dissolved in the oil layer than when it was in water. The reverse was true with coconut oil. Although addition of glycerol had no effect on the degree of splitting, addition of glacial acetic acid to the coconut oil system decreased fat splitting to a considerable extent. Addition of coconut fatty acids to the coconut oil system had little effect, but soybean fatty acids added to the soybean oil system markedly increased the degree of splitting.

For the first time it has been demonstrated that, at 35 ± 0.1°C., splitting of a fat by the Twitchell process occurs in a stepwise way. Coconut oil in contact with 1 *N* sulfuric acid containing the sulfonic acid, corresponding to 1% by the weight of the oil, was about 90% split in 15 to 30 days, depending on the area of contact of the two layers. The diglyceride concentration reached a maximum during the early days of the reaction and then decreased somewhat. Monoglyceride concentration appeared to reach a maximum more slowly and then continued at that level as the concentrations of free fatty acids and glycerol steadily increased.

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The Relationship of Diet to Life Expectancy and Atherosclerosis

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THERE HAS BEEN considerable discussion recently concerning the role that dietary fats and fatty foods play in the development of atherosclerosis. Some conclusions have been derived from the comparison of data obtained for various classes of people in carefully selected localities. For example, the Bantus of Africa have been compared directly with the population of the United States, and certain groups in Italy, Spain, and England compared with purportedly similar groups in Minnesota. This has been summarized in a number of review articles (1, 2, 3).

Such comparisons have several obvious and serious limitations. Perhaps the most serious is that of attempting to correlate a specific effect with only one of a large number of potentially causative variables. Thus, whereas dietary fats differed substantially in the groups studied, so too did such factors as total caloric intake, types of all food-stuffs, climate, racial characteristics, energy output, standards of living, sanitation, medical care, economic standards, age of the population, and many others. Another fallacy is that an intermediate factor may be overlooked in such comparisons. For example, any factor that would tend to increase life span would obviously result in more people being susceptible to atherosclerosis. Yet a simple correlation would implicate that factor as a direct cause of the increased incidence of atherosclerosis. Further it can be very misleading to draw conclusions from data of this type unless there are represented many different populations taken on a random basis rather than a few groups selected on some arbitrary basis.

Data are readily available on food consumption

TABLE I
Compilation of Some Components of the Diet and Various Vital Phenomena

Country	Food Consumption Kg./cap./year							Caloric intake per cap. per day	Vital Phenomena				
	Fats and oils					Meat	Eggs		Deaths/ 100,000, arterio-sclerotic heart disease[a]	Deaths/ 1,000, males 40–44 (all causes)[b]	Life expect-ancy, males age 40[c]	Surviv-ors age 40, per 100,000 born (males)[d]	Percent-age of males age 40 and over[e]
	Total	Meat	Veg.	Butter	Marine								
Norway	25	2.5	7.1	5.5	8.6	31	7	3140	135.3	2.8	35.2	90,196	37.0
Netherlands	23	4.5	14.7	3.6	1.8	29	5	2960	147.3	2.7	34.9	91,661	31.3
United Kingdom	22	1.6	10.2	7.4	2.3	50	11	3100	298.0	3.1	31.4	92,430	40.5
Ireland	21	1.3	0.7	18.7	0	56	14	3340	274.8	4.7	30.6	82,462	35.8
Sweden	21	2.5	6.6	14.0	0.7	48	11	3120	229.5	2.8	33.8	91,697	40.0
United States	19	5.6	10.5	4.8	0	74	21	3130	282.3	5.0	31.4	90,207	35.0
Denmark	19	4.7	9.2	4.5	2.6	65	9	3160	202.2	2.7	33.8	90,088	35.2
Belgium	19	5.0	7.2	10.8	0.7	45	13	2770	111.8	3.9	30.6	84,882	41.4
Canada	18	....	....	....	....	69	17	3060	222.9	3.9	32.4	89,649	36.0
Switzerland	16	3.9	8.8	5.8	0	40	9	3150	238.1	3.6	30.4	86,063	38.4
New Zealand	15	....	....	....	....	96	13	3250	252.2	2.7	32.6	92,250	34.1
West Germany	15	5.9	5.3	5.5	0.6	23	4	2640	163.3	3.5	32.3	87,102	40.0
Austria	15	7.9	6.1	3.4	0.1	29	4	2620	195.2	3.7	30.7	85,111	42.2
Israel	15	....	....	....	....	15	12	2630	120.9	2.7	33.1	90,900	27.6
Australia	14	....	....	....	....	108	12	3160	240.9	3.7	31.2	90,823	35.0
Finland	12	....	....	....	....	27	4	3000	191.7	5.5	28.0	86,799	30.0
France	12	4.2	5.9	6.0	0.2	54	10	2770	37.6	4.8	30.4	87,940	41.0
Portugal	10	3.3	9.5	0.4	0	19	2	2110	67.1	5.3	30.3	75,466	28.0
Italy	9	3.4	6.0	1.2	0	18	5	2340	176.3	4.4	32.3	....	....
Cyprus	8	....	....	....	....	17	2	2740	39.9	2.9	32.9	85,159	26.6
Brazil	6	....	....	....	....	39	3	2340	....	14.1	25.4	69,782	18.0
Chile	6	....	....	....	....	38	2	2360	36.2	9.2	27.2	51,210	22.6
Mexico	6	....	....	....	....	23	2	2050	5.5	11.5	24.8	50,376	21.4
Egypt	3	....	....	....	....	10	1	2290	28.2	14.2	26.1	47,243	22.5
Japan	1	....	....	....	....	2	1	2100	44.7	5.1	30.6	84,834	24.8

[a] Number of deaths from arteriosclerosis per 100,000 population.
[b] Deaths of males in age group 40–44 per 1,000 males in that age group.
[c] Average number of years of life remaining for males reaching age 40.
[d] Number of survivors per 100,000 males born 40 years previously.
[e] Percentage of males of all ages that are 40 and more years old.

and certain vital statistics for the various nations of the world (4, 5, 6). Some of these data for 25 countries, having both similarities and dissimilarities, have been assembled in Table I. They are listed in the order of decreasing consumption of food fats. Coefficients of correlation have been calculated in order to express mathematically the relationships between the several factors. High coefficients of correlation between any two factors shown are not to be interpreted as inferring causal relationship. The calculations are simply to be used as a convenience in discussion. These data are shown in Table II.

TABLE II
Coefficients of Correlation of Dietary Components with Vital Phenomena

	Deaths/ 100,000, arteriosclerotic heart disease	Life expectancy, males age 40	Deaths/ 1,000, males age 40–44	Survivors age 40, per 100,000 born
Caloric intake Cal./cap./day	+0.82	+0.54	−0.59	+0.69
Fat consumption Kg./cap./yr.	+0.70	+0.70	−0.67	+0.69
Meat consumption Kg./cap./yr.	+0.63	+0.20	−0.18	+0.45
Egg consumption Kg./cap./yr.	+0.70	+0.29	−0.54	+0.64
Survivors age 40 per 100,000 born	+0.64			

It is immediately evident that consumption of meat, eggs, and fats show impressive coefficients of correlation with the incidence of deaths because of arteriosclerotic heart disease (in the available data atherosclerosis is not reported separately). But the highest coefficient of correlation is shown with total caloric intake. Here, then, could be a case where the intermediate factor, total caloric intake has been ignored and responsibility placed solely upon one of the many components of total calories.

In fact, it is apparent that food fat consumption shows significant coefficients of correlation with those measurements of general well-being, *i.e.,* proportion of the population that attain age forty, and life expectancy at age forty. An equally significant negative coefficient of correlation exists between consumption of food fats and deaths of males in the 40–44 age range. One can now reasonably ask whether the apparent relationship between food fats and deaths because of arteriosclerotic heart disease is not the result of the beneficial effect of food fats upon longevity. Recently it has been demonstrated with college women that when total calories are maintained at equivalent levels, better over-all health was attained when food fats represented 35.5% of the calories than when lower levels of fat were taken (7).

It is clear from the data in Table I that one could prove several divergent hypotheses by simply selecting the proper countries to study. For example, a comparison of Norway and Australia would indicate that high fat consumption is protective against death because of arteriosclerotic heart disease. On the other hand, the comparison of the United Kingdom and France would indicate just the opposite. And, finally, comparison of the Netherlands and the United Kingdom would lead to the conclusion that some other factor(s) must be responsible. One could indulge in this kind of speculation indefinitely.

Although the data in Table I led to rather impressive coefficients of correlation, there are a number of examples that may be selected to support the previously stated conclusion that the correlations do not establish causal relationships beyond reasonable doubt. For example, the two consumers of the largest amounts of food fats (Norway and the Netherlands) compare well in vital statistics with those countries that consume only half as much fat per capita. The same is true for Belgium. Sweden and Norway, which one would expect to be quite similar in most respects, also represent an anomaly; Sweden with a lower fat consumption reports a much higher incidence of deaths because of arteriosclerosis.

Such observations raise the question of the reliability of some of the data. It seems reasonable that the data on life expectancy and survival should be fairly accurate; however information on deaths because of arteriosclerotic heart disease could be subject to sizable error for many reasons. Recently data were published on the incidence of deaths from atherosclerosis in the various states of the U.S.A. (8). It was observed that the state-by-state differences were surprisingly great, with some having only half the death rate as others. A number of factors were suggested that might be influential in causing the observed differences. One that was not mentioned is that, in general, in those states with a low apparent incidence of atherosclerosis, each physician had many more potential patients and more square miles of responsibility. How much does the consequent reduction in available medical attention affect the reported incidence of atherosclerosis?

It must be concluded that population statistics such as the above, while yielding interesting information, must be interpreted with caution and great discretion. Over-interpretation of limited data, especially that of uncertain validity, could do irreparable harm.

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