Ischaemic heart disease and consumption of hydrogenated marine oils in England and Wales

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Abstract

Study objective—The aim was to examine the hypothesis that hydrogenated fats, particularly those obtained from marine oils, may present a health hazard.

Design—Storage fat specimens obtained at necropsy were collected from several areas in England and Wales during 1975–1978. Cases ($n = 136$ samples) consisted of males dying of ischaemic heart disease, male deaths from unrelated causes acting as controls ($n = 95$ samples). The fatty acid compositions of the specimens were determined, and analysis included those acids—16:1 *trans* and “higher” C-20 plus C-22 (H)—highly characteristic of partially hydrogenated marine oils.

Measurements and main results—The case samples, which had been shown to be the richer in 16:1 *trans* ($p < 0.005$), were now found to have a significantly higher value of the ratio 16:1 *trans* to H ($p < 0.002$), arising from consumption of differing hydrogenated marine oil types.

Conclusions—It is concluded that the cases had consumed a greater amount ($p < 0.001$) of hydrogenated marine oils of a certain type, ie, that manufactured from certain highly unsaturated raw oils. The process of partial hydrogenation results in conversion to a product containing large amounts of polyunsaturated acids (PUFA) which are no longer in the natural all-*cis* methylene interrupted configuration. Such isomeric PUFA may obstruct or compete with utilisation of natural PUFA. It is further concluded that the case excess did not rise from medical advice favouring margarine or from any difference in social class status, but rather from fortuitous selection of margarine brand.

Following an observation (1975) that mortality from arteriosclerotic disease in the UK is highest in those areas and social classes which consume highest amounts of hydrogenated fat and lowest amounts of ruminant fat, I suggested¹ that those fatty acids arising from commercial hydrogenation of liquid oils (which are structurally different from those present in the natural oil) may have adverse effects. Using *trans* acids as a “marker” of such chemically modified acids, a programme was set up in which we examined^{2–4} the fatty acid compositions of body storage fat taken from male persons who had died of ischaemic heart disease, and compared them with samples from male persons who had died of

unrelated causes. The specimens were supplied through the courtesy of Dr P C Elwood (Director) and the MRC Epidemiology Unit (South Wales), and were collected from a number of areas in England and Wales differing widely in mortality.

Whereas there are difficulties³ in using 18:1 *trans* as a marker (about one half of which is contributed by ruminant fat), it was found⁴ that 16:1 *trans*, which is highly characteristic of hydrogenated marine oils, was present in significantly ($p < 0.005$) higher concentration in the storage fat of cases than in that of the controls. On the other hand, the control samples were richer in certain branched and odd number acids characteristic of ruminant animal fat (butter, beef and mutton fat). It was concluded that the cases had consumed a higher proportion of hydrogenated marine oils and a lower proportion of ruminant animal fat than had the controls.

At the time however one feature of the results was puzzling, namely that the tissue concentrations of “higher” (C-20 plus C-22) acids—which also are characteristic of hydrogenated marine oils—were virtually identical for the two populations.

More recently⁵ the work was extended to the Glasgow area with similar results, but again there was little difference in amounts C-20 plus C-22 acids (collectively labelled H) between cases and controls. By this time however it had become apparent that hydrogenated marine oils are variable in composition, particularly in regard to relative amounts of 16:1 *trans* and H. Thus an analysis of 10 popular brands of margarine⁵ had shown contents of 16:1 *trans* which were not proportional to H content. Indeed, the values of the ratio 16:1 *trans*/H (= R) varied from 0.10 to 0.39, so that at a given intake of H, consumption of 16:1 *trans* may vary by a factor about four times.

In the Glasgow study of adipose tissue fat we found that the cases had not only the higher content of 16:1 *trans*, but also that the case mean value of R (0.199) was significantly higher ($p < 0.005$) than the mean control value (0.160). It was concluded that the cases had consumed not only a greater total amount of hydrogenated marine oils than the controls, but in particular that the excess could be attributed essentially to a “higher risk” type of oil, ie, that having a high R value. In order to ascertain whether this conclusion was peculiar to the Glasgow area or of wider significance, the present paper re-examines levels of 16:1 *trans* and “higher” acids obtained in the earlier England and Wales project. It is also hoped thereby that some insight may emerge as to which component or components are responsible for the risk we attach to hydrogenated marine oils.

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Methods

COMPARABILITY OF METHODS OF DETERMINATION OF HIGHER ACIDS

In order to relate the two studies properly it is first necessary to compare the methods employed in determination of the higher acids. In the Glasgow project, the percentages of higher acids (H) in the samples were determined via full catalytic reduction to saturated acids so that H included 20:4 and any polyenoic C-22 acids originally present. For both cases and controls the ratio C-22/H was sensibly constant over the whole range of H values. For the England and Wales study on the other hand, the higher acids were determined and defined as the sum of the percentages of 20:0, 20:1, 20:2, 20:3, 22:0, and 22:1 which we label h; in other words h did not include 20:4 or polyenoic C-22 acids. Amounts of 20:4 were not related to h but were sensibly constant at 0.25 for both cases and controls.²

Writing θ as the (unmeasured) polyenoic C-22 acids in a given sample and x as the percent 22:0 as previously determined, then the new percent 22:0 in the sample becomes $x(100-\theta)/100$. Likewise the previous 22:1 (y) becomes $y(100-\theta)/100$, and h becomes $h(100-\theta)/100$. From analogy with the Glasgow results we now write

(1/100) (100-θ) (x+y) + θ = ψ
(1/100) (100-θ) (h + 0.25) + θ

in which ψ is a constant. Rearrangement gives

x + y = ψ (h + 0.25) + θ (ψ - 1) { 100 / (100 - θ) }

The plots of $(x+y)$ against $(h+0.25)$ as set variable were manifestly linear ($p \leq 0.001$) for both cases and controls throughout the range, and they did not pass through the origin. In other words, whereas θ is not zero, it is sensibly independent of total higher acids (an alternative assumption that θ is a constant fraction of total higher acids would require the plots to pass through the origin). Furthermore, analysis of covariance of $(x+y)$ on $(h+0.25)$, cases versus controls, showed that the two regression lines did not differ significantly either in slope or in intercept. Combination of the two therefore gave $\psi = 0.26$ (cf Glasgow 0.27) and $\theta = 0.53$. It is

concluded then that—within the limits of experimental accuracy—H differs from h by a sensibly constant small amount, the same for cases and controls ie, that $H = h + 0.8$.

THE RATIO 16:1 TRANS TO HIGHER ACIDS IN UK HYDROGENATED MARINE OILS

Of the various marine oils used in the manufacture of margarines and shortenings, herring^{6 7} is the least unsaturated and characteristically contains much 20:1 (14%) and 22:1 (21%). On hardening by hydrogenation to margarine specification^{6 7} it is converted to a product containing around 2% 16:1 trans and 48.5% H, giving $R \approx 0.04$. Other sources—anchovy, pilchard, sardine, menhaden—rather closely resemble one another and are sharply differentiated from herring. They all contain high amounts of unsaturated C-16 acids and although having a lower total C-20 plus C-22 content, they characteristically contain much polyunsaturated 20:5 and 22:6. They therefore require hydrogenation in greater degree than herring, and the product (taking the well studied menhaden as typical^{6 8}) contains around 9% 16:1 trans and 26% H giving $R \approx 0.35$. Although some variability in these “limiting” values of R may be expected—particularly in the lower value, which is based on small amount 16:1 trans—the published values agree well with those observed in our 10 margarines, viz 0.10 and 0.39.⁵

Our extensive examination of body storage fat likewise shows a range in R values. Of the 285 samples examined only two had values <0.06 (which may well have been due to experimental error in determining very small amounts 16:1 trans) whereas the next nine values in increasing magnitude fell between 0.06 and 0.08; the four highest values lay between 0.33 and 0.37. We may conclude therefore that the hydrogenated marine oils of UK margarines consist essentially of the two basic types in varying ratio, and that this fact is well reflected in body fat composition.

On balance it seems reasonable to set the R limits in UK experience at, say, 0.08 and 0.35; respective percentage amounts of 16:1 trans and H will then be 3.9 and 48.5 for herring and 9.1 and 26.0 for menhaden type.

One conclusion is immediately obvious, namely that of two samples of hydrogenated marine oils containing the same amount H, the amount of 16:1 trans will be the higher in that sample richer in menhaden relative to herring (ie, in that sample

Table 1 Parameter mean values and respective SD for cases (C) and controls (K) in nine areas

Hospital areas	Number of samples		% HMO to ± 0.1		log10 { 16:1 trans × 10 / H } and respective SD				% Hydrogenated menhaden type			
	K	C	K	C	K	SD	C	SD	K	SD	C	SD
Luton and Dunstable	15	22	10.2	10.9	0.00876	0.14496	0.08292	0.13388	1.5760	1.7520	3.0689	2.6046
Winchester, Poole, Folkstone	6	9	9.8	12.9	0.11527	0.14685	0.14926	0.11624	2.9562	2.0469	4.4635	1.9691
Carmarthen, Llanelli, Aberystwyth	9	16	9.9	10.5	0.09425	0.24365	0.14068	0.18623	3.2038	3.7214	3.8898	2.8820
Total	30	47										
Huddersfield, Halifax	7	14	11.0	11.4	0.10472	0.12781	0.21748	0.11212	3.3711	2.3313	5.2402	2.0432
Bradford	16	15	11.5	12.8	0.09906	0.12741	0.20270	0.10626	3.3435	2.5829	5.8992	3.5266
Manchester	18	19	13.1	12.5	0.14290	0.14201	0.21037	0.10067	4.3098	2.5193	5.5304	1.9795
Total	41	48										
Cardiff, Bridgend, East Glamorgan	12	17	12.0	12.2	0.22057	0.16418	0.25373	0.11132	5.7470	3.4102	6.4222	2.6429
Liverpool (Walton)	5	12	14.2	12.5	0.22561	0.03134	0.23647	0.10453	6.8084	1.8227	6.3311	2.4681
Chatham	7	12	12.3	12.6	0.28442	0.10053	0.28369	0.14252	7.0463	2.5526	7.4296	3.3260
Total	24	41										
Overall means and SD	95	136	11.6	12.0	0.12976	0.16274	0.19159*	0.13817	3.9706	3.0041	5.2291†	2.9052

*Student's t test on difference in mean values (0.06183) gives $t = 3.11$, $p \sim 0.002$
†Student's t test on difference in mean values (1.2585) gives $t = 3.19$, $p < 0.002$

having the higher value of R)—whether in margarine as such or in body storage fat where it is “diluted” by other fats. But we have already established⁴ that the concentration of 16:1 trans is significantly higher ($p<0.005$) in the case samples, whereas there is virtually no case versus control difference in H. The case samples are therefore the richer in menhaden type, but whether the difference in this respect is statistically significant remains to be examined.

Results

CASE VERSUS CONTROL STATISTICAL ANALYSIS
Table I lists case and control mean values arranged in increasing parameter order for nine areas; the previously reported separate data for “Manchester Conurbations 1 and 2” have been combined. The results fall into three broad groups, each accounting for approximately equal numbers of samples—with lowest parameter values in the first group. It can be seen that the ratio cases to controls for the first group (1.6) is very similar to the ratio (1.7) for the highest parameter group at the bottom of the table; under such circumstances there is little bias in selection and the overall mean case minus mean control difference can then be taken as representative of the nine areas at large.

The mean case value of R is 0.1639 compared with the control mean 0.1450, but the distribution of R is somewhat skew both for cases and controls. The skewness is however well removed by log transformation, and the transformed means are virtually identical with the median values. To avoid negative numbers, the tabulated figures are of $\log_{10}(10R)$. The mean value of $\log_{10}(10R)$ for the 95 controls is 0.12976 and for the 136 cases 0.19159, and a Student’s *t* test gives $t=3.11$, $p\approx0.002$. As previously for the Glasgow area, therefore, the body fat hydrogenated marine oil of the cases was significantly the richer in the menhaden type.

Two way analysis of variance yields, as anticipated, a very similar result for the case versus control difference, the Fisher variance ratio (*F*) being 10.45, $t=3.23$. It also showed however a highly significant area to area variation, $F=7.03$ (df 8, 213; $p<0.001$)—a fact which is considered further below.

Table II Analysis of variance, percentage hydrogenated menhaden in adipose tissue

Source of variation	df	Sum of squares	Mean square	F
Areas	8	2.3203	0.2900	8.33
Cases versus controls	1	.4089	0.4089	11.75 ($t=3.43$, $p<.001$)
Interaction	8	.1675	0.02094	0.60
Individuals	213	7.4150	0.03481	
Total	230	10.3117		

Table III Social class composition; mean percentages hydrogenated menhaden and linoleic acid for cases (C) and controls (K) within classes I, II, III NM (A) and III M, IV, V (B).

		No of samples			Mean % menhaden in			Mean % 18:2 in	
		All classes	In A	Fraction in A	A	B	All classes	A	B
Section 1	C	59	17	0.29	4.11	3.89	3.96	8.53	8.40
	K	30	9	0.30	3.51	1.89	2.37	8.52	8.15
Section 2	C	67	16	0.24	6.49	6.03	6.14	7.57	7.86
	K	51	17	0.33	4.97	4.75	4.83	7.13	7.98

It should be realised that the case versus control difference in R in no way depends on the assumption that commercial marine oils consist of mixtures of two specified types, viz herring and menhaden, but only on the demonstrable fact that UK margarines contain hydrogenated marine oils of differing R value. From previous and other considerations in an earlier paper (see reference 5, p155), however, it is reasonable (if not obvious) to regard hydrogenated marine oils as mixtures of the two dissimilar types in varying proportion so that the higher case value of R arises from excess of the menhaden type. Using the above numerical values of 16:1 trans and H for the two materials, it is easy to show that the fraction (β) of menhaden in a given mixture with herring is given by

$$\beta = 2.156(R - 0.0804)/(0.2311 + R) \tag{1}$$

in which R is the ratio 16:1 trans/H for that mixture.

Remembering that the previous values of 16:1 trans must be “corrected” for inclusion of C-22 polyunsaturated acids (θ) in the new total acid content, ie, by multiplying by the factor 0.995, the percentage total hydrogenated marine oils (HMO) in adipose tissue becomes

$$\% \text{ HMO} = 6.584(\% 16:1 \text{ trans} + 0.232 \text{ H}) \tag{2}$$

and the percentage menhaden in the tissue becomes

$$\% \text{ menhaden} = 14.19(\% 16:1 \text{ trans} - 0.0808 \text{ H}) \tag{3}$$

In these equations the percentage amounts of 16:1 trans and “higher acids” are now those previously reported—except that $H=h+0.8$. The form of equations (2) and (3) is such that case *v* control differences are not affected by the precise value of θ , so long as it has been shown to be the same for the two populations.

Mean area values of percentage menhaden intake corresponding to tissue values of 16:1 trans and H can now be calculated by means of equation (3). These are shown in the final columns of table I. The control mean value is 3.971 and the case mean value 5.229, the difference being highly significant. Analysis of variance gives a very similar result, but in addition furnishes information regarding area variability. The calculations involved are set out in table II [for arithmetic convenience the figures are for the function 16:1 trans -0.0808 H, ie, for the percentages divided by 14.19; see equation (3)]. For the case versus control difference, *F* is 11.75 ($t=3.43$, $p<0.001$), and for area variation 8.33 (df 8, 213; $p<0.001$).

Use of equation (2) yields mean percentage values of total hydrogenated marine oils shown in the left hand columns of table I. The overall mean case value is higher than the mean control value, but the difference is not significant. In other words, a significant difference arises only through “selection” of oil type.

The mean control value for the fractional content of menhaden type in total hydrogenated marine oil (3.97/11.6) is 0.34. This agrees well with the value (0.35) obtained from information

released by the Ministry of Agriculture, Fisheries and Food for the 1973–80 period.⁹

The above calculations are little affected by the choice of limiting R values. Thus using the published value^{6,7} of 0.04 as the lower limit in place of 0.08 leads to the test function (16:1 *trans* –0.050 H) instead of (16:1 *trans* –0.081 H) of equation (3). Analysis of variance then gives a case versus control difference significant at $t = 3.23$ in place of $t = 3.43$.

SOCIAL CLASS COMPOSITION OF THE DECEDENTS

We were able to obtain the social class status of 126 cases and 81 controls, ie, for 90% of the total samples (the previous figures³ were incomplete). Since those from classes I, II, and III NM accounted for only 28% of the total, the numbers of samples within this category for some of the nine areas were small (two or less)—particularly for controls. The overall effect of social class on the calculations is best judged by dividing the results into two sections, with the top four areas of table I (lowest parameter values) forming the first section and the remaining five areas (highest parameter values) forming the second section. Mean percent values of hydrogenated menhaden in the two sections are shown in table III for cases and controls within classes I, II, III NM (category A), and IV, V, III M (category B).

It can be seen that in both sections—surprisingly perhaps—the mean values for category A exceed those for category B within both cases and controls. The overall mean value for the 33 case samples within A is 5.26 compared with 5.06 for the 93 case samples within B, but the difference is far from significant. There was likewise no significant difference between category mean values for the 81 control samples.

If the social class composition of the cases and controls was identical, the case population would of course have exactly matched that of the control population. This is however not quite the case (column 4) and in both sections the controls have somewhat higher status thereby resulting in higher control percentages than would otherwise have appertained.

The overall (weighted) difference in percent menhaden (A minus B) is 0.041 and adjustment of control mean values to the class status of the cases may be made. The mean control values for sections 1 and 2 (2.37 and 4.83) became 2.36 and 4.77 respectively—an adjustment so small that the effect on the statistical calculations would be negligible. The sense of the correction however is such as to increase the observed case excess.

Discussion

In view of the primary observation that the case samples are significantly higher in 16:1 *trans* acids, it must remain a possibility that such acids present a risk to health purely by virtue of their *trans* configuration. But we have now established the further fact that over nine widely differing areas in England and Wales, as well as previously for the Glasgow area, the cases have a significantly higher intake of hydrogenated menhaden type. Consequently it is logical to enquire which fatty acids are present in this type in higher proportion than in hydrogenated herring. One obvious

difference (there are others such as, for example, positional, *cis*-isomers of the natural ω 7 palmitoleic acid) is that the hardened menhaden is particularly rich in C-20 and C-22 di- and tri-enoic acids which do not have the methylene interrupted all-*cis* structure characteristic of natural polyunsaturated acids (PUFA). Amounts of such isomeric PUFA in the hardened materials are substantial (around 18% in menhaden and 9% in herring) and replace virtually all the biologically active penta- and hexaenoic acids originally present in the raw fish oils.^{6–8} In view of the currently popular opinion that C-20 and C-22 PUFA of raw fish oils confer some measure of protection against heart disease, such destruction in itself should surely cause some concern. But further, the high consumption of hydrogenated marine oils in the UK leads to an intake of isomeric PUFA amounting to around 2 g daily—an amount far in excess of amounts of 20:5 and 22:6 consumed in an average fatty diet. (Interestingly, there is little doubt that present day consumption of fatty fish is much reduced from former times.) Isomeric PUFA have no essential fatty acid activity and may obstruct or compete with utilisation of natural PUFA.

I have been unable to examine my hypothesis with regard to hydrogenated vegetable oils (the 18:1 *trans* marker acid characteristic of this type is obscured by its presence in both hydrogenated marine oils and ruminant fat), but it may be prudent to remember that such material contains, depending on the extent and nature of the hardening process, appreciable amounts of isomeric C-18 polyenoic acids which may interfere with utilisation of both linoleic and linolenic acids.

I have previously observed¹ that the pattern of consumption of hydrogenated fat is one in which a positive gradient from smaller towns through larger towns to urban conurbations is superimposed on a geographical south to north increase. Such pattern is reflected in a general way in the figures for total hydrogenated marine oils and hydrogenated menhaden shown in table I. Although I do not wish to overstate the matter (it is well known that ischaemic heart disease mortality may vary markedly from suburb to suburb within a given city), the lowest values are shown by the more affluent small to medium sized towns in south and east England—in keeping with the low mortality experience of the region as a whole (standardised mortality ratio 89; male, ages 25–64 years; 1968–71¹). The highest values are shown by the densely populated areas in the north and northwest (standardised mortality ratio 116; north and northwest standard regions). Intermediate values would appear to appertain for the Yorkshire areas (standardised mortality ratio 109; Yorkshire and Humberside). The position of Chatham is perhaps surprising.

The figures for Wales are of particular interest. The principality as a whole is well known to have high ischaemic heart disease mortality experience, a fact which was not in keeping with my thesis¹ that high mortality is associated with high consumption of hydrogenated fat (HF) and low consumption of ruminant-animal fat. The National Food Survey for household food consumption showed Wales to have in fact a low

intake of HF; the data applied however to the principality as a whole, and separate information for the Registrar General's "Wales, South East" and "Wales, remainder" was not available.

According to the present paper however, intake of total hydrogenated marine oils and menhaden in the more rural parts of Wales is low and comparable with amounts for south and east England; amounts for the three hospital areas in Glamorgan on the other hand are among the highest found. This sharp difference is reflected in the differing standardised mortality ratios of 108 and 127 respectively. It may be of relevance to add that for females in the principality the standardised mortality ratios are 91 and 143¹—the latter figure being the highest in England and Wales. The often expressed statement that Wales has high ischaemic heart disease mortality is therefore less than the whole truth and may well hide a significant gradient in fatty diet from the more traditional and butter consuming rural parts to the industrialised south east.

It is perhaps surprising that as total hydrogenated marine oils increase, so also does R, and thus the richer the oil becomes in menhaden content; the area to area differences in menhaden intake thereby become highly significant. This may perhaps reflect no more than regional brand preferences as such, but it seems more likely that cheaper brands on average have higher menhaden content. Examination of the composition of the 10 margarines suggests that the latter may in fact be true. Thus the brands at the top of the table (see reference 5, p 156) have mean R value 0.12 (low in menhaden type) and contain on average about 40% (cheap) hydrogenated marine oils and 16% 18:2 derived from more expensive vegetable oils; per 100 g total hydrogenated marine oils they therefore provide around 40 g of 18:2. The three brands at the foot of the table have mean R = 0.35 (virtually all menhaden) and, per 100 g total hydrogenated marine oils they provide only around 5 g of 18:2. It may then be reasonably concluded that the lower content of menhaden in the more affluent areas arose from choice of better quality and higher priced brands.

It is noteworthy that the above inverse relationship between percentages menhaden and 18:2 in the margarines is reflected in some measure in adipose tissue. Thus the eight mean percent menhaden values of table III are inversely correlated with the eight values of 18:2 (Spearman rank coefficient, $R_s = -0.71$; $p < 0.05$).

On such a basis, the control samples (lower R value) would be expected to have the higher 18:2 content by an amount which may readily be estimated from the data of table I. Thus for the cases, the percent 18:2 arising from consumption of hydrogenated marine oil is $(0.05 \times 5.23) + (0.40 + 6.77) = 2.97$; that for the controls is 3.25. The control excess arising in this way would therefore be only 0.3%. In fact we found⁴ that the percent 18:2 in adipose tissue is 8.0, virtually the same for cases and controls.

The discrepancy could in principle have been due to, say, slightly differing consumption of pig and poultry fat which together were responsible for about one half of the total intake of 18:2 in the UK at the time. A more likely explanation is that, acting on medical advice, a fraction of those cases

with a history of heart disease had changed to high priced margarines "rich in polyunsaturates" (50% or more 18:2 and zero hydrogenated marine oils content) which were becoming available at the time. Thus it is readily calculated that if only seven of the total of 136 cases had changed say one half of their intake of margarine to such brands, the case mean adipose tissue percentage of 18:2 would in a few years reach that of the controls. In this connection it is interesting to realise that if a higher fraction of the cases had so adjusted their fatty diet, the case percentage of 18:2 (and the accompanying 18:3) would exceed the control percentage—an eventuality we have observed for the Glasgow area for which the difference in linolenic acid was statistically significant.⁵

We believe that dietary adjustment may well have been responsible for the observed lower case consumption of ruminant animal fat and for the slightly higher (0.38%) consumption of total hydrogenated marine oil. The greater case excess menhaden (1.26%), however, is highly significant and arose from the higher case value of R. Arguing as previously that the more likely adjustment would have favoured higher priced brands (lower value R), there would then have been a decrease in the case mean value of R towards the lower control value. In other words, in the absence of such adjustment, the case excess menhaden would have been even greater than that observed. Consumers have of course no information regarding the type of hydrogenated marine oil in margarines, and it is difficult to see how the case excess menhaden could have arisen other than by chance selection over a period of time of those materials which happened on average to be richer in menhaden relative to herring than those consumed by the controls. Furthermore, since we are dealing with an inherent feature of one particular type of fat (hydrogenated marine oil), the higher case ratio menhaden to herring is not confounded by compliance with any other medical advice, such as total fat and smoking reduction, which may have been given. The ratio of the two types of hydrogenated marine oil is not of course affected by the total amount consumed.

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