Modulation of fluid absorption and the secretory response of rat jejunum to cholera toxin by dietary fat

F A Sagher, J A Dodge, R Moore, C McMaster, G McCaughey

Abstract
To study the effects of dietary fat on jejunal water and ion absorption and on cholera toxin-induced secretion, 3 week old Sprague Dawley rats were fed isocaloric diets. Forty per cent of the total calories were given as fat, as butter (high saturated fat), olive oil (high monounsaturated fat), or corn oil (high polyunsaturated fat), with one group on low fat (10% of calories) standard laboratory diet as controls. During in vivo jejunal perfusion studies we found that (i) a polyunsaturated fat (corn oil) supplemented diet improves jejunal absorption of water and electrolytes and these changes are independent of the observed concentrations of luminal prostaglandins; (ii) high dietary fat appreciably reduced the secretory response to cholera toxin, probably without fundamentally changing the mechanism by which cholera toxin induces secretion. We conclude that dietary fat composition alters the permeability and transport characteristics of the small intestine. This observation might have relevance to some human diarrhoeal disorders.

Interest in dietary modification of intestinal ion transport and eicosanoid synthesis has arisen both as an alternative approach to studying their physiological importance and for possible therapeutic value. Evidence is gradually accumulating for a protective effect of dietary fat in diarrhoeal disorders. Epidemiological studies suggest a possible relation between low milk fat intake in children and an increased incidence of gastrointestinal illnesses, especially diarrhoea. Careful analysis failed to explain the association and it was concluded that there may be a non-immunoglobulin factor in milk fat which prevents diarrhoeal illness.1

In a clinical study children with chronic nonspecific diarrhoea who had no evidence of malabsorption were found to be taking low fat diets, and they responded well to polyunsaturated fat supplementation.13 In the laboratory fractionation of human milk by ammonium sulphate precipitation and column chromatography yields a non-immunoglobulin fraction which has been shown to protect rabbits against enterotoxin-induced intestinal fluid secretion in vitro. Preliminary experiments showed that the inhibitory activity was extracted by chloroform and methanol, which suggests the involvement of lipid.1 A glycoprotein or a glycolipid compound in the non-immunoglobulin fraction of human milk has recently been shown to inhibit the adherence of certain enterotoxigenic Escherichia coli strains to guinea pig intestinal tract.1

The present study was designed to investigate further the relations between diet and function and to explore the possible protective value of different types of dietary fat in a rat model of secretory diarrhoea.

Methods

PREPARATION OF ANIMALS AND DIETS
Three week old male Sprague Dawley rats were fed one of three synthetic diets. The diets contained 20% fat, which provided 40% of the total calories, as butter, olive oil, or corn oil. The diets were isocaloric and nutritionally adequate, providing all known essential requirements (Special Diet Service, Essex, England). A control group was maintained on a standard low fat (10% of calories) diet (Beta-Laboratory Diets – Witham) (Table I). The fatty acid composition of the diets is shown in Table II. To reduce coprophagy animals were housed individually with alternate 12 hours of light and dark, in cages with a mesh wire bottom, and their intake was calculated daily.

The diet high in saturated fatty acids (butter) provided 24-6% of the total fatty acids and 9-8% of the total calories (% wt/wt) as oleic acid, and approximately 2-5% of the total fatty acids and 0-95% of the total calories as linoleic acid, whereas only approximately 0-6% of the total fatty acids and 0-2% of the total calories were provided as α linolenic acid, with a w6/w3 ratio of 4-75.

The diet high in polyunsaturated fatty acids (corn oil) provided 29-78% of the total fatty acids and 11-9% of the total calories as oleic acid, approximately 58% of the total fatty acids and 23-2% of the total calories as linoleic acid, and only approximately 0-2% of the total fatty acids and 0-8% of the total calories were provided as α linolenic acid, with a w6/w3 ratio of 29.

The diet high in monounsaturated fatty acids (olive oil) provided 79-4% of the total fatty acids and 31-7% of the total calories as oleic acid, approximately 7% of the total fatty acids and 2-8% of the total calories as linoleic acid, but only approximately 1% of the total fatty acids and

<table>
<thead>
<tr>
<th>Table 1</th>
<th>The basic diet per 1 kg of complete diet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Synthetic (g)</td>
</tr>
<tr>
<td>Fat*</td>
<td>200</td>
</tr>
<tr>
<td>Protein (casein) vitamin free</td>
<td>200</td>
</tr>
<tr>
<td>Carbohydrate:</td>
<td></td>
</tr>
<tr>
<td>Corn starch</td>
<td>250</td>
</tr>
<tr>
<td>Sucrose</td>
<td>250</td>
</tr>
<tr>
<td>Vitamin mix</td>
<td>20</td>
</tr>
<tr>
<td>Salt mix</td>
<td>40</td>
</tr>
<tr>
<td>Fibre (non-nutritive)</td>
<td>40</td>
</tr>
</tbody>
</table>

* Dietary fat as high monounsaturated (olive oil), or high saturated (butter), or high polyunsaturated (corn oil).
TABLE II  Fatty acid profile of diets*  

<table>
<thead>
<tr>
<th>Fatty acid</th>
<th>Olive oil (HMU) (% wt)</th>
<th>Butter (HS) (% wt)</th>
<th>Corn oil (HPU) (% wt)</th>
<th>Standard diet: 3-5% fat (% wt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6:0</td>
<td>—</td>
<td>0.28</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>8:0</td>
<td>—</td>
<td>0.86</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>10:0</td>
<td>—</td>
<td>2.82</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>12:0</td>
<td>—</td>
<td>2.89</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>14:0</td>
<td>0-12</td>
<td>17.02</td>
<td>0.26</td>
<td>0.57</td>
</tr>
<tr>
<td>14:1</td>
<td>—</td>
<td>1.32</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>15:0</td>
<td>—</td>
<td>1.21</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>16:0</td>
<td>9.22</td>
<td>31.22</td>
<td>8.46</td>
<td>13.87</td>
</tr>
<tr>
<td>16:1</td>
<td>—</td>
<td>1.72</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>17:0</td>
<td>—</td>
<td>0.86</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>18:0</td>
<td>2.94</td>
<td>10.38</td>
<td>1.76</td>
<td>2.15</td>
</tr>
<tr>
<td>18:1</td>
<td>79.36</td>
<td>24.58</td>
<td>29.78</td>
<td>22.51</td>
</tr>
<tr>
<td>18:2 w6</td>
<td>6.99</td>
<td>2.59</td>
<td>5.78</td>
<td>14.13</td>
</tr>
<tr>
<td>18:3 w3</td>
<td>0.89</td>
<td>0.58</td>
<td>1.97</td>
<td>6.28</td>
</tr>
<tr>
<td>20:1</td>
<td>—</td>
<td>1.84</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Others</td>
<td>—</td>
<td>1.84</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Saturated</td>
<td>12.28</td>
<td>67.57</td>
<td>10.48</td>
<td>16.59</td>
</tr>
<tr>
<td>Monounsaturated</td>
<td>80-16</td>
<td>27.62</td>
<td>29.78</td>
<td>23.01</td>
</tr>
<tr>
<td>Polyunsaturated</td>
<td>7.88</td>
<td>2.97</td>
<td>59.75</td>
<td>60.41</td>
</tr>
</tbody>
</table>

*Diets analysed by Department of Food Biochemistry, Faculty of Agriculture, Queen's University of Belfast.
HMU=high monounsaturated; HS=high saturated; HPU=high polyunsaturated.

0.4% of the total calories were provided as α-linolenic acid, with a w6/w3 ratio of 7.

STEADY STATE JEJUNAL PERFUSION STUDIES
The non-stimulated perfusions were performed on adult male Sprague Dawley rats after an overnight fast (18-20 hours) during which water only was allowed ad libitum. Before laparotomy, anaesthesia was induced with intraperitoneal sodium pentobarbitone (60 mg/kg) and maintained when necessary with intramuscular doses of 0.3 mg. Rectal temperature was maintained throughout the experiment at 37°C (mean (SEM)) by an electrically heated pad. The perfusion studies were essentially as described by Sladen and Harries with some modification as follows.

The abdomen was opened with a midline incision and about 20 cm length of jejunum from the duodenojejunal junction was prepared. The inlet catheter (polyvinyl tubing bore 0.63 mm, diameter 1.4 mm) and the outlet catheter (polyvinyl tubing bore 2 mm, diameter 3 mm) were inserted into the gut and tied firmly in position with silk ligature. Faecal material, if present, was removed from the loop by washing with the perfusate at 37°C. The loop was then rinsed with perfusate and prepared for perfusion as previously described.

Cholera toxin was obtained from Sigma Chemical Company (No C3012).

A very low (1-6 mmol/l) glucose concentration was used as an additional marker, because luminal disappearance will reflect active transport or mucosal metabolism, or both, and thus viability of mucosa. It was assumed that any effect of prolonged anaesthesia on the absorption would be similar to all four groups.

LUMINAL PROSTAGLANDIN ASSAY
One ml of each collection of good recovery from non-stimulated jejunal perfusates was reserved at −20°C after centrifugation at 2500 rpm for 10 minutes, extracted as described by Powell, and assayed for prostaglandin E2 and F2α using prostaglandin radioimmunoassay kit (Seragen Inc, Boston) within three weeks. For each prostaglandin a standard curve of percentage binding vs pg prostaglandin was plotted using several standards. The amount of PGE2 and PGF2α contained in each sample was then calculated using the appropriate curve and corrected for 100% recovery. Each assay included non-specific binding standards – that is, no added antibody – and controls. The results were corrected for the flow rate and the wet weight of the perfused segment and expressed in pg/ml per min per g wet weight.

The cross-reactivity of PGE2 with PGF2α is only 0.3% and PGF2α with PGE2 1.3%. The
Dietary fat and ion transport

<table>
<thead>
<tr>
<th>Water (μl/g per min)</th>
<th>Na (μmol/g per min)</th>
<th>Cl (μmol/g per min)</th>
<th>K (μmol/g per min)</th>
<th>Glucose (μmol/g per min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=8)</td>
<td>7.67 (1.4)</td>
<td>1.93 (0.245)</td>
<td>0.452 (0.173)</td>
<td>0.0456 (0.006)</td>
</tr>
<tr>
<td>Olive oil (HMU) (n=8)</td>
<td>8.59 (2.7) NS</td>
<td>1.70 (0.34 NS)</td>
<td>0.32 (0.49 NS)</td>
<td>0.11 (0.036 NS)</td>
</tr>
<tr>
<td>Butter (HS) (n=6)</td>
<td>0.161 (0.86)**</td>
<td>-0.181 (0.56)**</td>
<td>-0.012 (0.102 NS)</td>
<td>0.147 (0.03 NS)</td>
</tr>
<tr>
<td>Corn oil (HPU) (n=6)</td>
<td>12.4 (2.0)**</td>
<td>2.57 (0.285)</td>
<td>0.075 (0.35 NS)</td>
<td>0.067 (0.016 NS)</td>
</tr>
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Values are expressed as mean (SEM). Values v control: *p<0.05; **p<0.025. NS=not significant.

recovery of total counts is better than 95%. The coefficient of variation of PGE2 is 8.5% (intra-assay) and 5.0% (interassay) and for PGF2α, 8.1% (intra-assay) and 5.0% (interassay).

Rats were sacrificed at the end of the experiment by cardiac puncture and the perfused segment removed and weighed after stripping fluid from the lumen. In all the experiments the concentration of glucose in the effluent was checked by a specific glucose oxidase method (Beckman glucose analyser), Na⁺ and K⁺ by ion selective electrode (Beckman Astra), and Cl⁻ by the colorimetric titration technique (Corning 920). ³C-polyethylene glycol activity in 100 μl aliquots (in duplicate) were counted in Scintillation HV (BDH) in a Beckman scintillation counter. Only 100 (5%) of ³C PEG levels were accepted as good recovery.

STATISTICAL ANALYSIS OF THE RESULTS

One way analysis of variance (Anova) and unpaired Student’s t test were used to analyse the statistical significance of the differences between the means of water and electrolyte absorption. The values obtained were reported as mean (SEM).

The non-parametric Kruskal-Wallis test of variance and Mann-Whitney U test were used to analyse the statistical significance of the differences of prostaglandin concentration between the groups. The values obtained were reported as mean (SEM) and range.

Results

CHARACTERISTICS OF ANIMALS

The weight of animals at the beginning and at the end of the experiments and the duration of feeding did not significantly differ between the four groups (Table III). No change in the frequency or in the consistency of stool was observed during the feeding period.

EFFECTS ON JEJUNAL ION TRANSPORT

Feeding rats corn oil enhanced the absorption of water (p<0.05 v control; p>0.005 v olive oil and butter), Na⁺ (p<0.05 v olive oil; p<0.005 v butter), and Cl⁻ (p<0.025 v butter only), but did not significantly change the absorption of K⁺ or glucose.

Feeding butter fat reduced the absorption of water, Na⁺, and Cl⁻ (p<0.025 v control) without significantly changing the absorption of K⁺ and glucose (Table IV). Olive oil did not significantly alter the transport of water or electrolyte during perfusion studies.

EFFECTS ON LUMINAL EICOSANOIDS

Feeding rats either butter or corn oil did not significantly change the luminal PGE2 and PGF2α, during in vivo steady state perfusion studies as compared to low fat chow diet.

Feeding rats olive oil increased the production of luminal PGE2 (p<0.005 v control; p<0.025 v butter and corn oil) and also increased the production of luminal PGF2α (p<0.025 v control and butter; p<0.005 v corn oil). Comparing the effects of dietary fat on the mean PGE2/PGF2α ratio, it seems that feeding rats olive oil increased both prostaglandins, particularly PGF2α, whereas feeding corn oil appreciably decreased PGE2 compared to the low fat diet (Table V).

EFFECTS ON CHOLERA TOXIN-INDUCED SECRETION

Feeding rats high dietary fat did not significantly alter the absorption of glucose at the low concentration used. High monounsaturated and high polyunsaturated dietary fat, compared with low fat intakes, reduced the effects of cholera toxin on secretion of water and electrolytes (Figs 1 and 2).

Taking into consideration the baseline absorption of unstimulated jejunum, it seems that all three high dietary fat regimens reduced the net secretory response to cholera toxin by about 50%.

Discussion

The absorption values of these perfusion studies were lower than those previously reported. The reason behind these variations may be related to the variation in the Na⁺ content of the perfusate, the relatively higher osmolality, lower glucose, lower total CO₂, and lower flow rate in this study, the variation in the ages of the animals, and the units used to express the data. Since our aim was to compare the effects of different dietary fat on ion transport under the same conditions, however, the comparisons are valid.

Prostaglandin synthesis in the intestine occurs predominantly in the subepithelial cells, whereas the capacity to degrade prostaglandins lies mainly in the epithelial cells. Prostaglandins are not stored in tissue but are continually formed and

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**Table IV** Dietary fat and ion transport

<table>
<thead>
<tr>
<th>Diet</th>
<th>Water (μl/g per min)</th>
<th>Na (μmol/g per min)</th>
<th>Cl (μmol/g per min)</th>
<th>K (μmol/g per min)</th>
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<td></td>
</tr>
</tbody>
</table>

Values are expressed as mean (SEM). Values v control: *p<0.05; **p<0.025. NS=not significant.

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**Table V** Effects of dietary fat on luminal prostaglandins (PG)

<table>
<thead>
<tr>
<th>Diet (no of perfusions)</th>
<th>PGE2</th>
<th>PGF2α</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=8)</td>
<td>24.1 (3.6)</td>
<td>2.9 (0.9)</td>
</tr>
<tr>
<td>Olive oil (HMU) (n=8)</td>
<td>37.04 (4.3)</td>
<td>7.4 (1.7)</td>
</tr>
<tr>
<td>Corn oil (HPU) (n=6)</td>
<td>17.9 (2.4)</td>
<td>2.57 (0.04)</td>
</tr>
</tbody>
</table>

Values are expressed as mean (SEM) and range.

* *p<0.005 v control, and p<0.025 v butter and corn oil.
** p<0.025 v control and butter, and p<0.005 v corn oil.

All unstimulated perfusion studies were performed on male Sprague Dawley rats after feeding one of the synthetic diets (Table I).
Modulation of fluid absorption and the secretory response of rat jejunum to cholera toxin by dietary fat

released. Determination of luminal PGE\(_2\) concentrations provides a simple and reproducible index of intestinal prostaglandin production which may be used for studies of pathophysiological mechanisms.\(^6\) The use of a valid marker perfusion technique may provide a reliable index of local prostaglandin synthesis after exclusion of the luminal fluids from the stomach, pancreas, and bile.

The sequence of biochemical events that occurs between ingestion of dietary fat and tissue prostaglandin production is complex, with several steps of autoregulation, and the effects of dietary manipulation on prostaglandin synthesis in various body organs are difficult to predict.

Permeability properties of the lipid bilayer are greatly dependent on the nature and chain length of the fatty acid constituents of lipid molecules.\(^1\) High dietary polyunsaturated fat is known to increase the fluidity of the enterocyte plasma membrane,\(^2\) which in turn produces a more permeable membrane due to reduced attractive forces between adjacent lipid molecules.\(^3\) The observations that high polyunsaturated fat appreciably increased the activity of Na\(^+\)/K\(^+\) ATPase in the basolateral membranes,\(^4\) significantly increased tissue concentration of somatostatin in the proximal small intestine (submitted data),\(^5\) and appreciably increased villous heights in the jejunum (submitted data)\(^6\) might lead to an expectation of enhanced absorption.\(^7\) The alterations in prostaglandins produced by diet may be relevant, since luminal PGE\(_2\) in rats fed corn oil was reduced to lower levels than in the control groups\(^8\) (Table V). Polyunsaturated fat may therefore inhibit a prostaglandin mediated step in intestinal secretion beyond the generation of cAMP.\(^9\)

In contrast, rats on high monounsaturated dietary fat showed no significant alteration in ion transport compared with low fat control groups. Their luminal PGE\(_2\) and PGF\(_{2\alpha}\) were significantly increased during perfusion (Table V), however, and the fact that an increase in luminal PGE\(_2\) has been shown to increase the inhibition of Na\(^+\)/K\(^+\) ATPase and stimulate adenyl cyclase\(^10\) may explain the relative reduction in the absorption of water and electrolyte compared with the corn oil group.

High saturated fat reduces the permeability of the enterocyte plasma membrane,\(^11\) thereby greatly reduces Na\(^+\)/K\(^+\)ATPase activities,\(^12\) and also reduces the jejunal villus height.\(^13\) It also increases total phospholipids in intestinal microvillus membranes and in particular membrane bound phosphatidylinositol,\(^14\) the hydrolysis of which is accompanied by more gating of Ca\(^{2+}\) across the basolateral membrane. This calcium-gating process increases ionised Ca\(^{2+}\) levels in the cell and this is thought to stimulate secretion through Ca\(^{2+}\)-dependent protein kinases. This is under investigation in our laboratory. These factors in combination may explain the appreciable reduction in water and electrolyte transport that we observed in the high saturated fat group (Table IV).

We used a single, very low glucose concentration as a marker to show the viability of the mucosa. Further studies using different concentrations of glucose are required to elucidate the possible effects of fat on glucose absorption. Previous in vitro studies showed that the rate of jejunal uptake of D-glucose varied from 0.5–20 mmol and was higher in animals fed high polyunsaturated fat.\(^15\)

Cholera toxin-induced stimulation serves as a useful model of secretory diarrhea in experimental studies. Cholera toxin is a protein molecule, which consists of a B subunit containing five non-cova-lently linked molecules and an A subunit containing two peptides, A\(_1\) and A\(_2\), linked by a disulphide bond.\(^16\) In a comprehensive review of the pathogenesis of cholera, Hendrix\(^17\) concluded that there is no evidence that cholera toxin increases epithelial permeability or osmotic or hydrostatic gradients, and the increased intestinal fluid secretion stimulated by cholera toxin arises from increased filtration through normal calibre 'channels.' The gross effect of cholera toxin is the production of water and electrolyte secretion by a complex biochemical mechanism, not yet well understood. It involves a rise in cAMP, which causes active secretion of Cl\(^-\) from the crypts and inhibits active electrogenic Na\(^+\)-coupled glucose absorption.\(^18\) It is not definitely known how the binding of the B subunit to a specific membrane glyco-

![Figure 1: Secretory (water) response of jejunum to cholera toxin in rats fed diets with differing fatty acid contents.](image1)

![Figure 2: Secretory (electrolyte) response of jejunum to cholera toxin in rats fed diets with differing fatty acid contents.](image2)
lipid mediates the entry of the A subunit or whether this is the only mechanism by which cholera toxin induces a secretory state.

In the present studies, if we can extrapolate from baseline studies in male rats to the cholera toxin-induced studies in female animals, it seems that the pre-existing absorptive/secretory state of the mucosa has a pronounced effect on the secretory response to toxin. Thus, bearing in mind that a butter diet produced a small net secretory state when unstimulated whereas corn oil apparently enhanced water and electrolyte absorption, all three fat supplements reduced the net secretory response by about half. This suggests that the fundamental mechanism by which cholera toxin activates secretion is unchanged, although a number of other theories may explain variations in both the unstimulated absorption and the toxin response.

The changes in membrane composition induced by dietary fat may determine the binding of bacteria or toxin to the intestinal cells. Studies on animals have shown that more cholera toxin binds to the microvillus membrane of the newborn rat intestine compared with adults28 and these effects may be related to the reduced ratio of protein/phospholipids commonly found in the immature membranes of the newborn.27 28 Although high polyunsaturated dietary fat reduces the total phospholipids in microvilli,29 which may in turn reduce the binding sites available for cholera toxin, this is unlikely to be the only explanation for our observations. Polyunsaturated fat, however, can increase activity of Na+/K+ ATPase in the basolateral membrane,30 31 and this could counteract the secretory effect of adenyl cyclase induced by cholera toxin.

The Na+-coupled glucose absorption route is unlikely to be involved in the protective mechanism of unsaturated dietary fat because cholera toxin does not interfere with Na+-coupled glucose transport32 and dietary fat did not appreciably alter the absorption of glucose at the low concentration used in this study.

Polyunsaturated fat significantly increased the tissue concentration of somatostatin in the proximal segment of rat small intestine compared to the low fat group (submitted data).33 Somatostatin inhibits in vivo jejunal secretion induced by PGE2 and tetrophiline in rats,34 improves diarrhoea in human carcinoid syndrome35 and in severe refractory secretory diarrhoea,36 and inhibits adrenergic and cholinergic neurotransmission in smooth muscle.37 High tissue concentrations of somatostatin may therefore contribute to the protective effects of corn oil in secretory diarrhoea.

The protective mechanism of olive oil may involve different mediators. Rats maintained on high monounsaturated dietary fat (olive oil) had high levels of luminal PGE2 and PGF2α, during unstimulated perfusion studies. This might be expected to alter the interdigestive motility pattern of the small intestine38 and contribute to sodium and chloride secretion.39

The possibility exists that stimulation of adenyl cyclase by prostaglandins40 and by cholera toxin41 is through different and antagonistic or competitive pathways.

The applicability of these studies to diarrhoeal disorders in humans is not known, but preliminary clinical observations suggest that the fatty acid composition of cell membranes may be related to the varying severity of childhood diarrhoea in different populations.42

We thank Mr T McFarlane, Royal Belfast Hospital for Sick Children, and Mr T Oliver, Department of Food Biochemistry, Faculty of Agriculture, Queen's University of Belfast, for their technical help.


27 Cho SW, Walker WA. Development of the gastrointestinal mucosal barrier: changes in phospholipid head groups and
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