“The China Study”: A Formal Analysis and Response

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Introduction

When I first embarked on an analysis of T. Colin Campbell's "The China Study," I did not anticipate the range or magnitude of responses it would invoke—reactions that have been at times controversial, at times impassioned, and at times downright heated, but above all else intellectually provocative. It seems "The China Study" is a book that, in many cases, is either intensely revered or vehemently criticized, and its ability to generate ongoing discussion signifies a deep-seated division in the scientific community.

I would like to thank Mr. Campbell for his cordial response to my critique, as well as for the time he has taken to elucidate his philosophy of nutrition and his approach to research. While I do not agree with some of his conclusions, I honor his contributions to the field of health and nutrition, and deeply admire his courage to promote an unpopular message amidst a research sector dominated by special interests and opposing views.

I propose that Campbell's hypothesis is not altogether wrong but, more accurately, incomplete. While he has skillfully identified the importance of whole, unprocessed foods in achieving and maintaining health, his focus on wedding animal products with disease has come at the expense of exploring—or even acknowledging—the presence of other diet-disease patterns that may be stronger, more relevant, and ultimately more imperative for public health and nutritional research.

Having lit a proverbial fuse, I feel called and compelled to make the sum of my findings available to the public so that they may add, in whatever extent or direction, to the symphony of voices engaged in this discourse. My intent with this paper is not to discredit Campbell as a scientist, nor to promote or discourage a particular diet—but rather, to present new ways of looking at the China Study data and related research while highlighting the shortcomings in Campbell's specific conclusions. I hope this information can be valuable to readers while—above all else—encouraging the use of independent, critical thought to advance our understanding of health.

Section 1:
Reiteration and Expansion of Criticisms

Although some of the following points have been discussed previously, they were largely dismissed as "reductionist." Given Campbell's preference to examine nutrition from a holistic perspective with less focus on individual components, his assessment is understandable, albeit inaccurate. I cite these points not to split nutritional hairs, but to reveal a consistent pattern of bias and misrepresentation as it relates to Campbell's hypothesis.

Here, I present my original points once more with additional information and references to highlight their relevance.

1. **An attempt to link animal protein with cancer by way of cholesterol—a chain of variables that exhibits several logical and statistical shortcomings**.

In citing the China Study data, Campbell states that total cholesterol is "positively associated with most cancer mortality rates" and also "positively associated with animal protein intake."[1] However, he provides no indication that he examined or accounted for the cancer-risk-raising variables associated with cholesterol, including schistosomiasis and hepatitis B infection.[2]

Additionally, per Campbell's own assessment, cholesterol is only one of several variables that tend to cluster alongside Western-type diseases: The others include higher blood glucose levels, increased consumption of refined carbohydrates, higher beer intake, and industrial rather than agricultural employment[3,4]—with the latter bringing changes in lifestyle and increased work hazards such as benzene exposure, an extensively studied cause of lung cancer, leukemia, and other lymphatic and hematopoietic malignancies in Chinese factory workers.[5,6]

This entanglement of risk-raising factors casts doubt on the usefulness of cholesterol as an indicator of animal food consumption rather than of accompanying variables—especially considering the lack of a known biological mechanism that causes cholesterol to rise from increased protein consumption.

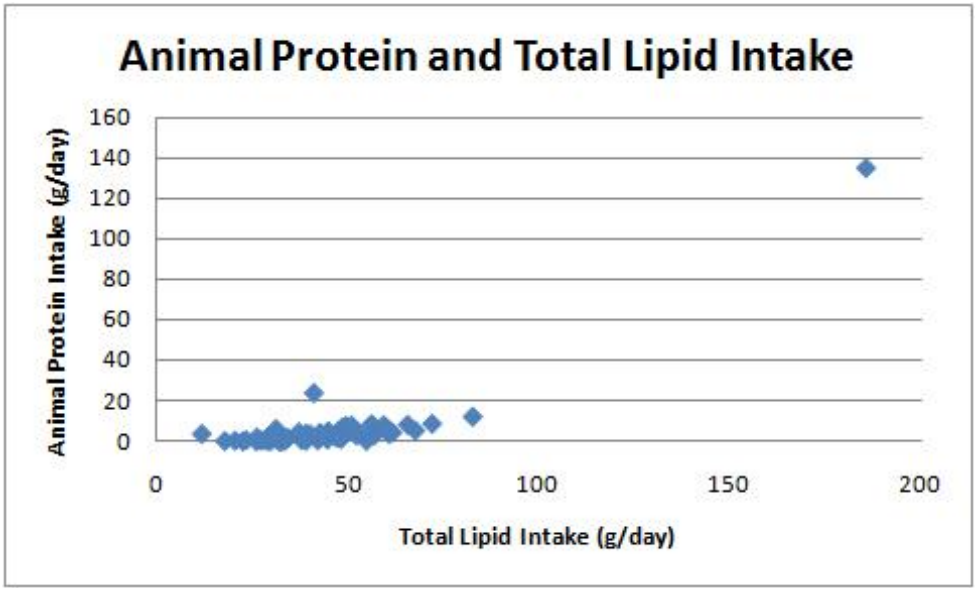
As previously mentioned, Campbell also fails to cite direct links between animal foods themselves and cancer, relying instead on biomarkers as a liaison. Since epidemiological data can only identify trends and not cause-and-effect sequences, and because some diseases intrinsically alter blood profiles, the assumption that higher cholesterol precedes disease is also unsubstantiated. To link specific foods or a category of foods with disease requires evidence that the foods themselves—independent of confounding factors—influence disease risk; the reliance on biomarkers that only partially relate to dietary items is too indirect to yield truly meaningful conclusions.

2. **The association of breast cancer with lipid intake—and lipid intake with animal protein consumption—as a means to link breast cancer with animal foods.**

Although many of Campbell's observations about breast cancer in rural China align with widely accepted risk factors, such as earlier menarche and greater exposure to hormones, Campbell relies on an intermediary variable (fat consumption) to forge a link that does not exist directly between animal foods and breast cancer. If an intermediary variable is introduced, a positive association can superficially emerge where the direct association is actually neutral or mildly negative. On page 86 of "The China Study," Campbell suggests that in China, "the association between fat and breast cancer might really be telling us that as consumption of *animal-based foods* goes up, so does breast cancer"—an idea spawned from his observation that animal protein consumption correlates strongly with lipid intake. On the same page, he notes that the correlation between fat consumption and animal protein was "very high, at 70 - 84%"—with 70% expressing the linear relationship between animal protein and percentage of calories as fat, and 84% expressing the linear relationship between animal protein and total lipid intake.[7]

However, these figures—especially the persuasively high 84%—may be overestimated. Campbell has noted that the data for the county with the highest animal protein and fat intake, Tuoli, was "clearly not accurate on the 3 days that the data were being collected," because "on those days, they were essentially eating as if it were a feast to impress the survey team."[8] Further, Campbell states that Tuoli was "intentionally excluded from virtually all our analyses" because of its misleading values for meat intake.[9]

Tuoli County was not, however, excluded from the calculation for the association between animal protein consumption and lipid intake, which has a correlation of 0.84 or 84% only when using all 65 counties. A visual graph of the data reveals Tuoli's strong influence in this correlation, as represented by the data point at the far right.



If the Tuoli data is ultimately unreliable and reflects a short-term "feast" of meat, its inclusion in the animal protein/lipid correlation may be erroneous. Since Campbell has provided no method for

correcting the data nor indicated what more reliable values may be, omitting this county from the calculation may be warranted.

With the removal of Tuoli, the correlation between animal protein intake and total lipid intake drops from 0.84 to 0.52 ($p<0.001$). While still high and statistically significant, this correlation is low enough to undermine Campbell's immediate implication of animal foods with breast cancer via lipid consumption.

More importantly, once the data set is corrected for Tuoli County, the correlation between plant oils and total fat intake rises drastically. Using all 65 counties, the variable "Oil intake other than rapeseed"—which includes soybean oil, corn oil, cottonseed oil, peanut oil, and sesame oil—has a non-significant linear correlation of 0.18,[10] and the variable "Rapeseed oil intake" correlates at 0.08.[11] When Tuoli is removed from calculation, these numbers rise to 0.42 ($p<0.001$) and 0.25 ($p=0.514$), respectively.

Using the data set with the flawed inclusion of Tuoli, Campbell cites a strong association between animal protein and lipid intake as a reason to implicate animal foods with breast cancer. Yet using the revised data set, animal foods do not contribute significantly more fat to total lipid intake than do plant oils. As a result, any association between breast cancer and dietary fat could be linked to either animal or plant-sourced foods, and there is no justification for indicting only animal products.

This may be particularly relevant because, according to one of Campbell's publications, certain plant oils such as corn have carcinogenic properties; he notes that "Increased intake of corn oil has previously been shown to promote the development of L-azaserine-induced preneoplastic lesions in rats,"[12] a similar phenomenon to what his research demonstrated with casein.

Nonetheless, the association with lipid intake and breast cancer may not even be noteworthy. From his paper "Additional ecological evidence: lipids and breast cancer mortality among women age 55 and over in China," Campbell et al conclude:

> Although the result is consistent with a positive association between lipid intake and breast cancer risk, the observed association is weaker than the association previously observed. This finding provides only modest support for the possibility of a diet-breast cancer link.[13]

Neither the association between animal foods and lipid intake nor lipid intake and breast cancer, then, is particularly strong. This casts doubt on the accuracy of Campbell's statement that the China Study data showed a "connection of breast cancer with dietary fat, [and] thus with animal-based foods."[14]

Moreover, another variable may be more relevant than lipids when exploring the mechanisms behind breast cancer occurrence. Although Campbell emphasizes the importance of biological models and clinical research to corroborate epidemiological data, particularly univariate correlations, he does not examine the positive correlations between breast cancer and blood glucose or processed starch and sugar consumption in the China Study data,[15] even in light of research showing these associations may be highly relevant. Research spanning the previous decade has revealed a potential role of blood glucose levels in the development of breast cancer,[16] has linked hyperinsulinaemia with both early menarche and

breast cancer,[17] and has shown that high insulin levels are a risk factor for breast cancer independent of estrogen.[18] Additional discussion of the relationship between blood glucose, insulin, and cancer is included under item 6.

The animal food/breast cancer hypothesis is directly testable in the China Study data, given the records for total animal protein consumption as well as amount and frequency of meat intake, fish intake, dairy intake, and egg intake. Yet despite his stated hypothesis that animal-based foods correspond with increased breast cancer rates, Campbell provides no indication of excavating a direct link through any of his analyses.

3. **The claim that animal products and plasma cholesterol increase rates of liver cancer in high-risk populations.**

Stratified data shows that high-risk areas for liver cancer have a nearly neutral association with animal food intake, and inverse associations with meat, eggs, and dairy.[19] Of all animal food variables, only fish and fish protein are strongly correlated with liver cancer[20]—an issue Campbell et al address in the publication "Fish consumption, blood docosahexaenoic acid and chronic diseases in Chinese rural populations":

> [It] is not difficult to visualise the reason for the link with liver cancer [and fish consumption]. The coastal, estuarine and lacustrine regions with the high fish and sea food intakes are also those with the highest humidities. Storage of food in regions of high humidity is known to encourage the spread and growth of hepatitis B virus and Aspergillus flavus which produces aflatoxin, both are major causes of primary carcinoma of the liver.[21]

Moreover, an identifiable relationship between cholesterol and liver cancer does not prove causality. Although Campbell implies that elevated cholesterol levels increase liver cancer risk, the inverse may be true: Hypercholesterolemia has been identified as a complication of liver cancer,[22] often in conjunction with hypoglycaemia and hypercalcaemia.[23]

4. **The incomplete statement that cardiovascular disease is inversely associated with green vegetable consumption, and the three-variable linkage between animal protein intake, apolipoprotein B (apo-B), and cardiovascular disease.**

In his 1998 publication "Diet, lifestyle, and the etiology of coronary artery disease: the Cornell China Study," Campbell states that coronary artery disease mortality rates were "inversely associated with the frequency of intake of green vegetables ($r = -0.43$, $p<0.01$)"[24] in rural Chinese populations—a statement cited elsewhere as a significant finding of the China Study.[25]

Although frequency of green vegetable consumption does boast a strong inverse correlation with heart disease in the unadjusted data, the actual amount of green vegetables consumed has a weak positive correlation ($r = 0.05$)[26]—a paradox Campbell does not mention or seem to explore. Had Campbell examined this discrepancy closer, he would notice the strong regional patterns associated with frequency of green vegetable consumption, including humidity ($r = 0.68$, $p<0.001$), heat ($r = 0.61$, $p<0.001$), elevation ($r = -0.48$, $p<0.001$), and latitude ($r = -0.60$,

$p<0.001$),[27] all of which suggest this variable serves as a geographical marker and thus is likely associated with other regional risk factors and protective factors for disease.

While Campbell has noted he prefers to view nutritional patterns in the aggregate rather than individually, the "green vegetable paradox," as I've termed it, is representative of similar and repeated oversights, potentially weakening his hypothesis as a whole. When referring to the China Study data, Campbell cites misleading figures when they endorse plant food consumption—without first completing the analytical steps necessary to prove their accuracy and eliminate confounding. Likewise, he consistently omits similar correlations that indicate a neutral or protective effect between animal foods and disease, even when those trends, too, seem to form an overarching pattern.

Furthermore, Campbell cites a chain of three variables to implicate animal protein with coronary heart disease: He notes that animal protein associates with the low-density lipoprotein fraction apo-B, that apo-B associates with increased mortality from coronary artery disease, and therefore concludes that animal protein associates with heart disease.[28] Although the first two statements are correct in isolation, the leap to the latter is unsupported by logic and contradicted by the China Study data.

While Campbell found it appropriate to cite an unadjusted correlation for frequency of green vegetable intake, had he done the same for animal food variables, he would find only neutral or inverse correlations between cardiovascular disease and:

- amount of meat consumed ($r = -0.28$)*
- frequency of meat consumption ($r = -0.15$)
- amount of fish consumed ($r = -0.15$)
- frequency of fish consumption ($r = -0.14$)
- amount of eggs consumed ($r = -0.13$)
- frequency of egg consumption ($r = -0.14$)
- animal protein intake ($r = 0.01$)
- fish protein intake ($r = -0.11$)[29]

** When Tuoli county is omitted, this correlation becomes $r = -0.36$, $p<0.05$. Other listed correlations do not significantly change.*

The only animal food with a positive (though still not significant) correlation with heart disease is dairy, both in amount ($r = 0.06$ for full data set; $r = 0.12$ adjusted for Tuoli) and frequency ($r = 0.11$ for full data set; 0.12 adjusted for Tuoli).[30] However, considering dairy is generally only consumed in three counties, the accuracy of these correlations is difficult to determine.

Non-dairy animal foods do not consistently correlate with shared geographical features,[31] vegetable consumption,[32] or plasma antioxidants,[33] thus minimizing the possibility of common protective factors masking their true effect on heart disease. In light of this—and given Campbell's interest in finding "overarching" themes in nutrition—it seems curious he does not explore the consistent inverse relationships between most animal foods and cardiovascular disease. While it is possible that any or all of these figures require additional adjustments to

account for confounding, Campbell offers no indication that he did so for patterns with vegetable consumption before embracing its inverse association with heart disease.

Likewise, while Campbell readily accepts favorable correlations with plant-food variables and disease, he does not account for the numerous correlations that run contrary to his hypothesis—particularly the association between all non-rice grains and heart disease, including: yearly ration of wheat ($r = 0.51$, $p<0.001$), yearly ration of corn ($r = 0.31$, $p<0.05$), yearly ration of sorghum ($r = 0.31$, $p<0.05$), yearly ration of millet ($r = 0.37$, $p<0.05$), wheat flour per day ($r = 0.67$, $p<0.001$), and "other cereal" intake per day, which includes corn, millet, sorghum, and barley ($r = 0.39$, $p<0.01$). Additional plant variables exhibit a positive association as well, including total fiber, per food composite ($r = 0.30$, $p<0.05$) and total plant protein, per food composite ($r = 0.21$), likely due to the influence of these grains.

Because these are unadjusted correlations, they are only preliminary and illustrative, not conclusive. However, if Campbell is seeking overarching patterns of nutrition and disease, it seems this is one worth inspecting.

Lastly, although Campbell also cites a correlation between apo-B and cardiovascular disease, the biomarker he typically uses to connect animal foods with health afflictions—total plasma cholesterol—is ineffective in the case of heart disease. In the paper "Erythrocyte fatty acids, plasma lipids, and cardiovascular disease in rural China," Campbell et al conclude:

> Within China neither plasma total cholesterol nor LDL cholesterol was associated with CVD [cardiovascular disease]. The results indicate that geographical differences in CVD mortality within China are caused primarily by factors other than dietary or plasma cholesterol.[34]

5. **The use of unadjusted univariate correlations to link fiber with reduced colorectal cancer rates, green vegetables with reduced stomach cancer rates, and plant-food biomarkers with reduced stomach cancer rates.**

Drawing from unadjusted China Study data, Campbell cites several perceived effects of plant foods and plant biomarkers on colorectal health and stomach cancer. While Campbell has stated his approach to nutrition is one of holism rather than reductionism, repeated use of unreliable correlations ultimately weakens the hypothesis they help construct.

In citing an inverse association between 14 fiber fractions and colorectal cancer,[35] Campbell provides no indication that he tested for confounding variables—a significant oversight, given that schistosomiasis infection appears to be both a major risk factor for colorectal cancer ($r = 0.89$, $p<0.001$) and less common in regions with high fiber consumption ($r = -0.23$ for total fiber intake). As demonstrated visually and verbally in my first response to Campbell, the protective effect of each fiber faction convincingly echoes its correlation with schistosomiasis, suggesting schistosomiasis may potentially create or accentuate the inverse relationship between fiber and colorectal cancers.[36]

Similarly, Campbell cites other unadjusted correlations related to plant foods. He notes an inverse association with stomach cancer and green vegetable intake, plasma beta-carotene, and plasma vitamin C[37]—patterns aligning with his hypothesis that plant foods are protective against disease.

The problem isn't that the correlations are invalid; they may, after more analysis, persist. However, Campbell selects them above stronger associations that contradict his hypothesis and provides no indication of adjusting for appropriate factors. This approach allows him to build a repertoire of supportive evidence that may only be superficially congruous with his hypothesis. Scientific rigor mandates more even-handed analyses, which Campbell has not done.

6. **The use of a three-variable chain to connect animal-based foods with cholesterol and cholesterol with "Western" diseases.**

To form a comprehensive method for examining disease patterns, Campbell creates two dichotomous sets of diseases—one associated with affluent living conditions and one associated with poverty. While searching for underlying nutritional patterns characterizing the diseases of affluence, he observes that plasma cholesterol has a positive correlation with the collective group, and concludes that "one of the strongest predictors of Western diseases was blood cholesterol."[38]

Given that a variety of factors—dietary and otherwise—can influence cholesterol and the cause-and-effect relationship between cholesterol and disease is not always clear, Campbell's use of cholesterol as an intermediary between animal foods and disease is unsubstantiated. For instance, a shift from a highly active lifestyle in agriculture-dominated regions to a more sedentary one in industrialized areas may, in itself, be enough to explain higher cholesterol levels in certain areas[39]—a plausible theory, given that regions with greater industry employment in the China Study tended to have higher total cholesterol ($r = 0.45$, $p<0.001$) but exhibited no significant association with animal protein intake ($r = 0.04$).[40]

More importantly, a different plasma variable may be even more relevant than cholesterol in the occurrence of Western diseases: blood glucose.

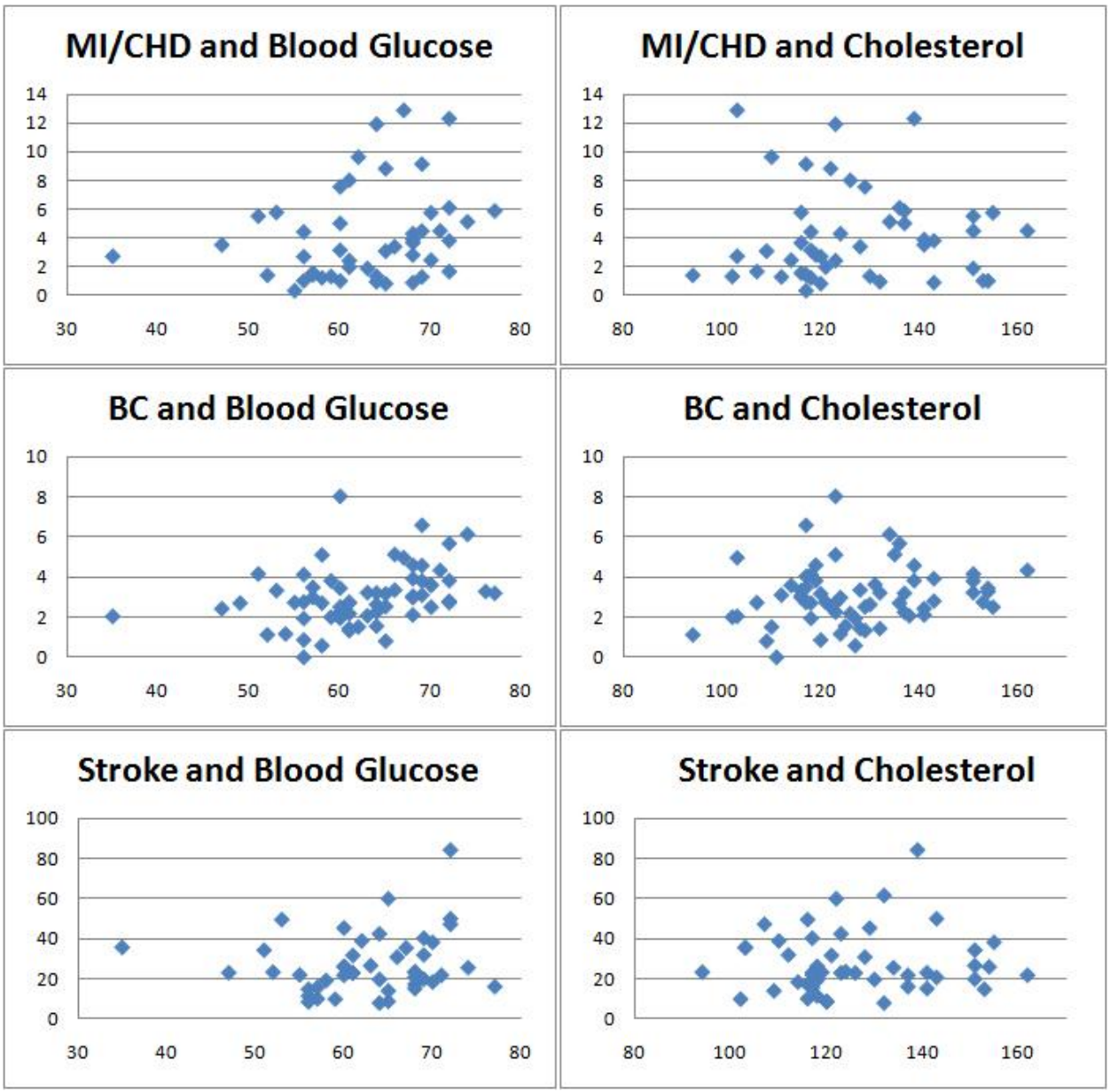
Blood glucose: an overlooked clue

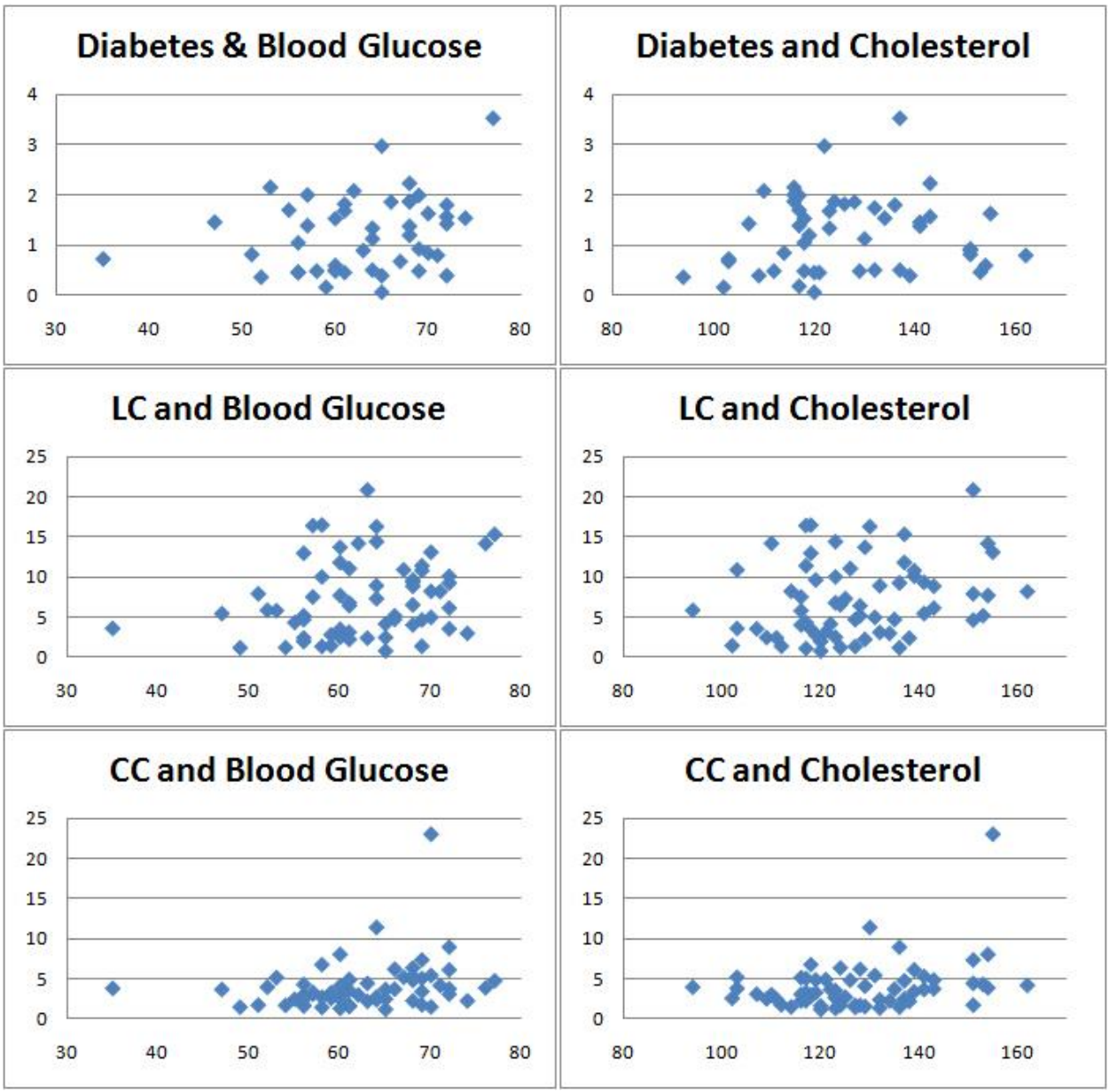
Although Campbell cites cholesterol as a consistent risk factor for Western diseases, blood glucose also exhibits associations with the chronic conditions most prevalent in affluent nations—including the ones less convincingly linked to cholesterol.

Notably, blood glucose tends to have a distinct nonlinear relationship with disease, and its association with cardiovascular diseases, cancer, and diabetes may therefore appear diminished when studying only linear correlations, as Campbell has generally done with cholesterol. The following graphs present the subtle "U-curve" pattern between blood glucose and diseases common to Western nations, juxtaposed with the same diseases and cholesterol.

On all left-side graphs, the horizontal axis represents blood glucose levels as mg/dL, while the vertical axis represents disease mortality per 1000 deaths. On all right-side graphs, the

horizontal axis represents total cholesterol as mg/dL, while the vertical axis represents disease mortality per 1000 deaths.
Acronyms: MI/CHD = myocardial infarction/coronary heart disease; BC = breast cancer; LC = lung cancer; CC = colorectal cancer.





An examination of the Third National Health and Nutrition Examination Survey (NHANES) revealed that every 50 mg/dL increase in plasma glucose corresponded with a 22% increase in cancer mortality risk, possibly due to the proliferative and anti-apoptotic properties of glucose and insulin.[41] In another study, a cohort of more than 140,000 Austrian adults revealed that elevated blood glucose is associated with multiple cancers in both men and women, including liver cancer, breast cancer, bladder cancer, gallbladder cancer, non-Hodgkin's lymphoma, and thyroid cancer.[42]

Although averaged blood glucose levels in China all fell within the normal range, if Campbell hypothesizes that increases in even very low cholesterol collide with greater disease risk, it

would not be a stretch—by his logic—to suggest that small increases in even normal-range blood glucose could do the same.

On pages 78-79 of "The China Study," Campbell writes:

> As blood cholesterol decreased from 170 mg/dL to 90 mg/dL, cancers of the liver, rectum, colon, male lung, female lung, breast, childhood leukemia, adult leukemia, childhood brain, adult brain, stomach and esophagus (throat) decreased.

However, citing the same univariate correlations, he could have also written:

> As plasma glucose decreased, cancers of the liver, rectum, colon, male lung, female lung, breast, childhood leukemia, adult leukemia, childhood brain, adult brain, stomach, bladder, and cervix, as well as childhood and adult lymphoma, decreased.

In addition to potentially spurring cancer growth, blood glucose and insulin may play a pivotal role in the development of atherosclerosis. A 2009 paper by Nunes and Silva[43] reveals that, "among several systemic parameters studied, plasma glucose was found to be correlated to coronary artery atherosclerosis lesions" and remained strongly correlated after accounting for other variables. The researchers note:

> In the context of the present investigation, one may speculate that higher plasma glucose, probably in the presence of elevated plasma insulin, could be associated to a growth-stimulating effect on atherosclerotic lesions, perhaps involving magnesium as a cofactor for insulin-stimulated growth.

In addition, Nunes and Silva mention that "in the present study, we could find no evidence of an association between lipid fractions and CADB [coronary artery disease burden]."

A separate study by Brunner et al determined that glucose intolerance associates with increased mortality risk from all causes, stroke, and respiratory disease. The researchers state that the findings of their study "provide clear evidence that coronary mortality is raised among those with marginal postload hyperglycemia,"[44] and suggest several possible explanatory mechanisms:

> A raised glucose level at baseline may indicate emerging insulin resistance and a downward trajectory in glycemic control, with increased risk of glucose intolerance, diabetes, and CHD in subsequent years. … Other pathways include oxidative stress and formation of advanced glycation end products that accelerate atherosclerosis when blood glucose is only slightly raised.

It is, of course, impossible to determine a cause-and-effect relationship in epidemiological data, but given that there is already corroborating research and biological plausibility for links between glucose, insulin, and disease, it seems the glucose-disease trend is one worth exploring. A combination of diet and lifestyle changes—especially ones associated with industry-dominated regions, where diseases of affluence tended to occur—could serve as precursors for metabolic

abnormalities and insulin resistance, contributing to or occurring alongside heightened risk for cardiovascular disease and many cancers.[45,46]

Dismissing other relevant variables

Campbell claims that "even small increases in the consumption of animal-based foods" were "associated with increased disease risk" in the China Study data,[47] but dismisses other relevant food variables because they are eaten in lower quantities in China than in most Western nations, and are thus "probably more indicative of general economic conditions and other local circumstances than of biological relationships to disease."[48] The illogic in this statement becomes apparent when comparing the actual ranges of these variables. For instance, once the outlier Tuoli is omitted, animal protein consumption in China ranged from 0g to 23.92g per day, whereas processed sugar and starch consumption ranged from 0g to 27g and beer intake ranged from 0g to 357.1g per day. Yet even though Campbell finds the small intake animal protein intake to be relevant, he dismisses other variables with even wider ranges due to being "consumed in much lower quantities" than in the United States.[49]

How can Campbell be certain that increases of animal protein from 0g to 24g are relevant, but increases in processed starch and sugar from 0g to 27g are not—especially if those increases coincide with other shifts towards a more Western lifestyle, such as decreased physical activity?

The potential significance of other variables cannot be dismissed based on subjective assessments of their importance. As explored later in this paper, some ethnic groups who have subsisted on a constant traditional diet for centuries or millennia respond with disproportionate levels of disease to the introduction of new, Western-style foods and lifestyle habits.[50] In studies of immigrants, Asians eating Western diets appear to have excessively high rates of diabetes and insulin resistance compared to their non-Asian counterparts,[51] suggesting a "predisposition to insulin resistance and its metabolic abnormalities."[52] In addition, studies of lean Chinese adults have demonstrated that insulin resistance often occurs independent of obesity, and even normal-weight Chinese are susceptible to impaired glucose tolerance.[53]

If there is widespread inability to handle high-glycemic foods like sugar and a predisposition to metabolic abnormalities, then what Campbell deems an insignificant intake by Western standards may, in actuality, be highly pertinent to the Chinese.

Projection of casein's carcinogenic properties to all forms of animal protein

As I explored in my original critique and clarified again in my follow-up post, Campbell's extrapolation of his casein research to all forms of animal protein—as well as the assumption that casein behaves the same way in whole-food form as when isolated—is supported by neither clinical evidence nor logic.

Campbell draws his animal protein/cancer hypothesis from a series of experiments conducted on aflatoxin-exposed rats, which revealed dramatic differences in the cancer growth depending on the level of protein consumed. As Campbell explains, rats fed a diet of 5% protein in the form of casein exhibited dramatically fewer lesions than rats fed a diet of 20% casein.[54] Additional experiments showed that wheat and soy protein did not promote cancer growth, even when fed at the 20% level.[55] From this,

Campbell concludes that casein could be the "most relevant carcinogen ever identified."[56]

Yet in a 1989 study, Campbell discovered that wheat protein exhibited similar carcinogenic properties as casein when lysine, its limiting amino acid, was restored.[57] This suggests that any complementary combination of amino acids will spur cancer growth under certain experimental conditions, and that carcinogenic qualities are not unique to casein nor to animal protein at large. The sole reason plant protein appeared protective in rat studies was due to a deficiency in one or more amino acids, a scenario that rarely occurs in real-world situations when a variety of foods—whether plant or animal in origin—are consumed. Campbell himself notes that eating a variety of plant foods provides a full spectrum of amino acids[58]—indicating that even a plant-only diet can yield the complete protein Campbell claims to be carcinogenic.

However, the notion that complete protein is inherently carcinogenic is contradicted by more recent literature. Although Campbell's focus on casein is understandable, given the research is chiefly his own, he does not acknowledge the abundance of similar studies showing that whey—another milk protein—consistently boasts anti-cancer properties,[59,60,61] including when studied under the same experimental conditions that demonstrate the carcinogenic qualities of casein.[62,63] This is significant, as even a single example of animal protein inhibiting rather than spurring cancer invalidates Campbell's hypothesis that the effects of casein can be extrapolated to all animal protein.

Section 2:
Biological Models and Cited Papers

An evaluation of Campbell's cited papers and relevance to biological models

In Campbell's second response to my critique of "The China Study," he notes:

> The China project encouraged us not to rely on independent statistical correlations with little or no consideration of biological plausibility. In the book, I drew my conclusions from six prior models of investigation to illustrate this approach: breast cancer, liver cancer, colon cancer (minimally), energy utilization/body weight control, affluent disease-poverty disease and protein vs. body growth rates.[64]

Campbell then cites "a few representative publications" in which he applies supportive data to biological models, purportedly demonstrating their congruency.

Do these publications and the biological models they employ implicate animal products as causative of disease? To answer this question, I've examined several of Campbell's cited papers, evaluating in each case whether the biological models Campbell draws from support his use of specific data to verify an animal food/disease hypothesis.

Breast cancer

Publication: Marshall JR, Qu Y, Chen J, Parpia B, and Campbell TC. "Additional ecological evidence: lipids and breast cancer mortality among women age 55 and over in China." Eur J Cancer. 1992;28A(10):1720-7.

In the first paper he cites for breast cancer, Campbell et al use the China Study data to explore the relationship between breast cancer mortality and a variety of risk indicators— searching specifically for associations between fat consumption and higher cancer rates. They note that, although animal experiments consistently demonstrate greater breast cancer risk from increasing dietary fat, that "human data are less consistent," and that "several well-designed and executed case-control studies apparently failing to show any association."

Drawing from plasma samples, red blood cell samples, diet survey results, and questionnaire answers, Campbell et al study breast cancer mortality in relation to:

- total cholesterol
- high density lipoprotein (HDL) cholesterol

- low density lipoprotein (LDL) cholesterol
- triglyceride levels
- apolipoprotein A-1
- apolipoprotein B
- total lipid saturates
- total lipid polyunsaturates
- ratio of total lipid polyunsaturates to saturates
- total dietary lipid intake
- total caloric intake
- percentage of caloric intake from lipids
- physical height
- physical weight
- Quetelet index
- alcohol consumption
- age at menarche
- age at first pregnancy, and
- total live births

For the diet variables, the researchers do not distinguish between animal and plant-derived fats—an important observation, given the equivalent contribution of plant versus animal fats to total fat intake after adjusting for the outlier county Tuoli, as explained in section 1.

From this study, Campbell et al conclude that, among cholesterol fractions, the "strongest and most consistent predictor of risk is apolipoprotein A-1," and that "higher levels are consistently associated with greater breast cancer risk" even after adjusting for other variables. Incidentally, apolipoprotein A-1 appears to be significantly associated with rapeseed oil intake, but not with animal protein.[65]

The researchers also note that "increased red blood cell saturated fat is associated with an insignificantly lower risk of breast cancer" ($r = -0.16$), although the plasma lipid indicators may provide unreliable measures of county status.

Although researchers do cite a weak positive relationship between lipid intake and breast cancer rates, no examination of specific animal foods such as meat, fish, dairy, eggs, or total animal protein is included in this paper's analyses. Similarly, researchers offer no rationale for using lipid intake or blood markers as a liaison specifically between animal products and disease.

Moreover, Campbell et al state that the China Study data offers only a weak indication of a relationship between diet and breast cancer. As mentioned in section 1, they conclude from their findings that the data "provides only modest support for the possibility of a diet-breast cancer link."

Additionally, Campbell et al acknowledge that their results "may well be confounded," particularly because their analysis unveiled several anomalous associations—such as a relationship between higher parity (number of live-born children) and increased cancer risk, as

well as higher age at first birth and lower cancer risk. These associations, the researchers note, "contradict nearly all individual-based studies." The accuracy of this particular data set and the trends extracted from it may thus be in doubt.

At best, the associated biological model involves a possible—but "modest"—role of lipid consumption in the development of breast cancer, with no distinction made between plant or animal sources of fat. While Campbell attempts to implicate animal foods due to a univariate correlation between animal protein and total lipid intake, the estimated correlation—as addressed earlier—is likely to be steeply overestimated due to the inclusion of Tuoli county in the calculation, an outlier whose survey data Campbell deemed "unreliable."

Publication: Key TJA, Chen J, Wang DY, Pike MC, and Boreham J. "Sex hormones in women in rural China and in Britain." British Journal of Cancer 1990(62):631-636.

In the second paper Campbell cites, researchers examine plasma concentrations of hormones implicated with breast cancer in Chinese versus British women—namely oestradiol, testosterone, sex hormone binding globulin, and prolactin. In comparing several physical and reproductive variables between Chinese and British women, researchers highlight significant differences in height, weight, age of menarche, age of first pregnancy, and age of menopause.

Results of the analysis reveal that British women generally had higher concentrations of oestradiol and testosterone than did the Chinese group, but that variations in testosterone "may be due to the difference in body weight."

The researchers note several shortcomings in the comparisons between these two countries, stating:

- The Chinese samples were collected at a different time of day than the British samples, and were only collected during a three-month period rather than year round—a significant observation, because hormone levels often fluctuate throughout the day as well as varying seasonally.
- Blood sample collection and processing methods differed between the two countries, possibly confounding the results.

Potential data inaccuracies aside, the researchers hypothesize that the differences in oestradiol between Chinese and British women may be due not only to the Chinese's low fat diet, but also to their greater levels of exercise, additional dietary practices, or other lifestyle factors that differ between the two populations. As with the previous paper Campbell cited, this publication does not discuss a potential role of animal foods as either direct or indirect causative agents of breast cancer. More specifically, it offers rationale for assuming animal foods, as a collective group, cause elevated hormone levels more than other dietary or lifestyle constituents.

Liver cancer

Publication: Campbell TC, Chen J, Liu C, Li J, Parpia B. "Nonassociation of aflatoxin with primary liver cancer in a cross-sectional ecologic survey in the People's Republic of China." Cancer Res 1990(50):6882-6893.

In exploring risk factors for primary liver cancer in rural China, Campbell et al conclude that liver cancer mortality was unassociated with exposure to the carcinogen aflatoxin, but was positively and significantly associated with hepatitis B infection, total cholesterol, and intake of cadmium from plant foods. The paper notes a high correlation between cholesterol and liver cancer, and posits animal foods as the cause:

> Plasma cholesterol was highly significantly associated with PLC mortality rates. … In this study, even though it was very low compared to the United States, it tended to be associated with the intakes of foods of animal origin.

The referenced table cites a list of univariate correlations between cholesterol and 12 variables, as follows:

Table 3 ***Univariate correlation coefficients for various dietary and nutritional factors with average (males and females) plasma cholesterol in 65 rural Chinese counties***

Variable	*r*
Dietary intake	
Lipid (% kcal)	0.30[a]
Dietary fiber (g/day)	−0.27[a]
Dietary cellulose (g/day)	−0.37[b]
Total protein (g/day)	0.07
Animal protein (g/day)	0.24
Fish protein (g/day)	0.33[b]
Dairy (g/day)	0.21
Eggs (g/day)	0.21
Meat (g/day)	0.26[a]
Legumes (g/day)[c]	−0.35[b]
Blood constituents	
Plasma albumin (g/dl)	0.33[b]
Other characteristics	
Height (cm)	0.28[a]

[a] $P < 0.05$.
[b] $P < 0.01$.
[c] Data based on amount rationed in each county; mostly soy beans.

Campbell et al used only univariate correlations to link animal food variables to cholesterol, without adjusting for other cholesterol-raising factors that may cluster alongside them. Moreover, had the researchers omitted Tuoli county from these calculations—a reasonable choice, given that the erroneous data of this region strongly influences any correlations involving meat, dairy, fat intake, and animal protein intake—several of the figures would be somewhat attenuated. The corrected data set yields a correlation of 0.18 instead of 0.24 with animal protein

and cholesterol, 0.03 instead of 0.21 for dairy, and 0.18 instead of 0.26 for meat. These numbers are lower than other variables correlating with cholesterol, such as daily beer consumption ($r = 0.32$, $p<0.01$), daily liquor consumption ($r = 0.20$), total daily alcohol consumption ($r = 0.21$), intake of soybean, corn, cottonseed, sesame, or peanut oil ($r = 0.20$) and industry employment ($r = 0.45$, $p<0.001$)—with the latter accompanying other lifestyle factors that may lead to higher cholesterol, such as reduced physical activity.

Most importantly, cholesterol may not actually be a cause of liver cancer—but rather, an effect. In a study of 792 Chinese patients with liver cancer, Hwang et al discovered that 11.4% of subjects were hypercholesterolaemic, but they exhibited a return of normal cholesterol levels following surgery and chemoembolization for their conditions:

> Serum cholesterol levels fell to the normal range after treatment and rose to abnormal levels again when tumours recurred after surgery. … Serum cholesterol levels may serve as another marker in identifying tumour recurrence and the presence of a viable tumour mass in hypercholesterolaemic HCC patients.[66]

Thus, liver cancer itself may cause cholesterol to rise, independent of diet or lifestyle factors. If this is the case, the influence of animal products on blood cholesterol would be irrelevant, and a direct link between animal foods and liver cancer would be necessary to prove their association. With the exception of fish—which Campbell et al have explained associates with liver cancer due to climatic and geographical factors[67]—such a relationship is not apparent.

In addition, it should be noted that this paper has received criticism from other cancer researchers who consider its conclusions unfounded. In 1991, Christopher P. Wild and Ruggero Montesano of the International Agency for Research on Cancer submitted a letter to *Cancer Research* stating:

> We were concerned by the conclusions drawn by Campbell et al. in their recent paper in which they reported (a) a lack of association between urinary aflatoxin metabolites and primary hepatocellular carcinoma in 48 counties in the People's Republic of China and (b) a positive association with plasma cholesterol. We consider the conclusions unsubstantiated and misleading …[68]

Wild and Montesano proceed to outline flaws in the experimental methods used by Campbell et al—including problems associated with the urinary assay of aflatoxin, the researchers' lack of adjustment for urine concentration, and seasonal variations in aflatoxin exposure that could yield misrepresentative data. Therefore, the validity of the paper itself may be in question.

Energy utilization

Publication: Campbell TC and Chen J. "Energy balance: interpretation of data from rural China." Toxicol Sci. 1999 Dec;52(2 Suppl):87-94.

In this paper, Campbell and Chen synthesize information from earlier lab rat studies, other animal research, and the China Study—particularly data recorded during a three-day diet survey,

which revealed that rural Chinese citizens have a high average calorie intake compared with most Americans. Based on this apparent calorie paradox, they hypothesize that a low-protein diet increases thermogenesis, and that:

> some unknown but significant, and probably difficult to measure, amount [of extra calorie intake] could be due to increased energy expenditure associated with non-post-prandial basal metabolism.

The implication is that low-protein diets may be effective for maintaining a healthy body weight because they divert a "biologically meaningful but difficult to measure" amount of energy away from weight gain and into body heat. Although the researchers do not specifically describe how this mechanism would occur in humans, they draw from animal involving brown adipose tissue metabolism.

However, the researchers concede that the increased calorie intake exhibited by the Chinese may simply be due to exercise:

> Undoubtedly, much of the increased energy intake but lower body weight in rural China, as measured in this survey, was attributable to their greater physical activity (i.e., it is common to see most office workers riding bicycles to work) …

Moreover, given Campbell's earlier disclosure that one county was "essentially eating as if it were a feast to impress the survey team"[69] during the three-day survey, the validity of the recorded energy intake is in question. How certain is Campbell that other counties were not altering their eating habits to give the impression of greater wealth or food abundance, thus leading to an overestimate of the average calorie intake for the Chinese?

Given the possible overestimation of calorie intake in the China Study, the reliance on animal rather than human studies, and the inability to calculate whether the increased calorie consumption was or was not fully offset by physical activity, this hypothesis rests on the accuracy of many unknowns. While its validity is still possible, the evidence at hand is insufficient to confirm it.

Affluent-poverty diseases

Publication: Campbell TC, Junshi C, Brun T, Parpia B, Yinsheng Q, Chumming C, and Geissler C. "China: From diseases of poverty to diseases of affluence. Policy implications of the epidemiological transition." Ecology of Food and Nutrition 1992(27):133-144.

The publication Campbell cites to explain his "diseases of poverty" and "diseases of affluence" model has already been mentioned throughout this paper, but briefly, its premise is that two disease clusters naturally emerged from the China Study data. Diseases in one group are "generally associated with impoverished conditions," while diseases in the second group are "characteristic of more affluent societies." The second self-clustered group includes stomach cancer, liver cancer, colon cancer, lung cancer, breast cancer, leukemia, diabetes, and coronary heart disease. By viewing each assembly of diseases in the aggregate, Campbell seeks to identify

underlying nutritional patterns in their collective emergences—potentially deciphering the source of rising disease rates in affluent nations.

While this method may be useful for examining general disease patterns, Campbell's chief errors are as follows.

1. **Disregard for potentially critical variables in disease proliferation**. Although Campbell acknowledges that numerous variables associate with the "diseases of affluence" cluster—including intake of processed starch and sugar, beer intake, fish consumption, egg consumption, and industry work—he dismisses all but cholesterol, citing the rest as "probably more indicative of general economic conditions and other local circumstances" than as causative of disease. In addition, other biomarkers such as plasma glucose may be of equal or greater relevance compared to cholesterol but receive no mention in his publication. Campbell's disregard for these variables appears to be subjective, rather than a result of the thorough analysis necessary for deeming them insignificant.
2. **"Reductionist" use of cholesterol as a disease indicator.** In examining health and nutritional trends, Campbell takes the same reductionist approach he censures elsewhere by targeting cholesterol as the chief predictor for disease. By linking cholesterol solely to animal food consumption and disregarding the numerous other variables that may cause it to rise, Campbell overlooks the larger context of disease mechanisms as they pertain to diet and lifestyle.
3. **Inaccurate representation of true diseases of affluence**. Campbell's dichotomization of diseases, while useful in some cases, does not accurately reflect disease rates in developed countries:
    - Stroke, the third leading cause of death in the United States,[70] does not fit cleanly into either the affluent or poverty disease group, so Campbell omits it entirely.
    - Heart disease correlates only weakly positively or, in three cases, inversely with the other diseases in the affluent cluster, suggesting it may not be strongly associated with the other conditions and is potentially a result of separate geographic, nutritional, or lifestyle variables.
    - Liver cancer is relatively uncommon in affluent nations, but exhibits strong correlations with the variables Campbell ascribes to diseases of affluence, most notably cholesterol. This provides further indication that cholesterol may not be an appropriate or dependable biomarker for examining true Western diseases in relation to diet.
4. **Oversight of a third, potentially significant disease cluster.** Myocardial infarction, hypertensive heart disease, stroke, brain and neurological diseases, and diseases of the blood and blood-forming organs share strongly statistically significant correlations with each other and with shared nutritional variables, such as non-rice grain consumption, while correlating inversely with the variables associated with diseases of affluence. Despite this, Campbell forces myocardial infarction into a disease cluster it does not naturally align with, and ignores the remaining diseases rather than attempt to explain their anomalous nonassociation with other Western conditions.

Consequently, Campbell's use of these disease clusters to identify relationships between diet and diseases of Western nations may be unsound, especially given a myopic focus on cholesterol to the point of excluding other pertinent factors.

Summary

While biological models, as Campbell notes, are essential for developing a comprehensive understanding of nutrition and disease mechanisms, the ones he employs do not validate the claim that animal foods are unhealthful—the hypothesis that inspired my original skepticism and critique. The biological models he cites fail to support the three-variable chains he creates to implicate animal products with cancer, heart disease, and other chronic conditions, and his use of univariate correlations to impose these links remains unfounded.

Moreover, the models Campbell cites center on individual biomarkers in disease mechanisms—examples of the same reductionism Campbell claims to oppose. If disease mechanisms work in a "symphony," as Campbell has described, and if animal products are harmful in the aggregate rather than due to single nutrients, then a direct relationship between animal food consumption and disease should be identifiable.

Section 3:
Response to Points Raised by Campbell

Although the previous sections have covered—directly and indirectly—my rationale for citing certain errors in Campbell's work, I will use the following pages to address more specific concerns Campbell raised regarding my critique.

Wheat: confounded variable or legitimate concern?

In his second response to my critique, Campbell cites a rhetorical remark I included in my analysis—an inquiry as to why he did not appear to have explored the strong ($r = 0.67$, $p<0.001$) correlation between wheat and heart disease,[71] despite citing far weaker correlations as a means to implicate animal products with various conditions. In offering possible explanations for the wheat-heart disease link, Campbell presents several relevant correlations that are "all highly statistically significant ($p<0.01$ to $p<0.001$)," including:

> Higher wheat flour consumption, for example, is correlated, as univariate correlations, with lower green vegetable consumption (many of these people live in northern, arid regions where they often consume meat based diets with little no consumption of vegetables).[72]

Although Campbell is correct in noting a highly statistically significant ($r = -0.63$, $p<0.001$) inverse correlation between wheat flour and green vegetable consumption, he uses the same variable whose speciousness I discussed earlier: frequency, rather than quantity, of green vegetable intake. Citing this correlation only shows—somewhat redundantly—that wheat is consumed in northern regions where many crops grow seasonally rather than year-round. The correlation with quantity of green vegetable consumption, however, is an attenuated -0.16, and wheat happens to positively correlate with protective plant foods such as light-green vegetables (0.10) and carrots (0.27, $p<0.05$).

Additionally, Campbell's claim that "many of these people … consume meat based diets" is fairly incongruous with the China Study data itself, which reveals that only one county—the now-discredited Tuoli—consumed any significant portion of meat, even though 22 counties consumed at least 100g of wheat flour per day. And in contrast to what Campbell asserts, wheat flour correlates at -0.22 with frequency of meat consumption and at -0.09 with amount of daily meat intake (reducing further to -0.23 and -0.22, respectively, when recalculated without Tuoli). Although animal protein, correlates at 0.17 with wheat flour consumption when using all 65 counties, this figure, too, reduces when Tuoli is excluded from calculation, dropping to -0.06.

Campbell is likely aware of the lack of a wheat-meat link, as a paper he coauthored in 1998, entitled "Diet, lifestyle, and the etiology of coronary artery disease: the Cornell China Study," presents the following conclusion:

> Nonetheless, the wheat-flour effect appears to be independent of meat consumption, so enhancement of coronary artery disease risk by wheat consumption may be a possibility.[73]

Despite the "possibility" that one of the most widely-consumed grains may contribute to heart disease, Campbell does not pursue this issue further through research or in his book, continuing to focus instead on animal foods.

In his response, Campbell points to another pertinent correlation with wheat: "greater body weight (higher risk of heart disease)," which correlates at 0.59 ($p<0.001$) with wheat flour intake.[74] This, of course, raises an important issue: Why do people in wheat-eating regions tend to have significantly higher body weight than citizens of other areas? The answer does not appear to be calories, as wheat flour only correlates at 0.07 with total caloric intake.[75] Nor is the answer lower activity associated with industrial employment, as wheat flour correlates at -0.24 with percentage of the population employed in industry.[76] And given the lack of association with meat or other animal foods, animal protein is an equally unlikely solution. Does wheat encourage body mass gain or spur growth in a way that rice, for instance, does not?

Given that wheat flour does not have an obvious relationship with other energy-dense foods Campbell ascribes to increased growth, it does seem wheat itself may be a factor. This is not a topic that can be feasibly analyzed in the span of this paper, but it may be a relevant one to explore in the future.

Another correlation Campbell notes is higher serum levels of urea, which he mentions is a biomarker of protein consumption. Although Campbell's implication may be that animal protein is the cause—thus lending credence to his animal protein-disease theory—an examination of the data reveals otherwise. A notable feature about grain consumption in China is its dichotomization: Rice dominates southern regions while strongly inversely correlating with wheat consumption ($r = -0.76$, $p<0.001$) and other cereal grains ($r = -0.68$, $p<0.001$)[77] in the north. Given that wheat flour, depending on whether whole-grain or refined, is nearly twice as high in protein compared to white rice on a per-calorie basis,[78] it logically follows that areas where wheat is a staple have higher protein intakes—and thus higher serum urea—than areas where rice is a staple.

Indeed, wheat flour correlates at 0.34 ($p<0.01$) with plant protein and at 0.35 ($p<0.01$)[79] with total protein intake, whereas rice intake correlates at -0.20 with plant protein and -0.23 with total protein intake.[80] Coupled with the fact that wheat flour inversely associates with all forms of animal food except for milk—which is generally only consumed in three counties—a logical interpretation is that a greater intake of plant protein results in these higher urea levels. A higher intake of plant protein, perhaps, may also contribute to the higher body weights exhibited in wheat-eating regions, particularly given Campbell's supposition about protein-restricted diets limiting weight gain and higher-protein diets fueling it.[81]

The next point Campbell raises may be significant: the relationship between wheat flour and certain serum lipid fractions. Although Campbell states that wheat flour intake is associated with "lower serum levels of monounsaturated fats," which he notes can increase risk of heart disease, wheat also correlates with lower total lipid docosahexaenoic acid (DHA) ($r = -0.34$, $p<0.05$)[82]—an essential fatty acid linked to cardiovascular health by abundant research,[83,84] including a China Study-based publication Campbell coauthored.[85]

Campbell implies that unfavorable lipid profiles may be responsible for the high rates of heart disease independent of wheat, thus creating a false correlation between wheat consumption and cardiovascular conditions. However, another possibility is that the wheat itself contributes to unfavorable blood lipid profiles, especially in the absence of more heart-protective foods such as fish—which is rarely consumed in wheat-eating regions ($r = -0.37$, $p<0.01$) but more frequently in rice-eating regions ($r = 0.32$, $p<0.05$) where heart disease is far less common ($r = -0.58$, $p<0.001$).[86] If wheat as a dietary staple is nutritionally inadequate, this would suggest that wheat-based diets may require careful planning or supplementation to reduce heart disease risk, especially in supplying certain fatty acids difficult to obtain from plant foods.

Continuing in this vein, Campbell writes:

> [The] correlation of wheat flour and heart disease is interesting but I am not aware of any prior and biologically plausible and convincing evidence to support an hypothesis that wheat causes these diseases.[87]

Because my initial mention of wheat's correlation with heart disease was intended to be speculative rather than assertive, I did not offer corroborating theories or evidence to substantiate a wheat-heart disease link. However, Joel Fuhrman—a plant-based diet advocate whom Campbell cites as one of his "physician colleagues"—has stated:

> Many scientific studies show a strong association between the consumption of white flour products, such as pasta and bread, with diabetes, obesity, and heart disease. … Whole grains are the least nutrient-dense food of the seed family, and they do not show the powerful protection against disease that is apparent in the scientific studies of fresh fruit, vegetables, beans, raw nuts, or seeds.[88]

Since the China Study data provides no indication as to whether the wheat flour consumed was whole-grain or refined, the following can only be guesswork. However, one noteworthy feature of refined grains such as white flour is their connection with elevated triglyceride levels, a condition widely associated with heart disease.[89,90]Another of Campbell's colleagues, John McDougall, asserts that refined grains cause blood triglycerides to increase, and states in his October 2006 newsletter:

> My experience has been that people who are having problems getting their … triglycerides under control need to stop using refined flour products and simple sugars.[91]

Indeed, wheat flour in the China Study is strongly associated with high triglyceride levels ($r = 0.51$, $p<0.001$).[92] Given that omega-3 fats have a mitigating effect on triglycerides,[93] it could be posited that rice-eating regions in China, with their frequent consumption of omega-3 rich

seafood, could be more protected from heart disease than wheat-eating regions—even though white rice alone may exhibit the same effects as other refined grains.

Of course, epidemiological data cannot prove causative relationships, only highlight correlations that may or may not be meaningful. And also importantly, the above web of univariate correlations is in no way conclusive, as many of these values may change when accounting for nonlinearity and confounding. When searching for overarching themes and material for future research, however, such univariate correlations are a useful place to start, as they are often the first indication of patterns that gain magnitude once fully excavated and analyzed. Given that connections between processed grains and heart disease are already corroborated by research as well as by biological plausibility, an authentic connection between wheat and heart disease is not unfeasible.

Selection of univariate correlations and confirmation bias

My largest concern with Campbell's conclusions, as stated in this paper and elsewhere, is that his approach to both China Study data and related research has been angled by the pursuit of a specific hypothesis—rather than an evenhanded evaluation of information and subsequent formation of a theory. In Campbell's explanation of his approach, he writes:

> I first inquired whether a collection of variables in the China survey (ranging from univariate correlations to more sophisticated analyses) could consistently and internally support each of these biologically plausible models and, second, I determined whether the findings for each of these models were consistent with the overarching hypothesis that a whole food, plant-based diet promotes health.[94]

Had Campbell approached the data from a different angle—or, better, from diverse and opposing perspectives in search of the most accurate one—he may have found multiple biologically plausible ways of incorporating China Study data trends with known physical mechanisms. By not testing alternative hypotheses alongside his own, Campbell runs the risk of investigative tunnel vision, and cannot truly determine whether his hypothesis is more valid than another.

In his second response to my critique, Campbell also writes:

> As I've said many times, not all the evidence in the China database supported this conclusion, although the large majority did.[95]

Considering the complexity and abundance of trends in the raw data, I would like to know what methods Campbell used to analyze and adjust the majority of the 8,000 statistically significant correlations in a way that yielded supportive results for his hypothesis. I propose that the China Study has generated enough material to bolster nearly any theory, regardless of actual validity—and for this reason, mandates an impartial and multi-perspective approach rather than a search for a predetermined outcome.

While the limitations of using univariate correlations are clear, Campbell has expressed willingness to employ them when they "consistently and internally support … biologically

plausible models."[96] Yet appears Campbell's chief criterion for deeming correlations valid is not just whether they're objectively plausible, but whether they support his hypothesis. In the instances they do, as explained earlier in this paper, he cites them without performing deeper analyses; in the instances they do not, he gives them no mention nor delineates a methodology for explaining their inconsistency with his theory. Rather than evaluating discrepancies within the data, he dismisses them—a choice ultimately leading to confirmation bias, potential misrepresentation of true trends, and a missed opportunity to rework his hypothesis to account for apparent anomalies.

Tuoli county and erroneous data

In discussing my observation of the apparent good health of a Chinese county whose diet, per China Study data, was high in animal protein, Campbell clarifies:

> [Tuoli county was] intentionally … excluded from virtually all our analyses on meat consumption because this county ranked very high when meat consumption was documented at survey time, but much lower when responding to the questionnaire on frequency of meat consumption. That is, these nomadic people migrate for part of the year to valleys, where they consume more vegetables and fruits.[97]

Although the information Campbell provides is useful, meat was not the dietary feature noted in my discussion of Tuoli: dairy was. Both the three-day diet survey and the frequency questionnaire reveal high intakes of dairy for Tuoli citizens, with the questionnaire indicating milk products are consumed an average of 330.3 days per year, and closer to 350 in one township.[98] In addition, despite Campbell's comment that the Tuoli migrate seasonally and consume more vegetables and fruit for part of the year, the China Study frequency questionnaire indicates Tuoli's vegetable intake is only twice per year and fruit intake is less than once per year on average.[99]

If Campbell believes both the three-day diet survey and frequency questionnaire were in error, I must question why Tuoli county was not excluded entirely from the data set—especially given its pronounced influence on virtually all associations involving meat, dairy, and animal protein, many of which Campbell cited as verification for his animal foods-disease hypothesis.

Efficacy of whole-food, plant-based diets versus whole-food diets with animal products

In his second response to my critique, Campbell states:

> [The] results of people using a diet of whole, plant-based foods, as shown by physician colleagues (previously mentioned, McDougall, Esselstyn, Ornish, Barnard, Fuhrman, et al) as well as by many of the readers of our book are nothing less than incredible.[100]

Campbell cites several examples of physician colleagues who have successfully employed plant-based diets—often in conjunction with other lifestyle modifications—to improve patients' health and reverse chronic conditions such as heart disease. Although these doctors unanimously advise limiting animal food consumption, their diet programs are characterized by more than just plant-based nutrition: They also drastically reduce or eliminate refined carbohydrates, processed sugar,

and hydrogenated oils—foods that tend to feature prominently in Western-style cuisines alongside animal-based products.

1. **John McDougall.** While McDougall's program embraces whole plant foods, he also advises against consuming refined flour, refined and sugar-coated cereals, soft drinks, vegetable oils, white rice, and other processed carbohydrates.[101]
2. **Caldwell Esselstyn, Jr.** The diet promoted by Esselstyn involves not only the elimination of animal products, but also the avoidance of vegetable oils and refined grains—including white rice, white flour, and products made from enriched flour such as pastas, breads, bagels, and baked goods.[102]
3. **Dean Ornish.** Along with eschewing meat, Ornish's program—as outlined in the book *Eat More, Weigh Less*—also involves reducing "sugar and simple sugar derivatives" such corn syrup, white flour, and white rice, avoiding margarines and vegetable oils, limiting alcohol, and avoiding commercial products with more than two grams of fat per serving, which is likely to disqualify most ready-made processed foods from dieters' menus.[103] Ornish also notes that his program involves more than just a plant-based diet: He emphasizes increased exercise[104] and other lifestyle changes to achieve better health.
4. **Neal Barnard.** In his book *Dr. Neal Barnard's Program for Reversing Diabetes*, Barnard advises his readers to "keep vegetable oils to a minimum" and "favor foods with a low glycemic index,"[105] which ultimately eliminates refined carbohydrates, most processed foods, high fructose corn syrup, and other common sweeteners. Barnard also recommends avoiding fried foods, including fried starches such as potato chips and French fries.[106]
5. **Joel Fuhrman.** Along with reducing or eliminating animal products, the diet Joel Fuhrman espouses shuns refined grains, refined oils, and refined sweets; Furhman lists these foods as less healthful than all forms of animal food in terms of nutrient density,[107] and notes that "eating a diet that contains a significant quantity of sugar and refined flour … leads to an earlier death."[108] Fuhrman also notes that "a low-fat diet can be worse than a higher-fat diet" if it centers on refined carbohydrates and contains trans fat,[109] stating specifically:

> A vegetarian whose diet is mainly refined grains, cold breakfast cereals, processed health food store products, vegetarian fast foods, white rice, and pasta will be worse off than a person who eats a little turkey, chicken, fish, or eggs but consumes large volumes of fruits, vegetables, and beans.[110]

Although plant-based diets eschewing white sugar, refined grain products, trans-fatty acids, high fructose corn syrup, and other highly processed ingredients are likely to improve health compared to a standard Western diet, research comparing unprocessed plant-food diets with unprocessed omnivorous diets is sparse. The success of whole foods, plant-based diets is not itself an indication that animal foods are deleterious; to determine this would require juxtaposing the results of whole-food vegan diets with equally "clean" omnivorous eating plans and demonstrating consistent superiority of the former.

Non-Westernized omnivorous diets

As current research indicates, other dietary paradigms may offer similar benefits to the plans promoted by McDougall, Esselstyn, Ornish, et al, without the reduction of animal products. Recent studies have shown "Paleolithic" style diets—which eschew grains, dairy, legumes, processed carbohydrates, and refined fats while embracing minimally processed meat, fish, vegetables, eggs, fruit, and nuts—may reduce fasting glucose levels, improve diastolic blood pressure, promote weight loss, improve glycemic control, lower triglycerides, raise HDL or "good" cholesterol, and generally reduce risk factors for cardiovascular disease, all while allowing for liberal consumption of non-dairy animal products.[111] In a study of diabetic patients, these effects were more pronounced in a Paleolithic diet group than in a group fed a standard low-fat diet including abundant plant-based foods such as whole-grain bread, other whole-grain cereal products, vegetables, and fruit, along with low-fat dairy.[112]

Similarly, a 1999 study by Frassetto et al discovered that non-obese subjects consuming a diet "comprising lean meat, fruits, vegetables and nuts" while excluding cereal grains, legumes, and dairy led to consistent and nearly immediate improvements in blood pressure, reduction in plasma insulin, lowered total cholesterol, reduced low-density lipoproteins, and decreased triglycerides.[113] In their publication, Frassetto et al conclude:

> Even short-term consumption of a paleolithic type diet improves BP [blood pressure] and glucose tolerance, decreases insulin secretion, increases insulin sensitivity and improves lipid profiles without weight loss in healthy sedentary humans.[114]

Additionally, a study conducted by Lindeberg et al showed that a grain-free diet with animal products improved glucose tolerance and reduced waist circumference more effectively than an unprocessed "Mediterranean" diet featuring whole grains, abundant plant foods, low-fat dairy, and minimal red meat.[115]

Additional clues herald from Australia. Research from O'Dea on the health and dietary patterns of Australian aborigines reveals that those eating a traditional cuisine—typically high in animal foods such as organ meats, fat deposits, and bone marrow along with tubers, vegetables, seeds, and fibrous fruits—exhibited "no evidence of the chronic diseases" common to Westerners,[116] including heart disease, diabetes, and obesity.[117]

Yet despite the apparent lack of adverse effects from their native high-meat diet, aborigines exhibit disproportionately high rates of diabetes and obesity after adopting a Western diet and lifestyle,[118] indicating genetics alone is not what protects them. If this surge in disease rates occurs with other groups shifting from a traditional omnivorous diet to a Western one, it suggests that factors other than animal food consumption may be responsible for the diseases plaguing developed countries.

In addition to the health of non-Westernized aborigines, virtually nonexistent rates of Western diseases have been reported of the Kitava, a traditional Melanesian society consuming no grains or processed carbohydrates but subsisting on a native diet of tubers, fish, coconut, and fruit.[119,120] A compilation of research collectively known as the Kitava Study revealed that "stroke and

ischaemic heart disease appear to be absent in this population,"[121] despite their consumption of animal products and lack of purportedly "heart-healthy" grains.

The Masai of East Africa—who consume copious amounts of meat and milk—also patently defy Campbell's hypothesis, particularly as it relates to animal foods and heart disease. After conducting a field survey of 400 Masai in the 1960s, researchers Mann et al observed that "Despite a long continued diet of exclusively meat and milk the men have low levels of serum cholesterol and no evidence for arteriosclerotic heart disease."[122]

In a guest editorial published in the American Journal of Clinical Nutrition, these researchers expand on their findings, describing the high animal-fat diet of the Masai and their paradoxically low serum cholesterol—a mean of 135.4 mg/dL on average, a level on par with the rural Chinese consuming plant-based cuisines:

> The average daily caloric intake was estimated to be about 3,000 kcal, with 66% of the calories derived from fat. The estimated average daily cholesterol intake was from 600 to 2,000 mg per person. The serum cholesterol levels of 254 Masai of various ages were determined; a low average value of 135.4 +/- 33.5 mg/100 ml … was observed.[123]

To provide further evidence of the Masai's noteworthy lack of heart disease, the authors note that "gross, histochemical, and chemical studies of the aortas and coronary arteries of 10 consecutive autopsies gave direct proof of the paucity of atherosclerosis in the Masai."[124]

In Alaska, researchers have observed rising rates of cardiovascular disease coinciding with a shift away from traditional dietary patterns and towards Western-style eating and lifestyle habits—a testimony to the health risks conferred by processed foods rather than animal foods in the aggregate. In 2009, cardiovascular disease risk factors were examined in relation to differing diet patterns among Alaska Eskimos, including a native diet featuring abundant animal products, wild foods, and no sugar or other refined carbohydrates:

> Participants following … the "traditional" diet consumed fish, native sea and land mammals and their fats and oils, wild greens, stew with mostly meat, stew with mostly rice or noodles, native birds, wild berries, and native berry agutuk.[125]

Compared to Eskimos following other eating patterns, including a Western-style diet and "healthy" store-bought diet, individuals consuming traditional foods had the most "desirable cardiovascular risk factor profile," including lower blood pressure and lower homocysteine.[126]

In a separate study, Alaskan Natives who replaced processed store-bought foods with traditional Eskimo foods—including meat from sea and land creatures—resulted in reduced diastolic blood pressure, lower total and low-density lipoprotein cholesterol, lower fasting glucose, and improved glucose tolerance.[127] Additional research shows that native diets emphasizing marine mammals, fish, game animals, berries, and wild greens results in lower triglycerides, increased high-density lipoprotein cholesterol, and better cardiovascular health—even while providing levels of animal fat exceeding those of most governmental recommendations.[128]

Conclusion

If both whole-food vegan diets and non-Westernized omnivorous diets yield similar health benefits, this is a strong indication that the results achieved by McDougall, Esselstyn, Ornish, et al are not due to the avoidance of animal products but to the elimination of other health-harming items. Western diets involve far more than increased consumption of animal products, and for some groups—such as Alaskan Natives—a switch from a traditional diet to a Westernized one entails *reduced* animal food consumption, with the caloric void replaced by refined carbohydrates, hydrogenated oils, grains, sugar, and convenience foods. The fact that a dietary shift towards Western fare inevitably leads to proliferation of "diseases of affluence"—regardless of changes in animal food consumption—suggests that another factor, or lattice of factors, instigates this decline in health.

The success of the Chinese on plant-based diets does not invalidate the experiences of other populations who evade disease while consuming animal products. Nor does individual success on a vegan program nullify the disease reversal seen by those adhering to specific omnivorous diets. Rather than studying the dissimilarities between healthy populations, perhaps we should examine their areas of convergence—the shared lack of refined carbohydrates, the absence of refined sweeteners and hydrogenated oils, the emphasis on whole, unprocessed foods close to their natural state, and the consumption of nutritionally dense fare rather than empty calories or ingredients concocted in a lab setting. Modern foods, and the diseases they herald, have usurped the dietary seats once occupied by more wholesome fare. It is this commonality—the thread bonding healthy populations—that may offer the most meaningful insight into human health.

A theory as purportedly universal as Campbell's should, by definition, unite the various health and disease patterns of global cultures without generating frequent anomalies. By naming animal products as the source of Western afflictions, Campbell has created a hypothesis valid only under hand-picked circumstances—one that cannot account for other epidemiological trends or even recent case-controlled studies. This is a symptom of a deficient theory, embodying only partial truths about broader diet-disease mechanisms.

I propose that the China Study remains a largely untapped resource for revealing potential diet-health patterns, expanded awareness of the source of disease, and inlets for future nutritional research—possibilities Campbell has not fully explored in his quest to validate a predetermined hypothesis. I invite Campbell, if he has the time and the interest, to present a more detailed account of his methodology, such as the unpublished book chapter he cited in his first response to my critique.[129] It is only through ongoing discussion and clarification that the field of nutrition can continue to evolve, progressing towards an increasingly unified understanding of health.

Lastly, I suggest that the "symphony" Campbell has heard thus far is only a partial opus. To cease listening now would be—at best—a missed opportunity for heightened health awareness, and at worst a perpetuation of the misinformation already degrading public and scientific understanding of diet and disease. I thank Dr. Campbell for both the harmonies and the dissonance his work has supplied to the field of nutrition, but implore him to continue listening. The final note has not yet sounded.

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