

## Feature

# The twisted tale of saturated fat

Jeff S. Volek<sup>1</sup>, Brittanie M. Volk<sup>1</sup>, Stephen D. Phinney<sup>2</sup>

<sup>1</sup> Department of Kinesiology, University of Connecticut, Storrs, CT; E-mail: jeff.volek@uconn.edu

<sup>2</sup> School of Medicine (emeritus), University of California, Davis, Davis, CA

### Summary

*Recent research results mandate a careful re-evaluation of the widespread belief that dietary saturated fat is harmful. Specifically, multiple recent reports find no association between dietary saturated fat intakes and cardiovascular disease (CVD). There is, however, a consistent pattern of increased risk for both CVD and type-2 diabetes associated with increased levels of saturated fatty acids (SFA) in circulating lipids. This raises the important question as to what contributes to increased levels of saturated fat in the blood? Whereas dietary intake of saturated fats and serum levels of SFA show virtually no correlation, an increased intake of carbohydrate is associated with higher levels of circulating SFA. This leads to the paradoxical conclusion that dietary saturated fat is not the problem; rather it's the over-consumption of carbohydrate relative to the individual's ability to metabolize glucose without resorting to de novo lipogenesis. From this perspective, insulin resistant states like metabolic syndrome and type-2 diabetes can be viewed as carbohydrate intolerance, in which a high carbohydrate intake translates to increased serum SFA and therefore increased risk.*

### Dietary saturated fat recommendations and unintended consequences

A reduction in the consumption of dietary saturated fat (SFA) has been the focus of nutritional recommendations for decades in the United States and many other countries. The fundamental justification for this dietary recommendation is the diet heart hypothesis. This hypothesis holds that dietary SFA, on average, increases circulating levels of low density lipoprotein cholesterol (LDL-C), which in turn increases cardiovascular risk. In the four decades since the diet heart hypothesis was proposed, it has not been proven. In fact, repeated studies involving the restriction of dietary SFA have failed to decrease risk of heart disease, suggesting (if not proving) that this paradigm has inherent flaws.

According to dietary trends over the past few decades, Americans have actually decreased their consumption of SFA; but at the same time they have replaced those calories with an even greater amount of carbohydrate. Thus our current epidemics of obesity and diabetes have occurred during a time when the population has increased their consumption of carbohydrate, not SFA. A recent meta-analysis showed that this dietary manipulation – switching saturated fat by carbohydrate – significantly increased risk of coronary events [1]. By continuing to promote lower intake of saturated fat, if even more people replace those calories with carbohydrate, this misbegotten campaign to decrease dietary SFA could have the unintended effect of accelerating coronary disease rates.

### What is the association of dietary SFA intake and risk for chronic disease?

Given the clear and consistent message to Americans that saturated fat is 'unhealthy'; you might assume there is a substantial amount of evidence to support such a dietary recommendation. After four decades of research focused on proving the diet heart hypothesis, the empirical basis for targeting saturated fat has

come under intense scrutiny. The massive Women's Health Initiative, which cost over half a billion dollars, showed no significant effect of reducing SFA intake on risk of cardiovascular disease, diabetes or cancer. In addition, three recent meta-analyses of large study populations published since 2009 all came to the same conclusion – that there was no association between SFA intake and incidence of cardiovascular disease [2]. In one of these large studies, however, replacing dietary SFA with carbohydrate significantly increased the risk of coronary events [1]. The bottom line: *recent evidence shows no link between dietary SFA and heart disease.*

### What is the association between serum SFA level and risk for chronic disease?

Although dietary intake of SFA is not associated with cardiovascular disease, this does not rule out a role of serum (i. e., blood) SFA in promoting heart disease. The primary factor determining the amount of SFA in the blood stream is their rate of release from the liver. When the liver is converting carbohydrate into fat (i. e., *de novo* lipogenesis), there is a higher incorporation of SFA into triacylglycerols which are then secreted into the blood. Serum triacylglycerols enriched with SFA are highly correlated with insulin resistance and adiposity. It should come as no surprise then that people with higher levels of serum SFA have increased risk of developing metabolic syndrome [3] and diabetes [4], as well as a significantly greater risk of having heart attack [5] and heart failure [6].

### What is the association between dietary SFA intake and serum SFA levels?

One of the most common nutritional axioms is: 'you are what you eat'. If this were true, it then it follows that any nutrient that causes harm in the body should be avoided. As noted above, however, the amount of SFA in the blood has no relationship with how much of them we eat. For example, King et al. [7]

reported lower serum levels of saturated fat in response to diets that contained 2–3 fold greater intake of saturated fat but were lower in carbohydrate. This inconsistency notwithstanding, this logical fallacy is still used to justify recommendations to restrict saturated fat. In practice, this means foods containing SFA (e.g., beef, dairy and eggs) are discouraged based on theoretical concerns associated with their saturated fat content which are baseless.

### What is the role of dietary carbohydrate in determining serum SFA?

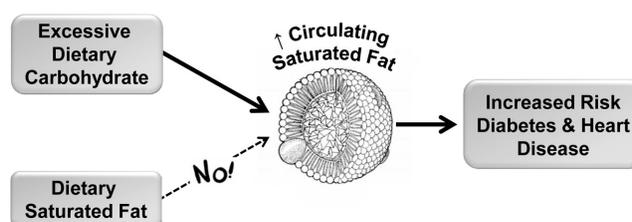
This lack of association between dietary intake and serum levels of saturated fat promoted us to examine SFA levels in people consuming varying levels of carbohydrate. In two recent studies from our laboratory we made the striking observation that dietary carbohydrate exerts a significant regulatory effect on serum saturated fat levels.

In the first study [8], overweight men and women consumed diets for 12 weeks containing ~1500 kcal/day that were either very low carbohydrate or low fat. Despite containing 3-fold higher dietary SFA than the low fat group (36 vs 12 g/day), the low carbohydrate diet group had a significantly greater reduction in the proportionate and absolute amounts of circulating saturated fat.

In the second study [9], we provided all the food to participants at a caloric level to maintain body weight. Despite the fact that there was no weight loss, we confirmed that a very low carbohydrate diet significantly decreased circulating levels of saturated fat despite a relatively high intake of dietary SFA. Thus, from the body's perspective, a low carbohydrate diet reduces circulating saturated fat levels irrespective of dietary saturated fat intake.

These results can best be explained by the metabolic adaptations induced by carbohydrate restriction, notably less stimulation of insulin and greater reliance on fats (including saturated fatty acids) as the body's primary fuel source. Lower insulin levels may have also simultaneously decreased activity of key enzymes in *de novo* lipogenesis which produces saturated fatty acids. In support of this mechanism, in our above two studies, we observed highly uniform reductions in cholesteryl ester palmitoleic acid, a biomarker indicative of increased conversion of dietary carbohydrate to fat, in subjects consuming very low carbohydrate diets [8, 9].

In summary, increased circulating levels of saturated fat are associated with increased risks for insulin resistance, type 2 diabetes, and heart attack. However when it comes to SFA, clearly you are not what you eat. Consumption of carbohydrates at levels that exceed an individual's ability to directly oxidize them contributes to increased circulating saturated fat (Figure 1) by driving *de novo* lipogenesis. Instead of telling everyone to restrict



**Figure 1.** Over consumption of carbohydrate, not saturated fat, drives up circulating levels of saturated fat, which in turn is associated with higher risk of diabetes and cardiovascular disease.

dietary saturated fat, a more rational and effective strategy would be to focus on ways to help people find their 'right' level of carbohydrate. Specifically for people with insulin resistance (i.e., carbohydrate intolerance), a low carbohydrate lifestyle can have a profoundly beneficial effect on a host of metabolic risk factors, including blood SFA. For a more detailed discussion of the clinical aspects of low carbohydrate diets see Volek and Phinney [10].

### References

- [1] Jakobsen MU, O'Reilly EJ, Heitmann BL, Pereira MA, Balter K, Fraser GE, Goldbourt U, Hallmans G, Knekt P, Liu S *et al.*, *Am J Clin Nutr* 2009, 89, 1425–1432.
- [2] Siri-Tarino PW, Sun Q, Hu FB, Krauss RM, *Am J Clin Nutr* 2010, 91, 535–546.
- [3] Warensjo E, Riserus U, Vessby B, *Diabetologia* 2005, 48, 1999–2005.
- [4] Patel PS, Sharp SJ, Jansen E, Luben RN, Khaw KT, Wareham NJ, Forouhi NG, *Am J Clin Nutr* 2010, 92, 1214–1222.
- [5] Wang L, Folsom AR, Eckfeldt JH, *Nutr Metab Cardiovasc Dis* 2003, 13, 256–266.
- [6] Yamagishi K, Iso H, Yatsuya H, Tanabe N, Date C, Kikuchi S, Yamamoto A, Inaba Y, Tamakoshi A., *Am J Clin Nutr* 2010, 92, 759–765.
- [7] King IB, Lemaitre RN, Kestin M, *Am J Clin Nutr* 2006, 83, 227–236.
- [8] Forsythe CE, Phinney SD, Fernandez ML, Quann EE, Wood RJ, Bibus DM, Kraemer WJ, Feinman RD, Volek JS, *Lipids* 2008, 43, 65–77.
- [9] Forsythe CE, Phinney SD, Feinman RD, Volk BM, Freidenreich D, Quann E, Ballard K, Puglisi MJ, Maresh CM, Kraemer WJ *et al.*, *Lipids* 2010, 45, 947–962.
- [10] Volek JS, Phinney SD, *The Art and Science of Low Carbohydrate Living*. Beyond Obesity LLC (2011). <http://www.amazon.com/Art-Science-Low-Carbohydrate-Living/dp/0983490708/>