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Influence of triacylglycerol structure of stearic acid-rich fats on postprandial lipaemia

Sarah E. E. Berry* and Thomas A. B. Sanders
Nutrition Food and Health Research Centre, King's College London, Franklin Wilkins Building, 150 Stamford Street, London SE1 9NH, UK

Exaggerated postprandial lipaemia may increase the risk of CHD by contributing to both thrombotic and atherogenic processes. Previous research has focused on the quantity and composition of dietary fat, whereas the effect of triacylglycerol (TAG) structure on postprandial lipaemia and clotting factor VII activity has received little attention. TAG with similar fatty acid composition may have different biochemical and physical properties that are dependent on their TAG structure, and these differences may affect lipid metabolism. Recent findings suggest that differences in the physical properties of stearic acid-rich fats are associated with differences in postprandial lipaemia, and may play an important role in determining their rates of digestion and absorption.

Postprandial lipaemia: Triacylglycerol structure: Stearic acid

Dietary fat intake is a major dietary factor implicated in the causation of CHD, and has been shown to influence both the atherosclerotic and thrombotic processes that cause the disease. It is well established that a diet high in long-chain saturated fatty acids (SFA; C_{14}–C_{16}) increases plasma LDL-cholesterol concentration, which promotes foam cell formation and the early stages of atherogenesis. Dietary fat intake may also influence risk by other mechanisms, particularly by influencing haemostatic function and insulin sensitivity, and this aspect has recently been reviewed in detail (Sanders, 2003). Studies in animals have shown that chylomicron remnants are atherogenic (Zilversmit, 1979). Subsequent observations in human subjects have shown that chylomicron remnants, but not chylomicrons, are associated with increased risk of coronary atherosclerosis (Patsch *et al*. 1992). Gianturco & Bradley (1999) have shown that triacylglycerol (TAG)-rich remnant particles, as well as resulting in arterial fatty steak formation, are toxic to the vascular endothelium. More recent research has shown that prolonged elevations of serum TAG-rich lipoproteins result in lipid transfer reactions catalysed by cholesteryl ester transfer protein that result in increased HDL turnover and the formation of small dense LDL particles, which are more atherogenic (Griffin, 1999). Meals containing 50 g fat compared with isoenergetic low-fat high-carbohydrate meals result in an increase in factor VII coagulant activity (Oakley *et al*. 1998) that reflects an increase in the concentration of the activated form of the clotting factor and impaired endothelial function (Ong *et al*. 1999). There has been a lack of information on the influence of TAG structure on postprandial lipaemia. The present review describes recent work that demonstrates that the TAG structure of stearic acid (18:0)-rich fats influences postprandial lipaemia.

Postprandial lipaemia

Postprandial lipaemia can be defined as the increase in plasma TAG concentration following a fatty meal. An intake of >15 g fat containing long-chain fatty acids results in detectable postprandial lipaemia (Dubois *et al*. 1998); however, intakes of >30 g fat are normally required to result in marked lipaemia in adults. Larger amounts of fat are required to result in lipaemia in young healthy subjects, particularly among premenopausal women (Cohen *et al*. 1988; Chen *et al*. 1992). Although the magnitude of postprandial lipaemia depends on the fat content of a meal, it can be enhanced by consecutive fat-containing meals (Jackson *et al*. 2002). Both the background diet as well as

Abbreviations: SFA, saturated fatty acid; TAG, triacylglycerol.

***Corresponding author:** Dr Sarah Berry, fax +44 20 7848 4185, email sarah.e.berry@kcl.ac.uk

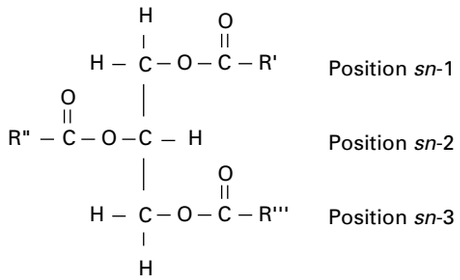

Fig. 1. The structure of the triacylglycerol molecule.

the fatty acid composition of the test meal can influence the extent of lipaemia (Weintraub *et al*. 1988; Zampelas *et al*. 1994*a*; Sanders *et al*. 1997, 2000; Oakley *et al*. 1998; Roche *et al*. 1998). High intakes of long-chain *n*-3 fatty acids tend to decrease postprandial lipaemia, probably by acutely suppressing VLDL synthesis and thus reducing competition from VLDL for removal of chylomicron remnants. There appears to be little difference in the postprandial lipaemic response between *cis*- and *trans*-fatty acids (Sanders *et al*. 2000, 2003*b*). Generally, fats rich in the fatty acid oleic acid (18:1*n*-9) have been found to cause pronounced lipaemia, with a tendency for an early peak and larger chylomicron particles. SFA differ in their capacity to cause lipaemia according to the fatty acid chain length. Short- and medium-chain SFA ($<C_{12}$) do not lead to marked lipaemia because they are absorbed and transported via the hepatic portal vein. Very-long-chain SFA such as arachidic (20:0), behenic (22:0) and lignoceric (24:0) acids are poorly absorbed, and variable results have been obtained with fats rich in stearic acid (18:0), as will be discussed. Sanders *et al*. (2001) first proposed that the configuration of stearic acid-rich TAG affects their capacity to cause postprandial lipaemia; in particular it was suggested that asymmetric disaturated TAG (with 18:0 in the *sn-1* and *sn-2* position) results in less lipaemia than disaturated TAG that are symmetrical (with SFA in the *sn*-1 and *sn*-3 positions; Fig. 1). A series of studies has subsequently been conducted to investigate this hypothesis (Berry & Sanders, 2003*a*,*b*; Sanders *et al*. 2003*a*).

Triacylglycerol structure

It was believed that the overall melting point of a fat is the most important factor influencing fat digestibility (Cheng *et al*. 1949). However, the melting point of individual SFA is generally higher than those of the TAG that contain them; for example, the melting points of palmitic acid (16:0) and 18:0 are 64 and 69°C respectively and those of 1-stearoyl 2, 3-dipalmitoylglycerol and 1-palmitoyl 2, 3-distearoylglycerol are 62·7 and 65·2°C respectively. Subsequent studies have indicated that both the content of SFA and the arrangements of those SFA on the glycerol backbone influence digestibility (for reviews, see Small, 1991; Bracco, 1994; Kritchevsky, 1995; Decker, 1996). The positional composition of dietary TAG can affect their absorption from the gut and also their metabolism in enterocytes, subsequent chylomicron metabolism and their distribution into tissues.

Table 1. Positional distribution of fatty acids of some common fats and oils

		Fatty acid			
	Position	16:0	18:0	18:1*n*-9	18:2*n*-6
Cocoa butter	1 and 3	38	52	7	tr
	2	3	4	86	7
Palm oil	1 and 3	43	7	41	7
	2	7	1	71	19
Shea butter	1 and 3	4	80	10	5
	2	1	3	79	17
Peanut*	1 and 3	14	5	59	19
	2	2	tr	59	39
Lard*	1 and 3	10	30	51	6
	2	72	2	13	3
Cow's milk*	1 and 3	34	10	30	2
	2	32	10	19	4
Human milk*	1 and 3	16	15	46	11
	2	58	3	13	7

tr, Trace.
*From www.lipidlibrary.co.uk/lipids.html; accessed 25.8.2004.

The positional composition of some common vegetable and animal fats are shown in Table 1. In fats of animal origin there is a high proportion of SFA in the *sn-2* position, whereas in vegetable fats SFA are found predominantly in the external *sn*-1 and *sn*-3 positions.

The importance of these differences in TAG structure becomes apparent when the specificity of mammalian lipases is considered. Both pancreatic lipase (Yang & Kuksis, 1991) and lipoprotein lipase (Nilsson-Ehle *et al*. 1973) preferentially hydrolyse the fatty acids in the *sn*-1 and *sn*-3 positions of the TAG, leaving the fatty acid in the *sn*-2 position as a 2-monoacylglycerol (Fig. 2). The positional specificity of pancreatic lipase may be advantageous in infants because of the improved absorption of SFA as a 2-monoacylglycerol rather than as NEFA. Long-chain SFA present in the intestine as NEFA (hydrolysed from the *sn*-1 and *sn*-2 positions of TAG) have high individual melting points above body temperature (18:0, 69°C; 16:0, 64°C), which are less soluble in the liquid phase, and tend to form hydrated acid–Ca soaps that are insoluble in aqueous media at the pH of the intestine. Indeed, feeding SFA esterified to the *sn*-2 position results in a reduction in Ca excretion (Carnielli *et al*. 1995). It has, therefore, been commonly held that long-chain SFA are better absorbed if situated in the *sn*-2 position than if located in the *sn*-1 and *sn*-3 positions (Mattson *et al*. 1979; Tomarelli *et al*. 1968).

It is well documented in animals and infants that 16:0 and 18:0 in the *sn*-2 position of the TAG are absorbed more efficiently than when they are in the *sn*-1 and *sn*-3 positions, and this factor accounts in part for the lower digestibility of the fat from cow's milk compared with that of human milk (Tomarelli *et al*. 1968). The food industry has utilised this characteristic to manipulate the absorption of long-chain SFA. SALATRIM^{TM} has been developed as a cocoa-butter substitute with a lower energy value that consists of randomised TAG consisting of 18:0, propionic

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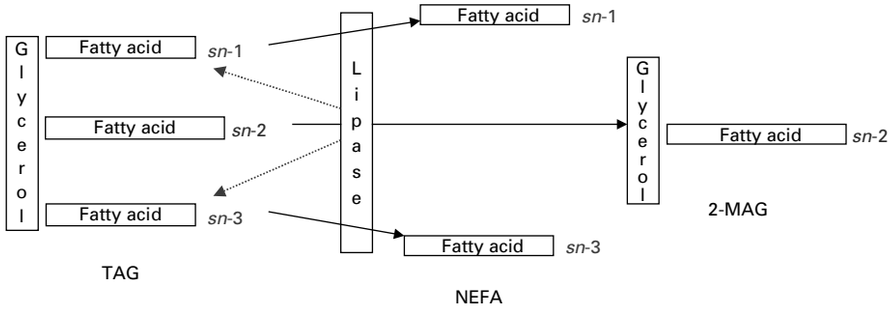

Fig. 2. Lipolysis of triacylglycerol by lipoprotein or pancreatic lipase. TAG, triacylglycerol; 2-MAG, 2-monoacylglycerol.

acid (3:0) and acetic acid (2:0), supplying 24 kJ (5 kcal)/g as opposed to 37·8 kJ (9 kcal)/g. Betapol^{TM} has been developed to enhance the absorption of fat in infants and involves enzyme-directed esterification of mainly 16:0 into the *sn*-2 position.

The fatty acid in the *sn*-2 position is retained on absorption and subsequent metabolism (Yang & Kuksis, 1991; Innis *et al*. 1994). Fatty acids attached to the *sn*-2 position may be preferentially transported to the liver instead of extrahepatic organs because of the positional specificity of lipoprotein lipase for the *sn*-1 and *sn*-3 positions of TAG. The residual TAG remaining in the chylomicron remnant after lipoprotein lipase action are an important source of hepatic fatty acids and are estimated to account for 73% of the newly-synthesised VLDL-TAG in mice (Jung *et al*. 1999).

Mortimer *et al*. (1994) have reported that the plasma clearance of TAG-rich lipoproteins depends on the positional composition of the TAG rather than the overall saturation of the TAG. Both 16:0 and 18:0 in the *sn*-2 position of dietary TAG slow down the clearance of chylomicrons (Mortimer *et al*. 1988, 1992; Redgrave *et al*. 1988). The rates of hydrolysis by lipoprotein lipase have also been shown to be affected by the positional distribution of SFA in some (Redgrave *et al*. 1988; Emken, 1992), but not all (Mortimer *et al*. 1988; Pufal *et al*. 1995; Hodge *et al*. 1999; Yli-Jokipii *et al*. 2002), studies.

This evidence would suggest that dietary fats containing TAG with a predominance of SFA in the *sn*-2 position might be absorbed more rapidly and cleared from the circulation more slowly than TAG containing SFA at the *sn*-1 and *sn*-3 positions, resulting in elevated chylomicron remnant concentrations and a more pronounced and prolonged postprandial lipaemia. Although the role of TAG molecular structure in influencing SFA absorption is supported by data from studies in animals and human infants, there are questions in the human adult relating to the relevance of positional distribution on absorption, digestion and postprandial lipaemia. Recent studies suggest that the human adult can absorb most dietary fatty acids efficiently, whether in the form of NEFA or 2-monoacylglycerol.

Betapol, a synthetic TAG with 74% 16:0 in the *sn*-2 position that can be prepared by enzyme-directed interesterification, has been compared with palm oil (approximately 7% 16:0 in *sn*-2) and has been found to produce a similar level of postprandial lipaemia (Zampelas *et al*. 1994*b*). Summers *et al*. (1999) have also found no difference in postprandial lipaemia in subjects receiving the structured TAG 1-stearoyl 2,3-dioleoylglycerol and 1,3-dioleoyl 2-stearoylglycerol, also prepared by enzyme-directed inter-esterification. In contrast, studies using random inter-esterification (randomisation) to prepare test fats have reported differences in postprandial lipaemia, and the presence of 18:0 or 16:0 in the *sn*-2 position of TAG (randomised fat) produces a lower postprandial lipaemia than when present in the *sn*-1 or *sn*-3 position (unrandomised fat; Sanders *et al*. 2000, 2001; Yli-Jokipii *et al*. 2001).

Stearic acid, triacylglycerol structure and postprandial lipaemia

18:0 is the second most abundant SFA in the Western diet; in the UK approximately 8·54 g is consumed daily, accounting for 24% of the SFA intake. The early studies of Hegsted *et al*. (1965) and Keys *et al*. (1965) noted that 18:0 has a neutral effect on serum cholesterol concentrations. Since these early studies numerous feeding studies have examined the effects of cocoa butter (Connor *et al*. 1969; Kris-Etherton *et al*. 1993) and synthetic fats with a high 18:0 content (Bonanome & Grundy, 1988), and have shown that 18:0 elicits a hypocholesterolaemic effect compared with other SFA. The reduction in cholesterol concentrations on a high-18:0 diet may be a result of its direct effects on cholesterol absorption and excretion (Schneider *et al*. 2000), the rapid conversion of 18:0 to 18:1*n*-9 (Bonanome & Grundy, 1988; Emken *et al*. 1993) or a result of its reduced bioavailability. These metabolic effects of 18:0 may also influence the postprandial response following an 18:0-rich meal.

Sanders *et al*. (2000) have reported that a randomised 18:0-rich TAG, prepared by inter-esterifying totally-hydrogenated sunflower oil with unhydrogenated sunflower oil, decreases the postprandial increase in plasma TAG and activated factor VII concentrations compared with an 18:1*n*-9-rich TAG (high-18:1*n*-9 sunflower oil). This observation has been confirmed by Tholstrup *et al*. (2001). A subsequent study, using cocoa butter as a source of 18:0-rich TAG, has found that cocoa butter results in a similar postprandial increase in plasma TAG and activated factor VII concentrations compared with an 18:1*n*-9-rich

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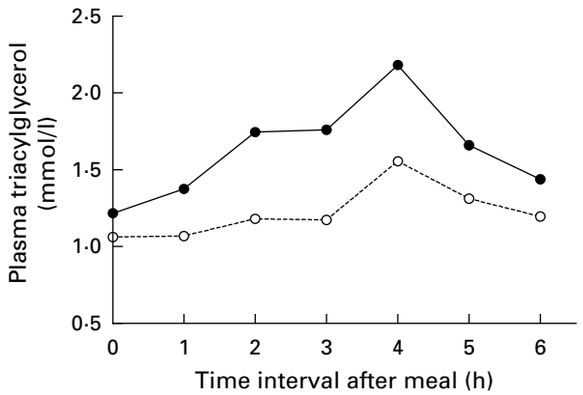

Fig. 3. Plasma triacylglycerol concentrations after 50 g unrandomised cocoa butter (●) or the randomised 18:0-rich fat (randomised inter-esterified hydrogenated sunflower oil with unhydrogenated sunflower oil) used in the study of Sanders *et al.* (2000), containing 43 mol 18:0 in the *sn*-2 position/100 mol (○), in six male subjects (Berry & Sanders, 2003*b*). Values are geometric means for six subjects. The diet × time interaction ($P=0{\cdot}033$) and the time effect ($P<0{\cdot}001$) were significant (repeated-measures ANOVA).

TAG (high-18:1*n*-9 sunflower oil), but SALATRIM (a synthetic randomised 18:0-rich TAG) has a neutral effect (Sanders *et al.* 2001). It has been suggested that the position of the fatty acids on the glycerol backbone may affect the level of postprandial lipaemia. However, SALATRIM also contains the fatty acids butyric acid (4:0) and 2:0, which could provide an alternative explanation. To investigate this result further, a series of studies have been undertaken to compare the effect of randomised *v.* unrandomised 18:0-rich fats on postprandial lipaemia in healthy male subjects.

A randomised crossover trial with seventeen male subjects aged 20–60 years compared the effects of meals containing 50 g fat provided as native unrandomised cocoa butter or as randomised cocoa butter (Sanders *et al.* 2003*a*). Cocoa butter, although of vegetable origin, is rich in SFA, i.e. 16:0 (24–27 g/100 g) and 18:0 (32–36 g/100 g) that are primarily in the *sn*-1 and *sn*-3 position, as well as 18:1*n*-9 (33–37 g/100 g) that is primarily in the *sn*-2 position (Bracco, 1994). Randomisation of cocoa butter increases the proportion of 18:0 in the *sn*-2 position from 4 mol/100 mol to 37 mol/100 mol, and decreases the postprandial area under the curve for plasma TAG by 41% ($P<0{\cdot}01$). Activated factor VII levels are not increased at 6 h postprandially following the randomised cocoa butter, but are increased following the unrandomised cocoa butter.

To ascertain whether there is a difference between cocoa butter and a randomised 18:0-rich fat, a comparison has been made between 50 g cocoa butter and 50 g randomised 18:0-rich fat (randomised inter-esterified hydrogenated sunflower oil with unhydrogenated sunflower oil used in the study of Sanders *et al.* (2000), containing 43 mol 18:0/100 mol in the *sn*-2 position) in six male subjects (Berry & Sanders, 2003*b*). The randomised inter-esterified hydrogenated sunflower oil with unhydrogenated sunflower oil was found to result in a markedly lower postprandial lipaemia (Fig. 3) and at 4 h the plasma concentration of 18:0 was observed to be 70% lower than that with cocoa butter.

As cocoa butter contains a high proportion of 16:0, another study has been undertaken to investigate the effect of feeding an 18:0-rich TAG with 18:0 as its only long-chain SFA. The postprandial response to 50 g native unrandomised shea butter (rich in the TAG species 1,3-distearoyl 2-oleoylglycerol, 3·1 mol 18:0/100 mol in *sn*-2 position) was compared with that of randomised shea butter (22·8 mol 18:0/100 mol in *sn*-2 position), in sixteen healthy male subjects (SE Berry and TA Sanders, unpublished results). Contrary to the observation with cocoa butter, the postprandial lipaemia for the two fats was not different. To investigate this finding further the postprandial response to 50 g high-18:1*n-9* sunflower oil, previously found to produce a similar level of lipaemia to that of cocoa butter (Sanders *et al.* 2001), has been compared with that of unrandomised shea butter in thirteen healthy male subjects (Berry & Sanders, 2003*a*). The postprandial TAG response was found to be markedly lower after consumption of unrandomised shea butter than after the high 18:1*n-9* sunflower oil, which suggests that shea butter, despite having a similar TAG structure to cocoa butter, produces a reduced postprandial lipaemia. Consequently, an alternative explanation to TAG structure is needed for the lower postprandial lipaemic response to shea butter. In all these studies the fatty acid in the *sn*-2 position in the dietary TAG was retained in that position when absorbed into the chylomicron TAG.

There are several potential mechanisms by which the randomised 18:0-rich fats and shea butter reduce the postprandial lipaemic response, including an increased rate of clearance of chylomicrons, which occurs with fish oil feeding (Weintraub *et al.* 1988) because of reduced competition with VLDL for chylomicron lipolysis and clearance. However, this explanation is unlikely, as studies measuring lipase activity have not shown an acceleration of lipase activity following SFA. Another possibility is that the physical properties of the fats retard the rate of absorption.

Stearic acid, triacylglycerol structure and digestion

Early reports suggested that 18:0 is poorly absorbed in both animal and man, and that its digestibility is greater as a mixed glycerol than as tristearin (Mattil & Higgins, 1945; Mattson, 1959). However, more recent human studies have reported that 18:0-rich fats are generally well absorbed (>90%), especially when fed with other dietary fats; for example, cocoa butter is relatively well absorbed, with digestibility values of 89–99% (Mitchell *et al.* 1989; Denke & Grundy, 1991; Shahkhalili *et al.* 2000). More variable results have, however, been obtained with randomised fats rich in 18:0. Bononome & Grundy (1988) have found that when human subjects are fed liquid diets rich in 18:0 (47 g 18:0/d, with 18:0 randomly distributed) 18:0 was well absorbed (97%). However, the synthetic randomised 18:0-rich fat SALATRIM (34 g 18:0/d) has been found to result in a low fat digestibility and a high 18:0 excretion of 7–12 g/d (Finley *et al.* 1994). Snook *et al.* (1999) have reported increased faecal

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excretion of 18:0 (3·9 g/d) following the consumption of a randomised 18:0-rich fat (28 g 18:0/d), and Dougherty *et al*. (1995) have reported lower fat digestibility following consumption of a diet high in native shea butter (25 g 18:0/d) compared with a low-18:0 diet. In contrast, the digestibility of liquid oils is >95%. The digestibility of randomised and unrandomised shea butter has been estimated (SE Berry, L Chen and TA Sanders, unpublished results) in a randomised crossover design study in which sixteen healthy male subjects consumed 30 g test fat (15 g 18:0; either as native unrandomised shea butter or randomised shea butter), in the form of two muffins, daily for 21 d. Faecal collections (3 d) were made on the last 3 d (days 18–21) of the two treatment periods. With the unrandomised shea butter virtually all the 18:0 was in *sn*-1 and *sn*-3 position, whereas in the randomised shea butter 23 mol 18:0/100 mol was in the *sn*-2 position. Total fat excretion was found to be 3·9 and 3·5 g/d, and 18:0 excretion 2·1 and 1·9 g/d following the randomised and unrandomised shea butter respectively. The amount of 18:0 in the faecal fat was found to be approximately 50 g/100 g total fatty acids following both test fats, with the second-most-abundant fatty acid being 16:0 (12–16 g/100 g total fatty acids). No differences were found in the total amount of fat excreted or the fatty acid composition of the faeces between the randomised shea and unrandomised shea, suggesting that the positional composition does not affect digestibility of 18:0-rich fats when fed in moderate amounts. Furthermore, the level of faecal fat excretion was observed to be inside the normal range, suggesting that both forms of shea butter are well digested. These results are in line with those reported by Shahkhalili *et al*. (2000) for cocoa butter.

Physical properties of stearic acid-rich fats

Melting point is considered to have an important influence on fat digestibility (Langworthy, 1923), because fats that are crystalline solids at body temperature form micelles less readily and the rate of micelle formation is a critical step in determining the rate of lipolysis. The differences in postprandial responses reported following 18:0- and 16:0-rich TAG may be a consequence of differences in the melting properties of the test fats. The melting points of the major TAG of 18:0-rich fats are shown in Table 2. Whilst the melting points (°C) of TAG with 18:0 or 16:0 in the *sn*-2 or *sn*-1, -3 positions are similar (1, 3-distearoyl 2-oleoylglycerol 41·6, 1, 2-distearoyl 3-oleoylglycerol 41·6, 1-stearoyl 2-palmitoyl 3-oleoylglycerol 40·5, 1-stearoyl 2-oleoyl 3-palmitoylglycerol 37·5), the melting points of trisaturated TAG are well above body temperature (63–73). During the process of random inter-esterification fatty acids are randomly redistributed on all three positions of the glycerol backbone, generating a fat with a variety of trisaturated, monounsaturated and diunsaturated TAG. Randomisation of shea butter or cocoa butter increases the proportions of trisaturated TAG, which have a high melting point. In addition to the presence of high-melting-point TAG, there may also be effects of the fat matrix in which the TAG are embedded.

Table 2. Melting temperatures of some triacylglycerols (adapted from Small, 1991)

Triacylglycerol	Melting temperature (°C)
Trisaturated:	
SSS	73·1
PPP	66·4
SSP	65·2
SPP	62·7
SPS	68·5
PSP	68·6
Monounsaturated:	
SOS	41·6
SOP	37·5–38
POP	35·2
SSO	41·6
SPO	40·5–41
PSO	41–41·5
PPL	36–38
SSL	36–38
Diunsaturated:	
OPO	22·0
OSO	25·2
OOP	18·2
OOS	24·0
Triunsaturated:	
OOO	5·5
LLL	–13·1

S, stearic acid; P, palmitic acid; O, oleic acid; L, linoleic acid.

Fats usually contain a variety of TAG, each with their own individual melting points, and thus do not have a sharp melting point but melt over a temperature range, so a typical fat may contain TAG with melting points from –50°C to ≥70°C. Thus, simple methods, such as slip point, that give one melting point are inadequate for the determination of the melting characteristics of a fat mixture. An additional complication is the ability of long-chain SFA and their TAG to exist in more than one crystal form, which results in different patterns of molecular packing in the crystals. The 18:0-rich TAG may occur in three main polymorphic forms, α, β′ and β, which have increasing stability and melting point, and differ in their hydrocarbon chain packing. When fats are cooled crystals of a lower melting form may be produced, which may change slowly or rapidly into a more stable form. When a fat is crystallised in an unstable form and heated to a temperature slightly above its melting point it may re-solidify into a more stable form. Thus, polymorphism results in the phenomenon of multiple melting points.

The organoleptic properties of cocoa butter and cocoa-butter substitutes have been extensively studied in order to better understand their melting characteristics, which give chocolate its unique mouth-feel (Timms, 1984). The two main techniques used are differential scanning calorimetry (McFarlane, 1994) and low-resolution pulsed NMR analysis (Talbot, 1994). Differential scanning calorimetry measures the melting profile of a fat, and is based on the principle that the transformation from the liquid to the solid state is accompanied by the release of heat (latent heat of crystallisation); the reverse, the transformation from a solid to liquid, is accompanied by a negative heat

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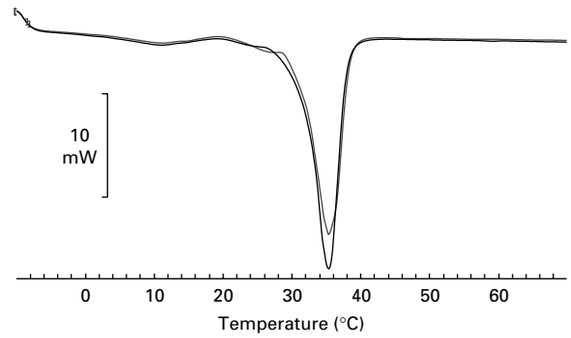

Fig. 4. Melting profile of unrandomised cocoa butter determined using differential scanning calorimetry.

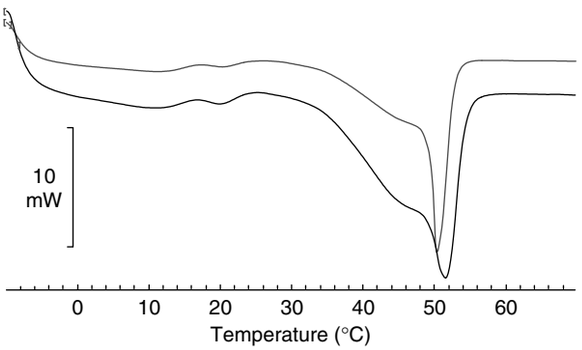

Fig. 5. Melting profile of randomised cocoa butter determined using differential scanning calorimetry.

effect (endothermic reaction). Determination of solid fat content by NMR analysis is based on the principle that protons in a solid state of a fat behave differently from those in the liquid state when they are subjected to radiofrequency energy in a sample contained in a magnetic field. Both these methods have been applied to the fats used in the authors' experimental studies, and large differences have been found in the melting profiles of the randomised and unrandomised fats, with the randomised fats eliciting higher peak melting temperatures (R Dhami, personal communication). This finding is illustrated by differential scanning calorimetry analysis of unrandomised and randomised cocoa butter (Figs. 4 and 5). Unrandomised cocoa butter shows a single sharp melting peak at approximately 37°C, whereas the randomised cocoa butter shows a broad melting peak with the highest melting point at approximately 50°C.

Measurement of the solid fat content of test fats at different temperatures using low-resolution NMR (Fig. 6) reveals an association with the postprandial TAG response. The randomised 18:0-rich fats (randomised cocoa butter and randomised inter-esterified hydrogenated sunflower oil with unhydrogenated sunflower oil), which are found to reduce the postprandial response when compared with unrandomised cocoa butter, have a high solid fat content at 37°C (37·4 and 23·3 g/100 g respectively), whilst unrandomised cocoa butter has almost no solid fat (<1 g/100 g). However, unrandomised shea butter, despite having a

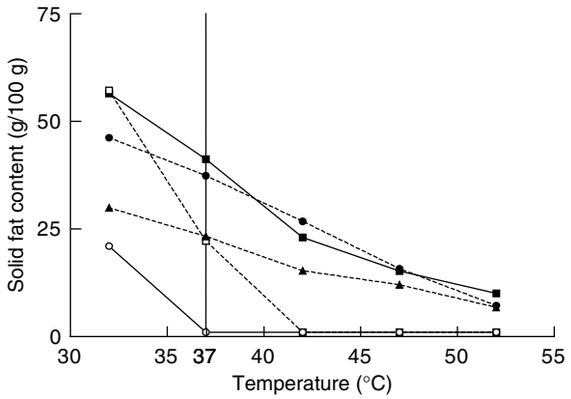

Fig. 6. Solid fat content of stearic acid-rich fats measured at different temperatures using low-resolution NMR. (○–○), Cocoa butter; (●---●), randomised cocoa butter; (▲---▲), randomised inter-esterified hydrogenated sunflower oil with unhydrogenated sunflower oil, used in the study of Sanders *et al.* (2000), containing 43 mol 18:0 in the *sn*-2 position/100 mol; (□---□), shea butter; (■---■), randomised shea butter.

similar TAG structure to unrandomised cocoa butter, has markedly different physical characteristics, with 22·2 g solid fat/100 g at 37°C. The proportion of solid fat at 37°C is therefore associated with the extent of postprandial lipaemia.

Conclusion

The results indicate that the lower postprandial lipaemia associated with the consumption of randomised 18:0-rich fats is likely to be a consequence of the higher solid fat content at 37°C, which appears to retard the absorption of fat. These findings have broader public health implications, because the food industry is using randomisation to harden fats as an alternative to partial hydrogenation, a process that results in the formation of *trans*-fatty acids that are linked to increased risk of CHD. The findings also show that randomisation of 18:0-rich fats has favourable effects on postprandial lipaemia and activation of factor VII. However, further long-term studies are required to ensure that a higher proportion of SFA in the *sn*-2 position in chylomicron TAG does not have adverse effects on lipid metabolism and cardiovascular risk.

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