T. COLIN CAMPBELL
Center for Nutrition Studies

Dr. T. Colin Campbell Timeline Slides - Full Text

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Raised on a dairy farm, milking cows. I was not especially aware of anything nutritional except that the milk that we were producing was the most perfect food. Indeed, milk was priced in the marketplace at that time according to its fat content, the higher the better. But nutritionally speaking, the main health value of milk was said to be its protein content and its protein 'quality'. It was our business and I was proud to be drinking more than most because, for us, it was so readily available. We also churned our own butter and made our own ice cream.

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Attended Penn State and University of Georgia Veterinary School (BS in Pre-veterinary Medicine, Penn State). My major at Penn State was pre-veterinary medicine and I received early admission (after 3 years) to a couple vet schools, but chose the University of Georgia because it served the Southeastern States without tuition. But near the end of the first year, I received an unsolicited offer of a scholarship for graduate studies at Cornell University in animal nutrition and biochemistry from a well-known professor, Clive McCay. I had become aware of the emergence of biochemistry as a relatively new science thus I was also pleased to drop vet school plans to do graduate study research at Cornell.

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Undertook graduate studies (Masters Thesis, 1957) in animal nutrition at Cornell University, doing experimental research on the ability of gut microorganisms to adopt the digestion of dietary fiber. This information demonstrated, to some extent, the concept of dietary adaptation that was, for me, a concept that later proved to be especially important. The adaptation in this case was attributed to a change in the gut microflora.

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Technician in a commercial laboratory, testing chemicals and irradiated foods for cancer-producing capability in experimental animals. This type of testing was the beginning of a long-standing program, still in existence even today, supported by government regulations to test for the ability of chemicals intentionally or unintentionally added to food to cause cancer in laboratory animals. It was triggered by a 1957 congressional amendment to the Food and Drug Regulations a called the Delaney Amendment. This grew out of a political storm that arose from the finding that a chemical herbicide added to cranberry plants could cause cancer. It became a very big political issue, in part because this finding occurred somewhat before the Thanksgiving Holiday, thus causing serious harm to the cranberry fanning business. Such chemicals took the name ‘chemical carcinogen‘. The subsequent congressional amendment stated that no amount of a chemical shown to be carcinogenic in experimental animals was allowed in foods destined

for human consumption, that is, it was interpreted as zero tolerance. The laboratory that I had joined was carrying out these studies for companies who had to make this determination.

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Graduate studies for PhD degree (PhD Dissertation, 1962) in animal nutrition (With minors in biochemistry and microbiology) at Cornell University, doing experimental research on utilization by ruminants of a nitrogen waste product to produce animal-based protein(1). I was testing for the ability of a non-protein nitrogen waste product, biuret, to be utilized by the microflora in the rumens of ruminants (cattle, sheep) to produce their own microbial protein that then could be digested and absorbed by the animal. In this way, biuret could be a new and cheap source of producing animal based proteins. Like my earlier study, ruminal bacteria were able to adapt to this never before seen compound and actually find a way to create an organism to utilize it. What a demonstration of nature! Although other laboratories subsequently used our research findings and continued to pursue the possibility of using this compound, I am not aware that it was ever successfully marketed. I should also mention that for 5 semesters during my graduate program I was a teaching assistant and on 3 of those occasions, had to teach students in the feeding of farm animals.

1. Campbell T.C., Loosli, J.K., Warner, R.G. & Tasaki, I. Utilization of biuret by ruminants. J. Animal Science 22, 139-145 (1963).

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Director of a commercial program in microbiology and radioisotope technologies, while testing, for the marketplace, chemicals for safety, using FDA guidelines. This was a spin-off toxicology testing laboratory of the earlier company, again testing for the possibility of commercial chemicals (herbides, pesticides, medicinals, etc.) causing cancer in experimental animals. While there, I also collaborated With the FDA to develop a biological testing procedure (bioassay) to evaluate a very toxic chemical found in the feed of poultry thought to be present in human food as well. It was called the ‘chick edema factor‘. The FDA official was then offered a position at the Massachusetts Institute of Technology (MIT) to develop a new toxicology program that could lead to a new department. He offered me a position to come with him to set up and run the laboratory.

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Continued investigating this so-called ‘chick edema factor‘. Isolated a material that clearly gave us the impression that it was the most toxic chemical ever discovered (2) but later identified by government scientists as dioxin. Other colleagues at FDA also continued to work on this same proiect, leading to the death of the one investigator who worked most closely with the

material. Eventually, this 'dioxin' was shown by others to be a contaminant of a defoliant being used in Vietnam called ‘Agent Orange‘.

2. Campbell, C. & Friedman, L. Chemical assay and isolation of chick edema factor in biological materials. J. Am. Assoc. Agri. Chem. 49. 824-828 (1966).

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Assistant to Full Professor at Virginia Tech, Department Of Biochemistry and Nutrition . Lecturer in biochemistry and toxicology. Coordinator of US State Department project in the Philippines to develop nationwide network of feeding centers for malnourished children, especially to insure adequate protein consumption. Set up laboratory to investigate effect of aflatoxin, a mold toxin found in peanuts and chemical carcinogen (for rats), as a cause of primary liver cancer in humans(3,4). l observed, anecdotally, that children most susceptible to primary liver cancer surprisingly consumed the most protein similar to consumption levels in U.S. (later, after discovery of hepatitis B virus by other researchers, this effect also was undoubtedly related to their chronic infection with hepatitis B virus). This observation in children corresponded with results of experimental animal (rats) studies in India showing that diets containing 20% protein (somewhat similar to human consumption in U.S.) compared with 5% protein (cow's milk protein) dramatically increased aflatoxin-initiated liver cancer(5). Many experimental rodent studies initially were undertaken(1) to confirm the effect of this protein on cancer and(2) to investigate by what mechanism this effect is this protein on this cancer and(2) to investigate by what mechanism this effect is expressed. Early studies showed that 20% dietary protein feeding substantially increased the metabolic activation of aflatoxin (in the liver) to form a highly reactive metabolite that chemically binds to DNA (the main component of genes), thus increasing the genetic potential for cancer initiation and growth. Multiple other explanatory mechanisms also contributed to this effect, suggesting that a cancer effect (or any other disease-producing effect?) is not attributed to a single mechanism(6-8)

3. Campbell, T. C. Present day knowledge on aflatoxin. Phil J Nutr 20, 193-201 (1967).

4. Campbell, T. C., Caedo, J. P., Jr., Bulatao-Jayme, J., Salamat, L. & Engel, R- W. Aflatoxin M1 in human urine. Nature 227, 403-404 (1970).

5. Madhavan, T. V. & Gopalan, C. The effect of dietary protein on carcinogenesis of aflatoxin. Arch. Path. 85, 133-137 (1968).

6. Campbell, T. C. & Hayes, J. R. Role of nutrition in the drug metabolizing system. Pharmacol. Revs. 26, 171-197 (1974).

7. Campbell, T. C. & Hayes, J. R. The role of aflatoxin in its toxic lesion. Tox. Appl. Pharm. 35, 199-222 (1976).

8. Preston, R. S., Hayes, J. R. & Campbell, T. C. The effect of protein deficiency on the in vivo binding of aflatoxin B1 to rat liver macromolecules. Life Sci. 19, 1191-1198 (1976).

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U.S. Senate Select Committee on Nutrition (chaired by Senator George McGovern) recommended decreased consumption of red meat and increased consumption of vegetables, fruits and whole grain cereals in order to decrease risk of heart disease(9). This activity of the McGovern Committee proved to be enormously troubling for politicians because it brought into question the main staple of the American diet, meat. McGovern later told me that he took more pride in this work than anything he had ever done, even if it caused him and some of his colleagues in the Midwestern States their political careers. This was one of my first deeply troubling experiences with the politics of science. This Senate committee report was based on emerging evidence in the scientific literature and focused on the effect of diet on heart disease. It was then that some of McGovern's colleagues wondered whether these same recommendations to prevent heart disease were consistent with the prevention of cancer. In order to answer that question, the Director of the National Cancer Institute (of NIH) was invited to give his views to a congressional hearing, testimony that I and other colleagues were first invited to review. It was then that I first learned that only 2-4% of the total budget was assigned to dietary and nutritional effects on cancer causation while it was also acknowledged by the same organization that 35% Of total cancers were attributed to diet and nutrition. I wondered how could this organization - the leading cancer research agency in the world - agree that one-third of all cancer could be prevented by diet (I believed that it was much higher) while simultaneously saying that we did not really know how diet works, yet then spend only 2-4% of its research budget trying to answer this question? A smell of obfuscation?

9. Select Committee on Nutrition and Human Needs (U.S. Senate). 83 (U.S. Government Printing Office Washington, DC, 1977).

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Accepted a full professorship at Cornell University, where experimental laboratory work was continued until about 1997. Made several observation that helped to establish nutrition

principles showing one: that experimental cancer development could be turned on an off, both early and late in cancer development, by modest changes in nutrient intake(6-8), two: that by appropriate nutritional means, early initiated cancer could be kept dormant for much of a lifetime, then recalled to growth later in life(10), three: that promotion of cancer was only for animal-based casein not for plant-based wheat and soy proteins(11) and four: that the usual dose-response observed for chemical carcinogens high dose, high response was completely obliterated by low protein feeding(12), among many other findings.

6. Campbell, T. C. & Hayes, J. R. Role of nutrition in the drug metabolizing system. Phamacol. Revs. 26, 171-197 (1974).

7. Campbell, T. C. & Hayes, J. R. The role of aflatoxin in its toxic lesion. Tox. Appl.

8. Preston, R. S., Hayes, J. R. & Campbell, T. C. The effect of protein deficiency on the in vivo binding of aflatoxin B1 to rat liver macromolecules. Life Sci. 19, 1191-1198 (1976).

10. Youngman, L. D. The growth and development of aflatoxin B1 –induced preneoplastic lesions, tumors, metastasis, and spontaneous tumors as they are influenced by dietary protein level, type, and intervention. (Cornell University, Ph.D. Thesis, Ithaca, NY, 1990).

11. Schulsinger, 0. A., Root, M. M. & Campbell, T.C. Effect of dietary protein quality on development of atlatoxin B1 -induced hepatic preneoplastic lesions. J. Natl. Cancer Inst. 81, 1241-1245 (1989).

12. Dunaif, G. E. & Campbell, T. C. Relative contribution of dietary protein level and Aflatoxin B1 dose in generation of presumptive preneoplastic foci in rat liver. J. Natl. Cancer Inst. 78, 356-369 (1978).

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Member of National Academy of Sciences panels on saccharin carcinogenicity(13) that showed difficulties of assessing cancer risks, especially as related to public perceptions of risk. This was another experience demonstrating how science can be so corrupted and distorted both by commercial interests and by politicians. Soft Drink companies went to extraordinary lengths to defend their use of saccharin, for example, claiming that it was the last of the non-nutritive, non-caloric sweeteners on the market. They made great claims throughout the marketplace that they were serving the public who wished to lose weight by consuming low calorie soft drinks. Ironically, however, not only was the experimental testing of saccharin for its carcinogenicity seriously challenged but in reality, experimental rats consuming levels of saccharin equivalent to human use actually became somewhat hypoglycemic and consumed MORE calories only to gain body weight!

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Dr. Junshi Chen from Chinese Institute of Food and Nutrition Science, one of first Chinese scholars to visit U.S. after rapprochement, does 8-month research sabbatical in Campbell laboratory. Dr. Chen had headed up a research group in China who were showing an effect of selenium supplementation on the reduction of a heart condition in children, Keshan Disease. Dr. Chen also was the first senior scientist in China to visit the U.S. for a sabbatical leave. Although those early findings became more complex over time, they illustrated a nutrient effect on the development of a chronic degenerative disease. While he was in the U.S., we learned of the release of an impressive series of atlases showing a highly unique geographic distribution of various cancers, based on a nationwide survey of cancer mortality rates in 2400 counties. We therefore decided to arrange for joint funding from NIH in the U.S. and the Chinese Ministry Of Health in Beijing to undertake a diet and lifestyle survey in 130 carefully selected villages in rural China. At about that same time, we invited Sir Richard Peto at the University Of Oxford, a world-renowned epidemiologist, and Dr. Li Junyao, lead author of the cancer atlas report, to join our group. This project became the first joint project between the U.S. and China.

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Member of National Academy of Sciences committee on Diet, Nutrition and Cancer(14) that was the first (reasonably) official report recommending for cancer prevention increased consumption of vegetables, fruits, and whole cereal grains and decreased consumption of total dietary fat to 30% of total calories. I was a member of this panel and on several occasions represented the committee in congressional hearings and in news media reports. It was an experience demonstrating the enormous public sensitivity and interest in diet and cancer.

14. Committee on Diet Nutrition and Cancer. Diet, nutrition and cancer: directions for research (ed. National Research Council, N. A. o. S.) (National Academy Press, Washington, D.C., 1983).

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Summary of 22 studies by R. Peto and colleagues consistently showing higher lung cancer rates with lower levels of circulating beta-carotene(15). This report generated considerable public interest in a role for a nutrient in cancer causation. Unfortunately, the commercial sector interpreted this finding to mean that beta-carotene supplementation could help to reduce cancer risk. Large human studies were organized and funded to test this hypothesis that eventually led to consistent findings that beta-carotene in the form of a supplement actually was associated with higher lung cancer risk while beta-carotene in the form of food was associated with lower lung cancer risk(16,17).

15. Peto, R., Doll, R. & Buckley, J. D. Can dietary beta-carotene materially reduce human cancer rates? Nature 290, 201-208 (1981).

16. Omenn, G. S. et al. Effects of a combination of beta carotene and vitamin A on lung cancer and cardiovascular disease. New Engl. J. Med. 334, 1150-1155 (1996).

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Atlas of Cancer Mortality in the People's Republic of China published in response to Chinese Premier Chou En Lai's affliction with cancer. Largest survey of disease mortality ever under taken. Included death rates for about a dozen types of cancer and three dozen other causes of death for more than 2,400 Chinese counties and 880 million (96%) of their citizens. Findings confirmed that, in China, cancer was geographically localized

18. Li, J.-Y. et al. Atlas of cancer mortality in the People's Republic of China. An aid for cancer control and research. Int. J. Epid. 10, 127-133 (1981).

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Sir Richard DoII and Sir Richard Peto published research findings on diet, lifestyle and cancer and concluded that 10%-70% of human cancers were attributed to diet while only 2%-3% of all cancers were attributed to genes(19). This was a striking finding that suggested that diet was far more important than genes in causing cancer.

19. Doll, R. & Peto, R. The causes of cancer. Quantitative estimates of avoidable risks of cancer in the Unites States today. J Natl. Cancer Inst.66, 1192-1265 (1981).

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Studies showing that lung cancer cases among 14,000 smokers is substantially lower when consuming beta-carotene containing vegetables(20), that risk for lung cancer among heavy smokers is reduced after about 10 years of non-smoking and that latent but unobserved experimental cancer could be 'promoted'(21). These studies collectively suggested that cancer not only could be prevented by plant based foods but also that it might be reversed by these same foods.

20. Shekelle, R. B. & Raynor Jr., W. J. Dietary vitamin A and risk of cancer in the Western Electric Study. Lancet 2, 1185-1190 (1981).

21. Berenblum, I. & Shubik, P. The persistence of latent tumour cells induced in the mouse's skin by a single application of 9:10-dimethyl-1:2-benzanthracene. Brit. J. Cancer 3, 384-386 (1949).

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Nationwide survey of diet and lifestyle characteristics conducted in China, then combined with 1973-75 Chinese disease mortality data(18) to produce 1990 monograph(22), a joint collaboration of the Chinese Academy of Preventive Medicine the Chinese Academy of Medical sciences, the University of Oxford and Cornell university and jointly funded by the U.S. National Cancer Institute (NIH), the Chinese government and the U.K. Imperial Cancer Research Fund.

13. Li, J.-Y. et al. Atlas of cancer mortality in the People's Republic of China. An aid for cancer control and research. Int. J. Epid. 10, 121-133 (1981).

22. Chen, J., Campbell, T. C., Li, J. at Peto, R. Diet, life-style and mortality in China. A study of the characteristics of 65 Chinese counties (Oxford university Press; Cornell university Press; People's Medical Publishing House, Oxford, UK; Ithaca, NY; Beijing, PRC, 1990).

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Demonstrated reversibility of early pancreatic cancer in experimental animal studies With altered fat diets featured on cover of the Journal of the National Cancer Institute(23).

23. O'Connor, T. P., Roebuck, B. O., Peterson, F. & Campbell, T. C. Effect of dietary intake of fish oil and fish protein on the development Of L-azaserine-induced preneoplastic lesions in rat pancreas. J. Natl. Cancer Inst. 15, 959-962 (1985).

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Professor Kenneth Carroll summarized findings from multiple countries showing that a higher animal fat intake but not plant fat was associated with increased breast cancer mortality(24), similar to earlier reports in a 1975 conference on diet and cancer(25).

24. Carroll, K. K., Braden, L. M., Bell, J. A. 81 Kalamegham, R. Fat and cancer. Cancer 58, 1818-1825 (1985).

25. Carroll, K. K. & Khor, H. T. in Progress in Biochemical Pharmacology: Lipids and Tumors 808-845 (S. Karger, New York, 1975).

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Reports showing that putative osteoporosis (as bone fracture rates) was associated with higher consumption of calcium(26) and animal protein(27) and that urinary calcium is increased with modestly increased consumption of protein (mostly animal based)(28).

26. Hegsted, D. M. Calcium and osteoporosis. J. Nutr. 116, 2316-2319 (1986).

27. Abelow, B. J., Holford, T. R. & lnsogna, K. L. Cross-cultural association between dietary animal protein and hip fracture: a hypothesis. Calcif. Tissue Int. 50, 14-18 (1992).

28. Margen, S., Chu, J.-Y., Kaufmann, N. A. & Calloway, D. H. Studies in calcium metabolism. I. The calciuretic effect of dietary protein. Am. J. Clin. Nutr. 27, 584-589 (1974).

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Re-surveyed same 65 (plus 4) counties in mainland China along with 16 additional counties in Taiwan for a total of 85 counties and 170 villages(29). This data shows the effect of westernization on dietary habits upon the development of cancer and other chronic degenerative diseases, (cancer, cardiovascular diseases, etc.).

29. Chen, C. J., P610, R., Pan, W.-H., Liu, B. & Campbell, T. C. Mortality, biochemistry, diet and lifestyle in rural China (Oxford University Press, Oxford, U.K., 2006).

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Caldwell Esselstyn, Jr., MD, Of the Cleveland Clinic, at a Tucson, AZ conference, reported dramatically reversed advanced heart disease among 18 seriously ill patients consuming a diet free of animal based foods and any added oil(30,31).

30. Esselstyn, C. in First National Conference for the Elimination of Coronary Heart Disease (Tucson, AZ, 1991).

31. Esselstyn, C. B., Elli5, S. G., Medendorp, S. V. & Crowe, T. D. A strategy to arrest and reverse coronary artery disease: a 5-year longitudinal study of a single physician's practice. J. Family Practice 41, 560-568 (1995).

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Reports showing that lung cancer rates increased with beta-carotene from supplements but decreased with beta carotene with food(16, 17), demonstrating importance of consuming nutrients as foods not as dietary supplements.

16. Omenn, G. S. et al. Effects of a combination of beta carotene and vitamin A on lung cancer and cardiovascular disease. New Engl. J. Med. 334, 1150-1155 (1996).

17. The Alpha-Tocopherol Beta Carotene Cancer Prevention Study Group. The effect of vitamin E and beta carotene on the incidence of lung cancer and other cancers in male smokers. New Engl. J. Med. 330, 1029-1035 (1994).

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Closed experimental laboratory operations to focus on delivering message to the public. Considerable concern that experimental research had become too reductionist.

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Published "The China Study, Startling Implications for Diet, Weight Loss and Long-Term Health", co-authored by T. Colin Campbell and Thomas M. Campbell ll.

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Presented hundreds of lectures in U. S. and abroad to a diversity of audiences, thus gaining perspectives on this diet for multiple other ailments and diseases.