International Journal of Food Sciences and Nutrition,
2013; Early Online: 1–7

informa
healthcare

The influence of saturated fatty acids on complex index and *in vitro* digestibility of rice starch

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Abstract
In Asia, rice and rice products are the main sources of carbohydrate contributing to both dietary energy and glycaemic load. It is known that complexation of starch with lipids could potentially reduce the availability of starch to enzymatic degradation. The aim of this study was to investigate the effect of capric, lauric, myristic, palmitic and stearic acids, ranging from 0 to 2 mmol/g starch, on complexing index and *in vitro* digestibility of gelatinized rice starch. The results revealed that the ability of rice starch to complex with saturated fatty acids increased with increasing concentration; but reduced with increasing lipid chain length. The complexation of rice starch with capric, lauric, myristic and stearic acids did not reduce the *in vitro* starch digestibility, except rice starch–palmitic acid complexes.

Keywords: *diabetes, glycaemic index, glycaemic response, rice, starch digestibility*

Introduction

The prevalence of diabetes has been rising in Asia, in particular, affecting 8.2% of the population aged 18–69 years in Singapore (Heng et al. 2010). Rice and rice products remain the major source of carbohydrate in many Asian countries. For example, rice consumption constitutes at least 55–65% of the total caloric intake in India (Mohan et al. 2010) and 30% in China (Wang 2002) and Japan (Kenko Eiyo Joho Kenkyukai 2009). In addition to rice being a bulk energy source, rice-based carbohydrate contributes to the dietary glycaemic load (GL) of these populations (Villegas et al. 2007; Mohan et al. 2009; Nanri et al. 2010). The GL is a concept mathematically derived from both glycaemic index (GI) and the amount of carbohydrate intake intended to represent the overall glycaemic effect of a diet (Salmerón et al. 1997).

The GI is a system of classifying carbohydrate-containing foods according to the extent to which they raise blood glucose levels after ingestion. The GI value of highly refined white rice ranges from 37 to 139 (Foster-Powell et al. 2002). A diet high in carbohydrates and with a high GI was associated with increased risk of type 2 diabetes (Foster-Powell et al. 2002; Villegas et al. 2007). A significant positive association between white rice consumption and risk of diabetes was observed among two cohorts of Chinese and Japanese women (Villegas et al. 2007; Nanri et al. 2010).

Carbohydrate-rich foods are habitually consumed in the presence of foods rich in fat and protein. Lipids have long been known to form inclusion compounds with amylose, with the hydrocarbon portion of the lipid located within the helical cavity of amylose (Banks and Greenwood 1972). The amylose–lipid inclusion, when resistant to α-amylase, leads to prolonged enzymatic hydrolysis (Eliasson and Krog 1985; Seneviratne and Biliaderis 1991). The slow digestibility of these complexes has been reported to reduce the postprandial serum glucose and insulin response (Takase et al. 1994; Murray et al. 1998). Larsson and Miezies (1979) observed a reduction in the rate of enzyme degradation of potato starch complexed with monoolein. Schweizer et al. (1986) found an inverse relationship between the degree of

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ISSN 0963-7486 print/ISSN 1465-3478 online
DOI: 10.3109/09637486.2013.763912

α-amylase digestion and the amount of complex formed between wheat flour and oleic acid. Guraya et al. (1997) showed reduced *in vitro* digestibility of waxy and non-waxy rice starch when complexed with emulsifiers. Tufvesson et al. (2001) showed that the addition of glycerol monopalmitin lowered the enzymatic hydrolysis rate of potato starch heated at 80°C.

On the other hand, amylose–lipid complexation can impede the amylose–amylose double helix formation and crystallization during cooling of a heated starch sample (Kulp and Ponte 1981). This reduces resistant starch (RS) formation, and increases the starch hydrolysis rate. In the same study, Tufvesson et al. (2001) showed that the addition of glycerol monopalmitin increased the hydrolysis rate of autoclaved high-amylose maize starch. Autoclaving at a high temperature makes amylose more available to form complex with monoglycerides, and thereby decrease the amylose retrogradation. Putseys et al. (2010) also demonstrated the addition of glycerol monostearate into starch reduced the RS content and increased the hydrolysis rate. Therefore, the authors suggested the addition of monodisperse amylose–lipid complexes to increase the RS contents resulting from the amylose chains freed from the complexes upon dissociation and to lower the degradability of starch.

Epidemiological data indicated a preventive role of low GI foods against the development of type II diabetes (Salmeron et al. 1997; Frost et al. 1999). Complexation of starch with lipids to reduce digestibility can therefore serve as dietary approach to lower the glycaemic response of rice and rice products consumed by many people in Asia. The aim of this study was to investigate the effect of capric, lauric, myristic, palmitic and stearic acids on complexing index (CI) and *in vitro* digestibility of gelatinized rice starch.

Methods

Rice starch and saturated fatty acids

Remy DR is neutral rice starch powder received from Beneo (Leuven, Belgium). According to products specification, the moisture content was maximum 14%, lipid content was maximum 0.1% and starch content was minimum 97% of dry matter. Capric acid (C10:0), lauric acid (C12:0), myristic acid (C14:0), palmitic acid (C16:0) and stearic acid (C18:0) were obtained from Natural Oleochemicals SDN BHD (Pasir Gudang, Malaysia).

Chemicals used

α-Amylase (30 units/mg), amyloglucosidase (≥ 300 U/ml), pancreatin (P7545; 8X USP specifications), pepsin (800–2500 units/mg) and bile extract (porcine) were purchased from Sigma-Aldrich Company Ltd (Missouri, USA). Amyloglucosidase (E-AMGDF; 3260 U/ml) was obtained from Megazyme International (Wicklow, Ireland). Chemicals were obtained from Sigma-Aldrich, Kanto Chemicals (Kanto Kagaku, Singapore) and Merck Chemicals (Darmstadt, Germany). All reagents were of standard analytical grade.

The maleate (0.2 M/pH 6) and acetate (0.1 M/pH 5.2) buffers were prepared according to previously described methods (Monro et al. 2009).

Preparation of gelatinized rice starch–fatty acid complexes

Rice starch powder (3 g) was weighed into a 100 ml reagent bottle. Total weight was brought up to 50 g by adding MilliQ water (Merck Millipore, Massachusetts, USA). The 6% (w/w) rice starch–water mixture was gelatinized hermetically in the reagent bottle by stirring for 15 min at approximately 95°C on a hotplate stirrer (Fisher Isotemp®, Fisher Scientific, Massachusetts, USA). For the measurement of CI, mixtures of 5 g gelatinized starch with 0.0625, 0.125, 0.25, 0.5, 1 and 2 mmol/g-starch fatty acids in 50 ml capped tubes were warmed above each melting temperature of the fatty acids with intermittent vortex for 2 min. For the measurement of *in vitro* digestibility, an amount of 2.5 g gelatinized starch was mixed with fatty acids at the concentration of 0.5 and 1 mmol/g-starch. A sample without the addition of fatty acid was prepared as a control.

Measurement of CI

The CI of starch was measured by adapting the method of Gilbert and Spragg (1964). Gelatinized rice starch–fatty acid mixture was mixed with 25 ml MilliQ water in a 50 ml capped tube. The tube was vortexed at 500 rpm for 2 min, and then centrifuged at 3000 *g* for 20 min. Supernatant (500 μl) and MilliQ water (15 ml) were added to 2 ml of iodine solution. The iodine solution was prepared by dissolving 2 g of potassium iodide and 1.3 g of I_2 in 50 ml of MilliQ water and allowing it to dissolve overnight. MilliQ water was added to make 100 ml. Each tube was inverted several times to mix, and then the absorbance (ABS) was measured at 690 nm (UV-2600, Shimadzu, Singapore). The CI was calculated using the following equation: CI (%) = (ABS of control − ABS of sample) × 100/ABS of control. All tests were performed in triplicate.

Measurement of in vitro digestibility

The *in vitro* digestibility of starch was determined by adapting the method of Monro et al. (2010). Gelatinized rice starch–fatty acid–water mixtures were digested in the specimen pots inserted to their full depth in a 12-position aluminium block placed in a circulating water bath maintained at 37°C with stirring

at 130 rpm. The oral phase digestion was initiated by adding 0.1 ml of 10% α-amylase in MilliQ water. After stirring for 1 min, 0.8 ml of 1 M HCl was added to the samples in quick succession to attain pH 2.5 (±0.2) measured by the use of pH meter (PB-11, Sartorius, Germany). Gastric phase was then initiated by adding 1 ml of 10% pepsin dissolved in 0.05 M HCl. The mixtures were stirred at 130 rpm for 30 min at 37°C to complete the gastric digestion, followed by neutralizing the gastric HCl with 2 ml of 1 M $NaHCO_3$ and 5 ml of 0.2 M maleate buffer (pH 6). Five millilitres of 10% bile extract solution in MilliQ water were added into the pots and then immediately filled with MilliQ water to the 55 ml mark. The pancreatic phase digestion was started by adding 0.1 ml of amyloglucosidase (Sigma-Aldrich) and 1 ml of 5% pancreatin in maleate buffer in quick succession. An aliquot of 0.5 ml sample from baseline, the end of oral and gastric phase, at 20, 60, 90 and 120 min from the start of pancreatic digestion was transferred into the tubes containing 2 ml ethanol for subsequent reducing sugar analysis.

Measurement of reducing sugars released during in vitro digestion

Sugars released during *in vitro* digestion were measured as monosaccharides using a modified dinitrosalicylic acid (DNS) colorimetric method (Monro et al. 2010). The ethanolic digesta were centrifuged at 1000*g* for 10 min at 20°C (Sorvall Legend RT, Thermo Scientific, Massachusetts, USA). Fifty microlitre aliquots of the supernatant, standard (10 mg/ml glucose) and a blank (MilliQ water) were mixed with 0.25 ml of acetate buffer containing 1% megazyme amyloglucosidase at room temperature for 30 min to complete the depolymerization into monosaccharides. Reducing sugars were then measured by adding 0.75 ml of DNS mixture containing 0.5 mg/ml glucose, 4 M NaOH and DNS reagent at a 1:1:5 ratio. The mixtures were heated for 15 min at 95–100°C, cooled and then diluted with 4 ml MilliQ water. ABS of the samples and standards was read at 530 nm against the blank using a UV–vis spectrophotometer (UV-2600).

Statistical analysis

The amount of sugars released at baseline, oral, gastric and pancreatic phase at 20, 60, 90 and 120 min was calculated in mg of glucose/g of starch using the ABS values of samples and standard. Rapidly digested starch (RDS) was calculated as the amount of reducing sugars present in the sample aliquot taken at 20 min from the start of pancreatic digestion, whereas slowly digested starch was calculated as the difference between the amount of reducing sugars measured at 120 min and RDS (Misha and Monro 2009). Data processing was carried out on an Excel spreadsheet (Microsoft Corporation, Redmond, WA, USA). The data reported in Figures 1 and 2 were subjected to analysis of variance (ANOVA) performed with Tukey test using the Statistical Package for the Social Sciences (SPSS; Version 16, Chicago, IL, USA). Statistical significance was set at $p < 0.05$.

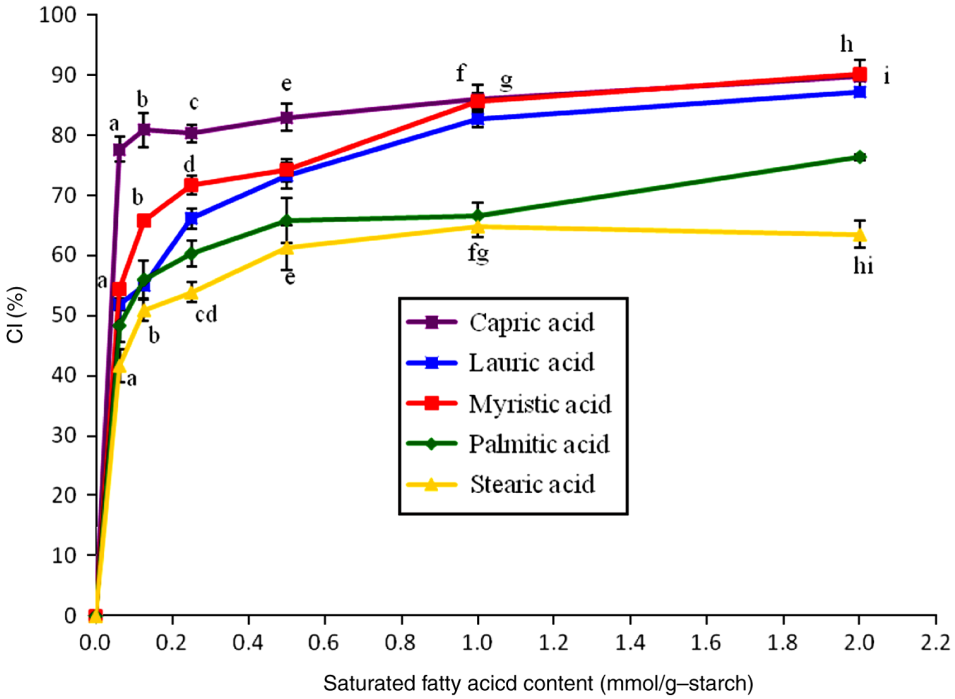

Figure 1. Effect of saturated fatty acids on the CI of gelatinized rice starch–fatty acid complexes. The CI values were expressed as mean ± SD ($n = 3$). Values with the same letter (a–i) at the same fatty acid concentration were significantly different at $p < 0.05$.

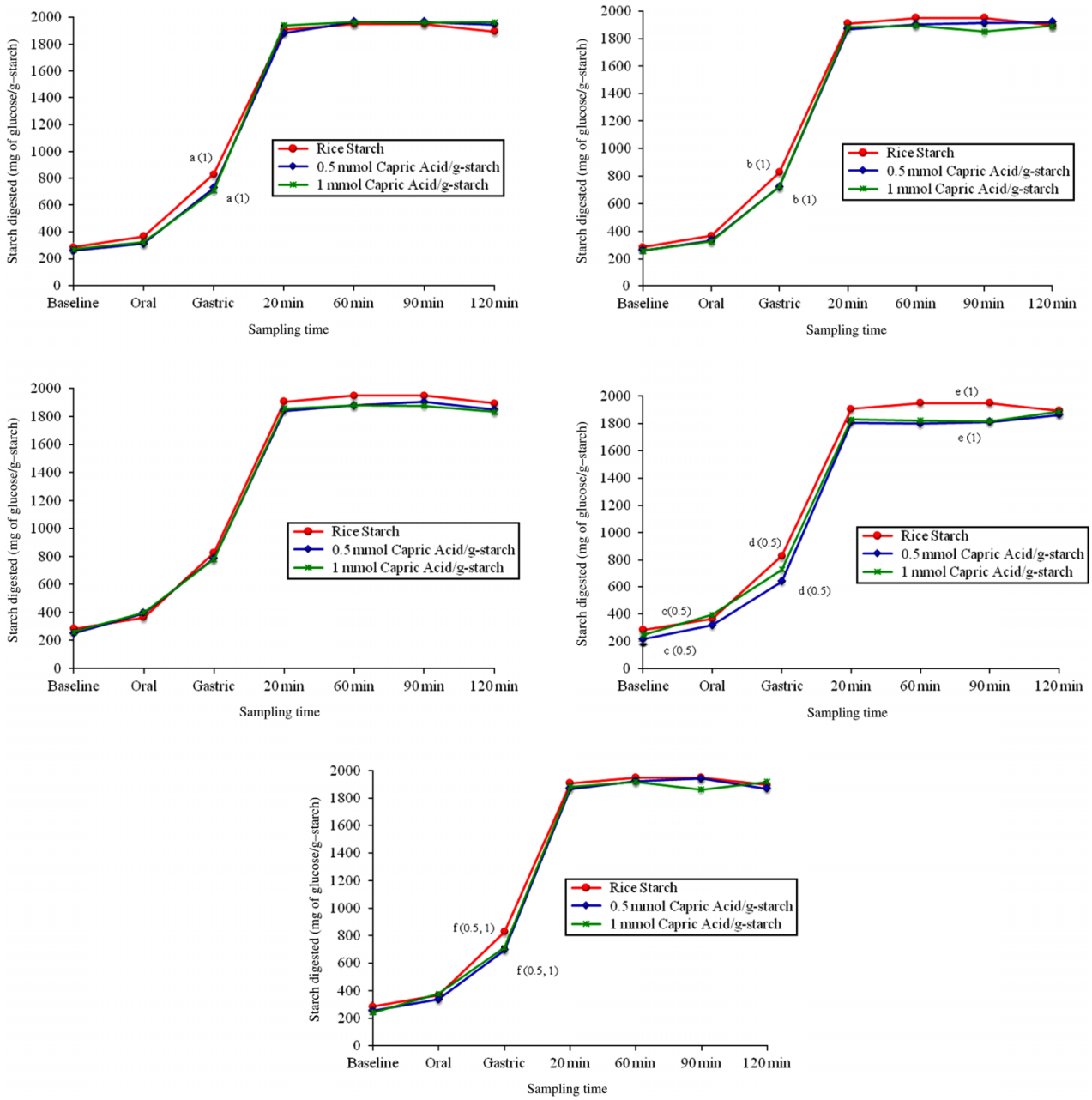

Figure 2. Effect of 0.5 and 1.0 mmol saturated fatty acids/g–starch on *in vitro* digestibility of gelatinized rice starch. The amount of sugar released across the digestion phases was expressed as mean ± SD ($n = 4$). Values with the same letter (a–f) at the same digestion phase were significantly different at $p < 0.05$. The concentration of fatty acid showing significant differences (compared to rice starch) was in parentheses.

Results

Effect of type and concentration of saturated fatty acids on CI

Inclusion of fatty acids reduced the iodine binding capacity of starch leading to an increase in CI. In this study, the ability of rice starch to complex with capric, lauric, myristic, palmitic and stearic acids was enhanced with increasing amount of fatty acids as shown in Figure 1. The optimum concentration of these saturated fatty acids forming a complex was in the range of 0.5–1 mmol/g-starch. Rice starch exhibited the highest complexing ability with capric acid, followed by myristic, lauric, palmitic acids and lowest with stearic acid. The CI value of capric and myristic acids was significantly higher than that of stearic acid ($p < 0.05$) as shown in Figure 1. For lauric and palmitic acids, the CI value was not significantly different from myristic and stearic acids, respectively, at all concentrations assayed ($p > 0.05$).

Effect of saturated fatty acids on in vitro starch digestibility

The *in vitro* digestion profiles were similar for rice starch complexed with saturated fatty acids at the concentration of 0.5 and 1 mmol/g starch (Figure 2). When compared with the digestibility of rice starch, the amount of free sugars was significantly reduced at

the gastric digestion phase when rice starch complexed with capric (1 mmol/g-starch), lauric (1 mmol/g starch), palmitic (0.5 mmol/g-starch) and stearic acids (0.5 mmol/g-starch) ($p < 0.05$). The free sugars reduction was approximately 15% in the gastric phase, except rice starch included with myristic acid. The latter showed similar susceptibility to enzymatic hydrolysis with rice starch alone. The RDS content of rice starch complexed with palmitic acid was lower than in the rice starch, and this led to a significant reduction of free sugars at 90 min of pancreatic digestion initiation ($p < 0.05$). For rice starch included with capric, lauric, myristic and stearic acids, the content of RDS was comparable with rice starch alone.

Discussion

This study systematically examined the ability of rice starch to complex with saturated fatty acids, and the *in vitro* digestibility of rice starch–fatty acid complexes. The fatty acids with a longer carbon chain provided a reduced amount of complex formation with amylose because of the low water solubility, and thus had poor dispersitivity in the gelatinized starch (Tufvesson et al. 2003). An inverse relationship between CI values and the number of carbons in saturated fatty acids was previously reported on potato and wheat starch (Tang and Copeland 2007; Kawai et al. 2012). The results of this study were consistent with those findings except that myristic acid with 14 carbon atoms was the preferred lipid chain length for rice starch than lauric acid containing 12 carbon atoms. It is still unknown which lipids form stronger complexes. Lipid chain lengths of 12, 14, 16 and 18 carbon atoms have been reported as the best complexing lipid with potato starch, corn starch, amylose and wheat starch, respectively (Lagendijk and Pennings 1970; Krog 1971; Hoover and Hadziyev 1981; Kawai et al. 2012).

The optimal concentration of saturated fatty acid forming a complex with rice starch was in concordance with a previous study using potato starch (Kawai et al. 2012). However, the maximum CI value of rice starch–fatty acid complex (lauric acid = 82.63%, myristic acid = 85.62%, palmitic acid = 66.47%, stearic acid = 64.70%) was twice that determined in potato starch–lipid inclusion at the same concentration (lauric acid = 49.9%, myristic acid = 42.4%, palmitic acid = 30.9%, stearic acid = 35.3%). The difference in CI may be in part due to the differences in amylose content, degree of polymerization and complex formation with endogenous lipids between rice starch and potato starch (Eliasson et al. 1998; Gelders et al. 2005; Mira et al. 2007).

Formation of amylose–lipid complexes could potentially reduce the availability of starches inducing implications on the glycaemic response of a food. It is important to note that starch digestibility is affected by both the quantity and quality of the single helix amylose–lipid complexes. In this study, the ability of capric and myristic acids complexed with rice starch was significantly higher than that of stearic acid, but the digestibility of these complexes was not significantly different. In addition, the CI value of rice starch and palmitic acid was relatively low, but a significant reduction of free sugars content was observed at the gastric phase and at 90 min of pancreatic digestion initiation. The lower hydrophilicity of longer lipid chains resulted in a stronger preference to reside within the hydrophobic helix cavity. A higher dissociation temperature was required to break the hydrophobic bonds (Raphaelides and Karkalas 1998). Therefore, the more hydrophobic interactions between the longer hydrocarbon chain, i.e. palmitic and stearic acids, and helix cavity could play a part in the resistance to enzymatic hydrolysis. On the other hand, the myristic acid-included rice starch gave a relatively high CI value but the amount of free sugars released during *in vitro* digestion was similar to rice starch indicating the weak physical interactions between myristic acid and rice starch. Based on the results, rice starch–palmitic acid complexes reduced the amount of free sugars significantly indicating that the amplitude of the glycaemic response may be lower than the rice starch alone. Therefore, co-ingestion of palm oil containing high concentrations of palmitic acid with rice may be proposed as a dietary suggestion to reduce the amplitude of glycaemic response in rice-based products.

The RDS content in food is a reflection of the amount of readily digestible starch that is largely responsible for short-term postprandial glycaemic response (Englyst et al. 1999). No significant difference was observed between the RDS content of rice starch and complexes formed in this study. Clegg et al. (2011) reported that the addition of fat to pancakes increased the digestibility of starch and release of sugars. Both studies demonstrated that the emulsification of lipids by bile at the beginning of pancreatic phase assisted in the breakdown of starch–fatty acid complexes leading to an increase in the amount of free sugars. The addition of lipase-containing pancreatin enzyme to simulate *in vivo* fat digestion might have improved the predictive power of the *in vitro* digestion model, particularly when carbohydrate foods with high fat content are analysed (Monro et al. 2010).

On the contrary, formation of amylose–lipid complexes was shown to reduce the susceptibility of amylose to enzymatic hydrolysis *in vitro* by Holm et al. (1983). Crowe et al. (2000) reported that lauric, myristic and palmitic acids inhibited the enzymatic hydrolysis of amylose by ~35%. Kawai et al. (2012) showed a significant reduction in the hydrolyzed starch content by inclusion with lauric, myristic, palmitic and stearic acids. The discrepancy could be attributed to the diversity in the *in vitro* digestion methods

employed and the quality of amylose–lipid complexes. The rate and extent of enzymatic degradation of amylose–lipid complexes reduced with increasing crystallinity (Seneviratne and Biliaderis 1991), where the extent of crystallinity increased with increasing amylose chain length (Godet et al. 1995). Therefore, the different digestibility of complexes formed between lipids and rice starch, wheat starch (Clegg et al. 2011) and potato starch (Holm et al. 1983; Crowe et al. 2000; Kawai et al. 2012) could be partly due to the different amylose content and degree of polymerization.

There is a considerable number of *in vivo* studies substantiating the addition of fat to a carbohydrate food which reduces the glycaemic response (Thomsen et al. 1999, 2003; Henry et al. 2008), and majority have found no difference between lipids (Thomsen et al. 1999, 2003; MacIntosh et al. 2003; Henry et al. 2008). However, the current *in vitro* data contradict the *in vivo* findings. Clegg et al. (2011) suggested that other factors such as absorption and transit time as opposed to digestion rate are responsible for reducing the glycaemic response when fats are added *in vivo*. Another hypothesis that may be playing a role is the potentiation of insulin by fat (Dobbs et al. 1975; Collier et al. 1988). Even though the *in vitro* digestion methods provide quick indicative data to reflect the *in vivo* glycaemic potential of carbohydrate foods (Monro and Mishra 2010), it does not necessarily reflect the *in vivo* starch digestion when fat is added.

Conclusions

The ability of rice starch to complex with saturated fatty acids increased with reducing lipid chain length. In this study, the formation of rice starch–fatty acid complexes did not necessary give rise to a reduction in *in vitro* starch digestibility. This contradicts the literature data from both *in vitro* and *in vivo* studies. It appears that the addition of bile to *in vitro* digestion assisted in the breakdown of the starch–lipid complexes leading to an increased amount of free sugars. Moreover, the lower GI when lipids are co-ingested with starchy foods could be attributed to the lower gastric emptying, higher insulinic response, decreased glucose absorption through the upper small intestine and lower starch accessibility to enzymes. Reduction in starch digestibility was only observed when rice starch complexed with palmitic acid. Future work will examine the effect of lipid type, chain length, degree of saturation, amylose content and degree of polymerization on the physiochemical properties, *in vitro* digestibility and *in vivo* glycaemic response of starch–lipid complexes.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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