

Assessing the impact of school canteen programs on the development of childhood obesity

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Abstract

The main aim of the school canteen is to provide children with enough food and essential nutrients such as calcium, iron, and essential fatty acids. The canteen must feed the children and, in particular, provide them with the energy they need for the afternoon and prevent them from having concentration problems. The canteen is also very useful in enabling children from disadvantaged backgrounds to eat foods that they cannot find at home. The canteen is also an opportunity to discover new flavors. Our work is essentially based on evaluating the school canteen program and detecting the impact of the menus presented to the children on the onset of childhood obesity, to monitor the lunches for a month in the "al afak" school canteen. We are determining the levels of these three macronutrients (lipids, glucose, and proteins) and also seeing whether the constituents of the meals comply with the ranges recommended by the European Food Safety Authority. From the results, we can conclude that the meals presented for children have increased protein content and a need for carbohydrates, which can lead to physical and psychological problems.

Keywords: School canteen; nutrients; daily intake; childhood obesity

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1. Introduction

A healthy, balanced diet is essential for children's development and growth. Nutritional intake should provide sufficient essential nutrients such as proteins, carbohydrates, fats, vitamins, and minerals. According to the World Health Organization (WHO), a healthy diet for children should include foods such as fruit, vegetables, wholegrain cereals, lean protein, and low-fat dairy products [1]. Proteins are important for the growth and repair of body tissues, carbohydrates provide energy; fats help absorb vitamins, and vitamins and minerals are needed to maintain a healthy immune system, strong bones, and a developing brain. It is also important to monitor sugar and salt intake in children, as excessive consumption can lead to health problems such as obesity, high blood pressure, and diabetes. It is recommended that children eat a variety of foods to get all the nutrients they need. Meals should be planned around a variety of food groups to provide a full range of nutrients [2]. As many children and teenagers eat at the canteen, we feel it is right to remind them of the canteen's nutritional objectives.

The first and foremost objective of the canteen should be to feed the children and, in particular, to provide them with the energy they need for the afternoon and prevent them from having concentration problems. Too many children do not eat enough in the canteen because the meals served are not always to their taste. So we need to offer foods that children enjoy. Starchy foods, a source of complex carbohydrates, are an essential component of meals. The second objective is to ensure the intake of certain essential nutrients such as calcium, iron, and essential fatty acids [3]. Canteens have no place in preventing childhood obesity. Firstly, it has little quantitative impact. On average, a child eats 140 days a year in the canteen. Energy intake is broken down as follows: 20% for breakfast, 20% for snacks, 30% for lunch, and 30% for dinner; the energy intake for this lunch therefore represents 11.5% of the total energy intake for a full year (365 days). What's more, the results of scientific studies show the limits of these actions in terms of effectiveness [4]. Many specialists point out that these nutritional information campaigns could encourage eating disorders and the stigmatization of obese children [5]. It is now well

established that childhood obesity is a multifactorial constitutional disease. The obesogenic environment of industrialized countries, combining a sedentary lifestyle with an abundance of food, encourages the onset of obesity in children with a genetic predisposition to weight gain. Ignoring this predisposition is tantamount to believing that obesity is simply the consequence of a "bad diet," for which children and parents alone are responsible [6].

The primary objectives of the canteen are to feed the children and contribute to their essential nutritional intake. The secondary objectives are to offer foods that are rarely consumed by the most disadvantaged families and to introduce them to new tastes. Given its low contribution to annual energy intake and the disappointing results of collective prevention through nutritional education, the role of the canteen is not to prevent childhood obesity. However, through the intermediary of the school medical service, the school can be the ideal place to detect children at risk, which is why our study is based on the evaluation of the canteen programme in relation to the daily nutritional needs of a child, as well as discovering the impact of the proposed menus on the appearance of childhood obesity in schoolchildren, and ending by proposing a menu that is appropriate, balanced and respects the recommended portions.

2. Materials and methods

2.1 Study population

The study was carried out within a private primary school in the city of Khemisset, in the region of Rabat, Morocco. We chose this school because it has a canteen that accommodates nearly 200 children per day. The weekly menu is proposed by the property manager and kitchen chefs and is based mainly on a starter, main course, and dessert. The purpose of this work is to involve children aged 6 to 12 (50 children) in this survey, as well as the evaluation of the school canteen program, and then to see if the daily menu meets the nutritional needs of the children, and finally to see if meals influence the onset of obesity in children. According to the EFSA (European Food Safety Authority), the breakdown of energy requirements between meals is as follows: 20% for breakfast, 30% for lunch, 20% for snacks, and 30% for dinner, the nutritional requirements for breakfast per day for a child aged 6 to 12 are as follows: Lipids 12 to 16.5g, and Protein 4.5 to 6g, and Carbohydrates 69 to 84 g.

2.2 Data collection

For each child, we collected data from the archives of the institution supervised by the school principal and with the help of the kitchen chefs (child age, type of diet (proteins, carbohydrates, and fats), school canteen program, first week of January, second week of January, week The third of January and the fourth week of January 2023), after that we tracked this type of diet on the health of children through protein consumption (insufficient protein intake, consequences of insufficient protein intake, kidney failure, excess protein intake, consequences of eating excessive protein, bone, cardiovascular risk, carcinogenesis), then fat consumption as it is broken down in the blood into four major components (fat overconsumption, fat deficiency), and finally carbohydrate consumption. All results made or provided by the institution are recorded.

2.3. The portions of food that constitute canteen meals

At school, children have to make enormous efforts from a young age in terms of attention and speed of processing information. The best academic results are achieved by children who can maintain their attention, alertness, and energy. These criteria are affected by heredity, and the social environment, such as lifestyle, diet, and living conditions, has the greatest effect. In this article, we presented the nutritional canteen of Al-Afak School as well as the impact of school canteen programs on the development of childhood obesity. Tables 1 - 8 present the nutritional canteen and the nutritional assessment for 4 weeks.

2.4. Statistical analysis

The statistical analysis of the results was performed using IBM SPSS Statistics v.26 software (IBM Corporation, Armonk, New York, USA). Descriptive statistics (Mean, Maximum, minimum, median, sum, standard deviation. SE of mean) were used to characterize our group. All analyses were performed with a set of risks of 5%, using the following guidelines for the interpretation of results: Involved children aged 6 to 12 (50 children), the evaluation of the school canteen program, the menu of the day meets the nutritional needs of a child, and finally, the meals influence the onset of obesity in children.

3. Results and Discussions

Descriptive statistics were first calculated to describe the nutritional status of the study sample stratified by children aged 6 to 12 years. They were also used to describe differences in the consumption of proteins, carbohydrates, and lipids for each daily menu, as well as the follow-up on children's health. Means and standard deviations were calculated for continuous variables. The statistical results obtained are illustrated in Table 9. Significant differences were observed between the nutritional elements (lipids, carbohydrates, and proteins) of the daily menu of children aged 6 to 12 years during the four days (see table 9). The average lipid nutrients consumed by children aged 6 to 12 years during the four days of the meal (Monday, Tuesday, Thursday, and Friday) vary respectively between 17.0 g and 41.73 g, as well as the minimum and maximum lipids consumed during the four days of the meal between 1.72 g and 20.60 g and between 42 g and 59 g, respectively (SD = 12–18 g). Then, the nutritional balance of carbohydrates consumed during the four days of the meal is distributed as follows: the average varies between 43 g and 80 g, the minimum varies between 5 g and 50 g, and the maximum carbohydrates consumed by children vary between 75 g and 102 g, as well as the standard deviation (SD = 12–34 g). Finally, the quantities of proteins consumed throughout the four days of meals are distributed as follows: the average is between 23 g and 65 g, the minimum of proteins consumed by the children is between 12 g and 36 g, the maximum of proteins consumed varies between 32 g and 122 g, and then the standard deviation is between 9 g and 48 g.

Table 1. The menu for week 1 of January

Days	Daily menu	Portions	Food	Nutrients in g
Monday	Moroccan salad	2 large spoons = 40g	Tomato, Onion Lettuce	0.04 lipids, 0.4 of proteins, 0.6 carbohydrates
	Turkey fillet	120g	Turkey	1.32g of lipids, 28.56g of protein, 0g carbohydrates, 89g of water
	Vegetable gratin	50g	Béchamel sauce (Milk /flour/butter), Carrots, Potato, Mushroom	4.23g of lipids, 1.6g of protein, 1.7g of carbohydrates, 41g of water
	An Apple	25g	Apple	0.05g lipids, 3.5g carbohydrate, 0.08g protein
Tuesday	Fruit salad	50g	Apple, Orange, Banana	0g lipids, 6.5g carbohydrates, 0.25g protein
	Eggplant with tomato sauce	50g	Eggplant, Tomatoes	0.14g lipids, 2.09g carbohydrates, 0.67g protein
	Baked sausage	120g	Sausage	0.24g carbohydrates, 9.36g protein, 14.4g lipids
	Mashed potatoes	100g	Potato; Butter, Milk, Cheese	4.2g lipids, 16.94 g carbohydrates, 1.96g protein
Thursday	Minced meat panini	175g	Bread, Minced meat sauce	33.25g lipids, 40.25g carbohydrates, 24.5g protein
	French fries	50g	Potato	6g lipids, 2.3g protein, 15g carbohydrates
	Apple salad	50g	Potato	1.05g protein, 8.6g carbohydrates, 0.69g lipids
	Caramel cream	50g	Eggs, Milk, Sugar	4.75g protein, 11.5g carbohydrates, 2g lipids
Friday	Couscous	200g	Couscous, Vegetables, Meat	1.2g lipids, 46.45g carbohydrates, 7.58g protein
	Yogurt Pear	1pot=125g	Dairy product	0.4g lipids, 3.6g carbohydrates, 10g protein
	Pear	50g	pear	0.12g lipids, 5.4g carbohydrates, 0.12g protein

Table 2. The nutritional assessment for week 1

Days/Nutrients	Lipids	Carbohydrates	Proteins
Monday	5.64g	5.8g	30.64g
Tuesday	18.74g	25.78g	12.24g
Thursday	41.94g	75.35g	32.6g
Friday	1.72g	55.44g	17.7g

Table 3. Menu for the second week of January 2023

Days	Daily menu	Portions	Food	Nutrients in g
Monday	Meat tagine	200g	Meat Potato Carrot Artichoke	27.81g protein. 38.2g carbohydrates . 11.47g lipids
	Moroccan salad	120g	Tomatoes. Onion. Cucumber	5.5g protein. 21.7g carbohydrates. 5.2g lipids
	A banana	1 fruit per child	Banana	1.06g protein. 19.7g carbohydrate. 0.39g lipids
Tuesday	Spaghetti minced meat	200g	Spaghetti. Tomato sauce. Minced meat	86.2g carbohydrate. 28.6g protein. 5.8g lipids
	Tuna and cheese salad	120g	Preserved tuna. Red cheese	41.5g protein. 32g lipids. 3.3g carbohydrates
	Fruit salad	100g	Strawberry. Orange. Banana	0g lipids. 13g carbohydrates. 0.5g protein
Thursday	Turkey Tacos	200g	Bread Turkey. Cheese	33.25g lipids. 40.25g carbohydrates. 24.5g protein
	Green bean salad	120g	Green bean	0.1g lipids. 7g carbohydrates. 1.8g protein
	French fries	100g	Potato	30g carbohydrate. 4.6g protein. 12g lipids
	An Apple	One fruit per child	Potato	0.2g lipids. 14g carbohydrates. 0.3g protein
Friday	Tuna pizza	200g	Pizza Paw. Tuna Cheese. Tomato sauce. Olives	16.70g lipids. 36.62g carbohydrates . 28.06g protein
	Rice and corn salad	120g	Rice. corn	7.20g lipid. 9.20g carbohydrate. 8.7g protein
	Yogurt	1pot=125g	Dairy product	0.4g lipids. 3.6g carbohydrates. 10g protein

Table 4. The nutritional assessment for week 2

Days/Nutrients	Lipids	Carbohydrates	Proteins
Monday	17.06g	79.6g	34.37g
Tuesday	37.8g	102.5g	70.6g
Thursday	45.55g	91.25g	31.2g
Friday	24.3g	49.42g	46.76g

Table 5. Menu for the third week of January 2023

Days	Daily menu	Portions	Food	Nutrients in g
Monday	Fish Tajine	200g	Vegetable. Fish	47g protein. 9.18g lipids. 0g carbohydrates
	Moroccan salad	120g	Tomato. Onion. Cucumber	5.5g protein. 21.7g carbohydrates. 5.2g lipids
	Pear	1 fruit per child	Pear	0.12g lipids. 5.4g carbohydrates. 0.12g protein
Tuesday	Baked Chicken	200g	Chicken	116g protein. 52.40g lipids. 2.34g carbohydrates
	Risotto	150g	Rice	6g protein. 50g carbohydrates. 0.6g lipids
	Fruit salad	120g	Strawberry. Banana. orange	0g lipids. 13g carbohydrates. 0.5g protein
Thursday	shawarma meat	200g	Bread. Meat. Tomato. Sauce	44g protein. 4g carbohydrates. 30g lipids
	French fries	100g	Potato	30g carbohydrate. 4.6g protein. 12g lipids
	Chocolate flan	100g	Milk. Eggs chocolate	2.09g protein. 4.6g lipids. 23.01g carbohydrates
Friday	Smoked turkey quiche	120g	Turkey. Quiche dough. Béchamel sauce. Cheese	11g protein. 23.5g carbohydrate. 19.2g lipids
	Turkey skewer	200 g	Turkey	33.25g lipids. 40.25g carbohydrates. 24.5g protein
	Banana	One fruit per child	Banana	1.06g protein. 19.7g carbohydrate. 0.39g lipids

Table 6. The nutritional assessment for week 3

Days/Nutrients	Lipids	Carbohydrates	Proteins
Monday	14.5g	27.1g	52.62g
Tuesday	53g	65.34g	122.5g
Thursday	46.6g	57.01g	50.69g
Friday	52.84g	83.45g	36.59g

Table 7. Menu for week 4 of January 2023

Days	Daily menu	Portions	Food	Nutrients in g
Monday	Chicken tagine	200g	Chicken. Vegetables	116g protein. 52.40g lipids. 2.34g carbohydrates
	Sauteed potato	120g	Potato	2g protein. 5.9g lipids. 16.7g carbohydrates
	Strawberry	3 fruits per child	Strawberry	0.6g lipids. 8g carbohydrates. 0.7g protein
Tuesday	Baked sausage	120g	Sausage	0.24g carbohydrates. 9.36g protein. 14.4g lipids
	Baked sausage Vegetable gratin	50g	Béchamel sauce (Milk /flour/butter). Carrots Potato. Mushroom	4.23g of lipids. 1.6g of protein. 1.7g of carbohydrates. 41g of water
	Caramel cream	50g	Eggs. Milk. Sugar	4.75g protein. 11.5g carbohydrates. 2g lipids
Thursday	Turkey sandwich	200g	Bread. Turkey. Tomato. Lettuce. Cheese	24g protein. 72.6g carbohydrate. 17.6g lipids
	Mashed potatoes	100g	Potato. Milk. Butter. Cheese	4.2g lipids. 16.94 g carbohydrates. 1.96g protein
	Yogurt	1pot=125g	Dairy product	0.4g lipids. 3.6g carbohydrates. 10g protein
Friday	Chicken pastilla	200g	Pastilla paste Almond Chicken	24.6g protein. 28.1g carbohydrates. 24.4g lipids
	Lemon juice	125ml	Lemon. Sugar. Water	0g protein. 12g carbohydrates. 0g lipids

Table 8. The nutritional assessment for week 4 of January

Days/Nutrients	Lipids	Carbohydrates	Proteins
Monday	58.9g	27.4g	118.7g
Tuesday	20.63g	13.44g	11.93g
Thursday	22.2g	93.14g	35.96g
Friday	24.6g	40.1g	26.6g

In light of this study, we can conclude that the maximum lipid consumed by children reaching 59 g of lipid was recorded on the fourth week of Monday; on the other hand. The minimum lipid consumed of 1.72 g was recorded on Friday of the first week. Then, the maximum carbohydrates consumed by the children, reaching 102 g of carbohydrate, were recorded the second week on Tuesday, on the other hand, the minimum lipid consumed, 5.8 g, was recorded on Monday of the first week. While the maximum amount of protein consumed by children is 122.5 g, this was recorded on Tuesday of the third week.

Monday: We noticed that the fat content of Monday's dish (5.64g) is lower than the portion size requested, which should be between 12 and 16.5. The same carbohydrate content (5.8g) is much lower than what we asked for the third protein nutrient. Higher than recommended; after calculation, we found protein = 30.64g, whereas the portion size requested should be between 4.5g and 6 g (**Fig. 1**).

Tuesday: For lipids, the value of 18.7 g is slightly higher than the requirement of 12 to 16.5 g. We note that carbohydrates are consistently lower than the recommended 25.78 g, so proteins are higher than the 12.24 g portion (**Fig. 2**).

Thursday: Thursday's meal contains 1.72 grams of fat, which is well below the ideal fat for breakfast (12 to 16.5 grams), and the carbohydrates on the day's menu are well below the required value of 75.35 grams but not the recommended value of 32.6 grams. Compared with this the protein has increased again (**Fig. 3**).

Friday: We noted that the ratio of fat and carbohydrates in the meal was below the required 1.72 g and 55.44 g, while the amount of protein in the Friday menu was well above the recommended value of 17.7 g.

3.2 Week 2 of January 2023

Monday: For today's menu, the fat and protein content of the food is higher than the 17.6 g and 34.37 g recommended, but the carbohydrate content is 79.6 g (see **Fig. 4**), within the range recommended by EFSA (69 g to 84 g).

Tuesday: At today's meal, all nutrients are higher than recommended Protein = 70.6g / Fat = 37.8g / Carbohydrates = 102.5g (**Fig. 5**)

Thursday: The nutrient content of the dishes on offer was also very high in relation to the required values, we found protein = 31.2g, carbohydrate = 91.25 g, and fat = 45.55 g.

Friday: The fat in the day's meal was 24.3 grams higher than required (from 12 to 16.5 grams), but the carbohydrate content of this dish was very low at 49.42 grams (from 69 grams to 84 grams), and the protein (47.76 grams) is still a substantial increase on the recommended intake for children (**Fig. 6**).

3.3 Week 3 of January 2023

Monday: The daily meal contains 14.5 g of fat, a reasonable amount compared to the recommended carbohydrates. Although the value is very low at 27.1 g, we

can still see an increase in protein compared with the required range of 52.62 g (**Fig. 7**).

Tuesday: The dishes of the day have high fat and protein values compared with the recommended values of 53 g and 122.5 g, respectively, but not for carbohydrates, which are much less comparable in this dish.

Thursday: Note that this day is high in fat and protein (46.6 g and 50.69 g), and 57.01 g of carbohydrates are listed, which is very low compared to what you need (**Fig. 8**).

Friday: The lunchtime meal contains a good amount of carbohydrates (83.45 g), which is within the required range (69–84 g), but protein and fat levels are below requirements of 36.59 g, and 52.84 g, respectively.

3.4 Week 4 of January 2023

Monday: At breakfast, we found that fat and protein were higher than the required 58.9 g and 118.7 g, but the amount of carbohydrates was low compared to the recommended value of 27.4 g, giving us

Tuesday: The amount of nutrients in a breakfast meal is shown as follows: Fat = 20.63 g; carbohydrate = 13.44 g. Protein = 11.93 g (**Fig. 9**).

Thursday: The daily meal contains 22.2 g of fat, more than recommended; 93.14 g of carbohydrates, again excluding the ideal carbohydrate range; and 35.96 g of protein (**Fig. 10**).

Friday: Note that the amount of protein is the same. The amount of carbohydrates in this meal is less than the recommended amount of 40.1 g, and the amount of protein in this meal is more than the recommended amount.

The results show over-consumption of proteins, carbohydrate deficiency, and the value of lipids varies from one meal to another.

4. Discussion

4.1 Protein consumption

The consumption of proteins of animal or plant origin has become a critical factor in terms of both health and the environment. In this context, it seems important to understand the early factors associated with the appreciation and consumption of foods rich in animal or plant proteins in children [7]. In the early 1950s, estimated protein requirements were much higher than the values currently used. Early observations in Africa showed that children suffering from kwashiorkor frequently consumed diets low in animal protein compared with the diets of children in rich countries [8].

4.1.1. Insufficient protein intake

Protein deficiencies need to be identified, prevented, and corrected. The circumstances in which deficiencies occur are numerous, particularly as a result of new dietary practices and the explosion in bariatric surgery, obesity... which explains the frequency of these nutritional situations at all ages. The consequences of protein deficiencies can be severe, especially as they are rarely isolated [9]. In adolescents, and more particularly in adolescent girls the refusal to eat meat could lead to protein deficiencies. Although meat is not essential (only the nutrients are essential), if there are no other sources of animal protein (ovo-lactate and marine) a vegan diet leads to low intakes not only of protein but also of iron, calcium, zinc, iodine, and long-chain omega-3 fatty acids [10]. Which can slow growth and alter lean body mass (oste-

muscular) thus, reducing the peak bone mass reached around the age of 20. It also seriously alters vitamin B12 status [11]. Vegetarian diets, on the other hand, are compatible with satisfactory protein and calcium intakes [12]. In adolescence in addition to eating patterns with specific exclusions, anorexia nervosa is also a frequent occurrence, leading to severe deficiencies and sometimes life-threatening emaciation. It is also worrying to note that, from an epidemiological point of view, in France, thinness increased sharply among 7-9 year olds between 2000 and 2016 [13], and among 3-17 year olds between 2006 and 2015]. This could be a perverse effect of public health messages about the risk of obesity, which are poorly relayed by certain health professionals and/or poorly understood by parents, and which can exacerbate the thinness ideal. More and more mothers are exercising excessive control over their children's dietary intake [14].

4.1.2. Consequences of insufficient protein intake

If the diet is vegetarian (ovo-lacto-vegetarian), protein intake may be sufficient. On a vegan diet (with no animal products), the risk of protein deficiency is low, but the risk of iron, calcium, zinc, omega-3, and iodine deficiency is high. A vitamin B12 deficiency is delayed [15]. This will affect the child's growth and may lead to mixed iron deficiency and megalocytic anemia! Vitamin B12 deficiency impairs cognitive function in adolescents [16]. Peak bone mass may be reduced. There is no life-threatening risk. It is essential to supplement people on a vegan diet with vitamin B12. It should be remembered that the consequences of vitamin B12 deficiency set in after around 3–4 years. However, if the choice and quantities of plant proteins are adequate, in particular by favoring soya and paying attention to cereal and dried vegetable supplementation, protein intake may be sufficient. In the course of anorexia nervosa, energy and protein intake are extremely low, leading to emaciation, which may temporarily justify artificial feeding. Although no longer a diagnostic criterion, anovulation and amenorrhoea very often occur.

4.1.3 On renal failure

Despite the theoretical arguments and numerous animal studies proving the efficacy of a reduced protein intake in slowing the progression of renal failure, current knowledge does not provide a definitive answer to the question of whether this reduction can have the same effect in humans. Most non-randomised studies show a protective effect on renal function of a protein-restricted diet. However, if we examine the results of the seven randomised studies involving more than 100 patients [17], two reports an effect of the protein-restricted diet but without quantifying the loss of function, one reports a limited effect and four find no effect, including the only study conducted in children. In this study [18], the group on a diet theoretically limited to the minimum recommended by the World Health Organisation (WHO) even lost more glomerular filtration rate than the group on an unrestricted protein diet after 3 years of observation. The same was true of the major study carried out in the United States on over 500 patients observed for 3 years [19].

4.1.4 Excess protein intake

Some sports, particularly resistance sports, lead to excessive protein intake. In these closed environments, inappropriate speeches and advice lead followers, especially boys and young men, to ingest "protein supplements" in excessive and inappropriate quantities. The preparations are based on skimmed milk powder and, increasingly, vegetable protein powders (peas, soya) with various incentives to over-consume these products, sometimes in addition to high or very high intakes of eggs or meat in order to increase muscle mass [20].

4.1.5 Consequences of excessive protein intake

The issue of excessive intakes is more complex in terms of its consequences very high protein diets can be observed. Up to 2.2 g/ kg/day, there is no deleterious effect recognised by the Agence nationale de sécurité sanitaire de l'alimentation, de l'environnement et du travail (ANSES) in the absence of renal failure [21]. However, this excessive intake is unnecessary, even if it is sought after in certain sporting activities. When sport is stopped, obesity may set in. Excessive intakes of eggs (up to 10 or more per day) will lead to hypercholesterolaemia. Excessive intakes of red meat are associated with increased cardiovascular and carcinogenic risk.

4.1.6 on the bone

It is now known that excess animal protein can contribute to the acidification of the internal environment, and therefore lower the pH [22]. However, we now know that this is not limited to proteins rich in sulphur-containing amino acids (i.e. animal proteins), but is also observed in plant proteins (excess of which is, however, less easy to achieve [23]). However, this is particularly true if excretion of H⁺ ions is reduced as a result of impaired renal function. Moreover, this is corrected by a sufficient intake of fruit and vegetables (alkalinising) [24]. Finally, while acidification of the internal environment may lead to a leakage of calcium from the bones, this is compensated for by adequate calcium [25] or dairy product intake.

4.1.7 On cardiovascular risk

Although studies have shown associations between animal proteins and ischaemic heart disease [26]. The negative role of animal proteins has not been established. Rather it appears to be the deleterious effect of excessive meat consumption, combined with low consumption of 'protective' plant foods.

4.1.8 On carcinogenesis

It cannot be ruled out that excess protein plays a role in cell proliferation, probably via endogenous production of IGF-1, which could increase the risk of adenoma or fibroma. This could go as far as a tumour-promoting role, but in no case as far as an initiating effect (mutagenesis). In the strictest sense of the word, therefore, this is not a carcinogenic effect. For the moment, this hypothesis has been rejected by ANSES with regard to excess consumption of dairy products [27]. However, no excess can be encouraged. Situations where there is a risk of protein deficiency are common. They can affect all age groups: infants who replace milk with vegetable juices, teenagers with vegan diets, and people on restrictive weight-loss diets or after bariatric surgery.

The consequences vary but can be severe. ranging from stunted growth to loss of bone mass and undernutrition. Situations where there is a risk of excess are less frequent and less immediately dramatic. They may affect infants. teenage athletes or people on unbalanced diets. Long-term consequences exist, with various somatic repercussions that are discussed.

4.2 Consumption of lipids

4.2.1 Lipids in the blood are divided into four main components

Phospholipids. which make up cell membranes. Lipids are insoluble in their own right in the blood. so they circulate bound to proteins: these are known as lipoproteins. Depending on their density. there are chylomicrons. Very low density lipoproteins (VLDL). Low density lipoproteins (LDL) and High density lipoproteins (HDL). The lipid composition of each varies. In a healthy subject. 80% of triglycerides are found in VLDL and 75% of cholesterol in LDL the distribution of estimated usual intakes of total lipids. various fatty acids and cholesterol. The median intake of total lipids. higher in boys than in girls. was 57g and 53g per day respectively. In terms of the different types of fatty acids. the median usual intakes of saturated and monounsaturated fats were around 21 g in both cases for boys. and around 20 g and 19 g respectively for girls. As for polyunsaturated fats. the median usual intake is around 9g a day for boys and almost 8g a day for girls. For linoleic acid the median usual daily intake is estimated at 7g for boys and 6g for girls. while for α -linolenic acid it is around 1g for both sexes. In all cases. boys have higher median intakes than girls [28].

Only linoleic acid (from the omega-6 family) and α -linolenic acid (from the omega-3 family) have reference values (AI). which are based on median intakes in a population that apparently has no deficiencies in these fatty acids. In the case of linoleic acid. regardless of gender. median usual intakes appear to be below the AI value of 10g (IOM. 2002). Moreover. even the 75th percentile estimate is below this value. In the case of α -linolenic acid the median usual intake for boys corresponds to the AI value of 0.9 g. while for girls it is slightly lower (0.8 g). Thus. in the case of α -linolenic acid. the results for boys suggest that the likelihood of inadequate intakes is low. However. for linoleic acid. in both sexes. and for α -linolenic acid in girls. it is not possible to comment on the risk of inadequate intakes in this population [29]. As for cholesterol the estimated median usual intake was higher in boys than in girls. at 178 g compared with 160 g per day. Although there is no established reference value (DRI) for this fatty substance. it is nevertheless recommended that consumption be limited. More specifically. with a view to preventing chronic disease. it is generally recommended that. for the population as a whole. intake of this nutrient should not exceed 300 mg per day. However. according to the survey data. a very small proportion of children have a usual daily intake exceeding 300 mg (less than 1%) [30].

4.2.2 Over-consumption of fats

The quantitative and qualitative aspects of excess lipid levels have been particularly studied in developed countries. Excessive weight and the dyslipidaemia it causes are responsible for a very high morbidity and mortality rate. and the progressive westernisation of the diet of the Ibbat et al., 2023

inhabitants of the major cities of black Africa is also threatening them. Coronary heart disease currently has a low prevalence among Africans. It ranks 5th among cardiovascular diseases in the CORONAFRIC study [31]. with a prevalence of 3.17% of hospitalised patients. well behind arterial hypertension (39%). cardiomyopathy (16%) and rheumatic valve disease (16%). In industrialised countries. it ranks 2nd among cardiovascular diseases. At the Abidjan Heart Institute. 5.3% of Africans and 39% of Europeans are hospitalised for coronary heart disease. This relative protection against coronary heart disease seems to be linked less to an ethnic factor than to environmental factors. particularly diet. While cholesterol levels in Westerners and Africans are identical at birth [31]. they are much lower in the latter group in adulthood [32]. With economic development. there is reason to believe that the incidence of coronary heart disease will worsen: because of the increase in fat intake. which is proportional to the increase in gross national product. and because of the increase in life expectancy in Africa (38 years in 1950, 50 years in 1980). which will allow clinical expression. This trend towards an increase in coronary heart disease is currently real. but it is small [31]. and predominantly affects the more affluent sections of the population. In addition to coronary heart disease. certain conditions for which dietary factors are well known could see their prevalence rise. Obesity and its many consequences immediately spring to mind. but we can also expect an increase in cholesterol lithiasis and certain cancers such as colon and breast cancer. for which the role of excess lipids has been incriminated on the basis of epidemiological and experimental data. These diseases are currently rare in the African population. A global nutritional policy is difficult to determine in African countries today. A small proportion of the population has an excessive lipid intake with pathological consequences similar to those seen in rich countries; the majority of the population has an overall lipid deficiency which. However. does not seem to lead to any specific pathology. as the endogenous lipidogenesis of carbohydrates compensates for the dietary fat deficiency. This apparently protective deficiency should not blind us to two particular nutritional characteristics of lipids: their high nutritional power and their organoleptic qualities.

4.2.3 Lipid deficiency

Lipid deficiencies are exceptionally responsible for pathological manifestations. except in very specific circumstances where essential fatty acid intakes are no longer guaranteed (unbalanced artificial diet, deficient newborns). However. this deficiency has been cited as a cause of juvenile tropical pancreatitis (JTP). Juvenile tropical pancreatitis is a chronic calcifying pancreatitis that differs from alcoholic pancreatitis in a number of epidemiological respects: it occurs exclusively in tropical areas. no alcohol is consumed. onset is in adolescence or young adulthood. and the sex ratio is irrelevant. A number of pathogenic hypotheses have been put forward for JTP: protein-energy malnutrition [33]. kwashiorkor in childhood [34]. hyper-consumption of manioc [35]. viral. immunological and genetic factors. None of these has been fully confirmed by comparative epidemiological studies. The role of lipid deficiency was suggested as early as 1978 by Durbec and Sarles. and then supported by multicentre nutritional surveys revealing a similar geographical distribution between PTJ and major lipid

deficiency. Other studies have not found this role of lipids in the onset of PJD. The pathogenesis of this condition is probably complex, including protein-energy malnutrition but also genetic and environmental factors. The role of lipid deficiency in the genesis of tropical ataxias has been suggested on the basis of a case-control study showing a statistically significant association. However, no causal relationship can be established, as a number of associated factors, such as high manioc consumption, may explain both the low lipid content of the diet and the occurrence of neurological disorders. The specific pathology associated with lipid deficiency therefore appears to be very limited, but its role in overall energy deficiency is certainly predominant [36]. The lipidophobia of parents and the rejection of lipids by some health professionals over a long period have undoubtedly been more harmful for children than for adults. In young children, current fat consumption, as a percentage of energy intake, is well below the recommendations. Consumption measurements described in the French EDEN cohort (comprising 1,275 children aged 8 to 12 months) show that 95% of 8-month-olds have fat intakes that are below their needs, and that less than 5% of them reach the threshold of 40% of energy from fat (the low value recommended by the Food and Agriculture Organization of the United Nations FAO) [37]. At the same time, this study shows that 95% of these children reach or exceed a proportion of 45% of energy from carbohydrates. The Anses recommends a fat intake of 45-50% of total energy for these ages, in infant foods and follow-on formulas. Children's energy requirements are very high between birth and 1 year of age, as well as between 1 and 3 years of age, due to a very intense basal metabolism linked to their very strong growth, particularly during the first 6 months [38]. The development of the nervous system (central and peripheral) and the myelination of nerves also require specific lipids, including essential fatty acids [39]. In 2013, the French Nutri-Bébé survey showed that 80% of children aged over one year had intakes below the values recommended by the European Food Safety Authority (EFSA) [40]. The choice of dietary lipids is important as part of a strategy to reduce cardiovascular risk factors, morbidity and mortality, obesity, and type 2 diabetes. Advances in knowledge have led to a better understanding of the role of diet in the development of cardiovascular disease. Lipid nutrients are not a homogenous group. We now know that not all SFAs are harmful and not all PUFAs are protective, but we also know that sterols have different effects depending on their origin.

4.3 Carbohydrate consumption

There is currently a strong presumption that the consumption of free sugars, and sweetened beverages in particular, plays a role in the pathogenesis of obesity and its co-morbidities. This presumption is based on the plausible mechanisms by which the fructose content of sugars disrupts carbohydrate and lipid stimulation, exerts oxidative stress, and provokes toxic secondary mediators, particularly in the liver. Data from large cohort studies showing a positive association between sugar consumption and the risk of developing obesity, diabetes or cardiovascular disease is compatible with the hypothesis of a deleterious effect of excessive sugar consumption [41]. Carbohydrates are divided into sugars, or simple carbohydrates, and starches, or complex carbohydrates [42]. The main dietary sugars are

monosaccharides (glucose, fructose, galactose) and disaccharides (sucrose, lactose, maltose). They are naturally present in fruit and vegetables, as well as in natural syrups such as honey, maple syrup and agave syrup. Sucrose is also isolated from sugar cane or sugar beet to produce crystalline sucrose. Lastly, syrups known as glucose-fructose syrup or isoglucose (often referred to in the Anglo-Saxon literature as "High Fructose Corn Syrup", HFCS) containing glucose and fructose in varying proportions, are prepared industrially from cereals or potatoes [43]. Sugar consumption has increased considerably over the last two centuries, reaching an average of 10-20% of total energy intake in most European countries, or an average of 100-150g of total sugars excluding lactose, of which 50-75g is fructose. In France, 20-30% of the adult population consumes more than 50g of fructose a day [42]. Some of the sugars consumed are naturally present in staple foods such as fruit, vegetables and dairy products. However, a portion of our daily intake comes in the form of so-called 'free' or 'added' sugars in sweetened drinks, biscuits, chocolate and other sweet snacks, etc. There is currently a strong suspicion that an excessive dietary intake of 'free' sugars may lead to the development of metabolic diseases. It has been suggested that the fructose component is mainly responsible for the deleterious effects of this class of sugars [44]. Non-communicable diseases such as obesity, diabetes mellitus, and cardiovascular disease, cancer and neurodegenerative diseases are nowadays the major cause of morbidity and mortality in most of the world's countries. These diseases are influenced by environmental factors, including diet [45]. Moreover, the prevalence of obesity has increased dramatically since the mid-twentieth century, and is a predisposing factor in the development of other non-communicable diseases. The suspicion that consumption of fructose-containing sugars plays a role in the pathogenesis of some of these diseases, in particular obesity, type 2 diabetes and cardiovascular disease, is based mainly on epidemiological data. Large prospective studies report an association between sugar consumption and weight gain over time. This association is explained by increased energy intake in subjects with a high sugar intake. Most of these prospective studies looked specifically at the effects of sugar-sweetened beverage consumption. They show a positive relationship between the intake of sugary soft drinks and weight gain [46]. They also show a positive relationship between sweetened beverage intake and overall mortality. Unexpectedly, this relationship was observed not only with caloric sweetened beverages such as soft drinks and fruit juices, but also with non-caloric artificially sweetened beverages, suggesting the possible presence of confounding factors in these analyses [47]. In these cohort studies, it should also be noted that sweetened beverages are only one of several dietary factors, such as the consumption of Chips and crisps, red meat and cold meats, associated with weight gain [48]. Finally, there is a positive association between the intake of sweetened beverages and the risk of diabetes [49], cardiovascular disease [50] and gout [51]. These relationships are partly independent of the degree of adiposity. Furthermore, the development of industrial techniques for preparing glucose-fructose syrups in the 1960s led to a spectacular increase in the consumption of HFCS in the USA at the end of the 20th century. This increase in consumption coincided with a marked rise in the prevalence of obesity, leading to the suggestion that the fructose contained in these syrups could

play a pathogenic role [52]. Dietary sugars account for around 10-20% of total energy intake in European countries. The fructose component of dietary sugars could play a role in the pathogenesis of metabolic diseases. Fructose is mainly metabolised in cells of the small intestine, liver and proximal convoluted tubules of the kidney expressing the enzymes fructokinase, Aldolase B and triokinase. In these cells, fructose is completely degraded to triose phosphate in an unregulated manner, and then to lactate, glucose, glycogen or fatty acids.

In healthy subjects, short-term fructose overfeeding is accompanied by the development of hepatic insulin resistance, an increase in blood triglycerides, intrahepatic triglyceride storage and an increase in uric acid concentration. In the long term, these changes could lead to the development of obesity and its cardiometabolic complications. Public health initiatives aimed at reducing the consumption of 'free' sugars have been introduced in several countries. Their long-term effect on sugar consumption and the incidence of metabolic diseases is not yet known.

Table 9. The descriptive statistical study of the nutritional assessment

Nutrients	N total	Mean	Standard Deviation	SE of mean	Sum	Minimum	Median	Maximum
Week 1								
<i>Lipids</i>	4	17.01	18.14	9.07	68.04	1.72	12.19	41.94
<i>Carbohydrates</i>	4	40.60	30.86	15.43	162.37	5.8	40.61	75.35
<i>Proteins</i>	4	23.30	9.90	4.95	93.18	12.24	24.17	32.6
Week 2								
<i>Lipids</i>	4	31.17	12.87	6.43	124.71	17.06	31.05	45.55
<i>Carbohydrates</i>	4	80.70	22.84	11.42	322.77	49.42	85.42	102.5
<i>Proteins</i>	4	45.73	17.88	8.94	182.93	31.2	40.56	70.6
Week 3								
<i>Lipids</i>	4	41.73	18.40	9.20	166.94	14.5	49.72	53
<i>Carbohydrates</i>	4	58.22	23.50	11.75	232.9	27.1	61.17	83.45
<i>Proteins</i>	4	65.6	38.60	19.30	262.4	36.59	51.65	122.5
Week 4								
<i>Lipids</i>	4	31.58	18.28	9.142	126.33	20.63	23.4	58.9
<i>Carbohydrates</i>	4	43.52	34.82	17.41	174.08	13.44	33.75	93.14
<i>Proteins</i>	4	48.30	47.96	23.98	193.19	11.93	31.28	118.7



Figure 1. The nutritional balance of carbohydrates during week 1 of January 2023

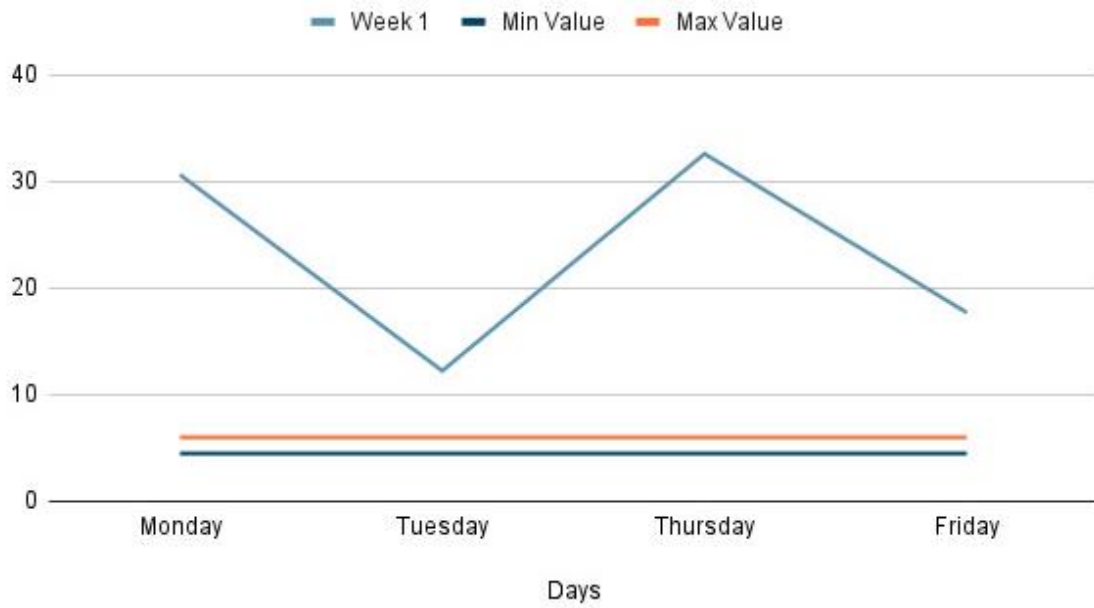


Figure 2. The nutritional balance of the protein during week 1 of January 2023



Figure 3. The nutritional assessment of lipids during week 1 of January 2023

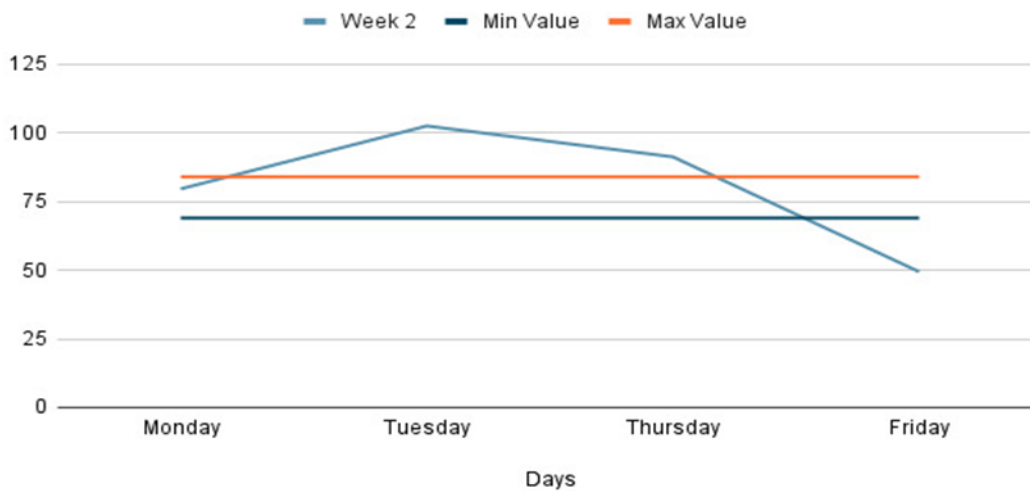


Figure 4. The nutritional balance of carbohydrates during week 2 of January 2023

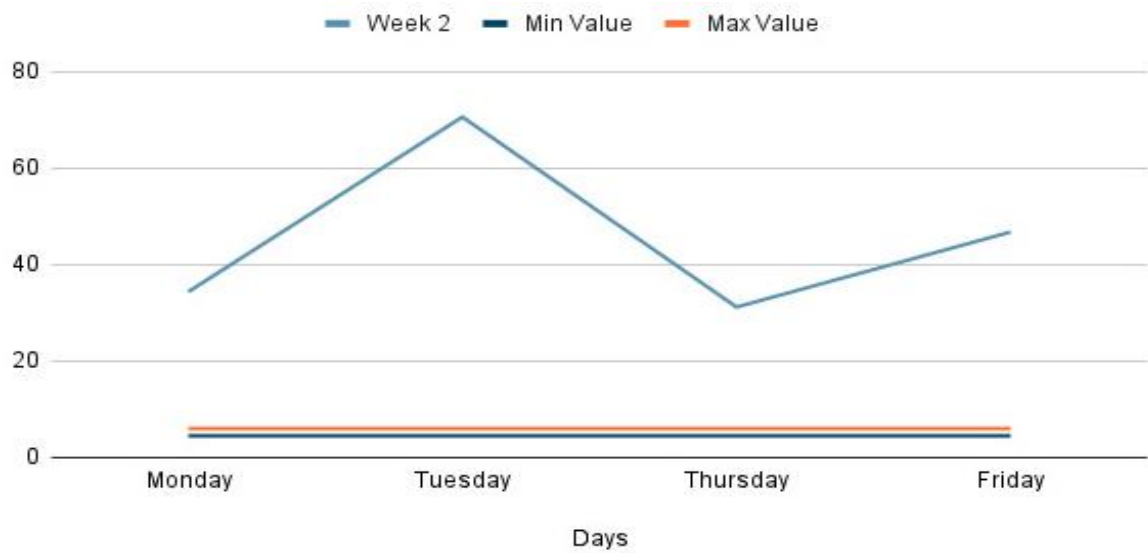


Figure 5. The nutritional balance of the protein during week 2 of January 2023

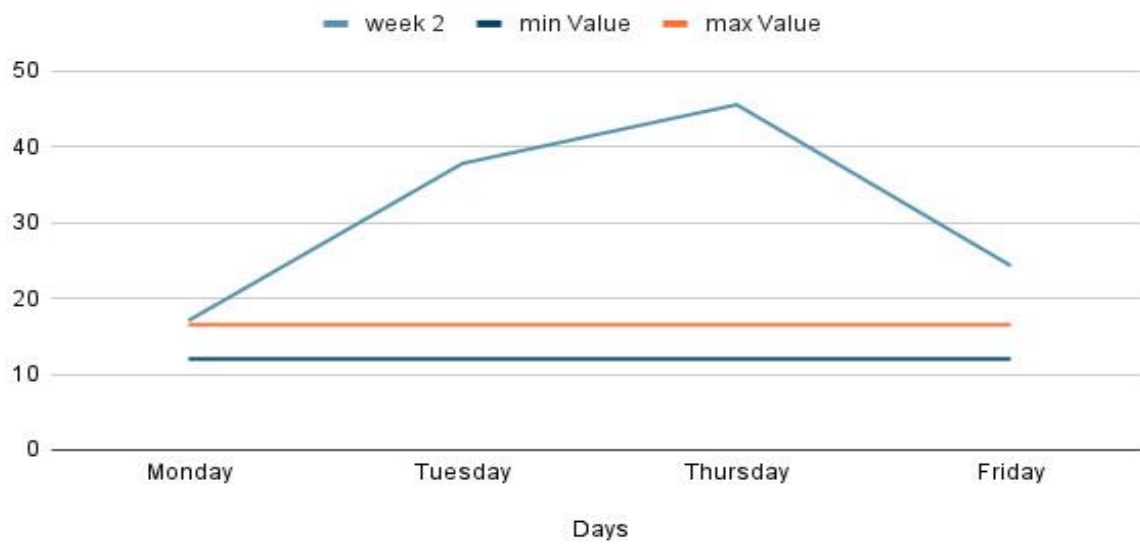


Figure 6. The nutritional assessment of lipids during week 2 of January 2023



Figure 7. The nutritional balance of carbohydrates during week 3 of January 2023

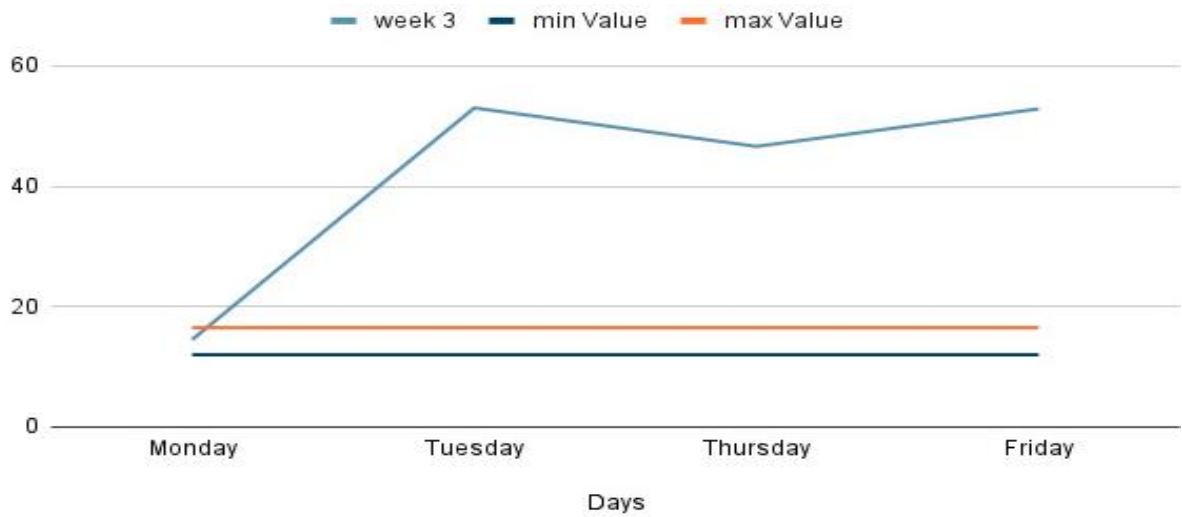


Figure 8. The nutritional assessment of lipids during week 3 of January 2023

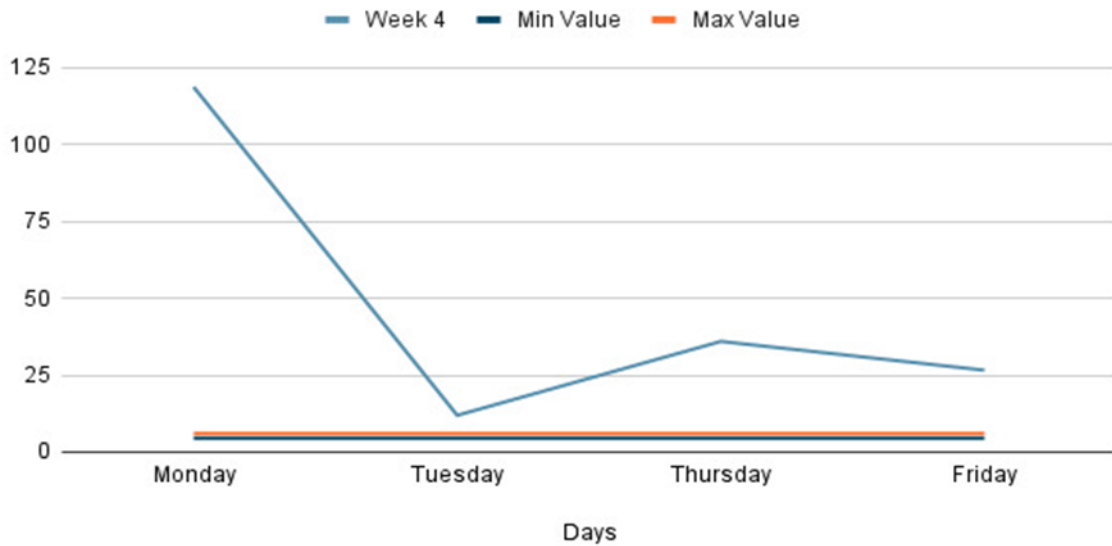


Figure 9. The nutritional balance of carbohydrates during week 4 of January 2023

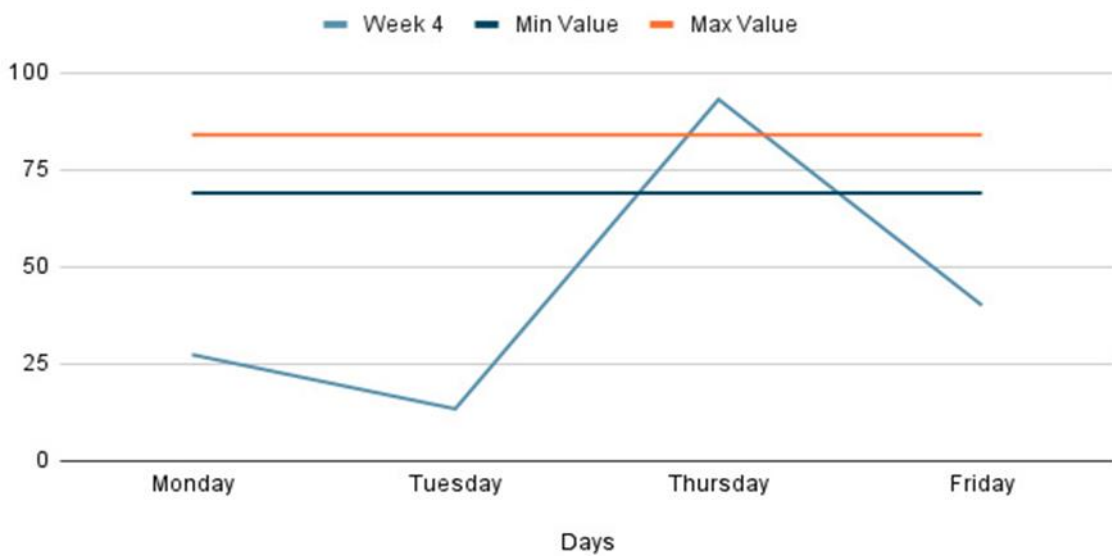


Figure 10. The nutritional balance of the protein during week 4 of January 2023

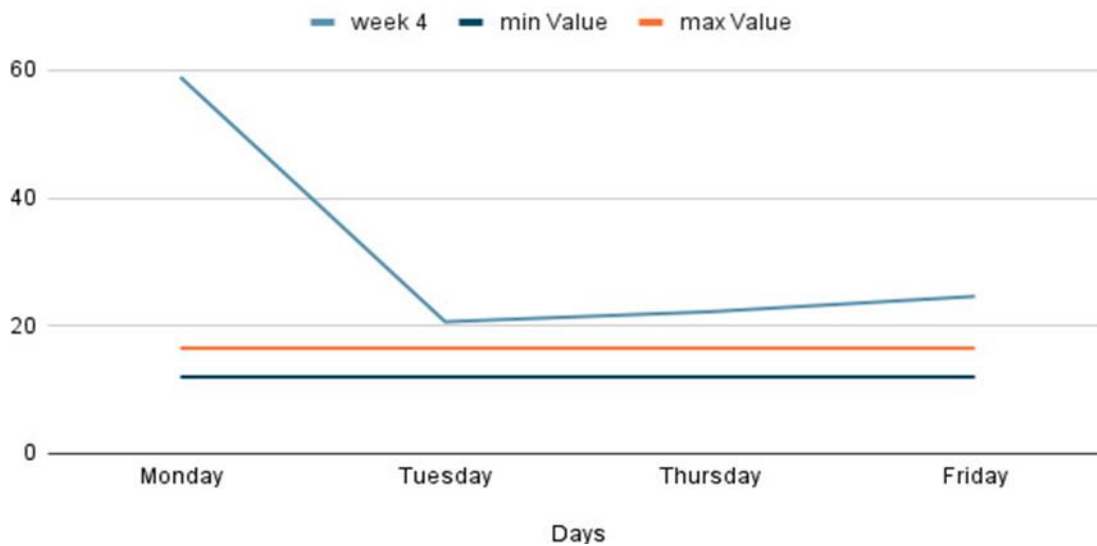


Figure 11. The nutritional assessment of lipids during week 4 of January 2023

5. Conclusion

Canteens have no place in preventing childhood obesity. Firstly, it has little quantitative impact. On average, a child eats 140 days a year in the canteen. Energy intake is broken down as follows: 20% for breakfast, 20% for snacks, 30% for lunch, and 30% for dinner. The energy intake for this lunch therefore represents 11.5% of the total energy intake for a full year (365 days). In addition, the results of our study show an overconsumption of proteins and a deficiency of carbohydrates, and the value of lipids varies from one meal to another. These nutritional values could encourage eating disorders but not have an intense impact on the onset of obesity. It is now well established that childhood obesity is a multifactorial constitutional disease. The obesogenic environment of industrialized countries, combining a sedentary lifestyle with an abundance of food, encourages the onset of obesity in children with a genetic predisposition to weight gain. Ignoring this predisposition is tantamount to believing that obesity is simply the consequence of a 'bad diet, for which children and parents alone are responsible.

Declarations

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Availability of data and materials

The datasets used and analyzed during the current study are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interest.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Ethics approval and consent to participate

Not applicable.

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