CHAPTER ONE

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1.1. Introduction:

Sudan is one of the [illegible] l (*Camelus dromedarius*) populated countries in the world. The total camel population of Sudan is estimated as 3 million heads (Ministry of Animal Resources, Sudan, 1999). In Sudan Camels provide mankind with a range of products and services, like wool, meat, milk and draught power.

Camel is a unique animal with economic and logistic benefit in Arab peninsula. Besides use in transportation, it is of great economical value for meat, milk and hides supplementation. It belongs to Artiodactyla suborder, which comprises other ruminants like cattle and sheep. Although, the camel is a ruminating animal that chews the cud, it is being classified as pseudorumenant (Mior, 1965).

Despites of the noticed importance of the dromedary, it still needs further investigation to resolve its physiological peculiarity that meets its arid and hot habitat. Besides its osmotic fragility testing that reported to be the most resistant than other related ungulates (Peshin *et al.*, 2010) plasma lipoproteins play a vital nutritive and metabolic roles in dromedary. There are assembled as lipid conjugated proteins with five subclasses namely chylomicron, very low density lipoprotein (VLDL), intermediate density lipoprotein (IDL), low density lipoprotein (LDL) and high density lipoprotein (HDL). Their function is to transport lipids (fats) such as cholesterol around the body in the blood. LDL carries cholesterol from liver to cells of the body, which are sometimes referred to as (bad cholesterol) lipoprotein, while HDL collected cholesterol from the body tissue and bring it back to the liver. HDL is known as good

cholesterol lipoprotein. Studies in cholesterol, triglycerides and lipoprotein in domestic animals have made it clear that species variations exit and that even within species. The normal concentration of serum lipids and lipoproteins of sheep, cow, horse, pony, calf and camel in various physiological conditions have been reported (Nazifi *et al*., 2003; Kaneko *et al*., 2008).

Javad and Nazifi (2011) reported that water buffalo had higher serum cholesterol and triacylglycerol than the reference values of dromedary camels reported by Nazifi *et al*. (2009) in which he found a significant correlation between the serum HDL cholesterol and total lipid.

Nazifi *et al.* (2003) reported that age had significant effect on the serum concentration of cholesterol, triglyceride, total lipid, the HDL cholesterol, LDL cholesterol and VLDL. Bennis *et al*., (1992) reported that in turkman horses the concentration of all lipids was similar to mature goats. When (Hugi and Blum, 1997) found that In calves, the cholesterol concentration increase transiently with age, but triglyceride showed no consistent change.

Apolipoproteins are proteins that bind lipids (oil, soluble substances such as fat and cholesterol) to form lipoproteins, they transport the lipids through the lymphatic and circulatory system. There are many types of apolipoproteins (Apolipoprotein A, B, C and E). Apolipoprotein A1 (APoA1), the main protein included in HDL cholesterol transport (Reichl and Miller, 1989). But it has been published that a genetic variation (-276 base pairs G/A) in promoter region of this apolipoportein is associated with altered post-prandial lipid metabolism (Marin *et al*., 2002).

Apolipoproteins B100 and B48 are the only non-exchange apoproteins with large size water insoluble characters that assembled into

lipoproteins in intestines and liver. Apo CII is absolutely required for activation of lipoprotein lipase in the peripheral tissue, but mainly in muscles and adipose tissue. However, sufficient Apo CII remains to ensure that most of the triacylglycerol are removed. In contrast, Apo CIII the component of HDL inhibits lipoprotein lipase and binding of lipoproteins to receptors at cell surface, furthermore, plasma Apo CIII concentration are obesity associated with triglyceride concentration (Ooi *et al*., 2008).

1.2. Objectives of the study:

1-To study the effect of different feeding regimens in dromedary camel on different serum biochemical constituents.

2-To study the difference in distribution pattern of plasma proteins during different feeding regimens as monitored by gel permeation chromatography and SDS PAGE electrophoresis.

3-To study the transcriptomic pattern of apolipoprotein as a function of feeding regimens, to better understand the influence of the diet on the degree of expression of lipid carrier in such animal with great lipid adaptation.

4-To study the expression levels of different lipoproteins during different feeding regimens as performed by protein footprinting.

CHAPTER TWO

Literature Review

2.1.Camel in Sudan:

The one-humped camel (Camelus dromedarius) belongs to the order Artiodactyla, suborder Tylopoda. The family Camelidae

consists of the genus Llama, to which all the new world Camelidae belong, and the genus Camelus. The genus Camelus is represented by the two-humped bactrian camel (Camelus bactrianus) and the one-humped dromedary camel (Camelus dromedarius) (Wilson, 1984). The one-humped camel populates the semi arid, arid tropical and subtropical regions of Africa, Asia and other regions such as Australia (Djemali and Alhadrami, 1998) . From about 19.4 million camels worldwide, the dromedary camel accounts for 95% (FAO, 2003). The Sudan owns 3.1 millions of the dromedary camel population that comprises 11% of animal biomass. The main area of the Sudan inhabited by camels extends between latitudes 10º and 20º north and is bound by the Ethiopian mountains and the Red Sea hills on the East and by the Ingasana mountains and Bahr El Arab in the South (Babiker, 1984). Camel produce milk, meat, wool, hair and hides; serves for riding, and as a draft animal for short-distance transport (Schwartz and Dioli, 1992) .Camel milk is one of the most important components of the diet of nomads in the Sudan, it is consumed by the owners and herders (El Amin, 1984).

In Sudan, dromedary camels are maintained for meat production, as baggage carriers and for riding. Furthermore, camel hair is an important by-product for nomads, where it is frequently used for making robes, tents, saddle girths, blankets, clothes and carpets (El Amin, 1984) .Small numbers of heavy camels are exported to neighbouring African states, where the bulk of camel export-trade goes to Egypt. Most of these camels are drawn from western of Sudan (Babiker, 1984). Racing camels are also exported to Saudi Arabia and other Gulf States (Schwartz and Dioli, 1992). The practice of Nomadism by the herders showed a decrease along years the 1984, 1994 and

2003 with percentage of 73.3 %, 33 % and 22 %, respectively. Each year is independent of the other and not on an additive basis. The results reflected high illiteracy among herders groups of age (30–49 years) and (above 50 years) old was 67.7 %. Natural disasters as drought and man made halters as encroachment of mechanized agriculture on pasture, insecurity and tribal conflicts and wars were the main constrains of camel production. In conclusion, Nomadism is declining and giving a way for settlement and increase of production.

2.1.1.Camel types in Sudan:

Sudan is rated second in numbers of camel population in the world after Somalia with an estimation of 4078 thousand head (Ministry of Animal Resources, 2006), concentrated in two main regions; the Eastern states (Butana plain and Red Sea mountains) and Western regions (Darfour and Kordofan) (Agab, 1993). The main camel keeping tribes in Butana region are the Shukriya, Rashaida, Kawahla, Lahawiyin, Bija and Bawadra.

Camels are classified as heavy and light (riding) types according to the function which they perform (Zaid *et al*., 1991). The Sudanese pack camel is the heavy type which makes up the majority of the camels maintained by nomads.They are raised mainly in northern Darfur and northern Kordofan states. In eastern Sudan there is Rashaida camel which is slightly shorter than the Arab camel (Zaid *et al*., 1991). Also the Anafi and Bishari camel are the riding camel in the east of the country (Wardeh, 2004a).

2.1.2.Camel Raising:

Camel research in the Sudan has been focused mainly on functional anatomy, diseases and reproduction. However, research on husbandry and

management systems, feeding and nutrition and production performance are scanty (Majid, 2000, Majid, 2006). In the last few years some new modes of camel husbandry practices were developed for the improvement of the traditional systems of camel keeping methods. Camel population in Sudan is 4.5 million heads (FAO , 2009). The camel, a multipurpose animal, is an important component in the dry and semi dry eco-systems, where it makes optimal utilization of the meager vegetation and limited water resources better than any other domestic animal species. The survival of the pastoralists is dependent on camel especially during severe prolonged drought which is difficult to other animals to produce or live. Camels are owned by nomadic tribes who took complete responsibility and care of their animals (Darosa, 2000). The predominant management system of camels is by migratory pastoralists in subsistence production system (Schwartz and Dioli, 1992), however camels are kept for milk production and transport purposes (Buron and Saint-Martin 1988, Sooud *et al*., 1989, Abedl-Rahim *et al*., 1994). Both features are important for the mobile pastoral system (Kaufmann, 1998). Recent technologies in husbandry, breeding, nutrition, disease diagnosis and management had not been adopted by animal's owners. This is either because of their ignorance to these advances or the negligence of the authorities on the importance of camels. Recent changes in nomadic system such as expansion of mechanized agricultural schemes and their socio-economic impact on camel herds have not been quantified.

Darosa *et al*., (2011) reported that there were three production systems, the nomadic migratory 22 %, semi-nomadic 36 % and the sedentary production system 42 %. It also reflected a negative decrease (-10.97) in camel herd population under migratory system

and a positive increase of (5.2 %) in camel herd population in sedentary system. The mean herd size was 62 head for migratory and 118 for sedentary, with male to female percentage of 25.6 % and 74.4 %, respectively.

2.1.3.Feed intake:

With few exceptions, camels are associated with nomadic or semi-nomadic production systems. However, these systems are undergoing rapid adaptive change and transformations to cope with emerging demographic and economic factors (Hashi,1991). Many herders are becoming more and more attached to quasi-permanent settlements, The resulting short-range management system differs considerably from the traditional long-range mobility patterns which used to balance the feed budgets of the herds. These included, for example, the exploitation of the camel's water turnover capacity by reducing the frequency of watering during the dry season and the driving of the herds to remote pastures. Another development in pastoral communities, is increasing cropping in very dry lands and the emergence of agropastoralism as a major production system.

A related trend within formerly purely pastoral systems, is the increasing commercialization of milk, and various forms of less mobile camel dairying are expanding. In some cases, producer-traders may keep lactating animals (taken from the main mobile herd) near settlements where they can regularly market the milk. The milking herd has access to range enclosures or reserves around the settlements. At the extreme end of these trends, camels may be raised, on a permanent basis, in ranches or in agricultural areas (with access to fallow lands, stubble grazing and crop residues) and in and around urban centers where they are provided purchased

feedstuffs. Camel feeding management and strategies must take into account these incredibly complex production patterns based on different resources (in terms of feed and physical environment) and guided by different producer/production targets (increase milk production, prolonged lactation for subsistence, herd growth and stability, etc.). Guidelines for camel feeding have often been extrapolated from the feeding standards for cattle, assuming that the digestibility of 'foods by camels and their efficiency of utilization of nutrients for various functions do not differ significantly from those of true ruminants (Bhattacharya *et al*., 1988; Gihad *et al.*, 1989; King, 1983; Wilson,1989).

Camels feed intake depends primarily on its selective feeding of a wide variety of vegetation and different parts of forage browse which differ in quality. However, feed intake studies, often based on uniform standard diets, do not take into account that ability. As a matter of fact, the few feed intake values reported for the camel in its natural conditions, are superior to those obtained under stall-fed conditions. The DMI values for camels grazing natural pastures have been estimated to be 1.6 – 3.8 kg per 100 kg lw (Richard,1989).

In Sudan there is a current change in the mode of camel husbandry when it recorded a percentage of 22% of camel herders as transhumant nomads in contrast to 42% as sedentary camel herders. Abbas (1997) and (Jasra and Isani , 2000) reported a global change in the lifestyle of camel herders towards settlement and sedentary mode of camel production. This new shift in the mode of camel husbandry in Butana indicates that the classical lifestyle is now giving the way to new emerging production systems. This is accompanied with increased and encouraged rain-fed and irrigated crop production

activities which may result in pushing the remaining camel nomads southward to un-hospitable eco-climate for camels due to the soil nature and high prevalence of diseases such as trypanosomiasis which may lead to further deterioration in the situation of traditional camel pastoralism. The climate influences both distribution of animals and the chemical composition and nutritive value of pasture plants (Parker and Blowey, 1976). Moreover, it has been reported that forage quality influences feeding patterns of camels, where the time available for grazing under adverse pasture conditions would be a limiting factor to their dry matter (DM) and nutrient intake (Kassilly, 2002). Very low crude protein (CP) content of forage has been reported during dry season (Abdelrahman *et al*., 1998). The protein content of the diets selected by other animals was higher, and correspondingly the crude fibre content was lower during the green season compared to the dry season. However, camels were consistently able to select best qualities of minor differences between dry and green season (Schwartz and Dioli, 1992). The chemical composition of camel browse species in terms of crude protein, crude fibre, fat, nitrogen free extract and ash were reported to be 12.5%, 18.3%, 4.2%, 53.2% and 10.9%, respectively (Le Houérou, 1980). (Mohamed, 2007) reported that growing male Maghraby camels fed on the diet containing black cumin seed-cake performed better than those offered the control ration. Moreover, feeding camel on black cumin ration is more better economically, which lower feed cost/unit gain or as a net income than feeding control ration. Hashi and Kammoun (1995) found that young growing camels achieved an average daily gain(ADG) of 285 g when offered a diet providing approximately 8.5 MJ ME per kg DM and hence an energy intake level at the lower end of the daily ME allowance for maintenance and live weight gain of cattle. Both

results would suggest that camels have lower energy requirements and/or extract more from fibrous feeds. However, more field work is needed to determine the metabolism of camels' diets and the energy costs of feeding and production to develop feed budgets within defined production pattern. Mohamed *et al*. (2009) reported that (yeast culture)YC supplementation of diets of Maghraby camel calves at 5 g/kg (DM basis) improves weight gain, ADG and feed utilization, while supplementation with (zinc bacitracin) ZnB at 2 g/kg (DM basis) has no effect on the same traits.

2.1.4. Biochemical parameters and feeding:

The nutritive value of tropical grasses has been reported to be of low protein and mineral contents (Dougall *et al.,* 1964). However, El Shami *et al.* (1985) reported that the mineral content of the browse plant is adequate in terms of Ca, Mg, and K, while deficiencies in Ca, P and Na level in forage during dry season were reported by Abdelrahman *et al*. (1998). Aichouni *et al.* (2013) reported that plasma glucose, creatinine and serum urea concentrations were significantly higher during the wet season. Whereas the concentration of triglycerides and cholesterol of serum increased significantly during the dry season. Babeker *et al.* (2013) detected differences between dry and green season in serum content of total protein, also found that glucose levels were significantly higher during Dry wet winter. Amin *et al*. (2007) reported that the serum levels of total proteins, globulin and triglycerides increased significantly during the dry season, while the concentrations of the plasma glucose, creatinine and serum urea increased significantly during green season.

Concentrations of glucose was estimated in venous plasma. The rate of glucose elimination was markedly lower in camels than in sheep and ponies. (El mahadi *et al*, 1997). Ilamas and alpacas clear glucose more

slowly than other domestic species after challenge, mainly because of a weak insulin response and slow cellular uptake. This response may impair the assimilation of exogenous glucose as well as make llamas and alpacas prone to diabetes – like disorder when an abundance of endogenous or exogenous glucogenic agents are present.(Christopherk *et al*, 2001). AL-Sultan (2003) found that sex has no significant effect on serum total protein, glucose, creatinine, serum urea, cholesterol and triglycerides in camels.

Glucose tolerance test:

Concentrations of glucose and insulin were estimated in venous plasma. The rate of glucose elimination was markedly lower in camels than in sheep and ponies. The insulin response after glucose infusion was more pronounced in ponies and sheep than in camels. It is concluded that the markedly higher plasma concentration of glucose compared to sheep and ponies may be caused by a poorer insulin response and/or reduced tissue sensitivity to insulin (El mahadi *et al*, 1997).

It had been suggested that marked species differences in glucose tolerance tests were due to differences in insulin resistance. To determine insulin responsiveness, euglycemic hyperinsulinemic clamps were carried out in camels. Porcine insulin was infused as primed-continuous infusions for 2 h (6 mU x kg(-1) x min(-1)). The steady state glucose infusion rates in the camels were 6.1 micromol x kg(-1) x min(-1). The maximal plasma insulin concentrations during the insulin infusions were 2,700 microU x ml(-1) in the camels (Kaske *et al.* 2001).

2.2. Lipid metabolism in camels:

Studies on cholesterol, triglyceride and lipoproteins in domestic animals have made it clear that species variations exist, and that, even

within species, significant differences occur. The normal concentrations of serum lipids and lipoproteins of the cat, dog, cow, horse, goat, reindeer calf and cheetah, in various physiological conditions, have been reported (Kaneko, 1989; Chand and Georgie, 1989; Soveri *et al*., 1992; Barrie *et al*., 1993; Kataria *et al*., 1993; Noro *et al*., 1993; Zitnan *et al*., 1993; Duncan *et al*., 1994; Kraft *et al*., 1994; Hugi and Blum, 1997; Backues *et al*., 1997; Gueorguieva and Gueorguiev, 1997). However, there is little information about the serum lipids in dromedary camels, apart from reports on serum concentrations of cholesterol and triglyceride in normal camels by Wasfi *et al*., 1987 and by Al-Ani *et al*., 1992. Mohamed (2008) assessed lipid status in Sudanese camels (Camelus dromedarius) in relation to age, sex and breed. No differences were reported due to sex,but cholesterol, triglyceride and total lipid varies significantly as a function of age thus adult camel have higher lipid status compared to yearling and neonates. Also breed-related significant differences, Arabi being higher in lipid status than Anafi.

The concentrations of cholesterol, triglyceride, total lipid, HDL-cholesterol, LDL-cholesterol and VLDL-cholesterol in the different age groups were significantly different . Moreover, (Nazifi *et al*., 2000) found that age has a significant effect on the serum lipids and lipoproteins of the camels as, with an increase in age of animals, there is an increase in the cholesterol , triglyceride ,HDL-cholesterol and VLDL-cholesterol concentrations, whereas, the concentration of LDL-cholesterol decreased. In 5- to 6-year-old camels, there were significant correlations between cholesterol and both HDL-cholesterol and LDL-cholesterol.Khajeh *et al.* (2008) found in camels, that there was a negative correlation between triglyceride and HDL. There were positive correlation between

cholesterol and LDL, VLDL. The serum cholesterol concentration decreased with age, whereas, the percentage of LDL and VLDL increased

Age had a significant effect on the serum concentration of cholesterol, triglyceride, total lipid, HDL cholesterol, LDL cholesterol and VLDL cholesterol of Turkoman horses, with the values being higher in older animals. Whereas, sex had no significant effect on the concentrations of cholesterol, triglyceride, total lipid, HDL cholesterol, LDL cholesterol and VLDL cholesterol (Nazifi *et al*., 2003). In Iranian goats The concentrations of triglyceride, and VLDL-cholesterol, in the different age groups were significantly different. Age had a significant effect on the serum lipids and lipoproteins of the goats, as with an increase in age of animals there was a decrease in the triglyceride ,VLDL-cholesterol concentrations. So age had a significant effect on the serum triglyceride and VLDL-cholesterol of the male goats and the values were lower in older animals (Nazifi *et al*., 2002). In water buffaloes the average age of the female was significantly more than that of the male. There were no significant differences between male and female in the serum concentration of cholesterol, triglycerides, total lipids, LDL-cholesterol and VLDL- cholesterol. The differences in the serum concentration of the HDL- cholesterol between the sexes was marginally significant (Javad and Nazifi, 2011). Sivakanesan and Mariathasan (1996) reported that in buffalo calves, up to seven months of age, the blood concentration of total cholesterol and HDL-cholesterol gradually increased until 22 weeks of age and then declined, but the blood triglyceride had no significant differences between different ages. Bennis *et al.* (1992) reported that in kids, the concentration of all lipids was similar to that in mature goats. Hugi and Blum (1997) reported that, in calves, the concentration of cholesterol increased transiently with age,

but triglycerides did not show a consistent change. Nazifi *et al*. (2000) reported that in dromedary camels the concentrations of cholesterol, triglyceride, total lipid, HDL cholesterol and VLDL cholesterol increased and the concentration of LDL cholesterol decreased with increasing age. In humans, Braunwald (1995) and Kleinveld (1996) reported that there was a statistically significant increase in the concentrations of serum cholesterol and triglyceride in advanced age. Noguchi (1993) reported that the concentrations of LDL and VLDL increased and the concentration of HDL decreased with increasing age. Khoshvaghti *et al.* (2012) reported that gray necked ostriches had higher serum cholesterol but a lesser serum triglyceride. And there were no significant differences between the male and female ostriches . The serum cholesterol had significant correlations with the HDL- and VLDL-cholesterol, and there was a significant correlation between HDL- and LDL-cholesterol. There were significant correlations between the HDL-, LDL-, VLDL-cholesterol and total lipids. Nazifi *et al*. (2009) found the same correlations and results in camels, but the correlation between the serum VLDL-cholesterol and total lipids was severe.

The concentration of lipoproteins and lipids in the sera of several species of healthy adult laboratory animals (guinea-pig, rabbit and rat), domestic animals (cattle, sheep, goat, horse and swine) and wild animals (deer, wild boar, mink and fox) have been investigated under physiological conditions. Special attention was paid to the lipoproteins which are precipitated with heparin-MnCl2 and to the ratio of lipids in separated alpha and beta lipoproteins. Most of the studied animals had significantly lower concentrations of total lipoproteins, beta lipoproteins and cholesterol than those in man. Only some wild animals (mink and fox) had concentrations of total lipoproteins, beta lipoproteins,

phospholipids and cholesterol which were significantly higher than man's. The ratio of lipids in the separated lipoprotein fractions also differed between the examined animals. Thus, in the sheep, rabbit, guinea-pig and wild boar most of the cholesterol (70-76%) was in beta lipoproteins. However, in the horse (60%), mink (60%), fox (65%), goat (70%) and cattle (73%) the high density lipoprotein fraction was the main carrier of cholesterol.With the exception of the guinea-pig (42%) most of the serum phospholipids were found in the high density lipoprotein fraction. (Vitić and Stevanovic, 1993).

As isolated by Oschry and Eisenberg (1982) Plasma lipoproteins from male rats by rate zonal centrifugation, four lipoproteins were identified: VLDL, LDL, HDL_I, and HDL_2. LDL, HLD_I, and some HDL_2 distributed within the salt density interval of 1.006-1.085 g/ ml, while HDL_2 was found in the 1.063-1.21 g/ml interval. HDL_3 was not identified in the rat. Rat VLDL is poor in cholesteryl esters (1.5-3.0% of total mass) and nearly lacks the smaller and denser particle subpopulation which is predominant in humans. Rat LDL, containing a relatively large amount of triglyceride (20.2% of total mass) and a small amount of cholesteryl ester (27.5%), could be isolated free of apoproteins other than apoB. HDL_l is a cholesteryl ester-rich lipoprotein that occupies a density interval overlapping both LDL and HDL_2. ApoE is the major protein constituent of HDL_l; apoA- I, A-IV, and C are also present. ApoA-I-rich HDL_2 is the only human-like HDL subpopulation found in rats. Lipoproteins from fasted and non-fasted rats were essentially similar. Arachidonic acid contributed 56.7% and 72.3% of total cholesteryl ester fatty acids in HDL_1 and HDL_2, respectively, but only 7.9% and 27.3% in VLDL and LDL, respectively. Palmitic, palmitoleic, and oleic acids were the major cholesteryl ester

fatty acids in VLDL and LDL. *In vitro* incubation of biosynthetically labeled HDL_2 cholesteryl ester with rat plasma demonstrated minimal transfer of the labeled cholesteryl ester to VLDL and LDL. These results indicate biological immis- cibility of HDL cholesteryl esters with those of lower density 1ipoproteins. The finding of cholesteryl ester-poor VLDL and LDL and the presence of HDL as larger and less dense subpopulations is compatible with the absence of cholesteryl ester transfer activity in an animal with pronounced LCAT(lecithin:cholesterol acyltransferase.) activity.

The high density lipoproteins (HDL) are a family of protein–lipid complexes that play a central role in reverse cholesterol transport and are a risk factor for atherosclerosis (Barter and Rye,1996). Human HDLs collectively contain apolipoproteins (apo) A-I and A-II as their major protein components, together with apoC, E, and A-IV (Alaupovic, 1984). In animals such as rats, in which they are the predominant lipoproteins, the HDL contain more apoE and A-IV than do human HDL. Changes in HDL composition occur during normal metabolism, as the result of genetic manipulations, and as a consequence of disease (Barter and Rye,1996 and Alaupovic, 1984). The protein composition of HDL can be analyzed by chromatography, electrophoresis, or immunoassay. Sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS PAGE) resolves proteins according to molecular size, while charge separation can be achieved by zone electrophoresis or isoelectric focusing. Quantification of protein bands after electrophoresis is achieved by immunoblotting or by staining with Coomassie Blue. However, due to differences in the chromogenicity of different apolipoproteins, the staining methods are only semi-quantitative (Tall *et al.,* 1985 and Kane *et al.,* 1975). Immunoassays offer good sensitivity and precision, but results can vary

with different antisera. Furthermore, immunoassays do not distinguish between different apolipoproteins isoforms.

Asadi *et al*.(2008) used the method of density-gradient ultracentrifugation to determine the lipid composition and electrophoretic pattern of plasma lipoproteins in camels. In which, Plasma samples were subjected to density-gradient ultracentrifugation for separation of plasma lipoproteins, including very-low-density lipoproteins (VLDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL). Purity of the separation was assessed using polyacrylamide gel disk electrophoresis. Concentrations of triglycerides, cholesterol, and phospholipids were measured in each lipoprotein fraction, and lipoprotein electrophoretic patterns were determined in plasma samples. The author found that phospholipid was the major constituent of VLDL concentration, LDL and HDL . Low-density lipoprotein, VLDL, and HDL were important plasma lipoprotein carriers for cholesterol , triglyceride and phospholipid respectively. The lipoprotein profile in one-humped camels differed substantially from that of other ruminants.

2.3.Apolipoproteins:

Apolipoproteins play a central role in the assembly, secretion, processing, and catabolism of lipoproteins. Because of their importance in atherosclerosis research, the structure and biosynthesis of these proteins have been studied by many laboratories. At the molecular level, the structure and organization of many apolipoprotein gene in humans and research animals have been elucidated. Comparative analysis of sequence data suggests that the soluble apolipoproteins are all members of a multigene family (Li *et al.* 1985). Sequence data have also been used to infer the structure-function relationships and the evolution of the multigene family (Boguski *et al.* 1985).

Members of the family have been shown to consist of arrays of internal repeats, and their evolutionary relationships have been inferred (Fitch *et al.*, 1986; Li *et al.*, 1985). Since apolipoproteins evolve rapidly, and since members of the gene family are well differentiated so that problems due to concerted evolution are minimized, they are particularly suitable for mammalian phylogenetic analysis. Biochemical analyses have revealed variation in the tissue distribution of apoA-I among species (Cheung and Chan 1983; Miller *et al.*, 1983; Pan *et al.*, 1987). Consequently, differences both in functional constraints and in evolutionary rates of apoA-I among different lineages may occur. To date, apoA-I has been sequenced in many vertebrate species, including four mammalian orders.

ApoB- 100 sequences appear to have evolved more rapidly than apoA-I. Because apoB- 100 is very large (4,536 amino acid residues), many residues may have evolved partly by repeated duplication of short sequences (Loof *et al.*, 1987). The higher evolutionary rate may reflect looser structural constraints of much of the protein. A gradient of functional constraint is suggested by the steady decline, from 3´ to 5´ along the sequence, in substitution rates. Thus, the 3´ -most 1,089 nt of the human-rat comparison evolve four times faster than the 5´-most 1,155 nt. Such a gradient may reflect the importance of global-rather than local-protein structure. The functionally important regions of apoB- 100 include two putative LDL receptor-binding domains at residues 3147-3157 and 3359-3367. These occur in moderately conserved regions suggestive of some flexibility in receptor-binding requirements. This interpretation is supported by the ability of another apoprotein, apoE, to bind to the same receptor. Despite its large size, apoB- 100

evolves at only twice the average rate for mammalian genes, far short of the quadrupled rate found in h-interferon (Li *et al*. 1985a).

ApoC-III is synthesized mainly by the liver (Wu and Windumeller,1978) and participates in the formation of the triglyceride-rich lipoproteins (chylomicrons and VLDL). It has been reported that apoC-III may inhibit lipoprotein lipase, an enzyme responsible for the clearance of triglyceride-rich lipoproteins from the circulation (Brown and Baginsky, 1972) and. Chung and Scanu, 1977). In addition, it has been shown that lipoprotein uptake by hepatic tissues is stimulated by apoE and inhibited by apoC-III (Quarfordt *et al.*,1982).

Apolipoprotein (apo) CII is a polypeptide composed of a single chain of 79 amino acids. It is present in plasma at a concentration of 2.7 ± 0.6 mg/dl (mean ± SEM) (Alaupovic *et al*., 1982) and resides in both the very low density lipoprotein (VLDL) and high density lipoprotein (HDL) fractions (Smith *et al*. , 1978). Purified apo CII has been shown to have cofactor activity for the enzyme lipoprotein lipase (triacylglycerol acylhydrolase, EC 3.1.1.3) (Havel *et al*., 1970 and LaRosa *et al*., 1970), which catalyzes the hydrolysis of triglycerides in chylomicrons and very low density lipoproteins (Nilsson *et al*., 1980).

Bauchart *et al*. (1989) evaluated The role of the liver in lipoprotein homeostasis in the preruminant calf. To this end, the hydrodynamic and physicochemical properties, density distribution, apolipoprotein content, and flow rates of the various lipoprotein particle species were determined in the hepatic afferent (portal vein and hepatic artery) and efferent (hepatic vein) vessels in fasting, 3-week-old male preruminant calves.

It has been suggested by (Steinberg, 1979) that exogenous cholesterol influences the lipoprotein profile by regulating the proportion

of VLDL, IDL and LDL secreted by the liver. The synthesis of these lipoproteins is limited by the amount of apoprotein B available, and in the presence of large amounts of exogenous cholesterol, there is an increase in the synthesis of VLDL and a decrease in the synthesis of LDL. LDL can also arise from VLDL that is released from the liver. The VLDL contains apoprotein B, apoprotein C and apoprotein E (arginine-rich protein). The apoprotein C is involved in the reaction with lipoprotein lipase in extrahepatic tissues in the formation of IDL, also called remnant particles. During this process, 90% of the apoprotein C is transferred to HDL, 50% of the apoprotein E is lost but there is no loss of apoprotein B. There are probably two pathways for the metabolism of the remnants. Probably the most important pathway occurs in the liver, where, the remaining apoprotein C and the apoprotein E are removed. It may well be that apoprotein E in HDL necessary for the removal of the apoprotein E from the remnant particle (Glomset, 1979). The other possible pathway for the conversion of remnants to LDL is via the pathway by which LDL is degraded in peripheral tissues (Goldstein and Brown, 1974). In the cholesterol-induced hypercholesteremia in the chickens, there was an increase in cholesterol content in the VLDL and IDL particles. Furthermore, the size of the IDL particles was increased. This would suggest that the rate of conversion of IDL to LDL is not sufficient to metabolize the exogenous cholesterol thus preventing the hypercholesteremia. Woollett *et al*. (1997) investigated the mechanism whereby diet modification alters the plasma concentration of high density lipoprotein (HDL) cholesteryl ester and apoA-I and to determine whether diet-induced alterations in circulating HDL levels are associated with changes in the rate of reverse cholesterol transport. Rates of HDL cholesteryl ester and apoA-I transport were measured in hamsters fed a control low-cholesterol, low-fat diet or the same diet supplemented with

soluble fiber (psyllium) or with cholesterol and triglyceride (western-type diet). The Westem-type diet increased the plasma concentration of HDL cholesteryl ester by 46% compared to the control diet and by 86% compared to the psyllium-supplemented diet; nevertheless, the absolute rates of HDL cholesteryl ester transport to the liver were identical in the three groups. Diet-induced alterations in circulating HDL cholesteryl ester levels were due to changes in the rate of HDL cholesteryl ester entry into HDL (whole body HDL cholesteryl ester transport) and not to regulation of HDL cholesteryl ester clearance mechanisms. The Western type diet increased the plasma concentration of HDL apoA-I by 25% compared to the control diet and by 45% relative to the psyllium-supplemented diet. Diet-induced alterations in plasma HDL apoA-I concentrations were also due entirely to changes in the rate of apoA-I entry into HDL (whole body HDL apoA-I transport). These studies demonstrate that the absolute flux of HDL cholesteryl ester to the liver, which reflects the rate of reverse cholesterol transport, remains constant under conditions in which plasma HDL cholesteryl ester concentrations are altered over a nearly 2-fold range by diet modification. Jiao, *et al*. (1990) assessed genetic variation of murine lipoprotein profiles, plasma lipoproteins of 11 inbred strains, were analyzed by gel-permeation chromatography (fast peptide liquid chromatography) and nondenaturing gradient gel electrophoresis. as a result, HDL sizes were positively correlated with HDL-cholesterol concentrations and LDL-cholesterol concentrations, but LDL sizes did not correlate with lipoprotein concentrations.

(Kupke and Worz-Zeugner, 1986) described a method for sequential separation of high density, very low density, and low density lipoproteins (HDL, VLDL, and LDL, respectively) from 100 µl of

serum, using an air-driven ultracentrifuge (Airfuge, Beckman). Cesium chloride was used for density adjustment. In order to test the results of this combined procedure, a group of healthy young men and women were examined. The results suggested that the procedure is highly suitable for lipoprotein analyses and thus can be applied to studies on children, including mature and premature neonates, as well as on small animals.

Lipids are transported through the circulation as lipoprotein complexes consisting of a hydrophobic core of cholesterol ester and triglyceride surrounded by phospholipids, free cholesterol and various apolipoproteins. Several classes of lipoproteins have been distinguished on the basis of density. Among these are chylomicrons and very low density lipoproteins (VLDL), which are secreted by intestine and liver, respectively, low density lipoproteins (LDL), which are derived in the circulation by lipolysis of VLDL, and high density lipoproteins (HDL), which are derived from precursor particles secreted by liver (Alaupovic *et al.,* 1982 and Smith *et al.,* 1978). Apolipoprotein B (apoB) is the major protein component of chylomicrons, VLDL and LDL, and it serves at least two crucial functions in lipoprotein metabolism. First, it is essential in the assembly and secretion of both VLDL and chylomicrons, since individuals with a rare genetic defect in the production of apoB, termed a betalipoproteinemia, are unable to produce these lipoproteins .(Havel *et al.,* 1970). Second, it serves as a recognition marker for the removal of LDL from the circulation by receptor-mediated uptake into a variety of cells (LaRosa *et al.,* 1970). It differs from other apolipoproteins in that it is an integral component of lipoproteins; while other apolipoproteins can exchange between lipoprotein particles and are soluble in the absence of detergents, apoB is extremely hydrophobic and remains tightly associated

with lipoprotein particles. Also, whereas, other apolipoproteins range in size from about 6 kDa-45 kDa, apoB is among the largest peptides known with molecular weight estimates ranging from 250-550 kDa (Havel *et al*., 1970, Nilsson *et al*., 1980- Elovson *et al.*, 1985). The hydrophobicity and size of apoB have made its study difficult. Furthermore, the protein exhibits size heterogeneity, with at least two major forms being present in the circulation. In humans, the liver synthesizes and incorporates into VLDL a species which we estimate has a molecular weight of about 400 kDa, while intestine incorporates into chylomicrons a smaller 210 kDa species. These 400 and 210 kDa species have been referred to as B100 and B48, respectively, and are synonymous with the PI and PIII species in Elovson' s nomenclature (Havel *et al.,* 1970, Kane *et al*., 1980, Elovson *et al.,* 1981). The B100 but not the B48 species contains the recognition marker for uptake by the LDL receptor which has been extensively studies by (Havel *et al*., 1970). The mechanisms leading to the production of the two species are unclear. Although most forms of abetalipoproteinemia are characterized by failure in the production of both apoBl00 and apoB48, others are characterized by continued expression of apoB48 but not apoBl00, raising the possibility that the two species are encoded by separate genes (Havel *et al*.,1970). On the other hand, peptide finger printing and immunological studies are consistent with their being derived from a common gene (Elovson 1981).

The small intestine absorbs fat very efficiently, and the ultrastructural pathway of absorbed lipid through intestinal epithelial cells has been well described (Jersild, 1966 and Sabesin and Frase, 1977). Interest is focused on the regulation of fat absorption, and it is clear that the various apoproteins associated with lipid particles during their absorption play an important role. One of these proteins,

apolipoprotein B (apoB), following lipid absorption from the intestinal lumen into the absorptive cell, apoB is essential for the formation and transport of lipid particles out of the absorptive cell into the lacteals (Schwartz *et al.*, 1978).

Many studies in recent years have explored the structure and function of the apoproteins involved in lipid metabolism. It is now well established that the intestine, as well as the liver, is a major site of synthesis of many of these apoproteins in rats (Windmueller *et al.*, 1973 and Wu and Windmueller., 1978) and in man (Rachmilewitz and Fainaru, 1979). Some quantitative work has been done; for example, Wu and Windmueller (1979) determined the relative contributions of liver and intestine to different plasma apoproteins in the rat. Their data for apoB showed that in fat-fed animals, the intestine contributes approximately 16% of the circulating apoB. In a later study, these investigators (Windmueller and Wu, 1981) found that when dietary or biliary fat was withheld, the intestinal contribution to circulating apoB was reduced to about 5%, supporting the idea that this apoprotein plays an active role in the synthesis of transportable lipid particles.

Apolipoprotein (apo) B normally exists in two molecular variants (Kane *et al.*, l980), apoB-100, which is synthesized in the liver and secreted into plasma as the major protein component of very low density lipoproteins (VLDL), and apoB-48, which is secreted from the intestine in chylomicrons after absorption of dietary lipids. ApoB is a marker of lipoprotein particle number, as there is only one apoB molecule per lipoprotein particle and, in contrast to other lipoprotein-bound apolipoproteins and lipids, apoB does not exchange between different lipoprotein particles (Chan, 1992).

human plasma, apolipoprotein B is heterogenous and exists in two major forms, apolipoprotein B-48 and B-100 (Kane, 1983). Apolipoprotein B-48 is synthesized exclusively by the intestine, and secreted on triglyceride-rich chylomicrons that undergo hydrolysis by lipoprotein lipase resulting in the formation of chylomicron remnants that are ultimately removed by the liver through an apolipoprotein E-mediated receptor process (Kane, 1983 and Brewer *et al*.,1983). Apolipoprotein B-100 (apo B-100) is synthesized by the liver (Kane, 1983 and Edge *et al*., 1983) and is secreted on triglyceride-rich very low density lipoprotein (Jackson *et al*., 1976, Kane *et al*.,1983 and Brewer *et al.*,1983). Liver very low density lipoproteins also undergo hydrolysis by lipoprotein lipase and are converted to intermediate density lipoproteins and, finally, to LDL (Kane *et al*.,1983 and Brewer *et al.*,1983). ApoB-100, the major apolipoprotein on LDL, is the protein determinant that interacts with the high-affinity LDL receptor and initiates the process of receptor mediated endocytosis that culminates in LDL catabolism (Goldstein and Brown, 1977). The structure and physicochemical properties of apo B-10 have been extensively studied for nearly a decade. The analysis of apo B-100 has been difficult because delipidated apo B-100 insoluble in aqueous solution, and generally aggregates in buffers containing NaDodSO4, urea, and guanidine hydrochloride (Jackson *et al*.,1976 , Kane *et al.*,1983). Structural studies of apo B-100 have met with limited success due to the insolubility of the native protein and its peptide fragments. The molecular size of apo B-100 remains controversial, and values ranging from 8 to 400 kDa have been reported (Kane *et al*.,1983 and . Elovson *et al.*, 1985). The heterogeneity in apo B-100 molecular size has been attributed to the propensity of delipidated apo B-10 to aggregate and to the reported sensitivity of apo B-100 t protease cleavage (Osborne and Brewer,1977 and Cardin *et al*.,1984). The structure of delipidated apo B-100 and

LDL-apo B-100 has been explored with monoclonal apo B-100 antibodies. Monoclonal antibodies have been characterized that will block the interaction of LDL with the LDL receptor (Marcel *et al.*,1982, Tikkanen *et al.*, 1982), and studies with a Fab fragment have been interpreted as indicating that there is a single apo B-100 per LDL particle (Marcel *et al.*,1982). Analysis of the epitopes present on apo B-100 and apolipoprotein B-48 has suggested that apo B-100 contains structural domain which is not present in apolipoprotein B-48 receptor (Marcel *et al.*, 1982).

Apolipoprotein B (apoB) plays a central role in cholesterol metabolism. It is an obligatory constituent of chylomicrons, very low density lipoproteins (VLDL), and low density lipoproteins (LDL) (Kane *et al.*,1983). Circulating apoB exists in two forms which are immunologically related. ApoB-100 has a molecular weight of about 400,000 and is made in the liver, whereas, apoB-48 has a molecular weight of about 200,000 and is made in the intestine (Elovson *et al.*, 1985 and Kane *et al.*, 1983). The liver secretes apoB-100 into circulation as a structural protein of triacylglycerol-rich VLDL, which is converted to cholesterol-rich LDL by the action of lipoprotein lipase (Havel *et al.*, 1980). Despite the abundance of apoB in plasma, its structure and biosynthesis have not been well-characterized. This in large part is due to its size, its insolubility when delipidated, its tendency to aggregate, and its susceptibility to oxidation. Some progress has recently been made, however, with the identification and sequencing of partial cDNA clones for human and rat apoB (Mehrabian *et al.*, 1985 and Lusis *et al.*,1985). However, the question of whether the production rate of apoB contributes to elevated LDL cholesterol levels remains unclear, although evidence from a number of studies suggests that alterations in the regulation of

apoB synthesis may contribute to hypercholesterolemia and atherosclerosis (Chait, 1980 and Kissebah *et al*., 1981).

Karpe and Hamsten, (1994) described a procedure for determining apolipoproteins (apo) B-100 and B-48 in subfractions of triglyceride-rich lipoproteins by analytical sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) with Coomassie staining. The chromogenicity of the two apoB species was found to be almost equal, and independent of lipoprotein particle size. Both proteins were sensitive to over- loading of the gel, which resulted in low dye uptake. This was particularly evident for apoB-48. The precision of this analytical SDS-PAGE-based procedure to determine the plasma concentrations of apoB-100 and B-48 in triglyceride-rich lipoproteins was found to be appreciably low (coefficients of variation ranging between 3.1 and 14.1%).

Various modes of chromatography are available for lipoprotein separation. Gel permeation and affinity chromatography are used for preparative purposes and to separate lipoproteins according to size and apolipoprotein content, respectively. Development of rigid supports for gel permeation has led to large improvements in speed and resolution. Reversed-phase high-performance liquid chromatography (HPLC) of apolipoproteins offers the best performance in terms of speed and resolution of structural variants. Due to its high speed and superior resolving power, the recently developed technique of capillary electrophoresis should emerge as an important method for lipoprotein analysis. (Tadey and Purdy, 1995)

Gabelli *et al.* (1986) developed an agarose-acrylamide gel electrophoretic method to separate the two major apoB forms. The gel is a mixture of 0.5% agarose and 2% acrylamide. The author found that

the agarose-acrylamide method is fast, has the advantage of being able to be used on an analytical or preparative scale in a vertical slab gel apparatus, and the gel is of sufficient strength to be used for immunoblotting or radioautography.

ApoB is the essential structural protein part of apoB-containing lipoproteins (Young, 1990 and, Chan, 1992). In humans, two different forms of apoB are present in plasma. These are classified according to the centile system as apoB-100 and apoB-48. These apoB proteins are essential for the assembly of VLDL in the liver and chylomicrons in the small intestine, respectively. Both apoB-100 and apoB-48 are products of the apoB gene. ApoB-100 results from the translation of the full length apoB mRNA, whereas, apoB-48 is the translation product of apoB mRNA that has been edited at cDNA position 6666 (C to U) which introduces a stop codon and terminates protein translation at 48% of the full length protein (Hodges and Scott,1992). Detailed structure-function studies of apoB are hampered by the large size of this protein (550,000 Da) and its highly lipophilic nature. Mutant forms of apoB are beginning to provide us with valuable information about structure-function relationships of the apoB protein. In recent years several truncated forms of apoB have been identified in humans (Linton *et al.*,1993). Most of these are due to mutations at the gene level that result in the introduction of premature termination codons. The resultant proteins are truncated at the C-terminal end. Affected individuals heterozygous for truncated apoB species are hypobeta-lipoproteinemic, with total and LDL-cholesterol concentrations of < 5th percentile and very low total apoB con- centrations.

The size of the truncated apoB protein influences the size and density of the particle that is formed. The physiologic mechanisms

responsible for the hypobetalipoproteinemia are not well understood, but decreased rates of production of apoB, increased fractional catabolic rates of VLDL-, IDL-, and LDL-apoB or various combinations of physiologic defects may be involved (Parhofer *et al.,* 1992 and Krul *et al.,*1992).

2.4. Gene expression of apolipoprotrein:

Apolipoprotein B is a large, amphipathic protein that plays a central role in lipoprotein metabolism. Because its over production and deficiency leads to metabolic and pathologic disorders, much effort has been paid to investigate the mechanisms of how its homeostasis is achieved. Earlier and recent studies have showed that apoB gene locus might reside in different chromatin domains in the hepatic and intestinal cells, and two sets of very distinct regulatory elements operate to control its transcription. Post transcriptional modification of apoB mRNA is performed by a multicomponent enzyme complex, several possible pathways regulate the editing efficiency. Understanding of the mechanism responsible for apoB mRNA editing will provide the basis for C-to-U editing in gene therapy. In addition to apoB mRNA abundance and stability, its translation can be also regulated at the steps of elongation. The translocation of apoB into the ER is an important and complicated process that is less understood. Successful transport and correct folding of apoB may lead to its final secretion, otherwise subject to intracellular degradation, which is accomplished by proteasomal and nonproteasomal pathways at multiple levels and may differ among cell types (Ai-Bing Wang,2003). Davidson *et al*, (1988) provided evidence for tissue-specific, independent regulation of apolipoprotein gene expression *in vivo*. Furthermore, the data suggest that aspects of hepatic triglyceride

assembly and secretion and apolipoprotein gene expression may be coordinately responsive to alterations in thyroid hormone status.

Greeve *et al.* (1993) measured apoB mRNA in liver and intestine from 12 different mammalian species by a quantitative primer extension analysis of reverse-transcribed and polymerase chain reaction (PCR) amplified apoB mRNA in order to determine whether i) editing of apoB mRNA is generally restricted to the intestine or may also be found in the liver of other species than rodents, and ii) hepatic expression of apoB mRNA editing influences lipoprotein concentrations in plasma. Intestinal apoB mRNA was edited at high levels in all species, 40% in sheep, 73% in horse, 82% in pig, 84% in dog, 84% in cat, 87% in guinea pig, 88% in rat, 89% in mouse, and >90% in human, monkey, cow, and rabbit. In liver apoB mRNA was edited to 18% in dog, to 43% in horse, to 62% in rat, and to 70% in mouse. Low levels of editing below 1% were detected in liver of rabbit and guinea pig. In contrast, hepatic apoB mRNA from human, monkey, pig, cow, sheep, and cat liver was not edited. The results of the primer extension analysis were confirmed by cloning and sequencing of the PCR products from dog, horse, cat, guinea pig, sheep, and cow for all of which the apoB cDNA sequence had not been established by previous investigations. Primer extension analysis of apoB mRNA from dog intestine and dog liver indicated C/U editing at c.5655 in addition to c6666. Cloning and sequencing of apoB cDNA from dog liver and intestine confirmed additional C/U editing at c6655 which changes ACA for threonine at amino acid residue 2149 into AUA for isoleucine. Synthesis and secretion of apoB-48-containing lipoproteins from liver was demonstrated by pulse labeling of freshly isolated horse hepatocytes

and immunoprecipitation with apoB-specific antibodies or density gradient ultracentrifugation.

The concentrations of VLDL, LDL, and HDL in all species were determined after fractionation by density gradient ultracentrifugation. The ratio (VLDL + LDL)/HDL was calculated for humans (1.92), pig (1.40), cow(1.04), monkey (0.91), sheep (0.65), cat (0.47), horse (0.44), rat (0.41), rabbit (0.32), dog (0.26), and mouse (0.25). Therefore, in four (dog, horse, rat, and mouse) out of six species (dog, horse, rat, mouse, cat, rabbit) with low ratios of below 0.5, apoB mRNA was edited in liver. In contrast, none of the species with high amounts of apoB-containing plasma lipoproteins expressed apoB mRNA editing in liver. I Taken together, these results indicate that i) editing of apoB mRNA is not intestine-specific but is also found in liver of many mammalian species, and ii) editing of apoB mRNA in liver appears to be one important genetic determinant for plasma concentrations of apoB-containing lipoproteins.

Andrzej et al. (1991) suggested that dietary fiber can modify apo B expression in the intestine. The increased fecal bile salt excretion might be involved in such a modification. Apolipoprotein B gene expression in rat intestine is effect by dietary fiber.

CHAPTER THREE

Materials and Methods

3.1. Materials:

3.1.1. Animals:

Forty five males clinically healthy camels (*Camelus dromedarius*) purchased from a local livestock market, two types Butana and Darfurian at the age of (18-24 month) and average body weight of (225.5±35 kg) were divided into two groups as zero browsing and free

browsing group. This study was conducted in The Camel Research Centre Tumbool, Sudan. It was carried out during the period July 2011-October 2011.

3.1.2. Experimental designs:

Twenty five male camels (*camelus dromedaries*) as zero browsing. 15 Darfurian and 10 Butana types were involved in this study. All animals were clinically healthy. After arrival at the site of the experiment all animals were sprayed against ecto-parasites, drenched with Albendazol, and injected with a complete prophylactic dose of ivomec and Oxytetracycline HCL. The first two weeks served as adaptation period Followed by a 120 days as experiment period. Animals were individually weighted weekly. The camels were injected with multivitamins monthly. Blood serum, tissues and DNA samples were collected from zero browsing and naturally browsing.

3.1.3. Experimental diets

In this study the camels supplemented with complete ration (table 1) formulated according to National Research Council, (NRC, 1994) to meet the camel's requirements. The former diet was composed of traditional ingredients of crushed sorghum grains and sugar cane molasses as energy sources, groundnut cake as source of protein and urea as nonprotein nitrogen. Wheat bran was added to the diets to adjust their total metabolize energy (ME) and crude protein contents. In addition, Dura husk (semema) was added to the concentrate diet as roughage. The salt licking stones, and water were offered *ad libitum* for four months (120 days).

Table (1) Ingredients percentage of the experimental diets (as fed basis):

Ingredients	%

Molasses	10
Crushed sorghum grains	50
Groundnut cake(GNC)	15
Dura husk	5
Wheat bran	5
Urea	2
Baggas	12
Common salt	1
Total	100

Table (2) The chemical composition and energy concentration of the experimental diet (as % of DM):

Particular	experimental diet%
Dry matter	96.5
Crude fiber	9.22
Crude protein	16.4
ME	11.32Mj/kg DM
	2466Kcal/kg DM
Ca	0.77
P	0.14

* Metabolizable energy was calculated according to MAFF (1975) formulae:

ME(MJ/kg DM) = 0.012CP+ 0.03EE+ 0.005CF+ 0.014NFE.

Where, CP is crude protein, g/kg DM; EE is ether extract, g/kg DM; CF is crude fiber, g/kg DM; and NFE is nitrogen free extract, g/kg DM.

3.1.4. Samples collection:

3.1.4.1. Blood samples:

The blood samples were taken from jugular vein by venipuncture, 10 milliliter blood samples were collected from each camel using

anticoagulant coated vacutainers as well as empty sterile tube to obtain plasma and serum, respectively. The plasma and serum samples were recovered from blood by centrifugation (3000 rpm at bench centrifuge for 10 minutes) and stored at -20°C till analysis.

3.1.4.2. Tissue samples:

The tissue samples were taken after sacrificing the animals, liver and muscle samples were collected in small pieces (2x2 cm cubes) and rapidly freezed in liquid nitrogen. Samples were then stored at -80°C till analysis.

3.2. Methods:

3.2.1. Blood lipid profile:

3.2.1.1. Cholesterol determination:

The total cholesterol was determined by (cholesterol oxidase, phenol & aminoantipyrine) CHOD-PAP- enzymatic colorimetric method using commercial kit, according to the method of (Ellefson *et al*., 1976).

Principle:

The reaction involved cholesterol esters were enzymatically hydrolyzed by cholesterol esterase to cholesterol and free fatty acids. Free cholesterol, is then oxidized by cholesterol oxidase (CHOD) to cholest-4-en-3-one and hydrogen peroxide, the last one combines with phenol and aminoantipyrine in the presence of peroxidase to form a chromophore (quinoneimine dye).

Procedure:

Ten microliters from standard and serum were pipetted into different centrifuge tubes. 1.0ml of reagent was added to each tube; sample, standard and blank. Then mixed and incubated for 10 min at

25ºC, and then the absorbance of specimen and standard measured against reagent blank within 30 min at 500nm.

Calculation

$$serum\ cholesterol\ concentration\ (mg/dl) = \frac{specimen}{standard}\ X\ 200$$

3.2.1.2. Triglyceride determination:

Triglycerides were determined by GPO- PAP enzymatic colorimetric method, using commercial kit according to (Bucolo *et al*., 1973).

Principle:

Triglycerides were hydrolyzed by LPL (lipoprotein lipase) to glycerol. Glycerol then phosphorylated to glycerol-3-phosphate by ATP in reaction that catalyzed by glycerol kinase. Glycerol-3-phosphate oxidized by glycerol phosphate oxidase (GPO) to dihydroxyacetone phosphate and hydrogen peroxide (H_2O_2). In the presence of proxidase the formed H_2O_2 causes the oxidative coupling of 4-chlorophenol &4-aminoantipyrine substrate to form a red color quinoneimine dye product that was measured at 456nm (Spectrophotometer).

Procedure:

Ten microliters from standard and sample were pipetted into different centrifuge tubes. 1.0ml of reagent was added to each tube; sample, standard and blank. Then mixed and incubated for 10 min at room temperature. Absorbance of specimen and standard were measured against blank within 30 min.

Calculation:

$$Serum\ Triglyceride(mg/dl) = \frac{Specimen}{Standard} X\ 200$$

3.2.1.3. High density lipoprotein cholesterol determination:

HDL cholesterol was measured by precipitation method using commercial kit (Friedwald *et al*., 1972), in which LDL and VLDL in sample precipitated with phosphotungstate and magnesium ions. After centrifugation, the cholesterol concentration in HDL fraction which remains in the supernatant was determined.

Procedure:

In a microcentrifuge tube, 0.5ml of reagent and 0.2ml specimen were mixed and incubated for 10 mins at room temp., then centrifuged for 10min at 4000rpm. The supernatant was collected, and then 50 µl of the sample was pipetted into a centrifuge tube. Then 1ml of cholesterol reagent was added into each tube. Absorbance was read against distilled water at 546nm within 60min.

Calculation:

$$HDL\ cholesterol(mg/dl) = sample\ X\ 570$$

3.2.1.4. Low density lipoprotein cholesterol determination:

LDL cholesterol concentration calculated according to (Friedwalds *et al*.,1972) formula:

$$LDL\ cholesterol(mg/dl) = total\ cholesterol - \frac{triglyceride}{5} - HDL\ cholesterol$$

3.2.1.5. Very low density lipoprotein cholesterol determination:

VLDL cholesterol concentration was estimated as one-fifth of the concentration of triglycerides (Friedewald *et al*., 1972)

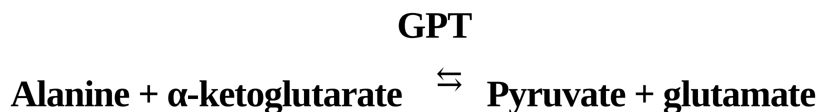
3.2.2. Liver function tests:

3.2.2.1. Determination of Serum alanine aminotransferase activity (ALT):

It was performed according to the method of (Reitman and Frankel, 1957).

Principle:

Colorimetric determination of GPT (ALT) activity was determined according to the following reaction:



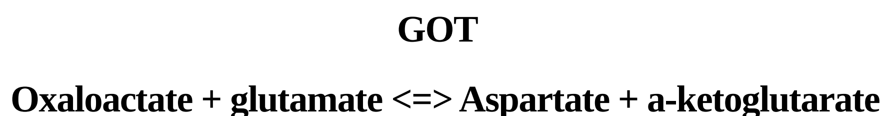
The keto acid pyruvate formed was spectrophotometrically measured in its derivative form 2, 4-dinitrophenylhydrazone at 505 nm.

3.2.2.2. Determination of Serum aspartate aminotransferase activity (AST):

It was performed according to the method of Reitman and Frankel (1957).

Principle:

Colorimetric determination of GOT (AST) activity according to the following reaction:



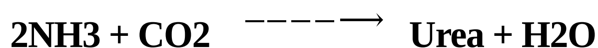
The keto acid oxaloacetate formed was spectrophotometrically measured in its derivative form 2, 4- dinitrophenylhydrazone at 505 nm.

3.2.2.3. Determination of Serum Urea concentration:

It was performed according to the method of Patton and Crouch (1977).

Principle:

Enzymatic determination of urea was done according to the following reaction:



$$2NH_3 + CO_2 \longrightarrow Urea + H_2O$$

In an alkaline medium the ammonium ions react with hypochlorite and salicylate to give a green colored iodophenol.

The intensity of color is directly proportional to concentration of urea and was measured against a reagent blank at a wave length of 580 nm.

3.2.3 Kidney function tests:

3.2.3.1. Determination of serum sodium and potassium concentration:

Sodium and potassium were estimated by flame photometery as described by (Gowenlock, 1988) using corning 410 (USA) flame photometer.

Principle:

Flame photometery is the measerment of the concentration of an ionic material in a solution introduced into a flame, where the intensity of light emitted by the flame under these conditions was measured. The diluted biological fluid was atomized directly into the flame, the resulting emissions were those of all inorganic ions present. For measurement, the emitted light was passed through a monochromator where the wave length most characteristic of the material under analysis and which most closely follows Beers low was selected.

Time of dilution:

Sodium : 1:200

Potassium : 1: 20

3.2.3.2. Determination of Serum Creatinine Concentration:

It was kinetically performed according to the method of Henry (1974).

Principle:

Creatinine in alkaline solution reacts with picric acid to form a colored complex. The colored complex formed was spectrophotometrically measured at 495 nm.

3.2.3.3. Determination of Serum Uric acid concentration:

It was measured using colormetric method, by commercial kit (Koch, 1937).

serum uric acid was determined using the following formula :

$$Uric\,acid\,(mg/dl) = \frac{Sample}{standard}\,X8$$

3.2.4. Blood protein profile:

3.2.4.1. Total protein:

Total protein concentration was measured by a colorimetric method (Biuret reagent) Cannon *et al*., (1974). In alkaline medium, the copper reacts with peptide bonds of proteins to form the characteristic pink to purple Biuret complex. Sodium potassium tartrate prevents copper hydroxide precipitation, and potassium iodide prevents the auto reduction of copper.

Procedure:

Twenty µl of standard or samples were added to 1.0ml of the reagent using empty blank tube with only reagent. Tubes were then incubated for 10 mins at room temp and absorbance was measured at 495 nm against blank within 30min.

Calculation

$$serum\,concentration\,(mg/dl) = \frac{specimen}{standard}\,X6$$

3.2.4.2. Determination of Serum Albumin concentration:

Serum albumin was performed according to the method of Bartholomew and Delany (1966).

Principle:

When albumin binds with bromocresol green (BCG) in a suitable buffer, pH 4.15 - 4.25, an intense blue colored albumin-BCG complex is formed:

Albumin + BCG Albumin-BCG complex

(yellow) (blue)

The intensity of the blue color is proportional to the amount of albumin in the sample, was measured at 630 nm.

Procedure:

1. There were test tubes Labeled as blank, standard and sample.
2. Then 5 ml of serum sample were pipetted into the sample tube
3. Then 0.02 ml saline were added to the blank tube
4. five ml bromocresol green (BCG) reagent was added to all tubes
5. All tubes were mixed by complete inversion
6. Then they were let to stand for 10 minutes at room temperature.
7. The ABS of each reaction tube was read at 630 nm.

Calculations:

$$Sample\,Conc.(mg/dl) = \frac{|Sample|}{|STD|}\,XCONC\,STD$$

3.2.4.3. Determination of Serum Globulin concentration:

The difference between serum total proteins and calculated albumin values for each sample was used to estimate globulin concentration (Kenneth *et al.,* 1990).

3.2.5. Determination of plasma glucose concentration:

It was performed according to the method of Trinder (1969).

Principle:

Enzymatic colorimetric determination of glucose according to the following reactions:

Glucose oxidase

Glucose + O_2 ------------------------------► Gluconic acid + H_2O_2

Peroxidase

$2H_2O_2$ + Phenol +4-aminoantipyrine► Red quinine + 4 H_2O

The red quinine was spectrophotometrically measured at 546 nm.

3.2.6. Determination of glucose tolerance test:

Glucose tolerance test was done according to the method described by (Elmahdi *et al.,* 1997), by infusing 1mmol glucose per kg body weight intravenously within 3min, blood samples were collected immediate before, and at 0, 5, 15, 25, 35, 45, 60, 90,120,150 and 180 mins. Determination of glucose level was determinated according to the enzymatic colorimetric method described by (Trinder, 1969).

3.2.8. Major Proteins in serum:

3.2.8.1. Gel Permeation of serum proteins using sephadex G200:

Serum protein were fractional separated by Sephadex G-200 (Pharmacia Aldrich chemical Co. Ltd) gel filtration.

Column Properties and preparation:

The column length was (75 cm) with 1cm^2 internal diameter and antisephadex glass wool pluged at the lower end. The dried sephadex material (5 gm dried weight) was equilibrated over night in phosphate buffer saline (PBS) (Bioshop, PBS Tablet in 100ml ddtt, ph 7.4, NaCl 137mM, phosphate buffer 10 Mm KCl 2.7Mm) . The column was filled with sephadex slurry by wet method 10 ml below the top. The volume of the column was determined with dextran blue. Serum samples (50 µl) were then added to the top of the column and fractions (1ml) were collected by Gilson fraction collector (FC 203B). The protein concentration was measured by tracing optical density at 280 nm of each fraction using Gilson UV/visible light detector (115 UV Detector). The data acquisition was then blotted using Graphpad Prism Software.

3.2.9. Serum lipoproteins fractions:

3.2.9.1. LDL-cholesterol fractionation:

LDL-cholesterol precipitated by LDL kit. The precipitate was collected and solved using PBS buffer(phosphate buffer saline) (Bioshop, PBS Tablet in 100ml ddtt, ph 7.4, NaCl 137mM, phosphate buffer 10 mM KCl 2.7mM).(20 µl)were then used in polyacrylamide gel electrophoresis.

3.2.9.2. HDL-cholesterol fractionation:

HDL-cholesterol was prepared by using HDL-cholesterol kit. Then the supernatant was collected (30 µl) and then was loaded in polyacrylamide gel electrophoresis.

3.2.9.3. Liver homogenate:

The liver was homogenated using PBS buffer, then centrifuged at 300 rpm, the supernatant was collected and (20µl) was loaded in polyacrylamide gel electrophoresis.

3.2.10. Determination of the number and molecular weight of protein subunits using SDS-polyacrylamide gel electrophoresis (SDS-PAGE):

The various fractions of camel lipoproteins were obtained by using polyacrylamide gel electrophoresis as described by (Lammli, 1970).

Principle:

To define the molecular weight of subunits, gel electrophoresis were used in the presence of SDS.

The quaternary, tertiary, and secondary structures of protein were all broken down. The chain was unfolded and surrounded by SDS molecules to form a micelle.

The polypeptide chain is therefore transformed into an elongated micelle, the length and charges of which was each proportional to the length (and hence molecular weight) of the chain, such particles were migrated in gel electrophoresis with relative mobilities depending only on chain lengths.

Chemicals and solutions used for SDS-PAGE:

Low molecular weight Marker:

The low molecular weight marker was obtained from BIO-RAD.

Preparation of 30% polyacrylamide:

Acrylamide	28.1 gm
N, N-Methylene-bis acrylamide	0.8 gm
Deionized bidistilled H_2O	up to100 ml

Preparation of separating buffer (pH8.6):

Tris	18.2 gm
SDS 10%	5 ml
Distilled water	up to 100 ml

Preparation of Stacking buffer:

Tris	12.1 gm
SDS 10%	5 ml
Distilled water	up to100 ml

Preparation of Ammonium Persulfate:

Ammonium Persulfate (APS)	400 mg
Distilled water	1 ml

Preparation of sample buffer:

20 mM tris Hcl(pH 6.8)	0.121 gm
40 % glycerol	20 gm
2 %SDS	1gm
2% Mercaptoethanol	1 gm
6 mg bromophenol blue	6 gm
Distilled water	50 ml

Preparation of 10% separating gel:

Bidistilled water	6 ml
30% Acrylamide mix	6 ml
Separating buffer	4.5 ml
Glycerol	1.5 ml
Amm. Persulfate	25 µl
N,N,N, N'-tetramethyl ethylene diamine	15µl

Preparation of 4% stacking gel :

Bidistilled water	3.8 ml
30% Acrylamide mix	0.7 ml
Stacking buffer	1.5 ml
Amm. Persulfate	10 µl
TEMED	10 µl

stock electrophoresis buffer 10 X:

Tris	15 gm
Glycine	72 gm
SDS	5 gm
Distilled water	500 ml

Destain Preparation:

Methanol	125 ml
Glacial Acetic acid	35 ml
Distilled water	up to 500 ml

Coomassie Briliant Blue R- 250

Methyl alcohol	300 ml
Acetic acid	100ml
Distilled water	900 ml

Procedure:

Preparation of the separating gel:

- The vertical slab gel unit was assembled in the casting mode by using the 1.5 mm spacers.
- The prepared solution of the separating gel was pipetted into the sandwiches to the level about 4 cm from the top.
- The gel was kept for about 30-45 minutes

Preparation of the stacking gel:

- About 1-2 ml of the stacking gel solution was added to each sandwich.
- The combs were inserted into each sandwich.
- The gel was allowed to set for at least half an hour.

Preparation of the sample and the marker:

- Twenty µl of the sample was diluted by 10 µl of sample buffer
- After dilution of the sample with the sample buffer, it was placed in a sonicator for 5 minutes.

Loading and running the gel:

- The combs were removed and each well was washed with distilled water.
- The samples and the marker (7µl) were inoculated in the wells.
- The chambers were then filled with running buffer.
- The power supply was then switched on (120 Volt).
- When the dye reached the bottom the power supply was turned off.

Staining and destaining of the gels:

- The gel was disassembled then put in the stain and shaked gently for 20 minutes on the shaker.
- The gel was then washed gently with deionized water
- The gel was removed and put on destaining solution for 30-45 minutes.
- The gel was then destaind again for at least 4 times till the destaining solution become clear from the stain.
- The gel was then dried between two cellophane membranes.

3.2.11.1 RNA Isolation

Principle :

The isolation of RNA was made by using QIAamp kit . (Sambrook *et al.*, 1989). The kit combines the selective binding properties of a silica-gel-based membrane with the speed of microspin or vacuum technology. The sample is first lysed under highly denaturing conditions to inactivate RNases and ensure isolation of intact tissue RNA. Buffering conditions provide optimum binding of the RNA to the QIAamp membrane, and the sample loaded onto the QIAamp spin column. The RNA bound to the membrane and contaminants washed away into two steps using two different wash buffers. The RNA eluted in a special RNase-free buffer until use.

Procedure:

- Five hundred and sixty µl of Viral Lysis Buffer (AVL) containing RNA carrier was pipetted into 1.5ml tube.
- Then 40 µl 0f the sample was added to the tube. Then vortexed for 15 seconds and incubated at room temperature for 10min, centrifuged at 6000xg for 1 min.

- After that 560 µl of ethanol was added, vortexed for 15 sec, and centrifuged at 6000xg for 1 min.
- Six hundred and thirty µl of the sample was taken from the 1.5 tube and pipetted into QIAamp spin column, centrifuged at 6000xg for 1min.
- Then 500 µl of AW1 (Wash Buffer 1)was added, centrifuged at 6000xg for 1min.
- After that 500 µl of AW2 (Wash Buffer 2)was added. centrifuged at 20000xg(QIAamp column was putted in 1.5 ml tube)
- Sixty µl of AVE buffer (Elution buffer) was added and incubated at room temperature for 1min. centrifuged at6000xg for 1min.
- RNA was stored at -20°c.

3.2.11.2. Two Step RT-PCR:

<u>Principle:</u>

Two step RT-PCR Kits were used to form full –length cDNA from isolated RNA in PCR. M-MuLV RNase H synthesizes complementary DNA strand initiating from camelus dromedarius partial mRNA for apolipoprotein B (apoB gene), editing region (Accession # AJ399508 ,175 bp mRNA linear):

Primer design:

APO-f 5'- TAGACAATGCCAAAATCAAGC -3'

APO-r 5'- AATCTTAGCAATAGCTGCCT -3'

The specifications for the primers we used

Primer pair 3

	Sequence (5'->3')	Template strand	Length	Start	Stop	Tm	GC%	Self complementarity	Self 3' complementarity
Forward primer	TAGACAATGCCAAAATCAAGC	Plus	21	18	38	55.10	38.10	3.00	2.00
Reverse primer	AATCTTAGCAATAGCTGCCT	Minus	20	142	123	54.48	40.00	6.00	1.00
Product length									125

The sequencing of the amplified fragment was performed (Sequi-Gen Nucleic Acid Sequencing Cell, BioRad). The amplified sequence is shown below:

TGATTTACAAATGGCATTAGACAATGCCAAAATCAAGCTTAATG
AGAAACTATCTCAACTACAAACATATGTGATACAATTTGATCAGT
ATATTAAAGATAATTATGATTTACATGATTTTAAGGCAGCTATTGC
TAAGATTATTGATCAAATCATTGAAAAATTGAAAATTCTT

Note the yellow mark and the green mark are the forward and reverse primers alignment, respectively.

The below figure shows the forward primer alignment at the gene bank.

TGATTTACAAATGGCATTAGACAATGCCAAAATCAAGCTTAATG
AGAAACTATCTCAACTACAAACATAT

GTGATACAATTTGATCAGTATATTAAAGATAATTATGATTTACATG
ATTTTAAGGCAGCTATTGCTAAGA

TTATTGATCAAATCATTGAAAAATTGAAAATTCTT

<u>Procedure:</u>

The RNA primer mixture was prepared in 0.2ml microcentrifuge tube:

- Ten ml of RNA was pipetted into 0.2 ml microcentrifuge .

- Then 1 µl dNTPs and 1µl oligo d(T) were added, incubated at 65° C for 5min. and chilled on ice for 2min, then spin down the mixture.

- Ten µl cDNA Synthesis (Mix of 10X Buffer M-MuLV 2µl, M-MuLV (RT) 1µl and Water top to 10µl) was added. and incubated at 42° C for 60 min, then incubated at 85° C for 5 min to stop the reaction. Then was used in PCR.

3.2.11.3. DNA Amplification:

DNA amplification by using 2X Taq Master Mix.

- First all solutions were mixed after thawing.
- Then was Spin down and kept on ice.
- The following components were pipetted into a 0.2ml PCR tube on ice . preparation of 50µl reaction volume:

- Two x Taq Master Mix	25µl
- Primer1(APO-f for100pmol/µl dissolve in170µl)	1µl
- Primer 2 (APO-r µl dissolve in 262µl)	15µl
- cDNA	1µl
- DNase free water (PCR Grade H_2O)	8µl

Cycling conditions (100bp-5kb):

Denaturation: 94° C for		2min.
Denaturation : 94° C for	for 30 cycles	2sec.
Annealing : 51° C for 30sec.		

Extension : 72° C for 30s.

Final Extension: 72°C for 7 minutes.

3.2.11.4. Determination of the molecular weight of DNA using Agarose gel electrophoresis:

Electrophoresis of the amplified DNA was carried out on 1.5% agarose gels at 80 V for 2 hr. The amplification products were visualized directly with ethidium bromide in UV Transilumenetor .

Procedure:

- 1.5g agarose was mixed with 100 ml 1X TAE buffer

  and solved in the microwave
- Then four µl ethidium bromide were added to the gel
- The gel stayed for thirty minutes for gel polymerization
- Then 3 µl loading buffer were mixed with 15 µl sample
- After that 1µl ladder were added to 3 µl loading buffer
- Then were loaded in the gel. Then the gel was run
- Finally the cDNA were visualized on a UV transilluminator and photographed

3.2.12. Statistical analysis:

The statistical analysis was performed using SPSS. The analysis of variance (two paired Student *t* test) was used to evaluate the effects of high quality diet on camel blood constituents and weight gain.

CHAPTER FOUR

Results

4.1. Feeding:

Body weight and feeding of Zero browsing camels:

Table (4.1) shows feedlot performances of feed intake, initial body weight, Final body weight, weight gain, dry matter intake and feed conversion ratio (FCR) in zero browsing and free browsing camels.

The results revealed a significant increase ($P < 0.05$) in mean body weight and average growth rate in zero browsing groups (321.5± 38.5kg) as compared to free browsing groups (272 ±32.3kg). The average total gain was almost double in zero browsing groups than free browsing group. Table (4.2) shows no significant differences in weight gain, dry matter intake and feed conversion ratio between Darfuri & Butana types in zero browsing camel group.

Figure (4.2) revealed that the rate of food consumption during the whole experimental period did not show major variation among different time intervals in zero browsing group.

Table (4.1) Weight gain of Zero browsing and free browsing camels:

Parameters	Period of experiment(day)120	
	Zero browsing n(25)	free browsing n(25)
Initial body wt. kg	225.5± 35.2	230 ±25.9

Final body wt. kg	321.5± 38.5	272 ±32.3
Total weight gain kg	96 ±17.3	42 ±19.5
Average daily gain/kg	0.800 ± 0.08	0.322±0.04
DM intake kg /day	4.44 ±	
Feed Conversion Ration	5.8 ±.1.09	

*= significant , (Means± SE)
NS= not significant

Table (4.2) Weight gain of Darfuri and Butana camels:

Parameters	Period of experiment(day)120	
	Darfuri n(15)	Butana n(10)
Initial body wt. kg	227 ± 32.2	224 ± 30.5
Final body wt. kg	321.8± 35.3	321.2± 34.5
Total gain kg	94.8±19.3	97.8±17.2
Average daily gain/ kg	0.790± 0.11	0.810 ± 0.06
DM intake/ kg /day	4.44± 0.12	4.34 ± 0.25
Feed Conversion Ration	5.8 ± 1.58	5.5 ± 1.53

*NS = not significant, (Means± SE)

Sig= significant

Fig. (4.1) Weight gain in zero browsing animals

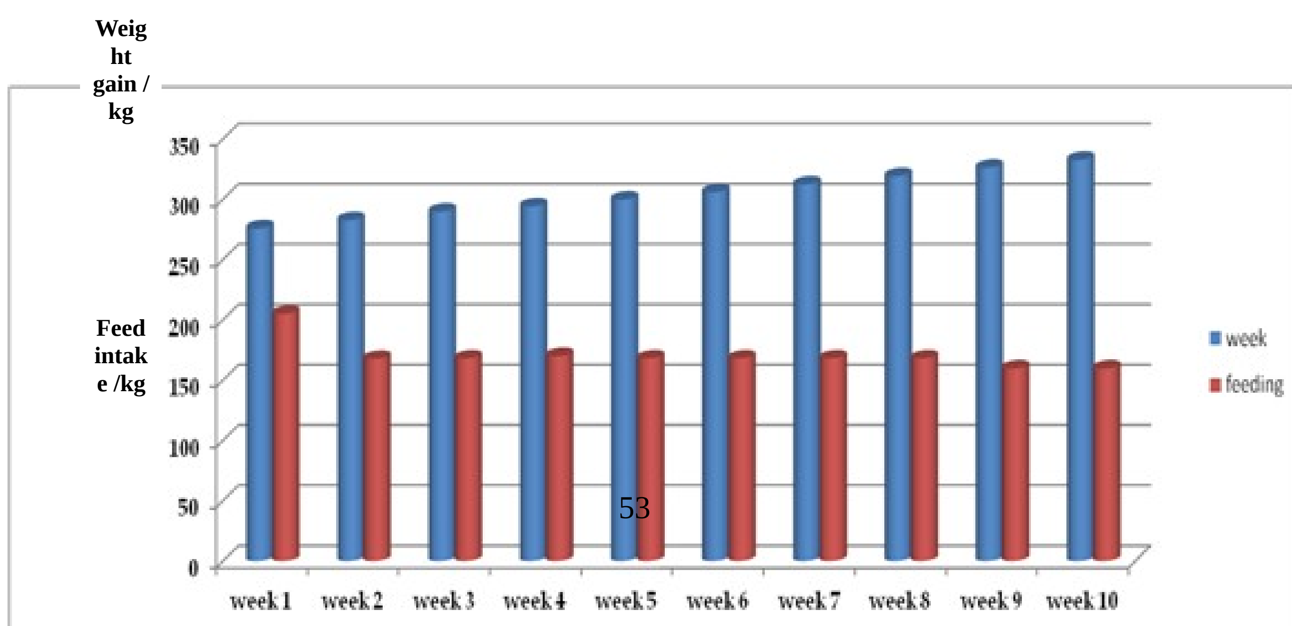


Fig. (4.2) Feed intake in zero browsing animals

4.2. Biochemical Findings:

Table (4.3) shows the biochemical parameters of zero browsing and free browsing animals. The results revealed that a significant effect ($P < 0.05$) was recorded for the concentrations of plasma glucose, serum urea and creatinine concentrations.These are higher in zero browsing group (88.1mg%, 24.04mg/dl,1.34mg/dl) respectively. Than the free browsing (57.2mg%, 15.8mg/dl, 1.2mg/dl, respectively. Serum albumin concentrations was slightly higher in zero browsing (4.3mg/dl) camels when compared with the free browsing (4.0mg/dl). However, the differences were not statistically significant in globulin (3.5mg/dl), uric acid(2.9mg/dl)in zero browsing when compared with free browsing (3.2mg/dl) and (2.8mg/dl), respectively.

Table (4.4) shows the biochemical parameters of serum lipids and lipoproteins in zero grazing and free grazing camels. The results showed a significant ($P < 0.05$) high concentrations of cholesterol, HDL-cholesterol and triglycerides in zero grazing group (41.32mg/dl, 21.88mg/dl and49.71 mg/dl) compared with the free grazing (36.98mg/dl, 16.35mg/dl and 44.59 mg/dl, respectively. Moreover, the differences was not statistically significant in LDL-cholesterol, VLDL-cholesterol., for

zero grazing (10.79 mg/dl, 8.871mg/dl) and free grazing (10.68mg/dl, 9.294mg/dl), respectively.

Table (4.5) shows the biochemical enzymes and minerals in zero browsing camels and free browsing. A higher significant effect ($P < 0.05$) was recorded on the concentration of sodium and glutamate oxaloacetate transaminase (GOT) in the zero browsing group (147.5 mEq/L, 32.98 U/L) when compared to the free browsing camels (141.3 mEq/L, 28.71 U/L, respectively. The differences were not statistically significant for Potassium and glutamate pyruvate transaminase (GPT), for zero browsing (4.194 mEq/L 20.69 U/L) compared to free browsing (3.973 mEq/L, 19.51 U/L), respectively.

Table (4.6) shows the biochemical parameters of Darfuri and Butana in zero browsing animals. The results revealed that there was no significant differences between the two types in all parameters measured.

Table (4.7) shows the biochemical parameters of serum lipids and lipoproteins of Darfuri and Butana in zero browsing camels. The results showed high significant increase ($P < 0.05$) in the concentration of HDL-cholesterol which was observed in Darfuri group (22.82mg/dl) as compared to the Butana (20.17mg/dl). There was no significant variations in all other parameters.

Table (4.8) shows the biochemical enzymes and minerals of Darfuri and Butana in zero browsing camels. The results revealed no significant differences in all parameters.

Table (4.9) shows the biochemical parameters of Darfuri and Butana in free browsing animals. The results revealed no significant differences in all parameters measured.

Table (4.10) shows the biochemical parameters of serum lipids and lipoproteins of Darfuri and Butana in free browsing camels. The results showed a significant increase ($P < 0.05$) in the concentrations of

triglycerides which was observed in The Butana group (53.00mg/dl) as compared to the Darfuri (47.91mg/dl). No significant differences were observed in other parameters.

Table (4.11) shows the concentration of serum enzymes and minerals of Darfuri & Butana in free grazing camels. The results revealed no significant differences in all parameters measured.

Table (4.12) and figure (4.3) show glucose tolerance tests for experimental animals. The results showed (GTT) increased at min5 which was found to be more than150 and decreased to lower than 80 at min15,it began to rise to more than158 at min45 then decreased to 116 at min60 after that it increased to more than 200 at min90 then decreased at min180 to 159.

Table (4.13) shows correlation of blood metabolites level for zero and free browsing camels. The results revealed that serum cholesterol had significant correlations with triglycerides, HDL and LDL- cholesterol. However, triglycerides had significant correlations with VLDL-cholesterol and HDL-cholesterol. HDL-cholesterol was negatively correlated with triglycerides, while total cholesterol was positively correlated with LDL-cholesterol, urea was significantly and positively correlated ($P < 0.01$) with creatinine, glucose and HDL-cholesterol, and negatively correlated with triglycerides. Whereas, creatinine was significantly and positively correlated with GOT, K, HDL, total protein and total cholesterol, and negatively correlated with GPT and triglycerides. GOT was significantly and positively correlated ($P < 0.01$) with Na, K, glucose and HDL, while it was negatively correlated with GPT. Glucose was significantly and positively correlated with total protein, HDL, total cholesterol, and negatively correlated with triglycerides. Albumin, K, total protein, triglycerides and HDL were significantly and positively correlated with globulin, glucose, HDL, VLDL and total cholesterol. Total cholesterol was negatively correlated with triglycerides, while it was positively correlated with LDL-

cholesterol and HDL-cholesterol, but there was no significant correlation with uric acid.

Table (4.14) shows correlation of blood metabolites level of Darfuri and Butana in zero browsing camels. The results revealed significant and positive correlations ($P < 0.01$) between creatinine and uric acid, triglyceride and VLDL. While GPT, albumin and K were significantly and negatively correlated ($P < 0.05$) with albumin, LDL and HDL cholesterol, respectively. But there were no significant correlations with urea, GOT, Na, total cholesterol and total protein.

Table (4.15) shows correlation of blood metabolites level of Darfuri and Butana in free browsing camels. The results showed albumin had significant and positive correlation ($P< 0.01$) with globulin and LDL had significant and positive correlation with total cholesterol. While, urea was significantly and positively correlated ($P< 0.05$) with GPT. However, creatinine and glucose were significantly and negatively correlated ($p<0.05$) with sodium and total protein, respectively. But there were no significant correlations with uric acid, GOT, GPT, K, HDL and triglycerides.

Figure (4.4) shows the biochemical parameters of zero browsing and free browsing. The concentraion of creatinine, urea and sodium were significant increased ($P<0.05$) in zero browsing group when compared to the free browsing. While, there was no variation in uric acid and Potassium concentraion between the two groups.

Fig (4.5) shows significant elevation($P<0.05$) in blood glucose, total protein and GOT levels in zero browsing when compared to the free browsing group. However, Serum globulin, albumin and GPT levels showed no significant difference.

Fig (4.6) shows significant increase ($P<0.05$) in triglycerides level in free browsing when compared to the zero browsing group.while, HDL-Cholesterol reveled significant increase ($P<0.05$) in HDL-Cholesterol level in zero browsing when compared to the free browsing.

There were no differences in LDL-Cholesterol, VLDL-cholesterol levels between the two groups.

Table (4.3) Serum concentration of some biochemical parameters of zero browsing and free browsing animals:

Parameters	Unit	zero browsing	Free browsing	Sig.
Glucose	mg%	88.10±1.66	57.20±1.27	*
Total protein	mg/dl	7.04±0.28	7.91±0.32	NS
Albumin	mg/dl	4.32±0.15	4.01±0.12	NS
Globulin	mg/dl	3.52±0.22	3.20±0.16	NS
Creatinine	mg/dl	1.34±0.01	1.24±0.01	*
Urea	mg/dl	24.04±0.59	15.80±0.38	*
Uric acid	mg/dl	2.94±0.04	2.87±0.05	NS

*=significant difference at($p<0.05$),NS=not significant, (values were mean ± SE), n=40

Table (4.4) Biochemical parameters of serum lipids and lipoproteins in zero and free browsing camels:

Parameters	Unit	zero browsing	Free browsing	Sig
Triglycerides	mg/dl	50.71±1.05	44.09 ±1.09	*
Total cholesterol	mg/dl	41.32±0.63	36.08±0.84	*

HDL- cholesterol	mg/dl	21.88±0.51	16.35±0.45	*
LDL- cholesterol	mg/dl	10.79±0.32	10.98±0.42	NS
VLDL-cholesterol	mg/dl	8.87±0.22	9.29±0.28	NS

HDL, high-density lipoprotein; LDL, low-density lipoprotein; VLDL, very low-density lipoprotein. *=significant difference at($p<0.05$)
NS=not significant,(values were mean ± SE), n = 40

Table (4.5) Serum concentration of some enzymes and minerals in zero and free browsing camels:

Parameters	Unit	zero browsing	Free browsing	Sig
GOT	U/L	32.98±0.9595	28.01±0.6455	*
GPT	U/L	20.69±0.5060	19.51±0.4767	NS
Sodium	mEq/L	147.5±1.656	141.3±2.247	*
Potassium	mEq/L	4.194±0.1260	3.973±0.1124	NS

GOTglutamate oxaloacetate transaminase;GPT glutamate pyruvate transaminase
NS=not significant, *=significant difference at($p<0.05$)
(values were mean ± SE), n = 40

Table(4.6) Serum concentration of some biochemical parameters of Darfuri and Butana in zero browsing camels:

Parameters	Unit	Darfuri	Butana	Sig.
Glucose	mg%	88.91±1.59	86.62±2.88	NS
Total protein	mg/dl	7.56±0.128	8.00±0.314	NS
Albumin	mg/dl	4.27±0.18	4.28±0.127	NS
Globulin	mg/dl	3.08±0.21	3.41±0.172	NS
Creatinine	mg/dl	1.82±0.027	1.88±0.024	NS
Urea	mg/dl	67.26±3.45	65.92±7.72	NS
Uric acid	mg/dl	2.99±0.07	3.043±0.08	NS

NS=not significant
(values were mean ± SE)n=20

Table(4.7) Serum lipids profile of Darfuri and Butana in zero browsing camels:

Parameters	Unit	Darfuri	Butana	Sig.
Triglycerides	mg/dl	43.17±1.315	46.17±1.88	NS
Total cholesterol	mg/dl	41.25±0.726	41.47±1.29	NS
HDL- cholesterol	mg/dl	22.82±0.519	20.17±0.703	*
LDL- cholesterol	mg/dl	10.55±0.477	10.42±0.46	NS
VLDL-cholesterol	mg/dl	8.67±0.266	9.16±0.34	NS

*=significant difference at(p<0.05)
NS=not significant, (values were mean ± SE), n = 20

Table(4.8) Serum concentration of some enzymes and minerals of Darfuri and Butana in zero browsing camels:

Parameters	Unit	Darfuri	Butana	Sig.
GOT	U/L	43.21±1.47	43.80±0.82	NS
GPT	U/L	35.81±1.93	33.00±1.88	NS
Sodium	mEq/L	146.7±2.43	149.00±1.58	NS
Potassium	mEq/L	4.28±0.18	4.56±0.19	NS

NS=not significant
(values were mean ± SE), n = 40

Table(4.9) Serum concentration of some biochemical parameters of Darfuri and Butana in free browsing camels:

Parameters	Unit	Darfuri	Butana	Sig.
Glucose	mg%	57.00±1.69	59.83±1.83	NS
Total protein	mg/dl	7.06±0.28	6.98±0.30	NS
Albumin	mg/dl	4.37±0.18	4.28±0.13	NS
Globulin	mg/dl	3.5±0.29	3.51±0.17	NS
Creatinine	mg/dl	1.32±0.03	1.33±0.03	NS

Urea	mg/dl	53.56±1.45	51.11±2.72	NS
Uric acid	mg/dl	2.94±0.07	3.00±0.08	NS

NS=not significant
(values were mean ± SE), n=20

Table (4.10) Serum lipids profile of Darfuri and Butana camels in free browsing:

Parameters	Unit	Darfuri	Butana	Sig.
Triglycerides	mg/dl	47.91±1.05	53.00±1.59	*
Total cholesterol	mg/dl	37.03±1.25	36.88±.85	NS
HDL- cholesterol	mg/dl	16.55±0.59	16.00±0.73	NS
LDL- cholesterol	mg/dl	10.83±0.63	10.42±0.44	NS
VLDL-cholesterol	mg/dl	9.24±0.32	9.40±0.55	NS

*=significant difference at($p<0.05$)
NS=not significant, (values were mean ± SE), n=20

Table (4.11) Serum concentration of some enzymes and minerals of Darfuri and Butana in free browsing camels:

Parameters	Unit	Darfuri	Butana	Sig.
GOT	U/L	34.26±1.81	37.90±1.92	NS
GPT	U/L	41.55±0.90	44.53±1.15	NS
Sodium	mEq/L	141.1±1.16	142.7±1.78	NS
Potassium	mEq/L	3.82±0.12	3.65±0.13	NS

NS=not significant, (values were mean ± SE), n=20

Table(4.12) glucose tolerance test:

Time/mintes	0	5	15	25	35	45	60	80	120	150	180
Glucose concentrations mg/dl	136	153	74	126	157	129	116	214	127	176	159

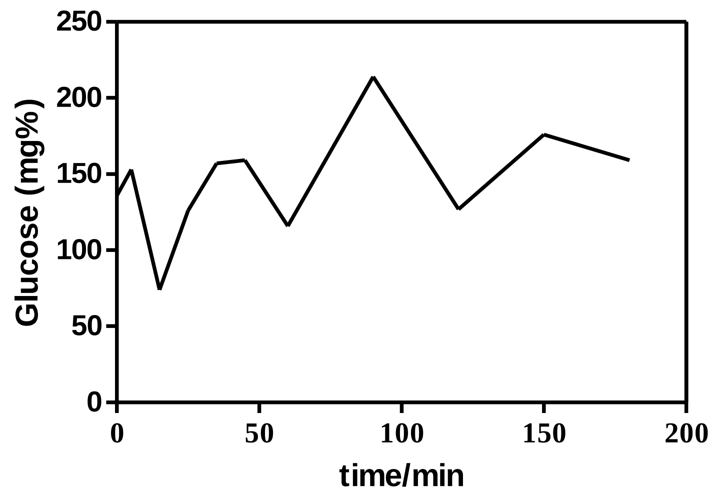


Fig. (4.3) glucose tolerance test

Table(4.13) Correlation between blood parameters in zero browsing and free browsing camels

	Urea	Creatin.	uric acid	GOT	GPT	Albu.	N	K	glucose	total prote.	Glob.	Trigly.	HDL. Chol.	LDL	VLDL	total chol.
Urea	1															
creatinine	0.57**	1														
uric acid	0.078	0.319	1													
GOT	0.334	0.578**	0.213	1												
GPT	-0.268	-0.577**	-0.291	-0.445**	1											
Albumin	-0.068	-0.068	-0.009	-0.14	-0.324	1										
N	0.316	0.361*	-0.165	0.467**	-0.344*	0.059	1									
K	0.374*	0.621**	0.285	0.456**	-0.503**	-0.003	0.072	1								
glucose	0.522**	0.879**	0.264	0.573**	-0.608**	0.006	0.342*	0.559**	1							
total protein	0.191	0.526**	0.187	0.307	-0.292	-0.051	0.222	0.363*	0.436**	1						
globulin	-0.154	-0.139	0.125	-0.248	-0.158	0.762**	-0.129	-0.078	-0.187	-0.094	1					
triglyceride	-0.521**	-0.53**	-0.059	-0.294	0.384*	0.028	-0.14	-0.314	-0.477**	-0.206	0.162	1				
HDL.Cholesterol	0.469**	0.763**	0.111	0.437**	-0.403*	0.017	0.409*	0.261	0.762**	0.377*	-0.223	-0.525*	1			

LDL	**-0.03**	**-0.015**	**-0.095**	**-0.14**	**0.115**	**-0.269**	**-0.073**	**0.084**	**-0.063**	**-0.1**	**-0.068**	**0.097**	**-0.079**	**1**		
VLDL	**-0.308**	**-0.158**	**0.055**	**-0.308**	**0.307**	**-0.058**	**-0.229**	**0.018**	**-0.243**	**0.015**	**0.214**	**0.497****	**-0.333**	**0.225**	**1**	
total cholesterol	**0.329**	**0.467****	**-0.178**	**0.264**	**-0.371***	**0.019**	**0.393***	**0.145**	**0.501****	**0.186**	**-0.124**	**-0.351***	**0.6****	**0.351***	**-0.061**	**1**

* *Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

	Urea	**Creatini.**	**uric acid**	**GOT**	**GPT**	**Albumin**	**N**	**K**	**Gluco.**	**total prote.**	**Globul.**	**Triglyce.**	**HDL. Chol**	**LDL**
Urea	**1**													
creatinine	**0.203**	**1**												
uric acid	**-0.030**	**0.740****	**1**											
GOT	**-0.277**	**0.036**	**0.117**	**1**										
GPT	**-0.004**	**-0.177**	**-0.338**	**-0.384**	**1**									
Albumin	**-0.024**	**-0.231**	**-0.161**	**-0.285**	**-0.506***	**1**								
N	**0.108**	**-0.119**	**-0.369**	**0.430**	**-0.091**	**0.006**	**1**							
K	**0.076**	**0.511***	**0.247**	**0.156**	**-0.462**	**0.198**	**-0.209**	**1**						
glucose	**-0.074**	**0.432**	**0.537**	**-**	**-0.263**	**0.264**	**-**	**0.305**	**1**					

			*	0.260			0.385							
total protein	**-0.092**	**0.008**	**0.207**	**0.087**	**0.084**	**-0.115**	**-0.143**	**0.009**	**-0.066**	**1**				
globulin	**0.000**	**0.001**	**-0.047**	**-0.153**	**-0.463**	**0.605***	**-0.106**	**0.233**	**0.010**	**-0.213**	**1**			
triglyceride	**-0.382**	**0.065**	**-0.128**	**-0.008**	**0.349**	**-0.377**	**0.013**	**0.042**	**-0.305**	**0.291**	**-0.245**	**1**		
HDL.Cholesterol	**-0.075**	**-0.204**	**0.032**	**-0.307**	**0.320**	**0.124**	**-0.012**	**-0.584***	**0.240**	**-0.129**	**-0.240**	**-0.210**	**1**	
LDL	**0.186**	**0.422**	**0.095**	**0.096**	**0.167**	**-0.563***	**-0.038**	**0.333**	**0.093**	**-0.214**	**-0.113**	**0.370**	**-0.319**	**1**
VLDL	**-0.444**	**0.040**	**-0.127**	**0.072**	**0.256**	**-0.317**	**0.008**	**0.156**	**-0.334**	**0.256**	**-0.233**	**0.972****	**-0.287**	**0.298**
total cholesterol	**0.072**	**-0.406**	**-0.377**	**-0.107**	**-0.096**	**0.119**	**0.284**	**-0.313**	**-0.363**	**-0.326**	**0.000**	**-0.016**	**0.202**	**-0.263**

Table (4.14) Correlation between blood parameters in zero browsing camels Darfuri and Butana

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

	Urea	**create.**	**uric acid**	**GOT**	**GPT**	**Albu.**	**N**	**K**	**Gluco.**	**total prote.**	**Glob.**	**Trigl.**	**HDL. Chol.**	**LDL**
Urea	**1**													
creatinine	**0.044**	**1**												

uric acid	**0.121**	**0.424**	**1**											
GOT	**0.343**	**-0.114**	**0.236**	**1**										
GPT	**0.545***	**-0.054**	**-0.113**	**0.211**	**1**									
Albumin	**-0.110**	**0.093**	**0.195**	**-0.038**	**-0.372**	**1**								
N	**-0.104**	**-0.519***	**-0.026**	**0.075**	**-0.117**	**0.256**	**1**							
K	**0.151**	**0.200**	**0.285**	**0.200**	**0.302**	**-0.214**	**-0.383**	**1**						
glucose	**0.147**	**-0.345**	**0.174**	**0.125**	**-0.156**	**0.008**	**-0.130**	**-0.148**	**1**					
total protein	**-0.480**	**0.033**	**0.034**	**-0.218**	**-0.056**	**0.085**	**0.132**	**0.225**	**-0.591***	**1**				
globulin	**-0.154**	**0.234**	**0.345**	**-0.161**	**-0.296**	**0.899****	**0.032**	**-0.155**	**-0.001**	**0.234**	**1**			
triglyceride	**-0.262**	**-0.358**	**0.186**	**0.091**	**-0.335**	**0.387**	**0.368**	**-0.178**	**0.419**	**-0.124**	**0.297**	**1**		
HDL.Cholesterol	**0.238**	**0.158**	**-0.018**	**-0.097**	**-0.213**	**0.085**	**0.149**	**-0.090**	**-0.347**	**-0.213**	**0.000**	**-0.211**	**1**	
LDL	**-0.344**	**-0.101**	**-0.278**	**-0.266**	**0.022**	**-0.049**	**-0.081**	**-0.080**	**-0.155**	**0.046**	**-0.063**	**-0.161**	**0.178**	**1**
VLDL	**0.075**	**0.214**	**0.298**	**-0.382**	**0.243**	**0.114**	**-0.373**	**0.211**	**0.122**	**0.097**	**0.397**	**0.057**	**-0.264**	**0.169**
total cholesterol	**-0.153**	**-0.234**	**-0.279**	**-0.213**	**0.021**	**0.022**	**0.032**	**-0.250**	**0.021**	**-0.115**	**-0.009**	**-0.111**	**0.303**	**0.91****

Table(4.15) Correlation between blood parameters in free browsing camels Darfuri and Butana

* * Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Serum Con. gm/dl

Fig. (4.4) Some biochemical parameters in serum of zero browsing and free browsing animals.

Serum Con mg

Fig. (4.5) Serum lipids and lipoproteins in zero browsing and free browsing animals.

Serum Con

Fig. (4.6) Serum concentration of some biochemical enzymes and minerals in zero browsing and free browsing animals.

Serum Con. gm/dl

Fig. (4.7) Serum levels of some biochemical parameters of Darfuri and Butana in zero browsing

U/L U/L mEq/L

Serum Con. gm/dl

Fig. (4.8) Serum lipids profile of Darfuri and Butana in zero browsing

Serum Con.

Fig. (4.9) Serum concentration of some biochemical enzymes and minerals in zero browsing animals Darfuri and Butana camels.

Serum Con. gm/dl

Fig. (4.10) Serum of some biochemical parameters of Darfuri and Butana camels in free browsing group.

U/L U/L mEq/L

Serum Con.mg/dl gm/

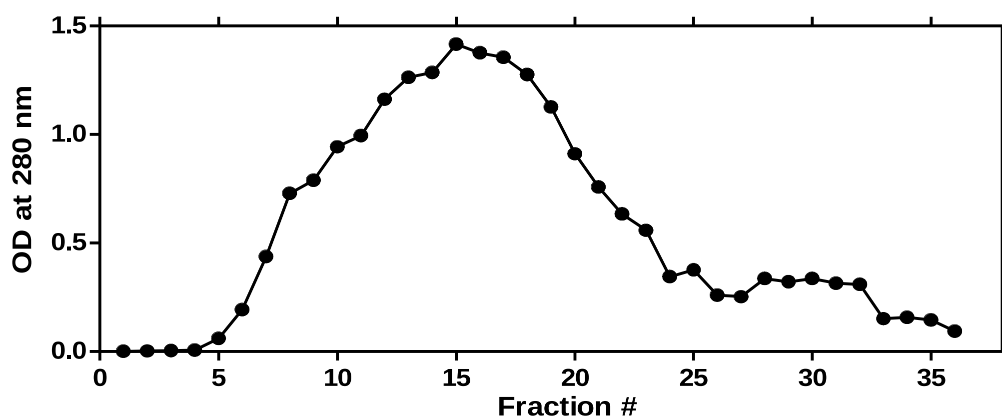
Fig. (4.11) Serum lipids and lipoproteins in free browsing Darfuri and Butana camels.

Serum Con.

U/L U/L mEq/L

Fig. (4.12) Serum enzymes and minerals of Darfuri and Butana free browsing camels.

The chromatograph of gel sizing chromatography in figure (4.13) and (4.14) showed no difference between the zero browsing and the free browsing groups except the long shoulder peak in range between 25 – 30 fractions that were noticed in the zero browsing group. This might be interpreted as a result of high concentration of low molecular weight protein in plasma of zero browsing animals when compared with the free browsing.



OD= optimum on optical density

Fig. (4.13) Major protein fractionation in zero browsing camels: (gel permeation):

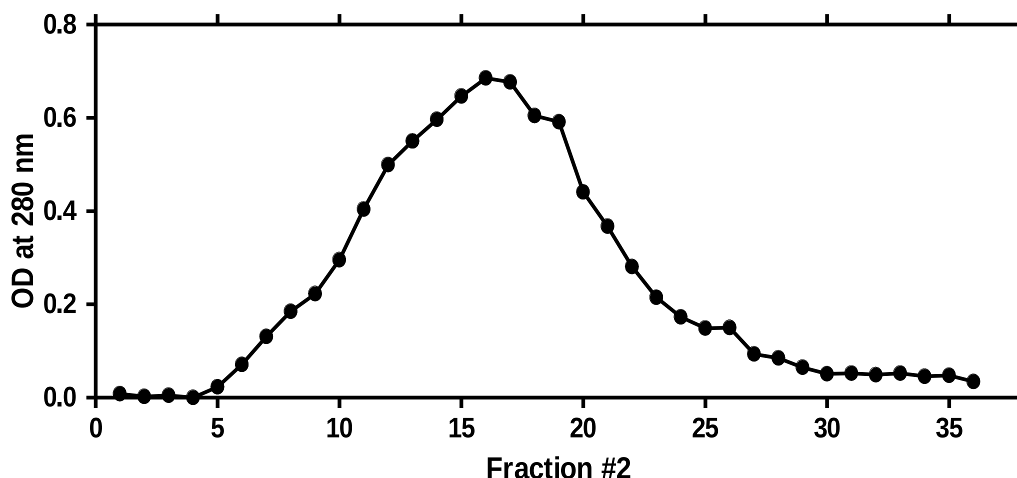


Fig. (4.14)Major proteins in free browsing animals:

4.3. Molecular Findings:

Table (4.15) shows molecular weight of protein subunits using SDS-polyacrylamide gel electrophoresis. Figure (4.15) shows a notable increase in the expression of protein in the HDL fraction as observed by wide protein bands at 27 KD when compared to the free browsing group. However, there are clear bands in two groups at145, 61KD.

Figure (4.16) shows neither apparent change in quantitative nor qualitative protein pattern distribution between zero browsing and free browsing camels regarding the LDL-rich fraction as revealed by SDS-PAGE with major dense banding at approximate 51,36,29and 22 KD.

Figure (4.17) shows dense clear banding at 175,95,70,51,40,29,22 KD in the two groups. Whereas, the zero browsing group shows dense clear banding at low molecular weight in the range of 10 KD. This dense band not clearly proved to appear in the free browsing group.

Figure (4.18) The specific band at 125 bp indicates the presence of the cDNA corresponds to ApoB gene (with reference to primer design) in our samples with higher rate of expression in the hepatic tissue (lane 5 when compared to others). Other tissues, however, like muscle tissues (lanes 3,

4, 7) showed ApoB lipoprotein gene down regulation as revealed by low rate of its mRNA expression that gave low concentration of its corresponding cDNA in their corresponding lanes.

Table (4.16): molecular weight of protein subunits using SDS-PAGE

Parameters	Zero browsing	Free browsing	Molecular weight
HDL-cholesterol	Lane 1,2,3	Lane 4,5	145KD
HDL-cholesterol	lane 1,2	Non	27 KD
HDL-cholesterol	lane1,2,3	lane4,5	61KD
LDL-cholesterol	lane 3,4,6	lane1,2,5,7	51,36,29.22KD
Liver homogenate	Lane1,2,3	Lane4,5,6,7	175,95,70,51KD
Liver homogenate	Lane1,2,3	Lane4,5,6,7	40,29,22KD
Liver homogenate	Lane1,2,3	Non	10KD

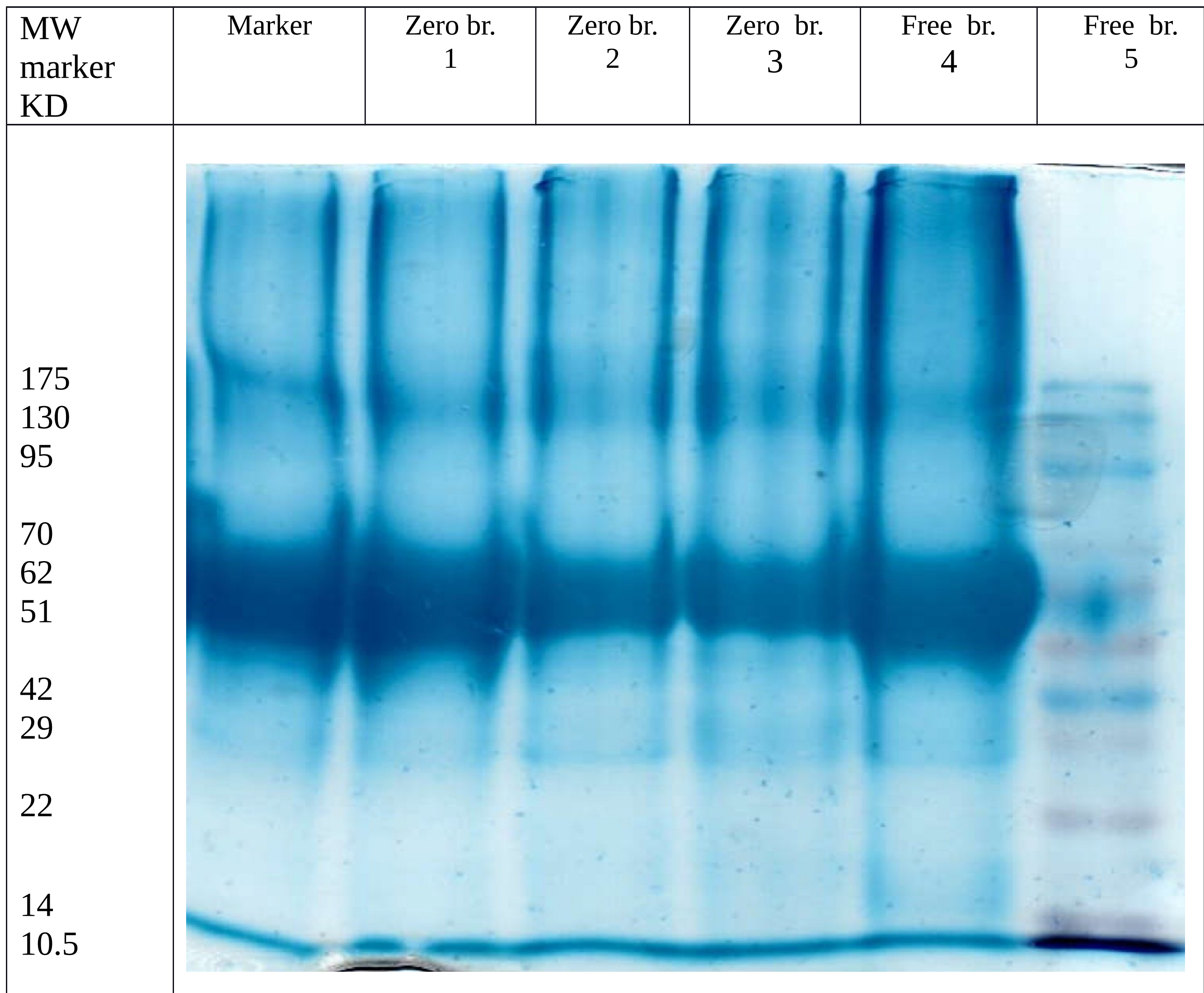


Fig. (4.15 **):** SDS PAGE of HDL in zero and free browsing camel plasma

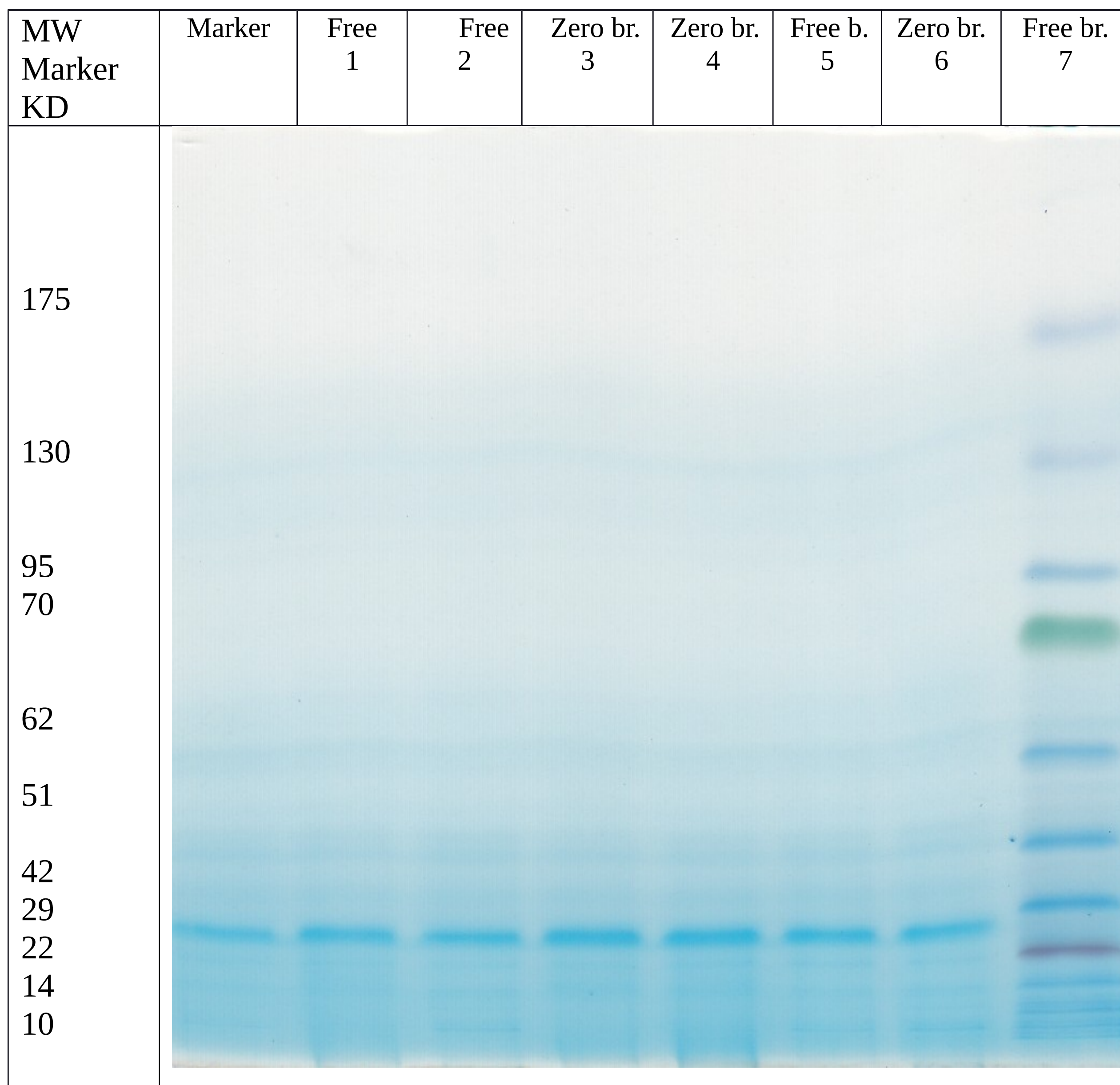


Fig. (4.16)SDS-Page of LDL in zero grazing and free browsing camels plasma

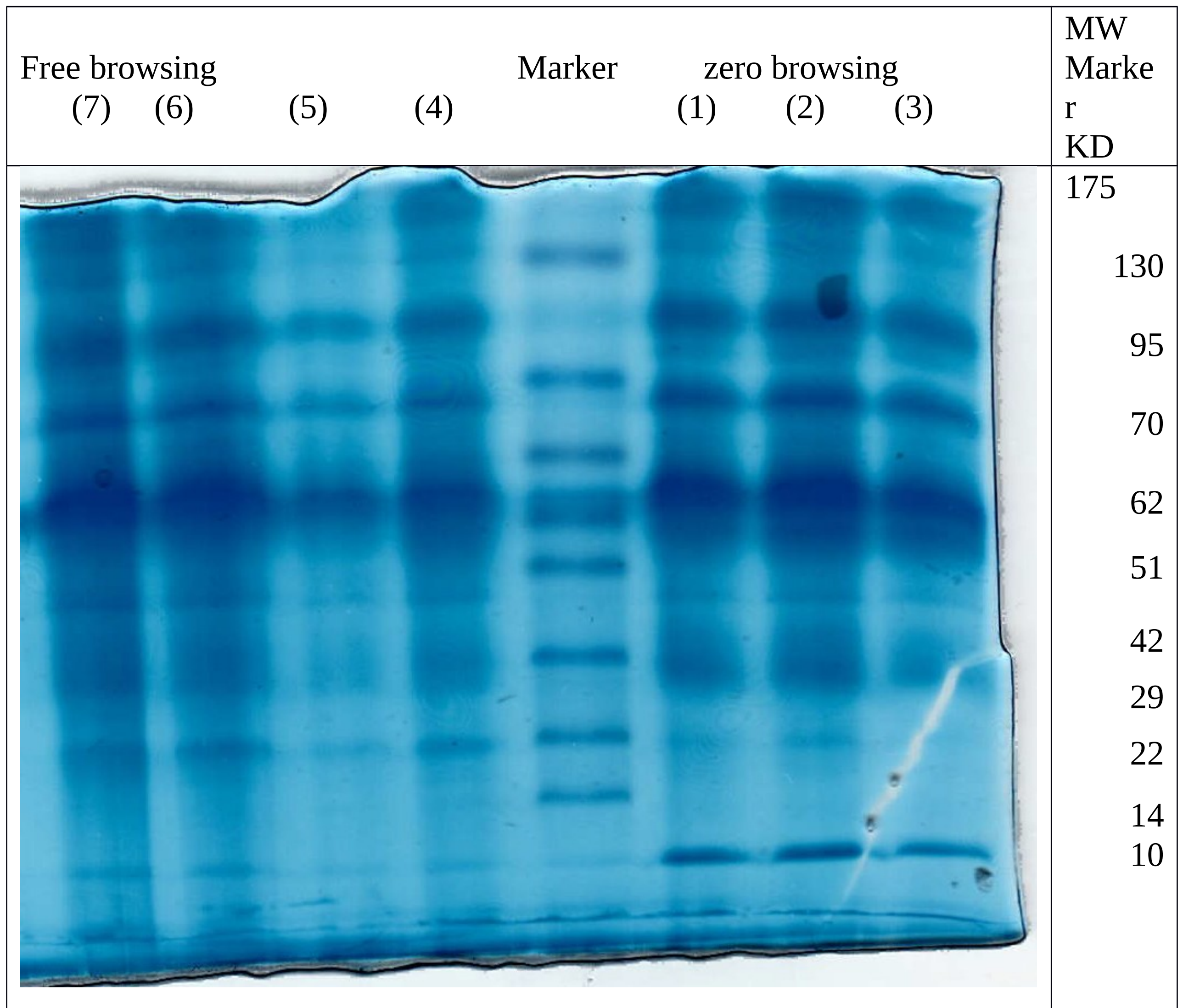


Fig. (4.17) SDS-PAGE of lipoprotein in zero browsing and free browsing camel plasma.

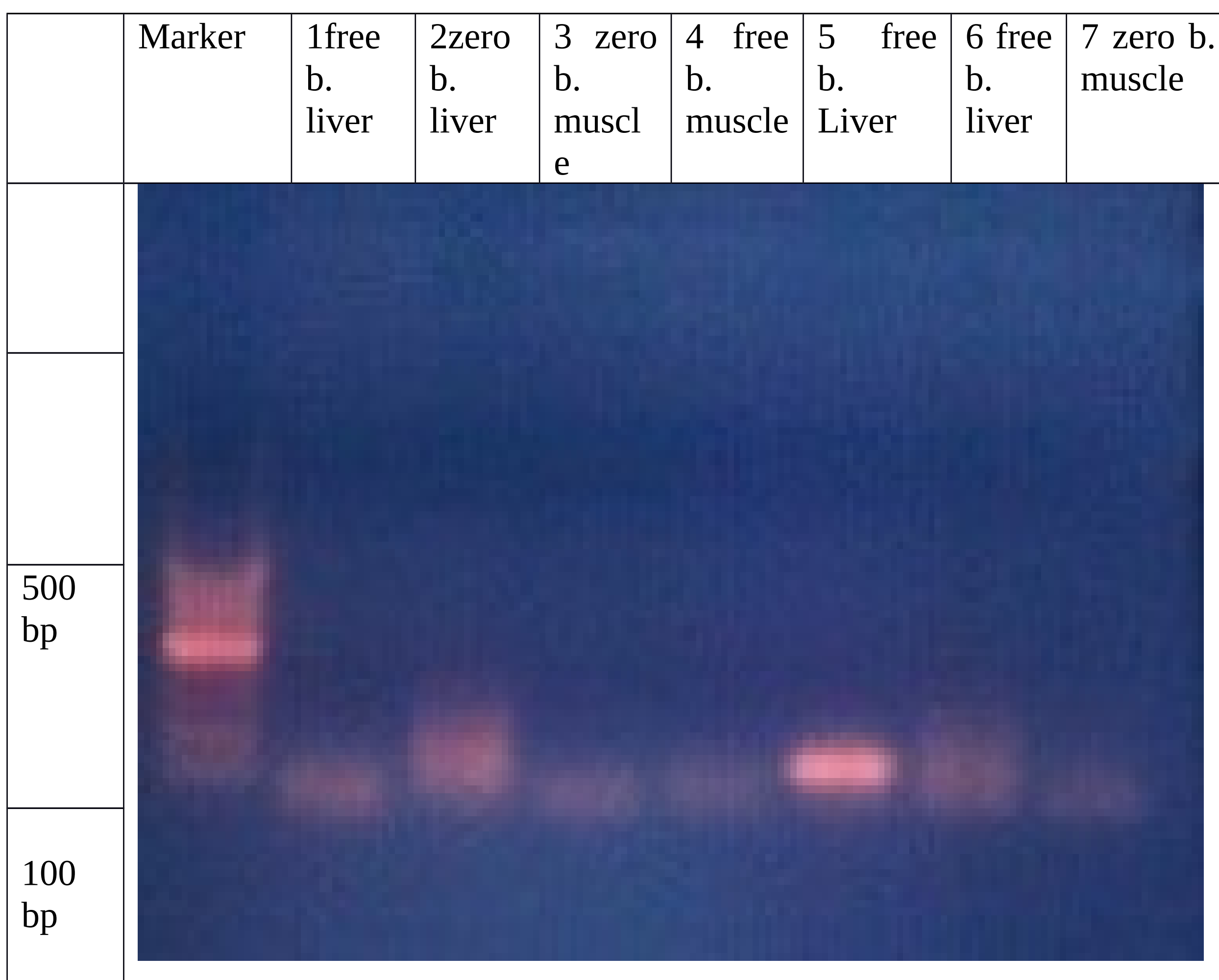


Fig. (4.18)The agarose gel electrophoresis of the RT-PCR products of apolipoproteins

CHAPTER FIVE

Discussion

The present investigation has been designed to study the effect of feeding regimens in dromedary camel. It appears from figure (4.1) the weight gain is gradually increased during the experimental period. Growth performance of camel-calves fattened with concentrate feed were presented in table (4.1) and (4.2). In this study the results revealed a significant increase ($P< 0.05$) in mean body weight and average growth rate in zero browsing group as compared to free browsing camel group.

These results agreed with those of Mohamed (2006), when fed growing dromedary camels ration containing black cumin seed cake (35%), molasses (18%) and a mixture of different straws (45%), Similar results were further obtained by Bakkar *et al*. (1998) who investigated that pelleted diet animals recorded high gain.

This work showed a higher value for average daily weight gain than that obtained by Sahani *et al*. (1998) and Faye *et al.* (2001) who reported that the daily gain for male camels between 18 to 24 months of age ranged from 0.111±0.015 to 0.219±0.24 kg/day, also higher than that recorded by Al Saiady *et al.* (2006) who recorded 0.741 kg average daily weight gain during 90 days when camels fed 75% concentrate and 25% hay. Further more the DWG (387 g/day) was not similar to our observations (Kaufmann, 1998), also higher than the values reported by (Hashi and Kammoun, 1995) which found that young growing camels achieved an average daily gain(ADG) of 285 g when offered a diet providing approximately 8.5 MJ ME per kg DM and hence an energy intake level at the lower end of the daily ME allowance for maintenance and live weight gain of cattle. Both results would suggest that camels have lower energy requirements and/or extract more from fibrous feeds. However, more field work is needed to determine the metabolism of camels' diets and the energy costs of feeding and production to develop feed budgets within defined production pattern.. In very intensive systems for fattening young camels, Faye, (1997) reported

(500g). These results could be due to the difference in the management system, the nutritive value of the diet distributed to the animals and the breed characteristics. However, the result for feed conversion ratio (FCR)was lower than that reported by (Mustafa *et al*., 1990; Eltayeb *et al*., 1990; Mohamed, 1999; Intesar, 2002). Guma

(1996), Itidal (2004) El Badawi and Yacout (1999) and Al Saiady *et al*., 2006).

This improvement in the Feed Conversion Ration might be attributed in part to absence of selective feeding behavior of the camel due to the complete diet system used in formulating the diet of high CP content (16%).

The difference in feeding performance showed a marked effect ($P< 0.05$) on the plasma biochemical constituents (Table 4.3) after feeding complete ration when compared to natural browsing. The observed increase in the level of plasma glucose ($P < 0.05$) in the group fed with complete ration could be due to the increased energetic value (Wensvoort *et al*., 2004). Abdel-Fattah *et al*. (1999) reported that the glucagon level in camels is higher than that in other mammals including man, and suggests that this is a probable species specificity, which would explain the higher level of glucose in the blood of camels than in that of other ruminants. The disappearance curve of injected glucagon in camel with exponential two-compartment function as previously reported in other mammals might explain the well-developed anti-insulin hormonal control in this creature. The hormone was rapidly distributed and was eliminated with a high rate of clearance. In this study, the plasma glucose concentration was in agreement with that previously reported by Sazmand *et al.,* 2013; Mohammed, 2007; Aichouni *et al*. 2013 and Amin *et al*., 2007 with lower value than that found by Osman and Al-Busadah, 2003; and Hassan *et al*., 2011.

The observed increase in the concentration of plasma creatinine in zero browsing group could be attributed to the higher intake of protein in the diet consumed by camels. However, In this study, The plasma creatinine levels was within the range reported by AL-Sultan,

(2003), with lower value than that found by Osman and Al-Busadah, 2003.

The observed increase in the concentration of plasma total protein in the free browsing group could be attributed to the stresses to which camels were subjected under dry conditions. Abokouider *et al*. (2001) reported an increase in plasma total protein in camel during the dry season. Similar values of serum proteins in this study were obtained by other workers (Al-Busada, 2007; Sazmand *et al*.,2013;Salah El-Din, 2005; Al-Sultan, 2003 and Osman and Al-Busadah, 2003), higher than the reference values reported by Shaker, 2011 and Mohammed, 2007.

Serum albumin concentration was similar to values reported by (Al-Busada, 2007;Salah El-Din *et al.,* 2005). Serum globulin was similar to the reference values obtained by Al-Busadah, 2007; Salah EL Din, 2005 and higher than that obtained by Osman and Al-Busadah, 2003; Mohammed, 2007 and Al Sultan, 2003.

The observed increase in the concentration of plasma triglycerides in the zero browsing group could be attributed to the dietary concentrate ration. The plasma triglyceride reported in this study, was within the range reported by (Hassan *et al*.,2011; Nazifi *et al*.,2000 ;Aichouni *et al*.,2010) but, lower than the values reported by (Mohamed, 2008; Asadi *et al.,* 2008) and higher than that obtained by (Khajeh *et al*., 2008; Sazmand *et al*., 2013and Osman and Al-Busadah, 2003).

The observed increase in the concentration of plasma urea in the zero browsing group could be attributed to the increased nitrogenous intake in the diet that was catabolized into urea nitrogen by dromedary liver. It has been reported that the level of plasma urea is related to the forage intake and consequently the energy and crude protein concentration (Grings *et al*., 1991). In this study, the plasma urea concentration was within the range of previous reports (Aichouni *et*

al.,2013;Hassan *et al*., 2011) but differ from the values obtained by (Osman and Al-Busadah, 2003).

The level of uric acid in both groups was higher than that reported by (Hassan *et al*., 2011) but it was within the normal range.

Logically the obvious increase ($P<0.05$) in the concentration of plasma HDL-Cholesterol in zero browsing group compared with free browsing one, could be attributed to mechanism of HDL-cholesterol to collect cholesterol from blood stream avoiding its precipitation in blood vesicles then causing atherosclerosis . In the present study, the HDL was higher than the values reported by Nazifi *et al* (2000) and lower than that reported by Asadi *et al.* (2008) and Khajeh *et al*. (2008)

In this study, the serum LDL-cholesterol concentration was similar to the values reported by Nazifi *et al*. (2000), and lower than that reported by Asadi *et al*. (2008) and Khajeh *et al.* (2008).

plasma VLDL-cholesterol concentration was higher than the values reported by Nazifi *et al*. (2000) but lower than that reported by Asadi *et al*. (2008) and Khajeh *et al*. (2008).

The observed increase in the concentration of plasma total cholesterol in zero browsing group could be attributed to the high lipid. In this study, the serum cholesterol concentration was similar to the values reported by Nazifi *et al*. (2000) and Mohamed (2008),lower than the values reported by (Al-Busadah, 2007; Al-Sultan, 2003 ; Osman and Al-Busadah, 2003 and Salah El-Din *et al*., 2005) and higher than the values reported by (Aichouni *et al*., 2013; Sazmand *et al*.,2011).

Serum sodium concentration and serum potassium concentration were within the range of values obtained by Al-Busadah, 2007; Mohammed *et al.,* 2007 but lower than the values reported by (Sazmand *et al*.,2011; Osman and Al-Busadah, 2003 and Aichouni *et al.,* 2010).

It appears from Table (4.5) and Figure (4.6) that plasma Glutamate Oxaloacetate Transminase (GOT)and Glutamate Pyruvate Transminase (GPT) assessed in blood serum, as an indicators of liver function showed a significant difference between the two groups. However, GOT and GPT levels were higher in zero browsing group when compared with the free browsing group, but the levels indicate healthy liver functions for such groups. In general, the values recorded for GOT and GPT are within the normal range reported by Fouda (2008), who found values ranging from 24 to 65 and 14 to 37 IU/L for GOT and GPT, respectively, in goats and sheep. The activity of GOT reported in this study were also similar to the values reported for the Arabian dromedary camels by Al-Busadah (2007) and lower than that reported by (Elrayah *et al*.,2012; Osman and Al-Busadah, 2003; Aichouni *et al*., 2010) and higher than that reported by Salah El-Din *et al*. (2005). GOT lacks organ specificity but is present in skeletal muscle, cardiac muscle and liver of large animals and pathological changes in these organs elevate the activity of GOT in the blood (Kaneko 1989). Like other animals the serum level of GPT in conjunction with other enzymes may be useful indicator for hepatic or muscular damage (Kaeneko 1989), but Kerr (1989) considers GPT as non specific index for liver investigations. In the present study the GPT activity was similar in both groups and in the range of the result of (Hassan *et al.,* 2011;Osman and Al-Busadah, 2003; Sazmand *et al*., 2013) but higher than the values obtained by (Salah El- Din *et al*.,2005),Al-Busada, 2007, Aichouni *et al*.,2010 and Elrayah *et al.*,2012). In this study the serum cholesterol had significant correlations with triglycerides, HDL- and LDL-cholesterol. However, triglycerides had significant correlations with VLDL-cholesterol and HDL-cholesterol, these are similar to results obtained by (Nazifi *et al*., 2009) who found the same correlations and results in

camels. Also it agree with Khoshvaghti *et al.* (2012) who reported that the serum cholesterol had significant correlations with the HDL- and VLDL-cholesterol. However, there were significant correlations between the HDL-, LDL-, VLDL-cholesterol and total lipids in gray necked ostriches. In this study, HDL-cholesterol was negatively correlated with triglycerides, while total cholesterol positively correlated with LDL-cholesterol, these were within the range of Khajeh *et al.* (2008) who found negative correlation between triglyceride and HDL.

In the present study, by using the technique of SDS-PAGE, HDL-cholesterol in figure (4.15) showed bands at molecular weight 145 K.D and clear one at 62KD in both groups. While there was a band at the range of 27KD in lane 1, 2 (zero browsing group). Whereas, the LDL-cholesterol constants in the two groups as shown in figure (4.16) and table (4.13), more abundant bands appears approximately with molecular weight 36 K.D. Also there are bands at51, 29 and 22 KD. These results were in line with (Asadi *et al.* , 2008) who found that the lipoprotein profile in one-humped camels differed substantially from that of other ruminants. when determined the electrophoretic pattern of plasma lipoproteins in dromedary camels. Results may be useful in the evaluation of metabolic disorders in camels. While lipoproteins from liver homogenate in figure (4.17) showed dense bands at molecular weight 175,95,51 in both group, and sharp band at 10KD in zero browsing camels.

The obtained results presented in figure (4.13) and figure (4.14) for chromatographic fraction of proteins of zero browsing and the free browsing camels on sephadex G-200, showed that no difference between the zero browsing and the free browsing groups except the long shoulder peak in range between 25 – 30 fractions that was noticed in the zero browsing group. This might be interpreted as a result of high

concentration of low molecular weight protein in plasma of zero browsing animals when compared with the free browsing.

The obtained results presented in figure (4.18) showed a band at 125 bp this specific band indicates the presence of the cDNA corresponding to ApoB gene in our samples (with reference to primer design) with higher rate of expression in the hepatic tissue (lane 5 when compared to others). Other tissues, however, like muscle tissues (lanes 3, 4, 7) showed ApoB lipoprotein gene down regulation as revealed by low rate of its mRNA expression that gave low concentration of its corresponding cDNA in their corresponding lanes. These results were in agreement with (Mehrabian *et al.*, 1985) who found that apoB with an exceptionally large 20 kb mRNA that is present in liver and intestine but not other tissues examined is consistent with the distribution expected from protein biosynthetic studies. The properties of the mRNA have implications for the biogenesis of the multiple apoB molecular weight forms secreted by liver and intestine.

Conclusion:

1- The values recorded for biochemical parameters were within the ranges reported for camels in Sudan. We concluded that complete diets improve the nutritive value for the animal. Furthermore, crushed sorghum which was added to the diet to a level of 50% without negative effect, but good results was obtained at level of 16% of crude protein.

2- The variations between the findings of workers may be attributed to the breed differences, nutrition, and husbandry or assay methodology. Findings of the current study provide baseline values that

may be used by clinicians for the Sudanese camels. Also the data obtained could be used as a base- line data for fattening camels.

Recommendations:

Further research is required to study the influence of the diet on the expression of lipid carrier protein in camels which have great lipid adaptation.

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Appendix 1

Arabi Pack Type . Rezaigat"UmGallol" (Dar Fur)

Appendix 2

Arabi Pack Type . Lahawi (Butana)

Appendix 3

Sephadex G-200 (gel filtration)

Appendix 4

SDS-polyacrylamide gel electrophoresis