



Metabolizable and digestible energy of some selected snacks from south-western Nigeria

Uthman-Akinhanmi YO¹, Yangomodou OD^{2*}, Solana OI³, Olugbemi MT⁴

¹⁻⁴Department of Home and Hospitality Management Faculty of Agricultural Management and Rural Development College of Agricultural Sciences Olabisi Onabanjo University, Yewa Campus, Ayetoro Ogun State, Nigeria

Abstract

Knowledge of Metabolizable Energy (ME) and Digestible Energy (DE) of any food is an essential tool in weight loss/gain program. This study was conducted to determine the value of spent and unspent energy of some randomly franchised fast foods (FF) using albino rats. Seventy-eight (78) albino rats were fed ten FF using a randomized block experiment. Feces and urine from each rat were collected daily in triplicate and analyzed for DE and ME using standard laboratory procedures. The animals were sacrificed after an overnight fast and some internal organs were weighed. Data were analyzed using descriptive statistics, analysis of variance and Pearson Product Moment Correlation (PPMC). Daily energy intake of rats ranged from 58.5 to 124.54 kcal. ME and DE value ranged from 9.03 to 44.44 kcal and 11.22 to 45.28 kcal respectively. ME was significant 8.41 g and 4.54 to 7.92 g respectively. Average weekly weight gain of rat models ranged from 8.68 to 11.93 g. PPMC for liver weight of rat models correlated significantly with zinc intake ($r = 0.433$; $P \leq 0.05$). Fast foods are good sources of digestible energy which might be useful in programs requiring use of foods high in unspent energy.

Keywords: fast foods, albino rats, fecal energy, urinary energy

Introduction

Improper dietary or food habits especially consumption of high caloric foods versus inadequate corresponding energy output has been associated with excessive weight gain coupled with genetic susceptibility can result in development of health problems associated with poor diet (Martinez, 2000) [48]. Gross [GE], Digestible [DE], and Metabolizable [ME] energies are all specific terms associated with the energy value of food; Fecal [FE] and Urinary [UE] energies are the energy value from a food that is excreted (Miller and Judd, 1984 [52]; Livesey, 1991) [44]. The potential energy a food supplied after complete oxidation of the organic matter in food is known as gross energy (GE), whose amount depends on the proportion of calories supplied by macronutrients in food; when fecal energy losses are discounted from GE, the apparent digestible energy content of food (DE) is obtained and ME represents DE less energy losses in urine and gases (Castrillo et al., 2009) [14]. The FE and UE excretion in healthy persons generally accounts for at least five to ten percent of the total energy from the diet; the energy lost from excretion is determined by the food eaten and the endogenous or metabolic fecal and urinary nitrogen from the breakdown of bodily components (Kleiber, 1975 [40]; Garcia, 2003) [31]. Thus, investigating the various nutritional components of fast foods in relation to energy spent and unspent are necessary since fast foods consumed contain various constituents that work together to aid or hinder the development of diseases. In this study animal models have been used to assess the ME and DE of snacks.

Materials and Methods

Study Area

The study area is South-Western, Nigeria. Nigeria is made up of six geo-political zones with a total of 36 states and the Federal Capital Territory. The south west zone comprises of six states (Lagos, Ekiti, Ondo, Oyo, Ogun and Osun) with an estimated population of 28 million people (NPC, 2009). The study areas are Ikeja the capital of Lagos State, Abeokuta the capital of Ogun State and Ibadan the capital of Oyo State.

Sampling Procedures

A randomized block experiment was used where seventy-eight, ten weeks old albino rats were housed initially in individual plastic cages with wire meshes bottoms. All rats were kept on a 12-hour light/dark regimen (0700 hours light/1900 hours dark) with free access to food and water. The rats were placed in the plastic metabolic cages for a one-week acclimation period before data collection was initiated at week two. All rats were fed the control (a commercial rat chow) diet non-meat based [NMB] for the first two weeks (week1 and week2).

Formulation of Feeds

A snack was selected randomly each from ten different fast food outlets in three south-western states in Nigeria; five non-meat based [NMB] and five meat based [MB] snacks. These snacks [FF] were freeze-dried and pin milled to 100 meshes in standard particle size according to Eunmi and Jinho, (2015) [22];

and coded as follows: B30S [NMB]), doughnut 1; C30R [NMB], doughnut 2; S30T [NMB], doughnut 3; S60T [NMB], popcorn; T30R [NMB], doughnut 4 ; B40S [MB], scotched egg; T20R [MB], sausage roll 1; S20T [MB], sausage roll 2; C50R [MB], chicken pie and T10R [MB], meat pie.

Adaptation, Data Collection and Sacrifice

The rats were stratified by weight and assigned randomly to one of four treatment groups: baseline (n=6), control (n=6), reference (n = 6) and Fast Foods [FF] (n=60) rats and was subjected to two weeks adaptation period; all four groups were fed the commercial rat chow during this period. After the adaptation period i.e. week three(3), the baseline and the control rats were fed commercially produced rat chows (normal rat chow), the reference group were fed commercially rat chow for growth which has a high protein content and the FF rats groups were fed the formulated feeds (each selected feed per group).

After the adaptation periods, the study was carried out in twelve weeks (six metabolic periods) i.e. week 3 through week 14 (Garcia, 2003) [31]. Feces and urine were collected from each rat in a metabolic cage daily at 08:00 hours. Each urine collection tube contained 1 ml of 10% HCl, which was added to reduce nitrogen loss in the urine (Ozelci and Leveille, 1997) [55]. The collected feces sample was sprayed with 0.5N sulfuric acid and dried for 48 hours at 75°C, then crushed and stored at -10°C to prevent decomposition and the volatilization of nitrogen content (Eunmi and Jinho, 2015) [22].

Food intake for baseline, control, reference and FF groups were recorded daily. Body weight, in grams, was measured and recorded daily for all rats. The baseline group was sacrificed after an overnight fast prior to the beginning of the metabolic periods (Garcia, 2003) [31]. The baseline group was included in this study to show differences in energy accumulation of the control, reference and FF group carcasses throughout the six metabolic periods (Garcia, 2003 [31]; Chiem *et al.*, 2009). The control, reference and FF rats were fasted overnight, weighed and sacrificed after Metabolic Period 6 (Week 14).

Energy from FF, reference and control diet, feces, urine, and carcasses were determined using the bomb calorimeter (Parr 1722 Bomb Calorimeter, Moline, IL). GE measurements from the bomb calorimeter were used to determine the DE and ME of the control diet, the reference and FF component. Baseline rat data was used to determine the total energy gained by control, reference and FF rats.

Livesey (1989) established a calculation for determining the Digestible (DE) and metabolizable energy (ME) of a component within a diet: ME= Gross intake energy – (gross fecal energy + gross urine energy) [Energy value was recorded in kcal]. DE= Gross intake energy - gross fecal energy, [Energy values was recorded as kcal].

The heat of combustion and the total GE values for the baseline, control, reference and FF groups were compared to obtain the energy retained within the tissues of the rats. The average GE value for the baseline was subtracted from each individual rat in the control, reference and the FF group to provide the value of GE gained (Garcia, 2003 [31]; Chiem *et al.*, 2009). The nutrient composition of these feeds were carried out separately using standard laboratory procedures, but was not presented as tables in this study, but was correlated with the outcome of the aim of correlated with the outcome of the aim of this study.

Results and Discussion

Results

Table 1 shows the metabolizable and digestible energies of rats fed sample snacks. Urinary energy was significantly higher in the MB groups in comparison with the NMB group, whereas; fecal energy was significantly higher in the NMB group in comparison with the MB group. Likewise ME and DE were significantly higher in the NMB group in comparison with the MB group. Average feed intake within the B40S-fed group was constant throughout the 6 metabolic periods such that the weight of actual intake was ≥ 28 g within the non-meat based group, S60T-fed group maintained a high value of ≥ 27 g throughout the study period with S30T- fed group exhibiting a haphazard value of actual feed throughout the study period.

Table 1: Metabolizable and digestible energy of feeds

Rats fed diet	Urinary energy (kcal)	Fecal energy (kcal)	GE (kcal)	ME (kcal/g)	DE (kcal/g)	Net energy (kcal)	Av intake daily(Kcal)
B30S NMB	1.00 ^c ±0.20	84.16 ^a ±0.37	115.66 ^b ±2.00	3.05 ^{bc} ±1.54	3.15 ^{bc} ±1.63	62.28 ^b ±2.00	113.84 ^b ±0.49
C30R NMB	0.90 ^c ±0.08	82.46 ^{ab} ±0.90	114.95 ^b ±3.07	3.16 ^b ±1.82	3.25 ^b ±2.41	61.57 ^b ±3.07	113.67 ^b ±0.76
S30T NMB	0.52 ^{cd} ±0.03	83.63 ^a ±6.12	111.69 ^b ±0.51	2.75 ^{bcd} ±6.60	2.81 ^{bcd} ±6.55	58.30 ^b ±0.51	109.45 ^b ±0.20
S60T NMB	0.83 ^c ±0.62	79.01 ^{bc} ±3.80	124.54 ^a ±2.57	4.44 ^a ±5.34	4.53 ^a ±5.81	71.17 ^a ±2.59	114.18 ^a ±0.58
T30R NMB	0.52 ^{cd} ±0.04	76.90 ^{cd} ±0.82	116.28 ^b ±0.86	3.90 ^a ±1.16	3.95 ^a ±1.19	62.90 ^b ±0.86	113.56 ^b ±0.19
B40S MB	2.22 ^a ±0.02	68.29 ^f ±0.91	79.55 ^d ±0.51	0.90 ^g ±1.19	1.12 ^f ±1.24	26.17 ^d ±0.51	76.88 ^{cd} ±0.19
T20R MB	1.70 ^b ±0.03	74.06 ^{de} ±0.65	99.33 ^c ±0.55	2.36 ^{def} ±0.90	2.53 ^{bcd} ±0.92	45.95 ^c ±0.55	97.66 ^{bc} ±0.15
S20T MB	1.70 ^b ±0.05	71.18 ^{ef} ±0.76	100.35 ^c ±1.04	2.75 ^{bcd} ±1.24	2.92 ^{bcd} ±1.21	46.97 ^c ±1.04	98.86 ^{bc} ±0.29
C50R MB	1.48 ^b ±0.13	36.14 ^h ±0.22	58.55 ^f ±1.70	2.09 ^{ef} ±2.05	2.24 ^{de} ±1.92	5.16 ^f ±1.70	56.66 ^c ±0.80
T10R MB	1.70 ^b ±0.04	44.07 ^h ±0.58	63.99 ^e ±2.74	1.82 ^f ±2.13	1.99 ^c ±2.17	10.61 ^e ±2.74	62.99 ^d ±1.20
Control NMB	0.05 ^d ±0.02	21.25 ⁱ ±0.46	45.92 ^g ±9.78	2.43 ^{cd} ±0.96	2.44 ^{cd} ±0.96	-7.46 ^e ±5.77	41.29 ^f ±3.1854.49 ^e ±0.18
Reference MB	2.22 ^a ±0.04	49.27 ^g ±0.77	54.60 ^f ±0.55	0.31 ^g ±0.12	0.53 ^f ±0.12	1.22 ^f ±0.55	
Baseline NMB	0.63 ^d ±0.03	21.29 ⁱ ±0.47	3.38 ^f ±2.32	3.14 ^{bc} ±0.19	3.15 ^{bc} ±0.19	NA	

^{abcdeghij} Means along the serial column with different superscripts have significant difference (p<0.05)

The average weight gain during the metabolic periods as shown in plate 1, revealed a peak in C30R-fed group. At the beginning of the metabolic periods, there appeared to be no significant difference between all groups, but at the end of the

6 metabolic periods, there were significant difference across all groups with the control and reference group having the least total weight but with no significant difference. Animals in the FF groups continued to gain weight and were heavier

than control animals throughout the study period.

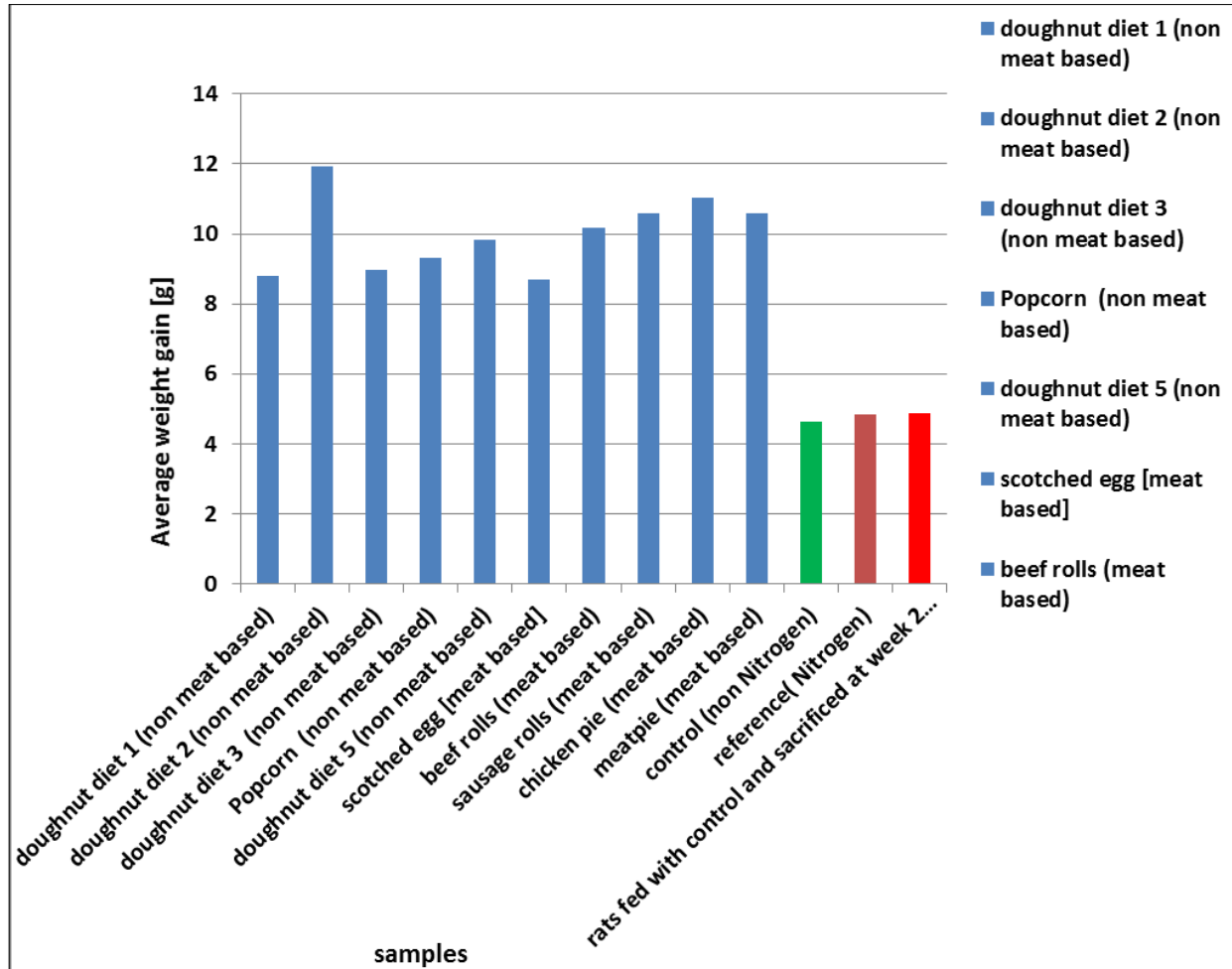


Fig 1: Average weekly weight gain of rat models

Table 2 shows the average and total weight gain of the rat models with both NMB and MB groups having no significant

difference in initial weight. There were variations in average and the overall weights among groups.

Table 2: Weight gain of rat models in grams

Rats fed diet	Initial weight(g)	Final weight (g)	Total weight gain (g)
B30S NMB	111.11 ^a ±21.66	216.52 ^{ab} ±29.89	105.41 ^{bc} ±10.47
C30R NMB	117.74 ^a ±13.26	260.92 ^a ±57.69	143.18 ^a ±44.52
S30T NMB	121.61 ^a ±21.80	229.17 ^{ab} ±18.57	107.56 ^{bc} ±6.36
S60T NMB	88.66 ^a ±21.17	200.30 ^{bc} ±16.15	111.56 ^{bc} ±5.75
T30R NMB	110.50 ^a ±5.76	228.59 ^{ab} ±25.09	118.08 ^{abc} ±19.47
B40S MB	92.32 ^a ±30.26	196.52 ^{bc} ±29.30	104.19 ^c ±4.86
T20R MB0	107.06 ^a ±35.49	229.06 ^{ab} ±40.84	122.03 ^{abc} ±5.34
S20T MB	102.97 ^a ±3.67	229.87 ^{ab} ±7.39	126.90 ^{abc} ±10.07
C50R MB	94.09 ^a ±17.09	226.54 ^{ab} ±12.63	132.45 ^{ab} ±4.47
T10R MB	120.35 ^a ±20.55	247.27 ^{ab} ±21.36	126.92 ^{abc} ±3.25
Control NMB	83.66 ^a ±8.01	139.33 ^{de} ±8.23	55.67 ^d ±3.32
Reference MB	100.86 ^a ±21.07	158.88 ^{cd} ±21.64	58.01 ^d ±0.67
Baseline NMB	98.09 ^a ±4.54	107.81 ^e ±4.02	9.27 ^e ±0.64

^{abcde} Means along the serial column with different superscripts have significant difference (p<0.05)

Table 3 shows the weight of internal organs of rats fed the different samples. There was significant difference (P≤0.05) in rats fed the same sample snack but obtained from different fast foods centers. C50R-fed groups had no significant

difference (P≤0.01) compared with the control group in GIT length. Heart weight in most groups had no significant difference (P≤0.01) with the highest being within the C50R-fed group. Caecum weight proved to be highest in the control

group and lowest in the baseline group. Within the meat based group, C50R-fed had the highest Caecum weight; within non-meat based group S60T-fed group had the highest Caecum weight. Liver weight was highest in the C50R fed group with the baseline group exhibiting the lowest value. There was no significant difference ($P \leq 0.01$) in kidneys weight in the non-meat based groups;

Kidneys weight was highest in reference group. There was no significant difference ($P \leq 0.01$) in stomach weight in all groups except the baseline group having the lowest value. Physical abdominal fat accumulation was significantly different ($P \leq 0.05$) across all groups, but there appeared to be no significant difference ($P \leq 0.01$) between the control and baseline groups.

Table 3: Weight of internal organs of the rat models

Rats	Heart [g]	Caecum [g]	Liver [g]	Kidneys [g]	GIT [g]	Stomach [g]	White fat [g]
B30S NMB	0.60 ^{ab} ±0.07	2.12 ^{bcde} ±0.85	6.036 ^{abc} ±1.35	1.04 ^{ab} ±0.10	7.84 ^{abcd} ±0.35	3.21 ^a ±0.89	7.90 ^{ab} ±0.60
C30R NMB	0.56 ^b ±0.35	2.02 ^{cde} ±0.50	5.29 ^{abc} ±1.08	0.98 ^{ab} ±0.16	8.63 ^a ±0.42	3.32 ^a ±0.13	6.99 ^b ±0.43
S30T NMB	0.78 ^{ab} ±0.08	2.29 ^{bcde} ±0.81	6.76 ^{abc} ±0.96	1.15 ^{ab} ±0.18	8.55 ^{ab} ±0.83	3.05 ^a ±0.11	6.77 ^b ±0.79
S60T NMB	0.61 ^{ab} ±0.18	2.98 ^{abcd} ±1.60	4.71 ^{abc} ±0.76	0.98 ^{ab} ±0.12	6.90 ^{cde} ±0.36	3.28 ^a ±0.43	4.69 ^c ±0.81
T30R NMB	0.64 ^{ab} ±0.05	1.91 ^{cde} ±0.37	7.87 ^{ab} ±3.15	1.13 ^{ab} ±0.16	7.79 ^{abcd} ±0.28	3.32 ^a ±0.25	8.41 ^a ±0.44
B40S MB	0.55 ^b ±0.21	1.66 ^{cde} ±0.72	5.91 ^{abc} ±0.53	1.20 ^{ab} ±0.46	7.73 ^{abcd} ±1.19	3.17 ^a ±0.27	6.71 ^b ±0.34
T20R MB	0.74 ^{ab} ±0.16	1.64 ^{cde} ±0.19	4.54 ^{bc} ±0.92	0.98 ^{ab} ±0.13	7.77 ^{abcd} ±1.05	3.32 ^a ±0.18	7.64 ^{ab} ±1.07
S20T MB	0.52 ^b ±0.09	1.48 ^{de} ±0.45	4.93 ^{abc} ±0.60	1.36 ^a ±0.53	8.60 ^a ±0.55	2.97 ^a ±0.16	7.39 ^{ab} ±0.43
C50R MB	0.90 ^a ±0.07	3.07 ^{abc} ±0.91	7.92 ^a ±2.71	1.03 ^{ab} ±0.12	8.12 ^{abc} ±1.02	3.10 ^a ±0.91	7.62 ^{ab} ±0.93
T10R MB	0.74 ^{ab} ±0.14	2.05 ^{cde} ±0.29	6.30 ^{abc} ±1.38	1.14 ^{ab} ±0.17	8.45 ^{ab} ±0.09	3.29 ^a ±0.41	7.32 ^{ab} ±0.84
Control NMB	0.61 ^{ab} ±0.04	4.31 ^a ±0.28	6.18 ^{abc} ±0.26	1.09 ^{bc} ±0.12	7.17 ^{bcd} ±0.37	3.40 ^a ±0.44	3.19 ^d ±0.31
Reference MB	0.60 ^{ab} ±0.02	3.56 ^{ab} ±0.74	7.11 ^{ab} ±2.93	3.99 ^a ±1.26	6.68 ^{de} ±0.30	3.33 ^a ±0.49	1.30 ^e ±0.08
Baseline NMB	0.60 ^{ab} ±0.01	1.27 ^c ±0.13	3.59 ^a ±0.59	0.61 ^c ±0.50	5.74 ^e ±0.37	1.63 ^b ±0.20	3.43 ^d ±0.37

^{abcde} Means along the serial column with different superscripts have significant difference ($p < 0.05$)

Table 4 shows an inverse correlation between average feed intake and dietary fiber ($r = -0.587$, $P \leq 0.01$) and positively correlates with fat content ($r = 0.583$, $P \leq 0.01$). There was significant difference ($p \leq 0.05$) within the baseline group when compared to other FFF groups in fecal output. There was no significant difference in the control and reference group, but when compared with the FFF groups a significant difference was observed. Fecal output of T30R-fed group [meat based group] was <1g per day throughout the study period. Fecal energy correlated inversely with moisture content of feeds ($r = -0.367$,

$P \leq 0.05$); directly with fat content ($r = 0.598$, $P \leq 0.01$) and with gross energy of feeds ($r = 0.759$, $P \leq 0.01$). Urinary energy showed a strong correlation with protein content of feeds ($r = 0.832$, $P \leq 0.01$); with moisture content of feeds ($r = 0.546$, $P \leq 0.01$) and inversely with carbohydrate ($r = -0.674$, $P \leq 0.01$) and gross energy ($r = -0.448$, $P \leq 0.01$). Metabolizable energy of feeds correlated with protein content of feeds ($r = 0.799$, $P \leq 0.01$) and with gross energy of feeds ($r = 0.642$, $P \leq 0.01$). Digestible energy correlated with protein content of feeds ($r = 0.776$, $P \leq 0.01$) and with gross energy of feed ($r = 0.693$, $P \leq 0.01$).

Table 4: Correlation of proximate composition, metabolizable and digestible energy of feeds

	Moisture	Dry matter	Fat	Ash	D fiber	Protein	Carbohydrate	G energy
Av Fd intake	0.291	-0.290	0.583**	-0.195	-0.587**	-0.212	-0.312	0.158
Fecal energy	-0.367*	0.369*	0.598**	0.013	-0.232	0.066	0.207	0.759**
Urine energy	0.548**	-0.547**	0.124	-0.225	-0.270	0.832**	-0.674**	-0.448**
G eng intake	-0.482**	0.483**	0.483**	0.164	-0.106	0.365*	0.384*	0.825**
M. energy	-0.526**	0.525**	0.085	0.375*	0.169	0.799**	0.584**	0.642**
D. energy	-0.512**	0.511**	0.096	0.375*	0.159	0.776**	0.564**	0.639**

** Correlation significant at $P \leq 0.01$. *Correlation significant at $P \leq 0.05$

Table 5 shows a correlation between dietary fiber content of feeds and caecum weight of rat models ($r = 0.607$, $P \leq 0.01$) and GIT weight ($r = 0.626$, $P \leq 0.01$). An inverse correlation was observed between dietary fiber and physical abdominal fat ($r = -0.764$, $P \leq 0.01$); dietary fiber and average feed intake ($r = 0.587$, $P \leq 0.01$). Protein consumption correlated with kidney weight ($r = 0.606$, $P \leq 0.01$). A correlation was observed in carbohydrate content of feeds and caecum weight ($r = 0.389$, $P \leq 0.05$) and inverse correlation existed between carbohydrate content and

physical abdominal fat ($r = -0.379$, $P \leq 0.05$). There was correlation between fat content and abdominal fat ($r = 0.712$, $P \leq 0.01$); GIT weight ($r = 0.541$, $P \leq 0.01$) and average feed intake ($r = 0.583$, $P \leq 0.01$); and an inverse correlation between fat and GIT length ($r = -0.706$, $P \leq 0.01$), caecum weight ($r = -0.714$, $P \leq 0.01$) and liver weight ($r = -0.407$, $P \leq 0.05$). A correlation existed between average feed intake and physical abdominal fat ($r = 0.863$, $P \leq 0.01$) and average feed intake and GIT weight ($r = 0.620$, $P \leq 0.01$).

Table 5: Correlation of proximate composition of feeds; average feed intake and weight of internal organs of the rat models

	moisture	D matter	fat	ash	D. fiber	Protein	CHO	G. energy	Av. feed
Heart wgt	0.263	-0.263	-0.036	-0.176	-0.213	-0.157	-0.153	-0.224	0.187
Caecum wgt	-0.284	0.284	-0.714**	0.399*	0.607**	-0.050	0.389*	-0.191	0.481**
Liver wgt	0.104	-0.105	0.407*	-0.182	-0.057	0.276	0.448**	-0.541**	-0.038
Whitfat wgt	0.385*	-0.385*	0.712**	0.488**	-0.764**	-0.290	-0.379*	0.099	0.863**
Kidney wgt	-0.111	0.112	-0.291	0.053	0.255	0.606**	0.034	-0.109	-0.366*
GIT wgt	0.346*	0.346*	0.541**	-0.520	0.626**	-0.198	-0.325	0.003	0.620**
Stomac wgt	-0.094	0.095	-0.095	0.065	0.110	0.047	0.091	0.057	0.271

** Correlation significant at $P \leq 0.01$ *Correlation significant at $P \leq 0.05$ CHO: Carbohydrates; GIT: Gastro intestinal tract

Discussion

The concentration of metabolizable and digestible energy were greater in non-meat based fed group $P < 0.05$ when compared to the meat based fed group, but when compared with the control group; the meat based fed group had higher values except sample B40S where there was no significant difference $P > 0.05$ with reference group. The rats fed FF containing high dietary fiber had the highest metabolizable energy which is similar to a study on human subjects where the group that consumed the highest dietary fiber content had a higher metabolizable energy when compared with the low dietary fiber group (Baer *et al.*, 1997) [7]. The metabolizable energy value of dietary fiber is a function of combustible energy content and digestibility (Livesey, 1990) [43]. Dietary fiber of feeds correlates positively with metabolizable energy at $P < 0.05$ in this study, because the combustible energy of dietary fiber is relatively consistent, changes in digestibility will affect the metabolizable energy value (Baer *et al.*, 1997) [7].

Protein content of samples correlated inversely with metabolizable energy (-0.799) and digestible energy (-0.776) at $P < 0.01$ however, there was a significant correlation between carbohydrate content of diet and metabolizable energy (0.584) and digestible energy (0.564) at $P < 0.01$ which is similar to work done by Wang *et al.* (2104) [73, 74]. There was an inverse correlation between protein intake and metabolizable energy. This tally with a study carried out on human (Baer *et al.*, 1997) [7].

Metabolizable energy and digestible energy correlate with gross energy intake at $P < 0.01$ in this study as the actual metabolizable energy value of mixed diets depends on the overall composition of the diet and, thus, it may be more difficult to predict the metabolizable energy content of mixed foods based on the amount of the individual macronutrients (Baer *et al.*, 1997 [7]; Garcia, 2003) [31]. Metabolizable energy correlates inversely with sodium, chlorine and potassium contents of diets at $P < 0.05$ and positive correlation with magnesium and zinc at $P < 0.05$. There was an inverse correlation between metabolizable energy and mono-sodium glutamate at $P < 0.01$.

The digestible energy (Kcal) for the FF was more compared to the control and reference groups ($P < 0.001$), this could be as a result of the fatty acids present in the fast foods since long chain unsaturated fatty acids are easily digestible than saturated fatty acids (Weiss and Wyatt, 2004 [75]; Harvartine and Allen, 2006) [33]. This indicates that the FF would provide more energy compared to the control or reference samples. Previous studies either measured the energy balance or the energy intake to output through bomb calorimetry (Behall and

Howe, 1996, Garcia, 2003) [31], or determined the energy value through specific calculations or general estimations (Mathers, 1992 [49]; Roberfroid, 1999; Garcia, 2003) [31]. The digestible energy in this study was calculated using the Livesey (1991) [44]. method. There was an inverse correlation between fecal energy and moisture content of feeds at $P < 0.01$. The sharp increase in fecal weight of group S20T (metabolic period 3), group T20R (metabolic period 3 and 5) and group S60T (metabolic period 3) might be due to the proliferation of beneficial bacteria in the colon (Hylla *et al.*, 1998) [35].

There was no significant difference in the non-meat based group in relation to average weight gain ($P > 0.05$) except in the C30R group where weight gain was highest at the end of the six metabolic periods. This agrees with the work done on resistance starch where there was no significant difference in weight gain in all groups (Garcia, 2003) [31]. Though when compared with the control and reference groups there appeared to be significant difference within groups. This could be attributed to the composition of the reference/control diet which has lower fat value and decreased overall energy intake (food consumption) in these groups in comparison to the fast foods sample groups. This corroborates the work carried out on rats fed resistance starch where energy consumption and weight gain was more in resistant starch group when compared with control (Garcia, 2003) [31]. There was no correlation between average/total weight gain and protein in this study.

There was significant correlation between protein intake and kidneys weight in rat models ($r = 0.606$; $P \leq 0.01$) in this study, this may be as a result of the protein being majorly from animal sources.

There was a significant inverse correlation between dietary fiber and saturated fatty acids ($r = -0.685$; $P < 0.01$), cholesterol ($r = -0.464$; $P < 0.01$) and trans fatty acids ($r = -0.420$; $P < 0.05$) in this study. Pereira *et al.* (2005) [58]. found association between increased intake of dietary fiber and lowering of blood lipids especially cholesterol, improving glycemic index and increasing hyper-insulinemia. Dietary fiber intake has been found to increase the process of digestion and bowel movement, while inadequate intake was reported to predispose consumers to cancer of the colon, constipation, irritable bowel syndrome, overweight and obesity, coronary heart disease and diabetes (Wu *et al.*, 2003) [78]. Dietary fiber has been shown to alter large bowel transit time in dogs (Lewis *et al.*, 1994), there was correlation between dietary fiber and fecal output at ($r = 0.378$; $P < 0.05$ in this study. The effects of dietary fiber on stool quality may be related to the length of cellulose fiber rather than the absolute amount of fiber (Wichert *et al.*, 2002) [77]. Similarly, in cats the addition

of dietary fiber in the form of long-fiber cellulose enhances stool quality (Prola *et al.*, 2010) [61].

The gross energy intake correlated significantly with fecal energy, metabolizable and digestible energy; protein efficiency ratio and fat at $P < 0.01$, and correlated inversely with urinary energy and protein value at $P < 0.05$ and $P < 0.01$ respectively which is similar to work done by Pederson *et al.* (2007) and Wang *et al.* (2014) [73, 74].

Increasing evidence associates the consumption of a diet high in carbohydrates, with high prevalence of metabolic syndrome (Gerritis and Tsalikian, 1993; Mendez *et al.*, 2013), calories from carbohydrates ranged from 20% to as high as 71.68% in this study. This seems to explain the relatively high weight gain seen in S60T-fed rats.

Fat content of the samples is inversely correlated with ash and dietary fiber content $P < 0.01$; and total carbohydrate at $P < 0.05$. This is similar to a research done on dietary fiber, metabolizable energy and nutrient digestibility of mixed diet fed humans (Baer *et al.*, 1997) [7]. where total carbohydrate content was inversely correlated with the amount of fat. Percentage contribution of dietary fat to this energy value ranged from 9.8% to 57.39%, this corroborates the work on non-alcoholic liver disease in rats fed westernized diets identified that typical westernized diet is rich in saturated fatty acids and cholesterol and provides about 35–40% energy from fat (Riserus, 2008 [62]; Tang *et al.*, 2014) [66]. This is particularly important for young children who have limited gastric capacity, provide essential fatty acids and their influence on the absorption of lipo-soluble nutrients (Jose *et al.*, 1989) [36].

Looking at each metabolic period according to weight gain there was significant difference in weight gain over the six metabolic periods within groups with weight gain increase between metabolic period 4 and 5 in samples C50R, T20R, T10R and S20T with ≥ 3 g. Average weight gain of rats correlated positively with white fat weight of rats (0.732 at $P \leq 0.01$). This is similar to Leibowitz *et al.* (2007) who measured the pre-pubertal weight gain in Sprague–Dawley rats on a high-fat diet at 30–35 days of age, the findings showed that weight gain is significantly, positively correlated with body fat accrual at maturity and more strongly related than the pre-pubertal measures of absolute body weight or daily energy intake. Average feed intake correlated strongly with white fat weight at ($r = 0.863$, $P \leq 0.01$), which is similar to previous research on winstar rats fed cafeteria diets; the cafeteria fed group became heavier in weight than the control group which was attributed to the consumption of high energy from fat and carbohydrate cafeteria diet (Pinto and Seraphim, 2012) [59].

There was an inverse correlation in average feed intake and dietary fiber of feeds which is attributable to an increase satiety in the diet of the FF group when compared with the control and reference group which are high in fiber value per 100g of edible portion and positive correlation between average feed intake and fecal energy at $P < 0.01$. Average weight gain correlated with ash and moisture content significant at $P < 0.05$.

Fecal excretion of rats is similar to human; fecal bulk when compared with the control and reference group in this study showed a significant difference in group T20R which had the

highest in all fast food samples. Such an increase in fecal bulk was observed with the consumption of resistant starch in rats (Garcia, 2003) [31], the greater the resistant starch composition of a diet, the greater the fecal bulk (Silvester *et al.*, 1995).

Organ weight can be the most sensitive indicator of the effect of an experimental compound (Michael *et al.*, 2007 [51]; Ajayi *et al.*, 2012) [3], as significant differences in organ weight between treated and untreated animals may occur in the absence of any morphological changes (Bailey *et al.*, 2004 [8]; Ajayi *et al.*, 2012) [3]. There were differences in organs weights across all groups when compared with the control and reference groups which could be attributed to variability in feed composition and intake (quantity), feed digestibility and utilization as observed by Ferrel and Koong, (1986) [25]. Observations from toxicological studies have reported complications often due to differences in body weights between groups (Ajayi *et al.*, 2012) [3]. Other parameters that are commonly used for analysis of organ weight are the ratio of the organ weight to body weight (to account for differences in body weight) and the ratio of the organ weight to the brain weight which are generally classified as relative ratios (Bailey *et al.*, 2004 [8]; Ajayi *et al.*, 2012) [3]. Base line information on organ-body weight data is very important in various spheres of veterinary medicine and biological science (Gangrade, 2009) [30], as any deviation from the normal weight is suggestive of the presence of some pathology (Tanna *et al.*, 2011) [67]. In this study, the average weight gain correlates positively with gastrointestinal tract (GIT) weight at $P \leq 0.01$ and inversely with GIT length, caecum weight and kidney weight at $P \leq 0.01$. This may reflect the functional capacity but not the functional efficacy of organs to support body size (Ozgun *et al.*, 2006) [56].

Liver weight of C50R-fed rats had a significant difference compared to other groups. Liver weight significantly correlates with GIT length ($r = 0.507$, $P \leq 0.01$), this reflects the roles of these organs in nutrients absorption and partitioning (Ozgun *et al.*, 2006) [56]. A correlation existed between liver and total carbohydrates and fat at $P \leq 0.05$ and $P \leq 0.01$ respectively and negatively with gross energy at $P \leq 0.01$ in this study; this can be attributed to liver being an organ that regulates the metabolism of glucose, proteins and fat. Storage of lipids occurs in hepatocytes as free fatty acids, triglycerides and cholesterol, whereby the liver is the main organ which synthesizes triglycerides and cholesterol (Donnelly *et al.*, 2005) [20].

Heart weight of C50R-fed rats had a significant difference from other groups; most of the fast food-fed rats had no significant difference in heart weight from the control, reference and baseline groups. These data were consistent with the overall trends and incidence and severity of cardiomyopathy seen in some comparable studies (Roe *et al.*, 1995 [64]; Kemi *et al.*, 2000 [39]; Keenan *et al.*, 2000 [39]; Faine *et al.*, 2002 [23]; Wan *et al.*, 2003; Keenan *et al.*, 2005) [37].

Kidneys weight was highest in the reference group, during the study there was high mortality rate within this group within 7th and 12th week of study which could maybe be associated with any form of renal disease, an important co-morbidity factor that contributed to the morbidity and the overall mortality in the ad libitum fed rats (Keenan *et al.*, 2005) [37]. these changes were gradual and progressive, and correlated with a steady

decline in renal function as measured by clinical biochemistry, creatinine clearance, urinalyses and urine protein electrophoresis, as reported by Keenan *et al.* (2000) ^[39]. The early events in the development of chronic nephropathy occurs as early as 14 study weeks, with measurable increases in kidney size, glomerular hypertrophy, hypertrophy of the entire nephron and a loss of renal function in response to the metabolic overload from increased food intake and rapid growth (Keenan *et al.*, 2000) ^[39]; and diet-induced hypercholesterolemia worsens kidney pathology produced by hypertension in Dahl-sensitive rats compared to rats fed a regular rat chow low in cholesterol (Tolins *et al.*, 1992 ^[69]; Danda *et al.*, 2005) ^[18].

Conclusion and Recommendations

This study revealed that the more protein and salts in the diet, the less the unspent energy derived from the diets; however, feeds with higher unspent energy tends to increase organ weights and thereby a risk factor to increased pathogenesis of diseases and mortality in rats. Based on the findings, it is recommended that feed should contain adequate nutrients so as to reduce unspent energy, risk factors, and spreading of diseases and even deaths in sampled rats.

References

1. Adeyeye EI. Waste Yield Proximate and Mineral Composition of Three Different Types of Land Snails Found in Nigeria. *International Journal of Food Science and Nutrition*. 1996; 47:111-116.
2. Adeyeye EI, Agesin OO. Dehulling the African Yam Bean (*Sphenostylis stenocarpa* Hochst) Seeds: Any Nutritional Importance? Note I. *Bangladesh Journal of Scientific and Industrial Research*. 2007; 42(2):163-174.
3. Ajayi IE, Shawulu JC, Nafarda WD. Organ Body Weight Relationship of Some Organs in the Male African Grasscutter. *Journal of Advanced Veterinary Research*. 2012; 2:286-90.
4. Ascherio A, Rimm EB, Herman MA, Giovannucci EL, Kawachi I, Stampfer MJ, et al. Intake of Potassium, Magnesium, and Fiber and Risk of Stroke among US men. *Circulation*. 1998; 98:1198-204.
5. Assis dos Passos Maria E, Moreira Carolina F, Pacheco Maria-Teresa B, Iracema T, Lopez, Maria-Lucia M, Valente-Mesquita, et al. Proximate and Mineral Composition of Industrialized Biscuits. *Food Science and Technology Campinas*. 2013; 33(2):2013.
6. Association of Official Agricultural Chemists (AOAC). *Official Methods of Analysis*, 20th ed., AOAC International, Gaithersburg, MD, 2010. <http://www.aoac.org>.
7. Baer DJ, Rimpler WV, Miles Carolyn W, Fahey GC. Dietary Fiber Decreases the Metabolizable Energy Content and Nutrient Digestibility of Mixed Diets Fed to Humans. *Journal of Nutrition*. 127(4):579-586.
8. Bailey SA, Zidell RH, Perry RW. Relationships Between Organ Weight and Body/Brain Weight in the Rat: What Is the Best Analytical End Point. *Journal of Toxicology and Pathology*. 2004; 32:448-466.
9. Behall KM, Howe JC. Resistant Starch as Energy. *Journal of American*, 1996
10. Bender A. Meat and Meat Products in Human Nutrition in Developing Countries, FAO Nutrition Paper 53, FAO: Rom, 1992, 1.
11. Bowman SA, Gortmaker SL, Ebbeling CB, Pereira MA, Ludwig DS. Effect of Fast Food Consumption on Energy Intake and Diet Quality among Children in a National Household Survey. *Journal of Paediatrics*. 2004; 113:111-120.
12. Bressani R, Sanchez M, Morales E. Chemical Composition of Grain Amaranth Cultivars and Effects on Processing on Nutritional Quality of Food. *Review Internationa*. 1992; 1:23-49.
13. Callabero B. Obesity in Developing Countries. Biological and Ecological Factors. *Journal of Nutrition*. 2001; 131:866-70.
14. Castrillo C, Herera M, Baucells MD. Methods for predicting the energy value of pet foods. *Revista Brasileira de Zootecnia*. No spe viciosa, 2009, 38.
15. Cheim Maria G, Oliveira EA, Arantes Vanessa C, Veloso RV, Reis Marise A, Gomes-da-Silva Maria H, et al. Effect of Nutritional Recovery with Soybean Flour Diet on Body Composition, Energy Balance and Serum Leptin Concentration in Adult Rats. *BioMed Central. Nutrition and Metabolism*. 2009; 6:34. doi:10.1186/1743-7075-6-34.
16. Cheim Maria G, Oliveira EA, Arantes Vanessa C, Veloso RV, Reis Marise A, Gomes-da-Silva Maria H, et al. Effect of Nutritional Recovery with Soybean Flour Diet on Body Composition, Energy Balance and Serum Leptin Concentration in Adult Rats. *BioMed Central. Nutrition and Metabolism*, 2009, 6:34. doi:10.1186/1743-7075-6-34.
17. College of Nutrition. 15(3):248-254.
18. Danda RS, Habib NM, Rincon-Choles H, Bhandari BK, Barnes JL, et al. Kidney Involvement in a Nongenetic Rat Model of Type 2 Diabetes. *Kidney International*. 2005; 68:2562-2571.
19. Dickinson A. Recommended Intakes of Vitamins and Essential Minerals. Council for Responsible Nutrition, 2002.
20. Donnelly KL, Smith CI, Schwarzenberg SJ, Jessurun J, Boldt MD, Parks EJ, et al. Sources of Fatty Acids Stored in Liver and Secreted via Lipoproteins in Patients with Nonalcoholic Fatty Liver Disease. *Journal of Clinical Investigation*. 2005; 115:1343-1351.
21. Drewnowski A. Nutrition Transition and Global Dietary Trends. *Journal of Nutrition*. 2000; 16:486-7.
22. Eunmi K, Jinho C. The evaluation of metabolizable energy in traditional Korean food for protein sources. *Journal of Ethnic foods*. 2015; 2(4):179-185.
23. Faine LA, Diniz YS, Almeida JA, Novelli ELB, Ribas BO. Toxicity of ad lib. Overfeeding: Effects on cardiac tissue. *Journal of Food Chemistry and Toxicology*. 2002; 40:663-8.
24. Fairweather-Tait SJ. The Importance of Trace Metal Specification in Nutrition Sciences. *Fresenius. Journal of Analytical Chemistry*. 1999; 363:536-40.
25. Ferrel CL, Koong KJ. Influence of Plane of Nutrition on Body Composition, Organ Size and Utilization of Sprague-Dawley rats. *Journal of Nutrition*. 1986; 116:2525-2535.

26. Flecks H. Introduction to Nutrition, 3rd edition Macmillan: New York, 1976, 1.
27. Food and Agriculture Organization (FAO). Roots, Tubers, Plantain and Bananas in Human Nutrition. Food and Agriculture Organization of the United Nations, Rome, Italy, 1998.
28. Food and Nutrition Board (FNB). Dietary, Functional, and Total Fiber. Retrieved August 6, from Dietary Reference Intakes for Energy, Carbohydrates, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids, 2000. <http://books.nap.edu/books/0309085375/html/265.html>.
29. Food and Nutrition Board, Institute of Medicine (FNB/IM). Dietary Reference Intakes: Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. National Academy Press, Washington, D.C, 2001.
30. Gangrade RP. Organ Weights and its Correlation with Body Weight- A Post Mortem Study. Journal of Forensic and Toxicology. 2009; 2:26.
31. Garcia Tanya A. The Metabolizable Energy Value of Physiologic Effects of HI MAIZE Resistant Starch in Male Rats. A Thesis in Human Ecology. Louisiana State University, U.S.A. (Dec.2003), 2003.
32. Gerrits PM, Tsalikian E. Diabetes and Fructose Metabolism. American Journal of Clinical Nutrition. 1993; 58:796S-799S.
33. Harvatine KJ, Allen MS. Effects of Fatty acid Supplements on Ruminant and Total Tract Nutrient Digestion in Lactating Dairy Cows. Journal of Dairy Science. 2006; 89:1092-1103.
34. He Q, Heo M, Heska S, Wang J, Pierson RN, Albu JZ, et al. Total Body Potassium differs by Sex and Race Across the Adult Age Span. American Journal of Clinical Nutrition. 2003; 78:72-77.
35. Hylla S, Gostner A, Dusel G, Anger H, Bartram HP, Christi SU, et al. Effects of Resistant Starch on the Colon in Healthy Volunteers: Possible Implications for Cancer Prevention. American Journal of Clinical Nutrition. 1998; 67:136-142.
36. Jose MB, Benjamin T, Moise BR, Nevin SS. Nutritional Goals for Health in Latin America. UNU Food and Nutrition Bulletin, 1989, 85.
37. Keenan KP, Hoe Chao-Min, Mixson Lori, McCoy Carol L, Coleman JB, Mattson Britta A, et al. Diabetes: A Polygenic Model of Dietary-Induced Obesity from Adlibitum Overfeeding of Sprague–Dawley Rats and Its Modulation by Moderate and Marked Dietary Restriction. Journal of Toxicology and Pathology. 2005; 33(6):650-674.
38. Keenan KP, Ballam GC, Dixit R, Soper KA, Laroque P, Mattson BA, et al. The Effects of Diet, Overfeeding and Moderate Dietary Restriction on Sprague–Dawley Rat Survival, Disease and Toxicology. Journal of Nutrition. 1997; 127:851S-6S.
39. Kemi M, Keenan KP, McCoy C, Hoe CM, Soper KA, Ballam GC, et al. The Relative Protective Effects of Moderate Dietary Restriction Versus Dietary Modification on Spontaneous Cardiomyopathy in Male Sprague–Dawley Rats. Journal of Toxicology and Pathology. 2000; 28:285-96.
40. Kleiber M. The fire of life: An Introduction to Animal Energetics. New York: R. E. Krieger, 1975.
41. Ledikwe JH, Blanck HM, Kettel KL, Serdula MK, Seymour JD, Tohill BC, et al. Dietary Energy Density is Associated with Energy Intake and Weight Status in United States Adults. American Journal of Clinical Nutrition. 2006; 83:1362.
42. Liebowitz KL, Chang GQ, Pamy PS, Hill JO, Gayles EC, et al. Weight Gain Model in Prepubertal Rats: Prediction and Phenotyping of Obesity-Prone Animals at Normal Body Weight. International Journal of Obesity, 2007, 1-12.
43. Livesey G. Energy Value of Unavailable Carbohydrate and Diets: an Inquiry and Analysis. American Journal of Clinical Nutrition. 1990; 51:617-637.
44. Livesey G. Calculating the Energy Values of Foods: Towards New Empirical Formulae Based on Diets with Varied Intakes of Unavailable Complex Carbohydrates. European Journal of Clinical Nutrition. 1991; 45:1-12.
45. Livesey G. Metabolizable Energy of Macronutrients. American Journal of Clinical Nutrition. 1995; 62:1135S-1142S.
46. MAFF. The Analysis of an Agriculture Materials. 2nd Edn., HMSO., London, 1981, 22.
47. Mariam S. Nutritive Value of Three Potential Complementary Food Based Cereals and Legumes. African Journal of Food and Agriculture. 2005; 5:1-15.
48. Martinez JA. Body Weight Regulation Causes of Obesity. Proceedings of Nutrition Society. 2000; 59:337-45.
49. Mathers JC. Energy Value of Resistant Starch. European Journal of Clinical Nutrition. 1992; 46:S129-130.
50. Mendez de Castro UG, Souza dos Santos RA, Silva ME, Geraldo de Lima W, Maria José CS, Alzamora A, et al. Age-dependent Effect of High-Fructose and High-Fat Diets on Lipid Metabolism and Lipid Accumulation in Liver and Kidney of Rats. Lipids in Health and Disease. 2013; 12:136. doi:10.1186/1476-511X-12-136.
51. Michael B, Yano B, Sellers RS, Perry R, Morton D, Roome N, et al. Evaluation of Organ Weights for Rodent and Non-rodent Toxicity Studies: A Review of Regulatory Guidelines and a Survey of Current Practices. Journal of Toxicologic Pathology. 2007; 35:742-750.
52. Miller DS, Judd PA. The Metabolizable Energy Value of Foods. Journal of Food Science and Agriculture. 1984; 35:111-116.
53. Moldawer M, Zimmerman SJ, Collins LC. Incidence of Osteoporosis in Elderly Whites and Elderly Negroes. Journal of American Medical Association. 1965; 194:859.
54. Otemuyiwa IO, Adewusi RSA. Nutrient Composition of Some Foods from a Nigerian Eatery. Journal of Food Chemistry and Nutrition. 2014; 2(1):11-18.
55. Ozelci A, Romas DR, Veleille GA. Influence of Diet Composition on Nitrogen Balance and Body Composition in Meal-eating and Nibbling Rats. Journal of Nutrition. 1997; 107:1768-1774.
56. Ozgur k, Armagan H, Ahmet Y, Zafer O, Filkrulla K, Emel B, et al. Reference Values for some Physiological and Biochemical Parameters in Rats Fed at Puberty.

- Journal of Animal and Veterinary Advances. 2006; 5(12):1121-1128.
57. Pedersen C, Boersma MG, Stein HH. Digestibility of Energy and Phosphorus in Ten samples of Distillers Dried Grains with Solubles Fed to Growing Pigs. *Journal of Animal Science*. 2007; 85:1168-1176.
58. Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR, et al. Fast food Increases the Risk of Obesity and Diabetes. *Lancet*. 2005; 365:36-42.
59. Pinto DAC Jnr, Seraphim PM. Cafeteria Diet Intake for Fourteen weeks can Cause Obesity and Insulin Resistance in Wistar Rats. *Review on Nutrition Campinas*. 2012; 25(3):313-319.
60. Prasad AS. Clinical, Biochemical and Nutritional Spectrum of Zinc Deficiency in Human Subject: An Update. *Nutrition Review*. 1983; 41:197-207.
61. Prola L, Dobenecker B, Mussa PP, Kienzle E. Influence of Cellulose Fibre Length on Faecal Quality, Mineral Excretion and Nutrient Digestibility in Cat. *Journal of Animal Physiology and Nutrition*. 2010; 94:362-367. doi:10.1111/j.1439-0396.2008.00916.x
62. Riserus U. Fatty Acids and Insulin Sensitivity. *Current Opinion in Clinical Nutrition and Metabolic Care*. 2008; 11:100-105.
63. Roberfroid MB. Caloric Value of Inulin and Oligofructose. *Journal of Nutrition*. 1999; 129:1436S-1437S.
64. Roe FJC, Lee PN, Conybeare G, Kelly D, Matter B, Prentice D, et al. The Biosure Study: Influence of Composition of Diet and Food Consumption on Longevity, Degenerative Disease and Neoplasia in Wistar Rats Studies for up to 30 Months Post Weaning. *Journal of Food Chemical and Toxicology*. 1995; 33(1):1S-100S.
65. Selvendran RR, Ring SG, Du Pont MS. *Journal of Chemistry and Industry*. 1979; 7:225.
66. Tang SY, Cheah IKM, Ng PE, Hoi A, Jenner AM. Heme Consumption Reduces Hepatic Triglyceride and Fatty Acid Accumulation in a Rat Model of NAFLD Fed Westernized Diet. *Hindawi Publishing Corporation ISRN Oxidative Medicine Article ID 659029*, 2014, 2014.
67. Tanna JA, Patel PN, Kalele SD. Relationship Between Organ Weights and Body Weight in Adult Population of Bhavnagar Region. A Post Mortem Study. *Journal of Indian Academy of Forensic Medicine*. 2011; 33(1):57-59.
68. Tee ES, Mizura SS, Anuar A, Kuladevan R, Young SE, Khor SC, et al. Nutrient Composition of Selected Cooked and Processed Snack Food. *Pertanika*. 1989; 12:15-25.
69. Tolins JP, Stone BG, Raij L. Interactions of Hypercholesterolemia and Hypertension in Initiation of Glomerular Injury. *Kidney International*. 1992; 41:1254-1261.
70. Uauy R, Olivares M, Gonzalez M. Essentiality of Copper in Humans. *American Journal of Clinical Nutrition*. 1998; 67:952S-959S.
71. Vaskonen MD. Dietary Minerals and Modifications of Cardiovascular Risk Factors. *Journal of Nutritional Biochemistry*. 2003; 14:492-506.
72. Wan R, Camandola S, Mattson MP. Intermittent Food Deprivation Improves Cardiovascular and Neuroendocrine Responses to Stress in Rats. *Journal of Nutrition*. 2003; 133:1921-9.
73. Wang TT, Liu DW, Huang CF, Liu L, Piao XS, Wang FL. Determination and Prediction of Digestible and Metabolizable Energy from the Chemical Composition of Chinese Corn Gluten Feed Fed to Finishing Pigs. *science*. 2014; 27(6):2014.
74. Wang TT, Liu DW, Huang CF, Liu L, Piao XS, Wang FL. Determination and Prediction of Digestible and Metabolizable Energy from the Chemical Composition of Chinese Corn Gluten Feed Fed to Finishing Pigs. *Asian Journal of Animal Science*. 2014; 27(6):2014.
75. Weiss WP, Wyatt DJ. Digestible Energy Values of Diets with Different Fat Supplements when Fed to Lactating Dairy Cows. *Journal of Dairy Science*. 2004; 87:1446-1454.
76. Whelton PK, He J, Cutler JA, Brancati FL, Appel LJ, Follmann D, et al. Effect of Oral Potassium on Blood Pressure, Meta-analysis of Randomized Control Clinical Trials. *Journal of American Medical Association*. 1997; 277:1624-32.
77. Wichert B, Schuster S, Hofmann M, Dobenecker B, Kienzle E. Influence of Different Cellulose Types on Feces Quality of Dogs. *Journal of Nutrition*. 2002; 132:1728S-1729S.
78. Wu H, Dywer KM, Fan Z, Anne S, Fan J, Dywer JH. Dietary Fibre and Progression of Arteriosclerosis: The Los Angeles Atherosclerosis Study. *American Journal of Clinical Nutrition*. 2003; 78:1085-1091.