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Smoking: the cause of human lung cancer.

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Abstract

Background. Some investigations found a strong association between smoking, particularly of cigarettes, and human lung cancer, the other not. The basic relation between smoking (nicotine) and human lung cancer still remains uncertain.

Methods. Sir William Richard Shaboe Doll**,** (28 October 1912–24 July 2005), analysed the relationship between smoking and human lung cancer in his historically important *case-control study* . Doll found that 647 of 649 lung cancer cases were smokers. In contrast to this, 622 of 649 non-cancer controls were smokers. Doll's study was reanalysed using the conditio sine qua non relationship and the mathematical formula of the causal relationship c. This methods are already known since 1989. All P values are one-sided; significance was indicated by a P value of less than 0.05.

Results. Using the conditio sine qua non relationship, it could be proofed that without smoking, particularly of cigarettes, no development of human lung cancer. On the other hand, using the mathematical formula of the causal relationship c, it could be proofed that smoking, particularly of cigarettes, is at the same time the cause of human lung cancer.

Conclusions. Without smoking, particularly of cigarettes, no development of human lung cancer. Smoking, particularly of cigarettes, is the cause of human lung cancer. Not smoking, particularly of cigarettes, will prevent from human lung cancer.

Key words: Causal relationship, Human lung cancer, Smoking, Cause, Effect, Barukčić

1. Introduction

Lung cancer as a cancer of the lungs is the most lethal of cancers today world-wide. This cancer is causing up to 3 million deaths annually. The five-year survival rate is about 10 per cent, that is to say one in ten patient diagnosed with lung cancer will survive the following five years. Lung cancer has been strongly associated with exposure to tobacco smoke. Tobacco smoke contains 19 known carcinogens such as nitrosamine, radioisotopes from the radon decay sequence and benzopyrene. Exposure to inhaled carcinogens, such as those present in tobacco smoke, is claimed to be by far the main contributor to human lung cancer. The amount smoked and length of time a person continues to smoke, particularly of cigarettes, is claimed to be able to increase person's chances of contracting lung cancer. Although lung cancer has been strongly associated with exposure is tobacco smoke, the role of smoking as the cause of lung cancer sill remains uncertain.

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2. Methods

2.1 Patients

Sir **William Richard Shaboe Doll,** (28 October 1912–24 July 2005), a British epidemiologist, was the first in the world who did pioneering work on the relationship between smoking and human lung cancer. Doll found in his historically important *case-control study* (Doll, 1950) that 647 of 649 lung cancer cases were smokers. In contrast to this, 622 of 649 non-cancer controls were smokers. Let us show this data in the following 2-2-table.

Smoking and human lung cancer.				
Doll R, Hill AB, "Smoking and carcinoma of the lung. Preliminary report", British Medical Journal, **2** (1950), 739 - 748.		Human lung cancer		
		Yes	No	
Smoking	Yes	647	622	1269
	No	2	27	29
		649	649	1298

2.1 Statistical Analysis

All statistical analyses were performed by self-programmed software. The new statistical techniques developed by Barukčić (Barukčić 1989, 2006a, 2006b; Thompson, 2006) were used. The formula of the conditio sine qua non was used to detect a **conditio sine qua non relationship** like **without** A **no** B between investigated random variables.
The mathematical formula of the **causal relationship c** (Barukčić 1989, 2006a, 2006b; Thompson, 2006) discovers causal relationships between experimental/non-experimental data. This formula was used to proof whether there is a significant causal relationship between smoking and human lung cancer. All P values are one-sided; significance was indicated by a P value of less than 0.05.

3. Results

3. 1. Without smoking no development of human lung cancer

Our hypothesis are:

Ho: Null-Hypothesis: p (Smoking ← Human lung cancer) = 1.
HA: Alternative-Hypothesis: p(Smoking ← Human lung cancer) < 1.

3.1.1 The probability of the conditio sine qua non relationship

The probability of the without smoking no lung cancer conditio sine qua non relationship was calculated from the data above (Barukčić 2006a, pp. 236-267) as

$$p(\text{Smoking} \leftarrow \text{Human lung cancer}) = \mathbf{0,999229583975346687211.}$$

3.1.2 The lower confidence bound of the conditio sine qua non relationship

The lower confidence bound of the conditio sine qua non relationship above was calculated from the data above (Barukčić 2006a, pp. 253-254) as

$$p_{lower} = \mathbf{0,986121815458665777014349956169676.}$$

The probability of the conditio sine qua non relationship

$$p(\text{Smoking} \leftarrow \text{Human lung cancer})$$

is higher then

$$p_{lower} .$$

In so far, the data above do support our Null-hypothesis:
without smoking
no human lung cancer.

We accept the Null-hypothesis and reject the Alternative-hypothesis ($p < 0.05$).

Without
smoking
no
development of human lung cancer.

3.2 Smoking: the cause of human lung cancer.

Smoking, particularly of cigarettes, could be the cause of human lung cancer. Let us reanalyse Doll's data (Doll, 1950) using Barukčić's mathematical formula of the **causal relationship c** (Barukčić 2006a, pp. 325-327) under this point of view. Our hypothesis are:

Ho: Null-Hypothesis: **c (Smoking $\Rightarrow$ Human lung cancer) $\leq$ 0**.
or there is no causal relationship between smoking, particularly of cigarettes, and human lung cancer.

HA: Alternative-Hypothesis: **c (Smoking $\Rightarrow$ Human lung cancer) > 0**.
or there is a causal relationship between smoking, particularly of cigarettes, and human lung cancer.

3.2.1 The calculated causal relationship $c_{calculated}$

The **causal relationship c** between smoking, particularly of cigarettes, and the development of human lung cancer was calculated according (Barukčić 2006a, p. 254, p. 317, p. 349) as

c = + 0,13031973814908427008619860372757 2.

3.2.2 The P value of the causal relationship c

The P value of the causal relationship c above was calculated (Barukčić 2006a, pp. 325-327) as

P value = 0,000001332191528.

3.2.3 The Power of the causal relationship c

The power of the causal relationship c above was calculated (Barukčić 2006a, pp. 332-335) as

Z_{β} = -1,23357441547384

power = 1 - p (Z_{β} = -3,0502755405678519377241000829213 7) = **0,9988568424726**,

a very strong and highly significant result. Thus, we reject our Null-Hypothesis and accept the alternative hypothesis. Conclusion.

There is a highly significant causal relationship between smoking,
particularly of cigarettes,
and the development of human lung cancer
(P value = 0,000001332191528, Power = 0,9988568424726).

Smoking is the cause of human lung cancer.

4. Discussion

The result above is highly significant. **Without** smoking, particularly of cigarettes, **no** development of human lung cancer. But smoking, particularly of cigarettes, is not only a conditio sine qua non of human lung cancer. Smoking, particularly of cigarettes, is at the same time the cause of human lung cancer (p value = 0,000001332191528, power = 0,9988568424726). **The cause of human lung cancer is identified.** Not smoking, particularly of cigarettes, will prevent from human lung cancer.

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