IS DIETARY ANIMAL PROTEIN CARCINOGENIC?

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The little nutritional science taught at conventional medical schools emphasises the notion that animal protein is the highest quality nutrient needed on a regular basis to build and maintain a healthy body. The word protein in fact comes from the Greek *proteios*, meaning "of prime importance". There have also been recent fad diets recommending only meats, eggs and cheeses, and no carbohydrates, to lose weight. To be on the safe side, most doctors would recommend a "balanced" diet, whatever "balanced" actually translates to at breakfast, lunch and supper.

This feature will therefore come as a surprise, outlining decades of published research pointing towards too much animal-based foods being the most important risk factor for the major types of cancer in both sexes. The farming lobbies are often accused of efforts to hinder exposure of these claims.

In 1968 an Indian research paper reported an experiment measuring hepatocellular carcinoma rates and protein consumption in two groups of laboratory rats. One group was given aflatoxin (a strong liver carcinogen) and diets containing 20% protein. The second group was given the same amount of aflatoxin and diets with only 5% protein. All the rats fed 20% protein got liver cancer or its precursor lesions, but not a single animal fed 5% protein got liver cancer or premalignant changes. An incredible 100% versus 0%. The protein used was cow's milk casein.[1]

Earlier, doctors in the Philippines claimed that hepatocellular carcinoma was more common in children than adults, compared to being commonest after 40 years of age in the West, and that the incidence of paediatric hepatic carcinoma was higher in rich best-fed Philippines families, possibly because they were eating much more animal protein than poor families.[2]

When the media reports a new chemical carcinogen, the public reaction is swift. In 1989 an apple industry growth regulator chemical (Alar) in the US was reported as "the most potent carcinogen in the food supply".[3,4] One woman called state police to chase a school bus and confiscate her child's apple, schools stopped serving apples, Alar use was halted, and the apple industry suffered a staggering financial loss.[5]

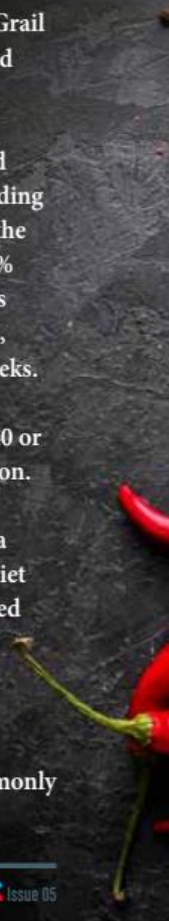
Sodium nitrite had been used as a meat preservative since the 1920s.[6] In 1970, the journal *Nature* reported that dietary nitrite may form nitrosamines in the body.[7] The US National Toxicology Program had declared that nitrosamines are "reasonably anticipated to be human carcinogens".[8,9] One nitrosamine, NSAR (N-nitrososarcosine), was given to 20 rats divided into two groups, one group getting twice the NSAR dose of the other group. Only 35% of rats on the low dose got throat cancer. All higher dose rats died of cancer in the second experiment year.[9] How much NSAR did the rats get? The low dose was equivalent to the NSAR a human would ingest by eating 270,000 sandwiches, each containing 500g cured ham, every day for over 30 years.[10] In another study, 10.2% of rats fed nitrite got lymphoma while only 5.4% of control animals not fed nitrite got lymphoma.[11] This created a public outcry. Marginal scientific results in animals fed exceptionally high levels of chemical for half their lifespan can make big waves in the public's perception but, when the dust settled, industry cut back on nitrite usage and the issue fell out of the spotlight.

The point does not relate to the safety of nitrite, but the mere possibility, however unlikely it may be, that it could cause cancer which alarms the public. So what if there was a chemical that experimentally turned on cancer in 100% of test animals and its relative absence limited cancer to 0% of the animals? Finding such a chemical would be the Holy Grail of cancer research. This is exactly what the aforementioned Indian research paper had claimed.[1]

The Indian animal experiment, whose results were initially not accepted by American scientists, was repeated and expanded upon in America. The effects of protein feeding on tumour development were spectacular and replicated the Indian findings. All rats administered aflatoxin, but fed 5% protein diet, were alive and active at 100 weeks and all rats given the same level of aflatoxin, and fed 20% protein diet, were either dead or moribund from liver cancer at 100 weeks. Again, the protein used was cow's milk casein.[12,13]

Furthermore, the diets of some rats were switched at 40 or 60 weeks to investigate the reversibility of cancer promotion. Animals switched from a high-protein to a low-protein diet had about 40% less tumour growth than animals fed a high-protein diet. Animals switched from a low-protein diet to a high-protein diet halfway through their lifetime started growing tumours. These findings confirmed nutritional manipulation can turn cancer "on" and "off".[12,13]

Cow's milk protein is undoubtedly a potent cancer promoter in rats dosed with aflatoxin. The fact that this promotion occurs at dietary protein levels (10-20%) commonly



"HE WHO DOES NOT KNOW FOOD, HOW CAN HE UNDERSTAND THE DISEASES OF MAN?"

HIPPOCRATES

used both in rodents and humans makes it especially provocative. So how does this research apply to human health and human liver cancer in particular? If casein's effect on cancer promotion is consistent across other species, other carcinogens and other organs, it might also apply to humans. Furthermore, and of great significance, experiments with plant protein (wheat and soya) did not promote tumour growth, even when fed high levels.[14]

In 1975 and 1982, studies claimed that chronic hepatitis B virus (HBV) infection was a major risk factor (20-40 times increased risk) for human liver cancer.[15,16] Researchers argued that both aflatoxin and HBV were key causes of human liver cancer but no one dared suggest that nutrition had anything to do with this disease. However, a subsequent study using two strains of transgenic HBV-transfected mice showed a similar result to the rat aflatoxin study. A 22% casein diet turned on expression of the viral gene to cause cancer, whereas a 6% casein diet showed almost no such activity.[17,18] It could now be concluded that cow's milk protein dramatically promotes liver cancer in rats dosed with aflatoxin and in mice infected with HBV.

Another study in rats dosed with two carcinogens showed that increasing intakes of casein promoted mammary cancer.[19] More studies used fish protein, dietary fats and carotenoid antioxidants, measuring the ability of these nutrients to promote liver and pancreatic cancer, thus proving nutrition controlled cancer evolution far more than the dose of the initiating carcinogen.[20]

Although there were strong arguments that these provocative findings were *qualitatively* relevant to humans, their *quantitative* relevance was unknown. In other words, is the animal protein and cancer relationship important for all humans in all situations, or is it marginally important for a minority of people in unique situations? Is this causative relationship responsible for one thousand, one million or more human cancers annually? The answer needed direct evidence from human research. This came from the so-called China Study, which The New York Times described as the "Grand Prix of Epidemiology", and "a story that needs to be heard" according to Robert C. Richardson, professor of Physics, Provost of Research at Cornell University and Nobel Prize Winner. Another feature will outline its findings.

Because nobody was taught the above at their medical school, readers might find its claims difficult to accept, also because the published material referred to is a few decades old. The American scientist (T. Colin Campbell PhD) leading these laboratory and epidemiological studies is the son of a farmer and, as would be expected, he originally dismissed the Indian paper (claiming animal protein is a cancer promotor) as bad research. So, as a good researcher would do, he repeated the Indian animal experiment and found its claim to be correct. I am unaware of any more recent published research disproving the above experimental claims of a relationship between dietary animal protein and cancer promotion.

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