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Index of relationship

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ABSTRACT

Aim.

Adding another measure of relationship to the numerous already known measures of relationship makes only sense if the new one measure of relationship brings some advantages over the already known one.

Methods.

A new statistical method, the index of relationship (IOR), is developed which goes far beyond the odds ratio. As an example, the study of Loeb et al. (see also Loeb et al. 2017) has been re-analysed.

Results.

Evidence from different studies on the relationship between testosterone and prostate cancer has been inconsistent. Especially an exposure to testosterone and prostate cancer risk is very controversial. However, the data of the study of Loeb et al. (see also Loeb et al. 2017) support the null-hypothesis:

without sexual activity **no** prostate cancer.

Conclusion.

Human prostate cancer is a sexually transmitted disease.

Keywords: Testosterone — prostate cancer — relationship

1. INTRODUCTION

The hypothesis of testosterone dependence of prostate cancer has been established by Charles Huggins in the year 1941 and later by other too. Huggins et al. (see also Huggins and Hodges 1941) wrote: "**... the activity of phosphatases in serum were found to provide objective indices of activity of the neoplasm when the enzymes were increased in amount above normal ... the acid phosphatase of serum is reduced in metastatic carcinoma of the prostate by decreasing the activity of androgens through castration or estrogenic injections and ... this enzyme is increased by injecting androgens ... Testosterone propionate caused an increase of serum acid phosphatase above the preinjection level in these patients. Following the cessation of injections, there was a decline to the preliminary level**. "As a result, medical or surgical castration became a first-line treatment of advanced prostate cancer and the male hormone testosterone were demonised. But, it is undeniable that testosterone has various(see also Emmelot-Vonk et al. 2008) especially positive effects on human body including sexual activity, sexual desire, erectile function(see also Rastrelli et al. 2018) and of course on human soul(see also Nead 2019) too. Thus it is hardly surprising that commercial sales(see also Baillargeon et al. 2013) of testosterone have increased substantially in recent years. However, following Huggins et al. (see also Huggins and Hodges

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1941), high levels of circulating testosterone might increase the risk of prostate cancer. The crux of the matter here is: what is true and what is false? Following several meta-analysis the uncertainty about testosterone is rapidly disappearing and vanishing from the unknown into the known. **Testosterone does not promote prostate cancer development**(see also Cui et al. 2014; Bruzzese et al. 2014; Loeb et al. 2017) . Therefore, the question is justified, is the male hormone testosterone at the end better than its current reputation? The new measure of relationship, index of relationship (IOR), is exemplified by investigating the relationship between testosterone and prostate cancer too.

2. MATERIAL AND METHODS

From the beginning of statistics onward the same is interrelated with probability theory. However, what kinds of 'things'are probabilistic statements, or more generally under which circumstances are probabilistic statements true or false and to what extent?

2.1. *Material*

The subject of study in statistics is among other the relation between data and hypotheses. Summing up, it remains problematic to study anything without some definitions.

2.1.1. *Definitions*

Contingency table —

Definition 2.1 (Contingency table)**.**

The relationship between two Binomial or Bernoulli(Barukčić 2021a) [1] distributed random variables A_t and B_t at a certain Bernoulli trial (or period of time) t can be illustrated by a 2 by 2 table. Furthermore, a 2 by 2 contingency table is able to provide a basic picture of the interrelation between two binomial distributed random variables and is of use to analyse the relationships between them in detail. Karl Pearson was the first to use the term contingency table in his paper "On the Theory of Contingency and Its Relation to Association and Normal Correlation"[2].

Relative risk		Outcome		Total
		YES	NO	
Exposed	YES	$p(a_t)$	$p(b_t)$	$p(A_t)$
	NO	$p(c_t)$	$p(d_t)$	$p(\underline{A}_t)$
	Total	$p(B_t)$	$p(\underline{B}_t)$	+1

where $p(a_t)$ denotes the joint probability of A_t and B_t, $p(b_t)$ denotes the joint probability of A_t and Not B_t, $p(c_t)$ denotes the joint probability of not A_t and B_t and $p(d_t)$ denotes the joint probability of not A_t and Not B_t.

Definition 2.2 (Basic relationships between probabilities of a 2 by 2 table)**.**

In general, it is

$$p(A_t) = p(a_t) + p(b_t) \tag{1}$$

[1] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html
[2] https://archive.org/details/cu31924003064833/page/n2/mode/2up

and

$$p(NotA_t) = 1 - p(A_t) = p(c_t) + p(d_t) \tag{2}$$

and

$$p(B_t) = p(a_t) + p(c_t) \tag{3}$$

and

$$p(NotB_t) = 1 - p(B_t) = p(b_t) + p(d_t) \tag{4}$$

where $p(a_t)$ denotes the joint probability of A_t and B_t. In general, it is

$$p(a_t) + p(b_t) + p(c_t) + p(d_t) = +1 \tag{5}$$

In general, the expectation values of $E(A_t)$, $E(\underline{A}_t)$, $E(B_t)$, $E(\underline{B}_t)$, N, a, b, c, d, follow under certain conditions as

$$\begin{aligned} E(A_t) \equiv A &\equiv N \times (p(A_t)) \\ &\equiv N \times (p(a_t) + p(b_t)) \\ &\equiv a + b \end{aligned} \tag{6}$$

and

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

and

$$\begin{aligned} E(B_t) \equiv B &\equiv N \times (p(B_t)) \\ &\equiv N \times (p(a_t) + p(c_t)) \\ &\equiv a + c \end{aligned} \tag{8}$$

and

$$\begin{aligned} E(\underline{B}_t) \equiv \underline{B} &\equiv N \times (p(NotB_t)) \\ &\equiv N \times (1 - p(B_t)) \\ &\equiv N \times (p(\underline{B}_t)) \\ &\equiv N \times (p(b_t) + p(d_t)) \\ &\equiv b + d \end{aligned} \tag{9}$$

where $p(a_t)$ denotes the joint probability of A_t and B_t. In general, it is

$$\begin{aligned} N &\equiv N \times 1 \\ &\equiv N \times (p(a_t) + p(b_t) + p(c_t) + p(d_t)) \\ &\equiv a + b + c + d \end{aligned} \tag{10}$$

Independence —

Definition 2.3 (Independence)**.**

The independence(Barukčić 2021a) [3] of two events A_t and B_t regarded from the standpoint of a certain observer was defined by de Moivre on page 7 as "... therefore, those two Events being independent, the Probability of their both happening will be 1/13 * 1/13 = 1/169 "[4] and Kolmogoroff [5] and other, as

$$p(a_t) = p(A_t \wedge B_t) = p(A_t) \times p(B_t) \tag{11}$$

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.

[3] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html

[4] https://doi.org/10.3931/e-rara-10420

[5] https://doi.org/10.1007/978-3-642-49888-6

Index of relationship (IOR) —

Definition 2.4 (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 (IOR) is defined as

$$\begin{aligned} IOR(A_t, B_t) &\equiv \left(\frac{p(A_t \wedge B_t)}{p(B_t) \times p(A_t)} \right) - 1 \\ &\equiv \left(\frac{p(a_t)}{p(B_t) \times p(A_t)} \right) - 1 \\ &\equiv \left(\left(\frac{N \times N \times p(a_t)}{N \times p(B_t) \times N \times p(A_t)} \right) - 1 \right) \\ &\equiv \left(\left(\frac{N \times a}{A \times B} \right) - 1 \right) \end{aligned} \tag{12}$$

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.

Dependence —

Definition 2.5 (Dependence)**.**

The Dependence(Barukčić 2021a) [6] of two events A_t and B_t regarded from the standpoint of a certain observer is defined as

$$p(a_t) = (p(B_t) \times p(A_t))^{1/2} \tag{13}$$

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 while the dependence of n events[7] follows as

$$p(a_{1,t}, a_{2,t}, ..., a_{n,t}) = (p(A_{1,t}) \times p(A_{2,t}) \times ... \times p(A_{n,t}))^{1/n} \tag{14}$$

Experimental event rate (EER) —

Definition 2.6 (Experimental[8] event rate (EER))**.**

$$EER\,(A_t, B_t) \equiv \frac{p(a_t)}{p(A_t)} = \frac{a_t}{a_t + b_t} \tag{15}$$

Definition 2.7 (Control[9] event rate (CER))**.**

$$CER\,(A_t, B_t) \equiv \frac{p(c_t)}{p(\underline{A}_t)} = \frac{c_t}{c_t + d_t} \tag{16}$$

Absolute risk reducation (ARR) —

Definition 2.8 (Absolute[10] risk reducation (ARR))**.**

$$\begin{aligned} ARR\,(A_t, B_t) &\equiv \frac{p(c_t)}{p(\underline{A}_t)} - \frac{p(a_t)}{p(A_t)} \\ &= \frac{c_t}{c_t + d_t} - \frac{a_t}{a_t + b_t} \\ &= CER\,(A_t, B_t) - EER\,(A_t, B_t) \end{aligned} \tag{17}$$

[6] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html

[7] Ilija Barukčić, Die Kausalität, Hamburg: Wissenschaftsverlag, 1989, pp. 57-59.

[8] https://grunigen.lib.uci.edu/sites/all/docs/gml/RRR_ARR_NNT.pdf

[9] https://grunigen.lib.uci.edu/sites/all/docs/gml/RRR_ARR_NNT.pdf

[10] https://grunigen.lib.uci.edu/sites/all/docs/gml/RRR_ARR_NNT.pdf

Number needed to treat (NNT) —

Definition 2.9 (Number needed to treat[11] (NNT))**.**

$$NNT\,(A_t, B_t) \equiv \frac{1}{CER\,(A_t, B_t) - EER\,(A_t, B_t)} \tag{18}$$

Number needed to harm (NNH) —

Definition 2.10 (Number needed to harm[12] (NNH))**.**

$$NNH\,(A_t, B_t) \equiv \frac{1}{EER\,(A_t, B_t) - CER\,(A_t, B_t)} \tag{19}$$

Relative risk (RR) —

Definition 2.11 (Relative [13] risk (RR))**.**

The degree of association between the two binomial variables can be assessed by a number of very different coefficients, the relative risk [14] is one of them. In this context, see also Sir Ronald Aylmer Fisher's (1890 - 1962) contribution in his publication "The Logic of Inductive Inference"[15] (see also Fisher 1935, p. 50). In general, relative risk is defined as

$$RR(A_t, B_t) = \frac{\frac{p(a_t)}{p(A_t)}}{\frac{p(c_t)}{p(NotA_t)}} = \frac{p(a_t) \times p(NotA_t)}{p(c_t) \times p(A_t)} = \frac{N \times p(a_t) \times N \times p(NotA_t)}{N \times p(c_t) \times N \times p(A_t)} = \frac{a \times (NotA)}{c \times A} = \frac{EER\,(A_t, B_t)}{CER\,(A_t, B_t)} \tag{20}$$

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 [16] [17] and relative risk [18] are already documented [19] [20] in literature.

Example.

According to the Centers for Disease Control and Prevention (CDC)[21], an outbreak of varicella (chickenpox) in Oregon (USA) in 2002 was diagnosed in 18 of 152 (12%) vaccinated students[22] compared with 3 of 7 (43%) unvaccinated students.

Table 1. Outbreak of varicella (chickenpox) in Oregon in 2002

		Varicella B_t		
		TRUE	FALSE	
Vaccinated	TRUE	$a_t = 18$	$b_t = 134$	$A_t = 152$
A_t	FALSE	$c_t = 3$	$d_t = 4$	$\underline{A}_t = 7$
		$B_t = 21$	$\underline{B}_t = 138$	$N_t = 159$

[11] https://pubmed.ncbi.nlm.nih.gov/7873954/
[12] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3083982/
[13] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2545775/
[14] https://www.ncbi.nlm.nih.gov/books/NBK430824/
[15] https://www.jstor.org/stable/pdf/2342435.pdf?seq=1
[16] https://www.ncbi.nlm.nih.gov/pubmed/9832001
[17] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6178613/
[18] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC522855/
[19] https://www.crcpress.com/Principles-of-Biostatistics-Second-Edition/Pagano-Gauvreau/p/book/9781138593145
[20] https://www.biometricsociety.org/wp-content/uploads/2018/07/IBS-IBC2012-Final-Programme.compressed.pdf
[21] https://www.cdc.gov/csels/dsepd/ss1978/lesson3/section5.html
[22] https://pubmed.ncbi.nlm.nih.gov/14993534/

The risk ratio RR is calculated as follows.

$$\begin{aligned} RR\,(A_t, B_t) &\equiv \frac{\frac{p(a_t)}{p(A_t)}}{\frac{p(c_t)}{p(NotA_t)}} = \frac{p(a_t) \times p(NotA_t)}{p(A_t) \times p(c_t)} \\ &\equiv \left(\frac{a_t \times \underline{A}_t}{c_t \times A_t}\right) \\ &\equiv \left(\frac{18 \times 7}{3 \times 152}\right) \\ &\equiv \frac{0.118}{0.42} \\ &\equiv 0.28 \end{aligned} \tag{21}$$

The risk ratio is RR = 0.28 and less than 1.0 which indicates a decreased risk or protective effect for the children which where vaccinated (exposed to vaccine). However, the risk ratio of 0.28 is completely misleading in this context as can be seen by table 2 .

Table 2. Vaccinated and Varicella.

		Varicella		
		YES	NO	
Vaccinated	YES	18	134	152
	NO	3	4	7
		21	138	159

Causal relationship k =	-0,1879
p Value left tailed (HGD) =	0,0493
p (SINE) =	0,9811
$\tilde{\chi}^2$ (SINE — B_t) =	0,4286
$\tilde{\chi}^2$ (SINE — $\underline{A}_t$) =	1,2857
p Value (SINE) =	0,0187
p(IOI)=	0,8239
p(IOU)=	0,0881

Taking the data as published by CDC for granted, we need to conclude the following: **without** vaccination **no** outbreak of varicella (chickenpox) in Oregon (USA) in 2002 (p(SINE) = 0,9811; p Value (SINE) = 0,0187; p(IOU)= 0,0881).

However, such a conclusion(Barukčić 2021a) [23] is neither completely justified nor free or errors. A negative causal relationship k (k = -0,1879), even if p(IOU) = 0,0881 is very impressive, does not support the hypothesis of necessary condition. In other words, the data as presented by CDC are self-contradictory[24] and cannot be used for such a conclusion. In contrast to this, the close connection between vaccination and outbreak of varicella (chickenpox) in Oregon in 2002 is rather masked than discovered by the risk ratio. Even though it is considered highly desirable, the conclusion that the vaccination protected against an outbreak of varicella (chickenpox) in Oregon (USA) in 2002 is not justified for sure due to the data published[25] by CDC. Reason: the study design with p(IOI)=0,8239 has been extremely biased.

Relative risk reduction (RRR) —

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

[23] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html

[24] https://pubmed.ncbi.nlm.nih.gov/14993534/

[25] https://pubmed.ncbi.nlm.nih.gov/14993534/

$$RRR(A_t, B_t) \equiv \frac{CER(A_t, B_t) - EER(A_t, B_t)}{CER(A_t, B_t)} = 1 - RR(A_t, B_t) \tag{22}$$

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.

$$VE(A_t, B_t) \equiv 100 \times (1 - RR(A_t, B_t)) \equiv 100 \times \left(\frac{CER(A_t, B_t) - EER(A_t, B_t)}{CER(A_t, B_t)}\right) \tag{23}$$

Historically, vaccine efficacy has been designed to evaluate the efficacy a certain vaccine by Greenwood and Yule[26] 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 to much on the study design, can lead to erroneous conclusions and is only of very limited value.

Odds ratio (OR) —

Definition 2.14 (Odds ratio[27,28] (OR))**.**

Odds(see also Yule and Pearson 1900, p. 273) ratio (OR) is a measure of association[29] which quantifies the relationship[30] between two binomial distributed random variables (exposure vs. outcome) and is related to Yule's (see also Yule and Pearson 1900, p. 272) Q(see also Yule 1912, p. 585/586). Hereafter, consider the table 3.

Table 3. The two by two table of Bernoulli 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

where
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).
The odds ratio (OR) is defined as

$$OR(A_t, B_t,) \equiv \left(\frac{a_t}{b_t}\right) \times \left(\frac{c_t}{d_t}\right) \equiv \left(\frac{a_t \times d_t}{b_t \times c_t}\right) \tag{24}$$

[26] https://journals.sagepub.com/doi/10.1177/003591571500801433
[27] https://bestpractice.bmj.com/info/toolkit/learn-ebm/how-to-calculate-risk/
[28] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2938757/
[29] https://www.cdc.gov/csels/dsepd/ss1978/lesson3/section5.html
[30] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2545775/

Remark. 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. 24. 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 recognise elementary relationships of objective reality. In fact, it would be a failure not to recognise how dangerous and less valuable odds ratio is.

Remark. Under conditions where (c = 0) odds ratio collapses too, because we need again to divide by zero as can be seen at eq. 24. 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 recognise 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.

Exclusion relationship —

Definition 2.15 (Exclusion relationship)**.**

The exclusion(Barukčić 2021a) [31] relationship (A_t excludes B_t and vice versa) is defined as

$$p(A_t \mid B_t) = p(b_t) + p(c_t) + p(d_t) = +1 \tag{25}$$

Necessary condition (conditio sine qua non) —

Definition 2.16 (Conditio sine[32] qua non relationship)**.**

The conditio sine qua non relationship (A_t is a necessary condtion of B_t) is defined as

$$p(A_t \longleftarrow B_t) = p(a_t) + p(b_t) + p(d_t) = +1 \tag{26}$$

Remark. Since thousands of years, human mankind is familiar with the concept of necessary conditions. For example, we all know that air or gaseous oxygen as such is a necessary condition for (human) life. In other words, without gaseous[33] oxygen, there is no (human) life. However, the first documented mathematiziation of the concept of a necessary condition (**conditio sine qua non**) has been published by Barukčić 1989 [34]. Conditions may be necessary without being sufficient and vice versa. Sufficient conditions need not to be necessary. However, there may exist conditions which are both, necessary and sufficient. Nonetheless, any form of a mechanical understanding of a necessary condition may not stand the test of reality forever.

Human experience knows about the relationship between water and human life. It is part of the established knowledge of all of us that **without** water **no** human life. In other words, water itself is a necessary condition of human life. However, there may be some circumstances under which something can turn into its own other and vice versa. In other words, a person who drinks over 1000 litres of water at once (i. e. sea water) will die. Under these certain circumstances, water which is a necessary condition of human life in general turns into the other of itself, into a sufficient condition of human death. It is of great importance to be exact and precise in describing the circumstances, the minima and the maxima, the terms, the definitions, the inclusion and exclusion criteria et cetera of an investigation. In this sense, it is more than appropriate to pay the necessary tribute to Giordano Bruno (1548-1600) who wrote: : "So ist denn von zwei Entgegengesetzten das eine zugleich das Prinzip des anderen $\cdots$ Wer also die tiefsten Geheimnisse der Natur ergründen will, beobachte und betrachte die Minima und die Maxima des Entgegengesetzten und Widerstreitenden."(see also Bruno 1583, p. 148/149). Translated into English: 'So it is from two opposites at the same time the one the principle of the other $\cdots$ So **if you want to discover the deepest secrets of nature, observe and consider the minima and maxima of the opposite and conflicting**.'

Sufficient condition (conditio per quam) —

Definition 2.17 (Conditio per[35] quam relationship)**.**

[31] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html
[32] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html
[33] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html
[34] Ilija Barukčić, Die Kausalität, Hamburg: Wissenschaftsverlag, 1989
[35] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html

The conditio per quam(Barukčić 2021a) [36] relationship (if A_t then B_t relationship) is defined [37] [38] [39] [40] [41] [42] [43] [44] as

$$p(A_t \longrightarrow B_t) = p(a_t) + p(c_t) + p(d_t) = +1 \tag{27}$$

Conditio per quam		**Street is wet**		
		YES	NO	
It is raining	YES	+1	+0	A_t
	NO	+1	+1	$\underline{A}_t$
		B_t	$\underline{B}_t$	

[36] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html
[37] https://aip.scitation.org/doi/abs/10.1063/1.3567453
[38] https://aip.scitation.org/doi/abs/10.1063/1.4773147
[39] https://www.scirp.org/journal/paperinformation.aspx?paperid=69478
[40] https://www.scirp.org/journal/paperinformation.aspx?paperid=67272
[41] http://www.ijapm.org/show-64-515-1.html
[42] https://www.sciencedirect.com/science/article/pii/S1875389211006626
[43] https://view.publitas.com/amph/rjr_2018_4_art-02/page/1
[44] http://jddtonline.info/index.php/jddt/article/view/3385

Remark. Chile's **Atacama desert** is a desert plateau covering about 1,000-km (600-mi) strip of land on the Pacific coast. In contrast to the equator where it rains very often, the Atacama desert is widely considered as world's driest nonpolar desert with an average rainfall of as little as 0.04 inches per year. However, a **conditio per quam** relationship between raining and a street which is wet can be investigated even under these circumstances.

Under conditions of the Atacama desert a thought experiment is performed and the following data were achieved. It rained seldom at this occasion thus that the experimenter put 999 times some water on the street by himself where he performed measurements in order to study what happens if it is not raining.

Conditio per quam (Atacama desert)		The street is wet		
		YES	NO	
It is raining	YES	1000	0	1000
	NO	999	1	1000
		1999	1	2000

Figure 1. Counterexample. Risk ratio.

The relative risk follows as

$$RR(A_t, B_t) = \frac{p(a_t) \times p(NotA_t)}{p(A_t) \times p(c_t)} = \frac{1000 \times 1000}{999 \times 1000} = 1.0010 \tag{28}$$

The relative risk has been calculated as RR = 1.0010 while the 95% CI is 0.9990 to 1.0030 and the P value is P = 0.3173. In other words, according to the relative risk, raining is not a risk factor of a wet street or raining and a wet street are independent of each other. For the better understanding, let us repeat this fact again. According to the risk ratio (RR), raining at a certain (period of) time t and a street which is wet at the same (period of) time t are independent of each other. Such a risk ratio based erroneous conclusion is far away from any possible reality and everyday human experience. Therefore, what is becoming more and more visible is how risk ratio is forcing us in an intolerable manner to see reality through foggy statistical glasses. The counterexample (see fig. 1) has provided evidence of the logical inconsistency of the risk ratio. The risk ratio collapsed by the counterexample (see fig. 1) at last like a rotten piece of wood. Formally, even if relative risk is able to recognise a **conditio per quam relationship** in reality the same does not. Above any suspicion, depending upon **study design and other factors**, the relative risk present us a false and completely misleading picture of objective reality. Without any doubt, it is really no longer necessary to hold onto relative risk at all.

Causal relationship k —

Definition 2.18 (Causal relationship k[45])**.**

Nonetheless, mathematically, the causal relationship Barukčić (1989, 1997, 2005, 2016, 2017) 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 Bernoulli trial t* in terms of statistics and probability theory as

$$\begin{aligned} k(U_t, W_t) &\equiv \frac{\sigma(U_t, W_t)}{\sigma(U_t) \times \sigma(W_t)} \\ &\equiv \frac{p(U_t \wedge W_t) - p(U_t) \times p(W_t)}{\sqrt[2]{(p(U_t) \times (1 - p(U_t))) \times (p(W_t) \times (1 - p(W_t)))}} \end{aligned} \tag{29}$$

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 4 illustrates the theoretically possible relationships between a cause and an effect.

Table 4. 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

Index Of Unfairness (IOU) —

Definition 2.19 (**Index Of Unfairness**)**.**

The quality of big data collected may depend upon several factors. Therefore, it is appropriate to quantify possible collection or extraction bias due to the method used. The index of unfairness (IOU) is defined (see Barukčić 2019a) as

$$\begin{aligned} p(IOU) &\equiv Absolute\left(\left(\frac{A+B}{N}\right) - 1\right) \\ &\equiv Absolute\left(\left(\frac{\underline{A}+\underline{B}}{N}\right) - 1\right) \end{aligned} \tag{30}$$

Index Of Independence (IOI) —

Definition 2.20 (**Index Of Independence**)**.**

Big data bias due to collection methods and other factors cannot be excluded completely. The index of independence (IOI) is of use in this context and defined (see Barukčić 2019b) as

$$\begin{aligned} p(IOI) &\equiv Absolute\left(\left(\frac{A+\underline{B}}{N}\right) - 1\right) \\ &\equiv Absolute\left(\left(\frac{\underline{A}+B}{N}\right) - 1\right) \end{aligned} \tag{31}$$

[45] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html

2.1.2. *Studies*

Married/sexual activity and prostate cancer I—Sex has received to little attention in the history of philosophy, science and of human mankind as such and the attention sex did receive was not always been completely unproblematic. Authors like Aristotle himself barely mentioned sex while Christian philosophers were dogmatically active to condemned sex. In turn, authors like Donatien Alphonse François Marquis de Sade (1740 - 1814) advocated to celebrate all(see also Marquis de Sade 1795) types of sexual acts in the extreme. Human sexual desire including the sexual act itself et cetera is at least very complex and directed under usual circumstances at the opposite human gender. However and above all, rooted in natural laws, sex is equally an everlasting ingenious invention of nature of giving and taking of (human) life. The influence of marital status or of regularly, mutual sex of both spouses on prostate cancer has been investigated many times. In general, married should suffer from prostate cancer more frequent than non-married. However, marital status does not exclude sex with other before a marriage and during a marriage. Therefore, marriage as such can be only a weak indicator of sexual intercourse as a path to prostate cancer. In order to detect a kind of a relationship, it is justified to accept a higher p Value. Loeb at al. (see also Loeb et al. 2017) relied on the National Prostate Cancer Register of Sweden and conducted a a nested case-control study which includes all 38,570 prostate cancer cases diagnosed from 2009 to 2012 and equally 192,838 age-matched men free of prostate cancer. Loeb at al. provided additionally data on the relationship between marriage and prostate cancer (see table 5) too.

Table 5. Married and prostate cancer I.

		Prostate		
		YES	NO	
Married	YES	25684	121279	146963
	NO	12886	71559	84445
		38570	192838	231408

p(IOI)=	0,468406451
p(IOU)=	0,19824293
Causal relationship k =	0,0286358656
p Value right tailed (HGD) =	0,0000000000
p (SINE) =	0,9443148033
p Value (SINE) =	0,0541631584

Married/sexual activity and prostate cancer II—The study of Loeb et al. (see also Loeb et al. 2017) is marked with several limitations. One serious limitation of this study includes a lack of data on sexual activity. In addition, it is assumed that married had sex during the marriage. However, it is not completely absurd to assume that more than 1.000 of 231.408 investigated subjects (= 0,432137178 %) which developed prostate cancer had already sex without being married. Under these assumptions the data of table 5 changes to the data as presented by table 6.

Table 6. Married/sexual activity and prostate cancer II.

		Prostate cancer		
		YES	NO	
Married/Sex	YES	26784	121279	148063
	NO	11786	71559	83345
		38570	192838	231408

p(IOI)=	0,47315996
p(IOU)=	0,193489421
Causal relationship k =	0,0508581440
p Value right tailed (HGD) =	0,0000000000
p (SINE) =	0,9490683122
p Value (SINE) =	0,0496564116

However and as already published(see also Barukčić 2019c) somewhere else, it is necessary to accept the null-hypothesis

without sexual activity **no** prostate cancer (see table 6).

Sexual activity and prostate cancer III—But it is more than probable that all 12886 of 231.408 investigated subjects (= 5,568519671 %) which developed prostate cancer had already sex without being married. Furthermore, it can reasonable be assumed that the men investigated were all after puberty and that all of them presumably had been sexually active. Under these assumptions, the data of Loeb et al. (see table 5) changes to the data as presented by table 7.

Table 7. Sexual activity and prostate cancer.

		Prostate cancer		
		YES	NO	
Sexual activity	YES	38570	121279	159849
	NO	0	71559	71559
		38570	192838	231408

p(IOI)=	0,524091648
p(IOU)=	0,142557734
Causal relationship k =	0,2992303770
p Value right tailed (HGD) =	0,0000000000
p (SINE) =	1,0000000000
p Value (SINE) =	0,0000000000

The data of Loeb at al. provide striking evidence for the null-hypothesis

without sexual activity **no** prostate cancer (see table 7).

Prostate cancer is caused by an infectious agent. In the same breath as sex has the potential to create human life, the same sex has the potential to destroy human life too. Sex is a creator and destroyer of human life and may be of life as such.

Testosterone and prostate cancer I—Testosterone is involved many step of the male sexual function even if the occurrence of sexual disorders is not automatically related to a decline in testosterone levels. In fact, testosterone may have an impact even on prostate cancer too. Loeb et al. (see also Loeb et al. 2017) provided data on the relationship between any prior prescription of testosterone and prostate cancer (see table 8).

Table 8. Testosterone and prostate cancer I.

		Prostate cancer		
		YES	NO	
Any prior prescription of testosterone	YES	284	1378	1662
	NO	38286	191460	229746
		38570	192838	231408

p(IOI)=	0,15949319
p(IOU)=	0,826142571
Causal relationship k =	+ 0,0009592321
p Value right tailed (HGD) =	0,3320389397
p (EXCL) =	0,9987727304
$\tilde{\chi}^2$ (EXCL— B_t) =	2,0912
p Value (EXCL) =	0,0012265168
IOR =	+0,02522

The right tailed p Value based on the hyper-geometric distribution is calculated as follows:

$$\begin{aligned} pValue_{\text{rt}}\left(X \geq 284\right) &\equiv 1 - \sum_{t=0}^{284-1} \left(\frac{\binom{1662}{t} \times \binom{231408-1662}{38570-t}}{\binom{231408}{38570}} \right) \\ &\equiv 0,3320389397 \end{aligned} \tag{32}$$

The data of Loeb et al. (see also Loeb et al. 2017) are self-contradictory. **Firstly.** The index of independence (p(IOI)=0,15949319) indicates that the data are suitable for the analysis of an exclusion relationship. The exclusion relationship is highly significant (p Value (EXCL) = 0,0012265168). In other words, testosterone excludes prostate cancer. **Secondly.** However, the relationship between testosterone and prostate cancer based on the data of Loeb et al. (see also Loeb et al. 2017) is not quite as simple as it is suggested. The causal relationship k is positive, even if not significant. The index of relationship (IOR) is positive too. However, this is a contradiction. If testosterone excludes prostate cancer then causal relationship k need to be negative and the index of relationship (IOR) need to be negative too. The data support both each other excluding situations. This is a contradiction. The question does arise, may we ignore the causal relationship k and the index of relationship (IOR) under conditions where p(IOI) ¡ 0.2. Nonetheless, the data of Loeb et al. (see also Loeb et al. 2017) are preliminary of limited value on the question testosterone and prostate cancer. Any prior description of testosterone does not necessarily mean an intake of testosterone too. A more differentiated assessment of the issue in question is necessary and warrants further investigation. Especially, it is necessary to consider an intake of testosterone at the time of the diagnosis of prostate cancer.

Testosterone and PSA > 100—In toto, 2548 prostate cancer patient with a prostate-specific antigen (PSA) of about PSA > 100 were not exposed to testosterone. In the same respect, 3 of the prostate cancer patient with a PSA > 100 were exposed to testosterone. These data are viewed by table 9.

Table 9. Testosterone and PSA > 100.

		PSA > 100		
		YES	NO	
Testosterone	YES	3	275	278
	NO	2548	36750	39298
		2551	37025	39576

p(IOI)=	0,057433798
p(IOU)=	0,928517283
Causal relationship k =	-0,0183811527
p Value $_{\text{left tailed}}$ (HGD) =	0,0000117
p (EXCL) =	0,9999241965
$\tilde{\chi}^2$ (EXCL— A_t) =	0,0324
$\tilde{\chi}^2$ (EXCL— B_t) =	0,0035
p Value (EXCL) =	0,0000758006
IOR =	-0,8326

The left tailed p Value based on the hyper-geometric distribution is calculated as follows:

$$\begin{aligned} pValue_{\text{left tailed}}\,(X \leq 3) &\equiv \sum_{t=0}^{3} \left(\frac{\binom{278}{t} \times \binom{39576-278}{2551-t}}{\binom{39576}{2551}} \right) \\ &\equiv 0,0000117244 \end{aligned} \tag{33}$$

In other words, a prostate-specific antigen or PSA > +100 excluded the prescription of testosterone for prostate cancer patients by physicians or vice versa. However, it is possible to take a very different view, considering this issue. The prescription of testosterone for prostate cancer patients excluded a prostate-specific antigen or PSA > +100. It remains to be found, what was first, the chicken or the egg.

2.1.3. *Axioms*

Axiom 1. Lex identitatis [46] [47] [48].

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

Axiom 2. Lex contradictionis[49] [50] [51].

$$+0 = +1 \tag{35}$$

2.2. *Methods*

2.2.1. *Proof methods*

Proof methods like a direct proof [52], proof by contradiction[53], modus ponens[54], modus inversus[55] [56] and other methods are of use to detect inconsistencies and inadequacies in scientific theories.

[46] https://www.scirp.org/journal/paperinformation.aspx?paperid=69478
[47] https://www.ncbi.nlm.nih.gov/nlmcatalog/101656626
[48] https://doi.org/10.22270/jddt.v9i2.2389
[49] https://www.ncbi.nlm.nih.gov/nlmcatalog/101656626
[50] https://doi.org/10.22270/jddt.v9i2.2389
[51] https://doi.org/10.22270/jddt.v10i1-s.3856
[52] http://www.ijmttjournal.org/Volume-65/Issue-7/IJMTT-V65I7P524.pdf
[53] https://aip.scitation.org/doi/abs/10.1063/1.3567453
[54] http://www.ijmttjournal.org/Volume-65/Issue-7/IJMTT-V65I7P524.pdf
[55] http://www.ijmttjournal.org/Volume-65/Issue-7/IJMTT-V65I7P524.pdf
[56] https://vixra.org/pdf/1911.0410v1.pdf

3. RESULTS

3.1. *The identity of distinguished I*

Theorem 1. *In general, the relationship*

$$p(A_t \wedge B_t) - p(A_t) \times p(B_t) = (p(a_t) \times p(d_t)) - (p(b_t) \times p(c_t)) \tag{36}$$

is valid.

Proof by direct proof. Theorem 1 or

$$+1 = +1 \tag{37}$$

is true. Multiplying by $p(A_t \wedge B_t)$, we obtain

$$p(A_t \wedge B_t) = p(A_t \wedge B_t) \tag{38}$$

Subtracting $p(A_t) \times p(B_t)$, it is

$$p(A_t \wedge B_t) - p(A_t) \times p(B_t) = p(A_t \wedge B_t) - p(A_t) \times p(B_t) \tag{39}$$

We recall the mathematical identity $p(a_t) = p(A_t \wedge B_t)$. Eq. 39 changes to

$$\begin{aligned} p(A_t \wedge B_t) - p(A_t) \times p(B_t) = & \; p(a_t) \\ & - p(A_t) \times p(B_t) \end{aligned} \tag{40}$$

or to

$$\begin{aligned} p(A_t \wedge B_t) - p(A_t) \times p(B_t) = & \; p(a_t) \times +1 \\ & - p(A_t) \times p(B_t) \end{aligned} \tag{41}$$

According to eq. 5 it is $p(a_t) + p(b_t) + p(c_t) + p(d_t) = +1$. Eq. 41 changes to

$$\begin{aligned} p(A_t \wedge B_t) - p(A_t) \times p(B_t) = & \; (p(a_t) \times (p(a_t) + p(b_t) + p(c_t) + p(d_t))) \\ & - p(A_t) \times p(B_t) \end{aligned} \tag{42}$$

According to eq. 1 it is $p(A_t) = (p(a_t) + p(b_t))$. Eq. 42 changes to

$$\begin{aligned} p(A_t \wedge B_t) - p(A_t) \times p(B_t) = & \; (p(a_t) \times (p(a_t) + p(b_t) + p(c_t) + p(d_t))) \\ & - (p(a_t) + p(b_t)) \times p(B_t) \end{aligned} \tag{43}$$

According to eq. 3 it is $p(B_t) = p(a_t) + p(c_t)$. Eq. 43 changes to

$$\begin{aligned} p(A_t \wedge B_t) - p(A_t) \times p(B_t) = & \; (p(a_t) \times (p(a_t) + p(b_t) + p(c_t) + p(d_t))) \\ & - ((p(a_t) + p(b_t)) \times (p(a_t) + p(c_t))) \end{aligned} \tag{44}$$

Rearranging eq. 44 we obtain

$$\begin{aligned} p(A_t \wedge B_t) - p(A_t) \times p(B_t) = & \; ((p(a_t) \times p(a_t)) + (p(a_t) \times p(b_t)) + (p(a_t) \times p(c_t)) + (p(a_t) \times p(d_t))) \\ & - ((p(a_t) \times p(a_t)) + (p(a_t) \times p(b_t)) + (p(a_t) \times p(c_t)) + (p(b_t) \times p(c_t))) \end{aligned} \tag{45}$$

In other words, it is

$$\begin{aligned} p(A_t \wedge B_t) - p(A_t) \times p(B_t) = & \; (p(a_t) \times p(a_t)) + (p(a_t) \times p(b_t)) + (p(a_t) \times p(c_t)) + (p(a_t) \times p(d_t)) \\ & - (p(a_t) \times p(a_t)) - (p(a_t) \times p(b_t)) - (p(a_t) \times p(c_t)) - (p(b_t) \times p(c_t)) \end{aligned} \tag{46}$$

or in general

$$p(A_t \wedge B_t) - p(A_t) \times p(B_t) = (p(a_t) \times p(d_t)) - (p(b_t) \times p(c_t)) \tag{47}$$

□

3.2. *The identity of distinguished II*

Theorem 2. *In general it is*

$$(N \times a) - (A \times B) \equiv (a \times d) - (b \times c) \tag{48}$$

Proof by direct proof. According to eq. 47, it is

$$p(A_t \wedge B_t) - p(A_t) \times p(B_t) = (p(a_t) \times p(d_t)) - (p(b_t) \times p(c_t)) \tag{49}$$

Multiplying eq. 49 by N, we obtain

$$N \times N \times p(A_t \wedge B_t) - N \times N \times p(A_t) \times p(B_t) = N \times N \times (p(a_t) \times p(d_t)) - N \times N \times (p(b_t) \times p(c_t)) \tag{50}$$

At the end, according to 1 until eq. 10, it is

$$(N \times a) - (A \times B) \equiv (a \times d) - (b \times c) \tag{51}$$

□

3.3. *Einstein's field equations and the beginning of our world*

As is known, Albert Einstein's (1879–1955) own neo-Kantianism philosophy of science culminated in the Einstein field equations(see also Einstein 1916, 1917) derived as $G_{\mu\nu}+(\Lambda\times g_{\mu\nu})=\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}$ where $G_{\mu\nu}$ is a rank-2 co-variant tensor describing the space-time curvature (the Einstein tensor) and $\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}$ is the stress–energy tensor or the stress–energy–momentum tensor or the energy–momentum tensor of matter while Λ is the cosmological constant(see also Einstein 1917; Einstein and Sitter 1932).

Theorem 3. *Thus far, let $R_{\mu\nu}=a_{\mu\nu}+b_{\mu\nu}+c_{\mu\nu}+d_{\mu\nu}$ denote the second-rank co-variant Ricci curvature tensor, the trace of the Riemann curvature tensor. Let $a_{\mu\nu}$, $b_{\mu\nu}$, $c_{\mu\nu}$ and $d_{\mu\nu}$ denote the four basic fields of nature, while $a_{\mu\nu}$ denote the stress-energy tensor of ordinary matter, $b_{\mu\nu}$ denote the stress-energy tensor of the electromagnetic field. In general, it is $\frac{8\times\pi\times\gamma}{c^4}\times T_{\mu\nu}=a_{\mu\nu}+b_{\mu\nu}$ and $G_{\mu\nu}=a_{\mu\nu}+c_{\mu\nu}$. In general, it is*

$$(R_{\mu\nu}\times a_{\mu\nu})-\left(\left(\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}\right)\times G_{\mu\nu}\right)=(a_{\mu\nu}\times d_{\mu\nu})-(b_{\mu\nu}\times c_{\mu\nu}) \tag{52}$$

Proof by direct proof. Theorem 1 or

$$+1=+1 \tag{53}$$

is true. Multiplying eq. 53 by the Ricci tensor $R_{\mu\nu}=a_{\mu\nu}+b_{\mu\nu}+c_{\mu\nu}+d_{\mu\nu}$, it is

$$(R_{\mu\nu})=(R_{\mu\nu}) \tag{54}$$

Multiplying eq. 54 by the stress-energy tensor of ordinary matter, $a_{\mu\nu}$, it is

$$(R_{\mu\nu}\times a_{\mu\nu})=(R_{\mu\nu}\times a_{\mu\nu}) \tag{55}$$

Subtracting the term $\left(\left(\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}\right)\times G_{\mu\nu}\right)$, we obtain

$$(R_{\mu\nu}\times a_{\mu\nu})-\left(\left(\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}\right)\times G_{\mu\nu}\right)=(R_{\mu\nu}\times a_{\mu\nu})-\left(\left(\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}\right)\times G_{\mu\nu}\right) \tag{56}$$

It is $R_{\mu\nu}=a_{\mu\nu}+b_{\mu\nu}+c_{\mu\nu}+d_{\mu\nu}$ Eq. 56 changes to

$$\begin{aligned}(R_{\mu\nu}\times a_{\mu\nu})-\left(\left(\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}\right)\times G_{\mu\nu}\right)&=(a_{\mu\nu}+b_{\mu\nu}+c_{\mu\nu}+d_{\mu\nu})\times a_{\mu\nu}\\&-\left(\left(\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}\right)\times G_{\mu\nu}\right)\end{aligned} \tag{57}$$

Additionally, it is $\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}=a_{\mu\nu}+b_{\mu\nu}$ and $G_{\mu\nu}=a_{\mu\nu}+c_{\mu\nu}$ Eq. 57 changes to

$$\begin{aligned}(R_{\mu\nu}\times a_{\mu\nu})-\left(\left(\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}\right)\times G_{\mu\nu}\right)&=(a_{\mu\nu}+b_{\mu\nu}+c_{\mu\nu}+d_{\mu\nu})\times a_{\mu\nu}\\&-((a_{\mu\nu}+b_{\mu\nu})\times(a_{\mu\nu}+c_{\mu\nu}))\end{aligned} \tag{58}$$

Simplifying eq. 58, it is

$$\begin{aligned}(R_{\mu\nu}\times a_{\mu\nu})-\left(\left(\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}\right)\times G_{\mu\nu}\right)&=((a_{\mu\nu}\times a_{\mu\nu})+(a_{\mu\nu}\times b_{\mu\nu})+(a_{\mu\nu}\times c_{\mu\nu})+(a_{\mu\nu}\times d_{\mu\nu}))\\&-((a_{\mu\nu}\times a_{\mu\nu})+(a_{\mu\nu}\times b_{\mu\nu})+(a_{\mu\nu}\times c_{\mu\nu})+(b_{\mu\nu}\times c_{\mu\nu}))\end{aligned} \tag{59}$$

and equally according to our today mathematical rules

$$\begin{aligned}(R_{\mu\nu}\times a_{\mu\nu})-\left(\left(\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}\right)\times G_{\mu\nu}\right)&=(a_{\mu\nu}\times a_{\mu\nu})+(a_{\mu\nu}\times b_{\mu\nu})+(a_{\mu\nu}\times c_{\mu\nu})+(a_{\mu\nu}\times d_{\mu\nu})\\&-(a_{\mu\nu}\times a_{\mu\nu})-(a_{\mu\nu}\times b_{\mu\nu})-(a_{\mu\nu}\times c_{\mu\nu})-(b_{\mu\nu}\times c_{\mu\nu})\end{aligned} \tag{60}$$

Equation 60 simplifies as

$$(R_{\mu\nu}\times a_{\mu\nu})-\left(\left(\left(\frac{8\times\pi\times\gamma}{c^4}\right)\times T_{\mu\nu}\right)\times G_{\mu\nu}\right)=(a_{\mu\nu}\times d_{\mu\nu})-(b_{\mu\nu}\times c_{\mu\nu}) \tag{61}$$

□

Remark. Depending upon several factors, eq. 61 dervied as

$$\left(R_{\mu\nu} \times a_{\mu\nu}\right) - \left(\left(\left(\frac{8 \times \pi \times \gamma}{c^4}\right) \times T_{\mu\nu}\right) \times G_{\mu\nu}\right) = \left(a_{\mu\nu} \times d_{\mu\nu}\right) - \left(b_{\mu\nu} \times c_{\mu\nu}\right) \tag{62}$$

can be equal to zero, less than zero or greater than zero. Does equation 62 allow something like a **creatio ex nihilio**, a creation or a beginning of our world our of nothing, however we may define nothing? Are question like these beyond any human experience?

Physics and the Einstein field equations many times study only something what exists. Consequently, one might expect physics including the Einstein field equations to have little to say about the special case in which nothing, an absence of something, exists. It is necessary to point out, **nothing exists**, it is a nothing, but it is given too. As far as simplicity is concerned, there is a tie between the Einstein field equations and Nothingness as such.

Let us imagine a manifold where $a_{\mu\nu}$, the stress energy tensor of ordinary matter vanish, where $a_{\mu\nu} = 0$. Equation 62 changes to

$$\left(R_{\mu\nu} \times 0\right) - \left(\left(\left(\frac{8 \times \pi \times \gamma}{c^4}\right) \times T_{\mu\nu}\right) \times G_{\mu\nu}\right) = \left(+0 \times d_{\mu\nu}\right) - \left(b_{\mu\nu} \times c_{\mu\nu}\right) \tag{63}$$

But by the same reasoning there is nothing rather than something or

$$-\left(\left(\left(\frac{8 \times \pi \times \gamma}{c^4}\right) \times T_{\mu\nu}\right) \times G_{\mu\nu}\right) = -\left(b_{\mu\nu} \times c_{\mu\nu}\right) \tag{64}$$

while $b_{\mu\nu}$ contains the entire stress-energy of the manifold studied while $c_{\mu\nu}$ contains the entire gravitational field. This is more or less one typical feature of the exact electrovacuum solution of the Einstein field equation and may be of any emptiness at all.

The question is justified, is emptiness or nothingness absolute or relative or both or none and can we know self-consciously anything at all about any emptiness, the void, which does not exist? Radical advocates of a creation out of nothing prefer the possibility of total nothingness. Equation 64 in turn implies that there can be some nothingness but the same is relative too. However, a beginning of our world out of the empty negative is possible, theoretically.

3.4. *Index of relationship (IOR) and odds ratio (OR)*

Under circumstances where random events like A_t and B_t are fighting(Barukčić 2021a) [57] amongst themselves or fight constantly one another and are opposed to each other, it is more likely that $IOR(A_t, B_t) < 0$. If $IOR(A_t, B_t) > 0$ then it cannot be excluded that A_t and B_t are somehow conditional upon each other. However the index of dependence and odds ratio are related.

Theorem 4. *In principle, the index of dependence is more or less a reformulation of odds(see also Yule and Pearson 1900, p. 273) ratio (OR) in the form*

$$IOR(A_t, B_t) = \frac{(OR(A_t, B_t) - 1) \times (b \times c)}{(A \times B)} \tag{65}$$

Proof. In general, according to theorem 2, eq. 51,it is

$$(a \times d) - (b \times c) = (N \times a) - (A \times B) \tag{66}$$

Dividing by $(b \times c)$ as long as this is allowed and possible, we obtain

$$\frac{(a \times d)}{(b \times c)} - 1 = \frac{(N \times a)}{(b \times c)} - \frac{(A \times B)}{(b \times c)} \tag{67}$$

or

$$OR(A_t, B_t) - 1 = \frac{(N \times a)}{(b \times c)} - \frac{(A \times B)}{(b \times c)} \tag{68}$$

Rearranging eq. 68, we obtain

$$\frac{(OR(A_t, B_t) - 1) \times (b \times c)}{(A \times B)} = \frac{(N \times a) \times (b \times c)}{(b \times c) \times (A \times B)} - \frac{(A \times B) \times (b \times c)}{(b \times c) \times (A \times B)} \tag{69}$$

or

$$\frac{(OR(A_t, B_t) - 1) \times (b \times c)}{(A \times B)} = \frac{(N \times a)}{(A \times B)} - 1 \tag{70}$$

or

$$\frac{(OR(A_t, B_t) - 1) \times (b \times c)}{(A \times B)} = IOR(A_t, B_t) \tag{71}$$

From eq. 71 follows that

$$OR(A_t, B_t) = \left(IOR(A_t, B_t) \times \frac{(A \times B)}{(b \times c)} \right) + 1 \tag{72}$$

□

However, there are circumstances where

$$OR(A_t, B_t) = \left(\frac{a_t \times d_t}{b_t \times c_t} \right) \sim IOR(A_t, B_t) + 1 \tag{73}$$

In contrast to odds ratio, which has to potential to collapse if b = 0 or if c = 0, IOR, the index of dependence does not collapse. However, the index of dependence need not to be completely identical with Yule's Q (see also Yule and Pearson 1900; Yule 1912) which itself is defined as

$$\begin{aligned} Q(A_t, B_t) &\equiv \frac{\left(\frac{a_t \times d_t}{b_t \times c_t} \right) - 1}{\left(\frac{a_t \times d_t}{b_t \times c_t} \right) + 1} \\ &\equiv \frac{OR(A_t, B_t) - 1}{OR(A_t, B_t) + 1} \\ &\equiv \frac{\left(IOR(A_t, B_t) \times \frac{(A \times B)}{(b \times c)} \right)}{\left(IOR(A_t, B_t) \times \frac{(A \times B)}{(b \times c)} \right) + 2} \end{aligned} \tag{74}$$

[57] https://www.matec-conferences.org/articles/matecconf/abs/2021/05/matecconf_cscns20_09032/matecconf_cscns20_09032.html

3.5. *Example: Without sexual activity no prostate cancer*

Loeb et al. (see also Loeb et al. 2017) provided data on the relationship between marriage (sexual activity) and prostate cancer. The data of Loeb et al. (see table 7) support the null-hypothesis

without sexual activity **no** prostate cancer.

However, this hypothesis is supported by other studies too. The risk for prostate cancer is reduced significantly among childless men (see also Giwercman et al. 2005).

According to data (see table 10) published by Brawley (see also Brawley 2012) the age distribution of Americans diagnosed with prostate cancer is as follows:

Table 10. Age distribution and prostate cancer.

Age range	Prostate cancer proportion (%)
85+	4,4
75–84	19,9
65–74	35,3
55–64	30,7
45–54	9,1
35–44	0,6

The same data are illustrated by table 11.

Table 11. Age distribution and prostate cancer.

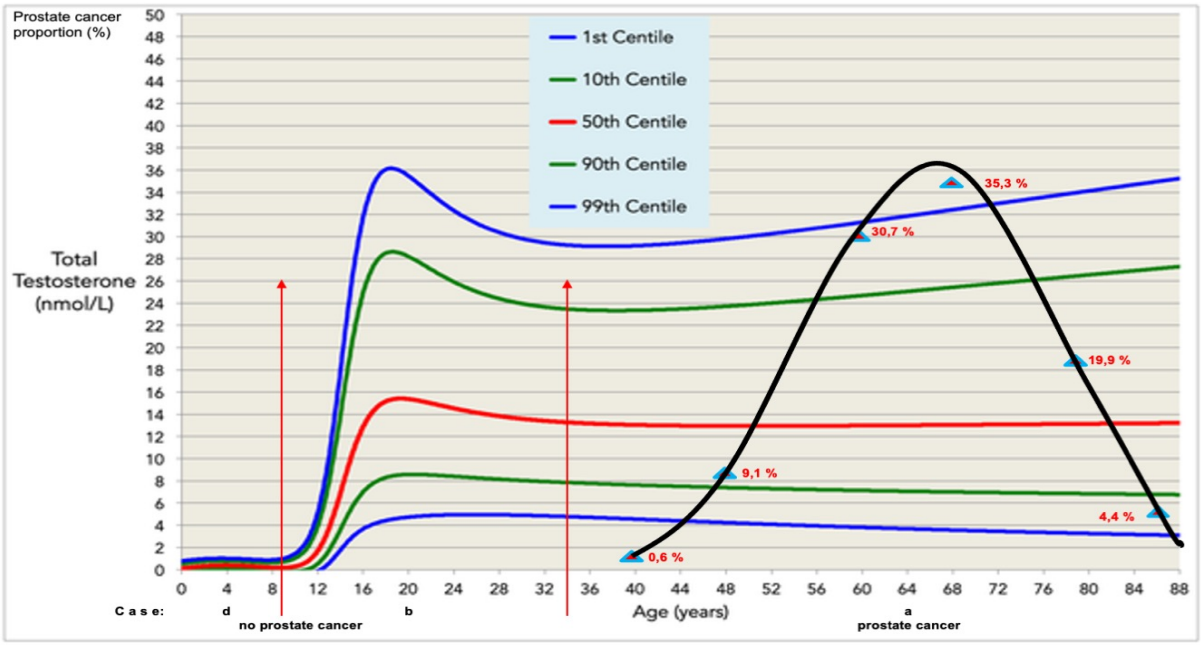
Postate cancer proportion (%)							
40,00							
35,00					35,30		
30,00				30,70			
25,00							
20,00						19,90	
15,00							
10,00			9,10				
5,00							4,40
0,50		0,6					
0,25							
0,00	0,00						
	0-34	35-44	45-54	55-64	65-74	75-84	84+
				Age range			

No American has been diagnosed with prostate cancer which has been younger than 35 years. In point of fact, everyday life supports the above hypothesis too. Sexually inactive male children up to an age of 5 years do not suffer frequently from prostate cancer and potentially not at all.

However, in contrast to expectation(see also Snyder 2008), healthy adult men maintain the total testosterone concentrations throughout life after total testosterone peaks at an average age of 19 years. In particular, Kelsey et al. (see also Kelsey et al. 2014) found "no evidence for a further fall in mean total testosterone with increasing age through to old age".

Combining the data of Brawley(see also Brawley 2012) et al. (see table 11) and the graph of Kelsey et al. (see also Kelsey et al. 2014) in one an the same graph, we obtain the following figure:

Fig. Total testosterone concentration, age distribution and prostate cancer.



Null-hypothesis: Without total testosterone concentration > 2 nmol/l no prostate cancer.

Fictive study

A fictive study under conditions of fair study design (see table 12) of the whole population should obtain data as follows.

Case a = 100 (see figure before):

Indicates the number of people with high/higher testosterone and prostate cancer.

Case b = 100 (see figure before):

Indicates the number of people with high/higher testosterone and no prostate cancer.

Case d = 100 (see figure before):

Indicates the number of people with no high/higher testosterone and no prostate cancer.

Case c = 0 (see figure before):

Would indicate the number of people with no high/higher testosterone and still prostate cancer. However, such cases are not observed.

Table 12. Total serum testosterone > 2 nmol/l and prostate cancer .

		Prostate cancer		
		YES	NO	
Total testosterone > 2 nmol/l	YES	100	100	200
	NO	0	100	100
		100	200	300

p(IOI)=	0,333333333
p(IOU)=	0
Causal relationship k =	+0,5000000000
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
IOR =	+0,5000

The right tailed p Value based on the hyper-geometric distribution is calculated as follows:

$$\begin{aligned} pValue_{\text{right tailed}}\left(X \geq 100\right) &\equiv 1 - \sum_{t=0}^{100-1} \left(\frac{\binom{200}{t} \times \binom{300-200}{100-t}}{\binom{300}{100}} \right) \\ &\equiv 0,0000000000 \end{aligned} \tag{75}$$

In other words, we have to accept the null-hypothesis:

without total serum testosterone > 2 nmol/l **no** prostate cancer (p Value = 0,0).

Testosterone is a necessary condition of prostate cancer. However, this does not mean at all that testosterone is the cause of prostate cancer too. Testosterone is responsible for many features of a male human being. Secondary sexual characteristics, spermatogenesis and especially the regulation of libido(see also Sharpe 1984) in the male are determined by the testosterone secretion from the Leydig cells in the testes in response to the gonadotrophin luteinizing hormone (LH) from the anterior pituitary gland. The libido itself leads to sexual activity, the same to a certain infection and the infection itself to prostate cancer. In this sense,

without total serum testosterone > 2 nmol/l **no** prostate cancer

is not incorrect.

Under conditions where a $\leq$ d, we expect an IOR of IOR $\geq$ 1,0, a cause effect relationship assumed to be given. Table 13 illustrates this consequence.

Table 13. Total testosterone $>$ 2 nmol/l and prostate cancer .

		Prostate cancer		
		YES	NO	
Total testosterone $>$ 2 nmol/l	YES	5	0	5
	NO	0	100	100
		5	100	105

p(IOI)=	0
p(IOU)=	0,904761905
Causal relationship k =	1,0000000000
p Value right tailed (HGD) =	0,0000000104
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
p (IMP) =	1,0000000000
$\tilde{\chi}^2$ (IMP — A_t) =	0,0000
$\tilde{\chi}^2$ (IMP — $\underline{B_t}$) =	0,0000
p Value (IMP) =	0,0000000000
p (SINE$\cap$IMP) =	1,0000000000
$\tilde{\chi}^2$ (SINE$\cap$IMP)$_1$ =	0,0000
$\tilde{\chi}^2$ (SINE$\cap$IMP)$_2$ =	0,0000
p Value (SINE$\cap$IMP) =	0,0000
p (EXCL) =	0,9523809524
$\tilde{\chi}^2$ (EXCL— A_t) =	5,0000
$\tilde{\chi}^2$ (EXCL— B_t) =	5,0000
p Value (EXCL) =	0,0465030452
IOR =	20,0000

The right tailed p Value based on the hyper-geometric distribution is calculated as follows:

$$\begin{aligned} pValue_{\text{right tailed}}\left(X \geq 5\right) &\equiv 1 - \sum_{t=0}^{5-1} \left(\frac{\binom{5}{t} \times \binom{105-5}{5-t}}{\binom{105}{5}} \right) \\ &\equiv 0,0000000104 \end{aligned} \tag{76}$$

We obtained an IOR = +0,5 (see table 12) while a $\leq$ d, which indicates a kind of a relationship. However, a causal relationship is under these conditions not really convincing.

4. DISCUSSION

Contrary to expectation, the study of Loeb et al. (see also Loeb et al. 2017) has not been able to clarify the relationship between testosterone and prostate cancer definitely. Nonetheless, the data suggest to some extent(see also Barukčić 2021b) that there is no relationship, however, the same data are self-contradictory too and of limited value with respect to this issue. Further and more differentiated investigations are necessary on this topic.

However, the limited data of Loeb et al. seems to indicate that prostate cancer is caused by an infectious agent. According to the data of Loeb et al.

without sexual activity **no** prostate cancer.

This is not really surprising since human papillomavirus has been identified as the cause(see also Barukčić 2019c) of human prostate(see also Lawson and Glenn 2020) cancer.

5. CONCLUSION

The relationship between testosterone and prostate cancer has not yet been conclusively clarified by Loeb et al. (see also Loeb et al. 2017) and by other too. This issue warrants further investigation.

6. IMPORTANT NOTE

The reader who is reading this article is invited to be aware that this article has not been published by a Web of Science, EBSCO, Scopus, PubMed/Medline et cetera and similar indexed journal. So one should be extremely cautious and very careful before taking the theorems derived in this publication formally as new or established scientifically validated knowledge.

7. PATIENT CONSENT OF PUBLICATION

Not required.

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9. TRANSPARENCY DECLARATIONS

The author declare that no conflicts of interest can be reported regarding the present study.

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REFERENCES

Stacy Loeb, Yasin Folkvaljon, Jan-Erik Damber, Joseph Alukal, Mats Lambe, and Pär Stattin. Testosterone replacement therapy and risk of favorable and aggressive prostate cancer. *Journal of Clinical Oncology: Official Journal of the American Society of Clinical Oncology*, 35(13):1430–1436, 5 2017. ISSN 1527-7755. https://doi.org/10.1200/JCO.2016.69.5304.

Charles M. Huggins and Clarence V Hodges. Studies on prostatic cancer: I. the effect of castration, of estrogen and of androgen injection on serum phosphatases in metastatic carcinoma of the prostate. *Cancer Research*, 1941(1):293–297, 1941. https://doi.org/https://doi.org/10.1016/s0022-5347(05)64820-3.

Marielle H. Emmelot-Vonk, Harald J. J. Verhaar, Hamid R. Nakhai Pour, André Aleman, Tycho M. T. W. Lock, J. L. H. Ruud Bosch, Diederick E. Grobbee, and Yvonne T. van der Schouw. Effect of testosterone supplementation on functional mobility, cognition, and other parameters in older men: a randomized controlled trial. *JAMA*, 299 (1):39–52, 1 2008. ISSN 1538-3598. https://doi.org/10.1001/jama.2007.51.

Giulia Rastrelli, Giovanni Corona, and Mario Maggi. Testosterone and sexual function in men. *Maturitas*, 112:46–52, 6 2018. ISSN 1873-4111. https://doi.org/10.1016/j.maturitas.2018.04.004.

Kevin T. Nead. Androgens and depression: a review and update. *Current Opinion in Endocrinology, Diabetes, and Obesity*, 26(3):175–179, 6 2019. ISSN 1752-2978. https://doi.org/10.1097/MED.0000000000000477.

Jacques Baillargeon, Randall J. Urban, Kenneth J. Ottenbacher, Karen S. Pierson, and James S. Goodwin. Trends in androgen prescribing in the united states, 2001 to 2011. *JAMA internal medicine*, 173(15):1465–1466, 8 2013. ISSN 2168-6114. https://doi.org/10.1001/jamainternmed.2013.6895.

Y. Cui, H. Zong, H. Yan, and Y. Zhang. The effect of testosterone replacement therapy on prostate cancer: a systematic review and meta-analysis. *Prostate Cancer and Prostatic Diseases*, 17(2):132–143, 1 2014. ISSN 1476-5608. https://doi.org/10.1038/pcan.2013.60.

Dario Bruzzese, Claudia Mazzarella, Matteo Ferro, Sisto Perdonà, Paolo Chiodini, Giuseppe Perruolo, and Daniela Terracciano. Prostate health index vs percent free prostate-specific antigen for prostate cancer detection in men with "gray" prostate-specific antigen levels at first biopsy: systematic review and meta-analysis. *Translational Research: The Journal of Laboratory and Clinical Medicine*, 164(6):444–451, 12 2014. ISSN 1878-1810. https://doi.org/10.1016/j.trsl.2014.06.006.

Ilija Barukčić. The causal relationship k. *MATEC Web of Conferences*, 336:09032, 2021a. ISSN 2261-236X. https://doi.org/10.1051/matecconf/202133609032.

Ronald Aylmer Fisher. The logic of inductive inference. *Journal of the Royal Statistical Society*, 98(1):39–82, 1935. ISSN 0952-8385. https://doi.org/10.2307/2342435.

Major Greenwood and G. Udny Yule. The statistics of anti-typhoid and anti-cholera inoculations, and the interpretation of such statistics in general. *Proceedings of the Royal Society of Medicine*, 8:113–194, 6 1915. ISSN 0035-9157. https://doi.org/10.1177/003591571500801433.

George Udny Yule and Karl Pearson. Vii. on the association of attributes in statistics: with illustrations from the material of the childhood society, &c. *Philosophical Transactions of the Royal Society of London. Series A, Containing Papers of a Mathematical or Physical Character*, 194(252–261):257–319, 1 1900. https://doi.org/10.1098/rsta.1900.0019.

George Udny Yule. On the methods of measuring association between two attributes. *Journal of the Royal Statistical Society*, 75(6):579–652, 1912. ISSN 0952-8385. https://doi.org/10.2307/2340126.

Giordano Bruno. *De la causa, principio et uno. Translated into German: Über die Ursache, das Prinzip und das Eine*. Reclams Universal-Bibliothek. Publisher: Unknown, translation by reclam, stuttgart 1986 edition, 1583. ISBN 978-3-15-005113-9.

Ilija Barukčić. *Die Kausalität*. Wiss.-Verl., Hamburg, 1. aufl. edition, January 1989. ISBN 3-9802216-0-1.

Ilija Barukčić. *Die Kausalität*. Scientia, Wilhelmshaven, 2., völlig überarb. aufl. edition, January 1997. ISBN 3-9802216-4-4.

Ilija Barukčić. *Causality: New statistical methods*. Books on Demand GmbH, Norderstedt, Germany, January 2005. ISBN 978-3-8334-3645-1.

Ilija Barukčić. The Mathematical Formula of the Causal Relationship k. *International Journal of Applied Physics and Mathematics*, 6(2):45–65, January 2016. https://doi.org/10.17706/ijapm.2016.6.2.45-65.

Ilija Barukčić. *Theoriae causalitatis principia mathematica*. Books on Demand, Norderstedt, 2017. ISBN 978-3-7448-1593-2.

Ilija Barukčić. Index of Unfairness. *Modern Health Science*, 2(1):p22, April 2019a. ISSN 2576-7305, 2576-7291. https://doi.org/10.30560/mhs.v2n1p22. URL https://j.ideasspread.org/index.php/mhs/article/view/260.

Ilija Barukčić. Index of Independence. *Modern Health Science*, 2(2):1–25, October 2019b. ISSN 2576-7305. https://doi.org/10.30560/mhs.v2n2p1. URL https://j.ideasspread.org/index.php/mhs/article/view/331.

Donatien-Alphonse-François Marquis de Sade. *La philosophie dans le boudoir ou les instituteurs immoraux. Dialogues destinés à l'éducation des jeunes demoiselles*. aux dépens de la Compagnie, 1795.

Ilija Barukčić. Human papillomavirus is the cause of human prostate cancer. *Journal of Drug Delivery and Therapeutics*, 9(4-s4-s):577–588, 8 2019c. ISSN 2250-1177. https://doi.org/10.22270/jddt.v9i4-s.3385.

A. Einstein. Die grundlage der allgemeinen relativitätstheorie. *Annalen der Physik*, 354(7):769–822, 1916. ISSN 1521-3889. https://doi.org/https://doi.org/10.1002/andp.19163540702.

Albert Einstein. Kosmologische betrachtungen zur allgemeinen relativitätstheorie. *Sitzungsberichte der Königlich Preußischen Akademie der Wissenschaften (Berlin)*, page 142–152, 1917.

A. Einstein and W. de Sitter. On the relation between the expansion and the mean density of the universe. *Proceedings of the National Academy of Sciences*, 18 (3):213–214, 3 1932. ISSN 0027-8424, 1091-6490. https://doi.org/10.1073/pnas.18.3.213.

Aleksander Giwercman, Lorenzo Richiardi, Magnus Kaijser, Anders Ekbom, and Olof Akre. Reduced risk of prostate cancer in men who are childless as compared to those who have fathered a child: a population based case-control study. *International Journal of Cancer*, 115(6):994–997, 6 2005. ISSN 0020-7136. https://doi.org/10.1002/ijc.20963.

Otis W. Brawley. Prostate cancer epidemiology in the united states. *World Journal of Urology*, 30(2):195–200, 4 2012. ISSN 1433-8726. https://doi.org/10.1007/s00345-012-0824-2.

Peter J. Snyder. Decreasing testosterone with increasing age: more factors, more questions. *The Journal of Clinical Endocrinology and Metabolism*, 93(7): 2477–2478, 7 2008. ISSN 0021-972X. https://doi.org/10.1210/jc.2008-0922.

Thomas W. Kelsey, Lucy Q. Li, Rod T. Mitchell, Ashley Whelan, Richard A. Anderson, and W. Hamish B. Wallace. A validated age-related normative model for male total testosterone shows increasing variance but no decline after age 40 years. *PloS One*, 9(10):e109346, 2014. ISSN 1932-6203. https://doi.org/10.1371/journal.pone.0109346.

R. M. Sharpe. Intratesticular factors controlling testicular function. *Biology of Reproduction*, 30(1):29–49, 2 1984. ISSN 0006-3363. https://doi.org/10.1095/biolreprod30.1.29.

Ilija Barukčić. The logical content of the risk ratio. *Causation*, 16(4):5–41, February 2021b. https://doi.org/10.5281/zenodo.4679509. URL https://doi.org/10.5281/zenodo.4679509.

James S. Lawson and Wendy K. Glenn. Evidence for a causal role by human papillomaviruses in prostate cancer - a systematic review. *Infectious Agents and Cancer*, 15:41, 2020. ISSN 1750-9378. https://doi.org/10.1186/s13027-020-00305-8.