, it is inexcusable if errors in reasoning are created intentionally in order to deceive a single reader or the scientific community as such. The high honour which scientist deserve implies above all the need to continue to meet the expectations with respect to a transparent, a methodological and a very precise scientific work. Therefore and apart from the permanent and intrinsic duty of every author to detect technical or other errors (of human reasoning) in his own publications and the publications of other authors and voluntarily to correct those errors which cannot be tolerated at all as soon as possible, there will always be the need to rely on different **scientific (proof) methods** (Barukčić, 2019c) including experiments which are able to identify among other cherished belief in science and to help us that logically inconsistent scientific positions, statement, theorems et cetera cannot be rescued from trouble any longer. In addition to that, the methods of investigation and the knowledge achieved especially in the natural sciences relies to a very great extent on mathematics too. However, objects studied in mathematics are not all the time located in space and time and the methods of investigation of mathematics differ markedly from the methods of investigation in the natural sciences. For these reasons and even if mathematics as such appears to enjoy a special esteem within the scientific community and is regarded more than above all other sciences due to the common belief that the laws or mathematics are absolutely certain and indisputable, are we allowed to regard today's mathematics as a science next to disciplines such as classical logic, physics and other? There may exit several distinct ways how the relationship between mathematics, other sciences and objective reality can be analysed. However, first and after all and in a slightly different way, **today's mathematics itself is more or less a product of human thought and mere human imagination** and belongs as such to a world of human thought and mere human imagination. In point of fact, **human thought and mere human imagination which produces the laws of mathematics is able to produce erroneous or incorrect results too** with the principal consequence that even mathematics or **mathematical theorems, rules or other results valid since thousands of years are in constant danger of being overthrown by newly discovered facts**. In addition to that, acquiring general scientific knowledge by deduction from basic principles, does not guarantee correct results automatically if the basic principles are not compatible with objective reality or classical logic as such. In other words, if **mathematics** has to be regarded more than science and less than **a religion formulated by numbers, definitions et cetera**, the same mathematics must be open to a potential revision. In general, and from a theoretical point of view, a mathematics or a mathematical theorem characterized by denial(ism) and resistance to the facts which do not offer itself to a potential refutation would not allow us to distinguish scientific knowledge from its look-alike. From a practical point of view, it is not enough to (mathematically) define how objective reality has to be, even **mathematics itself must discover how nature really is**. Due to the high status of science in present-day society, even **mathematics itself must pass the test of reality and does not stand above all and outside of reality**. The principles of mathematics should be logically compatible and receive strong experimental confirmation as much as possible. In this context, objective reality or practical or theoretical experiments and other check methods as such are a demarcation line between science and (sometimes fantastical) non-science. A very precise demarcation between non-science and science is possible and necessary for many theoretical and practical reasons, and especially in order to clarify or to identify what is **pure denialism or dogmatic resistance to the facts**. From a practical point of view, various proposals have been put forward which criteria of demarcation between science and non-science should be applied, including **modus tollens** as advocated especially by Karl Popper. Ever investigative and getting to the heart of matters, **Karl Popper** himself summarizes it very accurately.

> **“... it is possible by means of purely deductive inferences (with the help of the modus tollens of classical logic) to argue from the truth of singular statements to the falsity of universal statements. ”**
>
> (see Popper, Karl Raimund, 1935, p. 19)

However, **modus inversus** is an additional approach to solve the problem of demarcation between science and non-science.

2.1.2.1. Thought experiments Thought experiments (Cargile et al., 1994) are valid devices of the scientific (Sorensen, 1999) investigation [16], [17], [18] with the potential to play a central role in human medicine, natural sciences, in mathematics, in philosophy and in other sciences too. In point of fact, the saga of thought experiments is already going back at least two and a half millennia and has been practised even since the time of the Pre-Socratics (Rescher, 2005). It might be reasonably reiterated that there is still no standard definition for thought experiments, while the term is loosely characterized. More precisely, despite this shortcoming and deficiency, it is particularly important to underline the fact that thought experiments can be taken to provide evidence in favour of or against a (mathematical) theorem, a theory et cetera. Meanwhile, a general acceptance of the importance of thought experiments

[16] Eren Simsek. On thought experiments: Mach and Einstein (Part I), physics.hist-ph, arXiv:2003.04764, 2020

[17] Bunzl, M. The logic of thought experiments. Synthese 106, 227–240 (1996). https://doi.org/10.1007/BF00413701

[18] MacQueen, H., & Reid, D. (2013). Fraud or Error: A Thought Experiment? Edinburgh Law Review, 17(3), 343-69. https://doi.org/10.3366/elr.2013.0171

can be found in almost all disciplines of scientific inquiry. Thought experiments are conducted for diverse reasons in a variety of areas and are very common. A surprisingly large majority of impressive examples of thought experiments can be found especially in physics among some of its most brilliant practitioners are Galileo, Descartes, Newton and Leibniz (Sorensen, 1999) and other too.

2.1.3. Statistical methods

The probability of the exclusion (Barukčić, 2021c) relationship(see also Barukčić, 2021a) p(EXCL) has been calculated and tested for statistical significance. The chi-square goodness of fit test with one degree of freedom has been used to test whether the sample data published fit a certain theoretical distribution in the population. Additionally, the P Value has been calculated approximately (see also Barukčić, 2019f). The causal relationship k (Barukčić, 2016b, 2020a, 2021c) has been calculated to evaluate a possible causal relationship between the events. The hyper-geometric (Fisher, 1922, Gonin, 1936, Huygens and van Schooten, 1657, Pearson, 1899) distribution (HGD) has been used to test the one-sided significance of the causal relationship k. Bringing different studies together for analysing them or doing a meta-analysis is not without problems. Due to several reasons, there is variability in the data of the studies and there will be differences found. Usually, the heterogeneity among the studies is assessed through I^2 statistics [19], [20], [21]. Under usual circumstances, an I^2 value of 25%, 50% and 75% are regarded as low, moderate and high heterogeneity[22]. In this publication, the study (design) bias and the heterogeneity among the studies has been controlled by IOI, the index of independence (Barukčić, 2019d) and IOU, the index of unfairness (Barukčić, 2019e). All the data were analysed using MS Excel (Microsoft Corporation, USA).

P values less than 0.05 were considered statistically significant.

2.2. *Methods*

Definitions should help us to provide and assure a systematic approach to a scientific issue. It also goes without the need of further saying that a definition as such need to be logically consistent and correct.

2.2.1. Bernoulli distribution

A single event distribution is more or less a discrete probability distribution of any random variable X which takes a certain (observer independent) single value X_t at a **Bernoulli trial** (Uspensky, 1937,

[19] Cochran WG. The combination of estimates from different experiments. Biometrics 1954; 10(1): 101-29.

[20] Higgins JP, Thompson SG. Quantifying heterogeneity in a meta-analysis. Stat Med. 2002 Jun 15;21(11):1539-58. doi: 10.1002/sim.1186. PMID: 12111919.

[21] Higgins JP, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. BMJ. 2003 Sep 6;327(7414):557-60. doi: 10.1136/bmj.327.7414.557. PMID: 12958120; PMCID: PMC192859.

[22] Higgins JP, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. BMJ. 2003 Sep 6;327(7414):557-60. doi: 10.1136/bmj.327.7414.557. PMID: 12958120; PMCID: PMC192859.

p. 45) (period of time) t with the probability p(X_t). The same random variable X takes a certain single anti value $\underline{X}_t$ at a Bernoulli trial (period of time) t with the probability 1-p(X_t). There are conditions in nature where a random variable X can take only the values either +0 or +1 (see Birnbaum, 1961). Under these conditions, the random variable X takes the value 1 with probability p(X_t = +1) and the value 0 with probability $q(X_t = +0) = 1 - p(X_t = +1)$ while the single event distribution passes over into the **Bernoulli distribution**, named after Swiss mathematician Jacob Bernoulli (Bernoulli, 1713). Less formally, many times, the Bernoulli distribution is represented by a (possibly not biased) coin toss where 1 and 0 would represent 'heads'and 'tails'(or vice versa), respectively. However, the relationship between random variables (Gosset, 1914) can be investigated by many (Gosset, 1908) methods, including the tools of probability theory, too.

Definition 2.3 (Two by two table of single event random variables).

The two by two or contingency table which has been introduced by Karl Pearson (Pearson, 1904b) in 1904 harbours still a large variety of topics and debates. Central to this is the problem to apply the laws of classical logic on data sets, which concerns the justification of inferences which extrapolate from sample data to general facts. Nevertheless, a contingency table is still an appropriate theoretical model too for studying the relationships between random variables, including *Bernoulli (Bernoulli, 1713) (i.e. +0/+1) distributed random variables* existing or occurring at the same *Bernoulli trial* (Uspensky, 1937) (period of time) t.

In this context, let a random variable A at the *Bernoulli trial* (Uspensky, 1937) (period of time) t, denoted by A_t, indicate a risk factor, a condition, a cause et cetera and occur or exist with the probability p(A_t) at the *Bernoulli trial* (Uspensky, 1937) (period of time) t. Let E(A_t) denote the expectation value of A_t. In general it is

$$p(A_t) \equiv p(a_t) + p(b_t) \tag{3}$$

The expectation value E(A_t) follows as

$$\begin{aligned} E(A_t) &\equiv A_t \times p(A_t) \\ &\equiv A_t \times (p(a_t) + p(b_t)) \\ &\equiv (A_t \times p(a_t)) + (A_t \times p(b_t)) \\ &\equiv E(a_t) + E(b_t) \end{aligned} \tag{4}$$

Under conditions of +0/+1 distributed Bernoulli random variables it is

$$\begin{aligned} E(A_t) &\equiv A_t \times p(A_t) \\ &\equiv (+0+1) \times p(A_t) \\ &\equiv p(A_t) \\ &\equiv p(a_t) + p(b_t) \end{aligned} \tag{5}$$

Furthermore, it is

$$p(\underline{A}_t) \equiv p(c_t) + p(d_t) \equiv (1 - p(A_t)) \tag{6}$$

The expectation value $E(\underline{A}_t)$ is given as

$$\begin{aligned} E\left(\underline{A}_t\right) &\equiv A_t \times \left(1 - p\left(A_t\right)\right) \\ &\equiv A_t \times \left(p\left(c_t\right) + p\left(d_t\right)\right) \\ &\equiv \left(A_t \times p\left(c_t\right)\right) + \left(A_t \times p\left(d_t\right)\right) \\ &\equiv E\left(c_t\right) + E\left(d_t\right) \end{aligned} \tag{7}$$

Under conditions of +0/+1 distributed Bernoulli random variables we obtain

$$\begin{aligned} E\left(\underline{A}_t\right) &\equiv A_t \times \left(1 - p\left(A_t\right)\right) \\ &\equiv \left(+0 + 1\right) \times \left(1 - p\left(A_t\right)\right) \\ &\equiv \left(1 - p\left(A_t\right)\right) \\ &\equiv p\left(c_t\right) + p\left(d_t\right) \end{aligned} \tag{8}$$

Let a random variable B at the *Bernoulli trial* (Uspensky, 1937) (period of time) t, denoted by B_t, indicate an outcome, a conditioned, an effect et cetera and occur or exist with the probability $p(B_t)$ at the *Bernoulli trial* (Uspensky, 1937) (period of time) t. Let $E(B_t)$ denote the expectation value of B_t. In general it is

$$p\left(B_t\right) \equiv p\left(a_t\right) + p\left(c_t\right) \tag{9}$$

The expectation value $E(B_t)$ is given by the equation

$$\begin{aligned} E\left(B_t\right) &\equiv B_t \times p\left(B_t\right) \\ &\equiv B_t \times \left(p\left(a_t\right) + p\left(c_t\right)\right) \\ &\equiv \left(B_t \times p\left(a_t\right)\right) + \left(B_t \times p\left(c_t\right)\right) \\ &\equiv E\left(a_t\right) + E\left(c_t\right) \end{aligned} \tag{10}$$

Under conditions of +0/+1 distributed Bernoulli random variables it is

$$\begin{aligned} E\left(B_t\right) &\equiv B_t \times p\left(B_t\right) \\ &\equiv \left(+0 + 1\right) \times p\left(B_t\right) \\ &\equiv p\left(B_t\right) \\ &\equiv p\left(a_t\right) + p\left(c_t\right) \end{aligned} \tag{11}$$

Furthermore, it is

$$p\left(\underline{B}_t\right) \equiv p\left(b_t\right) + p\left(d_t\right) \equiv \left(1 - p\left(B_t\right)\right) \tag{12}$$

The expectation value $E(\underline{B}_t)$ is given by the equation

$$\begin{aligned} E\left(\underline{B}_t\right) &\equiv B_t \times \left(1 - p\left(B_t\right)\right) \\ &\equiv B_t \times \left(p\left(b_t\right) + p\left(d_t\right)\right) \\ &\equiv \left(B_t \times p\left(b_t\right)\right) + \left(B_t \times p\left(d_t\right)\right) \\ &\equiv E\left(b_t\right) + E\left(d_t\right) \end{aligned} \tag{13}$$

Under conditions of +0/+1 distributed Bernoulli random variables it is

$$
\begin{aligned}
E\left(\underline{B}_{\mathrm{t}}\right) &\equiv B_{\mathrm{t}} \times \left(1 - p\left(B_{\mathrm{t}}\right)\right) \\
&\equiv (+0+1) \times \left(1 - p\left(B_{\mathrm{t}}\right)\right) \\
&\equiv \left(1 - p\left(B_{\mathrm{t}}\right)\right) \\
&\equiv p\left(b_{\mathrm{t}}\right) + p\left(d_{\mathrm{t}}\right)
\end{aligned}
\tag{14}
$$

Let $p(a_t)= p(A_t \wedge B_t)$ denote the joint probability distribution of A_t and B_t at the same Bernoulli trial (period of time) t. In general, it is

$$
\begin{aligned}
E\left(a_{\mathrm{t}}\right) &\equiv E\left(A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \\
&\equiv \left(A_{\mathrm{t}} \times B_{\mathrm{t}}\right) \times p(A_{\mathrm{t}} \wedge B_{\mathrm{t}}) \\
&\equiv \left(A_{\mathrm{t}} \times B_{\mathrm{t}}\right) \times p(a_{\mathrm{t}})
\end{aligned}
\tag{15}
$$

Under conditions of +0/+1 distributed Bernoulli random variables, it is

$$
\begin{aligned}
E\left(a_{\mathrm{t}}\right) &\equiv E\left(A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \\
&\equiv \left(A_{\mathrm{t}} \times B_{\mathrm{t}}\right) \times p(A_{\mathrm{t}} \wedge B_{\mathrm{t}}) \\
&\equiv \left((+0+1) \times (+0+1)\right) \times p(A_{\mathrm{t}} \wedge B_{\mathrm{t}}) \\
&\equiv p(A_{\mathrm{t}} \wedge B_{\mathrm{t}}) \\
&\equiv p\left(a_{\mathrm{t}}\right)
\end{aligned}
\tag{16}
$$

Let $p(b_t)= p(A_t \wedge \neg B_t)$ denote the joint probability distribution of A_t and not B_t at the same Bernoulli trial (period of time) t. In general, it is

$$
\begin{aligned}
E\left(b_{\mathrm{t}}\right) &\equiv E\left(A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}\right) \\
&\equiv \left(A_{\mathrm{t}} \times \neg B_{\mathrm{t}}\right) \times p(A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}) \\
&\equiv \left(A_{\mathrm{t}} \times \neg B_{\mathrm{t}}\right) \times p(b_{\mathrm{t}})
\end{aligned}
\tag{17}
$$

Under conditions of +0/+1 distributed Bernoulli random variables, it is

$$
\begin{aligned}
E\left(b_{\mathrm{t}}\right) &\equiv E\left(A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}\right) \\
&\equiv \left(A_{\mathrm{t}} \times \neg B_{\mathrm{t}}\right) \times p(A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}) \\
&\equiv \left((+0+1) \times (+0+1)\right) \times p(A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}) \\
&\equiv p(A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}) \\
&\equiv p\left(b_{\mathrm{t}}\right)
\end{aligned}
\tag{18}
$$

Let $p(c_t)= p(\neg A_t \wedge B_t)$ denote the joint probability distribution of not A_t and B_t at the same Bernoulli trial (period of time) t. In general, it is

$$
\begin{aligned}
E\left(c_{\mathrm{t}}\right) &\equiv E\left(\neg A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \\
&\equiv \left(\neg A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \times p\left(\neg A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \\
&\equiv \left(\neg A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \times p(c_{\mathrm{t}})
\end{aligned}
\tag{19}
$$

Under conditions of +0/+1 distributed Bernoulli random variables, it is

$$
\begin{aligned}
E\left(c_{\mathrm{t}}\right) &\equiv E\left(\neg A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \\
&\equiv \left(\neg A_{\mathrm{t}} \times B_{\mathrm{t}}\right) \times p\left(\neg A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \\
&\equiv \left(\left(+0+1\right) \times \left(+0+1\right)\right) \times p\left(\neg A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \\
&\equiv p\left(\neg A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \\
&\equiv p\left(c_{\mathrm{t}}\right)
\end{aligned}
\tag{20}
$$

Let $p(d_t) = p(\neg A_t \wedge \neg B_t)$ denote the joint probability distribution of not A_t and not B_t at the same Bernoulli trial (period of time) t. In general, it is

$$
\begin{aligned}
E\left(d_{\mathrm{t}}\right) &\equiv E\left(\neg A_{\mathrm{t}} \times \neg B_{\mathrm{t}}\right) \\
&\equiv \left(\neg A_{\mathrm{t}} \times \neg B_{\mathrm{t}}\right) \times p\left(\neg A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}\right) \\
&\equiv \left(\neg A_{\mathrm{t}} \times \neg B_{\mathrm{t}}\right) \times p\left(d_{\mathrm{t}}\right)
\end{aligned}
\tag{21}
$$

Under conditions of +0/+1 distributed Bernoulli random variables, it is

$$
\begin{aligned}
E\left(d_{\mathrm{t}}\right) &\equiv E\left(\neg A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}\right) \\
&\equiv \left(\neg A_{\mathrm{t}} \times \neg B_{\mathrm{t}}\right) \times p\left(\neg A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}\right) \\
&\equiv \left(\left(+0+1\right) \times \left(+0+1\right)\right) \times p\left(\neg A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}\right) \\
&\equiv p\left(\neg A_{\mathrm{t}} \wedge \neg B_{\mathrm{t}}\right) \\
&\equiv p\left(d_{\mathrm{t}}\right)
\end{aligned}
\tag{22}
$$

In general, it is

$$
p\left(a_{\mathrm{t}}\right) + p\left(b_{\mathrm{t}}\right) + p\left(c_{\mathrm{t}}\right) + p\left(d_{\mathrm{t}}\right) \equiv +1 \tag{23}
$$

Table 5 provide us with an overview of the definitions above.

Table 5. The two by two table of Bernoulli random variables

		Conditioned B_t		
		TRUE	FALSE	
Condition	TRUE	$p(a_t)$	$p(b_t)$	$p(A_t)$
A_t	FALSE	$p(c_t)$	$p(d_t)$	$p(\underline{A}_t)$
		$p(B_t)$	$p(\underline{B}_t)$	+1

In our understanding, it is

$$
p\left(B_{\mathrm{t}}\right) + p\left(\Lambda_{\mathrm{t}}\right) \equiv p\left(a_{\mathrm{t}}\right) + p\left(c_{\mathrm{t}}\right) + p\left(\Lambda_{\mathrm{t}}\right) \equiv p\left(a_{\mathrm{t}}\right) + p\left(b_{\mathrm{t}}\right) \equiv p\left(A_{\mathrm{t}}\right) \tag{24}
$$

or

$$
p\left(c_{\mathrm{t}}\right) + p\left(\Lambda_{\mathrm{t}}\right) \equiv p\left(b_{\mathrm{t}}\right) \tag{25}
$$

Under conditions of Einstein's general theory of relativity, Λ denotes the Einstein cosmological (Einstein, 1917) 'constant '.

2.2.2. Binomial random variables

The binomial distribution (see Cramér, 1937) with parameters n and p has been developed by the Swiss mathematician Jakob Bernoulli (1655-1705) in a proof published in his 1713 book Ars Conjectandi (see Bernoulli, 1713) Part 1. In probability theory and statistics, the probability of getting exactly k successes in n independent Bernoulli trials is given by the probability mass function as

$$p\left(X_{\mathrm{t}}=k\right) \equiv \binom{n}{k} \cdot p^{k} \cdot q^{n-k} \tag{26}$$

is $\binom{n}{k} = \frac{n!}{k!(n-k)!}$ the binomial coefficient while the cumulative distribution function is given as

$$p\left(X_{\mathrm{t}} \leq k\right) \equiv 1-p\left(X_{\mathrm{t}}>k\right) \equiv \sum_{t=0}^{k}\binom{n}{t} \cdot p^{t} \cdot q^{n-t} \tag{27}$$

or as

$$p\left(X_{\mathrm{t}}>k\right) \equiv 1-p\left(X_{\mathrm{t}} \leq k\right) \equiv 1-\sum_{t=0}^{k}\binom{n}{t} \cdot p^{t} \cdot q^{n-t} \tag{28}$$

Furthermore, it is

$$p\left(X_{\mathrm{t}}<k\right) \equiv 1-p\left(X_{\mathrm{t}} \geq k\right) \equiv \sum_{t=0}^{k-1}\binom{n}{t} \cdot p^{t} \cdot q^{n-t} \tag{29}$$

or

$$p\left(X_{\mathrm{t}} \geq k\right) \equiv 1-p\left(X_{\mathrm{t}}<k\right) \equiv 1-\sum_{t=0}^{k-1}\binom{n}{t} \cdot p^{t} \cdot q^{n-t} \tag{30}$$

The binomial distribution is the mathematical foundation of a binomial test. The random variable X_{t} is counting for different things. The discrete geometric (see Feller, 1950, p. 61) distribution describes under certain circumstances the number of Bernoulli trials needed to get one success. The probability that the first occurrence of success requires k independent trials, each with success probability p, is given by the equation

$$p\left(X_{\mathrm{t}}=k\right) \equiv p \cdot q^{k-1} \tag{31}$$

The negative (see Fisher, 1941, Haldane, 1941) binomial probability is a discrete probability distribution which defines the number of successes (k) in a sequence of independent and identically distributed Bernoulli trials (n) before a specified (non-random) number of failures (denoted r) occurs. The probability mass function of the negative binomial distribution is

$$p\left(X_{\mathrm{t}}=r\right) \equiv \binom{k+r-1}{k-1} p^{k} \cdot q^{r} \tag{32}$$

where k is the number of successes, r is the number of failures, and p is the probability of success.

Definition 2.4 (Expectation value and variance of a binomial random variable).

The variance(see Pearson, 1904a, p. 66) of the binomial distribution with parameters n, the number of independent experiments each asking a yes–no question and p, the probability of a single event, is defined in contrast to Pearson (see Barukčić, 2022c) as

$$\sigma(X_t)^2 \equiv N \times N \times p(X_t) \times (1 - p(X_t)) \tag{33}$$

Definition 2.5 (**Two by two table of Binomial random variables**).

Let a, b, c, d, A, $\underline{A}$, B, and $\underline{B}$ denote expectation values. Under conditions where *the probability of an event, an outcome, a success et cetera is **constant** from Bernoulli trial to Bernoulli trial t*, it is

$$\begin{aligned} A &= N \times E(A_t) \\ &\equiv N \times (A_t \times p(A_t)) \\ &\equiv N \times (p(A_t) + p(B_t)) \\ &\equiv N \times p(A_t) \end{aligned} \tag{34}$$

and

$$\begin{aligned} B &= N \times E(B_t) \\ &\equiv N \times (B_t \times p(B_t)) \\ &\equiv N \times (p(A_t) + p(c_t)) \\ &\equiv N \times p(B_t) \end{aligned} \tag{35}$$

where N might denote the population or even the sample size. Furthermore, it is

$$a \equiv N \times (E(A_t)) \equiv N \times (p(A_t)) \tag{36}$$

and

$$b \equiv N \times (E(B_t)) \equiv N \times (p(B_t)) \tag{37}$$

and

$$c \equiv N \times (E(c_t)) \equiv N \times (p(c_t)) \tag{38}$$

and

$$d \equiv N \times (E(d_t)) \equiv N \times (p(d_t)) \tag{39}$$

and

$$a + b + c + d \equiv A + \underline{A} \equiv B + \underline{B} \equiv N \tag{40}$$

Table 6 provide us again an overview of a two by two contingency (see also Pearson, 1904b, p. 33) table of Binomial random variables.

Table 6. The two by two table of Binomial random variables

		Conditioned B_t		
		TRUE	FALSE	
Condition	TRUE	a	b	A
A_t	FALSE	c	d	$\underline{A}$
		B	$\underline{B}$	N

> "Such a table is termed a contingency table, and the ultimate scientific statement of description of the relation between two things can always be thrown back upon such a contingency table ⋯ Once the reader realizes the nature of such a table, he will have grasped the essence of the conception of association between cause and effect, and the nature of its ideal limit in causation. "
>
> (see also Pearson, 1911, p. 159)

2.2.3. Independence

Definition 2.6 (Independence).

The philosophical, mathematical(Kolmogoroff, Andreĭ Nikolaevich, 1933) and physical(Einstein, 1948) et cetera concept of independence is of fundamental(Kolmogoroff, Andreĭ Nikolaevich, 1933) importance in (natural) sciences as such. Therefore, it is appropriate to investigate the concept of independence as completely as possible. In fact, de Moivre sums it up in his book The Doctrine of Chances (see also Moivre, 1718). "Two Events are **independent**, when they have no connexion one with the other, and that the happening of one neither forwards nor obstructs the happening of the other. Two events are **dependent**, when they are so connected together as that the Probability of either's happening is alter'd by the happening of the other. "(see also Moivre, 1756, p. 6) We should consider Kolmogorov's position on independence before the mind's eye too. "The concept of mutual independence of two or more experiments holds, in a certain sense, a central position in the theory of probability."(see also Kolmogorov, Andreĭ Nikolaevich, 1950, p. 8) Furthermore, it is insightful to recall even Einstein's theoretical approach to the concept of independence. "*Ohne die Annahme einer ⋯ Unabhängigkeit der ⋯ Dinge voneinander ⋯ wäre physikalisches Denken ⋯ nicht möglich.*"(Einstein, 1948). In general, an event A_t at the Bernoulli trial t need not, but can be independent of the existence or of the occurrence, of another event B_t *at the same* Bernoulli trial t. De Moivre brings it to the point. "From what has been said, it follows, that if a Fraction expresses the Probability of an Event, and another Fraction the Probability of another Event, and those two Events are independent ; the Probability that both those Events will Happen, will be the Product of those two Fractions."(see also Moivre, 1718, p. 4). Mathematically, in terms of probability theory, independence (Kolmogoroff, Andreĭ Nikolaevich, 1933) of events at the same (period of) time (i.e. Bernoulli trial) t is defined as

$$p(A_{\mathrm{t}} \wedge B_{\mathrm{t}}) \equiv p(A_{\mathrm{t}}) \times p(B_{\mathrm{t}}) \equiv p(a_{\mathrm{t}}) \equiv \frac{\sum\limits_{t=1}^{N} (A_{\mathrm{t}} \wedge B_{\mathrm{t}})}{N} \equiv \frac{N \times (p(a_{\mathrm{t}}))}{N} \equiv 1 - p(A_{\mathrm{t}} \mid B_{\mathrm{t}}) \equiv 1 - p(A_{\mathrm{t}} \uparrow B_{\mathrm{t}}) \tag{41}$$

while $p(A_{\mathrm{t}} \cap B_{\mathrm{t}})$ is the joint probability of the events A_{t} and B_{t} at a same Bernoulli trial t, $p(A_{\mathrm{t}})$ is the probability of an event A_{t} at a same Bernoulli trial t, and $p(B_{\mathrm{t}})$ is the probability of an event B_{t} at a same Bernoulli trial t. With respect to a two-by-two table , **under conditions of independence**, it is

$$p(b_{\mathrm{t}}) \equiv p(A_{\mathrm{t}}) \times p(\underline{B}_{\mathrm{t}}) \tag{42}$$

or

$$p(c_{\mathrm{t}}) \equiv p(\underline{A}_{\mathrm{t}}) \times p(B_{\mathrm{t}}) \tag{43}$$

and

$$p(d_{\mathrm{t}}) \equiv p(\underline{A}_{\mathrm{t}}) \times p(\underline{B}_{\mathrm{t}}) \tag{44}$$

Example. In a narrower sense, the conditio sine qua non relationship concerns itself at the end only with the case whether the presence of an event A_{t} (condition) enables or guarantees the presence of another event B_{t} (conditioned). Thus far, as a result of the thoughts before, another question worth asking concerns the relationship between the independence of an event A_{t} (a condition) and another event B_{t} (conditioned) and the necessary condition relationship. To be confronted with the danger of bias and equally with the burden of inappropriate conclusions drawn, another fundamental question at this stage is whether is it possible that an event A_{t} (a condition) is a necessary condition of event B_{t} (conditioned) even under circumstances where the event A_{t} (a condition) (a necessary condition) is independent of an event B_{t} (conditioned)? Meanwhile, this question is more or less already answered to the negative (Barukčić, 2018b). An event A_{t} which is a necessary condition of another event B_{t} is equally an event without which another event (B_{t}) could not be, could not occur, and implies as such already a kind of dependence. However, it is not mandatory that such a kind of dependence is a causal one. It is remarkable that **data which provide evidence of a significant conditio sine qua non relationship between two events like A_{t} and B_{t} and equally support the hypothesis that A_{t} and B_{t} are independent of each other are more or less self-contradictory and of very restricted or of none value for further analysis**. In fact, if the opposite view would be taken as plausible, contradictions are more or less inescapable.

2.2.4. Dependence

Definition 2.7 (Dependence).

Whilst it may be true that the occurrence of an event A_{t} does not affect the occurrence of an other event B_{t} the contrary is of no minor importance. Under these other conditions, events, trials and random variables et cetera are dependent on each other too. The dependence of events (Barukčić, 1989, p. 57-61) is defined as

$$p\left(\underbrace{A_{\mathrm{t}} \wedge B_{\mathrm{t}} \wedge C_{\mathrm{t}} \wedge \ldots}_{n\ random\ variables}\right) \equiv \sqrt[n]{\underbrace{p\left(A_{\mathrm{t}}\right) \times p\left(B_{\mathrm{t}}\right) \times p\left(C_{\mathrm{t}}\right) \times \ldots}_{n\ random\ variables}} \tag{45}$$

2.2.5. Sensitivity and specificity

Definition 2.8 (Sensitivity and specificity)**.**

A (medical) test should measure what is supposed to measure. However, the extent to which a test measures what it is supposed to measure varies and is seldom equal to 100 %. In other words, it is necessary to check once and again the accuracy or the validity of a test, we have to fight it out in detail. In clinical practice, the concept of sensitivity and specificity is commonly used to quantify the diagnostic ability of a (medical) test. Sensitivity and specificity were introduced by the American [23], [24], [25], [26] biostatistician Jacob Yerushalmy (see also Yerushalmy, 1947) in the year 1947. The interior logic of sensitivity and specificity is best illustrated using a conventional two- by-two (2 x 2) table (see table 7).

Table 7. Sensitivity and specificity

		Disease B_t		
		present	absent	
Test	positive	a (true positive)	b (false positive)	A
A_t	negative	c (false negative)	d (true negative)	$\underline{A}$
		B	$\underline{B}$	N

The ability of a positive test (A_t) to correctly classify an individual as diseased (B_t) is defined as the proportion of true positives that are correctly identified by the test (a) divided by the individuals being truly diseased (B_t). In general, sensitivity follows as

$$Sensitivity\,(A \mid B) \equiv p\,(a \mid B) \equiv \frac{a}{B} \tag{46}$$

The specificity of a test is the ability of a negative test ($\underline{A}_t$) to correctly classify an individual as not diseased ($\underline{B}_t$ and is defined as the proportion of true negative that are correctly identified by the test (d) divided by the individuals being truly not diseased ($\underline{B}_t$). In general, specificity is given by the equation

$$Specificity\,(\underline{A},\underline{B}) \equiv p\,(d \mid \underline{B}) \equiv \frac{d}{\underline{B}} \tag{47}$$

The positive predictive value (PPV) is defined as

$$PPV\,(A,B) \equiv \frac{a}{a+b} \tag{48}$$

[23] Yerushalmy Jacob. Statistical problems in assessing methods of medical diagnosis, with special reference to X-ray techniques. Public Health Rep. 1947 Oct 3;62(40):1432-49. PMID: 20340527.

[24] Galen RS, Gambino SR. Beyond normality-the predictive value and efficiency of medical diagnosis. New York: NY:Wiley; 1975.

[25] Altman DG, Bland JM. Diagnostic tests. 1: Sensitivity and specificity. BMJ. 1994 Jun 11;308(6943):1552. doi: 10.1136/bmj.308.6943.1552. PMID: 8019315; PMCID: PMC2540489.

[26] Parikh R, Mathai A, Parikh S, Chandra Sekhar G, Thomas R. Understanding and using sensitivity, specificity and predictive values. Indian J Ophthalmol. 2008 Jan-Feb;56(1):45-50. doi: 10.4103/0301-4738.37595. PMID: 18158403; PMCID: PMC2636062.

The negative predictive value (NPV) is defined as

$$NPV\,(A,B) \equiv \frac{d}{c+d} \tag{49}$$

Example.

The importance of sensitivity and specificity in any research should certainly not be underestimated. However, it is essential not to lose sight of the major advantages and limitations [27] of these measures. In the following, in order to avoid misconceptions about sensitivity, specificity et cetera, let us consider a test with a sensitivity of 95 % and a specificity of 95 %. A two-by-two table is used as an illustration (see table 8).

Table 8. Sensitivity and specificity

		Disease B_t		
		present	absent	
Test	positive	95	5	100
A_t	negative	5	95	100
		100	100	200

Sensitivity is calculated as

$$Sensitivity\,(A \mid B) \equiv p\,(a \mid B) \equiv 100 \times \frac{a}{B} \equiv \frac{95}{100} \equiv 95\% \tag{50}$$

There are at least two kinds of medical tests, diagnostic tests and screening tests. Depending on the type of medical test, there are other logical implications. A screening test should correctly identify all people who suffer from a certain disease or all people with a certain outcome. Therefore, the sensitivity of a screening test should be at best 100 %. Under these conditions, we obtain **without** positive test **no** disease/outcome present. However, confusion should be avoided with regard to the adequacy and usefulness of the sensitivity of a screening test. The sensitivity of a test does not take into account events which are false positive (b) or which are true negative (d), the meaning of these events is ignored completely by sensitivity. Therefore, sensitivity is blind on one eye since its inception and underestimates the extent to which a screening test is able to identify the likely presence of a condition of interest. We calculated a 95 % sensitivity while the true possibility of the test to detect a disease is (see table 8)

$$SINE\,(A,B) \equiv 100 \times \frac{a+b+d}{N} \equiv \frac{95+5+95}{200} \equiv 97.5\% \tag{51}$$

In a way similar to sensitivity, specificity is not much better. Diagnostic tests are able to identify people who do not have a certain condition. Specificity is calculated as

$$Specificity\,(\underline{A} \mid \underline{B}) \equiv p\,(d \mid \underline{B}) \equiv 100 \times \frac{d}{\underline{B}} \equiv \frac{95}{100} \equiv 95\% \tag{52}$$

[27] Trevethan R. Sensitivity, Specificity, and Predictive Values: Foundations, Pliabilities, and Pitfalls in Research and Practice. Front Public Health. 2017 Nov 20;5:307. doi: 10.3389/fpubh.2017.00307. PMID: 29209603; PMCID: PMC5701930.

However, specificity does not take into account any individuals who suffer from a disease, who do have the condition and is well-known for being imperfect because of this fact too. Specificity underestimates the possibility of a diagnostic test to detect a disease. Above, the specificity has been calculated as being 95 %. In point of fact, the ability of the test to detect a disease or the relationship **if** test positive **then** disease present is much better and has to be calculated as (see table 8)

$$IMP(A,B) \equiv \frac{a+c+d}{N} \equiv \frac{95+5+95}{200} \equiv 97.5\% \tag{53}$$

As can be seen, the test detected the disease in 97.5 % while specificity allows only 95 %. How valuable is such a measure epistemologicallly? Measures like sensitivity and specificity are blurring of the issue, do risk leading us astray and disorient us systematically again and again. These measures should be abandoned.

2.2.6. Odds ratio (OR)

Definition 2.9 (Odds ratio (OR))**.**

Odds ratios as an appropriate measure for estimating the relative risk have become widely used in medical reports of case-control studies. The odds ratio(Fisher, 1935, p. 50) is defined(Cox, 1958) as the ratio of the odds of an event occurring in one group with respect to the odds of its occurring in another group. Odds(Yule and Pearson, 1900, p. 273) ratio (OR) is a measure of association which quantifies the relationship between two binomial distributed random variables (exposure vs. outcome) and is related to Yule's (Yule and Pearson, 1900, p. 272) Q(Yule, 1912, p. 585/586). Two events A_t and B_t are regarded as independent if $(A_t, B_t) = 1$. Let

a_t = number of persons exposed to A_t and with disease B_t

b_t = number of persons exposed to A_t but without disease $\underline{B}_t$

c_t = number of persons unexposed $\underline{A}_t$ but with disease B_t

d_t = number of persons unexposed $\underline{A}_t$: and without disease $\underline{B}_t$

a_t+c_t = total number of persons with disease B_t (case-patients)

b_t+d_t = total number of persons without disease $\underline{B}_t$ (controls).

Hereafter, consider the table 9. The odds' ratio (OR) is defined as

Table 9. The two by two table of random variables

		Conditioned/Outcome B_t		
		TRUE	FALSE	
Condition/Exposure	TRUE	a_t	b_t	A_t
A_t	FALSE	c_t	d_t	$\underline{A}_t$
		B_t	$\underline{B}_t$	N_t

$$
\begin{aligned}
OR(A_t, B_t) &\equiv \left(\frac{a_t}{b_t}\right) / \left(\frac{c_t}{d_t}\right) \\
&\equiv \left(\frac{a_t \times d_t}{b_t \times c_t}\right)
\end{aligned}
\tag{54}
$$

Remark 2.1. *Odds ratios can support logical fallacies and cause difficulties in drawing logically consistent conclusions. The chorus of voices is growing, which demand the immediate ending(Knol, 2012, Sackett, DL and Deeks, JJ and Altman, DG, 1996) of any use of Odds ratio.*

Under conditions where (b = 0), the measure of association odds ratio will collapse, because we need to divide by zero, as can be seen at eq. 54. However, according to today's rules of mathematics, *a division by zero is neither allowed nor generally accepted as possible. It does no harm to remind ourselves that in the case b = 0 the event* A_t *is a sufficient condition of* B_t*. In other words, odds ratio is not able to recognize elementary relationships of objective reality. In fact, it would be a failure not to recognize how dangerous and less valuable odds ratio is.*

Under conditions where (c = 0) odds ratio collapses too, because we need again to divide by zero, as can be seen at eq. 54. However, and again, today's rules of mathematics don't allow us a division by zero. In point of fact, in the case c = 0 it is more than necessary to point out that A_t *is a necessary condition of* B_t*. In other words, odds ratio or the cross-product ratio is not able to recognize elementary relationships of nature like necessary conditions. We can and need to overcome all the epistemological obstacles as backed by odds ratio entirety. Sooner rather than later, we should give up this measure of relationship completely.*

2.2.7. Relative risk (RR)

2.2.7.1. Relative risk (RR_{nc})

Definition 2.10 (Relative risk (RR_{nc})).

The degree of association between the two binomial variables can be assessed by a number of very different coefficients, the relative (Cornfield, 1951, Sadowsky et al., 1953) risk is one(Barukčić, 2021d) of them. In general, relative risk RR_{nc}, which provides some evidence of a necessary condition, is defined as

$$
\begin{aligned}
RR(A_t, B_t)_{nc} &\equiv \frac{\frac{p(a_t)}{p(A_t)}}{\frac{p(c_t)}{p(NotA_t)}} \\
&\equiv \frac{p(a_t) \times p(NotA_t)}{p(c_t) \times p(A_t)} \\
&\equiv \frac{N \times p(a_t) \times N \times p(NotA_t)}{N \times p(c_t) \times N \times p(A_t)} \\
&\equiv \frac{a_t \times (NotA_t)}{c_t \times A_t} \\
&\equiv \frac{EER(A_t, B_t)}{CER(A_t, B_t)}
\end{aligned}
\tag{55}
$$

That what scientist generally understand by relative risk is the ratio of a probability of an event occurring with an exposure versus the probability of an event occurring without an exposure. In other words,

relative risk = (probability(event in exposed group)) / (probability(the same event in not exposed group)).

A $RR(A_t,B_t) = +1$ means that exposure does not affect the outcome or both are independent of each other while $RR(A_t,B_t)$ less than +1 means that the risk of the outcome is decreased by the exposure. In this context, an $RR(A_t,B_t)$ greater than +1 denotes that the risk of the outcome is increased by the exposure. Widely known problems with odds ratio and relative risk are already documented in literature.

2.2.7.2. Relative risk (RR (sc))

Definition 2.11 (Relative risk (RR (sc))).

The relative risk (sc), which provides some evidence of a sufficient condition, is calculated from the point of view of an outcome and is defined as

$$
\begin{aligned}
RR(A_{\mathrm{t}}, B_{\mathrm{t}})_{\mathrm{sc}} &\equiv \frac{\frac{p(a_{\mathrm{t}})}{p(B_{\mathrm{t}})}}{\frac{p(b_{\mathrm{t}})}{p(NotB_{\mathrm{t}})}} \\
&\equiv \frac{p(a_{\mathrm{t}}) \times p(NotB_{\mathrm{t}})}{p(b_{\mathrm{t}}) \times p(B_{\mathrm{t}})} \\
&\equiv \frac{N \times p(a_{\mathrm{t}}) \times N \times p(NotB_{\mathrm{t}})}{N \times p(b_{\mathrm{t}}) \times N \times p(B_{\mathrm{t}})} \\
&\equiv \frac{a_{\mathrm{t}} \times (NotB_{\mathrm{t}})}{b_{\mathrm{t}} \times B_{\mathrm{t}}} \\
&\equiv \frac{OPR(A_{\mathrm{t}}, B_{\mathrm{t}})}{CPR(A_{\mathrm{t}}, B_{\mathrm{t}})}
\end{aligned}
\tag{56}
$$

2.2.7.3. Relative risk reduction (RRR)

Definition 2.12 (Relative risk reduction (RRR))**.**

$$
\begin{aligned}
RRR(A_{\mathrm{t}}, B_{\mathrm{t}}) &\equiv \frac{CER(A_{\mathrm{t}}, B_{\mathrm{t}}) - EER(A_{\mathrm{t}}, B_{\mathrm{t}})}{CER(A_{\mathrm{t}}, B_{\mathrm{t}})} \\
&= 1 - RR(A_{\mathrm{t}}, B_{\mathrm{t}})
\end{aligned}
\tag{57}
$$

2.2.7.4. Vaccine efficacy (VE)

Definition 2.13 (Vaccine efficacy (VE))**.**

Vaccine efficacy is defined as the percentage reduction of a disease in a vaccinated group of people as compared to an unvaccinated group of people.

$$
\begin{aligned}
VE(A_{\mathrm{t}}, B_{\mathrm{t}}) &\equiv 100 \times (1 - RR(A_{\mathrm{t}}, B_{\mathrm{t}})) \\
&\equiv 100 \times \left(\frac{CER(A_{\mathrm{t}}, B_{\mathrm{t}}) - EER(A_{\mathrm{t}}, B_{\mathrm{t}})}{CER(A_{\mathrm{t}}, B_{\mathrm{t}})}\right)
\end{aligned}
\tag{58}
$$

Historically, vaccine efficacy has been designed to evaluate the efficacy of a certain vaccine by Greenwood and Yule in 1915 for the cholera and typhoid vaccines(Greenwood and Yule, 1915) and best measured using double-blind, randomized, clinical controlled trials. However, the calculated vaccine efficacy is depending too much on the study design, can lead to erroneous conclusions and is only of very limited value.

2.2.7.5. Experimental event rate (EER)

Definition 2.14 (Experimental event rate (EER))**.**

$$EER\left(A_{t},B_{t}\right) \equiv \frac{p(a_{t})}{p(A_{t})} = \frac{a_{t}}{a_{t}+b_{t}} \tag{59}$$

Definition 2.15 (Control event rate (CER))**.**

$$CER\left(A_{t},B_{t}\right) \equiv \frac{p(c_{t})}{p(\underline{A}_{t})} = \frac{c_{t}}{c_{t}+d_{t}} \tag{60}$$

2.2.7.6. Absolute risk reduction (ARR)

Definition 2.16 (Absolute risk reducation (ARR))**.**

$$\begin{aligned} ARR\left(A_{t},B_{t}\right) &\equiv \frac{p(c_{t})}{p(\underline{A}_{t})} - \frac{p(a_{t})}{p(A_{t})} \\ &= \frac{c_{t}}{c_{t}+d_{t}} - \frac{a_{t}}{a_{t}+b_{t}} \\ &= CER\left(A_{t},B_{t}\right) - EER\left(A_{t},B_{t}\right) \end{aligned} \tag{61}$$

2.2.7.7. Absolute risk increase (ARI)

Definition 2.17 (Absolute risk increase (ARI))**.**

$$\begin{aligned} ARI\left(A_{t},B_{t}\right) &\equiv \frac{p(a_{t})}{p(A_{t})} - \frac{p(c_{t})}{p(\underline{A}_{t})} \\ &= EER\left(A_{t},B_{t}\right) - CER\left(A_{t},B_{t}\right) \end{aligned} \tag{62}$$

2.2.7.8. Number needed to treat (NNT)

Definition 2.18 (Number needed to treat (NNT))**.**

$$NNT\left(A_{t},B_{t}\right) \equiv \frac{1}{CER\left(A_{t},B_{t}\right)-EER\left(A_{t},B_{t}\right)} \tag{63}$$

An ideal number needed to treat(Cook and Sackett, 1995, Laupacis et al., 1988), mathematically the reciprocal of the absolute risk reduction, is NNT = 1. Under these circumstances, everyone improves with a treatment, while no one improves with control. A higher number needed to treat indicates more or less a treatment which is less effective.

2.2.7.9. Number needed to harm (NNH)

Definition 2.19 (Number needed to harm (NNH))**.**

$$NNH\left(A_{t},B_{t}\right) \equiv \frac{1}{EER\left(A_{t},B_{t}\right)-CER\left(A_{t},B_{t}\right)} \tag{64}$$

The number needed to harm (Massel and Cruickshank, 2002), mathematically the inverse of the absolute risk increase, indicates at the end how many patients need to be exposed to a certain factor, in order to observe a harm in one patient that would not otherwise have been harmed.

2.2.7.10. Outcome prevalence rate (OPR)

Definition 2.20 (Outcome prevalence rate (OPR))**.**

$$OPR\left(A_{t},B_{t}\right) \equiv \frac{p(a_{t})}{p(B_{t})} = \frac{a_{t}}{a_{t}+c_{t}} \tag{65}$$

2.2.7.11. Control prevalence rate (CPR)

Definition 2.21 (Control prevalence rate (CPR))**.**

$$CPR\left(A_{t},B_{t}\right) \equiv \frac{p(b_{t})}{p(\underline{B}_{t})} = \frac{b_{t}}{b_{t}+d_{t}} \tag{66}$$

Bias and confounding is present to some degree in all research. In order to assess the relationship of exposure with a disease or an outcome, a fictive control group (i.e. of newborn or of young children et cetera) can be of use too. Under certain circumstances, even a CPR = 0 is imaginable.

2.2.7.12. Absolute prevalence reduction (APR)

Definition 2.22 (Absolute prevalence reduction (APR))**.**

$$APR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) \equiv CPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) - OPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) \tag{67}$$

2.2.7.13. Absolute prevalence increase (API)

Definition 2.23 (Absolute prevalence increase (API))**.**

$$API\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) \equiv OPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) - CPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) \tag{68}$$

2.2.7.14. Relative prevalence reduction (RPR)

Definition 2.24 (Relative prevalence reduction (RPR))**.**

$$\begin{aligned} RPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) &\equiv \frac{CPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) - OPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right)}{CPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right)} \\ &= 1 - RR(A_{\mathrm{t}}, B_{\mathrm{t}})_{\mathrm{sc}} \end{aligned} \tag{69}$$

2.2.7.15. The index NNS

Definition 2.25 (The index NNS)**.**

$$NNS\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) \equiv \frac{1}{CPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) - OPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right)} \tag{70}$$

Mathematically, the index NNS is the reciprocal of the absolute prevalence reduction.

2.2.7.16. The index NNI

Definition 2.26 (The index NNI)**.**

$$NNI\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) \equiv \frac{1}{OPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right) - CPR\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right)} \tag{71}$$

Mathematically, the index NNI is the reciprocal of the absolute prevalence increase.

2.2.8. Index of relationship (IOR)

Definition 2.27 (Index of relationship (IOR))**.**

Due to several reasons, it is not always easy to identify the unique characteristics between two events like A_t and B_t. And more than that, it is difficult to decide what to do, and much more difficult to know in which direction one should think and which decision is right. Sometimes it is helpful to know at least something about the direction of the relationship between two events like A_t and B_t. Under conditions where $p(a_t) = p(A_t \wedge B_t)$, the index of relationship(Barukčić, 2021b), abbreviated as IOR, is defined as

$$\begin{aligned} IOR(A_t, B_t) &\equiv \left(\frac{p(A_t \wedge B_t)}{p(B_t) \times p(A_t)} \right) - 1 \\ &\equiv \left(\frac{p(a_t)}{p(B_t) \times p(A_t)} \right) - 1 \\ &\equiv \left(\left(\frac{N \times N \times p(a_t)}{N \times p(B_t) \times N \times p(A_t)} \right) - 1 \right) \\ &\equiv \left(\left(\frac{N \times a}{A \times B} \right) - 1 \right) \end{aligned} \tag{72}$$

where $p(A_t)$ denotes the probability of an event A_t at the Bernoulli trial t and $p(B_t)$ denotes the probability of another event B_t at the same Bernoulli trial t while $p(a_t)$ denotes the joint probability of $p(A_t$ AND $B_t)$ at the same Bernoulli trial t and a, A and B may denote the expectation values.

Even if a condition and a cause are deeply related, there are circumstances where a sharp distinction between a cause and a condition is necessary. However, exactly this has been denied by John Stuart Mill's (1806-1873) regularity view of causality (see Mill, 1843b). What might seem to be a theoretical difficulty for many authors is none for Mill. Mill simply reduced a cause to a condition and claimed that "... the real cause of the phenomenon is the assemblage of all its conditions." (see Mill, 1843a, p. 403)

2.3.1. Exclusion relationship

Definition 2.28 (Exclusion relationship [*EXCL*]).

Mathematically, the exclusion(see also Barukčić, 2021a) relationship [28] (EXCL), denoted by $p(A_t \mid B_t)$ in terms of statistics and probability theory, is defined(see also Barukčić, 1989, p. 68-70) as

$$
\begin{aligned}
p\left(A_{\mathrm{t}} \mid B_{\mathrm{t}}\right) &\equiv p\left(A_{\mathrm{t}} \uparrow B_{\mathrm{t}}\right) \\
&\equiv p\left(b_{\mathrm{t}}\right)+p\left(c_{\mathrm{t}}\right)+p\left(d_{\mathrm{t}}\right) \\
&\equiv \frac{N \times \left(p\left(b_{\mathrm{t}}\right)+p\left(c_{\mathrm{t}}\right)+p\left(d_{\mathrm{t}}\right)\right)}{N} \\
&\equiv \frac{\sum_{t=1}^{N}\left(\underline{A}_{\mathrm{t}} \vee \underline{B}_{\mathrm{t}}\right)}{N} \equiv \frac{b+c+d}{N} \\
&\equiv \frac{b+\underline{A}}{N} \\
&\equiv \frac{c+\underline{B}}{N} \\
&\equiv +1
\end{aligned}
\tag{73}
$$

Based on the 1913 Henry Maurice Sheffer (1882-1964) relationship, the Sheffer stroke(Nicod, 1917, Sheffer, 1913) usually denoted by ↑, it is $p\left(A_{\mathrm{t}} \wedge B_{\mathrm{t}}\right) \equiv 1-p\left(A_{\mathrm{t}} \mid B_{\mathrm{t}}\right)$ (see table 10).

Table 10. A_t excludes B_t and vice versa.

		Conditioned (COVID-19) B_t		
		TRUE	FALSE	
Condition (Vaccine)	TRUE	**+0**	$p(b_t)$	$p(A_t)$
A_t	FALSE	$p(c_t)$	$p(d_t)$	$p(\underline{A}_t)$
		$p(B_t)$	$p(\underline{B}_t)$	+1

[28] Barukčić, Ilija. (2021). Mutually exclusive events. Causation, 16(11), 5–57. https://doi.org/10.5281/zenodo.5746415

Example 2.1. *Pfizer Inc. and BioNTech SE announced on Monday, November 09, 2020 - 06:45am results from a Phase 3 COVID-19 vaccine trial with 43.538 participants which provides evidence that their vaccine (BNT162b2) is preventing COVID-19 in participants without evidence of prior SARS-CoV-2 infection. In toto, 170 confirmed cases of COVID-19 were evaluated, with 8 in the vaccine group versus 162 in the placebo group. The exclusion relationship can be calculated as follows.*

$$\begin{aligned} p\left(Vaccine: BNT162b2 \mid COVID-19(infection)\right) &\equiv p\left(b_t\right)+p\left(c_t\right)+p\left(d_t\right) \\ &\equiv 1-p\left(a_t\right) \\ &\equiv 1-\left(\frac{8}{43538}\right) \\ &\equiv +0,99981625 \end{aligned} \tag{74}$$

with a P Value = 0,000184.

Following Kolmogorov's definition of an n-dimensional probability density (see also Kolmogorov, Andreĭ Nikolaevich, 1950, p. 26) of random variables $\mathrm{A_t}$, $\mathrm{B_t}$ et cetera at the point t, we obtain

$$\begin{aligned} p\left(A_{\mathrm{t}} \mid B_{\mathrm{t}}\right) &\equiv p\left(\underline{A}_{\mathrm{t}} \cup \underline{B}_{\mathrm{t}}\right) \\ &\equiv 1-p\left(A_{\mathrm{t}} \cap B_{\mathrm{t}}\right) \\ &\equiv 1-\int_{-\infty}^{A_{\mathrm{t}}} \int_{-\infty}^{B_{\mathrm{t}}} f\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right)\, dA_{\mathrm{t}}\, dB_{\mathrm{t}} \\ &\equiv +1 \end{aligned} \tag{75}$$

while $p\left(A_{\mathrm{t}} \mid B_{\mathrm{t}}\right)$ would denote the cumulative distribution function of random variables and $f\left(A_{\mathrm{t}}, B_{\mathrm{t}}\right)$ is the joint density function.

2.3.2. Observational study and exclusion relationship

Under conditions of an observational study, the exclusion relationship follows approximately(see Barukčić, 2021a) as

$$p\left(A_{\mathrm{t}} \mid B_{\mathrm{t}}\right) \equiv p\left(A_{\mathrm{t}} \uparrow B_{\mathrm{t}}\right) \geq 1-\frac{p\left(a_{\mathrm{t}}\right)}{p\left(B_{\mathrm{t}}\right)} \tag{76}$$

2.3.3. Experimental study and exclusion relationship

Under conditions of an experimental study, the exclusion relationship follows approximately(see Barukčić, 2021a) as

$$p\left(A_{\mathrm{t}} \mid B_{\mathrm{t}}\right) \equiv p\left(A_{\mathrm{t}} \uparrow B_{\mathrm{t}}\right) \geq 1-\frac{p\left(a_{\mathrm{t}}\right)}{p\left(A_{\mathrm{t}}\right)} \tag{77}$$

2.3.4. The goodness of fit test of an exclusion relationship

Definition 2.29 (The $\tilde{\chi}^2$ goodness of fit test of an exclusion relationship).

Under some well known circumstances, testing hypothesis about an exclusion relationship p(A_t | B_t) is possible by the chi-square distribution (also chi-squared or $\tilde{\chi}^2$-distribution) too. The $\tilde{\chi}^2$ goodness of fit test of an exclusion relationship with degree of freedom (d. f.) of d. f. = 1 is calculated as

$$\begin{aligned}\tilde{\chi}^2{}_{\text{Calculated}}\left(\left(A_t \mid B_t\right) \mid A\right) &\equiv \frac{(b-(a+b))^2}{A} + \\ &\quad \frac{((c+d)-\underline{A})^2}{\underline{A}} \\ &\equiv \frac{a^2}{A} + 0 \\ &\equiv \frac{a^2}{A}\end{aligned} \tag{78}$$

or equally as

$$\begin{aligned}\tilde{\chi}^2{}_{\text{Calculated}}\left(\left(A_t \mid B_t\right) \mid B\right) &\equiv \frac{(c-(a+c))^2}{B} + \\ &\quad \frac{((b+d)-\underline{B})^2}{\underline{B}} \\ &\equiv \frac{a^2}{B} + 0 \\ &\equiv \frac{a^2}{B}\end{aligned} \tag{79}$$

and can be compared with a theoretical chi-square value at a certain level of significance α. The $\tilde{\chi}^2$-distribution equals zero when the observed values are equal to the expected/theoretical values of an exclusion relationship/distribution p(A_t | B_t), in which case the null hypothesis has to be accepted. Yate's (Yates, 1934) continuity correction was not used under these circumstances.

2.3.5. The left-tailed p Value of an exclusion relationship

Definition 2.30 (The left-tailed p Value of an exclusion relationship).

It is known that as a sample size, N, increases, a sampling distribution of a special test statistic approaches the normal distribution (central limit theorem). Under these circumstances, the left-tailed (lt) p Value (Barukčić, 2019f) of an exclusion relationship can be calculated as follows.

$$\begin{aligned} pValue_{\mathrm{lt}}(A_{\mathrm{t}} \mid B_{\mathrm{t}}) &\equiv 1 - e^{-(1-p(A_{\mathrm{t}}|B_{\mathrm{t}}))} \\ &\equiv 1 - e^{-(a/N)} \end{aligned} \tag{80}$$

A low p-value may provide some evidence of statistical significance.

2.3.6. Neither nor conditions

Definition 2.31 (**Neither A_t nor B_t conditions [*NOR*]).**

Mathematically, a neither A_t nor B_t condition (or rejection according to the French philosopher and logician Jean George Pierre Nicod (1893-1924), i.e. Jean Nicod's statement (Nicod, 1924)) relationship (NOR), denoted by $p(A_t \downarrow B_t)$ in terms of statistics and probability theory, is defined (Barukčić, 1989, p. 68-70) as

$$\begin{aligned} p(A_{\mathrm{t}} \downarrow B_{\mathrm{t}}) &\equiv p(d_{\mathrm{t}}) \\ &\equiv \frac{N - \sum_{t=1}^{N} (A_{\mathrm{t}} \vee B_{\mathrm{t}})}{N} \equiv \frac{\sum_{t=1}^{N} (\underline{A}_{\mathrm{t}} \wedge \underline{B}_{\mathrm{t}})}{N} \equiv \frac{N \times (p(d_{\mathrm{t}}))}{N} \\ &\equiv \frac{d}{N} \\ &\equiv +1 \end{aligned} \tag{81}$$

2.3.7. The Chi square goodness of fit test of a neither nor condition relationship

Definition 2.32 (**The $\tilde{\chi}^2$ goodness of fit test of a neither A_t nor B_t condition relationship).**

A neither A_t nor B_t condition relationship $p(A_t \downarrow B_t)$ can be tested by the chi-square distribution (also chi-squared or $\tilde{\chi}^2$-distribution). The $\tilde{\chi}^2$ goodness of fit test of a neither A_t nor B_t condition relationship with degree of freedom (d. f.) of d. f. = 1 may be calculated as

$$\begin{aligned} \tilde{\chi}^2{}_{\text{Calculated}}((A_{\mathrm{t}} \downarrow B_{\mathrm{t}}) \mid A) &\equiv \frac{(d-(c+d))^2}{\underline{A}} + \\ &\quad \frac{((a+b)-A)^2}{A} \\ &\equiv \frac{c^2}{\underline{A}} + 0 \end{aligned} \tag{82}$$

or equally as

$$\begin{aligned}\tilde{\chi}^2{}_{\text{Calculated}}\left((A_t \downarrow B_t) \mid B\right) &\equiv \frac{(d-(b+d))^2}{\underline{B}}+\\&\quad\frac{((a+c)-B)^2}{B}\\&\equiv \frac{b^2}{\underline{B}}+0\end{aligned} \tag{83}$$

Yate's (Yates, 1934) continuity correction has not been used in this context.

2.3.8. The left-tailed p Value of a neither nor B condition relationship

Definition 2.33 (The left-tailed p Value of a neither A_t nor B_t condition relationship).

The left-tailed (lt) p Value (Barukčić, 2019f) of a neither A_t nor B_t condition relationship can be calculated as follows.

$$\begin{aligned}pValue_{lt}(A_t \downarrow B_t) &\equiv 1-e^{-(1-p(A_t \downarrow B_t))}\\&\equiv 1-e^{-p(A_t \vee B_t)}\\&\equiv 1-e^{-((a+b+c)/N)}\end{aligned} \tag{84}$$

where $\vee$ may denote disjunction or logical inclusive or. In this context, a low p-value indicates again a statistical significance. In general, it is $p(A_t \vee B_t) \equiv 1-p(A_t \downarrow B_t)$ (see table 11).

Table 11. Neither A_t nor B_t relationship.

		Conditioned B_t		
		YES	NO	
Condition A_t	YES	0	0	0
	NO	0	1	1
		0	1	1

2.3.9. Necessary condition

Definition 2.34 (**Necessary condition [*Conditio sine qua non*]).**

Despite the most extended efforts, the current state of research on conditions and conditioned is still incomplete and very contradictory. However, even thousands of years ago and independently of any human mind and consciousness, water has been and is still a necessary condition for (human) life. **Without** water, there has been and there is **no** (human) life[29]. It comes therefore as no surprise that one of the first documented attempts to present a rigorous theory of conditions and causation (see also Aristotle, of Stageira (384-322 B.C.E), 1908, Metaphysica III 2 997a 10 and 13/14) came from the Greek philosopher and scientist Aristotle (384-322 BCE). Thus far, it is amazing that Aristotle himself made already a strict distinction between conditions and causes. Taking Aristotle very seriously, it is necessary to consider that

> "... everything which has a ... ... potency in question ... ... has the potency ... of acting ... not in all circumstances but on certain conditions ... " (see also Aristotle, of Stageira (384-322 B.C.E), 1908, Metaphysica IX 5 1048a 14-19)

Before going into details, Aristotle went on to define the necessary condition as follows.

> "... necessary ... means ...
>
> without ... a condition, a thing cannot live ... "
>
> (see also Aristotle, of Stageira (384-322 B.C.E), 1908, Metaphysica V 2 1015a 20-22)

In point of fact, Aristotle developed a theory of conditions and causality commonly referred to as the doctrine of four causes. Many aspects and general features of Aristotle's logical concept of causality are meanwhile extensively and critically debated in secondary literature. However, even if the Greek philosophers Heraclitus, Plato, Aristotle et cetera numbers among the greatest philosophers of all time, the philosophy has evolved. Scientific knowledge and objective reality are deeply interrelated and cannot be reduced only to Greek philosophers like Aristotle. Among many other issues, the specification of necessary conditions has traditionally been part of the philosopher's investigations of different phenomena. However, behind the need of a detailed evidence, it is justified to consider that philosophy or philosophers as such certainly do not possess **a monopoly on the truth** and other areas such as medicine as well as other sciences and technology may transmit truths as well and may be of help to move beyond one's self enclosed unit. Seemingly, **the law's concept of causation** justifies to say few words on this subject, to put some light on some questions. Are there any criteria in law for deciding whether one action or an event A_t has caused another (generally harmful) event B_t? What are these criteria? May causation in legal contexts differ from causation outside the law, for example, in science

[29] Barukčić, Ilija. (2022). Conditio sine qua non (Version 1). Zenodo. https://doi.org/10.5281/zenodo.5854744

or in our everyday life and to what extent? Under which circumstances is it justified to tolerate such differences as may be found to exist? To understand just what is the law's concept of causation, it is useful to re-consider how the highest court of states is dealing with causation. In the case *Hayes v. Michigan Central R. Co., 111 U.S. 228*, the U.S. Supreme Court defined 1884 conditio sine qua non as follows: "... **causa sine qua non – a cause which, if it had not existed, the injury would not have taken place**". (Justice Matthews, Mr., 1884) The German Bundesgerichtshof für Strafsachen stressed once again the importance of conditio sine qua non relationship in his decision by defining the following: "**Ursache eines strafrechtlich bedeutsamen Erfolges jede Bedingung, die nicht hinweggedacht werden kann, ohne daß der Erfolg entfiele**"(Bundesgerichtshof für Strafsachen, 1951) Another lawyer elaborated on the basic issue of **identity and difference between cause and condition**. Von Bar was writing: "Die erste Voraussetzung, welche erforderlich ist, damit eine Erscheinung als die Ursache einer anderen bezeichnet werden könne, ist, daß jene eine der Bedingungen dieser sein. Würde die zweite Erscheinung auch dann eingetreten sein, wenn die erste nicht vorhanden war, so ist sie in keinem Falle Bedingung und noch weniger Ursache. Wo immer ein Kausalzusammenhang behauptet wird, da muß er wenigstens diese Probe aushalten ... **Jede Ursache ist nothwendig auch eine Bedingung eines Ereignisses; aber nicht jede Bedingung ist Ursache zu nennen**."(Bar, Carl Ludwig von, 1871) Von Bar's position translated into English: *The first requirement, which is required, thus that something could be called as the cause of another, is that the one has to be one of the conditions of the other. If the second something had occurred even if the first one did not exist, so it is by no means a condition and still less a cause. Wherever a causal relationship is claimed, the same must at least withstand this test... Every cause is necessarily also a condition of an event too; but not every condition is cause too.* Thus far, let us consider among other the following in order to specify necessary conditions from another, probabilistic point of view. An event (i.e. A_t) which is a necessary condition of another event or outcome (i.e. B_t) must be given, must be present for a conditioned, for an event or for an outcome B_t to occur. A necessary condition (i.e. A_t) is a requirement which need to be fulfilled **at every single Bernoulli trial t**, in order for a conditioned or an outcome (i.e. B_t) to occur, but it alone does not determine the occurrence of such an event. In other words, if a necessary condition (i.e. A_t) is given, an outcome (i.e. B_t) need not to occur. In contrast to a necessary condition, a 'sufficient'condition is the one condition which 'guarantees'that an outcome will take place or will occur for sure. Under which conditions we may infer about the unobserved and whether observations made are able at all to justify predictions about potential observations which have not yet been made or even general claims which my go even beyond the observed (*the 'problem of induction')* is not the issue of the discussion at this point. Besides of the principal necessity of meeting such a challenge, a necessary condition of an event can but need not be at the same Bernoulli trial t a sufficient condition for an event to occur. However, theoretically, it is possible that an event or an outcome is determined by many necessary conditions. Let us focus to some extent on what this means, or in other words how much importance can we attribute to such a special case. *Example*. A human being cannot live without oxygen. A human being cannot live without water. A human being cannot live without a brain. A human being cannot live without kidneys. A human being cannot live without ... et cetera. Thus far, even if oxygen is given, if a brain is given ... et cetera, without water a human being will not survive on the long run. This example is of use to reach the following conclusion. Although it might seem somewhat paradoxical at first sight, **even under circumstances where a condition or an outcome depends on several different necessary conditions it is particularly important that every single of**

these necessary conditions for itself must be given otherwise the conditioned (i.e. the outcome) will not occur. Mathematically, the necessary condition (SINE) relationship, denoted by p($A_t \leftarrow B_t$) in terms of statistics and probability theory, is defined (Barukčić, 1989, p. 15-28) as

$$
\begin{split}
p(A_t \leftarrow B_t) \equiv p(A_t \vee \underline{B}_t) &\equiv \frac{\sum_{t=1}^{N}(A_t \vee \underline{B}_t)}{N} \equiv \frac{(A_t \vee \underline{B}_t) \times p(A_t \vee \underline{B}_t)}{(A_t \vee \underline{B}_t)}\\
&\equiv p(a_t) + p(b_t) + p(d_t)\\
&\equiv \frac{N \times (p(a_t) + p(b_t) + p(d_t))}{N} \equiv \frac{E(A_t \leftarrow B_t)}{N}\\
&\equiv \frac{a+b+d}{N} \equiv \frac{E(A_t \vee \underline{B}_t)}{N}\\
&\equiv \frac{A+d}{N} \equiv \frac{E(A_t \leftarrow B_t)}{N}\\
&\equiv \frac{a+\underline{B}}{N} \equiv \frac{E(A_t \vee \underline{B}_t)}{N}\\
&\equiv +1
\end{split}
\tag{85}
$$

where $E(A_t \leftarrow B_t) \equiv E(A_t \vee \underline{B}_t)$ indicates the expectation value of the necessary condition. In general, it is $p(A_t \prec B_t) \equiv 1 - p(A_t \leftarrow B_t)$ (see Table 12).

Table 12. Necessary condition.

		Conditioned B_t TRUE	FALSE	
Condition	TRUE	$p(a_t)$	$p(b_t)$	$p(A_t)$
A_t	FALSE	**+0**	$p(d_t)$	$p(\underline{A}_t)$
		$p(B_t)$	$p(\underline{B}_t)$	+1

A necessary condition A_t is characterised itself by the property that another event B_t will not occur if A_t is not given, if A_t did not occur (Barukčić, 1989, 1997, 2005, 2016b, 2017b,c, 2020a,c,d,e, Barukčić and Ufuoma, 2020). Taking into account Kolmogorov's definition of an n-dimensional probability density (see also Kolmogorov, Andreĭ Nikolaevich, 1950, p. 26) of random variables A_t, B_t et cetera at the (period of) time t, we obtain

$$
\begin{split}
p(A_t \leftarrow B_t) &\equiv +1\\
&\equiv +1 - p(c_t)\\
&\equiv +1 - p(\underline{A}_t \cap B_t)\\
&\equiv \left(\int_{-\infty}^{A_t}\int_{-\infty}^{B_t} f(A_t, B_t)\ dA_t\, dB_t\right) + \left(1 - \int_{-\infty}^{B_t} f(B_t)\ dB_t\right)
\end{split}
\tag{86}
$$

while $p(A_t \leftarrow B_t)$ would denote the cumulative distribution function of random variables of a necessary condition. Another adequate formulation of a necessary condition is possible too. If certain conditions are met, then necessary conditions and sufficient conditions are one way or another converses of each other, too. It is

$$p\left(A_t \leftarrow B_t\right) \equiv \underbrace{\left(A_t \vee \underline{B}_t\right)}_{\text{(Necessary condition)}} \equiv \underbrace{\left(\underline{B}_t \vee A_t\right)}_{\text{(Sufficient condition)}} \equiv p\left(B_t \rightarrow A_t\right) \tag{87}$$

These relationships are illustrated by the following tables.

Table 13. Without A_t no B_t

		B_t		
		TRUE	FALSE	
A_t	TRUE	a_t	b_t	A_t
	FALSE	$c_t = 0$	d_t	$\underline{A}_t$
		B_t	$\underline{B}_t$	$+1$

Table 14. If B_t then A_t

		A_t		
		TRUE	FALSE	
B_t	TRUE	a_t	$c_t = 0$	B_t
	FALSE	b_t	d_t	$\underline{B}_t$
		A_t	$\underline{A}_t$	$+1$

There are circumstances under which

$$p\left(A_t \leftarrow B_t\right) \equiv \underbrace{\left(A_t \vee \underline{B}_t\right)}_{\text{(Nessessary condition)}} \equiv \underbrace{\left(\underline{A}_t \vee B_t\right)}_{\text{(Sufficient condition)}} \equiv p\left(A_t \rightarrow B_t\right) \tag{88}$$

However, equation 87 does not imply the relationship of equation 88 under any circumstances.

Example I.

A wax candle is characterised by various properties, but is also subject to certain conditions. **Without** sufficient amounts of gaseous oxygen **no** burning wax candle, gaseous oxygen is a necessary condition of a burning candle. However, the converse relationship **if** burning wax candle, **then** sufficient amounts of gaseous oxygen are given is is at the same (period of) time t / Bernoulli trial t true. The following tables are illustrating these relationships.

Table 15. Without gaseous oxygen no burning candle

		Burning candle		
		TRUE	FALSE	
Gaseous	TRUE	a_t	b_t	A_t
oxygen	FALSE	$c_t = 0$	d_t	$\underline{A}_t$
		B_t	$\underline{B}_t$	$+1$

Table 16. If burning candle then gaseous oxygen

		Gaseous oxygen		
		TRUE	FALSE	
Burning	TRUE	a_t	$c_t = 0$	B_t
candle	FALSE	b_t	d_t	$\underline{B}_t$
		A_t	$\underline{A}_t$	$+1$

Example II.

Once again, a human being cannot live without water. A human being cannot live without gaseous oxygen, et cetera. Water itself is a necessary condition for human life. However, gaseous oxygen is a necessary condition for human life too. Thus far, even if water is given and even if water is a necessary condition for human life, without gaseous oxygen there will be no human life. In general, if a conditioned or an outcome B_t depends on the necessary condition A_t and equally on numerous other necessary conditions, an event B_t will not occur if A_t itself is not given independently of the occurrence of other necessary conditions.

Example III.

Another different aspect of a necessary condition relationship is appropriate to be focused upon here. As a direct consequence of a necessary condition **without** sufficient amounts of gaseous oxygen **no** burning wax candle is a special case of an exclusion relationship. The absence of sufficient amounts of gaseous oxygen A_t excludes (see Barukčić, 2021a) a burning wax candle B_t. Thus far, if we want to stop the burning of a wax candle, we would have to significantly reduce the amounts of gaseous oxygen A_t. Under these conditions, a wax candle will stop burning. The following tables (table 17 and table 18) may illustrate this aspect of a necessary condition in more detail.

Table 17. Without gaseous oxygen no burning candle

		Burning candle		
		TRUE	FALSE	
Gaseous	TRUE	a_t	b_t	A_t
oxygen	FALSE	$c_t = 0$	d_t	$\underline{A}_t$
		B_t	$\underline{B}_t$	+1

Table 18. Absent gaseous oxygen excludes burning wax candle

		Burning candle		
		TRUE	FALSE	
Gaseous	FALSE	$c_t = 0$	d_t	B_t
oxygen	TRUE	a_t	b_t	$\underline{B}_t$
		A_t	$\underline{A}_t$	+1

The necessary condition relationship follows approximately (see Barukčić, 2022b) as

$$p\left(A_t \leftarrow B_t\right) \geq 1 - \frac{p\left(c_t\right)}{p\left(B_t\right)} \tag{89}$$

and as

$$p\left(A_t \leftarrow B_t\right) \geq 1 - \frac{p\left(c_t\right)}{p\left(\underline{A}_t\right)} \tag{90}$$

2.3.10. The Chi-square goodness of fit test of a necessary condition relationship

Definition 2.35 (The $\tilde{\chi}^2$ goodness of fit test of a necessary condition relationship).

Under some well known circumstances, hypothesis about the conditio sine qua non relationship $p(A_t \leftarrow B_t)$ can be tested by the chi-square distribution (also chi-squared or χ^2-distribution), first described by the German statistician Friedrich Robert Helmert (Helmert, 1876) and later rediscovered by Karl Pearson (Pearson, 1900) in the context of a goodness of fit test. The $\tilde{\chi}^2$ goodness of fit test of a conditio sine qua non relationship with degree of freedom (d. f.) of d. f. = 1 is calculated as

$$
\begin{aligned}
\tilde{\chi}^2{}_{\text{Calculated}}\left(A_{\mathrm{t}} \leftarrow B_{\mathrm{t}} \mid B\right) &\equiv \frac{(a-(a+c))^2}{B}+ \\
&\quad \frac{((b+d)-\underline{B})^2}{\underline{B}} \\
&\equiv \frac{c^2}{B}+0 \\
&\equiv \frac{c^2}{B}
\end{aligned} \tag{91}
$$

or equally as

$$
\begin{aligned}
\tilde{\chi}^2{}_{\text{Calculated}}\left(A_{\mathrm{t}} \leftarrow B_{\mathrm{t}} \mid \underline{A}\right) &\equiv \frac{(d-(c+d))^2}{\underline{A}}+ \\
&\quad \frac{((a+b)-A)^2}{A} \\
&\equiv \frac{c^2}{\underline{A}}+0 \\
&\equiv \frac{c^2}{\underline{A}}
\end{aligned} \tag{92}
$$

and can be compared with a theoretical chi-square value at a certain level of significance α. It has not yet been finally clarified whether the use of Yate's (Yates, 1934) continuity correction is necessary at all.

2.3.11. The left-tailed p Value of the conditio sine qua non relationship

Definition 2.36 (The left-tailed p Value of the conditio sine qua non relationship).

The left-tailed (lt) p Value (Barukčić, 2019f) of the conditio sine qua non relationship can be calculated as follows.

$$
\begin{aligned}
pValue_{\mathrm{lt}}\left(A_{\mathrm{t}} \leftarrow B_{\mathrm{t}}\right) &\equiv 1-e^{-\left(1-p\left(A_{\mathrm{t}} \leftarrow B_{\mathrm{t}}\right)\right)} \\
&\equiv 1-e^{-(c/N)}
\end{aligned} \tag{93}
$$

2.3.12. Sufficient condition

Definition 2.37 (Sufficient condition [*Conditio per quam*]).

Mathematically, the sufficient (Barukčić, 2021c, p. 68-70) condition (IMP) relationship, denoted by $p(A_t \rightarrow B_t)$ in terms of statistics and probability theory, is defined (Barukčić, 1989, p. 68-70) as

$$
\begin{aligned}
p(A_t \rightarrow B_t) \equiv p(\underline{A}_t \vee B_t) \equiv \frac{\sum\limits_{t=1}^{N} (\underline{A}_t \vee B_t)}{N} &\equiv \frac{(\underline{A}_t \vee B_t) \times p(\underline{A}_t \vee B_t)}{(\underline{A}_t \vee B_t)} \\
&\equiv p(a_t) + p(c_t) + p(d_t) \\
&\frac{N \times (p(a_t) + p(c_t) + p(d_t))}{N} \\
&\equiv \frac{a+c+d}{N} \equiv \frac{E(\underline{A}_t \vee B_t)}{N} \\
&\equiv \frac{B+d}{N} \equiv \frac{E(A_t \rightarrow B_t)}{N} \\
&\equiv \frac{a+\underline{A}}{N} \\
&\equiv +1
\end{aligned}
\tag{94}
$$

In general, it is $p(A_t \succ\!\!- B_t) \equiv 1 - p(A_t \rightarrow B_t)$ (see Table 19).

2.3.12.1. Mackie's INUS Condition John Leslie Mackie (1917-1981) critically examined the theories of causation of various (see Ducasse, 1926) philosophers such as Hume (Book I, Part III, of the Treatise) (see Mackie, 1974, pp. 3-28), Kant (as well as Kantian approaches offered by Strawson and Bennett), Mill and other. Mackie rightly claims that Hume's regularity theory of causation offer only an incomplete picture of the nature of causation. Mackie writes: "It seems appropriate to begin by examining and criticizing it, so that we can take over from it whatever seems to be defensible but develop an improved account by correcting its errors and deficiencies." (see Mackie, 1974, p. 3). Nonetheless, in his trial to develop an improved account of Hume's theory of causation, Mackie's own account of the nature of causation follows Hume's principles of causation very closely (see Mackie, 1974, pp. 3-28). Mackie himself proposed already in 1965 that "the so-called cause is ... an *insufficient* but *necessary* part of a condition which is itself *unnecessary* but *sufficient* for the result ... let us call such a condition ... an INUS condition." (see Mackie, 1965, p. 245). However Mackie's account needs modification, and can be modified and when it is modified we can explain much more satisfactorily what Mackie ordinarily take to be a cause. Mackie is of the opinion that "... cause is ... part of a condition ... " (see Mackie, 1965, p. 245) and that "... a condition ... is ... *unnecessary* but *sufficient* for the result [i. e. effect, author]. " (see Mackie, 1965, p. 245). To put it very simply one could say that Mackie reduces a cause to a sufficient condition, "... cause is ... a condition which is itself ... *sufficient* ... " (see Mackie, 1965, p. 245). Indeed, there are circumstances, where several different events [30] might be necessary or sufficient et cetera at the same time in order to determine

[30] Barukčić, Ilija. (2022). Conditio per quam. Causation, 17(3), 5–86. https://doi.org/10.5281/zenodo.6369831

a compound/complex sufficient condition relationship. Thus far, it seems appropriate to take over from Mackie's INUS condition whatever seems to be acceptable but to develop an improved account by correcting its deficiencies and errors in order to do justice to the complexity of affairs. Equation 95 illustrates one real-world example of a compound/complex sufficient condition relationship in more detail.

$$
\begin{aligned}
p\left(\left(\left({}_{1}X_{t} \wedge {}_{2}X_{t} \wedge {}_{3}X_{t} \wedge \cdots\right) \wedge A_{t}\right) \rightarrow B_{t}\right) &\equiv p\left(\underline{\left(\left({}_{1}X_{t} \wedge {}_{2}X_{t} \wedge {}_{3}X_{t} \wedge \cdots\right) \wedge A_{t}\right)} \vee B_{t}\right)\\
&\equiv \frac{\sum_{t=1}^{N}\left(\underline{\left(\left({}_{1}X_{t} \wedge {}_{2}X_{t} \wedge {}_{3}X_{t} \wedge \cdots\right) \wedge A_{t}\right)} \vee B_{t}\right)}{N}\\
&\equiv +1
\end{aligned}
\tag{95}
$$

Again, taking into account Kolmogorov's definition of an n-dimensional probability density (see also Kolmogorov, Andreĭ Nikolaevich, 1950, p. 26) of random variables A_t, B_t et cetera at the (period of) time t, we obtain

$$
\begin{aligned}
p\left(A_{t} \rightarrow B_{t}\right) &\equiv +1\\
&\equiv +1 - p\left(b_{t}\right)\\
&\equiv +1 - p\left(A_{t} \cap \underline{B}_{t}\right)\\
&\equiv \left(\int_{-\infty}^{A_t}\int_{-\infty}^{B_t} f\left(A_{t}, B_{t}\right)\, dA_{t}\, dB_{t}\right) + \left(1 - \int_{-\infty}^{A_t} f\left(A_{t}\right)\, dA_{t}\right)
\end{aligned}
\tag{96}
$$

while $p\left(A_{t} \rightarrow B_{t}\right)$ would denote the cumulative distribution function of random variables of a sufficient condition. Another adequate formulation of a sufficient condition is possible too.

Table 19. Sufficient condition.

		Conditioned B_t		
		TRUE	FALSE	
Condition	TRUE	$p(a_t)$	**+0**	$p(A_t)$
A_t	FALSE	$p(c_t)$	$p(d_t)$	$p(\underline{A}_t)$
		$p(B_t)$	$p(\underline{B}_t)$	+1

Remark 2.2. *A sufficient condition A_t is characterized by the property that another event B_t will occur if A_t is given, if A_t itself occured (Barukčić, 1989, 1997, 2005, 2016b, 2017b,c, 2020a,c,d,e, Barukčić and Ufuoma, 2020).* ***Example****. The ground, the streets, the trees, human beings and many other objects too will become wet during heavy rain. Especially,* ***if*** *it is raining (event A_t),* ***then*** *human beings will become wet (event B_t). However, even if this is a common human wisdom, a human being equipped with an appropriate umbrella (denoted by R_t) need not become wet even during heavy rain. An appropriate umbrella (R_t) is similar to an event with the potential to counteract the occurrence of another event (B_t) and can be understood something as an* ***anti-dot*** *of another event. In other words, an appropriate umbrella is an antidote of the effect of rain on human body, an appropriate umbrella has the potential*

to protect humans from the effect of rain on their body. It is a good rule of thumb that the following relationship

$$p(A_t \rightarrow B_t) + p(R_t \wedge B_t) \equiv +1 \tag{97}$$

indicates that R_t is an antidote of A_t. However, taking a shower, swimming in a lake et cetera may make human hair wet too. More than anything else, however, these events does not affect the final outcome, the effect of raining on human body.

The approximate (see Barukčić, 2022a) value of the material implication is given as

$$p(A_t \rightarrow B_t) \geq 1 - \frac{p(b_t)}{p(A_t)} \tag{98}$$

and alternatively as

$$p(A_t \rightarrow B_t) \geq 1 - \frac{p(b_t)}{p(\underline{B}_t)} \tag{99}$$

2.3.13. The Chi square goodness of fit test of a sufficient condition relationship

Definition 2.38 (The $\tilde{\chi}^2$ goodness of fit test of a sufficient condition relationship).

Under some well known circumstances, testing hypothesis about the conditio per quam relationship $p(A_t \rightarrow B_t)$ is possible by the chi-square distribution (also chi-squared or $\tilde{\chi}^2$-distribution) too. The $\tilde{\chi}^2$ goodness of fit test of a conditio per quam relationship with degree of freedom (d. f.) of d. f. = 1 is calculated as

$$\begin{aligned}\tilde{\chi}^2{}_{\text{Calculated}}(A_t \rightarrow B_t \mid A) &\equiv \frac{(a-(a+b))^2}{A} + \\ &\quad \frac{((c+d)-\underline{A})^2}{\underline{A}} \\ &\equiv \frac{b^2}{A} + 0 \\ &\equiv \frac{b^2}{A}\end{aligned} \tag{100}$$

or equally as

$$\begin{aligned}\tilde{\chi}^2{}_{\text{Calculated}}(A_t \rightarrow B_t \mid \underline{B}) &\equiv \frac{(d-(b+d))^2}{\underline{B}} + \\ &\quad \frac{((a+c)-B)^2}{B} \\ &\equiv \frac{b^2}{\underline{B}} + 0 \\ &\equiv \frac{b^2}{\underline{B}}\end{aligned} \tag{101}$$

and can be compared with a theoretical chi-square value at a certain level of significance α. The $\tilde{\chi}^2$-distribution equals zero when the observed values are equal to the expected/theoretical values of the conditio per quam relationship/distribution $p(A_t \rightarrow B_t)$, in which case the null hypothesis is accepted. Yate's (Yates, 1934) continuity correction has not been used in this context.

2.3.14. The left-tailed p Value of the conditio per quam relationship

Definition 2.39 (The left-tailed p Value of the conditio per quam relationship).

The left-tailed (lt) p Value (Barukčić, 2019f) of the conditio per quam relationship can be calculated as follows.

$$\begin{aligned} pValue_{lt}(A_t \rightarrow B_t) &\equiv 1 - e^{-(1-p(A_t \rightarrow B_t))} \\ &\equiv 1 - e^{-(b/N)} \end{aligned} \tag{102}$$

Again, a low p-value indicates a statistical significance.

2.3.15. Necessary and sufficient conditions

Definition 2.40 (Necessary and sufficient conditions [*EQV*]).

The necessary and sufficient condition (EQV) relationship, denoted by $p(A_t \leftrightarrow B_t)$ in terms of statistics and probability theory, is defined(Barukčić, 1989, p. 68-70) as

$$\begin{aligned} p(A_t \leftrightarrow B_t) &\equiv \frac{\sum_{t=1}^{N} \left((A_t \vee \underline{B}_t) \wedge (\underline{A}_t \vee B_t) \right)}{N} \\ &\equiv p(a_t) + p(d_t) \\ &\equiv \frac{N \times (p(a_t) + p(d_t))}{N} \\ &\equiv \frac{a+d}{N} \\ &\equiv +1 \end{aligned} \tag{103}$$

2.3.16. The Chi square goodness of fit test of a necessary and sufficient condition relationship

Definition 2.41 (The $\tilde{\chi}^2$ goodness of fit test of a necessary and sufficient condition relationship).

Even the necessary and sufficient condition relationship $p(A_t \leftrightarrow B_t)$ can be tested by the chi-square distribution (also chi-squared or $\tilde{\chi}^2$-distribution) too. The $\tilde{\chi}^2$ goodness of fit test of a necessary and sufficient condition relationship with degree of freedom (d. f.) of d. f. = 1 is calculated as

$$\begin{aligned}\tilde{\chi}^2{}_{\text{Calculated}}\left(A_{\text{t}} \leftrightarrow B_{\text{t}} \mid A\right) &\equiv \frac{(a-(a+b))^2}{A}+\\ &\quad\frac{d-((c+d))^2}{\underline{A}}\\ &\equiv \frac{b^2}{A}+\frac{c^2}{\underline{A}}\end{aligned} \tag{104}$$

or equally as

$$\begin{aligned}\tilde{\chi}^2{}_{\text{Calculated}}\left(A_{\text{t}} \leftrightarrow B_{\text{t}} \mid B\right) &\equiv \frac{(a-(a+c))^2}{B}+\\ &\quad\frac{d-((b+d))^2}{\underline{B}}\\ &\equiv \frac{c^2}{B}+\frac{b^2}{\underline{B}}\end{aligned} \tag{105}$$

The calculated $\tilde{\chi}^2$ goodness of fit test of a necessary and sufficient condition relationship can be compared with a theoretical chi-square value at a certain level of significance α. Under conditions where the observed values are equal to the expected/theoretical values of a necessary and sufficient condition relationship/distribution $p(A_t \leftrightarrow B_t)$, the $\tilde{\chi}^2$-distribution equals zero. It is to be cleared whether Yate's (Yates, 1934) continuity correction should be used at all.

2.3.17. The left-tailed p Value of a necessary and sufficient condition relationship

Definition 2.42 (The left-tailed p Value of a necessary and sufficient condition relationship).

The left-tailed (lt) p Value (Barukčić, 2019f) of a necessary and sufficient condition relationship can be calculated as follows.

$$\begin{aligned}pValue_{\text{lt}}\left(A_{\text{t}} \leftrightarrow B_{\text{t}}\right) &\equiv 1-e^{-\left(1-p\left(A_{\text{t}} \leftrightarrow B_{\text{t}}\right)\right)}\\ &\equiv 1-e^{-((b+c)/N)}\end{aligned} \tag{106}$$

In this context, a low p-value indicates again a statistical significance. Table 20 may provide an overview of the theoretical distribution of a necessary and sufficient condition.

Table 20. Necessary and sufficient condition.

		Conditioned B_t		
		YES	NO	
Condition A_t	YES	1	0	1
	NO	0	1	1
		1	1	2

2.3.18. Either or conditions

Definition 2.43 (Either A_t or B_t conditions [*NEQV*]).

Mathematically, an either A_t or B_t condition relationship (NEQV), denoted by $p(A_t >\!\!-\!\!< B_t)$ in terms of statistics and probability theory, is defined(Barukčić, 1989, p. 68-70) as

$$
\begin{aligned}
p\left(A_{t} >\!\!-\!\!< B_{t}\right) &\equiv \frac{\sum_{t=1}^{N}\left(\left(A_{t} \wedge \underline{B}_{t}\right) \vee\left(\underline{A}_{t} \wedge B_{t}\right)\right)}{N} \\
&\equiv p\left(b_{t}\right)+p\left(c_{t}\right) \\
&\equiv \frac{N \times\left(p\left(b_{t}\right)+p\left(c_{t}\right)\right)}{N} \\
&\equiv \frac{b+c}{N} \\
&\equiv +1
\end{aligned}
\tag{107}
$$

It is $p\left(A_{t} >\!\!-\!\!< B_{t}\right) \equiv 1-p\left(A_{t} <\!\!=\!\!> B_{t}\right)$ (see Table 21).

Table 21. Either A_t or B_t relationship.

		Conditioned B_t		
		YES	NO	
Condition A_t	YES	0	1	1
	NO	1	0	1
		1	1	2

2.3.19. The Chi-square goodness of fit test of an either or condition relationship

Definition 2.44 (The $\tilde{\chi}^2$ goodness of fit test of an either or condition relationship).

An either or condition relationship $p(A_t >\!\!-\!\!< B_t)$ can be tested by the chi-square distribution (also chi-squared or $\tilde{\chi}^2$-distribution) too. The $\tilde{\chi}^2$ goodness of fit test of an either or condition relationship with degree of freedom (d. f.) of d. f. = 1 is calculated as

$$
\begin{aligned}
\tilde{\chi}^{2}{}_{\text{Calculated}}\left(\left(A_{t} >\!\!-\!\!< B_{t}\right) \mid A\right) &\equiv \frac{(b-(a+b))^{2}}{A}+ \\
&\quad \frac{c-((c+d))^{2}}{\underline{A}} \\
&\equiv \frac{a^{2}}{A}+\frac{d^{2}}{\underline{A}}
\end{aligned}
\tag{108}
$$

or equally as

$$
\begin{aligned}
\tilde{\chi}^2{}_{\text{Calculated}}\left(\left(A_{\mathrm{t}} \succ\!\prec B_{\mathrm{t}}\right) \mid B\right) &\equiv \frac{(c-(a+c))^{\,2}}{B}+\\
&\quad \frac{b-((b+d))^{\,2}}{\underline{B}}\\
&\equiv \frac{a^2}{B}+\frac{d^2}{\underline{B}}
\end{aligned}
\tag{109}
$$

Yate's (Yates, 1934) continuity correction has not been used in this context.

2.3.20. The left-tailed p Value of an either or condition relationship

Definition 2.45 (The left-tailed p Value of an either or condition relationship).

The left-tailed (lt) p Value (Barukčić, 2019f) of an either or condition relationship can be calculated as follows.

$$
\begin{aligned}
pValue_{\mathrm{lt}}\left(A_{\mathrm{t}} \succ\!\prec B_{\mathrm{t}}\right) &\equiv 1-e^{-\left(1-p\left(A_{\mathrm{t}}>-<B_{\mathrm{t}}\right)\right)}\\
&\equiv 1-e^{-((a+d)/N)}
\end{aligned}
\tag{110}
$$

In this context, a low p-value indicates again a statistical significance.

2.4. Causation

2.4.1. Causation in general

The history of the denialism of causality in Philosophy, Mathematics, Statistics, Physics et cetera is very long. We only recall David Hume's (1711-1776) account of causation and his inappropriate reduction of the cause-effect relationship to a simple habitual connection in human thinking or Immanuel Kant's (1724-1804) initiated trial to consider causality as nothing more but a '*a priori*' given category (Langsam, 1994) in human reasoning and other similar attempts too.

It is worth noting in this context that especially Karl Pearson (1857 - 1936) himself has been engaged in a long lasting and never-ending crusade against causation too. "**Pearson categorically denies the need for an independent concept of causal relation beyond correlation ... he *exterminated* causation from statistics before it had a chance to take root** "(see Pearl, 2000, p. 340).

At the beginning of the 20th century notable proponents of **conditionalism** like the German anatomist and pathologist David Paul von Hansemann (Hansemann, David Paul von, 1912) (1858 - 1920) and the biologist and physiologist Max Richard Constantin Verworn(Verworn, 1912) (1863 - 1921) started a new attack(Kröber, 1961) on the principle of causality. In his essay "Kausale und konditionale Weltanschauung"Verworn(Verworn, 1912) presented "an exposition of 'conditionism'as contrasted with 'causalism,'(Unknown, 1913) while ignoring cause and effect relationships completely. "**Das Ding ist also identisch mit der Gesamtheit seiner Bedingungen**."(Verworn, 1912) However, Verworn's goal to exterminate causality completely out of science was hindered by the further development of research.

The history of futile attempts to refute **the principle of causality** culminated in a publication by the German born physicist Werner Karl Heisenberg (1901 - 1976). Heisenberg put forward an illogical, inconsistent and confusing uncertainty principle which opened the door to wishful thinking and logical fallacies in physics and in science as such. Heisenberg's unjustified reasoning ended in an act of a manifestly unfounded conclusion: "**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**."(Heisenberg, Werner Karl, 1927) while 'Gleichung (1)'denotes Heisenberg's uncertainty principle. Einstein's himself, a major contributor to quantum theory and in the same respect a major critic of quantum theory, disliked Heisenberg's uncertainty principle fundamentally while Einstein's opponents used Heisenberg's Uncertainty Principle against Einstein. After the End of the German Nazi initiated Second World War with unimaginable brutality and high human losses and a death toll due to an industrially organised mass killing of people by the German Nazis which did not exist in this way before, Werner Heisenberg visited Einstein in Princeton (New Jersey, USA) in October 1954 (Neffe, 2006). Einstein agreed to meet Heisenberg only for a very short period of time but their encounter lasted longer. However, there where not only a number of differences between Einstein and Heisenberg, these two physicists did not really loved each other. "Einstein remarked that the inventor of the uncertainty principle was a 'big Nazi'... "(Neffe, 2006) Albert Einstein (1879 - 1955) took again the opportunity to refuse to endorse **Heisenberg's uncertainty principle** as a fundamental law of nature and rightly too. Meanwhile, Heisenberg's uncertainty principle is refuted (see Barukčić, 2011a, 2014, 2016a) for several times but still not exterminated completely out of physics and out of science as such.

In contrast to such extreme anti-causal positions as advocated by Heisenberg and the **Copenhagen interpretation of quantum mechancis**, the search for a (mathematical) solution of *the issue of causal inferences* is as old as human mankind itself ("*i. e. Aristotle's Doctrine of the Four Causes*") (Hennig, 2009) even if there is still little to go on.

It is appropriate to specify especially the position of D'Holbach(Holbach, Paul Henri Thiry Baron de, 1770). D'Holbach (1723-1789) himself linked cause and effect or causality as such to changes. "**Une *cause*, est un être qui e met un autre en mouvement, ou qui produit quelque changement en lui. *L'effet* est le changement qu'un corps produit dans un autre ...**"(Holbach, Paul Henri Thiry Baron de, 1770). D'Holbach infers in the following: "**De l'action et de la réaction continuelle de tous les êtres que la nature renferme, il résulte une suite de causes et d'effets ...**"(Holbach, Paul Henri Thiry Baron de, 1770).

With more or less meaningless or none progress on the matter in hand even in the best possible conditions, it is not surprising that authors are suggesting more and more different approaches and models for causal inference. Indeed, the hope is justified that logically consistent *statistical methods of causal inference* can help scientist to achieve so much with so little.

One of the methods of causal inference in Bio-sciences are based on the known *Henle(Henle, 1840) (1809–1885) - Koch(Koch, 1878) (1843–1910) postulates* (Carter, 1985) which are applied especially for the identification of a causative agent of an (infectious) disease. However, the pathogenesis of most chronic diseases is more or less very complex and potentially involves the interaction of several factors. In practice, from the 'pure culture' requirement of the Henle-Koch postulates insurmountable difficulties may emerge. In light of subsequent developments (PCR methodology, immune antibodies et cetera) it is appropriate to review the full validity of the Henle-Koch postulates in our days.

In 1965, Sir Austin Bradford Hill (Hill, 1965) published nine criteria (the '*Bradford Hill Criteria* ') in order to determine whether observed epidemiological associations are causal. Somewhat worrying, is at least the fact that, Hill's "... fourth characteristic is *the temporal relationship of the association* " and so-to-speak just a reformulation of the '*post hoc ergo propter hoc*'*(Barukčić, 1989, Woods and Walton, 1977)* logical fallacy through the back-door and much more then this. It is questionable whether association as such can be treated as being identical with causation. Unfortunately, due to several reasons, it seems therefore rather problematic to rely on Bradford Hill Criteria carelessly.

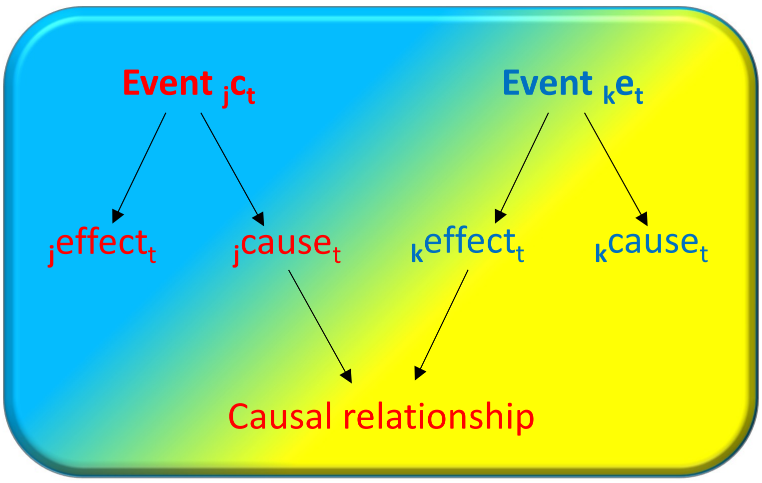
Meanwhile, several other and competing mathematical or statistical approaches for causal inference have been discussed by various modern authors (Barukčić, 1989, 1997, 2005, 2016b, 2017a,c, Bohr, 1937, Chisholm, 1946, Dempster, 1990, Espejo, 2007, Goodman, 1947, Granger, 1969, Hessen, Johannes, 1928, Hesslow, 1976, 1981, Korch, Helmut, 1965, Lewis, David Kellogg, 1973, 1974, Pearl, 2000, Schlick, Friedrich Albert Moritz, 1931, Spohn, 1983, Suppes, 1970, Todd, 1968, Zesar, 2013) or even established (Barukčić, 1989, 1997, 2005, 2016b, 2017a,c). Nevertheless, the question is still not answered, is it at all possible to establish a cause effect relationship between two factors while applying only certain statistical (Sober, 2001) methods?

2.4.2. Cause and effect

Besides all, there are several further aspects of causation for which our attention so far has not been adequately fixed in this context. In the causal relationship, cause and effect are united, a cause is an effect and an effect is a cause.

> "Thus, in the causal relation, cause and effect are inseparable; a cause which had no effect would not be a cause, just as an effect which had no cause would no longer be an effect. "
>
> (see Hegel, Georg Wilhelm Friedrich, 1991, p. 151)

The unity of cause and effect is a unity of two which are not the same. Cause and effect as inseparable in the causal relation are at the same time mutually related as sheer others; each of both as united in its own self to the other of itself is able to passes over into its own other and vice versa. Yet, to approach from a different point of view, a cause and an effect are separated in the same relation too, a cause is not an effect and an effect is not a cause, both are different in the same relation.



"Therefore, though the cause has an effect and is at the same time itself effect, and the effect not only has a cause but is also itself cause, yet the effect which the cause has, and the effect which it is, are different, as are also the cause which the effect has, and the cause which it is." (see Hegel, Georg Wilhelm Friedrich, 1991, p. 565/566)

2.4.2.1. What is a cause, what is an effect? An important fact to which we must pay attention here is that in a causal relation, under certain circumstances, an individual cause and an individual effect are related to each other in their own particular way. An effect which vanishes in its own cause in the same respect equally becomes again in it and vice versa. A cause which is merely extinguished in its own effect becomes again in the same. In fact, each of these determinations presupposes in its own other its own self and constitutes the intimate tie between an individual cause and its own individual effect. Thus far, under conditions of a **positive** causal relationship k, an event U_t which is for sure a cause of another event W_t is at the same time t a necessary and sufficient condition of an event W_t. Table 22 may illustrate this relationship. A matter of great theoretical importance is the fundamental

Table 22. What is the cause, what is the effect?

		Effect W_t		
		TRUE	FALSE	
Cause	TRUE	**+1**	**+0**	$p(U_t)$
U_t	FALSE	**+0**	**+1**	$p(\underline{U}_t)$
		$p(W_t)$	$p(\underline{W}_t)$	+1

relationship between a cause and a condition. Are both, cause and condition, at the end identical? As of now, following Mill (see Mill, 1843a, p. 403), Verworn (see Verworn, 1912), Mackie and others, we can give a clear 'Yes'in reply to this question: "... cause is ... a condition which is itself ... *sufficient* ... " (see Mackie, 1965, p. 245). However, this issue is not as simple as it sounds, according to Mackie. Thus far, it is essential to eliminate some errors. Indeed, there are circumstances where a cause and a condition are identical, a cause and a condition are equivalent. However, as outlined in this publication, both, a cause and a condition, are different too and a cause and a condition are not identical either.

> "Jede Ursache ist nothwendig auch eine Bedingung eines Ereignisses;
> aber nicht jede Bedingung ist Ursache zu nennen. "
>
> (see Bar, Carl Ludwig von, 1871, p. 4)

The crux of the matter is that not every condition is a cause too, in German: "... nicht jede Bedingung ist Ursache ... "(see Bar, Carl Ludwig von, 1871, p. 4). However, and in contrast to a condition, every cause as such is indeed a condition too, in German: "Jede Ursache ist ... auch eine Bedingung ... "(see Bar, Carl Ludwig von, 1871, p. 4). In general, a cause U_t is a necessary condition of an effect W_t. In other words, **without** a cause U_t **no** effect W_t. One consequence of the necessary condition relationship between cause and effect is that "... an effect which had no cause would no longer be an effect. " (see Hegel, Georg Wilhelm Friedrich, 1991, p. 151). However, a cause U_t being a necessary condition of an effect W_t is equivalent to an effect W_t being a sufficient condition of the same cause U_t and vice versa too. In our everyday words,

without

U_t

no

W_t

is equivalent with

if

W_t

then

U_t

and vice versa. As can be seen, there is a kind of strange mirroring between U_t and W_t at the same Bernoulli trial t. Lastly, both are converses of each other too. In other words, U_t's being a necessary condition of W_t's is equivalent to W_t's being a sufficient condition of U_t's (and vice versa). In general, it is

$$(U_t \vee \underline{W}_t) \equiv (\underline{W}_t \vee U_t) \equiv ((U_t \vee \underline{W}_t) \wedge (\underline{W}_t \vee U_t)) \equiv +1 \tag{111}$$

		Effect W_t		
		TRUE	FALSE	
Cause	TRUE	a_t	b_t	U_t
U_t	FALSE	$c_t = 0$	d_t	$\underline{U}_t$
		W_t	$\underline{W}_t$	+1

Table 23. Without U_t no W_t

		Cause U_t		
		TRUE	FALSE	
Effect	TRUE	a_t	$c_t = 0$	W_t
W_t	FALSE	b_t	d_t	$\underline{W}_t$
		U_t	$\underline{U}_t$	+1

Table 24. If W_t then U_t

The other side of the causal relation at the same (period of) time / Bernoulli trial t is the fact that a cause U_t is equally a sufficient condition of an effect W_t too or shortly **if** cause U_t **then** effect W_t. One straightforward consequence of this fundamental relationship between a cause and an effect is that "... a cause which had no effect would not be a cause ... " (see Hegel, Georg Wilhelm Friedrich, 1991, p. 151). But even this is not without difficulties, because a cause U_t being a sufficient condition of an effect W_t is equivalent to effect W_t being a necessary condition of the same cause U_t. In different words,

if

U_t

then

W_t

is equivalent with

without

W_t

no

U_t.

		Effect W_t TRUE	FALSE	
Cause	TRUE	a_t	$b_t = 0$	U_t
U_t	FALSE	c_t	d_t	$\underline{U}_t$
		W_t	$\underline{W}_t$	+1

Table 25. If U_t then W_t

		Cause U_t TRUE	FALSE	
Effect	TRUE	a_t	c_t	W_t
W_t	FALSE	$b_t = 0$	d_t	$\underline{W}_t$
		U_t	$\underline{U}_t$	+1

Table 26. Without W_t no U_t

To bring it to the point, necessary and sufficient conditions are at the end converses (see Gomes, Gilberto, 2009) of each other and far more than this. In fact, there is a kind of reciprocity or mirroring between cause and effect. Necessary and sufficient conditions are relationships used to describe the relationship between two events at the same Bernoulli trial t. In more detail, if U_t then W_t is equivalent with W_t is necessary for U_t, because the truth of U_t guarantees the truth of W_t. In general, it is

$$(\underline{U}_t \vee W_t) \equiv (W_t \vee \underline{U}_t) \equiv ((\underline{U}_t \vee W_t) \wedge (W_t \vee \underline{U}_t)) \equiv +1 \tag{112}$$

In other words, it is impossible to have U_t without W_t (Bloch, 2011). Similarly, U_t is sufficient for W_t, because U_t being true always implies that W_t is true, but U_t not being true does not always imply that W_t is not true. And we should use these relationships to make our point.

In general, **without** gaseous oxygen (U_t), there is **no** burning wax candle (W_t); hence the relationship **if** burning wax candle (W_t) **then** gaseous oxygen (U_t) is equally true and given. This everyday knowledge is known and secured since centuries and might be illustrated as follows.

		Wax candle B_t burning	not burning	
Gaseous	present	a_t	b_t	A_t
oxygen$_t$	not present	$c_t = 0$	d_t	$\underline{A}_t$
		B_t	$\underline{B}_t$	+1

Table 27. Without A_t no B_t

		Gaseous oxygen A_t present	not present	
Wax candle	burning	a_t	$c_t = 0$	B_t
	not burning	b_t	d_t	$\underline{B}_t$
		A_t	$\underline{A}_t$	+1

Table 28. If B_t then A_t

Nonetheless, and independently of this secured everyday knowledge, **a burning wax candle is a sufficient condition of gaseous oxygen but not the cause of gaseous oxygen**.

Given all the circumstances, it is at least this simple **counter-example** which provides us with a convincing evidence that **a sufficient condition alone is not enough to describe a cause completely**. In general, a cause as such cannot be reduced to a simple sufficient condition. In contrast to this obvious fact, other authors prefer another approach to the definition of a cause. "So that, more explicitly, if a given particular event is regarded as having been sufficient to the occurrence of another, it is said to have been its cause; if regarded as having been necessary to the occurrence of another, it is said to have been a condition of it; ..." (see Ducasse, 1926, p. 58). Therefore, in order to be a cause of oxygen, additional evidence is necessary that a burning wax candle is a necessary condition of gaseous oxygen too. However, even if the relationship **without** gaseous oxygen **no** burning wax candle is given, this relationship is not given vice versa. The relationship **without** burning wax candle, **no** gaseous oxygen is not given. Like other fundamental concepts, the concepts of cause and effect can be associated with difficulties too. Under certain conditions, the causal relationship between U_t and W_t, when correctly defined and recognized, is closely allied with the requirement that a certain study or that at least other, different studies provided evidence of a necessary condition between U_t and W_t and of a sufficient condition between U_t and W_t and if possible of **a necessary and sufficient condition** between U_t and W_t too.

Mathematically, a necessary and sufficient condition between U_t and W_t is defined as

$$(U_t \vee \underline{W}_t) \wedge (\underline{U}_t \vee W_t) \equiv +1 \tag{113}$$

However, I think it necessary to make a clear distinction between a necessary and sufficient condition and the converse relationship (Eq. 111) above.

$$((U_t \vee \underline{W}_t) \wedge (\underline{W}_t \vee U_t)) \neq (U_t \vee \underline{W}_t) \wedge (\underline{U}_t \vee W_t) \tag{114}$$

2.4.2.2. The direction of causation In general, a cause is related to its own effect in its own way and vice versa (see Mackie, 1966, p. 160) too. The effect (see Black, 1956) of this cause is itself related to its own cause in some way in which the cause is not related to its own effect (see Dummett and Flew, 1954). This can be considered as one of the reasons why the relation between cause and effect is taken to be asymmetrical.

2.4.2.3. The priority of cause to effect Contemporary discussions of causation are greatly influenced by the causal relation that 'an effect W_t is causally dependent upon a cause U_t'. However, under certain conditions (mono-causality), to say that 'an effect W_t is causally dependent upon a cause U_t'is to say that 'if a cause U_t had not occurred, then an effect W_t would not have occurred too.' (see Lewis, David Kellogg, 1973, 1974). However, what came first, the hen or the egg, the cause or the effect?

2.4.3. Definition causal relationship k

Definition 2.46 (Causal relationship k).

Nonetheless, mathematically, the causal(Barukčić, 2011a,b, 2012) relationship (Barukčić, 1989, 1997, 2005, 2016b, 2017a,c, 2021c) between a cause U_t (German: Ursache) and an effect W_t (German: Wirkung), denoted by k(U_t, W_t), is defined *at each single(Thompson, 2006) Bernoulli trial t* in terms of statistics and probability theory [31], [32], [33] as

$$\begin{aligned} k(U_t, W_t) &\equiv \frac{\sigma(U_t, W_t)}{\sigma(U_t) \times \sigma(W_t)} \\ &\equiv \frac{p(U_t \wedge W_t) - p(U_t) \times p(W_t)}{\sqrt[2]{(p(U_t) \times (1 - p(U_t))) \times (p(W_t) \times (1 - p(W_t)))}} \end{aligned} \tag{115}$$

where σ (U_t , W_t) denotes the co-variance between a cause U_t and an effect W_t *at every single Bernoulli trial t*, σ (U_t) denotes the standard deviation of a cause U_t at the same single Bernoulli trial t, σ (W_t) denotes the standard deviation of an effect W_t at same single Bernoulli trial t. Table 29 illustrates the theoretically possible relationships between a cause and an effect.

Table 29. Sample space and the causal relationship k

		Effect B_t		
		TRUE	FALSE	
Cause	TRUE	$p(a_t)$	$p(b_t)$	$p(U_t)$
A_t	FALSE	$p(c_t)$	$p(d_t)$	$p(\underline{U}_t)$
		$p(W_t)$	$p(\underline{W}_t)$	+1

However, even if one thinks to recognise the trace of Bravais (Bravais, 1846) (1811-1863) - Pearson's (1857-1936) "*product-moment coefficient of correlation*"(Galton, 1877, Pearson, 1896) inside the causal relationship k (Barukčić, 1989, 1997, 2005, 2016b, 2017a,c) both are completely different. According to Pearson: "*The fundamental theorems of correlation were for the first time and almost exhaustively discussed by B r a v a i s ('Analyse mathematique sur les probabilities des erreurs de situation d'un point.' Memoires par divers Savans, T. IX., Paris, 1846, pp. 255-332) nearly half a century ago.*"(Pearson, 1896) Neither does it make much sense to elaborate once again on the issue causation(Blalock, 1972) and correlation, since both are not identical (Sober, 2001) nor does it make sense to insist on the fact that "*Pearson's philosophy discouraged him from looking too far behind phenomena.*"(Haldane, 1957) Whereas it is essential to consider that the causal relationship k, in contrast to Pearson's product-moment coefficient of correlation(Pearson, 1896) or to Pearson's phi

[31] Ilija Barukčić, "The Mathematical Formula of the Causal Relationship k," International Journal of Applied Physics and Mathematics vol. 6, no. 2, pp. 45-65, 2016. https://doi.org/10.17706/ijapm.2016.6.2.45-65

[32] Barukčić, Ilija. (2015). The Mathematical Formula Of The Causal Relationship k. https://doi.org/10.5281/zenodo.3944666

[33] Ilija Barukčić. The causal relationship k. MATEC Web Conf., 336 (2021) 09032 DOI: https://doi.org/10.1051/matecconf/202133609032

coefficient(Pearson, 1904b), is defined at every single Bernoulli trial t. This might be a very *small* difference. However, even a small difference might determine a difference. However, in this context and in any case, this small difference *makes*(Barukčić, 2018a) the difference.

2.5. Axioms

Whether science needs new and obviously generally valid statements (axioms) which are able to assure the truth of theorems proved from them may remain an unanswered question. In order to be accepted, a new axiom candidate (see Easwaran, 2008) should be at least as simple as possible and logically consistent to enable advances in our knowledge of nature. The importance of axioms is particularly emphasized by Albert Einstein. "**Die wahrhaft großen Fortschritte der Naturerkenntnis sind auf einem der Induktion fast diametral entgegengesetzten Wege entstanden**." (see Einstein, 1919, p. 17). In general, lex identitatis, lex contradictionis and lex negationis have the potential to denote the most simple, the most general and the most far-reaching axioms of science, the foundation of our today's and of our future scientific inquiry.

2.5.1. Principium identitatis (Axiom I)

Principium identitatis or **lex identitatis** or axiom I, is closely related to central problems of metaphysics, epistemology and of science as such. It turns out that it is more than rightful to assume that

$$+1 \equiv +1 \tag{116}$$

is true, otherwise there is every good reason to suppose that nothing can be discovered at all.

Identity as the epitome of a self-identical is at the same time different from difference, identity is free from difference, identity is at the same time the other of itself, identity is not difference. Identity is in its very own nature different, it is in its own self the opposite of itself (symmetry). It is equally

$$-1 \equiv -1 \tag{117}$$

In general, +1 and -1 are distinguished, however these distinct are related to one and the same 1. Identity as a vanishing of otherness, therefore, is this distinguishedness in one relation. It is

$$0 \equiv +1 - 1 \equiv 0 \times 1 \equiv 0 \tag{118}$$

Identity, as the unity of something and its own other is in its own self a separation from difference, and as a moment of separation might pass over into an equivalence relation which itself is reflexive, symmetric and transitive. Nonetheless, backed by thousands of years of often bitter human experience, the scientific development has taught us all that human knowledge is relative too. Even if experiments and other suitable proofs are of help to encourage us more and more in our belief of the correctness of a theory, it is difficult to prove the correctness of a theorem or of a theory et cetera once and for all. The challenge for all the science is the need to comply with Einstein's position: **"Niemals aber kann die Wahrheit einer Theorie erwiesen werden. Denn niemals weiß man, daß auch in Zukunft eine Erfahrung bekannt werden wird, die Ihren Folgerungen widerspricht..."** (Einstein, 1919). Albert Einstein's position translated into English: 'But the truth of a theory can never be proven. For one never knows if future experience will contradict its conclusion; and furthermore, there are always other conceptual systems imaginable which might coordinate the very same facts.'Our human experience tells us that everything in life is more or less transitory, and that nothing lasts. As a result of our knowledge and experience, several scientific theories have a glorious past to look back on, but all the glory of such scientific theories might remain in the past if scientist don't continue to innovate. In a word, theories can be refuted by time.

> "No amount of experimentation can ever prove me right;
> **a single experiment can prove me wrong**."
>
> (Albert Einstein according to: Robertson, 1998, p. 114)

In the light of the foregoing, it is clear that appropriate axioms and conclusions derived from the same are a main logical foundation of any 'theory'.

> "**Grundgesetz (Axiome)** und **Folgerungen** zusammen bilden das was man **eine 'Theorie'** nennt. "
>
> (Einstein, 1919)

However, another point is worth being considered again. One single experiment can be enough to refute a whole theory. Albert Einstein's (1879-1955) message translated into English as: *Basic law (axioms) and conclusions together form what is called a 'theory'* has still to get round. However, an axiom as a free creation of the human mind which precedes all science should be like all other axioms, as simple as possible and as self-evident as possible. Historically, the earliest documented use of **the law of identity** can be found in Plato's dialogue Theaetetus (185a) as "... each of the two is different from the other and the same as itself "[34] . However, Aristotle (384–322 B.C.E.), Plato's pupil and equally one of the greatest philosophers of all time, elaborated on the law of identity too. In Metaphysica, Aristotle wrote:

> "... all things ... have some unity and identity. "
>
> (see Aristotle, of Stageira (384-322 B.C.E), 1908, Metaphysica, Chapter IV, 999a, 25-30, p. 66)

In Prior Analytics, [35] , [36] Aristotle, a tutor Alexander, the thirteen-year-old son of Philip, the king of Macedon, is writing: "When A applies to the whole of B and of C, and is other predicated of nothing

[34] Plato's dialogue Theaetetus (185a), p. 104.
[35] Aristotle, Prior Analytics, Book II, Part 22, 68a
[36] Kenneth T. Barnes. Aristotle on Identity and Its Problems. Phronesis. Vol. 22, No. 1 (1977), pp. 48-62 (15 pages)

else, and B also applies to all C, A and B must be convertible. For since A is stated only of B and C, and B is predicated both of itself and of C, it is evident that B will also be stated of all subjects of which A is stated, except A itself. "[37] , [38] For the sake of completeness, it should be noted at the outset that Aristotle himself preferred **the law of contradiction** and **the law of excluded middle** as examples of fundamental axioms. Nonetheless, it is worth noting that **lex identitatis** is an axiom too, which possess the potential to serve as the most basic and equally the most simple axiom of science but has been treated by Aristotle in an inadequate manner without having any clear and determined meaning for Aristotle himself. Nonetheless, something which is really just itself is equally different from everything else. In point of fact, is such an equivalence (Degen, 1741) which everything has to itself inherent or must the same be constructed by human mind and consciousness. Can and how can something be **identical with itself** (Förster and Melamed, 2012, Hegel, Georg Wilhelm Friedrich, 1812a, Koch, 1999, Newstadt, 2015) and in the same respect different from itself. An increasingly popular view on identity is the one advocated by Gottfried Wilhelm Leibniz (1646-1716):

> **"Chaque chose est ce qu'elle est. Et dans autant d'exemples qu'on voudra**
> **A est A,**
> **B est B**. "
> (Leibniz, 1765, p. 327)

or **A = A**, **B = B** or **+1 = +1**. In other words, a thing is what it is (Leibniz, 1765, p. 327). Leibniz' **principium identitatis indiscernibilium** (p.i.i.), the principle of the indistinguishable, occupies a central position in Leibniz' logic and metaphysics and was formulated by Leibniz himself in different ways in different passages (1663, 1686, 1704, 1715/16). All in all, Leibniz writes:

> "C'est
> le principe des indiscernables,
> en vertu duquel
> il ne saurait exister dans la nature deux êtres identiques.
> ...
> Il n'y a point deux individus indiscernables. "
> (see Leibniz, Gottfried Wilhelm, 1886, p. 45)

Exactly in complete compliance with Leibniz, Johann Gottlieb Fichte (1762 - 1814) elaborates on this subject as follows:

[37] Aristotle, Prior Analytics, Book II, Part 22, 68a, p. 511.
[38] Ivo Thomas. On a passage of Aristotle. Notre Dame J. Formal Logic 15(2): 347-348 (April 1974). DOI: 10.1305/ndjfl/1093891315

"Each thing is what it is ;
it has those realities which are posited when it is posited,
(A = A.) "
(Fichte, 1889)

Hegel preferred to reformulate an own version of Leibnitz principium identitatis indiscernibilium in his own way by writing that "All things are different, or: there are no two things like each other. "(see Hegel, Georg Wilhelm Friedrich, 1991, p. 422). Much of the debate about identity is still a matter of controversy. This issue has attracted the attention of many authors and has been discussed by Hegel too. In this context, it is worth to consider Hegel's radical position on identity.

"The other expression of the law of identity: A cannot at the same time be A and not-A, has a negative form; it is called
the law of contradiction. "
(Hegel, Georg Wilhelm Friedrich, 1991, p. 416)

We may, usefully (see Barukčić, 2019a), state Russell's position with respect to the identity law as mentioned in his book 'The problems of philosophy ' (see Russell, 1912). In particular, according to Russell,

"...principles have been singled out by tradition under the name of 'Laws of Thought.' They are as follows:
(1) **The law of identity**: 'Whatever is,is.
(2)**The law of contradiction**: 'Nothing can both be and not be.'
(3) The law of excluded middle: 'Everything must either be or not be.'
These three laws are samples of self-evident logical principles, but are not really more fundamental or more self-evident than various other similar principles: for instance, the one we considered just now, which states that what follows from a true premise is true. The name 'laws of thought' is also misleading, for what is important is not the fact that we think in accordance with these laws, but the fact that **things behave in accordance with them**; "

(see Russell, 1912, p. 113)

Russell's critique, that we tend too much to focus only on the formal aspects of the 'Laws of Thoughts' with the consequence that "... we thing in accordance with these laws" (see Russell, 1912, p. 113) is justified. Judged solely in terms of this aspect, it is of course necessary to think in accordance with the 'Laws of Thoughts'. But this is not the only aspect of the 'Laws of Thoughts'. The other and may be much more important aspect of these 'Laws of Thoughts'is the fact that quantum mechanical objects or that "... things behave in accordance with them" (see Russell, 1912, p. 113).

2.5.2. Principium contradictionis (Axiom II)

Principium contradictionis or **lex contradictionis** [39], [40], [41] or axiom II, the other of lex identitatis, the negative of lex identitatis, the opposite of lex identitatis, a complementary of lex identitatis, can be expressed mathematically as

$$+0 \equiv 0 \times 1 \equiv +1 \tag{119}$$

In addition to the above, from the point of view of mathematics, axiom II (equation 119) is equally the most simple mathematical expression and formulation of a contradiction. However, there is too much practical and theoretical evidence that a lot of 'secured'mathematical knowledge and rules differ too generously from real world processes, and the question may be asked whether mathematical truths can be treated as absolute truths at all. Many of the basic principle of today's mathematics allow every single author defining the real world events and processes et cetera in a way as everyone likes it for himself. Consequentially, a resulting dogmatic epistemological subjectivism and at the end agnosticism too, after all, is one of the reasons why we should rightly heed the following words of wisdom of Albert Einstein.

"I don't believe in mathematics."

(Albert Einstein cited according to Brian, 1996, p. 76)

In the long term, however, the above attitude of mathematics is not sustainable. History has taught us time and time again that objective reality has the potential to correct wrong human thinking slowly but surely, and many more than this. Objective reality has demonstrably corrected wrong human thinking again and again in the past.

Despite all the adversities, it is necessary and crucial to consider that a self-identical as the opposite of itself is no longer only self-identity but a difference of itself from itself within itself. In other words, in opposition, a self-identical is able to return into simple unity with itself with the consequence that even as a self-identical the same self-identical is inherently self-contradictory. A question of fundamental theoretical importance is, however, why should something be itself and at the same time

[39] Horn, Laurence R., "Contradiction", The Stanford Encyclopedia of Philosophy (Winter 2018 Edition), Edward N. Zalta (ed.), URL = https://plato.stanford.edu/archives/win2018/entries/contradiction/.

[40] Barukčić I. Aristotle's law of contradiction and Einstein's special theory of relativity. Journal of Drug Delivery and Therapeutics (JDDT). 15Mar.2019;9(2):125-43. https://jddtonline.info/index.php/jddt/article/view/2389

[41] Barukčić, Ilija. (2020, December 28). The contradiction is exsiting objectively and real (Version 1). Zenodo. https://doi.org/10.5281/zenodo.4396106

the other of itself, the opposite of itself, not itself? Is something like this even possible at all and if so, why and how? These and similar questions have occupied many thinkers, including Hegel.

"Something is therefore
alive only in so far as it contains contradiction within it,
and moreover is this power to
hold and endure the contradiction within it. "

(see Hegel, Georg Wilhelm Friedrich, 1991, p. 440)

However, as directed against identity, contradiction itself is also at the same time a source of self-changes out of itself of a self-identical.

"... contradiction
is the root of all movement and vitality;
it is only in so far as something has a contradiction within it
that it moves, has an urge and activity. "

(see Hegel, Georg Wilhelm Friedrich, 1991, p. 439)

The further advance of science will throw any contribution to scientific progress of each of us back into scientific insignificance, as long as principium contradictionis is not given enough and the right attention. **The contradiction [42] is existing objectively and real and is the heartbeat of every self-identical.** We have reason to be delighted by the fact that very different aspects of principium contradictionis have been examined since centuries from different angles by various authors. According to Aristotle, principium contradictionis applies to everything that is.

"... the same ... cannot at the same time belong and not belong to the same
... in the same respect ... This, then, is
the most certain of all principles "

(see Aristotle, of Stageira (384-322 B.C.E), 1908, Metaph., IV, 3, 1005b, 16–22)

Principium contradictionis or axiom II has many facets. As long as we follow Leibniz in this regard, we should consider that "**Le principe de contradiction est en general ...** "(Leibniz, 1765, p. 327). Scientist inevitably have false beliefs and make mistakes. In order to prevent scientific results from falling into logical inconsistency or logical absurdity, it is necessary to posses among other the methodological possibility to start a reasoning with a (logical) contradiction too. However and in contrast to the way of reasoning with inconsistent premises as proposed by para-consistent (Carnielli and

[42] Barukčić, Ilija. (2020, December 28). The contradiction is existing objectively and real (Version 1). Zenodo. https://doi.org/10.5281/zenodo.4396106

Marcos, 2001, da Costa, 1974, 1958, Priest, 1998, Priest et al., 1989, Quesada, 1977) and other logic, in the absence of technical and other errors of reasoning, the contradiction itself need to be preserved. In other words, **from a contradiction does not anything follows but the contradiction itself** while the theoretical question is indeed justified "What is so Bad about Contradictions? " (Priest, 1998). Historically, **the principle of (deductive) explosion** (Carnielli and Marcos, 2001, Priest, 1998, Priest et al., 1989), coined by 12th-century French philosopher William of Soissons, demand us to accept that anything, including its own negation, can be proven or can be inferred from a contradiction. In short, according to **ex falso sequitur quodlibet**, a (logical) contradiction implies anything. Respecting the principle of explosion, the existence of a contradiction (or the existence of logical inconsistency) in a scientific theorem, rule et cetera is disastrous. However, the historical development of science shows that scientist inevitably revise the theories, false positions and claims are identified once and again, and we all make different kind of mistakes. In order to avert disproportionately great damage to science and to prevent reducing science into pure subjective belief, a negation of the principle of explosion is required. Nonetheless, a justified negation of the **ex contradictione quodlibet principle** (Carnielli and Marcos, 2001) does not imply the correctness of para consistent logic (Carnielli and Marcos, 2001, da Costa, 1974, 1958, Priest, 1998, Priest et al., 1989, Quesada, 1977) as such as advocated especially by the Peruvian philosopher Francisco Miró Quesada (Quesada, 1977) and other (Carnielli and Marcos, 2001, da Costa, 1974, 1958, Priest, 1998, Priest et al., 1989). In general, scientific theories appear to progress from lower and simpler to higher and more complex levels. However, high level theories cannot be taken for granted because high level theories are grounded on a lot of assumptions, definitions and other procedures and may rest upon too much erroneous stuff even if still not identified. Therefore, it should be considered to check at lower at simpler levels like with like.

2.5.3. Principium negationis (Axiom III)

Lex negationis or axiom III, is often mismatched with simple opposition. However, from the point of view of philosophy and other sciences, identity, contradiction, negation and similar notions are equally mathematical descriptions of the most simple laws of objective reality. What sort of natural process is negation at the end? Mathematically, we define principium negationis or lex negationis or axiom III as

$$\text{Negation}(0) \times 0 \equiv \neg(0) \times 0 \equiv +1 \tag{120}$$

where $\neg$ denotes (logical (Boole, 1854) or natural) negation (Ayer, 1952, Förster and Melamed, 2012, Hedwig, 1980, Heinemann, Fritz H., 1943, Horn, 1989, Koch, 1999, Kunen, 1987, Newstadt, 2015, Royce, 1917, Speranza and Horn, 2010, Wedin, 1990b). In this context, there is some evidence that

$$\text{Negation}(1) \times 1 \equiv \neg(1) \times 1 = 0 \tag{121}$$

Logically, it follows that

$$\text{Negation}(1) \equiv 0 \tag{122}$$

In the following we assume that axiom I is universal. Under this assumption, the following theorem follows inevitably.

Theorem 2.1 (Zero divided by zero)**.** *According to classical logic, it is*

$$\frac{0}{0} \equiv 1 \tag{123}$$

Proof by direct proof. The premise

$$1 \equiv 1 \tag{124}$$

is true. It follows that

$$\begin{aligned} 0 &\equiv 0 \\ &\equiv 0 \times 1 \end{aligned} \tag{125}$$

In the following, we rearrange the premise (see equation 120, p. 67). We obtain

$$0 \times (\text{Negation}(0) \ \times 0) \equiv 0 \tag{126}$$

Equation 126 changes slightly (see equation 121, p. 67). It is

$$(\text{Negation}(1) \ \times 1) \times (\text{Negation}(0) \ \times 0) \equiv 0 \tag{127}$$

Equation 127 demands that

$$(\text{Negation}(1) \ ) \times (\text{Negation}(0) \ ) \times 0 \equiv 0 \tag{128}$$

Equation 128 is logically possible (see equation 118, p. 61) only if

$$(\text{Negation}(1) \ ) \times (\text{Negation}(0) \ ) \equiv 1 \tag{129}$$

Whatever the meaning of Negation(1) or of Negation(0) might be, equation 129 demands that

$$\text{Negation}(0) \ \equiv \frac{1}{\text{Negation}(1)} \tag{130}$$

and that

$$\text{Negation}(1) \equiv \frac{1}{\text{Negation}(0)} \tag{131}$$

Equation 130 simplifies as (see equation 122, p. 67)

$$\begin{aligned} \text{Negation}(0) \ &\equiv \frac{+1}{\text{Negation}(1)} \\ &\equiv \frac{+1}{+0} \end{aligned} \tag{132}$$

It follows that

$$\neg(0) \times 0 \equiv \frac{1}{0} \times 0 \equiv \frac{0}{0} \equiv 1 \tag{133}$$

To bring it to the point. Classical logic assumed to be universally valid would also be valid for the mathematical sciences too and demands without any possibility of beating around the bush that

$$\frac{0}{0} \equiv 1 \tag{134}$$

□

Concepts like identity, difference, negation, opposition et cetera engaged the attention of scholars at least over the last twenty-three centuries (see also Horn, 1989, Speranza and Horn, 2010). As long as we first and foremost follow Josiah Royce, negatio or negation "is one of the simplest and most fundamental relations known to the human mind. For the study of logic, no more important and fruitful relation is known." (see also Royce, 1917, p. 265) But, do we really know what, for sure, what negation is? Based on what we know about negation, Aristotle (see also Wedin, 1990a) has been one of the first to present a theory of negation, which can be found in discontinuous chunks in his works the Metaphysics, the Categories, De Interpretatione, and the Prior Analytics (see also Horn, 1989, p. 1). Negation (see also Newstadt, 2015) as a fundamental philosophical concept found its own very special melting point especially in Hegel's dialectic and is more than just a formal logical process or operation which converts true to false or false to true. Negation as such is a natural process too and equally '**an engine of changes of objective reality** " (see also Barukčić, 2019a). However, it remains an open question to establish a generally accepted link between this fundamental philosophical concept and an adequate counterpart in physics, mathematics and mathematical statistics et cetera. Especially the relationship between creation and conservation or creatio ex nihilio (see also Donnelly, 1970, Ehrhardt, 1950, Ford, 1983), determination and negation (see also Ayer, 1952, Hedwig, 1980, Heinemann, Fritz H., 1943, Kunen, 1987) has been discussed in science since ancient (see also Horn, 1989, Speranza and Horn, 2010) times too. Why and how does an event occur or why and how is an event created (creation), why and how does an event maintain its own existence over time (conservation)? The development of the notion of negation leads from Aristotle to Meister Eckhart (see also Eckhart, 1986) von Hochheim (1260-1328), commonly known as Meister Eckhart (see also Tsopurashvili, 2012) or Eckehart, to Spinoza (1632 – 1677), to Immanuel Kant (1724-1804) and finally to Georg Wilhelm Friedrich Hegel (1770-1831) and other authors too. One point is worth being noted, even if it does not come as a surprise, it was especially Benedict de Spinoza (1632 – 1677) as one of the philosophical founding fathers of the Age of Enlightenment who addressed the relationship between determination and negation in his lost letter of June 2, 1674 to his friend Jarig Jelles (see also Förster and Melamed, 2012) by the discovery of his fundamental insight that " **determinatio negatio est**" (see also Spinoza, 1674, p. 634). Hegel went even so far as to extended the slogan raised by Spinoza into to "Omnis determinatio est negatio" (see also Hegel, Georg Wilhelm Friedrich, 1812b, 2010, p. 87). Finally, it did not take too long, and the notion of negation entered the world of mathematics and mathematical logic at least with Boole's (see also Boole, 1854) publication in the year 1854. "Let us, for simplicity of conception, give to the symbol x the particular interpretation of men, then 1 - x will represent the class of 'not-men'." (see also Boole, 1854, p. 49). Finally, the philosophical notion negation found its own way into physics by the contributions of authors like Woldemar Voigt (see Voigt, 1887), George Francis FitzGerald (see FitzGerald, 1889), Hendrik Antoon Lorentz (see Lorentz, 1892, 1899), Joseph Larmor (see Larmor, 1897), Jules Henri Poincaré (see Poincaré, 1905) and Albert Einstein (see Einstein, 1905) by contributions to the physical notion "Lorentz factor".

3. Results

3.1. High blood pressure and coronary artery disease

For our purposes, we examine the relationship between high blood pressure and coronary artery disease in the form of a thought experiment, a purely theoretically experiment. Children up to the age of 6 years and a systolic blood pressure (SBP) of less than 90 mm Hg on the one side (placebo) and adults from the age of 30 years and more and a systolic blood pressure of more than 90 mm Hg on the other side (verum) are investigated with (multislice) computed tomography (MSCT) [43], [44] to detect and quantify coronary artery calcium levels.

Null-Hypothesis:

The data support the Null-Hypothesis:

without high blood pressure, **no** coronary artery disease.

Alternative-Hypothesis:

The data do not support the Null-Hypothesis:

without high blood pressure, **no** coronary artery disease.

The theoretical data obtained are the following (see table 30).

[43] Agatston AS, Janowitz WR, Hildner FJ, Zusmer NR, Viamonte M Jr, Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. J Am Coll Cardiol. 1990 Mar 15;15(4):827-32. doi: 10.1016/0735-1097(90)90282-t. PMID: 2407762.

[44] Budoff MJ, Young R, Burke G, Jeffrey Carr J, Detrano RC, Folsom AR, Kronmal R, Lima JAC, Liu KJ, McClelland RL, Michos E, Post WS, Shea S, Watson KE, Wong ND. Ten-year association of coronary artery calcium with atherosclerotic cardiovascular disease (ASCVD) events: the multi-ethnic study of atherosclerosis (MESA). Eur Heart J. 2018 Jul 1;39(25):2401-2408. doi: 10.1093/eurheartj/ehy217. PMID: 29688297; PMCID: PMC6030975.

Table 30. SBP > 90 mm Hg and CAD.

		CAD		
		YES	NO	
SBP > 90 mm Hg	YES	300	700	1000
	NO	0	300	300
		300	1000	1300

STATISTICAL ANALYSIS.	
Causal relationship k =	+0,3000000000
p Value right tailed (HGD) =	0,0000000000
p (SINE) =	1,0000000000
$\tilde{\chi}^2$ (SINE — B_t) =	0,0000
$\tilde{\chi}^2$ (SINE — $\underline{A}_t$) =	0,0000
p Value right tailed (HGD) =	0,0000
p Value (SINE) =	0,0000000000
RELATIVE RISK (RR).	
RR (nc) =	Division by zero!
RR (sc) =	1,4286
ADDITIONAL MEASURES.	
OR =	0,4615
IOR =	0,3000
STUDY DESIGN.	
p(IOU)=	0
p(IOI)=	0,538461538

The study design was fair (p(IOU) = 0), i.e. the data are basically suitable for examining the relationship of a necessary condition. The causal relationship is positive (k > + 0). Overall, the data cannot be regarded as contradictory or as biased. The data support the null hypothesis, **without** high blood pressure, **no** coronary artery disease (P-Value = 0.0).

4. Discussion

Our theoretical investigations confirmed the relationship between high blood pressure and coronary artery disease. Consequently, we need to consider whether there is a causal relationship between high blood pressure and coronary artery disease. In any case, a statistically significant necessary condition relationship between high blood pressure and coronary artery disease is proofed. **Without** high blood pressure, **no** coronary artery disease (P Value = 0.0).

However, taking the previous thought experiment seriously require just a few comments. A critical reader might rise the fundamental question whether these two different age groups can be compared at all. Under normal conditions, the answer would be of course not and by no means, due to many well-known reasons. However, the matter is by far not that simple.

The relationship between gaseous oxygen and human life is secured everyday knowledge. Neither children nor adults can exist without gaseous oxygen. Therefore, if we would examine the relationship between gaseous oxygen and human life, it would be permissible to compare the two groups above for sure. **Without** gaseous oxygen, **no** human life, it does not matter whether children up to the age of 6 years are compared with adults from the age of 30 years and more or not, **without** gaseous oxygen, **no** life of children up to the age of 6 years, **without** gaseous oxygen, **no** life of adults from the age of 30 years and more. In other words, if high blood pressure is a necessary condition of the coronary artery disease, it is the case in children up to the age of 6 years as well as in adults from the age of 30 years and over. Genetic or other factors which protect children from coronary artery disease are not known to date. Also, age itself is not a necessary condition of a coronary artery disease. There are enough old people who do not suffer from coronary artery disease, while only one of the old people free of coronary artery disease would suffice to serve as a counterexample against such a hypothesis. The definitive assessment is that a comparison of these two groups is permitted within the framework of the issue investigated.

However, various factors which can contribute to an increase in blood pressure need to be taken into account. This might include a human cytomegalovirus infection (see Barukčić, 2019b, 2020b) too.

5. Conclusion

High blood pressure is a necessary condition of coronary artery disease, **without** high blood pressure, **no** coronary artery disease (P Value = 0.0).

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6. Patient consent for publication

Not required.

Conflict of interest statement

No conflict of interest to declare.

Private note

The definition section of a paper need not and does not necessarily contain new scientific aspects. Above all, it also serves to better understand a scientific publication, to follow every step of the arguments of an author and to explain in greater details the fundamentals on which a publication is based. Therefore, there is no objective need to force authors to reinvent a scientific wheel once and again unless such a need appears obviously factually necessary. The effort to write about a certain subject in an original way in multiple publications does not exclude the necessity simply to cut and paste from an earlier work, and has nothing to do with self-plagiarism. However, such an attitude cannot simply be transferred to the sections' introduction, results, discussion and conclusions et cetera.

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