Effects of Dietary Fats on Gut Hormones Release and Satiety

Simon Toh Wai Hong*; Voon Phooi Tee* and Kalanithi Nesaretnam*

INTRODUCTION

Obesity is one of the greatest public health challenges of the 21st century. In 2008, the World Health Organisation (WHO) reported that 1.5 billion adults globally (age ≥ 20 years) were overweight and at least 500 million adults were obese. In addition, WHO forecasted that approximately 2.3 billion adults will be overweight and more than 700 million adults will be obese by 2015 (WHO, 2010). Meanwhile, more than 220 million people worldwide have diabetes (WHO, 2011). Gut hormones release has been shown to play a role in the prevalence of obesity through food intake reduction and appetite satisfaction. Release of gut hormones could be regulated by the consumption of dietary fats. Moreover, gut hormones such as glucagon like-peptide 1 and glucagon insulinotropic dependent polypeptide have been implicated in the treatment of patients with diabetes.

GUT HORMONES

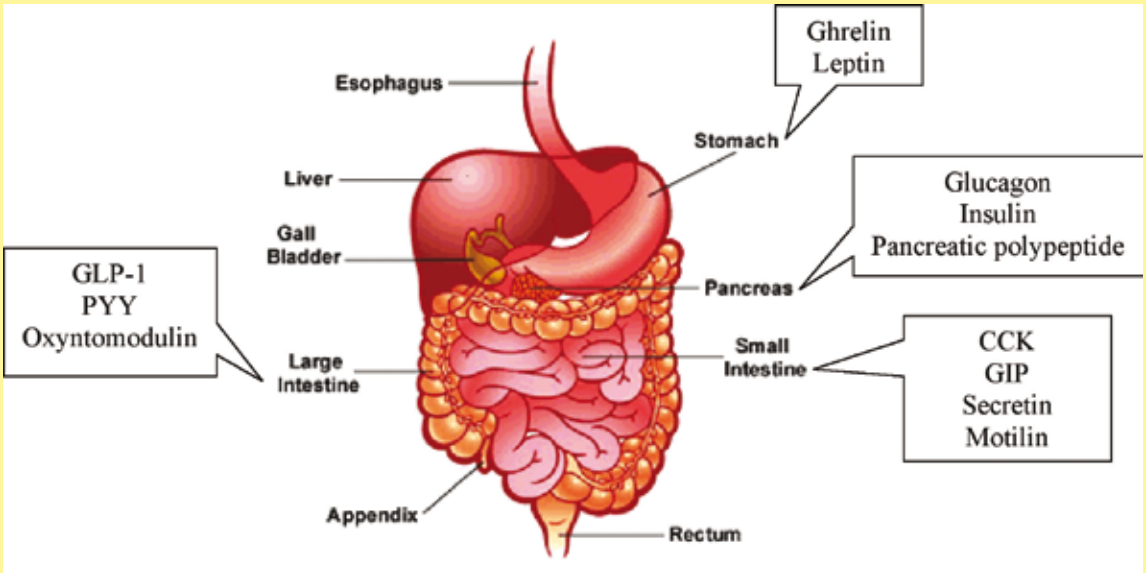
Gut hormones are satiety signals that are released from the gastrointestinal (GI) tract which modulates the activity of appetite centres within the brain. These signals play a fundamental role in regulating food intake and satiety as well as in energy balance. Gut hormones also play an important physiological role in postprandial satiety (fullness after consumption of a meal) and hence, regulate meal initiation and termination. *Figure 1* shows various gut hormones being secreted from different organs in the body.

Gut hormones such as glucose dependent insulinotropic polypeptide (GIP), glucagon-like peptide 1 (GLP-1), cholecystokinin (CCK) and peptide YY (PYY) are released from the small intestine while ghrelin is secreted from the stomach (*Figure 1*). PYY acts on hypothalamus of the brain to send signals that will suppress appetite while CCK sends a satiety signal that will reduce food intake. Meanwhile, secretion of ghrelin will stimulate food intake. *Table 1* shows the effects of various gut hormones on food consumption as well as in postprandial satiety. For an example, when CCK, GLP-1, GIP or PYY are secreted, it will reduce food consumption and appetite, thus increasing fullness. Therefore, secretion of these gut hormones when dietary fats are consumed is directly involved in preventing the development of obesity, satiety and food intake.

CURRENT FINDINGS OF DIETARY FATS AND POSSIBLE ROLE OF TRIACYLGLYCERIDES STRUCTURE ON GUT HORMONES SECRETION

Entry of dietary fat into the small intestine induces the release of gut peptides upon interaction between the gut wall and nutrients. The presence of fat in the small intestine modulates the secretion of gut hormones such as GIP, GLP-1, CCK, PYY and ghrelin, all of which have been implicated in the regula-

* Malaysian Palm Oil Board,
P. O. Box 10620, 50720 Kuala Lumpur,
Malaysia.
E-mail: simontoh15@gmail.com



Source: modified from Cummings and Overduin (2007).

Figure 1. Principal site of secretion of gut hormones.

TABLE 1. EFFECTS OF THE GUT HORMONES SECRETION ON FOOD CONSUMPTION AND POSTPRANDIAL SATIETY

Gut hormones	Food consumption	Satiety (fullness)
Cholecystokinin	Reduce	Increase
Glucagon-like peptide 1	Reduce	Increase
Peptide YY	Reduce	Increase
Glucose dependent insulinotropic polypeptide	Reduce	Increase
Ghrelin	Increase	Reduce

Source: adapted from Maljaars *et al.* (2007).

tion of gastrointestinal function and energy intake (Little *et al.*, 2007).

The degree of fat saturation affects hunger and food intake as well as the secretion of gut hormones. This was shown from studies carried out by several researchers related to the effect of fat saturation on hunger and food intake. Ingestion of unsaturated fats induced a greater reduction in food intake as compared to saturated fats.

Maljaars *et al.* (2009) reported that shea butter with C18:0 did not reduce hunger or fullness whereas unsaturated fats of canola oil and safflower oil did reduce hunger and increase fullness. Moreover, there was no significant difference in food intake between treatments (shea butter; C18:0, canola oil; C18:1; safflower oil; C18:2) given to human volunteers (Maljaars *et al.*, 2009). Macintosh *et al.* (2003) reported that the degree of fat saturation did not alter satiety responses.

On secretion of gut hormones, Maljaars *et al.* (2009) reported that polyunsaturated fatty acids (PUFA) from canola oil with C18:1 and safflower oil with C18:2 have shown that it increased the secretion of plasma CCK while there was no significant effect on secretion on gut hormones shown by saturated fats such as shea butter with C18:0. Furthermore, release of GLP-1 has been shown to increase after a MUFA-rich (olive oil) meal compared with a SFA-rich (coconut oil) meal (Thomsen *et al.*, 1999; 2003; Rocca *et al.*, 2001). Secretion of gut hormones after a SFA-rich meal was secreted to a lesser extent as compared to MUFA and PUFA-rich meal. Therefore, it would be interesting to explore whether palm oil will induce an effect on gut hormone secretion.

Palm oil is mainly used as cooking oil in Southeast Asia countries while its fractions such as palm stearin and palm mid fraction are used in confectionary products

such as instant noodles, bread and cookies as well as chocolates. Interestingly, palm oil has a unique positional distribution in its triacylglycerides (TAG) molecule as shown in *Figure 2*. About 89%-93% palmitic acid occupy the sn-1 and sn-3 positions of the palm triglyceride molecule, while the sn-2 position is occupied by 60%-70% of the monounsaturated oleic acid and 7%-11% of palmitic acid. It is hypothesised that the oleic acid at the sn-2 position will be absorbed into the body and does not alter the blood lipid profile.

Previous studies as described above have shown that the degree of saturation of fat affects satiety and food intake (Thomsen *et al*., 1999; 2003; Rocca *et al.,* 2001; Thomsen *et al*., 2003; Maljaars *et al.*, 2009). However, these studies were not designed to investigate the effects of the positional distribution of TAG (especially in palm oil) on the release of gut hormones as well as in satiety and food intake. Furthermore, it will be interesting to find out whether the degree of saturation of different test oils with different types of fatty acid at the sn-1 and sn-3 positions will affects gut hormones secretion and food intake.

IMPORTANCE OF GUT HORMONES SECRETION IN THE INCIDENCE OF OBESITY AND DIABETES

Gut hormones secretion from various forms of dietary fats has resulted in a great interest on the effects of gut hormones release on food intake, satiety as well as performing as anti-obesity 'tool'. A dietary fat intake will give a greater postprandial satiety (act of fullness), which in turn can facilitate weight loss. This suggests that the subject will get full when he is given an intake of dietary fat and when the subject will be eating less, and thus committing him to weight loss.

GLP-1 has been shown in obese subjects that it was effective in reducing appetite and food intake as well as calorie intake in humans. The GLP-1 interacts with gastric or duodenal satiety signals to increase satiety and reduce food intake. And thus, this contributes in the prevention of obesity. Meanwhile, Karra and Batterham (2010) reported that PYY acts as an anorectic (loss of appetite) effect in obese human volunteers.

Both GIP and GLP-1 hormones increase the amount of insulin released from the β cells of pancreas. The secretion of insulin has been implicated in the treatment of type 2 diabetes mellitus, a global disease phenomenon that affects 90% of people and is largely the result of excess body weight and lack of physical activity (WHO, 2009). Insulin facilitates the uptake of glucose by muscle tissue and the liver while simultaneously suppressing glucagon secretion, leading to reduced production of glucose (Drucker, 2007).

Studies have shown that gut hormones secretion induces a lower food intake and increases satiety, and hence this may reduce the percentage of obesity incidence occurrence. Therefore, this has prompted a research to be carried out to study the effects of sn-1 and sn-3 palmitic or stearic acid-rich fats on the release of gut hormones which may have a link to anti-obesity incidence.

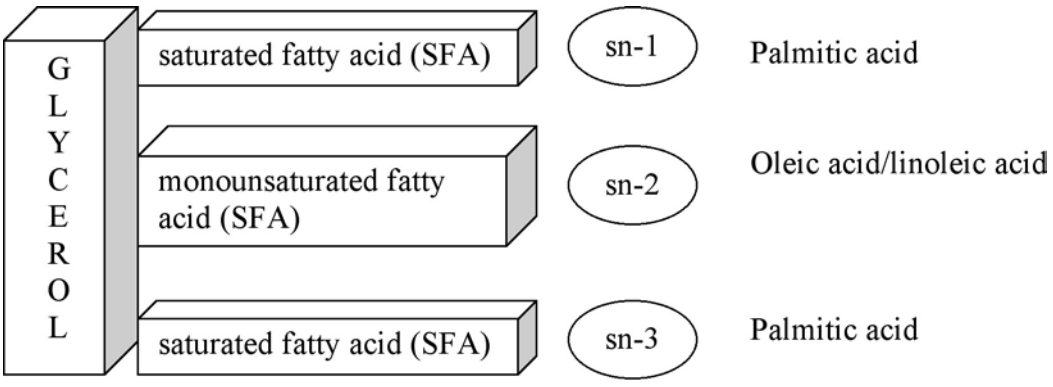


Figure 2. Triacylglycerides (TAG) structure of palm oil.

CONCLUSION

GI tract plays a key role in satiety through various gut hormones being secreted in response to dietary intake of food. Gut hormones established from the consumption of dietary fats consisting of fatty acids have brought upon an additional answer in tackling the question of the 21st century, obesity. It is inevitable that gut hormones plays a major role in the regulation of body weight and represent therapeutic targets for the future treatment of obesity, thus it has been an important area to be explored for palm oil. Further scientific research on palm oil on the effects of gut hormones will thus enhance the value of palm oil.

REFERENCES

CUMMINGS, D E and OVERDUIN, J (2007). Gastrointestinal regulation of food intake. *The Journal of Clinical Investigation, 117*: 13-23.

DRUCKER, D J (2007). The role of gut hormones in glucose homeostasis. *The Journal of Clinical Investigation, 117*: 24-32.

KARRA, E and BATTERHAM, R L (2010). The role of gut hormones in the regulation of body weight and energy homeostasis. *Molecular and Cellular Endocrinology, 316*: 120-128.

LITTLE, T J; HOROWITZ, M and FEINLE-BISSET, C (2007). Modulation by high-fat diets of gastrointestinal function and hormones associated with the regulation of energy intake: implications for the pathophysiology of obesity. *American Journal of Clinical Nutrition, 86*: 531-41.

MACINTOSH, C G; HOLT, S H A and BRAND-MILLER, J C (2003). The degree of fat saturation does not alter glycemic, insulinemic or satiety responses to a starchy staple in healthy men. *The Journal of Nutrition, 133*: 2577-2580.

MALJAARS, J; PETERS, H P F and MASCLEE, A M (2007). Review article: the gastrointestinal tract: neuroendocrine regulation of satiety and food intake. *Alimentary Pharmacology & Therapeutics, 26 (Suppl 2)*: 241-250.

MALJAARS, J; ROMEYN, E A; HADDEMAN, E; PETERS, H P F and MASCLEE, ADAM (2009). Effect of fat saturation on satiety, hormone release, and food intake. *The American Journal of Clinical Nutrition, 89*: 1019-24.

ROCCA, A S; LAGRECA, J; KALITSKY, J and BRUBAKER, P L (2001). Monounsaturated fatty acid diets improve glycemic tolerance through increased secretion of glucagon-like peptide-1. *Endocrinology*, *142*: *3*, 1148-1155.

THOMSEN, C; RASMUSSEN, O; LOUSEN, T; HOLST, J J; FENSELAU, S; SCHREZENMEIR, J and HERMANSEN, K (1999). Differential effects of saturated and monounsaturated fatty acids on postprandial lipemia and incretin responses in healthy subjects. *American Journal of Clinical Nutrition, 69*: 1135-43.

THOMSEN, C; STORM, H; HOLST, J J and HERMANSEN, K (2003). Differential effects of saturated and monounsaturated fats on postprandial lipemia and glucagon-like peptide 1 responses in patients with type 2 diabetes. *American Journal of Clinical Nutrition, 77*: 605-11.

WORLD HEALTH ORGANISATION, OBESITY AND OVERWEIGHT (2008). Available from <http://www.who.int/mediacentre/factsheets/fs311/en/>, accessed 6 January 2011.

WORLD HEALTH ORGANISATION, DIABETES (2009). Available from: <http://www.who.int/media centre/factsheets/fs312/en/>, accessed 10 January 2011.