Can Nutrition Explain the Pattern of International Epidemiology of Hormone-dependent Cancers?¹

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Summary

International data on the distribution of hormone-dependent cancers suggest that they are cancers of affluence. Their occurrence parallels that of bowel cancer fairly closely in population and less closely with regard to individual patient risk. The most plausible hypothesis, although based on extremely incomplete knowledge, is that some components of the Western high-protein, high-fat diet acting in early life make individuals prone to develop these cancers.

Introduction

For the purposes of this presentation, the "endocrine-dependent" cancers to be considered will be cancers of the testis, ovary, prostate, breast, and uterine corpus. It may well be that cancers of other endocrine organs and target tissues have related epidemiologies, but current information is sparse. Of these others only cancer of the thyroid has been well studied. Its incidence certainly is diet dependent but the diet factor, iodine, represents a special problem outside the scope of the present discussion.

This presentation will review evidence that has suggested that endocrine-dependent cancers are related causally to nutrition. The presentation will concentrate on descriptive and population epidemiology, using vital statistics data. It will not attempt to discuss separately cancers of different epidemiologies occurring in the same organ, although for each of these cancer sites histological differences have been associated with epidemiological differences (1, 2, 10, 38, 61). Rather the approach of this discussion will be to consider endocrine-dependent cancers as much as possible as a single group, seeking the epidemiology that they have in common. No attempt will be made to bring out all the special characteristics of each type of cancer. Recent reviews that do this include Refs. 12, 23, 35, 37, 41, 54, 56, 59, 60, and 62. The justification for the group approach lies mostly in the tendency of these cancers to occur in the same populations, but they can also cluster in the same family (55).

The 1st question to be answered about the role of nutrition in the etiology of endocrine-dependent cancers is whether there is evidence that any environmental factor is involved. The alternative is to ascribe at least the major variations in risk to genetic inheritance, be it racial, familial, or a random convergence of events that lead perhaps to ovarian dysfunction. If we decide that important risk variations are related to environment, there is a 2nd and more difficult question. Are the environmental factors primarily carcinogens, such as those that cause most lung and skin cancers, or are they factors that in a more general way serve to raise or lower those thresholds for the action of ubiquitous carcinogens or for "spontaneous" mutation?

Endocrine-dependent Cancers as Cancers of Affluence

In Iowa, as elsewhere in the United States 3rd National Cancer Survey areas, endocrine-dependent cancers were extremely prominent (7). Breast cancer was the single most common cancer in women. Prostate cancer was the 2nd most common cancer in men and, of course, would have undisputed 1st place were it not for cigarette smoking. Overall, the endocrine-dependent cancers comprised 30% of all non-skin cancers in Iowa. They represented 40% of all cancers in women and 19% of all cancers in men. Not including cigarette smoking, we estimate that endocrine-dependent cancers would make up 27% of all cancers in men and 36% of all non-skin cancers in the population.

This importance is not a general situation throughout the world (Table 1). In India and Japan endocrine-dependent cancers together represent only about 3% of the total in men and 16% in women (15). In African blacks the figures were about 5 and 20%, and in Latin Americans 8 and 18%. Figures from Central Europe suggest an increased importance in women (25%) but not in men (5%). Earlier but more extensive figures are given by Segi (48).

In terms of actual incidence the Japanese present the lowest incidence of endocrine-dependent cancer (15). The male rate of about 7.5/100,000 is about 10% of the United States incidence. The female rate of about 20/100,000 is 16% of the United States rate. The rates for both men and women are about twice as high in India and Central Europe as in Japan. In black Africans, the female rat also is about twice that of Japan, but the male incidence is 4 times higher because of the high incidence of prostate cancer.

Within a country the association of endocrine-dependent cancers with social class will be best seen when the country shows a wide range of economically determined cultural patterns. This has been particularly well documented for breast cancer (e.g., Ref. 4). The other endocrine-dependent cancers are less well documented in this respect, and the

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endocrine-dependent cancers are more frequent than in
blacks. (15).

A marked sociometric gradient in a genetically homo-
genous culture is 1 type of evidence that environment, not
heritability, is responsible for high cancer incidence. Even for
breast cancer we may not have this situation. Hence to settle
Japan, but the approach to the United States rate is slower
dependent cancer incidence are environmental and that
endocrine-dependent cancers are more frequent than in
blacks. The next problem is whether the endocrine-depend-
cancers have an epidemiology sufficiently similar to the
epidemiology of bowel cancer that we should seek a
common cause for both or whether the discrepancies favor
essentially different causal situations.

Endocrine-dependent Cancers and Bowel Cancers

At the moment most of us believe that bowel cancer is
almost surely a cancer of affluent nutrition (31). To the
extent that bowel cancers and endocrine-dependent cancers
occur together, endocrine-dependent cancers also could be
nutritional cancers. Internationally, the incidence and rates
for the endocrine-dependent cancers follow fairly closely the
rates for bowel cancer. Table 2 presents some of these
correlations. Breast cancer is linked particularly closely
with bowel cancer. The associations between bowel cancer
and corpus, ovarian, testis, and prostatic cancers become
progressively weaker in that order, but still all are statisti-
cally highly significant.

Within individual countries the correlations are weaker,
but the extent to which intracountry distributions can be
relied on for epidemiological clues is a moot point in any
case. Certainly, the lack of correlation of rates for Japanese
prefectures (29) can be explained by the low rates for all of
the cancers in question. Burbank's (6) stepwise analysis of
United States mortality rates links breast cancer fairly
closely, but prostate and testis cancer not at all
to bowel cancer. The links between these
cancers are stronger for United States black men and
women. Overall, population risks suggest a parallel between
bowel and endocrine-dependent cancer risk factors but not
necessarily an equivalence.

Another way to look for evidence of a common cause of
cancer is to see whether individuals who are victims of one
of the diseases in question are at special risk for the others.
We have not yet reached much sophistication in the
interpretation of these results. When we are convinced of a
common cause but find no association (e.g., the relationship
of diet fat to coronary heart disease and bowel cancer), we
postulate alternate metabolic pathways. When we report
associations we usually have not studied the individual case
material thoroughly enough to rule out genetic rather than
environmental linkage.

The studies that I was involved with (Table 3) showed that
patients with endometrial and ovarian cancer had somewhat
later bowel cancer than expected. There was no increased
risk of breast or prostate cancer in bowel cancer patients,
nor was there appreciable increased bowel cancer in patients

The available data, however, leave many unanswered ques-
tions, and more information certainly could be utilized.

There are many ways in which affluence can affect cancer
incidence. One of the more unusual suggestions is that the
increase in artificial lighting may act through the pineal to
produce earlier menarche (33). This would in turn produce a
higher risk for at least breast (41) and possibly endometrial
cancer (34).

The general picture, then, is that endocrine-dependent
cancers are 1 of the 3 major environmental cancer problems
of this country along with cigarette-caused cancers and
bowel cancers. We know from many studies that cigarettes
are not epidemiologically linked with endocrine-dependent
cancers. The next problem is whether the endocrine-depend-
cancers have an epidemiology sufficiently similar to the
epidemiology of bowel cancer that we should seek a
common cause for both or whether the discrepancies favor
essentially different causal situations.

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<table>
<thead>
<tr>
<th>Population</th>
<th>Breast</th>
<th>Prostate</th>
<th>Testis</th>
<th>Breast</th>
<th>Corpus</th>
<th>Ovary</th>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Miyagi</td>
<td>0.3</td>
<td>3.2</td>
<td>0.4</td>
<td>11.0</td>
<td>1.3</td>
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<tr>
<td>Okayoma</td>
<td>0.9</td>
<td>4.3</td>
<td>1.2</td>
<td>12.4</td>
<td>2.2</td>
<td>2.8</td>
</tr>
<tr>
<td>India</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bombay</td>
<td>0.2</td>
<td>6.5</td>
<td>1.2</td>
<td>20.4</td>
<td>1.5</td>
<td>6.1</td>
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<tr>
<td>African blacks</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ibadan</td>
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<td>0.1</td>
<td>13.7</td>
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</tr>
<tr>
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<td>0.0</td>
<td>35.5</td>
<td>3.6</td>
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<td>19.2</td>
<td>0.0</td>
<td>13.6</td>
<td>4.1</td>
<td>7.7</td>
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<tr>
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<td>23.2</td>
<td>0.1</td>
<td>11.9</td>
<td>4.7</td>
<td>6.4</td>
</tr>
<tr>
<td>Latin America</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Cali</td>
<td>0.2</td>
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<td>27.3</td>
<td>6.8</td>
<td>9.2</td>
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<tr>
<td>Jamaica</td>
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<td>0.0</td>
<td>33.5</td>
<td>6.4</td>
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<tr>
<td>Puerto Rico</td>
<td>0.7</td>
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<td>0.8</td>
<td>20.9</td>
<td>4.6</td>
<td>5.1</td>
</tr>
<tr>
<td>California, Alameda</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>0.5</td>
<td>38.0</td>
<td>3.5</td>
<td>62.4</td>
<td>16.8</td>
<td>12.6</td>
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<tr>
<td>Black</td>
<td>1.0</td>
<td>65.3</td>
<td>0.0</td>
<td>38.6</td>
<td>18.1</td>
<td>10.4</td>
</tr>
</tbody>
</table>

* Rates per 100,000 population age adjusted to "European" standard (15).
Correlations between cancer incidence rates in populations

<table>
<thead>
<tr>
<th>Cancer A</th>
<th>Cancer B</th>
<th>Cancer B after Cancer A</th>
<th>Cancer A after Cancer B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bowel</td>
<td>Breast</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td>Endometrium</td>
<td>1.2</td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td>Ovary</td>
<td>1.3</td>
<td>2.2</td>
</tr>
<tr>
<td></td>
<td>Prostate</td>
<td>1.0</td>
<td>1.2</td>
</tr>
<tr>
<td>Breast</td>
<td>Endometrium</td>
<td>0.4*</td>
<td>2.0</td>
</tr>
<tr>
<td></td>
<td>Ovary</td>
<td>2.1*</td>
<td>4.4*</td>
</tr>
<tr>
<td>Endometrium</td>
<td>Ovary</td>
<td>2.0</td>
<td>4.0</td>
</tr>
</tbody>
</table>

* Statistically significant decrease presumably due to postmastectomy hysterectomies and/or castration.
* Statistically significant increase.

Associations among bowel and endocrine cancers in the same patient

<table>
<thead>
<tr>
<th>Relative risk of</th>
<th>Cancer A</th>
<th>Cancer B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Breast</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td>Endometrium</td>
<td>1.2</td>
</tr>
<tr>
<td></td>
<td>Ovary</td>
<td>1.3</td>
</tr>
<tr>
<td></td>
<td>Prostate</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td>Endometrium</td>
<td>0.4*</td>
</tr>
<tr>
<td></td>
<td>Ovary</td>
<td>2.1*</td>
</tr>
<tr>
<td></td>
<td>Ovary</td>
<td>2.0</td>
</tr>
</tbody>
</table>

* Statistically significant decrease presumably due to postmastectomy hysterectomies and/or castration.
* Statistically significant increase.

Table 2

Table 3

Nutrition and Epidemiology of Hormone-dependent Cancers

path to endocrine-dependent and bowel cancer must vary more than do the paths to lung and bladder cancer in a heavy cigarette smoker. By contrast, breast, endometrial, and ovarian cancers occur in the same patient often enough that a single basic causal mechanism may well be active. Besides genetic associations referred to above, at least for women, evidence is accumulating that ovarian cancers (46), as well as endometrial and breast cancers, are associated with abnormal hormonal function and, particularly, with persistent postmenopausal sex hormone activity (41, 60). To my knowledge, such hormonal abnormalities have not been described for bowel cancer patients, but I do not know that they ever have been looked for. However, there are suggestions along this line from population data and especially from a study of cancer mortality in nuns (19). Their high breast, ovarian, and endometrial cancer mortality was explained by infertility and it was speculated that bowel cancer mortality, which also was increased, also was influenced by marital status and infertility as it seems to be in the general population. It is not clear whether the excess bowel cancer mortality in nuns and other single women is truly excess incidence or to what extent it could be related to special nutritional problems.

Another link pointing to some underlying common thread is the close genetic linking of bowel and breast cancer in a number of families (39). Further study of the gene lesions behind such linkage might indicate ways in which the environment affects the target organs. Other families, however, certainly show endocrine-dependent cancer risk without especial bowel cancer risk and vice versa. Overall, there seem to be enough hints that bowel and endocrine-dependent cancers could have etiological factors in common so that the search for these factors should go on, but there is not enough evidence to guarantee success in the search.

Endocrine-dependent Cancers and Food Patterns

Given the noted close country-by-country associations between endocrine-dependent cancers and bowel cancer, it is not surprising that international studies show correlations at least between breast and prostate cancer and the same food types that have been associated with bowel cancer, i.e., meat, animal fat, and sugar. The other endocrine-dependent cancers have not been studied from this point of view. Drasar and Irving (17) showed high correlations between national levels of breast cancer and per capita consumption of total fat, animal protein, animal fat, eggs, and total protein intake (Table 4). Sugar and sweets were somewhat less correlated with breast cancer ($r \approx 0.5$), but this correlation still is stronger than that of sugar with bowel cancer ($r \approx 0.3$). Howell (30) reports that prostate cancer mortality correlated with meats, fats, milk, and sugars in particular, all of the associations being slightly weaker than for intestinal cancers. In another review (31) Howell relates national rates of cancer mortality from intestinal, breast, and prostate cancer to the sugar, meat, fat, and milk food group. Hems (26) has shown that the diet-breast cancer correlations improve when only "postmenopausal" breast cancer rates are utilized. However, Howell in unpublished...
changes, Japanese children have been steadily growing in height and weight. Cancer mortality has shown increases (in mortality) of 2- to 4-fold in the 3rd National Cancer Survey, compared to the previous rate. The increase in endocrine-dependent cancer rates is as low, compared to that in prostate cancer, testicular cancer, and, although not breast cancer, ovarian cancer is a possibility of very specific factor-target links should not be ruled out. The inadequately explored high incidence of endometrial cancer in obese diabetic women (22, 43, 57) is a case in point, since these women have not been shown to be affected by diet.

Table 4

<table>
<thead>
<tr>
<th>Drasar and Irving: 37 counties</th>
<th>Pearson’s r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast cancer and...</td>
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</tr>
<tr>
<td>Total fat</td>
<td>0.8*</td>
</tr>
<tr>
<td>Combined fat</td>
<td>0.8</td>
</tr>
<tr>
<td>Animal protein</td>
<td>0.75</td>
</tr>
<tr>
<td>Animal fat</td>
<td>0.75</td>
</tr>
<tr>
<td>Eggs</td>
<td>0.75</td>
</tr>
<tr>
<td>Total protein</td>
<td>0.6</td>
</tr>
<tr>
<td>Sugar</td>
<td>0.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Howell (1974): 41 counties</th>
<th>Rank correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prostate cancer and...</td>
<td></td>
</tr>
<tr>
<td>Cattle meat</td>
<td>0.75</td>
</tr>
<tr>
<td>Meats</td>
<td>0.74</td>
</tr>
<tr>
<td>Fats</td>
<td>0.73</td>
</tr>
<tr>
<td>Milk</td>
<td>0.70</td>
</tr>
<tr>
<td>Sugars</td>
<td>0.67</td>
</tr>
<tr>
<td>Potatoes</td>
<td>0.54</td>
</tr>
<tr>
<td>Eggs</td>
<td>0.50</td>
</tr>
</tbody>
</table>

* Values from from figure.

work found that bowel cancer mortality correlated more closely with premenopausal breast cancer rate than with the rates in older women.

One obvious next step is to see whether the parallels in cancer risk hold for special groups. Earlier we emphasized that in Argentina where a high beef, low-sugar diet was associated with a high intestinal cancer rate but not a high coronary heart disease rate (24). In La Plata breast cancer mortality was also comparable with Anglo American rates (45). The story on breast cancer was not completely clear, however, because Bristol’s high death rate arose in a country where the incidence rate is substantially below that of the United States (15). If La Plata had the same incidence-to-mortality ratio as Bristol, its incidence rate would be only about two-thirds of the United States rate. Ovarian cancer mortality was high in Bristol and San Francisco but not in La Plata; therefore a definite discordance exists. The other endocrine-dependent cancers do not show enough difference between Anglo American and Latin American death rates to follow the same lines of logic.

A more explicit and crucial test will come when firm incidence rates are available from the Seventh Day Adventists and Mormons, 2 groups on “prudent” diets. If endocrine-dependent cancer rates are as low, compared to nearby populations, as their bowel cancer rates, we will have substantial evidence that diet plays a major role in the causation of these cancers. Preliminary evidence that this may be true comes from a report on the breast cancer incidence in Utah (18). Only crude incidence is given but the rate of 56/100,000 is substantially below the rate of 79/100,000 reported for white women in the 3rd National Cancer Survey.

Other key evidence comes from Japan and should be analyzed more thoroughly. Presumably because of diet changes, Japanese children have been steadily growing taller (53). Concomitantly with this change, ovarian cancer, prostatic cancer, and testicular cancer although not breast cancer have shown increases (in mortality) of 2- to 4-fold in less than 20 years (36, 49). Further and more detailed studies of these phenomena might well prove the most direct way of linking diet and endocrine-dependent cancer risk more closely.

Hypotheses Regarding the Endocrine-dependent Cancer-Food Associations

If we accept the initial hypothesis that the geographic and socioeconomic distribution of endocrine-dependent cancers implies an environmental factor (as opposed to inborn genetic susceptibility), we must postulate either a specific environmental carcinogen such as a virus or a general enhancement of host susceptibility by affluence. As long as we lack the evidence for the specific carcinogen, susceptibility must be a viable alternative. We know that at least for breast cancer both the endocrine state of the patient and the past reproductive history are determinants of cancer risk. The most economical hypothesis is to postulate that the environmental factors act by modifying the endocrine status and balance (58). Evidence for this now is available from Hawaii. Dickinson et al. (14) have shown that the endocrine profile of young Japanese women in Hawaii has become more like that of Caucasian women, and quite altered from the profile of women in Japan. To me, one of the most important facts about the endocrine story is the great influence on breast cancer risk exercised by events in early reproductive life (41). It seems most logical to assume that the environmental factor must be most influential when it is cotemporal with these early reproductive events. The culture in which a woman lived during her early reproductive period would be the culture most influencing her breast cancer risk. The parallel would be with stomach cancer, where risks are set early in life rather than with bowel cancer, or lung cancer, where risk seems to relate in good part to exposures of middle age.

The next question is how does diet affect endocrine profile? Hopefully, the endocrinologists may help us if we can phrase our questions correctly. One lead is in the relationships reviewed by de Waard and Baanders-van Halemwijn (13) between somatotype and breast cancer, and it is pointed out that both somatotype and breast biology (11) are influenced by growth hormone while growth hormone activity levels are in turn influenced by the level of dietary protein (42). In speculation one could go as far as to suggest that mankind generally evolved under conditions of prudent (i.e., low-fat and protein) nutrition and that the present affluent diet from childhood onward may overstimulate the endocrine system, producing the same effect that one would obtain running a diesel engine on high-octane airplane fuel. The onset and frequency of menstruation certainly is dependent upon nutrition (21). In this view, overnutrition is a general evil favoring endocrine hyperfunction and hyperstimulation of target organs.

While it probably is worthwhile to see what implications of a very general overnutrition hypothesis can be tested, the possibility of very specific factor-target links should not be ruled out. The inadequately explored high incidence of endometrial cancer in obese diabetic women (22, 43, 57) is a case in point, since these women have not been shown to be...
at appreciable excess risk for other cancers. If this is an association in search of an explanation, the suggestion of Hill et al. (28) that gut flora might convert cholesterol and bile steroids into estrogen-like breast carcinogens is a hypothesis in search of more epidemiological and chemical evidence. Perhaps support for this concept is found in the reports [reviewed by Carroll et al. (8)] that adding fat to the diet specifically increases breast cancer incidence in rat and mouse systems.

The cancer most in need of more specific hypotheses is prostate cancer. One could argue that blacks represent a population with specific susceptibility to disease. Certainly, in South Africa there is a contrast between the great white-black differences in breast, endometrial, and testicular cancer and the nearly equal rate for prostatic (and ovarian) cancer (15). Prostate cancer is only 1 of a spectrum of prostatic diseases that are common in Bantus (44). The prostatic cancer incidence in Rhodesia and Jamaica is 50% higher than English and European rates, while of course United States blacks have the highest prostatic cancer rates in the world. However, it seems more productive to avoid the concept of racial predisposition as long as we can and to search for additional etiological factors such as viral or even bacterial infection that might combine with dietary factors to produce an ultrahigh incidence rate (32, 62). The suggestion that prostate cancer and heart disease are linked also should be investigated (27).

Other situations where the incidence of 1 or more cancers deviates from what would be expected may also point to unique epidemiological complexes. For example, Maoris appear to be at high risk for endocrine cancer without sharing the high incidence risk of other New Zealanders for bowel cancer (15). Hawaiian Chinese appear to be high in male bowel and female endocrine cancers but low in prostatic and testis cancer incidence (15). Chinese American mortality rates differ somewhat in being low for breast and high for testis cancer (20). Both United States and Israeli data suggest that Jews of European stock have high rates of ovarian and breast cancer but relatively low rates for endometrial and especially prostatic cancer (15, 50).

These and other special risk situations seem to rule out any simple relationship between a specific dietary factor and all endocrine-dependent cancers. Other epidemiological variables may have to be identified and peeled away before we are in a position to see clearly what the key nutritional factors might be and how they operate. It therefore may be premature to consider endocrine-dependent cancers as a group in epidemiological studies. Concern with the peculiarities and local risk factors, however, should not obscure the point that there seem to be underlying forces acting generally to raise the frequency of endocrine-dependent cancers in populations like our own and that these factors could easily be nutritional.

References


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