LIPID COMPOSITION OF VARIETY MEATS (STOMACH, INTESTINE, LIVER) OF BULL

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Abstract

Lipid composition of the dietary oils of the stomach, intestine and liver of bull found in Nigeria was determined by gas chromatography. Crude fat ranged from 4.10–5.22 g/100 g. SFA level ranged from 60.7–94.7 % of total fatty acids. MUFA was also widely distributed among the samples and composed the second largest fraction of 4.65–34.4 %. The n–6 PUFA constituted the third largest group of 1.29–6.74 % whereas the n–3 PUFA of 0.047–1.15 % formed the fourth and lowest group. Most concentrated SFA was palmitic acid; highest MUFA was C18:1 (cis–9) (intestine), C18:1 (cis –9) (liver), C18:1 (trans – 11) (stomach); highest n–6 was C18:2 (cis–9, 12) (intestine), C18:2 (trans 9, 11) (liver), C18:2 (cis –9, 12) (stomach); highest n–3 was C18:3 (cis–9, 12, 15) in all the samples. Cholesterol was the only sterol detected 274–335 mg/100 g. The highest phospholipid was phosphatidylcholine having a range of 1.76–2.89 mg/100 g (58.6–62.3 %). 100 g bull liver would provide 1.85 g of SFA, 1.05 g of MUFA and 0.15 g of PUFA with corresponding energy levels (kJ/100 g) of 68.5, 38.9, 5.55. The n–6/n–3 range was 3.28–27.4 whereas LA/ALA was 0.302 –20.9 and EPSI was 0.143–0.341. Statistical analysis showed significant differences existed ($\alpha = 0.05$) in the SFA, MUFA (cis), MUFA (trans), MUFA (total), n–6/n–3 and LA/ALA among the three samples in each case. Results of correlation analysis between Liver/Intestine and Liver/Stomach showed that significant differences occurred in each case at $r_{= 0.05}$.

Keywords: *Bull, offal, lipid composition.*

INTRODUCTION

Offal, also called, especially in the United States, variety meats or organ meats, refers to the internal organs and entrails of a butchered animal (Oxford University Press, 2001). The word does not refer to a particular list of edible organs, which varies by culture and region, but includes most internal organs other than muscle and bone. As an English mass noun, the term "offal" has no plural form. Some cultures shy away from offal as food, while others use it as everyday food, or in delicacies. Some offal dishes are considered gourmet food in international cuisine. This includes *foiegras*, *pâté* and sweetbreads. Other offal dishes remain part of traditional regional cuisine and may be consumed especially in connection with holidays. This includes Scottish haggis, Jewish chopped liver, Southern US and African–American chitterlings, as well as many other dishes. Intestines are used as casing for sausages, although cheaper types may use artificial casing. Depending on the context, offal may refer to those parts of an animal carcass discarded after butchering or skinning; it may also refer to the by–products of milled grains, such as corn or wheat (Merreiam–Webster Online Dictionary, 2011). Offal not used directly for human or animal food is often processed in a rendering plant, producing material that isused for fertilizer or fuel; or in some cases, it may be added to commercially produced pet food. In earlier times, mobs sometimes threw offal and other rubbish at condemned criminals as a show of public disapproval (Norton, 1810): In 1809 Richard Thomas Dudman and Edward Wood were convicted of a "conspiracy" to commit sodomy, and sentenced to two years' imprisonment and to stand for one hour in the pillory, where they were pelted with offal supplied by the butchers of Newgate and Fleet Markets.

In some parts of Europe, scrotum, brain, chitterlings (pigs small intestine), trotters (feet), heart, head (of pigs, calves, sheep and lamb), kidney, liver, spleen, "lights" (lung), sweetbreads (thymus or pancreas), fries (testicles), tongue, snout (nose), tripe (reticulum) and maws (stomach) from various mammals are common menu items. In some Latin American countries, such as Mexico, almost all internal parts and organs are consumed regularly. Chicken hearts, gizzards and livers are usually eaten fried or boiled, either alone, or in broth. Brainstem is served as soup, *sopa de médulla*. The tongue is boiled to make tacos. Eyes are eaten as *tacos de ojo. Tripas* (intestines) are also eaten, but normally in tacos rather than stews. In Africa, sausage is made from the small intestine of a goat, cow or sheep, stuffed with chilli and small chunks of meat, fatty meat, and blood (although some people prefer the bloodless kind). In Kenya it is commonly referred to as 'mutura' which is the Kikuyu

name for it. Sheep's or goat's stomach is also stuffed in a similar way. In South Africa offal is enjoyed by South Africans of diverse backgrounds. Due to the popularity of this dish, it is one of the few customs that white (especially Africaners) and black South Africans share. In China, many organs and animal–parts are used as for food or traditional Chinese medicine. In Indonesia cow and goat internal organs are popular delicacies; it can be fried, made into Soto soups of grilled as satay and almost all the parts of the animal are eaten. In India and Pakistan, the goat's brain (*maghaz*), feet (*paey*), head (*siri*), stomach (*ojhari* or *but*) tongue (*zabaan*), liver (*kalayi*), kidney (*gurda*), udder (*kheeri*) and testicles (*kapooray*) as well as chickens' heart and liver are enjoyed. One popular dish, *Kata–Kat*, is a combination of spices, brains, liver, kidneys and other organs. Beef offal is relished with the above mentioned parts regularly used in food, especially fried delicacies. In Lebanon, lamb brain is used in *nikhaat* dishes and sometimes as a sandwich filling. Another popular dish in the region surrounding is *korouch* which is rice–stuffed sheep intestine. Overall, the consumption of variety meats is considerably less common in North America, the United States in particular. In Australia offal is most commonly consumed in meat pies or in ethnic dishes.

A cow is a ruminant animal, which means they have one stomach that contains four compartments. The feed passes through the oesophagus into the first compartment of the stomach, known as the rumen. This compartment is by far the largest stomach compartment, with a volume of 40 to 50 gallons in a typical cow. The rumen is also known as the "fermentation vat" because feedstuffs undergo a fermentation process while in the rumen. The inside surface of rumen is covered with papillae, small finger–like projections that aid in absorption by increasing the surface area of the rumen. The rumen contains five compartments. The reticulum is the second compartment of the ruminant stomach. After feed passes through the rumen and reticulum, it enters the omasum (many plies or many leaves). The final compartment of the stomach is the abomasum, otherwise known as the true stomach. This compartment is most similar to the stomach of humans and other monogastric (single–stomached) animals. Partially digested feed, known as digesta or chime, flows from the abomasum to the small intestine. The average small intestine is 130 feet long and holds 10 gallons of digesta. The small intestine is the primary site of nutrient absorption in all animals. The small intestine is made up of three different sections, the duodenum, the jejunum and the ileum. The liver secretes bile into the small intestine. Bile helps to neutralize the digesta as it enters the small intestine, and is important for the digestion of fats. In addition to secreting bile, the liver plays a tremendously important role in converting certain absorbed nutrient into compounds that are more useful to the animal: conversion of propionate and lactate absorbed from the rumen into glucose; conversion of absorbed fatty acids into forms better suited for transport through blood and use by the tissues, and conversion of absorbed ammonia into the less toxic compound urea.

There are many breeds of cattle found in Nigeria. They include White Fulani, SokotoGudale, Red Longhorn (Raheja), Shuwa cattle, Adamawa, Biu cattle, Chad, Nigerian Shorthorn (Muturu) and N'Dama. White Fulani is a strong, big, robust breed with a white coat with black ears and nuzzle, black tongue and eyebrows and often black hooves. The body colour is very variable from white, through red and roan to black (Phillips, 1977). There is hardly any information on the contribution of bull intestine, stomach and liver to dietary lipid source when used as food. The purpose of the present report was as food. The purpose of the present report was to expose evidence relating to the lipid composition of the bull offal as contributor to the availability of lipids when used as protein food source.

MATERIALS AND METHODS

Collection of Samples

The bull (White Fulani) offal was collected from butchers who daily slaughtered cattle for the meat from the slaughter house based in Ado Ekiti.

Samples Treatment

On arrival in the laboratory, the samples were rinsed with distilled water and dried in the oven for about 5 h until constant weight. After drying, the samples were ground, sieved and kept in freezer (–4^{o}C) in McCartney bottles pending analysis.

Extraction of Lipid

0.25 g of each sample was weighed into the extraction thimble. 200 ml of petroleum ether (40–60^{o}C 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, 2005). The lipid was extracted for 5 h. The extraction flask with the oil was oven dried at 105^{o}C for 1 h. The flask containing the dried oil was cooled in the desiccator and the weight of the cooked flask with the dried oil was taken.

Preparation of Methyl Esters and Analysis

50 mg of the extracted oil was saponified for 5 min at 95^{o}C with 3.4 ml of 0.5 M KOH in dry methanol. The mixture was neutralised by 0.7 MHCl. 3 ml of 14 % boron triflouride in methanol was added (AOAC,

2005). The mixture was heated for 5 min at 90ºC to achieve complete methylation process. The fatty acid methyl esterswere thrice 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 the GC. The fatty acid methyl esters were analysed using an HP 5890 powered with HP gas chromatograph (HP 5890 powered with HP ChemStation rev. A09.01 [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 at10 ºC/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 x 0.25 mm) packed with a polar compound (HP INNOWAX) with a 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 standard fatty acid methyl esters.

Sterol Analysis

Sterol was analysed as described by AOAC (2005). The aliquots of the extracted fat were added to the screw–capped test tubes. The sample was saponified at 95ºC for 30 min, using 3 ml of 10 % KOH in ethanol, to which 0.20 ml of benzene had been added to ensure miscibility. Deionised water (3 ml) was added and 2 ml of hexane was added in extracting the non–saponifiable materials. Three extractions, each with 2 ml 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 injection pot of GC. The peaks were identified by comparison with standard sterols. The sterols were analysed using similar chromatographic conditions as for fatty acid methyl ester analysis.

Phospholipids 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 phospholipids 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 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 analysed using an HP 5890 powered with HP gas chromatograph (HP 5890 powered with HP ChenStation rev. A09.01 [1206] software [GMI, Inc, Minnesota, USA]) fitted with a pulse flame photometric detector. Nitrogen was used as the carrier gas with a flow rate of 20–60 ml/min. The oven programme was: initial temperature at 50ºC, whilst detector temperature was 320ºC. A capillary column (30 m x 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 per 100 g in Sample

At the data source and reference database levels, values for individual fatty acids are usually expressed as percentages of total fatty acids. At the user database levels, values per 100 g of food are required. A conversion factor derived from the proportion of the total lipid present as fatty acids is required for converting percentages of total acids to fatty acids per 100 g of food. Total lipid level was multiplied by a conversion factor of 0.741 to convert it to total fatty acids (Anderson, 1976). (This calculation was only done for the liver because it had a conversion factor while others do not have.) For fatty acids, precision is best limited to 0.1 g/100 g of fatty acids (Greenfield & Southgate, 2003).

Statistical Analysis

Statistical analysis (Oloyo, 2001) was carried out to determine coefficient of variation in per cent (CV%), linear correlation coefficient (r_{xy}), coefficient of determination (r_{xy}^2), coefficient of alienation (C_A) and index of forecasting efficiency (IFE). The r_{xy} was subjected to the table (critical) value at $r_{=0.05}$ to see if significant differences existed in the values of Liver/Intestine and Liver/Stomach ratios. Also determined and subjected to the table values (Oloyo, 2001) were SFA (total), MUFA (*cis*), MUFA (*trans*), MUFA (total), PUFA (total), *n*–6, *n*–3, n–6/n–3, PUFA/SFA, MUFA/SFA, LA/ALA, EPSI for their

Chi Square (X^2) values at $\alpha = 0.05$ at both vertical and horizontal columns.

RESULTS AND DISCUSION

Table 1 depicts total lipids and calculated total fatty acid (for liver only) levels on dry weight basis. The values of total lipids between the three samples were close with the CV% of 12.0. The total fat of 4.10–5.22 g/100 g were slightly close to the value of 7 g/100 g in calf liver (Bender, 1992) but very much lower than in bull brain of 42.5 g/100 g (Adeyeye, 2012); chicken's meat and skin (18 g/100 g), beef fat (67 g/100 g), lamb fat (72 g/100 g) and pork fat (71 g/100 g) (Bender, 1992). Table 2 shows the saturated fats (SFA), monounsaturated fat (MUFA) and polyunsaturated fats (PUFA) of the samples. In the bull offal samples, the following SFA recorded 0.0 % value: C20:0, C24:0 with not detected (nd) for C2:0, C3:0, C5:0 in all the samples but specifically C6:0, C8:0 in both intestine and liver but C4:0 only in the stomach. For the MUFA, the three samples had 0.0 % value for C22:1 (*cis*–13) and C24:1 (*cis*–15). For the PUFA, the following recorded 0.0 % value: C20:3 (*cis*–8, 11, 14), C20:3 (*cis*–11, 14, 17), C20:4 (*cis*–5, 8, 11, 14), C22:2 (*cis*–13, 16), C20:5 (*cis*–5, 8, 11, 14, 17) and C22:6 (*cis*–4, 7, 10, 13, 16, 19).

When ruminants eat carbohydrates, the ruminal microorganisms release enzymes that break them down into monosaccharides. The monosaccharides are them converted by the microorganisms through fermentation into volatile fatty acids (VFA). The VFA (primarily acetate, propionate and butyrate) are absorbed across the wall of the rumen and the small intestine, and are used by an animal as an energy source. In the present report C4:0 levels were 0.0082 % (intestine) and 0.059 % (liver) but not detected in stomach. On the other hand higher VFA components in SFA were detected in the stomach: 0.141 % (C6:0) and 0.14 % (C8:0); whose values were higher than the VFA values in liver and intestine. C10:0 and C12:0 were of moderate values in the SFA with respective range values of 2.98–6.74 % and 2.86–5.85 %. Short–chain fatty acids have four to six carbon atoms. These fats are always saturated. Four–carbon butyric acid is found mostly in butterfat from cows, and six–carbon capric acid is found mostly in butterfat from goats. These fatty acids have antimicrobial properties– that is, they protect us from viruses, yeasts and pathogenic bacteria in the gut. They do not need to be acted on by the bile salts but are directly absorbed for quick energy. For this reason, they are less likely to cause weight gain than olive oil or commercial vegetable oils (Portillo *et al*., 1998). Short–chain fatty acids also contribute to the health of the immune system (Kabara, 1978). Medium–chain fatty acids have eight to twelve carbon atoms and are found mostly in butterfat and the tropical oils. Like the short–chain fatty acids, these fats have antimicrobial properties; are absorbed directly for quick energy; and contribute to the health of the immune system. Long–chain fatty acids have from 14 to 18 carbon atoms and can be either saturated, monounsaturated or polyunsaturated. Not all SFA are equivalent in their potential to raise plasma cholesterol levels (Ulbricht and Southgate, 1991). As shown above, short–and medium–chain fatty acids have not been shown to influence plasma cholesterol because they are absorbed directly into blood and rapidly metabolized in the liver. Stearic acid (18:0) does not affect LDL–cholesterol, possibly because it is rapidly metabolized to oleic acid (18:1); C18:0 occurred at levels of 15.8–22.5 % in the present samples. Lauric (12:0), myristic (14:0) and palmitic (16:0) acids were originally regarded as the three 'cholesrerol–raising' fatty acids affecting total and LDL–cholesterol concentrations. Myristic acid is probably the most potent and has been estimated to have four times the effect of the other two SFA. Palmitic acid is the principal SFA in most diets. This is the position in the present results where C16:0 values ranged from 26.4–34.1 %. There is now some evidence to suggest that it may not raise cholesterol as much as C14:0, provided that intakes of n–6 PUFA are above a certain threshold and intakes of dietary cholesterol are low (Hayes & Khosla, 1992). The importance of SFA as a group being responsible for 'cholesterol–raising' effect of diets may have to be re–evaluated. It is possible that SFA intakes are only important in those people consuming significant quantities of full–fat dairy products, food containing coconut and palm kernel oils (the major sources of lauric and myristic acids), and low levels of n–6 PUFA (British Nutrition Foundation, 1997). C14:0 in our samples ranged from 12.2–25.5 %. Despite much research, the mechanism by which specific SFAs raise LDL–cholesterol is not fully understood. Plausible explanations for why certain SFAs raise LDL levels include:

- Inhibiting removal of LDL from plasma by interfering with LDL receptors in the liver; and
- Stimulating LDL synthesis directly.

It should generally be noted however, that during rumen fermentation, most of the unsaturated fats in the diet are converted to saturated fats. This is why most of the fat in the milk and meat of ruminants is saturated fat. This is shown vividly in the samples as total SFA ranged between 60.7–94.7 %. The present SFA values were all greater than the following literature values (%) in: beef fat (43), lamb fat (50), pork fat (37), chicken–meat and skin (33), duck– meat and skin (27) and calf liver (30) (Bender, 1992). Long–chain SFAs

increase postprandial TG responses, especially if background diet is rich in SFAs. Behenic acid (C22:0) was at trace level (0.0058–0.0987 %) in the samples.

The total *cis*–MUFA ranged from 1.81–17.5 % made up of C14:1 (*cis*–9, 0.012–0.107 %), C18:1 (*cis*–6, 1.18–6.77 %), C18:1 (*cis*–9, 0.609–10.6 %) and C20:1 (*cis*–11, 0.00384–0.0654 %). The only *cis*–MUFA of nutritional significance is oleic acid (18:1 *n*–9), but it normally makes the greatest single contribution of all fatty acids to the diet and is also the single most important FA in the body in quantitative terms. Here, oleic acid did not make the highest single contribution of all fatty acids in the samples, but it is reasonable in quantity in liver (10.6 %) and stomach (6.32 %). Recent studies had found that when *cis*–MUFA was substituted for SFA, *cis*–MUFA lowered plasma cholesterol concentration almost as effectively as *n*–6 PUFA. The reduction was mostly in LDL–cholesterol. When substituted for carbohydrates, *cis*–MUFA resulted in a similarly low plasma LDL–cholesterol but did not elicit the rise in VLDL (and therefore triglycerides) often seen with high carbohydrate diets. Neither did they lower HDL–cholesterol. An overview of the effect of *cis*–MUFA by regression analysis showed no evidence of an independent effect of *cis*–MUFA on plasma cholesterol (Hegsted *et al*., 1993). In studies relating *cis*–MUFA intakes to CHD, four out of six investigations found higher MUFA intakes in people who subsequently had a heart attack compared with people who did not. It is possible then that any 'protective effect' of MUFA might result from effects other than an effect on lowering LDL–cholesterol. The 1994 CRG [Coronary Review Group of the Committee on Medical Aspects of Food Policy (UK)] Report concluded that substitution of SFA in the diet with oleic acid, lowers both total and LDL–cholesterol in the plasma (Department of Health, 1994). c*is*–MUFA are less susceptible to oxidation than PUFA and foods containing them may have a longer shelf–life. There is also some evidence that LDL–cholesterol particles containing a high proportion of oleic acid compared with linoleic acid are less susceptible to oxidation. Since oxidized LDL is now thought to be more important than native (not oxidized) LDL in the development of atherosclerosis; this could partly explain some of the beneficial effects of MUFA.

The total *trans*–MUFA ranged between 2.84–16.9 % and made up of C18:1 (*trans*–6, 0.714–5.14 %), C18:1 (*trans*–9, 0.974–5.76 %) and C18:1 (*trans*–11, 0.984–7.33 %). The effects of *trans* fatty acids on plasma cholesterol have been reviewed a number of times with conflicting conclusions (British Nutrition Foundation, 1987, 1995). High *trans* fatty acid intake (10 % of dietary energy, compared with current average UK intakes of 2 % dietary energy) have been shown to raise LDL–cholesterol and to lower HDL–cholesterol (Mensink and Katan, 1990). Data from a prospective study on over 80,000 women considered *trans* fatty acid intake calculated from dietary questionnaires. After adjustment for age and total energy intake, a positive relationship was found between *trans* fatty acid intakes and the risk of CHD (highest intakes 1.5 times lowest intakes) (Willett *et al*., 1993). The British Nutrition Foundation's most recent Task Force Report on *trans* Fatty Acids (1995) concluded that:

> '*trans* fatty acids are qualitatively different to saturated fatty acids in their effects on HDL–cholesterol–saturated fatty acids raise HDL–cholesterol while *trans* fatty acids lower it. *trans* fatty acids raise LDL–cholesterol to approximately the same extent as saturated fatty acids. There is convincing evidence that *trans* fatty acids have an adverse effect on plasma LDL and HDL–cholesterol concentrations and this would appear to be greater than the adverse effect of an equivalent amount of saturated fatty acids'.

The most consistent evidence for the effect of dietary component on Lp (a) [lipoprotein (a)] levels is the effect of *trans* fatty acids. Several groups have shown that a diet high in *trans* fatty acids increased Lp (a) levels, by about 30 % in some cases (British Nutrition Foundation, 1995). Tissues of ruminant animals, such as cows, sheep and goats, can contain a number of different 18:1 isomers like C18:1 *trans*–9 (5.0 %) and C18:1 *cis*–9 (85 %), C18:1 *trans*–11 (47 %) and C18:1 *cis*–11 (47 %) (Hay & Morrison, 1973) with the *cis*–isomers, 9–and 11–18:1 slightly predominating as might be expected. 11*t*–18:1 makes up 50 % of *trans*–monoenes in ruminant tissues (which can comprise 10–15 % of the total monoenes or 3–4 % of the total FAs). In the present report C18:1 *trans*–11 had a range of 0.984–7.33 % of total FAs and 34.6–56.0 % of the *trans*–monoenes or 17.5–32.6 % of the total monoenes. The *n*–6 PUFA levels were C18:2 (*cis*–9, 12) (0.984–3.06 %), rumenic acid or C18:2 (*trans*–9, *cis*–11, conjugated linoleic acid, CLA) (0.279–2.97 %), C18:3 (*cis*–6, 9, 12) (0.036–0.66 %) and C20:2 (*cis*–11, 14) (0.00384–0.0654 %). The C18:3, *cis*–6, 9, 12 (gamma–linolenic acid, GLA) is found in evening primrose, borage and black currant oils. The body makes GLA from C18:2, *cis*–9, 12 and uses it in the production of substances called prostaglandins, localized tissue hormones that regulate many processes at the cellular level. Eicosadienoic acid [C20:2 *cis*–11, 14 or 20:2 (*n*–6) all *cis*–11, 14–eicosadienoic acid] or homo–gamma–linoleic acid is an uncommon naturally occurring PUFA. It is not

enriched in any particular tissue, it is rare in all lipid classes. Dietary sources include herring and menhaden oils, cattle liver (low in the present result), swine brain lipid and shark oil (Yagallof *et al*., 1995). The acid inhibits the binding of [^{3}H]–ITB$_4$ to pig neurophil membrane with a Ki of 3 µm. The levels of C18:2 *cis*–9, *trans*–11 were very close to the levels of C18:2 *cis* –9, 12 or 0.279–2.97 % and 0.984–3.06 % respectively. CLAs make up a group of PUFA found in meat and milk from ruminant animals and exist as a general mixture of conjugated isomers of LA. Of the many isomers identified, the *cis*–9, *trans*–11 CLA isomer (rumenic acid or RA) accounts for up to 80–90 % of the total CLA in ruminant products (Nuernberg *et al*., 2002). Naturally occurring CLAs originate from two sources: bacterial isomerization and /or biohydrogenation of *trans*–fatty acids in the adipose tissue and mammary glands (Griinari *et al*., 2000). Microbial biohydrogenation of LA and ALA by an anaerobic rumen bacterium *Butyrivibriofibrisolvens* is highly depend on rumen pH (Pariza *et al.,* 2000). *De novo* synthesis of CLA from 11*t*–C18:1 (TVA) has been documented in rodents, dairy cows and humans. True dietary intake of CLA should therefore consider native 9 *c* 11*t*–C18:2 (actual CLA) as well as the 11 *t*–C18:1 (TVA, potential CLA) content of foods (Adlof *et al*., 2000). Significant health benefits attributable to the actions of CLA have been documented (Kritchevsky *et al*., 2000).

The major effect of substituting n–6 PUFA for SFA, is a reduction of plasma cholesterol, principally the LDL fraction. There is little reduction in HDL–cholesterol as long as the contribution of linoleic acid (LA) is not more than 12 % of dietary energy. This level is unlikely to be exceeded as shown in our results. Plausible explanations for why unsaturated FAs, such as C18:1 *cis*–9 and C18:2 *cis*–9, 12 might lower LDL include:

- Over–riding the inhibition in LDL receptor activity caused by certain SFA;
- Lowering LDL–cholesterol by counteracting the increase in LDL synthesis caused by certain SFA; and
- Simply substituting for SFA which raise LDL levels.

Only two out of six prospective studies found lower PUFA intakes in people who went on to have a heart attack (British Nutrition Foundation, 1992).

The only *n*–3 FA in the sample was C18:3 (*cis*–9, 12, 15) (ALA) with levels of 0.0471–1.145 %. In contrast to *n*–6 PUFA, the main effect of *n*–3 PUFA on the atherogenic lipid profile is to reduce the concentration of VLDL. Since the major lipid component of these lipoproteins is triglycerides, the main response is lowering of plasma triglyceride concentrations. Only at very high intakes does *n*–3 PUFA lower LDL or total cholesterol. The dramatic effect of a Mediterranean α–linolenic acid – rich diet in the secondary prevention of CHD (70 % reduction in coronary events and cardiac events) was achieved without reduction in serum cholesterol, TG or an increase in HDL–cholesterol compared with controls (de Lorgeril *et al*., 1994). The effect might have been due to an effect on reducing the risk of thrombosis. The fatty acid composition of the brain of bull (% total fatty acid): SFA (6.11), MUFA (all *cis*) (8.89), LA (2.27), GLA (1.90), *n*–6 PUFA (36.7), rumenic acid (2.30), PUFA (*n*–6 total) (39.0) and ALA (2.22) (Adeyeye, 2012). In rabbit, lean, C18:2 (13.5 %), C18:3 (0.7 %); brain, sheep, 18:2 (0.4 %), 18:3 (–); liver: ox, 18:2 (7.4 %), 18:3 (2.5 %), sheep, 18:2 (5.0 %), 18:3 (3.8 %), pig, 18:2 (14.7), 18:3 (0.5 %), calf, 18:2 (15.0 %) and 18:3 (1.4 %) (Paul & Southgate, 1978).

Table 1: Crude fat levels of bull variety organs (g/100 g dry weight)

Parameter	Intestine	Liver	Stomach	Mean	SD	CV%
Crude fat	4.75	4.10	5.22	4.69	0.562	12.0
Total fatty acid *****	-	3.04	-	-	-	-

*****Crude fat x 0.741; **CV% =**coefficient of variation

Table 2: Fatty acids compositions (%) of the intestine, liver and stomach of bull

Fatty acid	Intestine	Liver	Stomach	Mean	SD	CV%
C2:0	–	–	–	–	–	–
C3:0	–	–	–	–	–	–
C4:0	0.0082	0.059	–	0.034	0.036	106
C5:0	–	–	–	–	–	–
C6:0	–	–	0.141	–	–	–
C8:0	–	–	0.14	–	–	–
C10:0	6.74	2.98	3.71	4.48	1.20	44.5
C12:0	5.85	3.25	2.86	3.99	1.63	40.8
C14:0	25.5	12.2	16.2	18	6.82	37.9
C16:0	34.1	26.4	29.6	30	3.87	12.9
C18:0	22.5	15.8	17.1	18.5	3.56	19.2
C20:0	0.00	0.00	0.00	–	–	–
C22:0	0.0058	0.0177	0.0987	0.041	0.051	123
C24:0	0.00	0.00	0.00	–	–	–
SFA total	94.7	60.7	69.8	75.1	17.6	23.4
C14:1 (*cis–9*)	0.012	0.055	0.107	0.058	0.047	81.8
C18:1 (*cis–6*)	1.18	6.77	2.87	3.60	2.87	79.8
C18:1 (*cis*–9)	0.609	10.6	6.32	5.84	5.02	85.8
C20:1 (*cis* 11)	0.00384	0.0117	0.0654	0.027	0.034	124
C22:1 (*cis*–13)	0.00	0.00	0.00	–	–	–
C24:1 (*cis*– 15)	0.00	0.00	0.00	–	–	–
MUFA (*cis*)	1.81	17.5	9.40	9.57	7.85	82
C18:1 (*trans–6*)	0.88	5.14	0.714	2.24	2.51	112
C18:1 (*trans–9*)	0.974	5.76	5.09	3.94	2.59	65.7
C18:1 (*trans–11*)	0.984	6.01	7.33	4.78	3.35	70.1
MUFA (trans)	2.84	16.9	13.1	10.9	7.27	66.7
C18:2 (cis–9,12)	0.984	1.54	3.06	1.86	1.10	57.9
C18:2 (*trans–9,11*)	0.279	1.55	2.97	1.60	1.35	84.3
C18:3 (*cis*–6, 9,12)	0.036	0.66	0.647	0.445	0.357	79.6
C18:3 (*cis*–9,12,15)	0.0471	1.145	0.925	0.706	0.581	82.3
C20:2 (*cis–11,14*)	0.00384	0.0117	0.0654	0.027	0.034	124
C20:3 (*cis*–8,11,14)	0.00	0.00	0.00	–	–	–
C20:3 (*cis*–11,14,17)	0.00	0.00	0.00	–	–	–
C20:4 (*cis*– 5,8,11,14)	0.00	0.00	0.00	–	–	–
C22:2 (*cis*–13,16)	0.00	0.00	0.00	–	–	–
C20:5 (*cis*–5,8,11,14,17)	0.00	0.00	0.00	–	–	–
C22:6 (*cis*– 4,7,10,13,16,19)	0.00	0.00	0.00	–	–	–
PUFA total	1.34	4.92	7.67	4.64	3.17	68.4

Table 3. Calculated parameters from fatty acids

Parameter	Intestine	Liver	Stomach	Mean	SD	CV%	X^2	Remark
SFA *total*	94.7	60.7	69.8	75.1	17.6	23.4	8.25	S
MUFA *cis*	1.81	17.5	9.40	9.57	7.85	82	12.9	S
MUFA *trans*	2.84	16.9	13.1	10.9	7.27	66.7	9.71	S
MUFA *total*	4.65	34.4	22.5	20.5	15	73	21.9	S
PUFA *total*	1.34	4.92	7.67	4.64	3.17	68.4	4.34	NS
n-6	1.29	3.77	6.74	3.93	2.73	69.4	3.73	NS
n-3	0.047	1.15	0.925	0.707	0.583	82.4	0.961	NS
n-6/n-3	27.4	3.28	7.29	12.7	12.9	102	26.3	S
PUFA/SFA	0.014	0.081	0.11	0.068	0.049	72.4	0.071	NS
MUFA/SFA	0.049	0.567	0.322	0.313	0.259	80.5	0.229	NS
LA/ALA	20.9	0.747	0.302	7.31	11.8	161	37.9	S
EPSI	0.288	0.143	0.341	0.257	0.103	39.9	0.537	NS
X^2	632	315	345	-	-	-	-	
Remark	S	S	S					

S - Significant
NS- Not Significant,
EPSI- Essential PUFA Status Index

Table 4. Statistical analysis of the calculated parameters from fatty acids

Samples	r_{xy}	$r_{xy}2$	C_A	**IFE**	**Table value**	**Remark**
Liver/Intestine	0.7623	0.5811	0.6472	0.3528	0.576	S
Liver/Stomach	0.9594	0.9204	0.2821	0.7179	0.576	S

Table 3 contains some parameters calculated from Table 2. The Table was further subjected to statistical analysis. With exception of CV% in SFA total (23.4), all other CV% were high. On the analysis of chi square (X^2) at $\alpha = 0.05$, the following values were significantly different among the samples (horizontal lines): SFA total, MUFA *cis*, MUFA *trans*, MUFA total, *n*–6/*n*–3 and LA/ALA whereas all the parameters determined were significantly different among each sample (as shown vertically). Further comparisons (Table 4) were made in the fatty acid results in liver/intestine and liver/stomach subjecting them to correlation determination. The r_{xy} was significant at $r_{=0.05}$; whereas the index of forecasting efficiency (IFE) was low in liver/intestine, it was high in liver/stomach. In Table 5 are shown the FAs level in the bull per 100 g liver sample as food and their corresponding energy contributions. This conversion to g/100 g as food of liver FAs was possible because the conversion factor of crude fat to total fatty acids was available (crude fat x 0.741 = total fatty acids) (Anderson, 1976). The energy contribution from the SFA was very high at 60.9 % which doubles the expected contribution. The energy contributions from both MUFA *cis* and MUFA *trans* were close at 17.5 % and 16.9 %, respectively. Energy contribution from PUFA total was moderate at 4.93 %.

Table 5: Fatty acids level in the bull per 100 g liver sample as food and their corresponding energy contributions

Fatty acid	g/100 g as food	Energy kJ/100 g	% Energy contribution
C2:0	–	–	–
C3:0	–	–	–
C4:0	0.002	0.066	0.059
C5:0	–	–	–
C6:0	–	–	–
C8:0	–	–	–
C10:0	0.091	3.37	2.99
C12:0	0.099	3.66	3.26
C14:0	0.371	13.7	12.2
C16:0	0.803	29.7	26.4
C18:0	0.48	17.8	15.8
C20:0	–	–	–
C22:0	0.0005	0.02	0.018
C24:0	–	–	–
SFA *total*	1.85	68.5	60.9
C14:1 (*cis–9*)	0.002	0.062	0.055
C18:1 (*cis–6*)	0.206	7.62	6.78
C18:1 (*cis*–9)	0.322	11.9	10.6
C20:1 (*cis* –11)	0.0004	0.013	0.012
C22:1 (*cis*–13)	–	–	–
C24:1 (*cis*– 15)	–	–	–
MUFA (*cis*)	0.532	19.7	17.50
C18:1 (*trans–6*)	0.156	5.77	5.13
C18:1 (*trans–9*)	0.175	6.48	5.76
C18:1 (*trans–11*)	0.183	6.77	6.02
MUFA *trans*	0.514	19	16.9
MUFA *total*	1.05	38.9	34.5
C18:2 (*cis*–9,12)	0.047	1.74	1.55
C18:2 (*trans–9,11*)	0.047	1.74	1.55
C18:3 (*cis*–6, 9,12)	0.0201	0.74	0.658
C18:3 (*cis*–9,12,15)	0.035	1.3	1.15
C20:2 (*cis–11,14*)	0.0004	0.013	0.012
C20:3 (*cis*–8,11,14)	–	–	–
C20:3 (*cis*–11,14,17)	–	–	–
C20:4 (*cis*– 5,8,11,14)	–	–	–
C22:2 (*cis*–13,16)	–	–	–
C20:5 (*cis*–5,8,11,14,17)	–	–	–
C22:6 (*cis*– 4,7,10,13,16,19)	–	–	–
PUFA *total*	0.15	5.55	4.93
n–6	0.115	4.26	3.78
n–3	0.035	1.30	1.15

In the analysis of sterols (Table 6) only cholesterol was detected in both samples with values of (mg/100 g): 335 (intestine), 236 (liver) and 274 (stomach) with CV% of 17.7. Cholesterol is a fatty compound involved in the transport of fat in the blood stream and is also part of the structure of cell membranes of tissues of the body. It is not a dietary essential dietary ingredient. Confusion has arisen between the terms blood cholesterol and dietary cholesterol. For most individuals dietary cholesterol has little or no effect on blood cholesterol levels because reduced synthesis in the body compentates for increased dietary intake (Bender, 1992). However, there are individuals who are sensitive to dietary cholesterol (Reiser & Shorland, 1990) and most authorities advise a general reduction in cholesterol intake for everyone. These sterols recorded 0.0 mg/100 g in the samples: cholestanol, ergosterol, campesterol, stigmasterol, 5–avenasterol and sitosterol as observed in bull brain (Adeyeye, 2012). Meat supplies about one third of the dietary cholesterol in many western diets with the remainder from eggs and dairy products. Since all these foods are valuable sources of nutrients there could be some nutritional risk in restricting their intake. Most authorities, but not all, recommend a reduction in dietary cholesterol to around 300 mg or less per day (Bender, 1992); this is more than the level in liver and stomach in 100 g sample under discussion. Some literature values of cholesterol were as shown (mg/100): fish (50–60), egg yolk (1260), meat and poultry (60–120), brain (2000–3000), liver (300–350) (Bender, 1992). Sheep brain contains 2200 mg/100 g cholesterol level (Paul & Southgate, 1978). Garcia *et al.* (2008) reported (cholesterol g/100 g) 40.3 and 45.8 or 40300 and 45800 mg/100 g of tissue in pastured and grain–fed steers (castrated bulls), respectively ($p < 0.001$). Report of the cholesterol in the brain of the Nigeria bull is 974 mg/100 g (Adeyeye, 2012).

Table 6: Sterols level (mg/100g) of the intestine, liver and stomach of bull

Sterols	Intestine	Liver	Stomach	Mean	SD	CV%
Cholesterol	335	236	274	282	49.9	17.7
Cholestanol	0.00	0.00	0.00	–	–	–
Ergosterol	0.00	0.00	0.00	–	–	–
Campesterol	0.00	0.00	0.00	–	–	–
Stigmasterol	0.00	0.00	0.00	–	–	–
5– Avenasterol	0.00	0.00	0.00	–	–	–
Sitosterol	0.00	0.00	0.00	–	–	–
Total	**335**	**236**	**274**	**282**	**49.9**	**17.7**

Table 7: Phospholipids level (mg/100g) of the intestine, liver and stomach of bull

Phospholipids	Intestine	Liver	Stomach	Mean	SD	CV%
Phosphatidylethanolamine	0.639(14.2%)	0.642(19.9%)	0.647(15.7%)	0.651	0.0195	2.99
Phosphatidylcholine	2.63 (58.6%)	1.76 (54.5%)	2.89 (62.3%)	2.43	0.59	23.8
Phosphatidylserine	0.438(9.76%)	0.429(13.3%)	0.374(8.06%)	0.414	0.0348	8.39
Lysophosphatidylcholine	0.482(10.7%)	0.372(11.5%)	0.391(8.43%)	0.415	0.059	14.2
Phosphatidylinositol	0.294(6.55%)	0.0243(0.752%)	0.314(6.77%)	0.211	0.162	76.6
Total	**4.49**	**3.23**	**4.64**	**4.12**	**0.774**	**18.8**

Table 7 shows the levels of the various phospholipids. Phosphatidylcholine (PC) was the most abundant phospholipid in the three samples forming levels of (mg/100 g): 2.63 (58.6 %) (intestine), 1.76 (54.5 %) (liver), and 2.89 (62.3 %) (stomach). PC is the most abundant phospholipid in brain cell membranes comprising about 30 % of the total phospholipid content while phosphatidylethanolamine (PE) came second with levels of 0.639–0.647 mg/100 g or 14.2–19.9 %. Lecithin (PC) is usually the most abundant phospholipid in animals and plants, often amounting to almost 50 % of the total, and as such it is the key building block of membrane bilayers. This observation is true for lecithin values in these results with percentage values ranging from 54.5 % – 62.3 %. PC is a class of phospholipids that incorporate choline as a headgroup. They are a major component of biological membranes and can be easily obtained from a variety of readily available sources such as egg yolk or soy beans from which they are mechanically extracted or chemically extracted using hexane. They are also a member of the lecithin group of yellow–brownish fatty substances occurring in animal and plant tissues. At birth and throughout infancy, phosphatidylcholine concentrations are high (as high as 90 % of the cell membrane), but it is slowly depleted throughout the course of life, and may drop to as low as 10 % of the cellular membrane in the elderly. As is such, some researchers in the fields of health and nutrition have begun to recommend daily supplementation of phosphatidylcholine as a way of slowing down senescence (Mei–Chu, 2001) and improving brain functioning and memory capacity (Chung *et al*., 1995). It is the principal phospholipid circulating in plasma, where it is an integral component of the lipoproteins, especially the HDL (Whitney *et al*., 1994). The CV% levels ranged between 2.99–76.6.

Quality Assurance

The correlation determined for all the standards: fatty acids, phospholipids and sterols, all had values ranging as follows: 0.99833–0.99997 (fatty acids), 0.99909–0.99999 (phospholipids) and 0.99920–0.99994 (sterols); all the correlation values were greater than 0.95 which is the critical correlation for acceptance of these types of analytical results, thus attesting to the quality assurance of the determinations.

CONCLUSIONS

The findings of this study showed that the samples demonstrated the lipid composition of the offal (stomach, intestine, liver) of bull with unequal distribution of all the parameters determined. The samples were low in total fats, had SFA as the predominant fatty acids with percentage levels of 60.7–94.7 %, hence samples can be grouped into SFA group. Significant differences occurred in the fatty acid levels. Only cholesterol was the sterol found in levels greater than 0.00 mg/100 g in all the samples. Quality assurances of the determinations were highly satisfactory.

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