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Effect of dietary palm oil on arterial thrombus formation and prostanoid production in rats

G. Hornstra

Dept. of Biochemistry, Limburg University, P.O. Box 616, 6200 MD Maastricht, The Netherlands

Earlier studies, using a rat model of arterial thrombosis, demonstrated that oils rich in polyunsaturated fatty acids of the (*n*-6) or (*n*-3) families and poor in saturated fatty acids, have an antithrombotic effect. Oleic acid appeared neutral as to arterial thrombus formation: it is antithrombotic only if it replaces prothrombotic dietary lipids. Long chain (> 14 carbon atoms) saturated fatty acids were shown to promote arterial thrombus formation.

A palm oil enriched diet, however, appeared an exception to this rule: although palm oil contains about 50% long chain saturated fatty acids (mainly palmitic acid), it was shown to have a distinct antithrombotic effect [1].

Recently, the antithrombotic effect of dietary palm oil was confirmed (Table 1). Rats were fed diets containing 50% of its digestible energy (en%) physically refined palm oil (PO-R) or alkali-refined palm oil (PO-N). Two other groups of animals were given diets containing either 5 (negative control group) or 50 (positive control group) en% sunflowerseed oil. After 8–10 weeks of feeding, arterial thrombosis tendency in the PO-N group was lower than that in the negative control group and did not differ significantly from that in the positive control group, the thrombosis tendency of which was significantly reduced. Physically refined palm oil also tended to lower arterial thrombosis tendency; however, its effect did not reach statistical significance.

As compared with the positive control group, palm oil feeding significantly decreased collagen-induced platelet aggregation in whole blood, measured with the Chronolog whole-blood lumi-aggregometer. Platelet aggregation tended to be increased in the positive control group, rich in sunflowerseed oil. This is not in agreement with the very consistent antithrombotic activity of dietary sunflowerseed oil but has been observed before [2], using the "classical" turbidimetric platelet aggregation test.

In the same collagen-activated blood samples, the platelet release reaction was determined simultaneously with the aggregation reaction, by measuring the course of ATP-dependent luminescence. Platelets of palm oil fed animals released less ATP than those obtained from animals of both control groups.

Since the balance between prothrombotic thromboxane A_2 (TxA_2) and antithrombotic prostacyclin (PGI_2) has been suggested to determine platelet aggregability and arterial thrombosis tendency [3], TxA_2 and PGI_2 formation was determined in plasma of the blood samples used before for aggregation and release studies. Measurements were performed by applying a radioimmunoassay (RIA) for TxB_2, the stable hydrolysis product of TxA_2, and a RIA for 6-keto-$PGF_{1\alpha}$, the major metabolite of PGI_2. Feeding of palm oil caused a significant reduction in the formation of prothrombotic TxA_2 upon activation of platelets with collagen. The PGI_2 production (most probably from monocytes) was not significantly different between the groups. Therefore, palm oil feeding significantly reduced the TxA_2/PGI_2 ratio in collagen-activated whole blood.

Table 1
Effect of palm oil (PO)-enriched diets (50 en%) on arterial thrombosis tendency (obstruction time, OT, h), platelet function, and prostanoid formation (mean ± S.E.M., $n = 16–18$). C: negative control group (5 en% sunflowerseed oil, SO), SO: positive control group (50 en% SO), PO-R: physically refined palm oil, PO-N: alkali refined palm oil.

Parameter	C	SO	PO-R	PO-N
Arterial thrombosis				
log OT	1.91 ± 0.142[b]	2.09 ± 0.120[a]	1.99 ± 0.172	2.07 ± 0.157[a]
OT (h)	81	122	97	116
Platelet function				
Aggregation (Ω)	6.4 ± 0.50	8.3 ± 0.64	5.3 ± 0.45[b]	4.8 ± 0.52[b]
ATP-release (μmol/l)	4.5 ± 0.38	5.0 ± 0.34	3.3 ± 0.31[a, b]	3.5 ± 0.41[b]
Prostanoid production				
TxB_2 (ng/ml plasma)	73 ± 5.9	67 ± 4.6	53 ± 3.3[a]	56 ± 4.1
6-keto-$PGF_{1\alpha}$ (pg/ml plasma)	323 ± 20.0	295 ± 15.7	324 ± 17.4	368 ± 20.2[b]
TxB_2/6-keto ratio	217 ± 17.0	220 ± 16.0	168 ± 11.0[b]	159 ± 12.8[a, b]

[a] $P_2 < 0.05$ as compared with negative control group (C),
[b] $P_2 < 0.05$ as compared with positive control group (SO).
Bonferroni inequality test.

The lower TxA_2 production by collagen-activated platelets of palm oil fed animals is not due to a lower arachidonic acid (AA) content of the platelet phospholipids. Fluidity of platelet membranes, measured by steady state fluorescence polarization using DPH as a fluorescent probe, was not different in the palm oil group as compared with the lowfat control group but was enhanced in the positive control group [4].
The unexpected effects of palm oil on platelet function and arterial thrombosis are difficult to explain on the basis of its fatty acid composition. The importance of palm oil as a nutritional oil justifies further research to elucidate the mechanism by which it exerts its beneficial effects.

References

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