SATURATED FATTY ACIDS
A new paradigm is necessary

Nutrients before all....

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AGROCAMPUS - INRA, Rennes, FRANCE

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No conflict of interest for this conference
SATURATED FATTY ACIDS

- We eat them but we make «them» (one)

- Metabolism

- Important specific functions

- Problems with CVD and MS biomarkers
Saturated fatty acids

« We do synthesize them »: (human, animals, plants…)
In addition, mammary gland synthesizes the short and middle chain saturates (C4-C10), plus lauric (C12) and myristic acid (C14).
SATURATED FATTY ACIDS

- We eat them but we make them

- **Metabolism**

- Important specific functions

- Problems with CVD and MS biomarkers
Comparative absorption of saturated fatty acids

Intestine

Long chain fatty acids → Chylomicrons → Lymph → Circulation

Short and middle chain fatty acids → Portal vein → Liver

CATABOLISM (β-oxidation)

Adipose

Muscle

STORAGE
Myristic / Palmitic acid metabolism

C14:0

- Uptake
- Incorporation into lipids
- β-oxidation
- Elongation
- Desaturation
- Acylation of proteins (myristoylation)
- Gene regulation?

Secretion

HEPATOCYTE

Rioux et al. (2000), Rioux et al. (2002), Legrand et al. (2002), Rioux et al. (2003), Jan et al. (2004),
Rioux et al. (2005), Rioux et al. (2006), Rioux et al. (2007), Rioux et al. (2008),
METABOLIC DIFFERENCES

- Short and middle chain SFA have a specific and « safe » metabolism,
- Myristic acid and palmitic acid have not the same metabolic fate in the cell:
  - Myristic acid is rapidly β-oxidized, weakly secreted in the form of TG-VLDL, but strongly elongated into palmitic acid. No accumulation!
  - Palmitic acid is stored and secreted in the form of TG, weakly elongated into stearic acid. Also main product of de novo lipogenesis, it accumulates in the cell! « Sugaric acid ? »
- Stearic acid is less synthesized (than palmitic), actively desaturated into oleic acid. No accumulation! « Pre-Oleic acid ? »

Rioux et al. (2000), Rioux et al. (2002), Legrand et al. (2002), Rioux et al. (2003), Jan et al. (2004), Rioux et al. (2005), Rioux et al. (2006), Rioux et al. (2007), Rioux et al. (2008), Beauchamps et al., 2009
SATURATED FATTY ACIDS

- We eat them but we make them
- Metabolism
- Important specific functions
- Problems with CVD and MS biomarkers
Saturated fatty acids functions at a glance
(in addition to energetical function)

- fuel for intestinal mucosa and microbiota
- Immunostimulation
- Cellular differentiation and inhibition of tumor proliferation \textit{in vivo and in vitro} (Induction of apoptosis)
Saturated fatty acids functions at a glance
(in addition to energetical function)

C4 butyric
C6 caproïc
C8 caprylic
C10 capric
C12 lauric
C14 myristic
C16 palmitic
C18 stearic
C20 arachidic
C22 behenic
C24 lignoceric

- Less fat deposition
- C8 VLDL secretion (inhibition of apo B synthesis)
- Hypocholesterolemic effect (C8, C10)
- Ghrelin acylation (C8)
- Antifungal and antiviral properties
<table>
<thead>
<tr>
<th>Saturated fatty acids functions at a glance</th>
</tr>
</thead>
<tbody>
<tr>
<td>(in addition to energetical function)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Chain Length</th>
<th>Fatty Acid</th>
<th>Functions</th>
</tr>
</thead>
<tbody>
<tr>
<td>C4</td>
<td>butyric</td>
<td>- Specific acylation of proteins</td>
</tr>
<tr>
<td>C6</td>
<td>caproïc</td>
<td>- Activation of conversion from C18:3 n-3 towards EPA + DHA</td>
</tr>
<tr>
<td>C8</td>
<td>caprylic</td>
<td>- Activation of sphingolipids synthesis</td>
</tr>
<tr>
<td>C10</td>
<td>capric</td>
<td></td>
</tr>
<tr>
<td>C12</td>
<td>lauric</td>
<td></td>
</tr>
<tr>
<td>C14</td>
<td>myristic</td>
<td>- Specific acylation of proteins</td>
</tr>
<tr>
<td>C16</td>
<td>palmitic</td>
<td>- Activation of conversion from C18:3 n-3 towards EPA + DHA</td>
</tr>
<tr>
<td>C18</td>
<td>stearic</td>
<td>- Activation of sphingolipids synthesis</td>
</tr>
<tr>
<td>C20</td>
<td>arachidic</td>
<td></td>
</tr>
<tr>
<td>C22</td>
<td>behenic</td>
<td></td>
</tr>
<tr>
<td>C24</td>
<td>lignoceric</td>
<td></td>
</tr>
</tbody>
</table>
N-terminal myristoylation

myristoyl-CoA + S-CoA

Protein with an N-terminal glycine

Gly$_1$

AA$_2$-AA$_3$-

myristoylated protein

membrane association

subcellular localization

protein-protein interaction

protein activation
Myristoylation and palmitoylation of the $\alpha$-subunit of heterotrimeric G proteins

N-terminus: G C T L S A E D K A A V E R

myristic acid

palmitic acid

C 16:0
C 14:0

$\alpha$

$\beta\gamma$

GDP

Wedegaertner, 1998
Saturated fatty acids functions at a glance
(in addition to energetical function)

C4    butyric
C6    caproïc
C8    caprylic
C10   capric
C12   lauric
C14   myristic
C16   palmitic
C18   stearic
C20   arachidic
C22   behenic
C24   lignoceric

- Specific acylation of proteins
- Activation of conversion from C18:3 n-3 towards EPA + DHA
- Activation of sphingolipids synthesis
Role of myristic acid on PUFA metabolism (in vitro)

Jan et al., 2004; Beauchamp et al., 2007

NADH-cyt b5 reductase, component of desaturase complex is myristoylated
Effect of myristic acid on PUFAs composition in the rat *in vivo* and in human

**Rioux et al., 2005, Dabadie et al., 2005, Rioux et al., 2008**
Saturated fatty acids functions at a glance
(in addition to energetical function)

<table>
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<tr>
<td>C14</td>
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<tr>
<td>C16</td>
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<tr>
<td>C18</td>
</tr>
<tr>
<td>C20</td>
</tr>
<tr>
<td>C22</td>
</tr>
<tr>
<td>C24</td>
</tr>
</tbody>
</table>

- Specific acylation of proteins
- Activation of conversion from C18:3 n-3 towards EPA + DHA
- Activation of sphingolipids synthesis
Importance of SFA for sphingolipids biosynthesis in animals

Palmitic acid + serine

3-cetosphinganine

Sphinganine

dihydroceramide

De novo biosynthesis

dihydroceramide Δ4-desaturase is myristoylated

Saturated fatty acid

Ceramide

ceramide 1-P

Sphingosine

Sphingosine 1-P

Phosphosphingolipids

Glycosphingolipids

Beauchamp et al., 2007
Saturated fatty acids functions at a glance
(in addition to energetical function)

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C24 lignoceric

- Componant of sphingolipids and phosphoglycerids
- Acylation of some proteins (cell adhesion molecules, receptors, channels……..)
- Lung surfactant (Dipalmitoyl PC)
- Precursor of PEA (palmitoylethanolamide, neuro protective and neuroanti inflammatory effects)
Saturated fatty acids functions at a glance
(in addition to energetical function)

C4  butyric
C6  caproïc
C8  caprylic
C10 capric
C12 lauric
C14 myristic
C16 palmitic  - stearoylation of proteins (transferrine receptor
C18 stearic  - Active desaturation to oleic acid
            - component of phospholipids
C20 arachidic
C22 behenic
C24 lignoceric
Saturated fatty acids functions at a glance
(in addition to energetical function)

C4 butyric
C6 caproïc
C8 caprylic
C10 capric
C12 lauric
C14 myristic
C16 palmitic
C18 stearic
C20 arachidic
C22 behenic
C24 lignoceric

1 / 3 of phospholipids fatty acids : structural role
Saturated fatty acids functions at a glance
(in addition to energetical function)

- Nervous structure (myelinisation)
Saturated fatty acids functions at a glance
(in addition to energetical function)

C4  butyric
C6  caproïc
C8  caprylic
C10 capric
C12 lauric
C14 myristic
C16 palmitic
C18 stearic
C20 arachidic
C22 behenic
C24 lignoceric

SO WHAT?
Nutrients with important functions
SATURATED FATTY ACIDS

- We eat them but we make them
- Metabolism
- Important specific functions
- Problems with CVD and MS biomarkers
RCT with hard endpoints:

Even the most positive (Hooper et al, 2015) : NS on total mortality, CVD mortality, myocardial infraction, non-fatal myocardial infraction, stroke, coronary heart disease events and coronary heart disease mortality (only the composite « combined cvd events »)

RCT with surrogate endpoints:

Mensink et al, 2016 : effect of reduction/replacement on LDL, total, total/HDL..., but no evidence of beneficial effects ? Translation from markers to CVD endpoints ? And SFA/ PUFA without any distinction is not sufficient anymore (omega6)
Observational studies (meta-analysis, cohorts)

Association between SFA and CVD risk:

- Meta-analysis (Siri-Tarino 2010) : 21 cohorts
  "Overall, despite the conventional wisdom that reduced dietary saturated fat intake is beneficial for CVD health, there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD"

- Other meta-analysis: O’Sullivan, 2013; Chowdhury, 2014; Harcomb, 2015, De Souza 2015 same results

- In CAD patients: Puaschitz et al., 2015 No association either
Global risk for SFA

Figure 1: Association between estimated percentage energy from nutrients and total mortality and major cardiovascular disease (n=135,335) Adjusted for age, sex, education, waist-to-hip ratio, smoking, physical activity, diabetes, urban or rural location, centre, geographical regions, and energy intake. Major cardiovascular disease–fatal cardiovascular disease+myocardial infarction+stroke+heart failure.
Saturated fatty acids
General Problems?

- C4 butyric
- C6 caproïc
- C8 caprylic
- C10 capric
- C12 lauric
- C14 myristic
- C16 palmitic
- C18 stearic
- C20 arachidic
- C22 behenic
- C24 lignoceric

Problem with the CVD risk:
Deleterious effects … in case of excess
Accumulation of palmitic acid: endogenous + exogenous origins

No problem with the CVD risk! Praagman 2016, Mensink 2003…

No problem with the CVD risk!
- No reason for considering SFA “en bloc” anymore, in term of structure and metabolism, in term of functions and in term of deleterious effect as well.

- Absence of evidence for specific deleterious effects, need of more precise epidemiological studies (different saturated fatty acids, dose-effects approach, controls…) for the deleterious effects in case of excess

- Intervention studies with substitutions for oleic or n-6 polyunsaturated : not beneficial, except substitution for n-3 polyunsaturated
Plasma inflammatory markers

Linoleic acid (C18:2n-6): Physiopathological approach of excess

LA enrichment leads to increased inflammatory markers

( Marchix et al., 2015, J. Nutr Biochem.)
Linoleic acid (C18:2n-6): Physiopathological approach of excess

Expression of aortic NF-κB

CTL: 12% MUFA-2%LA
LA: 7% MUFA-7%LA
SFA: 12% SFA-2% LA

NF-κB: nuclear factor –kappa B.
CONCLUSION – SUMMARY

- No reason for considering SFA “en bloc” anymore, in term of structure and metabolism, in term of functions and in term of deleterious effect as well.

- Absence of evidence for specific deleterious effects, need of more precise epidemiological studies (different saturated fatty acids, dose-effects approach, controls…) for the deleterious effects in case of excess

- Intervention studies with substitutions for oleic or n-6 polyunsaturated: not beneficial, except substitution for n-3 polyunsaturated

- Intervention studies with substitutions for carbohydrates (sugars starch…) : not beneficial and maybe worse except complex carbohydrates (fibers)

Time for new paradigm and “up to date” recommendations without caricatural old statements of toxicity or eviction........
New paradigm for SFA guidelines
Let’s also speak physiology and nutrition and not only statistics and epidemiology

C4 butyric + fibers, probiotics……

C6 caproïc
C8 caprylic
C10 capric
C12 lauric
C14 myristic + omega 3

Problem with the CVD risk ?
Only one !…..in case of excess

C16 Palmitic (sugaric ?)
+ Fructose, glucose, alcool….
+ Total energy….
+ Linoleic ?

Need of a balanced and synergic reco for palmitic
……….. So, we tried it in France throw the recommendaations of the food safety agency (ANSES)
## ANC : NON ESSENTIAL FA : SATURATED FA

For an adult at 2000-2200 kcal/day  
Values expressed in % total energy.

<table>
<thead>
<tr>
<th>NON ESSENTIAL FA</th>
<th>Minimal physiological requirement</th>
<th>Metabolic syndrome, diabetes, obesity</th>
<th>Cardio-vascular diseases</th>
<th>Cancers : breast, colon</th>
<th>Neuro-psychiatric pathologies</th>
<th>Other pathologies : Macular degeneration</th>
<th>ANC 2010</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lauric acid (C12:0) + myristic acid (C14:0) + palmitic acid (C16:0)</td>
<td>-</td>
<td>-</td>
<td>≤ 8</td>
<td>_</td>
<td>-</td>
<td>-</td>
<td>≤ 8</td>
</tr>
<tr>
<td>Total Saturated FA</td>
<td>-</td>
<td>-</td>
<td>≤ 12</td>
<td>≤ 12</td>
<td>-</td>
<td>-</td>
<td>≤ 12</td>
</tr>
<tr>
<td>Oleic acid C18:1 n-9</td>
<td>-</td>
<td>-</td>
<td>≤ 20</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>15 - 20</td>
</tr>
<tr>
<td>Others non essential FA</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
Thanks for your attention
Les lipides laitiers augmentent le statut en DHA des rétines et du cerveau

DHA

Rétines

Cerveau

Acuité visuelle et
Neuro-développement

% des AGT des phospholipides

MGV MGL MGV+DHA MGL+DHA

MGV MGL MGV+DHA MGL+DHA

ALA C18:3 \(\Delta_6\) STA C18:4 \(\epsilon_5\) C20:4 \(\Delta_5\) EPA C20:5 \(\epsilon_2-5\) DPA n-3 C22:5 \(\epsilon_2\) C24:5 \(\Delta_6\) C24:6 \(\beta-ox\) DHA C22:6
# Recommendations for lipids and saturated fat

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Year</th>
<th>Lipids</th>
<th>SFA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canadian Dietary reference intakes (DRI)</td>
<td>2010</td>
<td>20-35% En</td>
<td>As low as possible</td>
</tr>
<tr>
<td>Dietary Guidelines for Americans (DGAC/USDA)</td>
<td>2010</td>
<td>20-35% En</td>
<td>&lt; 10% En</td>
</tr>
<tr>
<td>AHA/ACC Lifestyle Management Guideline</td>
<td>2013</td>
<td>none</td>
<td>&lt; 7% En</td>
</tr>
<tr>
<td>European Food Safety Agency (EFSA)</td>
<td>2010</td>
<td>20-35% En</td>
<td>As low as possible</td>
</tr>
<tr>
<td>World Health Organization (WHO)</td>
<td>2008</td>
<td>15-35% En</td>
<td>&lt; 10% En</td>
</tr>
<tr>
<td>ANSES, France</td>
<td>2011</td>
<td>35-40% En</td>
<td>&lt;12% (&lt;8%) En</td>
</tr>
</tbody>
</table>

4. EFSA Journal 8: 1461-1568.
Predicted changes (Δ) in the ratio of serum total to HDL cholesterol and in LDL- and HDL-cholesterol concentrations when carbohydrates constituting 1% of energy are replaced isoenergetically with lauric acid (12:0), myristic acid (14:0), palmitic acid (16:0...)

Individual saturated fatty acids

Lauric acid markedly increases cholesterol, whereas stearic acid lowers it somewhat when it is used to replace carbohydrates. However, the picture reverses if one looks at total:HDL cholesterol: both lauric and stearic acid are now more favorable than carbohydrates. Lauric acid—a major component of tropical oils such as coconut and palm kernel fat—has the largest cholesterol-raising effect of all fatty acids, but much of this is due to HDL cholesterol. As a result, lauric acid had a more favorable effect on total:HDL cholesterol than any other fatty acid, either saturated or unsaturated.
### Saturates and PUFAs bioavailability

<table>
<thead>
<tr>
<th></th>
<th>MY0</th>
<th>MY5</th>
<th>MY10</th>
<th>MY20</th>
<th>MY30</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>C14:0</strong></td>
<td>0.02</td>
<td>5.09</td>
<td>9.81</td>
<td>19.41</td>
<td>29.57</td>
</tr>
<tr>
<td><strong>C16:0</strong></td>
<td>9.25</td>
<td>14.65</td>
<td>14.83</td>
<td>15.29</td>
<td>15.51</td>
</tr>
<tr>
<td><strong>C18:0</strong></td>
<td>3.35</td>
<td>4.87</td>
<td>4.78</td>
<td>3.84</td>
<td>3.01</td>
</tr>
<tr>
<td>AG Saturés</td>
<td>13.26</td>
<td>32.69</td>
<td>38.17</td>
<td>45.04</td>
<td>52.52</td>
</tr>
<tr>
<td><strong>C 18:1 n-9</strong></td>
<td>76.13</td>
<td>51.94</td>
<td>46.34</td>
<td>41.33</td>
<td>35.47</td>
</tr>
<tr>
<td><strong>C 18:2 n-6</strong></td>
<td>6.57</td>
<td>7.04</td>
<td>7.00</td>
<td>7.03</td>
<td>6.91</td>
</tr>
<tr>
<td><strong>C 18:3 n-3</strong></td>
<td>1.32</td>
<td>1.38</td>
<td>1.40</td>
<td>1.39</td>
<td>1.38</td>
</tr>
</tbody>
</table>

*Between 2.2% and 6.6% myristic acid (energie) (trimyristin)*

Rioux et al. (2008) Animal 2, 636-644
Saturates and PUFAs bioavailability

<table>
<thead>
<tr>
<th>% AG</th>
<th>OL</th>
<th>BF</th>
<th>SAT</th>
<th>TMY</th>
</tr>
</thead>
<tbody>
<tr>
<td>C4:0-C10:0</td>
<td>8,93</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C12:0</td>
<td>2,62</td>
<td>2,83</td>
<td></td>
<td>0,11</td>
</tr>
<tr>
<td>C14:0</td>
<td>0,02</td>
<td>9,83</td>
<td>9,71</td>
<td>9,75</td>
</tr>
<tr>
<td>C16:0</td>
<td>7,83</td>
<td>15,27</td>
<td>16,02</td>
<td>7,51</td>
</tr>
<tr>
<td>C18:0</td>
<td>3,06</td>
<td>4,82</td>
<td>2,52</td>
<td>2,68</td>
</tr>
<tr>
<td>AG Saturés</td>
<td>11,54</td>
<td>43,83</td>
<td>31,75</td>
<td>20,83</td>
</tr>
<tr>
<td>C 18:1 n-9</td>
<td>71,05</td>
<td>33,22</td>
<td>51,28</td>
<td>62,26</td>
</tr>
<tr>
<td>C 18:2 n-6</td>
<td>11,82</td>
<td>12,55</td>
<td>11,99</td>
<td>11,69</td>
</tr>
<tr>
<td>C 18:3 n-3</td>
<td>2,49</td>
<td>2,61</td>
<td>2,53</td>
<td>2,53</td>
</tr>
</tbody>
</table>

Butterfat: myristic acid + short and middle chain FA +……..

Dairy fat: myristic acid + short and middle chain FA +……..

Legrand et al. (2010) Lipids 45, 975-986
# Saturates and PUFAs bioavailability

## 4 isocaloric and isolipidic diets

<table>
<thead>
<tr>
<th></th>
<th>Olive oil</th>
<th>Olive oil + SFAs</th>
<th>Fractionated butter</th>
<th>butter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Olive oil</td>
<td>76.0</td>
<td>48.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Canola oil</td>
<td>19.0</td>
<td>28.0</td>
<td>18.0</td>
<td>27.0</td>
</tr>
<tr>
<td>Corn oil</td>
<td>5.0</td>
<td>6.4</td>
<td>12.0</td>
<td>11.6</td>
</tr>
<tr>
<td>Fractionated butter</td>
<td></td>
<td></td>
<td>66.5</td>
<td></td>
</tr>
<tr>
<td>Butter</td>
<td></td>
<td></td>
<td></td>
<td>61.4</td>
</tr>
<tr>
<td>Trilaurin (C12:0)</td>
<td>2.0</td>
<td>3.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trimiystrin (C14:0)</td>
<td>4.9</td>
<td>3.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tripalmitin (C16:0)</td>
<td>8.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tristearin (C18:0)</td>
<td>1.6</td>
<td></td>
<td></td>
<td></td>
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</table>

(% lipidic mix)

<table>
<thead>
<tr>
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<th>Fractionated butter</th>
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<tbody>
<tr>
<td>C4:0</td>
<td></td>
<td></td>
<td></td>
<td>3.6</td>
</tr>
<tr>
<td>C6:0</td>
<td></td>
<td></td>
<td></td>
<td>1.9</td>
</tr>
<tr>
<td>C8:0</td>
<td></td>
<td></td>
<td></td>
<td>1.1</td>
</tr>
<tr>
<td>C10:0</td>
<td></td>
<td></td>
<td></td>
<td>2.4</td>
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<td></td>
<td>1.9</td>
</tr>
<tr>
<td>C14:0</td>
<td></td>
<td></td>
<td></td>
<td>9.8</td>
</tr>
<tr>
<td>C16:0</td>
<td>7.8</td>
<td>15.0</td>
<td>15.3</td>
<td>18.5</td>
</tr>
<tr>
<td>C18:0</td>
<td>3.1</td>
<td>3.8</td>
<td>4.8</td>
<td>6.7</td>
</tr>
<tr>
<td>Σ AGS</td>
<td>11.5</td>
<td>26.2</td>
<td>43.8</td>
<td>42.2</td>
</tr>
<tr>
<td>C18:1 n-9</td>
<td>71.1</td>
<td>56.5</td>
<td>33.2</td>
<td>33.3</td>
</tr>
<tr>
<td>Σ AGMI</td>
<td>74.2</td>
<td>59.4</td>
<td>40.0</td>
<td>39.2</td>
</tr>
<tr>
<td>C18:2 n-6</td>
<td>11.8</td>
<td>11.5</td>
<td>12.6</td>
<td>14.9</td>
</tr>
<tr>
<td>Σ AGPI n-6</td>
<td>11.8</td>
<td>11.5</td>
<td>13.5</td>
<td>15.2</td>
</tr>
<tr>
<td>C18:3 n-3</td>
<td>2.5</td>
<td>2.9</td>
<td>2.6</td>
<td>3.4</td>
</tr>
<tr>
<td>Σ AGPI n-3</td>
<td>2.5</td>
<td>2.9</td>
<td>2.7</td>
<td>3.4</td>
</tr>
<tr>
<td>C18:2 / C18:3</td>
<td>4.7</td>
<td>4.0</td>
<td>4.8</td>
<td>4.4</td>
</tr>
</tbody>
</table>

(% Total FA)

Thèse d'Hélène Ezanno (2012)
Saturates and PUFAs bioavailability

C18:3 n-3

Adipose Tissu

Liver

Plasma

Butter

Régimes

Δ6


C18:3 n-3 (% AGT)

a, b

Régimes

Δ6


C18:3 n-3 (% AGT)

a, b

Régimes

Δ6


C18:3 n-3 (% AGT)

a, b

Régimes

Δ6


C18:3 n-3 (% AGT)

a, b

Régimes

Δ6


C18:3 n-3 (% AGT)

a, b

Régimes

Δ6


C18:3 n-3 (% AGT)
LC-PUFAs n-3

Saturates and PUFAs bioavailability

\[ 18:3 \xrightarrow{\Delta 6} 18:4 \xrightarrow{\epsilon} 20:4 \xrightarrow{\Delta 5} 20:5 \xrightarrow{\epsilon} 22:5 \xrightarrow{\epsilon} 24:5 \xrightarrow{\Delta 6} 24:6 \xrightarrow{\beta} 22:6 \] n-3

Liver

Adipose tissue

Butter

AGPI n-3 (% AGT)

EPA

DPA

DHA

HO HOS BF BN

HO HOS BF BN

HO HOS BF BN

-SATURATED FATTY ACIDS (C12, C14, C16)

- Increase VLDL secretion by inhibition of apo-B dégradation (Kummrow et al, 2002)

INCREASE OF LDL-CHOLESTEROL (dose-dependent), be carefull about small and dense LDL (Katan 1995)
Free Fatty Acids
C14:0 ?
C16:0 ?
C18:0 ?
Trans ?
Unsaturates ?

ROS
O$_2^-$
Superoxide

Endothelial Progenitor Cells
EPCs

Endothelial Cells
Strivastava, 2006

Apoptosis
Jiang, 2010

Vascular permeability

Plaque erosion
Rössig, 2001
Choy, 2001

Plaque rupture

Oestradiol
Gong, 2003

Myristoylation
Busconi, 1993
Shaul, 1996

Myristic acid
C14:0

CD36
Isenberg, 2007

e NO synthase
Moers, 1997
Artwohl, 2008

No data really specific to SFA and or to dietary SFA (meaning fructose/glucose load may be worse)