Overnight Effects of Lecithin on Postprandial Response and Satiety Following a Standardized Breakfast: A Study in Healthy Young Adults

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Overnight Effects of Lecithin on Glycemic Response and Satiety Following a Standardized Breakfast: A Study in Healthy Young Adults

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Popular science summary

Timing is key: how long do the health benefits of plant fats last?

According to the World Health Organization, the number of people with diabetes has quadrupled over the last 30 years, with experts pointing out unhealthy diets and obesity as the major culprits. The use of appetite reducing medications has shown great promise in combatting this epidemic, but recent research suggests that a common plant fat could have similar benefits.

Our digestion of food is a complex system of biochemical reactions. From the moment food enters our mouths, specific enzymes work to release as much sugar as possible, to then be taken up in the intestines and distributed throughout the body through the bloodstream. In charge of controlling this entire process are hormones. They instruct us when to eat but they also instruct individual cells when to take up or release sugar from or to the bloodstream. However, having an energy-rich diet over the long-term risks making the cells unresponsive to insulin, the hormone responsible for promoting the uptake of sugar from the blood. This condition is called insulin resistance, and causes chronic illnesses, cardiovascular disease or even type 2 diabetes unless properly managed. The risk of developing type 2 diabetes and cardiovascular diseases can however be greatly reduced by maintaining a healthy diet that doesn't rapidly increase blood sugar levels after food intake.

Phospholipids are a type of fat that exists in all cells as the major constituent of the cell membrane. When removed as a byproduct in vegetable oil production, this brownish powder is called lecithin and is commonly used as an emulsifying agent in foods. Recent studies, however, have found that addition of lecithin to food reduces the release of insulin, the hormone that promotes sugar uptake from the blood. Lecithin also lowered blood sugar levels despite the lowered insulin levels and increased the release of hormones that make us feel full. Interestingly, this effect is not only limited to the meal where lecithin was added, but also a following lecithin-free meal eaten a few hours later. These findings suggest that lecithin might have an alternative area of use as a food additive, aimed to reduce the risk of developing type 2 diabetes by reducing overeating and elevated insulin levels.

Much is still unknown about lecithin, however. The appetite and insulin reducing effects were found immediately upon consumption and up to 6 hours later. This study wanted to fill the knowledge gap regarding the longer-term effects by investigating a later time span. This study focused instead on the effects between 11 to 14 hours after consuming lecithin enriched meals. The results showed that lecithin didn't have any effect on blood glucose levels, insulin or appetite in this later time span. This information is an important puzzle piece for the future research of novel uses of lecithin as well as for development of lecithin-containing functional foods.

Abstract

With the rise of global obesity, which is linked to the development of cardiovascular disease and diabetes, dietary strategies for prevention have become a subject of interest.

Previous studies at Lund University have demonstrated the potential of plant polar lipids as a functional food ingredient for improving the postprandial metabolic response as well as reducing the appetite for up to 6 hours. This thesis aims to investigate if these improvements remain after an overnight fast of 11 hours.

A randomized single-blind crossover study involving 17 healthy young adults was conducted. The participants consumed three different bread rolls in the late evening: one containing lecithin, one containing sunflower oil and one without added lipids. They then consumed a standardized breakfast in the morning, and their blood glucose levels, insulin levels and subjective feelings of appetite were measured the following two hours.

No significant differences were found in any of the tested variables, indicating that the positive effects were no longer present in this longer timeframe. These findings suggest that the improvements found from incorporating plant polar lipids into a carbohydrate meal subside sometime between the 6–11-hour period post-meal.

Keywords: Plant polar lipids, sunflower lecithin, postprandial response, second-meal effect, appetite regulation, satiety

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Abbreviations

BMI	Body mass index
CCK	Cholecystokinin
CVD	Cardiovascular diseases
FFA	Free fatty acids
GI	Glycemic index
GL	Glycemic load
GLP-1	Glucagon-like peptide 1
IAUC	Incremental area under the curve
PPGR	Postprandial glycemic response
PYY	Peptide YY
T2DM	Type 2 Diabetes Mellitus
VAS	Visual analogue scale

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1 Introduction

The worldwide prevalence of obesity, defined by having a body mass index (BMI) that equals or is greater than 30, has increased dramatically over the past three decades [1]. Studies have shown a clear link between obesity and mortality as well as chronic diseases such as cardiovascular diseases (CVD) and Type 2 Diabetes Mellitus (T2DM) [2, 3, 4]. In addition, individuals with T2DM are particularly susceptible to CVD [2]. Therefore, using lifestyle interventions, such as dietary modifications, is a synergistic complement to medical treatment for preventing CVD in these patients [2]. There is thus an interest in finding preventive measures for obesity and the development of T2DM and CVD. Recent studies at Lund University have investigated the beneficial metabolic and gut hormonal effects of plant polar lipids which have been showing promising results.

A study investigating the dose-dependent effects of plant polar lipids found that consumption of 17g of plant polar lipids as a spread on white bread significantly reduced the overall postprandial glycemic response (PPGR) as well as exhibiting a second-meal effect on postprandial glucose tolerance 3.5 hours later when compared to the reference sample oil [5].

Another study on 15 g of oat polar lipids (also as a spread on white bread) demonstrated similar effects on glycemic response but also reported lower concentrations of insulin and the hunger hormone ghrelin, higher concentrations of the satiety hormones glucagon-like peptide 1 (GLP-1) and peptide YY (PYY) and lower triglyceride levels immediately post-meal as well as after the second meal, which was served 3.5 hours later [6]. Sunflower lecithin, another polar lipid more commercially available than oat polar lipids, has been recently discovered to have the same metabolic and gut hormonal effects as oat polar lipids over a 6-hour period when 15 g is consumed [7].

A pilot study on the feasibility of incorporating polar lipids in a food matrix in the form of a muffin found that a breakfast enriched with plant polar lipids significantly decreased peak blood glucose levels after a subsequent standardized lunch. While not significant, likely because of the small sample size, the polar lipid enriched breakfast also trended towards improved overall as well as acute glycemic responses, higher ratings of fullness and lower ratings of hunger [8].

While all the mentioned studies have demonstrated beneficial metabolic and hormonal effects of polar lipids over a 6-hour period, the duration of these positive effects remains unknown. This master thesis aims to address this research gap.

2 Aim & objectives

This master thesis aims to investigate the postprandial metabolic and hormonal effects of polar lipids, specifically sunflower lecithin, **after an 11-hour period**. To achieve this, the following objectives will be addressed following a standardized breakfast, 11-14 hours after the consumption of the test meals:

- To quantify the postprandial **glycemic** response
- To quantify the postprandial **insulinemic** response
- To assess **subjective appetite sensations**
- To evaluate whether **sunflower lecithin** provides a **superior** metabolic and appetite-regulating effect compared to a reference oil (sunflower oil) and a no-lipid control

3 Theoretical background

3.1 Pathophysiology of obesity and Type 2 Diabetes

3.1.1 Mechanisms of insulin resistance and β-cell dysfunction

In healthy individuals, insulin, secreted by pancreatic β-cells, serves as the primary hormone responsible for regulating glucose homeostasis post-meal [9]. If muscle, fat and liver cells stop properly responding to insulin, a condition known as insulin resistance develops [10]. Prediabetes is a condition where individuals show early signs of insulin resistance, causing elevated blood glucose levels that exceed normal limits but remain below the threshold required for diagnosis of T2DM [10]. An example of this would be a fasting blood glucose value that is higher than 6 mmol/L but lower than 7 mmol/L [11]. Insulin resistance is caused by dysfunction of adipose tissue, which is linked to obesity [12]. The location of fat also plays a role in the development of insulin resistance, as visceral fat secretes a higher volume of resistance-inducing factors compared to subcutaneous fat [12]. Dysfunctional adipose tissue impairs insulin sensitivity through three distinct mechanisms [12].

First, and likely most critical, is the release of free fatty acids (FFA). These FFA accumulate in muscle and liver cells, where they compete with glucose for oxidation and disrupt intracellular insulin signaling pathways. The second mechanism involves the secretion of pro-inflammatory cytokines, triggering a state of chronic systemic inflammation. These cytokines activate cellular pathways that block insulin action. The third mechanism involves the altered secretion of specific adipokines. Adipokine hormones that enhance insulin sensitivity, such as retinol-binding protein 4 are downregulated while adipokine hormones that decrease insulin sensitivity, such as adiponectin are upregulated [12].

Collectively, these mechanisms inhibit the insulin signaling cascade, making the cells unresponsive. As insulin resistance develops, the pancreatic β-cells adapt to the decreased insulin sensitivity by increasing insulin secretion (compensatory hyperinsulinemia). This adaptation allows many insulin-resistant individuals to maintain normal blood glucose levels despite severe resistance. However, chronic exposure to elevated fatty acids exerts a direct toxic effect on the pancreas (lipotoxicity), which impairs the β-cells' ability to produce insulin. As the β-cells begin to deteriorate, glucose levels rise, initially as postprandial spikes, and later as fasting hyperglycemia. This combination of high fat and rising blood sugar levels creates a synergistic toxic effect known as glucolipotoxicity that accelerates the progression to clinical T2DM [12].

Not all obese individuals develop T2DM however, indicating that genetics play a decisive role. Obesity acts as a metabolic stressor that interacts with an underlying genetic susceptibility. In individuals with robust β-cells genetics, the pancreas successfully undergoes a compensatory expansion of β-cell mass and function, allowing the individual to counteract the toxic effect of elevated levels of FFA and maintain insulin production despite obesity. In individuals with genetically susceptible β-cells, however, this compensatory expansion does not occur, leading to dysfunction and eventual development of T2DM [12].

3.2 Glycemic control and satiety regulation

3.2.1 Starch digestion and glucose absorption

Starch is a complex carbohydrate consisting of long chains of glucose molecules. The glucose molecules are joined through glycosidic bonds, forming both linear amylose molecules and branched amylopectin molecules. Together, they make up the starch polymer. Found in plant-based foods, starch is the main source of energy in the human diet. Upon consumption, starch is broken down enzymatically into glucose, which serves as an energy substrate for cellular processes [9].

3.2.2 Postprandial glycemic response (PPGR)

Fasting blood glucose levels are heavily regulated in a narrow range as both high (hyperglycemia) and low (hypoglycemia) levels are harmful for the body [9]. For a healthy individual, the fasting blood glucose value ranges from 4-6 mmol/L [11]. The PPGR refers to the temporary spike in blood sugar levels that occurs after eating [9]. This happens because of dietary carbohydrates being broken down into glucose and absorbed into the bloodstream. The increase in blood sugar concentration, which can last for up to three hours, is counteracted by the release of the hormone insulin from pancreatic β-cells [9].

Insulin lowers blood glucose levels through tissue-specific mechanisms [9, 13]. In muscle and fat cells, insulin facilitates glucose uptake by triggering the movement of GLUT-4 transport proteins to the cell membrane [13]. Glucose uptake in the liver, however, does not rely on GLUT-4 [13]; here, the insulin suppresses gluconeogenesis and activates enzymes that convert glucose into glycogen for storage [9, 13]. The glycogen in the liver can be reconverted to glucose if blood glucose levels drop too low [9, 13]. Insulin stimulates lipogenesis in fat cells, and also in the muscle and liver cells if glycogen storages in those tissues are filled [9, 13].

The glycemic index (GI) was developed to assess the postprandial glycemic impact of carbohydrate-containing food. The metric is calculated by measuring the incremental area under the curve (IAUC) of blood glucose over a two-hour period when 50g of available carbohydrates from a test food is consumed, which is then compared with a reference standard containing 50g of glucose (dissolved in 250ml of water) or 50g available carbohydrates from white bread. The GI is then calculated by dividing the IAUC of the test food with IAUC of the reference standard and multiplying with 100. For starchy foods, GI is influenced primarily by enzymatic hydrolysis of starch, which in turn is dependent on how accessible the starch is to the digestive enzymes. This accessibility is affected by how the starch interacts with the food matrix as well as the degree of thermal or chemical modification applied during meal preparation [9].

While GI evaluates the glycemic potential of a standardized 50g of available carbohydrates in a food, this fixed portion rarely aligns with typical serving sizes. To address this, the glycemic load (GL) was introduced, which accounts for the actual quantity of carbohydrates consumed in a standard serving. The glycemic load is calculated by multiplying the GI for the food with the amount of available carbohydrate in a typical serving and then dividing by 100 [9].

The practical applications of GI and GL remain a subject of ongoing debate. A low GI classification does not necessarily mean nutritional quality; foods rich in saturated fats or added fructose often exhibit a low glycemic response due to delayed gastric emptying yet remain unhealthy choices. Furthermore, the accuracy of these measurements often decreases in actual daily diets. While GI values are determined by consuming single foods in isolation, typical diets consist of mixed meals where the presence of proteins, fats and other ingredients significantly

alters digestion rates and the resulting glucose response. Because of these complexities, recent reviews suggest that dietary fiber may be a more robust predictor of health outcomes than GI or GL alone. Substantial evidence highlights fiber's benefits for metabolism and gut health. Furthermore, a meta-analysis on current studies indicates that there is a stronger correlation between fiber intake and reduced chronic disease risk compared to low-GI alternatives [9].

Current epidemiological evidence suggests that PPGR is a more significant independent predictor of cardiovascular mortality than fasting blood glucose in both healthy individuals [14, 15] and individuals with prediabetes [14]. A Taiwanese study demonstrated that in nondiabetics, measuring 2-hour PPGR was a better predictor for risk of cardiovascular death than fasting blood glucose measurements [14]. Another European study focusing on strictly healthy individuals, meaning individuals who were neither diagnosed with T2DM or prediabetes, found that individuals whose postprandial glucose levels failed to return to baseline 2 hours post-meal faced significantly higher cardiovascular mortality risks [15]. These findings suggest that dietary interventions that target the PPGR are beneficial for susceptible T2DM patients [2] as well as healthy individuals.

An elevated PPGR is detrimental as it results in excessive protein glycation and generation of free radicals, both of which cause oxidative stress as well as inducing an inflammatory response. This creates an environment that promotes endothelial dysfunction, which is a risk factor for cardiovascular complications. These effects have been observed in healthy individuals as well as diabetic patients, suggesting that cardiovascular risk increases with the size and frequency of these spikes, rather than only occurring above a specific threshold [16]. Additionally, chronic elevated PPGR can cause insulin resistance [17], which as previously mentioned can lead to prediabetes and T2DM.

3.2.3 Hormonal regulation of appetite

Ghrelin is a hunger hormone, mainly produced in the stomach, that stimulates eating [18, 19]. Ghrelin levels rise during fasting and drop rapidly post-meal, suggesting it is responsible for initiating meals [19]. Clinical trials have shown that intravenous injection of ghrelin triggers hunger and increased caloric intake in humans [18].

Cholecystokinin (CCK) is a hormone secreted primarily in the duodenum and jejunum when nutrients, particularly fat and protein, are present [19, 20]. CCK slows gastric emptying, which induces a feeling of fullness and limits meal size by binding to CCK1 receptors on the vagus nerve, sending satiety signals directly to the brain [20].

GLP-1 and PYY are satiety hormones released by the intestinal cells primarily in the ileum and the colon under the presence of nutrients [21]. These hormones stimulate vagal afferent nerves, the nerve cells that carry signals from the gut to the brain, to transmit satiety signals [22, 23]. They can also cross the blood-brain barrier [22, 23] and bind directly to receptors in the brain [19]. In addition, GLP-1 and PYY also slows gastric emptying, prolonging the satiety signal transmission and dampening the postprandial glycemic response [19] [24]. GLP-1 also functions as an incretin hormone, promoting insulin secretion and inhibiting glucagon release to lower blood glucose [25]. This incretin effect is only active when blood glucose levels are above fasting values, preventing the risk of hypoglycemia [22]. Interestingly, previous studies have shown that postprandial insulin levels are decreased while GLP-1 levels are increased upon consumption of plant polar lipids, which is an indicator of improved glucose tolerance [6, 7].

While the aforementioned gut hormones are meal-related, leptin is a hormone that instead functions as a long-term regulator of energy homeostasis. Primarily secreted by adipose tissue, circulating leptin levels are proportional to total body fat mass. Leptin acts by crossing the blood-brain barrier and binding to specific receptors in the hypothalamus. Once bound, it decreases feelings of hunger while promoting feelings of satiety. During periods of fasting or weight loss, leptin levels drop, triggering adaptive mechanisms to conserve energy and increase appetite. In obese individuals, circulating leptin levels are elevated, but appetite is not suppressed. This is due to leptin resistance, where the brain becomes desensitized to the hormone's signaling [26].

3.2.4 The "Second-meal effect"

The second meal effect is a metabolic phenomenon where consumption of a previous meal influences the metabolic response to the next meal [27, 28]. Historically, this phenomenon was thought to be dependent on the GI of the first meal, as only meals with low GI values were shown to improve the postprandial responses [28]. However, recent research has demonstrated that the underlying physiological mechanisms appear to be distinct depending on the time interval between the meals [27, 29, 30].

In the short term, such as the period between breakfast and lunch (approximately 4 hours), the positive effect is caused by a prolonged postprandial phase due to the consumption of a previous low-GI meal [27, 28, 29, 30]. This sustained postprandial phase prevents the temporary hypoglycemia that often follows high-GI meals, thereby preventing the release of counter-regulatory hormones that would otherwise impair insulin action [28]. Furthermore, the prolonged digestion postpones the return to a fasting state, thus maintaining a suppression of FFA levels [30]. Since FFA competitively inhibits glucose uptake, their continued suppression is proposed as the primary mechanism for the improved insulin sensitivity observed at the start of the second meal [28, 30].

Recent evidence suggests that the positive postprandial effects from this delayed postprandial phase diminish over time [27, 29, 30, 31]. Studies comparing evening meals of equivalent low GI found that while pasta lowered acute postprandial responses, it failed to improve glucose tolerance the following morning, whereas evening meals rich in indigestible carbohydrates, such as barley kernels or breads enriched with resistant starch, successfully induced an overnight second meal effect [27, 29, 30, 31]. This indicates that while the short-term effect relies on digestion and absorption kinetics, the overnight effect is dependent on another mechanism: namely colonic fermentation [27, 29, 30, 31]. Indigestible carbohydrates are fermented in the large intestine, enabling the production of short-chain fatty acids overnight. These short-chain fatty acids have been shown to suppress FFAs and improve insulin sensitivity the following morning [27, 29, 30, 31]. This fermentation mechanism has additionally been demonstrated to result in higher feelings of subjective satiety, higher levels of GLP-1 and lower levels of inflammatory markers the following morning [31].

3.3 Polar lipids

3.3.1 Structure and classification

Polar lipids are a group of lipids characterized by their amphiphilic properties. The molecular structure consists of one polar hydrophilic end and one nonpolar hydrophobic end, which allows the molecule to be dissolved in both a water and an oil phase simultaneously. Polar lipids are for this reason often employed in the food industry as emulsifying agents, as they stabilize the

interface between the dispersed and continuous phase by preventing coalescence and precipitation of the droplets [32].

Whilst a rough initial classification of lipids may be done based on polarity, further division into groups requires an analysis of their chemical structure. Two major groups of polar lipids include glycolipids and phospholipids. Glycolipids are present in eukaryote cell membranes and play an important role in cell identification. Their structure consists of either a glycerol or sphingosine backbone, to which fatty acids and a mono- or disaccharide are bonded through either ester bonds or amide- and glycosidic bonds depending on the backbone.

Similarly, phospholipids' structure may either consist of a glycerol or sphingosine backbone with bonded fatty acids. The difference in structure from glycolipids stems from the sugar moiety being replaced by a phosphate group in phospholipids. The phosphate's polarity causes the glycerol end of the lipid to become hydrophilic, while the fatty acids remain hydrophobic, giving rise to its aforementioned amphiphilic properties. Different alcohol moieties may be joined to the phosphate to modulate the lipids polarity, allowing for further classification into groups such as phosphatidylcholine, phosphatidylglycerol and phosphatidylethanolamine amongst others. Phospholipids are found in all types of cells as the major constituent of the cell membrane, where its amphiphilic property allows it to form a bilayer [33].

3.3.2 Lecithin

Lecithin may be produced from many different food sources, one of which is as a byproduct of vegetable oil production. To stabilize the oil for commercial use or further processing, glycolipids and phospholipids must first be removed. Lecithin's major constituent is phospholipids from the groups of phosphatidylcholine, phosphatidylethanolamine, phosphatidylinositol and phosphatidic acid. For sunflower lecithin, a study found the ratios of these phospholipids to be 16, 6, 17 and 2 percent respectively based on a P-NMR analysis. The composition of fatty acids in lecithin roughly mimics the oil from which it was extracted. In sunflower lecithin 63 and 18 percent of the fatty acids were found to be poly- and monounsaturated steric acids respectively, followed by 11 percent saturated palmitic acid [34].

3.3.3 Mechanisms of action and metabolic effects

The linear amylose molecules in starch can interact with lipids if heated in the presence of water, forming amylose-lipid complexes [35]. A recent study demonstrated that when agitated in high temperatures, the branched amylopectin molecules can also form amylopectin-lipid complexes [36]. In both cases, the carbohydrate-lipid complexes reduce the enzymatic hydrolysis of the starch [35, 36], which is an indicator of a reduced PPGR [35]. The extent of digestibility reduction appears to be dependent on the stability of the formed complexes, with saturated FFAs forming the most stable complexes and unsaturated FFAs forming the least stable [35]. Although soy lecithin facilitated starch swelling, a process that typically promotes digestibility, the formation of lipid complexes ultimately resulted in reduced enzymatic hydrolysis compared to native starch [35].

Incorporating fat in bread modulates the PPGR by two mechanisms: delayed gastric emptying and reduced carbohydrate digestibility through the formation of carbohydrate-lipid complexes. While the extent of the modulation is dependent on the kind of fat used, the addition of fat does not appear to significantly alter overall insulin demand [37]. This is however not the case for plant polar lipids, which has been demonstrated to lower postprandial insulin demand [6, 7].

4 Material and methods

The trial was conducted using a randomized single-blind crossover study design. The project was conducted in three stages, the formulation of a lecithin containing test product and equivalent controls, collection of blood samples and analysis of these samples.

4.1 Bread formulation

The delivery method of lecithin was selected to be through consumption of a bread roll. For this reason, it was necessary to develop three bread formulations: one containing lecithin, as well as two controls, one with an equal mass of sunflower oil and one without any added lipids.

Initial trials started with a basic dough containing wheat flour, water, salt and dry yeast. To this dough, lecithin and sunflower oil were incorporated separately. Different water to flour ratios were tested, as well as different mixing orders to ensure a homogenous dough. Due to concerns that formation of bread crust may influence the breads digestion or lecithin content, different shapes and shaping techniques were also investigated. Due to the same concerns, different cooking surfaces were tested as these affected crust thickness on the underside of the bread roll. Using thermometers, a required cooking time to reach an internal temperature of 96°C could be confirmed.

Through the variations explained above, an optimal formulation could be determined. The ingredients and ratios per bread roll are shown in Table 1. The dry ingredients flour, salt, and lecithin if included, were mixed into a homogenous powder. The dry yeast was dissolved in water at 37°C together with oil, if included. The dry mix was then added to the liquid and worked into a dough. The dough was allowed to rise under a cloth for 30 minutes at room temperature. Dough balls of 149.2 ± 1 g if lipids were included, or 133 ± 1 g if lipids were not included, were portioned out on pieces of parchment paper and shaped into smooth semi-spheres using wetted hands. Cooking was performed at 200°C for 30 minutes by placing the parchment papers with dough onto an oven rack positioned in the middle of the oven, with trays placed above and below to shield from direct heating to minimize crust formation.

Table 1: Formulations for lipid free dough, dough with added sunflower oil and dough with added lecithin forming a single bread roll.

Ingredient	**Mass (g)**		
	Lipid free dough	**Sunflower oil dough**	**Lecithin dough**
Wheat flour	80	80	80
Water	50	50	50
Sunflower lecithin	N/A	N/A	16
Sunflower oil	N/A	16	N/A
Dry yeast	1.6	1.6	1.6
Salt	1.6	1.6	1.6
Total	133.2	149.2	149.2

4.2 Clinical trials and sampling

Prior to the start of the study, all participants were fully informed about the study's purpose and procedures, both orally and in writing. Written informed consent was obtained from each subject before participation. The participants were explicitly informed that their participation was voluntary and that they had the right to withdraw from the study at any time without providing a reason. The study was approved by the Swedish Ethical Review Authority.

The clinical trials took place on weekdays throughout the month of October of 2025 at the department of Processing and Life Science Engineering at Lund University. The participant group initially consisted of 18 healthy volunteers, of which 17 completed the trial (10 females and 7 males). They were aged 21-27, had no chronic diseases or allergies, and had a BMI between 18.7-26.3 kg/m^2. As the study was conducted on young healthy individuals, participants classified as underweight (BMI below 18.5) or obese (BMI above 30) were not eligible. The day before a trial, the participants were instructed to thaw one of the frozen bread rolls they had previously received over the day and consume it at 21:00 the same evening. The bread roll was to be consumed plain and without further baking or toasting, only accompanied by water, plain tea or plain coffee. After the consumption the participants were fasting until the sampling began the next day. Furthermore, participants were instructed to standardize their day before the trial, such as eating the same meals, abstaining from alcohol and high fiber foods and performing the same or similar activities.

Each participant came in for sampling on three occasions, at least 6 days apart. What test product they had consumed the day before was randomized. The participants arrived onsite at 07:30 and were given a couple of minutes to calm down and acclimatize to the waiting room. Before sampling began, the participants were instructed to rinse their hands under hot water to increase blood flow, as well as holding on to an electric handwarmer throughout the trial. Fasting blood glucose values were measured and a blood sample for insulin levels was collected for each participant. Participants were also asked if they had felt any side effects from the bread

roll they had consumed the day before. At 08:00 the participants were given a standardized breakfast consisting of 100 g (50 g available carbohydrates according to label) untoasted plain toast (JätteFranska, Pågen AB) together with 150g of water. The participants were instructed to consume the bread and water in an even pace over 10-12 minutes. From consumption start, the participants' capillary blood was sampled from the fingertips every 15 minutes the first hour, and every 30 minutes the following two hours. Both blood glucose and insulin samples were taken at each sampling time, except for the 15 and 150 minute mark where only blood glucose was measured. Sampling was performed in a room adjacent to the waiting room, and the blood glucose values were analyzed immediately using a HemoCue® Glucose 201+ device and HemoCue® Glucose 201 microcuvettes, while the samples for serum insulin were collected in Microtainer® SST™ and left in room temperature for 30-60 minutes before being centrifuged, transferred to a microcentrifuge tube and frozen for later analysis. While waiting for their next sampling time participants were free to spend the time as they pleased, provided they remained seated in the waiting room.

Just before each sampling time, participants were instructed to fill out a questionnaire rating their fullness, hunger and desire to eat. The fullness was explained to represent how filled their stomach felt, the hunger as how much discomfort they felt due to not eating and desire to eat how much they wanted to eat regardless of how they rated fullness and hunger. When applicable and as a reference, the participants were shown how they rated these sensations the previous time before breakfast consumption. The visual analogue scale (VAS) was made by making a mark on a 10 cm long horizontal line where only the midpoint was indicated.

4.3 Insulin and starch quantification

Following the trials, insulin levels in the collected samples were quantified. The method chosen was through ELISA and the chosen kit (Insulin ELISA 10-1113-01, Mercodia AB) had high specificity to human insulin and no cross-reactivity with C-peptide or Proinsulin, with a measurement range of 0.13-8.70 µg/L. As blood samples sometimes were less than 200 µL, the resulting serum was not enough to perform duplicates.

The available starch levels in the bread products were quantified by breaking down the starch enzymatically to glucose using Thermamyl® and amyloglucosidase. The released glucose was subsequently measured though spectrophotometry at 450 nm with glucosidase/peroxidase and determined using a calibration curve of glucose standards. The moisture content of the breads was also determined by weighing the sample before and after being dried over night at 105°C.

4.4 Statistical method

The determined statistical method for this project was to perform a One-Way repeated measures ANOVA to identify if there were significant differences in the measured variables for the test products, and if so, perform paired t-tests to identify where these differences occurred.

As One-Way repeated measures ANOVA was unavailable in the used spreadsheet software, a Two-Factor ANOVA without replication was performed instead, with the independent variables being test product and participant, and the dependent being the investigated effect. These two analyses are identical under the assumption of sphericity, which may be assumed in a randomized experiment with no risk of cross-over effects between the products [38]. As the participants consumed the test products in a randomized order with a minimum of a 6-day washout period between the test products, this assumption is likely fulfilled.

5 Results

5.1 Time intervals and handling of data

Due to external circumstances, one of the sampling days had to be cut short after the 120-minute mark. Because of this, as well as the peak in blood glucose occurring during the first 1-1.5 hours, all charts and analysis have been conducted on the time interval 0-120 minutes. In addition, occasionally too little serum was collected to give a reliable result on the ELISA. In these instances, all values at the affected time-point for all three products were disregarded when calculating IAUC and maximum value for an individual participant, as well as disregarded when calculating averages.

5.2 Blood glucose

As external factors may influence a specific participant's initial blood glucose values between trial days, the Δ blood glucose for each participant and product were calculated, setting the initial blood glucose as the baseline. The average absolute blood glucose values as well as the average Δ blood glucose for the three products are found in Appendix 1 and visualized in Figure 1.

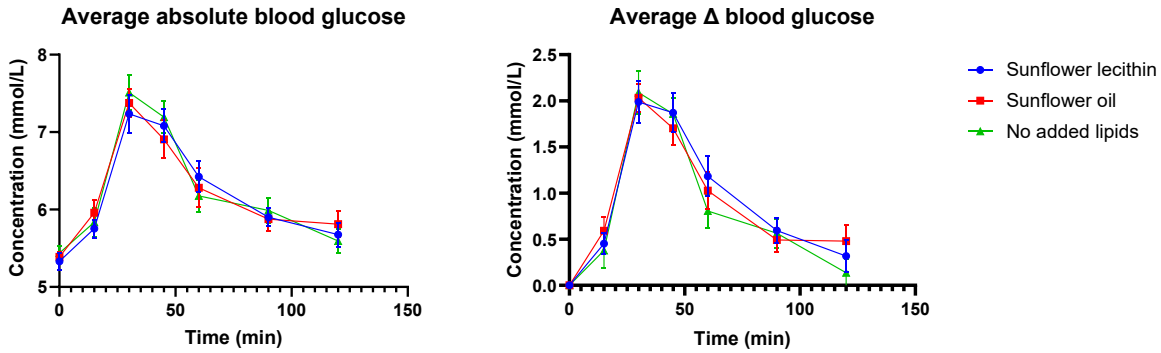

Figure 1: Average absolute (left) and Δ blood glucose (right) concentrations (in mmol/L) of the participants following the consumption of the standardized breakfast measured over time. The plotted curves illustrate the differential effect 11 hours after consuming a bread roll containing sunflower lecithin (blue), sunflower oil (red) or no added lipids (green) on the preceding evening. Error bars represent SEM (n = 17).

To analyze and compare the effects of the products on blood glucose, both the IAUC, as well as the maximum value was calculated for each participant's Δ blood glucose values. The average IAUC were found to be 113.9 ± 26.03, 109.9 ± 24.05 and 102.0 ± 25.77 mmol·min/L for the bread roll with sunflower lecithin, sunflower oil and with no added lipids respectively. An ANOVA showed no significant difference ($P<0.05$) between the groups in terms of IAUC or maximum value.

5.3 Serum insulin

To address the same concerns regarding external factors as mentioned for the glucose measurements, the Δ serum insulin was calculated by subtracting the initial fasting concentration from all subsequent values recorded for each participant. The Δ absolute average serum insulin levels as well as the average Δ serum insulin curve over time are found in Appendix 2 and visualized in Figure 2.

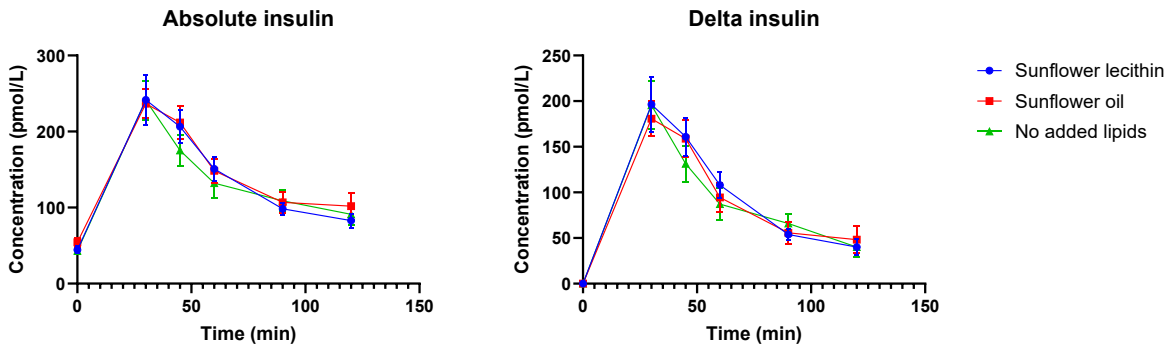

Figure 2: Average absolute (left) and Δ serum insulin (right) concentrations (in pmol/L) of the participants following the consumption of the standardized breakfast measured over time. The plotted curves illustrate the differential effect 11 hours after consuming a bread roll containing sunflower lecithin (blue), sunflower oil (red) or no added lipids (green) on the preceding evening. Error bars represent SEM (n = 17).

The IAUC for the Δ serum insulin curve and maximum Δ serum insulin were calculated for each participant. The average IAUC were calculated to 11485±2576, 10968±2364 and 10908 ±2609 pmol·min/L for the bread roll with sunflower lecithin, sunflower oil and with no added lipids respectively. An ANOVA showed no significant difference ($P<0.05$) between the groups in terms of IAUC or maximum value.

5.4 Subjective appetite responses

The average responses of the 17 participants that completed the trial when asked to report their subjective sensations of fullness, hunger and desire to eat are found in Appendix 3 and visualized in Figure 3. Three aspects of these responses were investigated: IAUC, initial response and maximum response, these values are also found in Appendix 3.

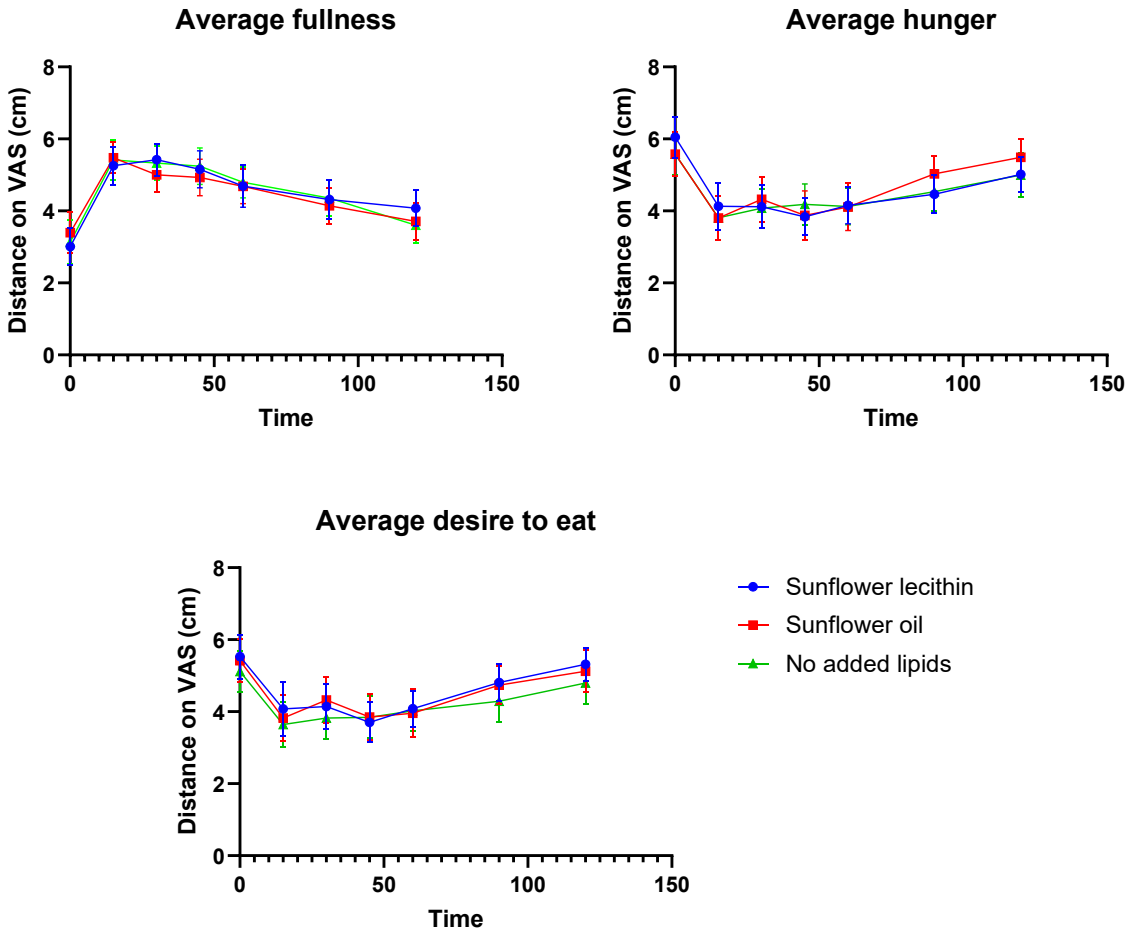

Figure 3: Average subjective appetite responses of participants over time following a standardized breakfast. The three panels display average fullness (top left), average hunger (top right), and average desire to eat (bottom). All values represent distance in centimeters on a visual analogue scale (VAS) and illustrate the persisting differential effects of having consumed a bread roll containing sunflower lecithin (blue), sunflower oil (red), and no added lipids (green) during the preceding evening. Error bars represent SEM (n = 17).

An ANOVA showed no significant difference ($P<0.05$) between the groups in terms of IAUC, initial value or maximum value for any of the three appetite variables investigated.

5.5 Starch analysis

The results from the starch analysis are shown in Appendix 4 and concluded that the average available starch based on fresh weight was 38, 42 and 41 percent for the bread roll with sunflower lecithin, the bread roll with sunflower oil and the bread roll with no added lipids respectively. The available starch in the toast used from the controlled breakfast was determined to 42 percent based on wet weight.

5.6 Side effects

Three participants reported minor side effects. One participant felt slightly bloated after having consumed the product containing sunflower oil. Another participant felt slightly bloated after having consumed both the product containing sunflower oil, and the product containing no added lipids. A third participant felt slightly bloated after having consumed all three products.

6 Discussion

Research within the field of lecithin's effect on glycemic response and insulin is limited, and even less information is available on sunflower lecithin specifically. The most complete work to date is the doctoral dissertation of Mukul Hossain. The dissertation investigated the glycemic and insulin effects of oat polar lipids, and a comparison to sunflower lecithin was made. The dissertation found a significant reduction in both blood glucose and insulin levels the two proceeding hours after consuming a breakfast containing 15 g sunflower lecithin, compared to an equivalent rapeseed oil and a lipid free control breakfast. In the interval 2.5-5.5 hours after breakfast, a significant reduction was found in insulin levels compared to both the rapeseed oil and lipid free control. In the same interval, the lecithin breakfast was found to cause a reduction in blood glucose levels compared to the lipid free control, but not the rapeseed oil control [39].

The study measured three variables: blood sugar, serum insulin and subjective appetite sensations at a controlled second meal 11 hours after consumption of a sunflower lecithin bread roll and two control bread rolls, one containing sunflower oil and one without added lipids. When the average values of the investigated variables were plotted over time, negligible to no difference could be visually observed in the curves between the lecithin and the controls. Further analysis was performed by calculating the IAUC and maximum value for each product. The ANOVA yielded P-values far above the desired threshold of 0.05, thus indicating no significant difference in maximum blood glucose and insulin levels, or blood glucose and insulin levels over time. Similarly, the investigated appetite sensations fullness, hunger and desire to eat, all yielded P-values far above the desired threshold regarding IAUC and maximum values, once again indicating no significant difference between the lecithin and the controls. As the ANOVA indicated no significant difference between the groups, no paired t-tests were performed as this might have resulted in false positives.

Blood sugar and insulin levels are closely related. High blood glucose promotes the secretion of insulin, which in turn decreases blood glucose levels by promoting glucose uptake. The dependent relationship between these two variables suggests that the same trends should be observed in the insulin data as in the blood glucose data. The fact that no significant difference was found in either insulin or blood glucose over time, or in terms of maximum value thus increases the credibility in the results. The results of this study differ from previous studies however, as the previous studies found significant differences in blood glucose, insulin levels and appetite after consumption of sunflower lecithin [5, 6, 7, 8]. In those studies, lecithin or its controls were consumed at breakfast, and the second-meal effects investigated were the following lunch. Those results indicated that lecithin helped reduce the postprandial response of both insulin and blood sugar after both breakfast and lunch and dampened subjective appetite, all of which are desired outcomes.

An explanation for the observed results in this study is the metabolic timeline of the intervention. The mechanism of delayed gastric emptying, which modulates the PPGR, relies on the presence of lipids in the gastrointestinal tract. Given the 11-hour fasting interval, it is highly probable that the lipids in the bread rolls were completely metabolized, as postprandial fat levels in healthy individuals typically return to baseline within 6-8 hours [40]. Consequently, the stimulus required to release satiety hormones, such as CCK, GLP-1 and PYY, was absent at the time of the breakfast. This complete return to a fasting metabolic state explains the lack of significant differences across all the tested variables and suggests that the metabolic effects of lecithin are relatively short-lived.

Furthermore, the starch analysis provides a structural explanation for the lack of an overnight clinical effect. Theoretically, lecithin should interact with amylose or amylopectin to form carbohydrate-lipid complexes, which resist enzymatic hydrolysis [35, 36]. However, the available starch content across the three bread formulations was virtually identical (~38-42%). Although the lecithin bread exhibited slightly lower starch availability, the difference was negligible. As the formation of these complexes are affected by both food processing as well as the specific formulation, it is possible that the baking, the freezing or even the specific formulation ratio disrupted the formation of the complexes [41]. For instance, baking under low temperature for longer time periods and refrigerating instead of freezing has been shown to decrease starch digestibility in bread [41]. Without the generation of these resistant complexes, there is no fermentable substrate in the colon. The absence of colonic fermentation means no production of short-chain fatty acids, thereby preventing the long term "second-meal effect" associated with indigestible carbohydrates [27, 29, 30, 31].

This study is not without limitations. The sample consisted of healthy young adults, a demographic that typically possesses robust homeostatic control over blood glucose. This metabolic resilience may have masked effects that could otherwise be observable in populations with impaired glucose tolerance, such as those with prediabetes or T2DM. Additionally, while the washout period was sufficient, control of external lifestyle factors such as sleep quality, evening meal composition and exercise, relies on participant compliance. These lifestyle factors could have affected the results, though the randomized study design attempts to mitigate this. This study also only investigated the effects of lecithin from sunflowers, which is a limiting factor in drawing wider conclusions regarding lecithin in this time interval, as lecithin from other sources could have other effects.

As the findings of this study were unable to confirm that consumption of sunflower lecithin had any significant effect on blood sugar, insulin or appetite after 11 hours, an interesting area of future research would be to determine when the effects of lecithin subside. Thus, the time interval between 6-11 hours is of particular interest. Furthermore, as this study incorporated lecithin into the food-matrix through baking and then freezing, as compared to Hossain who added lecithin as a spread on bread, it would also be of interest to investigate how this type of processing affects lecithin's therapeutic properties as compared to its unprocessed counterpart.

7 Conclusion

In conclusion, while sunflower lecithin has demonstrated potential as a functional food ingredient for acute glycemic and insulinemic control and lowered appetite over a 6-hour period, this study establishes that these benefits do not persist beyond 11 hours.

8 Future work

Lecithin from other sources have been investigated as a functional food component in a similar way to sunflower lecithin. The longevity of their effect is however unknown. An interesting future area of research would be to compare lecithin from different sources and compare when their glycemic effects subside. The conducted studies also lack a long-term perspective of what effects a diet containing lecithin would exert on the glycemic and insulinemic response, but also other health markers such as blood fats, inflammation markers and gut hormones such as ghrelin, CCK, GLP-1 and PYY, which may be expanded on.

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Appendix 1: Average absolute and Δ blood glucose (mmol/L) over time.

Time (min)	Aboslute blood glucose sunflower lecithin	Aboslute blood glucose sunflower oil	Aboslute blood glucose no added lipids	Δ blood glucose sunflower lecithin	Δ blood glucose sunflower oil	Δ blood glucose no added lipids
0	5.3	5.4	5.4	0.0	0.0	0.0
15	5.8	6.0	5.8	0.5	0.6	0.4
30	7.2	7.4	7.5	2.0	2.0	2.1
45	7.1	6.9	7.2	1.9	1.7	1.9
60	6.4	6.3	6.2	1.2	1.0	0.8
90	5.9	5.9	6.0	0.6	0.5	0.6
120	5.7	5.8	5.6	0.3	0.5	0.1
150	5.3	5.3	5.5	0.0	-0.1	0.1
180	5.2	5.1	5.1	-0.2	-0.3	-0.3

Appendix 2: Average absolute and Δ serum insulin (pmol/L) over time.

Time (min)	Aboslute serum insulin sunflower lecithin	Aboslute serum insulin sunflower oil	Aboslute serum insulin no added lipids	Δ serum insulin sunflower lecithin	Δ serum insulin sunflower oil	Δ serum insulin no added lipids
0	44.40	54.43	43.11	0.00	0.00	0.00
30	241.59	236.59	241.05	196.35	180.73	195.57
45	206.35	211.92	175.23	160.90	158.78	131.34
60	150.84	148.26	132.19	108.12	94.47	87.16
90	98.35	106.96	108.97	53.94	55.74	65.86
120	82.90	101.98	91.17	40.18	48.18	40.06
180	41.72	52.52	38.17	-6.92	-4.53	-6.81

Appendix 3: Average responses to subjective appetite variables over time, IAUC, initial value and maximum value for the appetite variables fullness, hunger and desire to eat.

	Fullness			Hunger			Desire to eat		
Time (min)	Sunflower lecithin	Sunflower oil	No added lipids	Sunflower lecithin	Sunflower oil	No added lipids	Sunflower lecithin	Sunflower oil	No added lipids
0	3.0	3.4	3.1	6.0	5.6	5.6	5.5	5.4	5.1
15	5.3	5.5	5.4	4.1	3.8	3.8	4.1	3.8	3.6
30	5.4	5.0	5.3	4.1	4.3	4.1	4.1	4.3	3.8
45	5.2	4.9	5.2	3.8	3.9	4.2	3.7	3.9	3.8
60	4.7	4.7	4.8	4.2	4.1	4.1	4.1	4.0	4.0
90	4.3	4.1	4.4	4.5	5.0	4.5	4.8	4.7	4.3
120	4.1	3.7	3.6	5.0	5.5	5.0	5.3	5.1	4.8
150	3.9	3.3	3.4	5.2	6.0	5.5	5.9	5.5	5.5
180	3.3	2.5	3.1	6.5	7.2	6.4	6.6	7.1	6.5
IAUC (cm min)	556.3	542.0	556.1	529.4	547.6	526.8	536.2	528.6	499.4
Initial value (cm)	3.009	3.394	3.138	6.047	5.574	5.576	5.529	5.418	5.118
Maximum value (cm)	5.838	5.788	6.071	6.638	6.403	6.341	6.438	6.409	5.897

Appendix 4: Dry substance and available starch in the four bread products used in the study.

Product/ replicate	Dry substance fresh bread	Average	Dry substance sample	Average	Sample mass for analysis (mg)	Measured glucose in diluted sample (µg)	Calculated starch in sample (mg)	Ratio starch in fresh bread	Avrage ratio starch in fresh bread
Bread roll with sunflower lecithin (1)	0.60	0.60	0.93	0.93	503.00	66.00	297.00	0.38	0.38
Bread roll with sunflower lecithin (2)	0.60		0.93			65.92	296.63	0.38	
Bread roll with sunflower oil (1)	0.60	0.60	0.92	0.92	501.00	72.34	325.51	0.42	0.42
Bread roll with sunflower oil (2)	0.60		0.92			72.22	324.97	0.42	
Bread roll with no added lipids(1)	0.55	0.54	0.91	0.91	504.00	76.81	345.65	0.41	0.41
Bread roll with no added lipids (2)	0.54		0.91			77.11	346.98	0.41	
Toast used as controlled breakfast (1)	0.61	0.61	0.93	0.93	503.00	72.31	325.40	0.42	0.42
Toast used as controlled breakfast (2)	0.61		0.93			72.45	326.04	0.42	