LIPID COMPOSITION OF THE JERKY MEAT (KILISHI) CONSUMED IN NIGERIA

Supported by tetfund

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Received: April 26, 2020 **Accepted:** June 19, 2020

Abstract: We have reported on the lipids constituent of kilishi. The followings were observed on this unit (g/100g): crude fat (14.2); fatty acid (13.0); other lipids (1.19); whereas energy results from the weights were (kJ/100g and kcal/100g): crude fat (525/128); fatty acid (481/117); other lipids (44.1/10.7). The SFAs ranged from 0.00 - 25.3% of total FA leading to total SFA of 33.4% with corresponding edible portion of FA (EPg/100g) range of nd to 3.30 and total of 4.34. For the monounsaturated fatty acids (MUFAs), FA range was 3.04e-4 to 21.9% with a total of 41.2% and corresponding EPg/100g range of 3.95e-5 to 2.84 and total of 5.36. For ω6 (n-6) PUFA, FA range was 1.32e-3 to 13.1% with a total of 18.9% and corresponding EPg/100g of 1.72e-4 to 1.70 and total of 2.46. For the ω3 (n-3) PUFA, FA range was 5.72e-3 to 4.85 with total of 6.54 and corresponding EPg/100g range of 7.44e-4 to 6.30e-1 with total of 8.51e-1. The energy contribution from the various constituents were (kJ/100g and kcal/100g): SFA (161/39.1); MUFA (198/49.2) and PUFA (122/29.8); meaning that energy contribution ran thus: MUFA>SFA>PUFA. The values of the following quality parameters were relatively high: MUFA/SFA, PUFA/SFA, EPA/DHA, LA/ALA, AA/DGLA, EPSI, C16: O: C18:1 cis-9, %C16: O in SFA, %C18: O in SFA, AA/EPA + DHA and TUFA. The sterol levels were with a total value of 81.1 mg/100g; cholesterol predominated at a value of 74.0 mg/100g (91.2%). The phospholipids levels were all high based on their high percentage levels at total value of 5586 mg/100g which ranged between 192-3262 mg/100g. Phosphatidylcholine (PC) predominated (3262 mg/100g and 58.4%). Hence, kilishi lipid is good nutritionally for man.

Keywords: Lipid, constituents, Nigerian, kilishi, meat product

Introduction

Kilishi (kilichi) meat in Nigeria is a version of jerky meat. It originated in Hausaland in the Northern part of Nigeria. It is a derivation form of suya, which can be made from deboned cow, sheep or goat meat. Selected muscle is normally sliced into sheets of about one metre or less; this makes for easy drying. The dried sheets of meat would then be collected and kept for the next processing stage (Wikipedia) [Jerky is lean trimmed meat that has been cut into strips and dried (dehydrated) to prevent spoilage.]

The drying process normally includes the addition of salt to prevent bacteria growth before the meat dehydration process is completed (Wikipedia) kilishi preparation takes about three stages which are shown below:

A paste that is made from peanut called *labu*, is diluted with enough water, spices, salt, ground onions and sweetener such as honey, to add sweetness. Usually a more natural way to add sweetness is to add date palm. The dried "sheets" of meat are then immersed one by one into the *labu* paste to coat them, before being left to dry for hours before roasting to taste (Nigeria Today, 2016).

After roasting, the final moisture content usually ranged between 10-12%, this value range decreases during storage at room temperature to 7.0%. When packaged in hemetically sealed low density plastic pack of 0.038 mm thickness, kilishi remains appreciably stable at room temperature for a period of about one year (Wikipedia).

Other ingredients or treatments to improve the quality of kilishi are: use of suya spice (suya pepper); cloves of garlic; one teaspoon of cloves (kanafuru); piece of ginger; dry cayenne pepper seeds. The meat used for kilishi should be free from fat and should be gotten from the red part of the beef. Each of these items give kilishi special characteristics such as aroma, etc. (Umuoke, 2018).

The above had given some information about the preparation, nutritional characteristics and the ingredients that improve the food quality of kilishi. Searching through literature, there is paucity of information on the lipid constituent of kilishi. The major concern of this report is to exhibit the lipid profile of kilishi as revealed to us from the chemical analysis of kilishi. The data obtained would also be discussed to show the health implications of the consumption of kilishi meat.

Materials and Methods

Collection of samples

Samples of packaged kilishi were purchased from RAO Supermarket in Ado-Ekiti, Ekiti State, Nigeria. The kilishi samples were actually prepared for sale by Golden Datol Ent., Akure, Ondo State, Nigeria. The kilishi sample was labelled as containing beef, onion, garlic, salt, honey, ginger, maggi, groundnut and pepper.

Sample treatment

The kilishi pack were further dried at 50°C in the oven, allowed to cool, blended and packaged in plastic containers and kept in the refrigerator (2.8°C or 37°F) pending analysis. A typical kilishi sample is shown in Fig. 1.

Fig. 1: Nigerian jerky meat (kilishi)

Extraction of lipid

0.25 g of the sample was weighed into the extraction thimble. 200 ml of petroleum ether (40-60ºC boiling range) was measured and then added to the dried 250 ml capacity flask. The covered porous thimble with the sample was placed in the condenser of the Soxhlet extractor arrangement that has been assembled (AOAC, 2006). The lipid was extracted for 5 h. The extraction flask with oil was oven dried at 105ºC for 1h. The flask containing the dried oil was cooled in the desiccator and the weight of the cooled flask with the dried oil was measured.

Preparation of methyl esters and analysis

50 mg of the extracted oil was saponified for 5 min at 95ºC with 3.4 ml of 0.5M KOH in dry methanol. The mixture was neutralized by 0.7M HCl. 3 ml of 14% boron trifluoride in methanol was added (AOAC, 2006).The mixture was heated for 5 min at 90ºC to achieve complete methylation process. The fatty acid methyl esters were trice extracted from the mixture with redistilled n-hexane. The content was concentrated to 1 ml for analysis and 1µl was injected into the injection pot of GC. The fatty acid methyl esters were analyzed using an (HP 5890 powered with HP ChemStation rev. AO9.01 [1206] software [GMI, Inc, Minnesota, USA]) fitted with a flame ionization detector. Nitrogen was the carrier gas with a flow rate of 20 – 60 ml/min. The oven programme was: initial temperature at 60ºC, first ramping at 10ºC/min for 20 min, maintained for 4 min, second ramping at 15ºC/min for 4 min and maintained for 10 min. The injection temperature was 250ºC whilst the detector temperature was 320ºC. A capillary column (30 m, 0.25 mm) packed with a polar compound (HP INNOWAX) with diameter (0.25 µm) was used to separate the esters. Split injection type was used having a split ratio of 20:1. The peaks were identified by comparison with standards of fatty acid methyl esters.

Sterol analysis

Sterol was analyzed as described by AOAC (2006). The aliquot of the extracted fats was added to the screw-capped test tubes. The sample was saponified at 90ºC for 30 min, using 3 ml of 10% KOH in ethanol, to which 0.20 ml of benzene had been added to ensure miscibility. Deionized water (3 ml) was added and 2 ml of hexane was used in extracting the non-saponifiable materials.

Three extractions, each with 2 ml of hexane, were carried out for 1 h, 30 min and 30 min, respectively. The hexane was concentrated to 1 ml in the vial for gas chromatographic analysis and 1 µl was injected into the injection pot of GC. The peaks were identified in comparison with standard sterols. The sterols were analyzed using similar conditions as for fatty acid methyl ester analysis.

Phospholipid analysis

Modified method of Raheja *et al*. (1973) was employed in the analysis of phospholipids. 0.01 g of the extracted fat was added to each test tube. To ensure complete dryness of the fat for phospholipid analysis, the solvent was completely removed by passing stream of nitrogen gas on the fat. 0.40 ml chloroform was added to the tube followed by the addition of 0.10 ml chromogenic solution. The tube was heated at 100ºC in water bath for 1 min, 20 sec. The content was allowed to cool to the laboratory temperature and 5 ml hexane was added and the tube shaken gently several times. The solvent and the aqueous layers were allowed to be separated. The hexane layer was recovered and concentrated to 1.0 ml for analysis. The phospholipids were analyzed using an HP 5890 powered with HP gas chromatography (HP 5890 powered with HP ChemStation rev. AO9.01[1206] software [GMI, Inc, Minnesota, USA]) fitted with a pulse flame photometric detector. Nitrogen was used as a carrier gas with a flow rate of 20-60 ml/min. The oven programme was: initial temperature at 50ºC whilst the detector temperature was 320ºC. A capillary column (30 m, 0.25 mm) packed with a polar compound (HP) with a diameter (0.25 µm) was used to separate the phospholipids. Split injection type was used having a split ratio of 20:1. The peaks were identified by comparison with standard phospholipids.

Quality assurance

Standard chromatograms were prepared for sterols, phospholipids and fatty acid methyl esters which were then compared with respective analytical results; calibration curves were prepared for all the standard mixtures and correlation coefficient determined for each fatty acid parameter, same for sterols and phospholipids. Correlation coefficient should be >0.95 for the result to be acceptable. It was performed with Hewlett Packard Chemistry (HPCHEM) software (GMI, Inc, 6511 Bunker Lake Blvd Ramsey, Minnesota, 55303, USA).

Calculation of fatty acid as food per 100 g in sample

At the data source and reference data base levels, values for individual fatty acids are usually expressed as percentages of total fatty acids. At the user database level, values per 100g of food are required. When the content of total fatty acid in food or fat is not given, it is necessary to calculate it by using the fatty acid conversion factor (XFA). The conversion factor reflects the ratio between the sum of fatty acid and total lipids (TL) in the food (Weihrauch *et al.,* 1977).

FACID (g/100gEP) = TL (g/100g) × XFA

Total lipid (TL = crude fat) was multiplied by a conversion factor of 0.916 to convert it to total fatty acids (Anderson, 1976). For fatty acids, precision is best limited to 0.1 g/100g of fatty acids (Greenfield and Southgate, 2003). Further calculations were the conversion of the edible portion (EP) into two different unit of energy: kJ/100g EP and kcal/100g EP.

Results and Discussion

Table 1 contained the result of the crude fat content and other lipid related calculations of the sample (including the corresponding energy values in kJ and kcal). The total crude fat content was 14.2 g/100g showing kilishi to be a moderate source of fat. Out of the crude fat content, 13.0g/100g was due to fatty acid content whereas 1.19 g/100g was due to the other lipid content. The total energy due to the crude fat was 525 kJ/100g (128 kcal/100g) with corresponding fatty acid energy content of 481 kJ/100g (117 kcal/100g). Other lipid content had energy value of 44.1 kJ/100g (10.7 kcal/100g). The fatty acid had a percentage value of 91.6 of the crude fat whereas other lipid had percentage value of 8.38 showing the crude fat to be majority of fatty acid content.

Table 1: Crude fat and other lipid related calculated values in Nigerian jerky meat (kilishi)

Parameter	Unit	Value
Crude fat	g/100g	14.2
Total fatty acid[a]	g/100g	13.0
Total energy[b]	kJ/100g	525
Total energy[c]	kcal/100g	128
Total fatty acid energy[d]	kJ/100g	481
Total fatty acid energy[e]	kcal/100g	117
Other lipid content	g/100g	1.19
Other lipid energy[f]	kJ/100g	44.1
Other lipid energy[g]	kcal/100g	10.7

[a]Crude fat × XFA= 14.2 × 0.916; [b]Total energy= crude fat × 37.0; [c]Total energy = crude fat × 9.00; [d]Total fatty acid energy= Total fatty acid×37.0; [e]Total fatty acid energy = Total fatty acid× 9.00; [f]Other lipid energy= Other lipid value× 37.0; [g]Other lipid energy = Other lipid value ×9.00

Table 2: Saturated fatty acid profile and corresponding edible portion (EP) of the Nigerian jerky meat (kilishi)

Fatty acid	Percentage total fatty acids	Edible portion of fatty acid (EPg/100g)
C6:0	0.00	-
C8:0	0.00	-
C10:0	8.92e-3	1.16e-3
C12:0	1.70e-1	2.21e-2
C14:0	5.71e-1	7.43e-2
C16:0	25.3	3.30
C18:0	7.16	9.31e-1
C20:0	9.35e-3	1.22e-3
C22:0	1.26e-1	1.63e-2
C24:0	1.07e-3	1.39e-4
SFA	33.4	4.34
CI4:1 (cis-9)	2.45	3.18e-1
C16:1 (cis-9)	1.27	1.65e-1
C18: 1(cis-6)	21.9	2.84
C18: 1 (cis-9)	12.5	1.63
C20: 1 (cis-11)	8.67e-1	1.13e-1
C22: 1 (cis-13)	2.19	2.85e-1
C24: 1 (cis-15)	1.07e-3	1.39e-4
MUFA (cis)	41.2	5.36
C18: 1 (trans- 6)	3.36e-3	4.37e-4
C18: 1 (trans- 9)	3.04e-4	3.95e-5
C18: 1(trans- 11)	1.74e-3	2.26e-4
MUFA (trans)	5.41e-3	7.03e-4
MJUFA (total)	41.2	5.36

Table 2 was a combination of saturated fatty acid (SFA) values and the monounsaturated fatty acid (MUFA) values. Among the SFAs, the palmitic acid (C16:0) had the highest concentration of 25.3% of total fatty acid (FA) and slightly followed by stearic acid (C18:0) of 7.16%. Both C6:0 and C8:0 recorded 0.00% each; ultra-trace SFA were C10: 0, C20:0 and C24:0 whereas trace level of FA were in C12: 0, C14:0 and C22:0 giving an overall total SFA of 33.4%. The edible portion of the SFA ranged as in the FA and the edible FA as food (EPg/100g) was 4.34. SFA with C12: 0, C14:0 and C16:0 are known to be primary contributors to elevated blood cholesterol, and so contribute to cardiovascular diseases; C14: 0 being the major culprit. SFA with 12, 14 and 16 carbons generally constitute about 25% of the total FA in animal foods. All the 12, 14 and 16 carbons SFA constituted a value of 26.0%; this was 1.00% greater than the 25% (the usual minimum value in animals). C18:0 (7.16% of total FA) may not be as hypercholesterolemic as the other SFA (apparently because it is converted to oleic acid) (Bonanome and Grundy, 1998).

The C16: 0 is usually the most prevalent SFA in human diet and is present to some extent in essentially all fats. The value of C16: O in the total SFA was 75.9% showing its pre-eminence in the total SFA whereas C18: 0 in total SFA was 21.4%

Considering the influence of C16:0 on the lipoprotein profile, 16:0 is known to be intermediate, that is, it can be neutral when placed on a triglyceride molecule with PUFA, MUFA or 18:0 or cholesterol raising when it is attached along with C12:0 + C14:0. In high amount 16:0 is able to raise TC and LDL when substituted for 18:0, MUFA or PUFA in people who already have elevated TC or who eat large amounts of cholesterol. Therefore, the general advice has been to remove as much SFA from the diet as possible (Hayes, 2002); although Enig and Fallon (2002) had both enumerated many important roles SFA can play in the body chemistry. Radio tracer studies have shown that fish can convert C16: 0 to omega-7 monoenoic acid and C18:0 to omega-9 fatty acid (Mead and Kayama, 1967) (Scheme 1). Both C18: 0 and C16: 0 are the preferred food for the heart, which is why the fat around the muscle is highly saturated giving the heart the ability to draw on this reserve in times of stress.

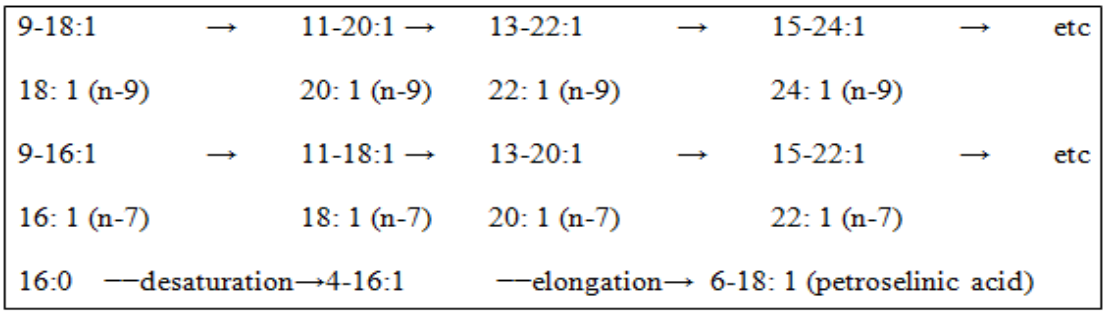
Short and medium-chain SFAs have important antimicrobial properties thereby protecting us against harmful microorganisms in the digestive tract. Short-chain fatty acids (SCFA) are fatty acids with aliphatic tail of five or fewer carbons (e.g. butyric acid) (Cifuentes, 2013). Medium-chain FAs (MCFA) are FAs with aliphatic tails of 6 to 12 (Roth, 2013) carbons, which can form medium-chain triglycerides. It has been established that the accumulation of certain long-chain FAs is associated with degenerative diseases of the central nervous system. Long-chain FAs (LCFA) are FAs with the aliphatic tail of 13 to 21 carbons (Beermann *et al.*, 2003). Also, very-long chain FAs (VLCFA) are FAs with aliphatic tails of 22 or more carbons. VLCFA include behenic acid (C22:0) with value of 1.26e-1% of total fat and corresponding food equivalent of 1.63e-2 EPg/100g lower than the literature value of 1% in beef (Whetsell, 2003) and lignoceric acid (C24:0) with analytical value of 1.07e-3% and EPg/100g value of 1.39e-4 which is also about 1% in beef (Whetsell, 2003). Both behenic and lignoceric acids were lower than 1.00% as occurred in beef.

Accumulation occurs because the enzymes needed to maintain turnover of those FAs are lacking (Lord and Bralley, 2000). Behenic acid has been detected to be a cholesterol-raising SFA factor in humans (Cater and Denke, 2001). Both lignoceric and behenic acids in the sample were of no significant values and therefore could not be of any nutritional health risk from kilishi. External body fat is softer than the internal fat that surrounds the organs owing to the higher content of unsaturated fat in external animal parts (Ahmad *et al.*, 2018). This could have been responsible for the relatively high level of SFA in kilishi.

It had been mentioned earlier that the monounsaturated fatty acids (MUFA) constituent could be seen in Table 2. MUFA of note in the sample were of levels (% of total FA): C14:1 cis-9(2.45) (EPg/100g = 3.18e-1), C16:1 cis-9 (1.27) (EPg/100g = 1.65e-1), C18:1 cis-6 (21.9) (EPg/100g = 2.84), C18:1 cis-9 (12.5) (EPg/100g = 1.63) and C22:1 cis-13(2.19) (EPg/100g = 2.85e-1).Insignificant MUFA cis values came from C20:1 cis-11 and C22:1 cis-15 whose ranges were 1.07e-3 to 8.67e-1% with EPg/100g range of 1.39e-4 to 1.13e-1. Some MUFA trans-constituents were also observed in the kilishi all be it at very low insignificant levels: C18:1 trans-6 (3.36e-3%; 4.37e-4 EPg/100g), C18:1 trans-9 (3.04e-4%; 3.95e-5 EPg/100g) and C18:1 trans-11 (1.74e-3%; 2.26e-4 EPg/100g).

Whilst the total cis-MUFA was 41.2%, those of trans-MUFA was 5.41e-3% with corresponding EPg/100g levels of 5.36 and 7.03e-4 (Ahmad *et al.*, 2018). The trans-FA in the kilishi was low in value. The total MUFA value of 41.2% was close to the values of 40.4% (turkey-hen) and 45.4% (duck) brains (Adeyeye and Aye, 2015) and the roam antelope brain which was 44.3% (Adeyeye and Aye, 2013). Many epidemiological studies comparing disease rates in different countries have suggested an inverse association between MUFA intake and mortality rates to CVD (Hu *et al.*, 1997; Kris-Etherton, 1999). Oleic acid (9c-18:1 or 18:1 (n-9) is by far the most abundant monoenoic fatty acid in plant and animal tissue, both in structural lipids and in deposit fats. However, this usual observation was in the reverse in this report as C18:1 cis-6 and C18:1 cis-9 had earlier been observed in the brain of male free-roaming domestic dog [*Canis lupus* var. *familiaris* (Linneaus, 1758)] whose C18:1 cis-6 was 23.8% and C18:1 cis-9 was 20.7% (Adeyeye *et al.*, 2018). 30% of the FA content in conventionally produced beef is composed of oleic acid (Whetsell *et al.*, 2003); this is a MUFA that elicits a cholesterol-lowering effect among other healthful attributes that includes a reduced risk of stroke and a significant decrease in both systolic and diastolic blood pressure in susceptible populations (Kris-Etherton, 1999). Oleic acid in turkey brain had a value of 13.9% (Adeyeye and Aye, 2015)

which was close to the present value of 12.5%. In roan antelope brain, C18:1 cis-6 was 18.5% (Adeyeye and Aye, 2013) which was lower than the present value of 21.9%. Oleic acid is the biosynthetic precursor of a family of fatty acids with the (n-9) terminal structure and with c\hain-lengths of 20-24 or more (Scheme 1). Petroselinic acid (6c-18:1) formed the largest single g broup in the MUFA with a value of 21.9% which was close to the value of 23.8% in *Canislupus* Var. *Familiaris* brain (Adeyeye *et al.*, 2018). Petroselinic acid occurs up to a level of 50% or more in seed oils of the Umbelliferae family, including carrot, parsley and coriander. *In vitro* studies by Weber *et al*. (1995) revealed that triacylglycerols containing petroselinoyl [18:1 (n-12)] moieties are hydrolyzed by pancreatic lipase at much lower rate than other triacylglycerides. Weber *et al*. (1995) data showed that petroselinic acid (6c-18:1) from dietary triglycerides is absorbed by rats as readily as oleic acid, however petroselinic acid reduces the concentration of arachidonic acid (AA) in tissue lipids suggesting [in view of earlier studies (Mohrhauer *et al.*, 1967) petroselinic acid mediated inhibition of AA synthesis]. C16:1 cis-9 is an important MUFA (1.27% and 1.65e-1EPg/100g); it has strong antimicrobial properties. It is found almost exclusively in animal fat. C16:1 is beneficial in reducing bad cholesterol (LDL) although it behaves like a SFA and not as unsaturated FA in its effect on HDL-cholesterol (Nestle *et al.*, 1994). It also reduces the fat deposition in blood vessels and blood clot formation (Grundy, 1994). Gadoleic acid-trivial name for cis-cos-9-enoic acid (20:1 n-11) is a common but minor constituent of animal tissues and fish oils (as here with a value of 8.67e-1% and 1.13e-1 EPg/100g) often accompanied by the 13-isomer. Erucic acid (C22:1n-13, cis) is a FA that is apparently responsible for a favourable response of persons with nervous system disorders (Christensen *et al.*, 1988). The administration of eruic acid in the serum reduces the serum levels and brain accumulation of very-long-chain SFAs (such as C26:0) responsible for demyelination (Rasmussen *et al.*, 1994; Sargent *et al.*, 1994). C22:1 cis-13 was 2.19% and 2.85e-1 EPg/100g in kilishi sample. The production of longer chain acids of the n-9 family and n-7 family as well as the production of petroselinic acid were shown in Scheme 1.



Scheme 1: Production of longer chain n-9 and n-7 families and petroselinic acid

In Table 3, we presented the PUFA ω6 (n-6) and ω3 (n-3) values. Among the ω6 levels, the significant members there were C18:2 (cis-9, 12) (13.1% and EPg/100g = 1.70); C18:3 (cis-6, 9, 12) (1.20% and EPg/100g = 1.20e-2); C20:4 (cis-5, 8, 11, 14) (4.27% and EPg/100 g = 5.56e-1) whereas minor valued ω6 members were C20:2 (cis-11, 14) (1.32e-3% and Epg/100g =1.72e-4); C20:3 (cis-8, 11, 14) (1.26e-1% and EPg/100g = 1.62e-2); C22:2 (cis-13, 16) (1.95e-1% and EPg/100g = 2.54e-2) and C18: 2(trans-9,12) (3.94e-3% and EPg/100g = 5.13e-4). The total ω6 PUFA was 18.9% and 2.46 EPg/100g. The values of ω3 PUFA were generally lower than in the ω6 levels. Values (in % total fat) were; C18:ω3 (cis-9, 12, 15) (1.44 and 1.88e-1 EPg/100g); C20:ω3 (cis-11, 14, 17) (5.72e-3 and 7.44e-4 EPg/100g) and C22:6 (cis-4, 7, 10, 13, 16, 19) (2.48e-1 and 3.23e-2EPg/100g). Total ω3 FA was 6.54% (8.51e-1 EPg/100g) which was about 1/3 of the total ω6 value. From Tables 2 and 3, ω6 and ω3 total (PUFA) was 25.4% (3.31 EPg/100g) whereas ω9 + ω6 + ω3 totaled 66.6% (8.66 EPg/100g). Oleic (C18:1), linoleic (C18:2) and linolenic (C18:3) and arachidonic (C20:4) acids are necessary constituents of mitochondria cell wall and other active metabolic sites (Ahmed *et al.*, 2018). Ahmed *et al.*(2018) reported on PUFA composition of beef as thus: C18:2 (2.1%), C18:3(1.4%), C20:3 (trace), C20:4 (1.1), C22:5 (trace), and C22:6 (nil); these values were all less than the observation in kilishi meat. Even in pork meat, values of PUFA were (%); C18:2 (7.5), C18:3 (1.0), C20:3 (nil), C20:4 (trace), C22:5 (trace), C22:6 (1.1). The essential FAs influence the fluidity, flexibility and permeability of the membranes; they are the precursor of the eicosanoids, are necessary for maintaining the impermeability barrier of the skin and are involved in the cholesterol transport and metabolism. The knowledge of the significance of the long chain PUFA of the n-3 type, particularly EPA and DHA, for human health has increased considerably since the 1970s (Stansby, 1990a, b). Omega-3 FAs hold great promise in the prevention and management of obesity.

Table 3: PUFA[a] n-6 and n-3 fatty acid profile and corresponding edible portion (EP) of the Nigerian jerky meat (kilishi)

Fatty acid	Percentage total fatty acids	Edible portion of fatty acid (EPg/100g)
C18:2 (cis- 9, 12)	13.1	1.70
C18: 3 (cis- 6, 9, 12)	1.20	1.20e-2
C20: 2 (cis- 11, 14)	1.32e-3	1.72e-4
C20: 3 (cis- 8, 11, 14)	1.26e-1	1.64e-2
C20: 4 (cis- 5, 8, 11, 14)	4.27	5.56e-1
C22: 2 (cis- 13, 16)	1.95e-1	2.54e-2
ω6 (n-6) PUFA (cis)	18.9	2.46
C18: 2 (trans- 9, 12)	3.94e-3	5.13e-4
ω6 (n-6) PUFA (total)	18.9	2.46
C18: 3 (cis- 9, 12, 15)	1.44	1.88e-1
C20: 3 (cis- 11, 14, 17)	5.72e-3	7.44e-4
C20: 5 (cis- 5, 8, 11, 14, 17)	4.85	6.30e-1
C22: 6 (cis- 4, 7, 10, 13, 16,19)	2.48e-1	3.23e-2
ω3 (n-3) PUFA (total)	6.54	8.51e-1
ω6 + ω3 (n-6 + n-3) PUFA	25.4	3.31
TUFA[b]	66.6	8.66

[a]PUFA = polyunsaturated fatty acid (Essential fatty acid); [b]TUFA= total unsaturated fatty acid

Omega-6 and omega-3 polyunsaturated fatty acids (PUFAs) are essential fatty acids that must be derived from the diet, cannot be made by humans and other mammals because of the lack of endogenous enzymes for omega-3 desaturation (Simopoulos, 2001; Kang, 2003). Omega-6 fatty acids are represented by linoleic acid (LA) (18:2ω-6) and omega-3 fatty

acid by alpha-linolenic acid (ALA) (18:3ω-3). LA is plentiful in nature and sources include from the seeds of most plants except coconut, cocoa and palm whereas ALA is found in the chloroplast of green leafy vegetables, in seeds of flax, rape, chia, perilla and walnuts (Simopoulos, 2016). Both EFAs are metabolized to longer-chain FAs of 20 and 22 carbon atoms. LA is metabolized to arachidonic acid (AA) (20:4ω6) whilst ALA is metabolized to eicosapentaenoic acid (EPA) (20:5ω3) and docosahexaenoic acid (DHA) (22:6ω3). This is achieved by increasing the chain length and the degree of unsaturation by adding extra double bonds to the carboxyl end of the FA molecule (Simopoulos, 1991) (Fig. 2). Humans and other mammals, except for certain carnivores such as lions, can convert LA to ALA and ALA to EPA and DHA, although the process is slow (De Gome Dumm and Brenner, 1975; Emken *et al.*, 1989). There is competition between omega-6 and omega-3 FAs for the desaturation enzymes. Both FA desaturase 1(FADS1) and FA desaturase 2(FADS2) prefer ALA to LA (De Gome Dumm and Brenner, 1975; Hague and Christoffersen, 1984; Hague and Christoffersen, 1986). However, a high LA intake, such as that characterizing Western diets interferes with the desaturation and elongation of ALA (Emken *et al.*, 1989; Hague and Christofferson, 1984; 1986; Indu and Ghafoorunissa, 1992). Also, trans- FA interfere with the desaturation and elongation of both LA and ALA (Simopoulos, 2016).

Important genetic variables in FA biosynthesis involving FADS1 and FADS2 that encode rate-limiting enzymes of FA metabolism are known. Ameur *et al.* (2012), performed genome-wide genotyping of the FADS region in five European population cohort and analyzed available genomic data from human populations, archaic hominins and more distant primates. Their results showed that present-day humans have two common FADS haplotypes A and D which differ dramatically in their ability in generating long-chain polyunsaturated fatty acids (LC-PUFAs). The more common haplotype, denoted haplotype D, was associated with high blood lipid levels, whereas the less common haplotype (haplotype A) was associated with low blood lipid levels. The haplotype D associated with the enhanced ability to produce AA and EPA from their precursors LA and ALA, respectively is specific to humans. The haplotype shows evidence of a positive selection in African populations in which it is presently almost fixed and it is less frequent outside Africa. Haplotype D provides a more efficient synthesis of LC-PUFAs and in today's high LA omega-6 dietary intake from vegetable oils, it leads to increased synthesis of AA from LA. As a result halotype D represents a risk factor for coronary heart disease (CHD), cancer, obesity, diabetes and the metabolic syndromes, adding further to health disparities in populations of African origin living in the West, in addition to lower socioeconomic status (Sergent *et al*., 2012; Mathias *et al.*, 2011). Also, FADS2 is the limiting enzyme and there is some evidence that it decreases with age (De Gomez Dumm and Brenner, 1975). Premature infants (Calson *et al.*, 1986), hypertensive individuals (Singer *et al.*, 1984) and some diabetics (Honigmann *et al.*, 1982) are limited in their ability to make EPA and DHA from ALA. These findings are important and need to be considered when making dietary recommendations. Genetic variants in FADS cluster are determinants of long chain PUFA levels in circulation, cells and tissues (Simopoulos, 2016). These genetic variants have been studied in terms of ancestry and the evidence is robust relative to ethnicity. Thus, 80% of African Americans and about 45% of European Americans carry two copies of the alleles associated with increased levels of AA. It is said to be quite probable that gene PUFA interactions induced by the modern Western diet are differentially driving the risk of diseases of inflammation (obesity, diabetes, atherosclerosis and cancer) in diverse populations (Simopoulos, 2016).

In Table 4, we reported the energy values of the various fatty acids of kilishi in both kJ/100g and kcal/100g. From Table 4, the total energy from TSFA was 161 kJ/100g (39.1 kcal/100g) making a percentage contribution of 33.4%. Out of this, C16:0 contributed 122 kJ/100g (29.7 kcal/100g) or 25.3%% whereas C18:0 contributed 34.5 kJ/100g (8.38 kcal/100g) or 7.16%. In MUFA, its total energy contribution was 198 kJ/100g (49.2 kcal/100g) or 41.2%. The 41.2% majorly came from C18:1 ω-6, 105 kJ/100g (25.6 kcal/100g) or 21.9%; C18: 1 ω-9, 60.4 kJ/100g (14.7 kcal/100g) or 12.5%; C14: 1 ω-9, 11.8 kJ/100g (2.86 kcal/100g) or 2.45%; C22: 1 ω-9 (cis-13), 10.6 kJ/100g (2.57 kcal/100g) or 2.19% and C16:1 ω-9, 6.10 kJ/100g (1.48 kcal/100g) or 1.27%.

Table 4: Energy values (and percentage values) of the fatty acid profile of Nigerian jerky meat (kilishi) (kJ/100g and kcal/100g, EP SFA and MUFA)

Fatty acid	Energy in kJ/100g (%value)	Energy in kcal/100g (%value)
C6: 0	-	-
C8: 0	-	-
C10: 0	4.29e-2 (8.92e-3)	1.01e-2 (8.92e-3)
C12: 0	8.18e-1 (1.70e-1)	1.99e-1 (1.70e-1)
C14: 0	2.75 (5.71e-1)	6.69e-1 (5.71e-1)
C16: 0	122 (25.3)	29.7 (25.3)
C18: 0	34.5 (7.16)	8.38 (7.16)
C20: 0	4.50e-2 (9.35e-3)	1.09e-2 (9.35e-3)
C22: 0	6.04e-1 (1.26e-1)	1.47e-1 (1.26e-1)
C24: 0	5.13e-2 (1.07e-2)	1.25e-3 (1.07e-2)
SFA	161 (33.4)	39.1 (33.4)
C14: 1 (cis- 9)	11.8 (2.45)	2.86 (2.45)
C16: 1 (cis- 9)	6.10 (1.27)	1.48 (1.27)
C18:1 (cis- 6)	105 (21.9)	25.6 (21.9)
C18: 1 (cis- 9)	60.4 (12.5)	14.7 (12.5)
C20: 1 (cis- 11)	4.17 (8.67e-1)	1.02 (8.67e-1)
C22: 1 (cis- 13)	10.6 (2.19)	2.57 (2.19)
C24: 1 (cis- 15)	5.13e-3 (1.07e-3)	1.25e-3 (1.07e-3)
MUFA (cis)	198 (41.2)	49.2 (41.2)
C18: 1 (trans- 6)	1.62e-2 (3.36e-3)	3.94e-3 (3.36e-3)
C18: 1 (trans- 9)	1.46e-3 (3.04e-4)	3.56e-4 (3.04e-4)
C18: 1 (trans- 11)	8.34e-3 (1.74e-3)	2.04e-3 (1.74e-3)
MUFA (trans)	2.60e-2 (5.41e-3)	6.34e-3 (5.41e-3)
MUFA (total)	198 (41.2)	49.2 (41.2)
SFA + MUFA	359 (74.6)	88.3 (74.6)

Table 5: Energy values (and percentage values) of the fatty acid profile of Nigerian jerky meat (kilishi) in kJ/100g and kcal/100g [EP n-6 (ω6) PUFA and n-3 (ω3) PUFA]

Fatty acid	Energy in kJ/100g (%value)	Energy in kcal/100g (%value)
C18:2 (cis- 9, 12)	63.0 (13.1)	15.3 (13.1)
C18: 3 (cis- 6, 9, 12)	4.46e-1 (1.20)	10.8e-1(1.20)
C20: 2 (cis- 11, 14)	6.37 e-3 (1.32e-3)	1.55e-3 (1.32e-3)
C20: 3 (cis- 8, 11, 14)	6.08e-1 (1.26e-1)	1.48e-1 (1.26e-1)
C20: 4 (cis- 5, 8, 11, 14)	20.6 (4.27)	5.00 (4.27)
C22: 2 (cis- 13, 16)	9.41e-1 (1.95e-1)	2.29e-1 (1.95e-1)
ω6 (n-6) PUFA (cis)	90.9 (18.9)	22.1 (18.9)
C18: 2 (trans- 9, 12)	1.90e-2 (3.49e-3)	4.62e-3 (3.94e-3)
ω6 (n-6) PUFA (total)	90.9 (18.9)	22.1 (18.9)
C18: 3 (cis- 9, 12, 15)	6.95 (1.44)	1.69 (1.44)
C20: 3 (cis- 11, 14, 17)	2.75e-2 (5.72e-3)	6.70e-3 (5.72e-3)
C20: 5 (cis- 5, 8, 11, 14, 17)	23.3 (4.85)	5.67 (4.85)
C22: 6 (cis- 4, 7, 10, 13, 16, 19)	1.20 (2.48e-1)	2.91e-1 (2.48e-1)
ω3 (n-6) PUFA (total)	31.5 (6.54)	7.66 (6.54)
ω6 + ω3 (n-6 + n-3) PUFA	122 (25.4)	29.8 (25.4)
TUFA	321 (66.6)	78.0 (66.6)

Energy values contributed by ω-6 and ω-3 PUFAs had been depicted in Table 5. Total ω-6 energy contribution was 90.9 kJ/100g (22.1 kcal/100g) or 18.9%. Major contributions were C18: 2 ω-6 (cis-9, 12), 63.0 kJ/100g (15.3 kcal/100g) or

13.1% and C20:4 ω-6 (cis-5, 8, 11, 14), 20.6 kJ/100g (5.00 kcal/100g) or 4.27%. Total energy contributions from ω-3 FA was 31.5 kJ/100g (7.66 kcal/100g) or 6.54%. The major contributors for the ω-3 energy contributions were C18:3 ω-3 (cis- 9, 12, 15), 6.95 kJ/100g (1.69 kcal/100g) or 1.44% and C20:5 ω-3 (cis- 5, 8, 11, 14, 17), 23.3 kJ/100g (5.67 kcal/100g) or 4.85%. All the energy calculations were made from edible portion EPg/100g of the kilishi sample. It is interesting to note that the percentage of the energy values were correspondingly equivalent to the FA values. Values that were significant in the FA levels were also significant in the energy levels. Most energy values were low.

It was observed that total energy contributed was 481 kJ/100g (117 kcal/100g) (Tables 1, 4, 5 and 6). This value was higher than in the innards of *N. masculatus* where we had 433 kJ/100g (105 kcal/100g) in the male and 407 kJ/100g (99.0 kcal/100g) in the female (Adeyeye*et al.,* 2019). For optimum weight loss, you are advised to reduce your overall fat/oil consumption to a sensible level. A level of 15-20% of your calories should come from fat and majority of that should be essential FAs. From Tables 4 and 5, we have the following FA group energy contributions: MUFA (41.2%) > SFA (33.4%) > PUFA (25.4%).

However, TUFA (66.6%) > SFA (33.4%). In determining how many grammes these calories (15-20%), you would multiply your total calories by 15% (20% for the high end of the range) and then divide by 9, which is the number of calories in a gramme of fat. An example goes thus:

2550 daily calories × 0.15 = 375/9 = 41.7 or 42 g of total fat per day- the bulk of which should be EFAs. It is known that 20% energy from fat is consistent with good health. With 41.7 g of total fat per day, the sample with value of 13.0 g/100g (31.2% of 41.7 g of total fat per day) FAs would be able to provide about 1/3.2 of the daily needs of energy to its consumers. Energy based on the different FAs components were: EFA EPg/100g = 3.31/13.0 = 25.5%; for energy contribution it was 3.31/41.7 × 100 = 7.94%; in MUFA we had 5.36/13.0= 41.2%; for energy contribution it was 5.36/41.7 × 100 = 12.9%; in SFA we had 4.34/13.0 = 33.4%; for energy contribution it was 4.34/41.7 × 100 = 10.4%.

Some of the quality parameters of the kilishi had been depicted in Table 6. The excerpts came from Tables 2 and 3. Number one parameter was MUFA/SFA ratio of 1.23. Phospholipid compositions have direct correlations to the relative proportions of MUFA/SFA and changes to this ratio have been claimed to have effects on such disease states as cardiovascular disease, obesity, diabetics, neuropathological conditions and cancer. They have cyto-protective actions in pancreative β-cells. cis- Monoenoic FAs have desirable physical properties for membrane lipids in that they are liquid at body temperature, but still relatively resistant to oxidation. They have since been recognized by nutritionists as being beneficial in the human diet. The MUFA/SFA in the sample was good enough and compared favourably with MUFA/SFA in the innards of *N. maculatus* (1.28 - 1.71) (Adeyeye *et al.,* 2019) as well as in *Acanthurus monroviae* and *Lutjamus gorensis* (1.16 – 1.78) (Adeyeye, 2015). The ratio of PUFA/SFA (P/S) in dietary oils is important in determining the detrimental effects of dietary fats. The higher the P/S ratio the more nutritionally useful is the dietary oil. This is because of the severity of atherosclerosis is closely associated with the proportion of the total energy supplied by SFAs and PUFA fats (Honatra, 1974). The value of P/S in kilishi was 7.62e-1; this is slightly more favourable towards SFA. However, the value of 7.62e-6 could still be regarded as reasonable. The AA/DGLA (C20:4n-6, cis-5, 8, 11, 14/C20:3n-6, cis- 8, 11, 14) value was high at 33.8. A high value of this ratio between AA and DGLA, as an indicator of Δ-5 desaturase activity in the skeletal muscle phospholipids has been related to good insulin activity (Benatti *et al.,* 2004). In assessing the essential PUFA status of an individual, the total amount of the various EFA and PUFA in the plasma or erythrocyte phospholipids has been a useful indicator (Hornstra, 1992). In assessing the EFA and PUFA qualities, the following were further used as additional status markers in order to reliably assess the functional PUFA status (Benatti *et al.,* 2004). The best known marker is mead acid [trivial name for all-cis-cosa- 5, 8, 11 – trienoic acid (20:3n-9)]. The synthesis of this FA is promoted if there are insufficient concentrations of LA and ALA to meet the need for the synthesis of long-chain PUFA. Both EPA and DHA inhibit mead acid synthesis; the presence of mead acid indicates a general shortage of all essential PUFA. The ratio of EPA/DHA value was 19.5 and no mead acid was observed. Another indicator of essential PUFA status is the essential PUFA status index (EPSI); this is a ratio between all essential PUFA (the sum of all n-6 and n-3 FAs) and all non-essential unsaturated FAs (the sum of all n-7 and n-9 FAs). The higher the EPSI status index the better is the essential PUFA status. Further in the status marker, if there is a functional shortage of DHA, the body starts to synthesise the most comparable long-chain PUFA of the n-6 family, osbond acid (C22:5n-6) (all-cis-4, 7, 10, 13, 16 – docosapentaenoic acid; an omega-6 FA with the trivial name asbond acid). It is formed by an elongation and desaturation of arachidonic acid 20:4 omega-6. In mammals clupanodonic acid deficiency is accompanied by an increase of this isomer; the osbond/DHA ratio is thus a marker of dietary DHA sufficiency (Hornstra, 2007). Therefore, under steady conditions, the ratio between DHA and osbond acid is a reliable indicator of the functional status (Neuringer *et al.,* 1986). No value of osbond was observed in the result; hence kilishi PUFA could not cause functional distress. In the Table 6, the ratios of C16:0:C18:1cis-9 (2.02) and C18:0:C18:1cis-9 (5.7e-1) were respectively close to the values of such ratios in the innards of *N. maculatus* (Adeyeye *et al.,* 2019). From the work of Sridhar *et al.* (2016) on FA composition of mangrove wild legume seeds (*Sesbania speciosa)* in south-western India, the following ratio values were reported in these respective samples: hot extracted, uncooked/cooked [TUFA/TSFA = 0.34-0.13], [TPUFA/TMUFA = 1.31 – 0.73], [C18:1/C18:2 = 0.74-1.34] and [ω-6/ ω-3 = 16.39 – 12.09]; cold extracted, uncooked/cooked [TUFA/TSFA = 1.48-3.78], [TPUFA/TMUFA = 2.55 – 4.87], [C18:1/C18:2 = 0.36 – 0.22] and [ω-6/ ω-3 = 11.21 – 8.79].

Table 6: Some quality parameters of the fatty acid of Nigerian jerky meat (kilishi) from Tables 2 and 3

Parameter	Value
MUFA/SFA	1.23
PUFA/SFA	7.62e-1
EPA/DHA	19.5
ω6/ω3 or n-6/n-3 (LA/ALA)	9.06
Total ω6/total ω3	2.89
AA/DGLA	33.8
EPA/DHA	5.09
EPSI[a] (MUFA/PUFA)	1.62
C16: 0: C18: 1 cis-9	2.02
C18: 0: C18: 1 cis-9	5.70e-1
%C16: 0 in SFA	75.9
%C18: 0 in SFA	21.4
AA/EPA+DHA	8.39e-1
TUFA[b] (MUFA+PUFA)	66.6
TFA[c] (SFA + MUFA +PUFA)	100.0
Total EPg/100g	13.0
Total energy (kJ/100g FA)	481
Total energy (kcal/100g FA)	117

[a]Essential PUFA status index; [b]Total unsaturated fatty acid; [c]Total fatty acid

Table 7: Omega-6/omega-3 ratios in different populations

Population	ω-6 /ω-3
Paleolithic	0.79
Greece prior to 1960	1.00-2.00
Current Japan	4.00
Current India, rural	5-6.1
Current UK and northern Europe	15.00
Current US	16.74
Current India, urban	38 – 50

Source: Simopoulos (2016)

Omega-6 and omega-3 PUFAs are essential FAs that must be derived from diet, cannot be made by humans, and other mammals because of the lack of endogenous enzymes for omega-3 desaturation (Simopoulos, 2001; Kang, 2003). However, due to agribusiness and modern agriculture, western diets contain excessive levels of omega-6 PUFAs but very low levels of omega-3 PUFAs, leading to an unhealthy omega-6/omega-3 ratio of 20:1, instead of 1:1 that was during evolution in humans (Simopoulos, 2001; Simopoulos, 2008). In Table 6, LA/ALA was 9.06 whereas total ω-6/total ω-3 was 2.89. An unbalanced ω-6/ ω-3 ratio in favour of ω-6 PUFAs is highly prothrombotic and proinflammatory, which contributes to the prevalence of atherosclerosis, obesity and diabetes (Simopoulos, 2013; Kromhout and de Goede, 2014). Both n-6 and n-3 FAs have critical roles to play in the membrane structure (Kinsella, 1990) and as precursors of eicosanoids which are potent highly reactive compounds. Because both compete for the same enzyme but having diffrerent biological roles, the balance between n-6 and n-3 FAs in the diet can be of considerable importance (FAO/WHO, 1994). Omega-6 and omega-3 FAs are not interconvertible, are metabolically and functionally distinct, and often have important opposing physiological effects; therefore their balance in the diet is important (Simopoulos, 2016). When humans ingest fish or fish oil, the EPA and DHA from the diet partially replace the ω-6 FAs, especially AA, in the membranes of probably all cells, but especially in the membranes of platelets, erythrocytes, neutrophils, monocytes and liver cells (reviewed in Simopoulos, 2008; Simopoulos, 1999). AA and EPA are the parent compounds for eicosanoid production. Because of the increased amounts for ω-6 in the Western diet, the eicosanoid metabolic products from AA, especially prostaglandins, thromboxanes, leukotrienes, hydroxyl fatty acids and lipoxins, are formed in larger quantities than those derived from omega-3 FAs, especially EPA (Simopoulos, 2008). The eicosanoids from AA are biologically active in very small quantities and, if they are formed in large amounts, they contribute to the formation of thrombus and atheromas; to allergic and inflammatory disorders, particularly in susceptible people; and to proliferation of cells (Simopoulos, 2011). Thus, a diet rich in ω-6 FAs shifts the physiological state to one that is proinflammatory, prothrombotic and proaggregatory, with increases in blood viscosity, vasospasm, vasoconstriction and cell proliferation (Simopoulos, 2016). There is competition between ω-6 and ω-3 FAs for the desaturation of enzymes. Both Fas desatutrase 1(FADS1) and FA desaturase 2 (FADS2) prefer ALA to LA (De Gomez Dumm and Brenner, 1975; Hague and Christoffersen, 1984; Hague and Christoffersen, 1986). However a high LA intake, such as that characterizing Western diets, interferes with the desaturation and elongation of ALA (Indu and Ghafoorunissa, 1986; Emken *et al.*, 1988) (Fig. 2). In the Table 6, n-6/n-3 ratio of 2.89 was better than the normal ratio of 5:1 and the ω-6/ ω-3 (LA/ALA) of 9.06 was within the ratio of 10:1. In many people, an ω-6 to ω-3 ratio of 4:1 is ideal: that is 4 ω-6s for every 1 ω-3. Anti-ageing experts suggest going even further, maintaining a 1:1 ratio or higher in favour of ω-3s (Table 7). In America, the average American eats a ratio of somewhere from 12:1 to 25:1 ω-6 to ω-3. The major reason for skewed ratio in the US and other countries that eat Western diets is the types of oil in their foods. The most common source of ω-6s is LA, found in corn oil, soybean, safflower oil, cotton seed oil, sun flower oil, poultry, some nuts and seeds (Adeyeye *et al.*, 2019). The increased LA and AA content of foods has been accompanied by a significant increase in the AA/EPA + DHA ratio within adipose tissue, leading to increased production in AA metabolites, PG12 which stimulates white adipogenesis and PGF2α which inhibits the browning process, whereas increased consumption of EPA and DHA leads to adipose tissue homeostasis through adipose tissue loss and increased mitochondria biogenesis (Simopoulos, 2016). The value of AA/EPA + DHA was low at 8.39e-1 (Table 6). This is a nutritional advantage of EPA + DHA over AA.

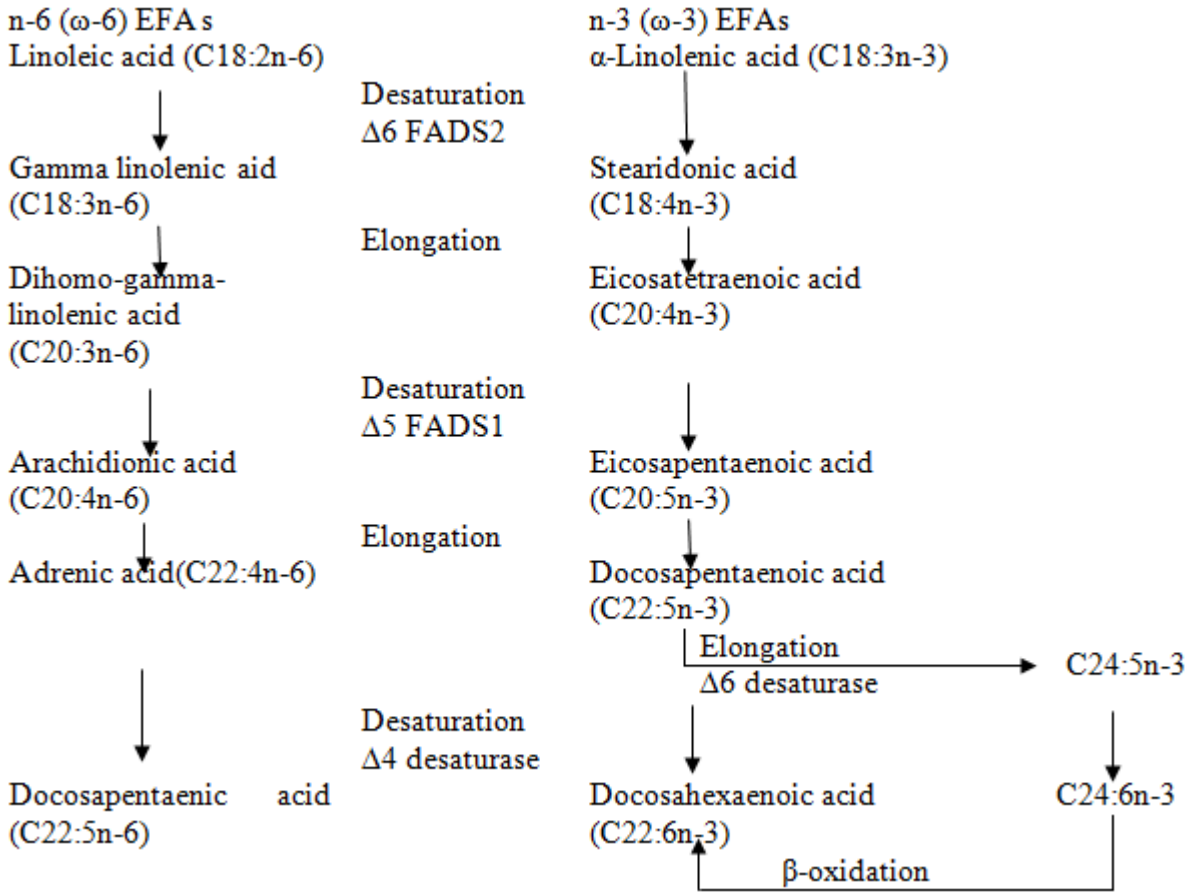


Fig. 2: Desaturation and elongation of ω-3 and ω-6 fatty acids by the enzymes fatty acid desaturases FADS2 (D6) and FADS1 (D5) (Simopoulos, 1991)

The sterol levels in the kilishi sample had been depicted in Fig. 3. Sterol of major significance was cholesterol having a concentration value of 74.0 mg/100g covering 91.2% of the total sterol level. Very far close to this value was sitosterol with a concentration value of 3.80 mg/100g occupying 3.80% of the total value. Low sterol values were observed for campesterol (1.46 mg/100g and 1.80%) and stigmasterol (1.73 mg/100g and 2.13%). Insignificant values of sterol were observed for cholestanol (1.08e-3 mg/100g; 1.33e-3%) and ergosterol (1.07e-5 mg/100g; 1.30e-5%). Only 5-avenasterol had a value close to 1.00 mg/100g with a concentration of 8.46e-1 mg/100g (1.07%). On the whole, the sterol levels in kilishi was generally low at a total sum of 81.1 mg/100g meaning that the virtual concentration of sterol in kilishi came from cholesterol (74.0 mg/100g) and percentage value of 91.2% whereas the other members collectively had a concentration of 7.10 mg/100g. Most authorities, although not all, recommend a reduction in dietary cholesterol to around 300 mg or less per day (Bender, 1992). Ahmad *et al.* (2018) gave the cholesterol level of meat sources which were (cholesterol mg/100g): mutton (81), beef (62) and pork (71) which were all highly comparable to kilishi cholesterol. Cholesterol in the diet is known to play an important role in maintaining the health of the intestinal wall (Alfin-Slater and Aftergood, 1980). This is why low-cholesterol vegetarian diets can lead to a leaky gut syndrome and other intestinal disorders.

Cholesterol is liable to damage on exposure to heat and oxygen. This damage or oxidized cholesterol seems to promote both injury to the arterial cells as well as a pathological build-up of plague in the arteries (Paul, 1990). Low cholesterol levels have been linked to aggressive and violent behaviour, depression and suicidal tendencies. Mother's milk is especially rich in cholesterol and contains a special enzyme that helps the baby utilize this nutrient.

Babies and children need cholesterol-rich food throughout their growing years to ensure proper development of the brain and other parts of the nervous system. In conjunction with SFA, cholesterol in the membrane gives our body cells necessary body stiffness and stability. When diet contains an excess PUFA, these replace SFA in the cell membrane, so that the cell wall actually becomes flabby. When this happens, cholesterol from the blood is driven into the tissues to give them structural integrity. This is the reason why serum cholesterol levels may go down temporarily when we replace SFA with PUFA oils in the diet (Jones, 1997). Other functions of cholesterol are: acts as a precursor to vital carticosteriods (hormones that help us deal with stress and protect the body against heart disease and cancer); to the sex hormones like androgen; precursor to vitamin D (an important fat soluble vitamin needed for healthy bones and nervous system); proper growth; mineral metabolism; muscle tone; insulin production; reproduction and immune function. The bile salts are made from cholesterol. Bile is vital for digestion and assimilation of fat in the diet. Cranton and Frackelton (1984) showed that cholesterol acts as an antioxidant. This gives a likely explanation for the fact that cholesterol levels go up with age. As an antioxidant, cholesterol protects us against free radical damage that leads to heart disease and cancer. It is needed for proper function of serotonin receptors in the brain (Engelberg, 1992).

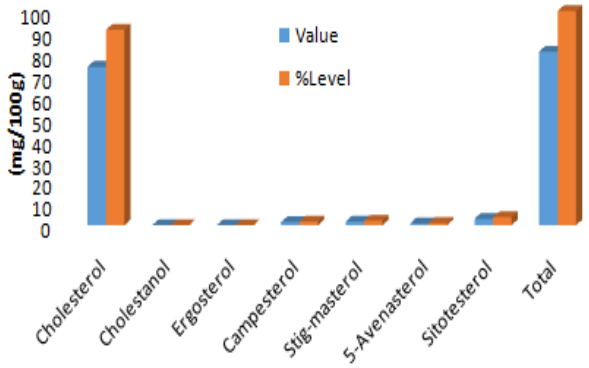
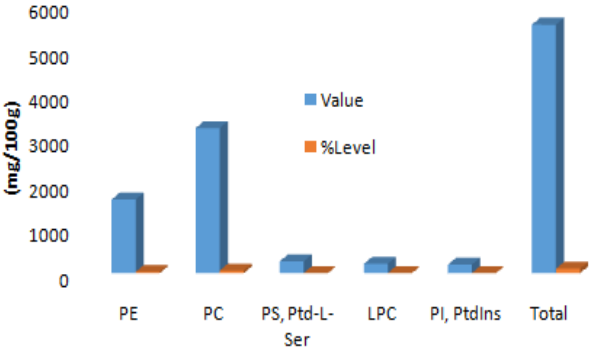


Fig. 3: Sterol levels (mg/100g) of Nigerian jerky meat (kilishi)



PE- Phosphatidylethanolamine (also called cephalin); PC- Phosphatidylcholine (also called lecithin); PS, Ptd-L-Ser- Phosphatidylserine; LPC- Lysophosphatidylcholine; PI, PtdIns- Phosphatidylinositol

Fig. 4: Phospholipid levels (mg/100g) of edible portion of Nigerian jerky meat (kilishi)

In Fig. 4, we have the chart for the phospholipids levels of kilishi. Individual phospholipid values were generally high with values that ranged between 192 to 3262 mg/100g. Concentration levels of the phospholipids in kilishi ran thus (mg/100g): phosphatidylcholine (PC), 3262 (58.4%) > phosphatidylethanolamine (PE), 1655 (29.6%) > phosphatidylserine (PS, Ptd-L-Ser), 267 (4.79%)> lysophosphatidylcholine (LPC), 210 (3.76%) > phosphatidylinositol (PI, PtdIns), 192 (3.43%) and a grand total concentration of 5586 mg/100g. The literature report of Viswanathan Nair and Gopakumar (1984) on the lipid and FA compositions of five species of lean fish, silver hew fish (*Johnius argentatus*), milk fish (*Chanos chanos*), pearl spot (*Etroplus suratensis*), cat fish (*Pseudarius jella* and *Tahysurus sp*) and three species of shell fish, mussed (*Perna viridis*), crab (*Neptuous palagius*) and fresh water prawn (*Macrobranchium rosenbergii*) showed that the PC was the major phospholipid in all the samples studied. In lean fish, its proportion varied from 55.9 – 63.8% and in shell fish, from 44.0 – 68.9% of total lipids. PE content was the second highest component (except in fresh water prawn). Its proportion in lean fish varied from 14.9 – 21.7% of total lipids. PE occupied the second highest concentrated position as it followed PC (first highest concentration) in *A. monronviae* and *L. goreensis* (Adeyeye, 2015) as well as in the innards of *N. maculatus* (Adeyeye *et al.*, 2019) as they occurred in the present sample. Lecithin (PC) is the building block of membrane bilayers, is also the principal phospholipid circulating in plasma, where it is an integral component of the lipoproteins, particularly HDL (Whitney *et al.*, 1994). PE occurs in all living cells, although in human physiology it is found particularly in the nervous tissue such as the white matter of brain, nerves, neural tissue and in the spinal cord (Adeyeye, 2011). PS occupied the third position in the concentration series. PS has been shown to enhance mood in a cohort of young people during mental stress and to improve accuracy during tee-off by increasing the stress resistance of golfers. The US Food and Drug Administaration (USFDA) had stated that consumption of PS may reduce the risk of dementia in the elderly (Adeyeye, 2011). PI (PtdIns) occupied the fifth important position in concentration in the kilishi sample. PI is a negatively charged phospholipid and is capable of being phosphorylated to form phosphatidylinositol phosphate (PIP), phosphatidylinositol biphosphate (PIP2) and phosphatidylinositol triphosphate (PIP3). PIP, PIP2 and PIP3 are collectively called phosphoinositides. Phosphoinositides play important roles in lipid signaling,cell signaling and membrane tracking (Adeyeye, 2011). LPC had significant concentration value of 210 mg/100g (3.76%) and occupied the fourth position in the concentration level. Partial hydrolysis of PC with the removal of only one FA yields an LPC. Generally, most values of the phospholipids in kilishi followed the trend observed in the innards of *N. maculatus* heterosexuals (Adeyeye *et al.*, 2019).

Quality assurance

In pursuance of quality assurance for the analysis, correlation coefficients (Cc) were determined for all the standards: fatty acids, phospholipids and sterols. The Cc values for the standards ranged thus: 0.99833 – 0.99997 (FAs), 0.99909 – 0.99999 (phospholipids) and 0.99920 – 0.99994 (sterols); all the correlation coefficient values being greater than 0.95 which is the critical correlation for acceptance of these types of analytical results. This attested to the quality assurance of the determinations.

Conclusion

Kilishi had average level of total lipid. Lipid constituents had the following trend of values: MUFA>SFA>PUFA and phospholipids>sterols. Whereas total lipid was 14.2 g/100g, total fatty acid was 13.0 g/100g (91.5%) ≡ 13.0 EPg/100g ≡ 481 kJ/100g ≡ 117 kcal/100g. The total energy from all unsaturated FA (TUFA) was 321 kJ/100g (78.0 kcal/100g) totalling 66.6% of the total energy contribution of the FAs. Both LA/ALA (9.06) and total n-6/total n-3 FA of 2.89 were within the acceptable ratio of 10:1. The following ratios were also good nutritionally: MUFA/SFA, PUFA/SFA, EPA/DHA, AA/DGLA, EPSI, AA/EPA+DHA, C16:0 to C18:1 cis-9 and C18:0 to C18:1 cis-9. Sterol levels were generally low and hence the corresponding cholesterol was low. However, the components of the phospholipids were all generally high. The ratio of sterols to phospholipids was 1:69. Therefore, kilishi lipid could be useful for people trying to watch their weight as far as nutritional fat is concerned.

Conflict of Interest

Authors declare that there is no conflict of interest reported in this work.

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