CHAPTER ONE
INTRODUCTION

1.1 Background

The one-humped camel (*Camelus dromedarius*), and the two-humped camel (*C. bactrianus*) from which it is thought to have evolved, are the two species comprising the genus Camelus. Together with the llamoids of South America they are members of the Camelidae family, believed to have evolved from the Protylopus which occupied the North American continent over 1 million years ago (Mobarak *et al.*, 1990). The dromedary was probably first domesticated in southern Arabia around 3000 B.C. Of the estimated 17 million camels of the world, 15 million are one-humped, and the vast majority of these (12 million) are found in Africa, especially in the five neighbouring East African countries of Somalia (5.4 million), Sudan (2.9 million), Djibouti (0.4 million), Ethiopia (0.9 million) and Kenya (0.5 million). The rest are mainly found in Asia. Camel populations are increasing only slightly, and in a few areas, such as Northern Kenya and West African countries, the numbers are actually declining, since camels in pastoral herds are being replaced by other livestock species (Mahmoud, 2006).

When there is damage to the smooth, skeletal or heart musculature, the level of some enzymes in the serum as creatine kinase (CK), lactic dehydrogenase (LDH) and a spartate transaminase (AST) are elevated. These diagnostic enzymes are therefore valuable tools used in the early detection of muscle wastage as a result of ischemia, injury or inflammation (Sacher *et al.*, 1991). There are a considerable number of pathological conditions, which affect the major blood vessels in humans and animals. In addition to the tissue enzymes, experimental work has shown that large

molecules in blood plasma can enter the vessel wall through endothelial cells, presumably by pinocytosis. Similarly, studies have shown that smaller molecules may enter the arterial wall by passing between endothelial cells at cell junctions.

The study of blood constituents can provide valuable information about the general health of an animal and, therefore, can be used for evaluating the health status of the animal. Observation of a deviation of certain blood parameters from their normal limits could be a guide for diagnosis or differential diagnosis of a disease condition. A number of tissue enzymes are a valuable tool as diagnostic agents. Alkaline phosphatase (ALP), alanine transaminase (ALT), aspartate transaminase (AST), creatine kinase (CK), glutamyl transpeptidase (GGP), lactate dehydrogenase (LDH), in addition to minerals are demonstrated to be constituents of some of the major camel blood vessels. Any damage to these blood vessels could result in a significant increase in serum values becoming a valuable diagnostic tool for vascular diseases. Studies in related to measurement of liver enzymes as diagnostic methods in camels are needed.

The objectives of this research were to

- Measure serum biochemical parameters of camels before and after the race and to
- Find out the effect of the race on these chemical blood functions in race camels.

CHAPTER TWO

LITERATURE REVIEW

2.1 Camel (*Camelus dromedarius*)

The Camelidae are very distinct from other families in it includes the Bactrian Camel (*Camelus bactrianus*), the Dromedary (*Camelus dromedarius*), Guanaco (*Lama guanicoe*), Llama (*Lama glama*), Viçuna (*Vicugna vicugna*), and Alpaca (*Vicugna pacos*) (Franklin, 2011). Around 95% of the camelids in the Old World are Dromedaries, with nearly all of the remaining 5% being domestic Bactrian Camels (there are no more than a few thousand wild Bactrian Camels). In the New World, around 47% of camelids are Llamas, 41% Alpacas, 8% Guanacos, and 4% Viçunas (Franklin, 2011).

After the New anatomical and DNA evidence on the relationship between Artiodactyla (even-toed ungulates like Camelids) and Cetacea (whales and dolphins) recently led to a merging of the two orders into a new group, Cetartiodactyla as reviewed by Kulemzina, (2009). Following this findings, experts had not agreed on whether to define Cetartiodactyla as an official taxonomic order that would replace Artiodactyla and Cetacea. Some continue to list camels in the order Artiodactyla (Franklin, 2011) or use the term Cetartiodactyla without defining it as an order (IUCN, 2008).

Camelids originated in North America during Eocene, 40-45 million years ago and become extinct later from their original places (Cui *et al.*, 2007). Camels that would give rise to the dromedary and Bactrian camel spread into Asia, across Europe, and as far west as Spain. Camelids would give rise to the current day South American species spread south across the Panamanian Land Bridge. Because of their ability to thrive under tough

conditions of extreme temperature and scarce food and water, domesticated Camelids have been extremely important to the development of human cultures in the steppes of Eurasia, the deserts of Africa, and the arid Andes of South America. Earliest Camelids were similar to modern guanaco but rabbit-sized (30 cm at shoulder). All Camelids are diurnal and are adapted to harsh and dry climates and are all highly social (Franklin, 2011).

There is unclear evolutionary relationship between *C. bactrianus, C. dromedarius,* and *C. ferus*. However, DNA studies show wild Bactrian camel is not the ancestor of two domesticated species as previously thought and *C. ferus* is separate lineage and not direct progenitor of *C. bactrianus* (Ji *et al*., 2009, Silbermayr, 2009) but previously, they were considered to be a sub-species, *C. bactrianus ferus* (Grubb, 2005). Dromedary camels, Bactrian camels, llamas, and alpacas all have 74 chromosomes (2x36 = 72 autosomes and one pair of sex chromosomes), and they are phylogenetically related to each other while the wild camel has 77 chromosomes (Potts 2004; Hare, 2008). Similarly, there is no plausible evidence to confirm Bactrian camels as ancestor of dromedaries (Kinne *et al*., 2010). According to FAO (2008) report, currently there are about 97 dromedary and 16 Bactrian camel breeds all over the world.

Dromedary (*Camelus dromedarius*) also named as one-humped/Arabian camel is associated with East and northern Africa as well as southwestern Asia. It belongs to the domain Eukarya, kingdom of Animalia, phylum of Chordate, class Mammalia, order Artiodactyla, family Camelidae, Genus Camelus and species of dromedarius. This camel is entirely domesticated (except for a free-ranging feral population in central Australia introduced in the late 1800s). The Dromedary was domesticated around 4000-5000 years ago; the wild form is believed to have gone extinct by 2000- 5000 years ago. In addition to their natural migration routes, archeological finds

indicate that both the dromedary and domestic Bactrian camel were imported into Northern provinces of the Roman Empire for military and civilian use (Pigière and Henrotay, 2012).

The Food and Agriculture Organization (FAO) estimates the total population of camel in the world today to be 25.89 million, of which 89% are one-humped dromedary camels and the remaining 11% are the two-humped (*Camelus bactrianus*) generally found in the cold deserts of Asia. Over 80% of the world's camel population is found in Africa with the highest concentration in North East Africa which accounts for 63% of the world camel population. Ethiopia is estimated to be the third largest camel herd in the world after Somalia, and Sudan (FAO, 2013).

2.2 special adaptive features of *Camelus dromedarius*

Dromedary camels are known by their peculiarity features and in this section the main special anatomy, physiology and behavioral adaptation mechanisms helping them to live with the hostile desert environment are entertained in brief and comprehensive manner. Series of distinctive anatomical and physiological adaptations help them to survive a long period of time without feed and water. Dromedary camels are hardy desert animal are famous for their exceptional tolerance of heat and deprivation of water or feed in arid climate (Farah *et al.*, 2004). The remarkable survival and performance in the desert attribute results from a very low basal metabolism and exceptional water-conserving adaptations.

The camels' thick coats insulate them from the intense heat radiated from desert sand and during the summer the coat becomes lighter in color, reflecting light as well as helping avoid sunburn. Dromedaries have a pad of thick tissue over the sternum called the pedestal. When the animal lies

down, the pedestal and other small areas of padded contact points on with the ground their legs raise the body from the hot surface and allow cooling air to pass under the body. As behavioral adaptation to the hot sand they face towards the sun while resting on the desert sand in a stream lined state to minimize the exposed parts of their body to the sun (Melaku, 2014).

The long legs they have are typically useful for them to keep far high from the hot sand in the desert and allow cold air to move in the under parts of the belly as cooling mechanism. Their large broad ‘elastic’ pads with two finger nail-like toenails on front are also important structures to easily walk on the desert sand which is not possible for other ungulates walk on tips of hoof-covered toes. The advantage of this broad leathery pad in camels is to disperse their weight in a wider surface area and their feet don’t sink in the loose sandy soil (Melaku, 2014).

Hump is another anatomical structure composed of fibrous tissue and fat but not water reservoir at all. The size and shape of the hump signifies availability of nutrition almost disappears during starvation. When the animal is well fed, the hump is plump and erect, but in starved animals it shrinks or flops to one side. The large fat reservoir in the hump sustains camels through times of food scarcity. When the fat is metabolized, it acts as a source of energy and water as every kilo gram of fat mobilization produces one milliliter of water. The other advantage of hump in the dromedary camel is to concentrate body fat in small area and minimizes its presence throughout the rest of body which indirectly reduces heat-trapping that occurs with insulating layers of fat in the hot climate (Melaku, 2014).

The Slit-like closable nostril protects against blowing sand and moistens air on its way to the lungs. When camel exhales water vapors becomes

trapped in their nostrils and is reabsorbed into the body to conserve water. Their eyes are also equipped with protective bony arch and long eyelashes to safeguard from sun shield and frequent blowing sand in the desert respectively. If sand gets lodged in their eyes, they can dislodge it using their transparent third eyelid (nictitating membrane) (Butler and William, 2005). Split upper lip assists feed selection and easy prehension during browsing and their mouths have a thick leathery lining, allowing them to chew thorny desert plants. The small wound ears covered with tufts of hair is protected from entering of the blowing sand.

The oval red blood cells in dromedary camels can easily flow quicker in a dehydrated state of the animal as compared to the round shaped red blood cells in other mammals. These red blood cells are also enormously expansible. In sections they appear similar to sickle cells of humans. In view of the peculiar nature of the red blood cells of the Camelidae, the study by Warda & Zeisig (2000) is especially interesting. They examined the composition of the dromedary red cell membranes. There was 28.8% sphingomyelin, 12% phophatidylcholine, and phosphatidylserine each. Because of the shorter and less saturated fatty acid chains that they identified, the dromedary red cell membranes are more fluid than those of human red cells and perhaps this explains the remarkable stretching ability in camels. The ellipsoid shape of camel erythrocytes is very stable and that the cytoskeleton differs from that of human red cells and they may expand with distilled water to 400% before they rupture (Omorphos et al., 1989).

The digestive and urinary tracts are well specialized in water conservation. Cattle lose 20 to 40 liters of fluid daily through feces, whereas camels lose only 1.3 liters. This is one of the primary methods for resisting water deprivation in the desert. Fluid is absorbed in the end part of the intestines, where the small fecal balls are produced (Breulmann et al., 2007). The long

loops of Henle, which are four to six times longer than in cattle, have the function of both, concentrating urine and reducing its flow. A dehydrated camel urinates only drops of concentrated urine being shown by white stripes of salt crystals on the hind legs and tail. This concentrated urine not only serves to conserve water, but also allows camels to drink water which is more concentrated than sea water (above 3% NaCl), and to eat salty plants (halophytes) that would otherwise be toxic. The body of camels can tolerate loss of water over 30% of body weight whereas most mammals die if they lose half of this value (Franklin, 2011). As a means of water conservation strategy, dromedary camels have very few (25%) of number of sweat glands in cattle and they can only start to sweat at very high temperature.

The large camel nasal surface absorbs the vapor and cools a network of small blood vessels, named the 'carotid rate'. This carotid vessel network surrounds the jugular vein and cools its blood. On the way to the heart the cooled venous blood meets the warm arterial blood going to the brain and eyes, cooling it by more than 4 ºC. .Brain cooling in camels at their rete mirabile, a complex of veins lying very close to the carotid artery utilizes countercurrent blood flow to cool blood flowing to the brain and eyes. This protects their brain from heat damage as it is the first organ affected by high temperature (Schmidt-Nielsen, 1998).

2.3 Physiology of camel

Changes in the environmental factors were found to exert pronounced effects on the blood characteristics to maintain the animal health and help animal to survive the adverse conditions (Al-Arfaj, *et al.* 1992). Camels physiology was different in many aspects when compared to other mammals, which help them to survive and flourish under drastic conditions

of harsh environments and fluctuating nutritional conditions where other species can not exist (Badawy, *et al.* 2008).

Blood is an index for several metabolic processes of the body, so differential concentrations and periodic change of blood metabolites may determine the genetic potential of a species. Plasma biochemical parameters can provide valuable information regarding health, sex, age, nutritional and physiological status of the animals (Osman and Al-Busadah, 2003).

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, sheep, cow, horse, pony, reindeer calf, cheetah and camel in various physiological conditions have been reported (Nazifi et al., 2003).

The serum cholesterol level generally varies inversely with thyroid activity (Gueorguieva and Gueorguiev, 1997; Bruss, 2008), but there are some contradictory findings regarding the relation between serum thyroid hormones and cholesterol and triglycerides, and in camels and goats the concentrations of thyroid hormones were not correlated with cholesterol levels (Nazifi et al., 2002).

2.4 Liver enzymes and diagnostic significance in camels

The physiological functional reserve and regenerative capacity of liver can be lost by diseases affecting liver. Camel liver infection is an imperative disease that leads to great losses to camel production and condemnation of large numbers of livers in slaughter houses. A number of diseases can affect camel liver but toxic substances, infectious diseases, parasitic

hepatitis, fatty liver, and tumors are considered as the usual causes. Clinical signs and pathological lesions of liver diseases are usually nonspecific hence, the disease is frustrating to diagnose and often difficult to treat. It is tricky to know the primary cause and even to differentiate the location of liver pathology (hepatic vs pre/post -hepatic) (Belina, *et al.* 2015). Hence its tentative clinical diagnosis should be confirmed by special techniques and diagnostic approaches such as necropsy, histopathology, serum liver enzyme (ALT, AST, GGT and ALP) tests, molecular pathology tests like IHC, PCR and in situ hybridization, and diagnostic cytology. Molecular pathology tests are employed in specific antigen detection, nucleic acid amplification and localization of cells containing specific nucleic acid sequences**.**

As in other domestic animals in dromedary camels liver is kept in position by the pressure of the neighboring organs and two groups of ligaments, a visceral group and a parietal group (Siddig, 2002). The liver possesses considerable functional reserve and regenerative capacity. In healthy animals including camels more than two thirds of the hepatic parenchyma can be removed without significant impairment of hepatic function and normal hepatic mass can be regenerated in a matter of days (Radostits, 2000).

Hepatocellular injury is one of the pathologic condition affecting domestic animals including camels. Liver infection is an imperative disease that affects all kinds of meat producing animals, leading to great losses to livestock production and national income due to condemnation of livers in the slaughter houses as it represents 2.8% to 5.7% of the dressed carcass weight (Sohair and Eman, 2009). The Influx of acute or chronic inflammatory cells in to the liver is termed hepatitis. Inflammatory cells may be limited to the sites of entry (portal tracts) or spill over into the

parenchyma and intracellular enzymes escape (Talukder, 2001). Besides this, infected liver constitute a good media for bacterial multiplication, transportation of microorganisms with the parasites occurs during the different stages of its life cycle. Anaerobic necrotic lesions of the liver produced by immature flukes occasionally provide a suitable environment for the germination of spores of Cl. novyi type B bacteria in the liver. The bacteria will release toxins into the blood stream resulting in what is known as black disease in camels (Sohair and Eman, 2009).The clinical signs of liver disease are usually non-specific and include anorexia, depression, weight loss and vomiting. Jaundice, when present, may assist the clinician to localize the disease process to the hepatobiliary system (provided that haemolytic anaemia is not the cause). However, jaundice is not always present in animals with liver disease and a variety of different etiologies can cause hepatobiliary disease and jaundice. Other clinical signs include: fever, abdominal effusion and CNS signs (Maddison, 2006).

The causes of liver disease in camel are numerous but primarily liver is affected by: Toxic substances, Infectious diseases, parasitic hepatitis, Fatty liver, Tumors and etc. It is difficult to examine liver because of its location in the cranial abdomen, and its` obvious malfunction occurs only after the liver has lost approximately two thirds of its functional capacity. Therefore, hepatic failure is seen only when there is extensive damage to the hepatic parenchyma or there is obstruction to biliary drainage. According to Green and Flamm (2002) most hepatitis allows escape of intracellular enzymes into the blood stream as the damage enhances permeability of membranes of the liver cells. The major intracellular enzymes involved in camels are alanine aminotransferase (ALT) and aspartate aminotransferase (AST). ALT is found primarily in the liver with a small amount in muscle. Its highest cellular concentration occur in the cytosol; therefore, the enzyme is

released following acute and diffuse hepatocellular damage (Maddison, 2006); whereas the AST is present in both the mitochondria and cytosol of hepatocytes. The cytosolic and mitochondrial forms of AST are true isoenzymes and immunologically distinct. On the other hand, Cholestasis (e.g., biliary obstruction or hepatic infiltration) obstructed bile ducts cause the induction of synthesis of alkaline phosphatase (ALP) and gammaglutamyltranspeptidase (GGT).

Abnormal liver tests may indicate an abnormality of the liver and provide clues as to the nature of the problem. However, in an asymptomatic diseased animal, mild abnormalities may not be clinically significant. A systematic approach in evaluating the animal and ordering further tests will help to identify underlying disease (Howard et al., 1998).

Liver performs different kinds of biochemical, synthetic and excretory functions, so no single biochemical test can detect the global functions of liver. All laboratories usually employ a battery of tests for initial detection and management of liver diseases and these tests are frequently termed "Liver function tests", although they are of little value in assessing the liver function per se. The role of specific disease markers, radiological imaging and liver biopsy is not underestimated (Thapa and Walia, 2007). However, liver disease is frustrating to diagnose and often difficult to treat, because it is difficult to differentiate the location of liver pathology (hepatic vs post-hepatic) using a single test, yet such differentiation can be important in deciding the appropriate diagnostic or therapeutic step to take next. Surgical correction of post-hepatic obstruction may be feasible whereas surgery is indicated in primary hepatocellular disease to obtain biopsies. Thus, when liver disease is suspected after a general clinical examination, special techniques and diagnostic approaches such as Necropsy Examination, Clinical pathology tests (total bile acid and liver enzyme),

Molecular pathology tests like Immunohistochemistry (IHC), Polymerase chain reaction (PCR) and In situ Hybridization(ISH), ultrasonography and magnetic resonance image (MRI) incorporation is better to determine further the status of the liver (Ayman, 2008). Diagnostic cytology is also helpful in identification and examination of individual cells involved in camel liver pathology.

2.4.1 Major causes of camel liver pathology

Liver disease is relatively common but often occurs in the absence of specific clinical signs. The liver has great powers of regeneration and more overt clinical signs associated with its failure do not appear until 70-80% of the functional capacity is lost. Obscure signs of liver disease are therefore much more common than overt signs of liver failure (BCMA, 2011). The liver is subject to many of the same pathologic conditions that affect other body organs. The inflammation of the liver refers to us hepatitis, which can be due to reactions to chemical agents, drugs, and toxins; disorders such as autoimmune diseases and infectious mononucleosis that cause secondary hepatitis; and by hepatotropic viruses that primarily affect liver cells or hepatocytes (Hennessy and Porth, 2004). Signs of liver failure include central nervous system disturbances and are usually acute, even if the underlying liver disease develops over a protracted period. This hepatic encephalopathy is associated with toxic blood levels of ammonia and intestinal amines, which would normally be detoxified by the liver (Mudron et al., 2004).

Concerning camel disease, camels are formerly considered resistant to most of the diseases commonly affecting livestock, but as more research is conducted, camels are found to be susceptible to a large number of pathogenic agents which contribute to hepatobiliary disease, such as

hepatic insufficiency. The major liver disease in ruminants that may also affect camel include: Infectious: septic abscesses, Chlamydia, Salmonella, and Listeria spp., tuberculosis, Johne's disease, Parasites: sarcosporidiosis, Fasciola hepatica, Ascaris spp, Metabolic: hepatic lipidosis, immune responses, neoplasms, Toxins: blue-green algae, pyrrolizidine alkaloid containing plants, mycotoxins (moldy hay, moldy tall fescue), cotton seed meal, Klein grass, Chemicals: iron, copper, phosphorus, arsenic, carbon tetrachloride, hexachloroethane, gossypol, cresols, coal tar pitch, nitrite, chlorinated naphthylenes, Drugs: like halothane, Extrahepatic bile duct obstruction: calculi, abscesses, Congenital: hepatic fibrosis, portosystemic venous shunting of blood, and congenital hyperbilirubinemia of Corrydale, and Idiopathic: hepatic fatty cirrhosis (Douglas, 2004).

2.4.2 Toxic substances

Liver is the most common site of toxic injury for two reasons: the liver receives approximately 80% of its blood supply from the portal vein which drains blood from the GI tract. Thus, ingested toxic substances, including plant, fungal, and bacterial products, as well as metals, minerals, and other chemicals that are absorbed into the portal blood, are transported to the liver in high concentrations. Second, the liver possesses the enzymes capable of biotransformation of a variety of endogenous and exogenous substances for elimination from body; this process may also bioactivate some substances to a more toxic form, thereby causing hepatic injury. The common causes of toxic hepatitis in camelides and other animals are either inorganic poisons such as copper (Junge et al., 1989), phosphorus, arsenic, possibly selenium or organic poisons like carbon tetrachloride, hexachloroethane, Gossypol, creosols and coal tar pitch, chlorophorm and some anaesthetic agents (Groom et al., 1995) and copper diethylaminequinoline sulfonate. Poisonous plants include Senecio,

Crotalaria, Panicumeffusumand water-damaged alfalfa hay. Alfatoxin contaminated stored camel food can also be toxic when its concentration is above 2.5 mg/kg of feed and is considered lethal when found in a concentration of 6.2 mg/kg feed (Osman et al., 2004). Ingestion of some insects such as sawfly larvae (Lophyrotomainterrupta) and insecticides can be toxic to the liver (Byars, 2003); as high incidence of toxicity due to Diazinon is reported by Agab (2003), from the misuse of this insecticide by the camel herders through administration via drinking water. Accidental accessibility of the camels to urea fertilizer in the farm as it is obtained for soil fertilization is also a cause for ureal poisoning.

2.4.3 Infectious Hepatitis

Infectious Hepatitis usually caused by infectious agents such as bacteria and viruses (Hennessy and Porth, 2004). Routes of infection into the liver can be haematogenous, direct penetration, and ascending via the biliary system. The most common route is haematogenous because the liver receives both arterial and venous blood. The severity of inflammation is dictated by the nature (acute or chronic) of infectious agents. Among which viral agents such as: infectious canine hepatitis virus, rift valley fever virus are reported in dromedary camels in Sudan, Egypt and Kenya (Ayman, 2008), herpes virus, Wessels born disease virus and infectious equine anaemia virus are also common; bacterial agents: Bacillary haemogloniaemia caused by Cl. haemolyticum, infectious necrotic hepatitis caused by Cl. novyi type B are reported in dromedary camels by Seifert (1992), Tyzzer`s disease caused by Bacillus piliformis, leptospirosis of L. grippotyphosa and liver abscesses caused by Fusobacteriumnecrophorum or Corynebacterium psuedotuberclosis are also recognized to affect camel liver (Rosa et al., 1989). Acute or chronic infectious hepatitis enhances influx of inflammatory cells into the liver and

the inflammatory cells may be limited to the sites of entry (portal tracts) or spill over into the parenchyma. There are two mechanisms of liver injury in viral hepatitis: direct cellular injury and immune responses against viral antigens in infected hepatocytes. On the other hand, a Physiological alteration of hepatocyte by infectious agents has a significant effect on other body systems which may be due to the development of cirrhosis, portal hypertension, liver failure and etc. If infectious hepatitis occurs with fulminant hepatitis, necrosis may wipe out entire lobules or destroy central and midzonal regions sparing periportal region of lobules (Thapa and Walia, 2007).

2.4.4 Parasitic Hepatitis

Parasitic liver affections in meat-producing animals are one of major factors that reduce the national income, either directly through condemnation of the pathologically affected livers, or indirectly by their effect on the animal growth and so its meat production (El-Hallawany and Abdel-Aziz, 2012). Hepatitis caused by migration of helminth larvae such as Ascaris spp, Strongylus spp, Fasciola spp and Schistosoma spp, through the liver is common in domestic animals. The migration of the larvae throughout the hepatic parenchyma causes local tracks of hepatocellular necrosis accompanied by inflammation. The tracks are eventually replaced with connective tissue leading to the production of fibrous scars on the capsular surface. Some Cestode including members of the genus Taennia occur within the hepatobiliary system of domestic animals and may result in hepatic infection (Neil et al., 2010). Hydatid liver disease caused by E. granulosus is also one of the most important liver problems in animals and man worldwide (Belina et al., 2012). For instance, an incidence of 59.8% of hydatidosis is reported in dromedary camels from different parts of Sudan (Omer et al., 2004).The hepatic migration of the immature flukes of

F.hepatica, a trematode commonly found in sheep, cattle and occasionally other species including camleidae, produces hemorrhagic tracks of necrotic liver parenchyma (Sohair and Nasr, 2009). These tracks are grossly visible in heavy infestations usually as dark red. The infective metacercariae usually migrate the liver capsule and hepatic tissue. This migration usually cause direct trauma with hemorrhages, necrosis and subsequent granulation tissue eventually ensuing liver cirrhosis (El-Hallawany and Abdel-Aziz, 2012). A variety of signs can follow this migration like acute peritonitis, hepatic abscesses and bacillary hemoglobinuria resulting from the proliferation of Cl.hemolyticum or Cl. Novyi in the formed necrotic tissue, the so called black liver disease. Mature flukes reside in the larger bile ducts and cause cholangitis or cholangiohepatitis which may lead to stenosis of the duct (Stuart, 2012).

2.4.5 Fatty Liver

The presence of excessive lipid within the liver is termed as fatty liver. Fatty liver occurs when the rate of triglyceride accumulation within hepatocytes exceeds either their rate of metabolic degradation or their release as lipoproteins. Fatty liver is not a specific disease entity but it occurs as a sequel to many perturbations of normal lipid metabolism which can be due to excessive entry of fatty acid into liver, abnormal hepatocyte function, excessive dietary intake of carbohydrate, increased esterification of fatty acids to triglycerides, decreased apoprotein synthesis and subsequent decreased production and release of lipoprotein and impaired secretion of lipoprotein from the liver (Smith, 2002). The other mechanism for this is activation of free radicals. For instance, Carbon tetrachloride (CCl4) is converted to the toxic free radical CCl3, principally in the liver; causing autocatalytic membrane phospholipid peroxidation, with rapid breakdown of the ER. Hence decline in hepatic protein synthesis of

enzymes and plasma proteins; swelling of the smooth ER and dissociation of ribosomes from the smooth ER have occurred. Thus, there is reduced lipid export from the hepatocytes, as a result of their inability to synthesize apoprotein to form complexes with triglycerides and thereby facilitate lipoprotein secretion; the result is the "fatty liver" of CCl4 poisoning (Althnaian, 2013). Liver degenerative changes in camels including cloudy swelling, hydropic degeneration, fatty change and amyloidosis are also described (Tej Singh et al., 2006). Liver diseases such as hepatic lipidosis with biliary hyperplasia, cholangiohepatitis, hepatic necrosis, lymphoplasmacytic cholangitis, pericholangitis, septic phlebitis and hydropic degeneration are notorious in Llamas and alpacas (Ayman, 2008). According to Tornquist et al. (1999), Females are more affected with fatty liver in llamas and alpacas than males, even though the sex distribution is not different from that of the camelid population in the diagnostic laboratory's data base. In view of this author, all affected females are pregnant and lactating in the age of 6 to 10 years and anorexia and recent weight loss are common.

2.4.6 Tumors of the liver

Damage to the cellular genome is a common feature for virtually all neoplasms (tumors), despite the facts that neoplasms arise in a broad variety of tissues and that diverse agents such as viruses, mutagenic chemicals, and radiation induce their outgrowth. The genetic damage produced by carcinogens is believed to be random, and many mutations may be inconsequential (Cullen et al., 2002). Primary hyperplastic and neoplastic proliferation of the hepatobiliary system arise from hepatocytes (e.g. hepatocellular nodular hyperplasia or hepatocellular carcinoma), epithelium of the bile ducts (cholangiocellular hyperplasia or carcinoma) and gall bladder (carcinoma), and mesenchymal elements such as

connective tissue and blood vessels. Multifocal lymphoma is reported in a 7- year old female dromedary camel which evaluated for inappetence, weight loss, polyuria, and polydipsia. Up on IHC staining the neoplastic cells shows uniformly CD3- positive, indicating a T-cell lymphoma (Simmons et al., 2005). On the other hand, in acute and chronic liver diseases there is occasionally neoplastic associated photosensitization. This is an injury of the cutaneous tissues resulting from activation of photodynamic pigments by ultraviolet light present in the sun rays. It is caused by the increased circulating concentration of phylloerythrin, a photodynamic derivative of chlorophyll, which is normally detoxified and excreted by the liver. In addition to this, Cholangiocarcinoma (CC), which is a malignant tumor arising from bile duct epithelium, is described in other domestic animals and recently in camel (Birincioglu et al., 2008). It is more often originates in intra hepatic bile duct epithelium than in extrahepatic bile ducts or the gall bladder (Ayman, 2008). The incidence of CC increases with age and most cases occur in animals over 10 years of age; neither a breed nor sex prevalence is accounted in animals including camel (Bergman, 1985). Intrahepatic CC mainly affects older domestic animals, particularly dogs and cats, though there is a doubt about intrahepatic CC in camel (Ayman, 2008).

2.4.7 Serum liver enzyme test as diagnostic approach

Morphological diagnosis is powerful and allows for the accurate classification and diagnosis of the majority of disease states within pathologically altered tissues. Necropsy may be defined as the systematic examination of an animal carcass aimed to search for lesions. Necropsy provides a firsthand look on what really happened along the course of the disease. It provides an opportunity to examine everything, both inside and outside. Obviously, the necropsy allows a thorough visualization of all

internal organs (Melissa, 2013). Necropsy examination is often performed to determine the cause of an unexpected death. However, a thorough and systematic postmortem examination also used to confirm a clinical diagnosis, identify the etiology of disease process, explain apparent unresponsiveness to treatment or reveal unrecognized disease process. integration of necropsy with clinical signs and laboratory data ultimately enhance the clinician`s understanding of the disease process and sharpen diagnostic skill (Lowenstine, 1986). Liver is the largest visceral organ in the body. To deal with liver necropsy, first examine the intact, and cut surfaces of the liver and note for color, texture, size and consistency. Several slices of the liver is made for closer inspection. By cutting and opening of the gall bladder, quality and color of bile is examined. While evaluating anatomic liver pathology at necropsy, organisms like liver flukes and lesions of different sizes and consistency can be detected (Sohair and Nasr, 2009). For instance, in examination of CC in camel liver, the liver is enlarged, firm, and yellow-greenish. Grayishwhite and centrally depressed multiple lesions are observed on the serosal surface of the liver. Similar lesions may also be appreciated in the cut surface of the liver. They may range from 0.5 to 3.0 cm in diameter and are generally distinct from the hepatic parenchyma (Birincioglu *et al.,* 2008). On the other hand, in liver affected by fascioliasis and secondary bacterial complication the liver is appeared hard, dark and brown in color with presence of multiple soft abscesses (ranged from 3-10mm in diameters) on the liver surface. On cut section, a viscous yellow material oozed from the cut ends. The abscesses may be surrounded by hyperemic zone (Sohair and Nasr, 2009).

Fibrosis and Cirrhosis of liver are the usually findings in abattoir surveys. In the case of fibrosis, fibrous tissue is formed in response to inflammation or direct toxic insult to the liver. The initial stage of fibrosis develops

around portal tracts or the central veins or within the spaces of disse. With continuing fibrosis, liver is subdivided into nodules of regenerating hepatocytes surrounded by scar tissue, termed cirrhosis. Cirrhosis is characterized by: bridging fibrous septa in the form of delicate bands or broad scars replacing multiple adjacent lobules, disruption of architecture of the entire liver and Parenchymal nodules created by regeneration of encircled hepatocytes (Talukder, 2001).

Serum liver enzymes are measured as biochemical parameters to provide information about hepatocellular injury or cholestasis but do not define how much functional liver is present (Douglas, 2004). There are two major categories of liver enzymes: leakage enzymes and cholestatic enzymes. Leakage enzymes are enzymes that leak into the plasma when hepatocyte injury or death occurs and their high activities in serum is an indication of hepatocellular injury. Commonly measured leakage enzymes include: AST, ALT, Sorbitol dehydrogenase (SDH), and Lactate dehydrogenase (LDH). Cholestatic enzymes are those induced by biliary obstruction or hepatic infiltration like ALP and GGT (Salem and Hassan, 2011).

2.4.8 Aminotransferases

Aminotransferases are the most frequently utilized and specific indicators of hepatocellular necrosis. These enzymes are AST-formerly serum glutamate oxaloacetic transaminase, SGOT and ALT-formerly serum glutamic pyruvate transaminase,SGPT catalyze the transfer of α amino acids of aspartate and alanine, respectively to α keto group of ketoglutaric acid (Thapa and Walia, 2007). Of the numerous methods used for measuring their levels, the most specific method couples the formation of pyruvate and oxaloacetate.

ALT is found primarily in the liver with a small amount in muscle. The highest cellular concentrations occur in the cytosol and it is released following acute and diffuse hepatocellular damage as ALT is specific marker of hepatocellular injury in camel, although occasionally severe muscle damage may also raise serum ALT activity (Salem and Hassan, 2011). In general, serum levels are not regarded as significant unless they are at least two to three times above normal. Liver can be regarded as a sympathy organ – it reacts in sympathy whenever any other organ in the body is damaged. Hence, small to moderate increases in ALT may occur in hypoxia, GI disease, sepsis, hyperthyroidism, diabetes mellitus, pancreatitis, hyperadrenocorticism and other non hepatic disorders. ALT may also be moderately increased in animals on anticonvulsant therapy and glucocorticoids and those with biliary stasis (Maddison, 2006).

AST is present in both mitochondria and cytosol of hepatocytes. It also found in tissues like the heart, skeletal muscle, kidney and brain. The cytosolic and mitochondrial forms of AST are true isoenzymes but immunologically distinct (Rosalki and Mcintyre, 1999). Large increases in mitochondrial AST occur in serum after extensive tissue necrosis. AST considers a nonspecific index for camel liver investigations as it elevated in skeletal or cardiac muscle disease as well as in liver disease. Mitochondrial AST is also increased in chronic liver disease (Salem and Hassan, 2011).

According to Tapasya and Chosdol (2007), the activity of ALT and AST in serum at any moment reflects the relative rate at which they enter and leave circulation. In cases of camel hepatitis there is significant increase in the activity of ALT and AST. Significant decrease in A/G ratio due to decrease in serum albumin and increase in serum globulin concentrations with insignificant change in BUN are observed in all cases of hepatic affection

(Salem and Hassan, 2011). Hepatic sinusoids are primary site for clearance aminotransferases, so they are absent in urine or bile.

2.4.9 Cholestatic enzymes

Cholestatic enzymes are enzymes for which their synthesis increased as a result of bile retention, biliary epithelium damage or administration of drugs. Bile retention usually results from intrahepatic or extrahepatic bile duct obstruction. For diagnostic purpose commonly measured cholestatic enzymes are ALP and GGT (Douglas, 2004). In normal condition average values of ALP vary with age and are relatively high in neonatal and at puberty but lower in middle age and higher again in old age in small animals. However, Serum ALP concentration is higher in lactating sows than their suckling and weaning calves though some controversy exist between different scholars (Omer *et al.*, 2007). ALP is a family of zinc metaloenzymes, with a serine at the active center and not liver specific. ALP isoenzymes are present in all tissues with high activity in liver, bone, kidney, intestine, and placenta. However, the highest level of ALP occurs in cholestatic disorders. Elevation occurs as a result of both intrahepatic and extrahepatic obstruction to bile flow. Its degree of elevation does not help to distinguish between the two though ALP levels are likely to be very high in EHBA.

The mechanism by which ALP reaches the circulation is uncertain; leakage from the bile canaliculi into hepatic sinusoids may result from leaky tight junctions and the other hypothesis is that the damaged liver fails to excrete ALP made in bone, intestine and liver (Thapa and Walia, 2007).

In acute viral hepatitis, ALP is usually either normal or moderately increased. Tumors may secrete ALP into plasma. Hepatic and bony

metastasis can also cause elevated levels of ALP. Other camel liver diseases like infiltrative liver diseases, cholestasis, abscesses, granulomatous, hepatic necrosis and amyloidosis increase serum activity of ALP (Salem and Hassan, 2011). Mildly elevated levels of ALP may be seen in cirrhosis and hepatitis of congestive cardiac failure. Even though it has not been well diagnosed in camel, corticosteroids and drugs like cimetidine, frusemide, phenobarbitone and phenytoin induce a marked increase in a specific hepatic isoenzyme in dogs (Douglas, 2004). Low levels of ALP occur in hypothyroidism, pernicious anemia, zinc deficiency and congenital hypophosphatasia and wilson's disease. When wilson's disease complicated by hemolysis the ratio of ALP and bilirubin declines. This might be the result of replacement of cofactor zinc by copper and subsequent inactivation of ALP. Regardless of the cause of acute hepatic failure a low ratio of ALP to bilirubin is associated with a poor prognosis (Thapa and Walia, 2007). GGT is a membrane bound glycoprotein which catalyses the transfer of gammaglutamyl group to other peptides, amino acids and water. Large amounts of GGT are found in the kidneys, pancreas, liver, intestine and prostate. In liver disease GGT activity correlates well with ALP levels but rarely the GGT levels may be normal in intra hepatic cholestasis as in some circumstances like familial intrahepatic cholestasis. In EHBA GGT is markedly elevated and in the case of hepatic lipidosis of camel GGT is only substantially increased when concurrent conditions such as pancreatitis and cholangiohepatitis are present (Salem and Hassan, 2011). Clinicians may faced a dilemma when they see elevated levels of ALP to differentiate between liver diseases and bone disorders; in such situations measurement of GGT is helpful as GGT is raised in cholestatic disorders and not in bone diseases (Rosalki and Mcintyre, 1999).

2.5 Total protein in camels and associated factors

Proteins are complex nitrogen containing organic compounds found in all animals and vegetable cells, where they constitute a major part of living protoplasm. All enzymes and many hormones that regulate biochemical reactions are functional proteins. In Indian camels the total protein and different fractions of proteins were reported by Kumar *et al*. (2006). Total serum protein was 6.40 ± 0.55 g/dl (range 5.6 - 7.3) in adult camels. The total protein levels in camels ranged from 6.19 to 8.02 g/dl (Bansal et al., 2010).

The variation in the serum protein due to age, sex, pregnancy, rut and infections was studied by Bhargava *et al*. (2004). They reported 3.0 ± 0.07, 6.6 ± 0.18, 6.2 ± 0.16, 5.9 ± 0.25 and 6.1 ± 0.24 g/dl total protein in camel calves of two years age, adult males in rut, adult non-rut males, non-pregnant and pregnant females, respectively. Koudier *et al.* (2008) also estimated total protein in the blood plasma of camels over one calendar year and reported it to be 7.8±0.61 g/dl during breeding season (October - January) and 6.53 ± 0.44 g/dl in quiescent period (February - September). The total protein in different age groups was estimated by Ghodsian *et al*. (2011). These workers reported total protein in the camel calves younger than one year, from one to five years and older than five years of age. The total protein concentration ranged from 5.5 to 8.0, 5.1 to 8.4 and 5.8 to 9.3 g/dl, respectively. The average protein level was 6.8 g percent in all age groups.

Total protein concentration in different age groups was reported in Indian camels by NRCC (2005). According to these reports the averages for total serum protein were 7.48, 7.42, 8.01, 8.76 and 7.84 g/dl in camels from 6 months to 1 year, 1 to 2 years, 2 to 3 years, 3 to 4 years and older than 4

years, respectively. In age groups of < 4, 4-10 and > 10 years, Kataria et al. (2012) reported total protein levels of 8.10 ± 0.19, 7.65 ± 0.15 and 6.86 ± 0.11 g/dl, respectively. The mean values according to age groups were significantly different from each other. It was highest in young animals below 4 years of age and then gradually declined as the age advanced.

Chartier *et al.* (2006) divided 132 samples from camels in 6 groups based on age and sex and the average serum proteins for camels younger than 1 year were 63.4 g/l and 63.l g/l for males and females respectively; and for camels 1-7 years old were 81.9 g/l and 82.7 g/l for males and females respectively; and for camels older than 7 years were 81.4 g/l and 80.7 g/l for males and females respectively. These workers observed that the serum protein level was significantly low in animals below one year of age. However, within age groups the protein levels were not affected by the sex. Chiericato *et al.* (2013) also reported similar results.

Mehrotra and Gupta (2009) studied seasonal variations in certain blood constituents in camels and found protein concentration as 6.8, 4.2, 6.1, 8.1, 6.8, 10.7, 9.0, 10.5, 10.4, 10.1, 9.6 and 7.7 g/dl from January to December, respectively. The protein concentration was relatively higher in the rainy season. Mohamed *et al.* (2010) studied diurnal variation in blood levels of some haematochemical and hormonal parameters in the grazing dromedary. Total serum protein concentration was 6.0 ± 0.1 and 6.4 ± 0.2 g/dl in samples collected at 15 and 60 days after the cessation of rainfall, respectively.

2.6 Blood urea nitrogen and creatinine and associated factors

Creatinine and blood urea nitrogen (BUN) are waste products removed from the blood by the kidneys. Creatinine is a breakdown product of

creatinine phosphate in the muscle. Serum creatinine is a marker used for renal function assessment (Kamili *et al.*, 2013; Patel *et al.*, 2013). High BUN usually means that kidney function is less than normal, but other factors may affect the BUN level. Blood urea ranged from 11.8 ± 0.3 (Elias and Yagil, 2004) to 78.12 mg/dl (Azwai *et al.* 2010).

Urea is a product of deamination of protein in liver and creatinine is produced in muscles during creatinine-phosphate usage. Uric acid is produced during Pyrimidine-base metabolism. Both have very high permeability to nephron membrane and their exchange is a simple diffusion in particular to urea. Change in kidney function is an ability of camels. It has shown that they reduce forestomach dilution rate and feed intake with continuous flow of saliva isotonic fluid and buffering capacity after 11 days water deprivation (Von Engelhardt *et al*., 2006). Perhaps high blood urea concentration in camels is related to their powerful kidney Jaber *et al.* (2004) implied that water restriction has a direct effect on blood urea concentration. They implied that water deficiency leads to increased water re-absorption in the distal tubule and particularly the collecting ducts of the nephron, consequently urea re-absorption is expected to increase as it is a highly permeable molecule. In addition, hypovolemia- due to water insufficiency- is expected to cause a decrease in renal blood flow and thus leads to a decreased filtration rate and high blood urea concentration. Alamer (2009) observed lower glomerular filtration rate in goat during hot summer. This cue was increased along with water deprivation for three consecutive days. But after 6 hours of rehydration, goat had urea and creatinine concentration similar to hydration time. In our results, camel had higher urea concentration than sheep. Therefore, the rise in serum urea concentration could be related to the maintenance of renal function at a lower level, which consequently reduces the clearance rate of serum urea.

Elias and Yagil (2004) made a comparison of blood urea in newborn versus their mothers up to 30 days postpartum. In newborn calves they measured the concentration of urea as 16.9±0.4, 10.3±0.6, 9.4±0.5, 7.7 ± 0.5 and 9.1 ± 0.4 mmol/l. In lactating females the urea concentrations were 12.0±0.3, 11.9±0.3, 10.1 ± 0.6, 9.0 ± 0.4 and 6.9 ± 0.3 mmol/l after 0, 7, 14, 21 and 30 days postpartum respectively, while in adult non-lactating camels the urea concentration was reported as 11.8 ± 0.3 mmol/l.

Chiericato *et al.* (2013) reported urea concentration as 39.9 and 36.6 mg/dl in males and females, respectively. They did not observe any sex related difference in urea concentration. The effect of the breeding season on urea concentration was studied by Koudier *et al.* (1988). Blood urea during breeding season was 4.89 ± 0.67 mmol/l while during the non-breeding season it was 6.84 ± 0.87 mmol/l. Azwai *et al*. (1990) analysed 142 blood samples from dromedaries aged 3 months to 25 years belonging to both sexes and reported overall mean concentration of blood urea as 31.72 mg/dl (range 21.82 - 78.12 mg/dl). Mohamed *et al.* (2010) observed urea concentration as 45.7 ± 1.6 and 39.2 ± 1.4 mg/dl on the 15th and 60th day after the cessation of rainfall. The probable cause of the difference in the two periods was attributed to availability and quality of forages.

2.7 Serum mineral contents in camels

Macro and micro minerals are essential elements for animal functioning and health. Trace elements such as cobalt, selenium, Cu, Zn and Fe are integral components of some enzymes and other important biological components. There are some published reports on haematological and biochemical values in camels. These include for example serum mineral values of Sudanese (Damir *et al*., 2008), Saudi Arabian (Al-Shami, 2009), Emirati (Faye *et al*., 2008), and Iranian (Mohri *et al*., 2008). There are also

some reports on the serum mineral values in the Bactrian camel (Wernery *et al*., 1999).

2.7.1 Iron

Iron is important for racing animals because of its role in haemoglobin synthesis in the blood. Fe had a wider range (54-214 μg dL) (Mohamed and Hussein, 1999). Wernery *et al*. (1999) reported a range of 87-135 μg dL^{-1} in 2-12 years old racing camels in the UAE. Osman and Al-Busadah (2003) reported a Fe level of 80 μg dL^{-1} in Saudi camels. Lower values of Fe (46.2 μg dL^{-1}) were reported in Iranian 5 years old female camels (Badiei *et al*., 2006).

There were no differences between values obtained for female camels as reported for non-rutting male camels (Zia-ur-Rahman *et al*., 2007). However, there are some reports that age has an effect on Fe levels in camels. For instance Faye *et al*. (2008) reported that older camels (>8 years) had highest Fe levels (283 μg/100 mL) compared to those of 3-7 years of age. Higher levels of Fe were reported in Bactrian camel (49-57 μg dL^{-1}) as reported mentioned Wernery *et al*. (1999).

2.7.2 Copper

Mohamed (2004) reported value of 57.6-72.4 μg/100 mL in Sudanese adult camels. Serum Cu levels could be quite variable as reported in temperate camels (7-114 μg 100 mL) by Faye *et al*. (1995). Normal Cu levels in ruminants range is 70-140 μg/100 or 11-22 μmol L^{-1} according to Damir *et al*. 2008) and 70-120 μg/100 mL (12-19 μmol L^{-1}) according to Faye *et al*. (2008). Plasma Cu levels were significantly lower in camels (61 μg/100 mL) than cattle (111 μg/100 mL) as reported by Bengoumi *et al*. (1998). Serum Cu levels below 50 μg/100 mL are regarded as Cu deficient. Copper deficiency in camels was also reported in East Africa (Faye and Bengoumi, 1997). Lower values of Cu (20.5 μg dL^{-1}) were reported in Iranian 5 years old female camels (Badiei *et al*., 2006).

Racing camels in Oman are usually kept in confined enclosure and their diet is strictly monitored.

The basic fodder is fresh alfa alfa, which contains higher levels of Cu compared to Rhodesgrass hay (4.6 vs. 2.8 ppm) the other common roughage (Damir *et al*., 2008). It is well established that excess molybdenum, inorganic sulfate, iron or zinc interferes with copper absorption in ruminants (Damir *et al*., 2008). Cupric oxide needle capsules, which are safe and effective compared to mineral blocks (Damir *et al*., 2008) may be a supplement of choice for Cu. There were no differences between values obtained for female camels (Zia-ur-Rahman *et al*., 2007). Also, no sex effects on Cu levels were observed by Bengoumi *et al*. (1995). However, Faye *et al*. (2008) reported that females had higher levels of Cu than males (62 vs. 56.7 μg/100 mL). Sex differences in mineral contents in camels may be more related to physiological status effect rather than sex per se.

2.7.3 Calcium

Calcium values of (3.39-8.9 mEq L^{-1}) were reported by Osman and Al-Busadah (2003) for Saudi camels (9.0 mEq L^{-1}) and Wernery *et al*. (1999) for UAE camels (9.5-11.5 mg dL^{-1}) but higher than the 2.8 mmol L^{-1} reported for Iranian camels (Mohri *et al*., 2008). However, values of a lower range in Saudi camels (7.6-13.1), reported by Al-Busadah, (2007), UAE camels (11.3 mmol L^{-1}; Ayoub and Saleh, 1998) and temperate camels (11.5 mg/100 mL, Faye *et al*., 1995). Nonetheless, extremely lower ranges (1.6-2.8 mmol L^{-1}) were reported by Sackey *et al*., 2007). There were no differences between values obtained for female camels for non-rutting male camels (Zia-ur-Rahman *et al*., 2007).

CHAPTER THREE
MATERIALS AND METHODS

3.1 Study area

Alshahanya region, State of Qatar was the study area.

Al Shahaniya (also *Ash Shahaniya*) is a municipality (3299 km2) in Qatar, (Qatar Statistics Authority, 2015). This area has a camel race tract which is used for international and local camel races; especially of racing camels in the time period of one month

3.2 Animals

Two hundred and sixty four (264) race camels from different ages and sexes (144female/120male) were used in this research. All of them were from Arabia strain hybridized with the Sudanese camels**.**

The camels were kept under reasonable hygienic conditions and veterinary supervision (Fig. 1). The animals were fed on hay and barley. Water was available *ad libitum*.

The camels were fed with concentrate (Super Camel Racing Mix, Baileys Horse Feeds, UK) and hay. Clean water was provided ad libitum.
Animals were fed on a ration of fresh clover, dry clover, barley, camel milk, pre-mix feeds, multi-vitamins and mineral. All camels had free access to drinking water.

3.3 Samples

Samples of blood from jugular vein the amount was 5 ml by 5 ml injection from the race camels into plane tubes (Fig 2a,b). Samples were taken in the early morning before eating and the race and considered as a control. Another 5ml of test blood were collected after the race.

To evaluate the serum biochemical profiles, the blood samples in plain tubes were allowed to clot, and the serum after centrifugation at 3000r/min for 10 min was stored in single test tube at −20°C until processing.

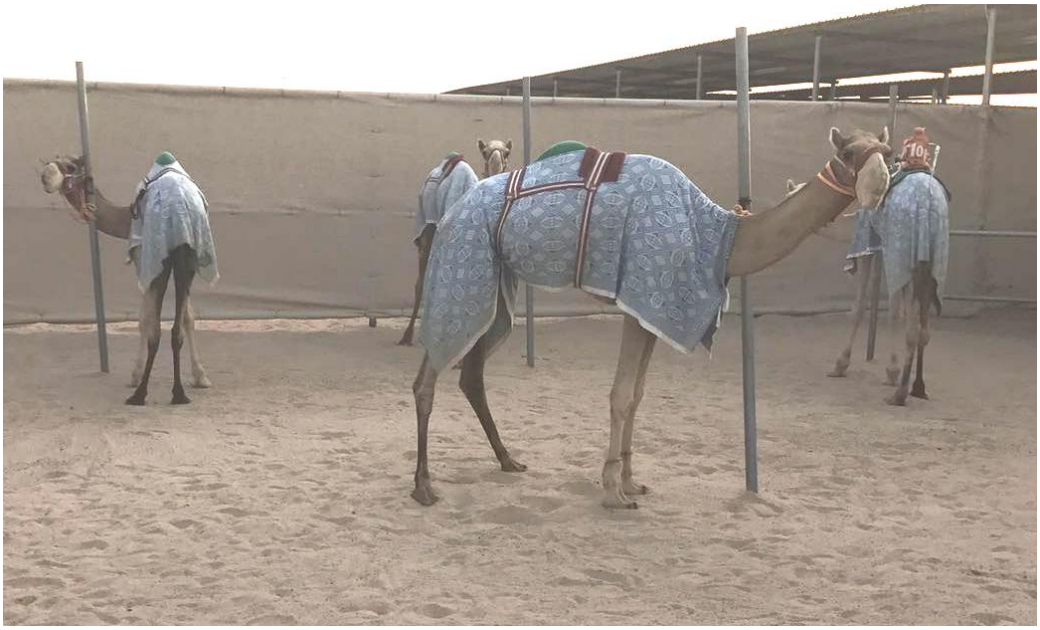

Fig. 1. Healthy racing camels under veterinarian supervision

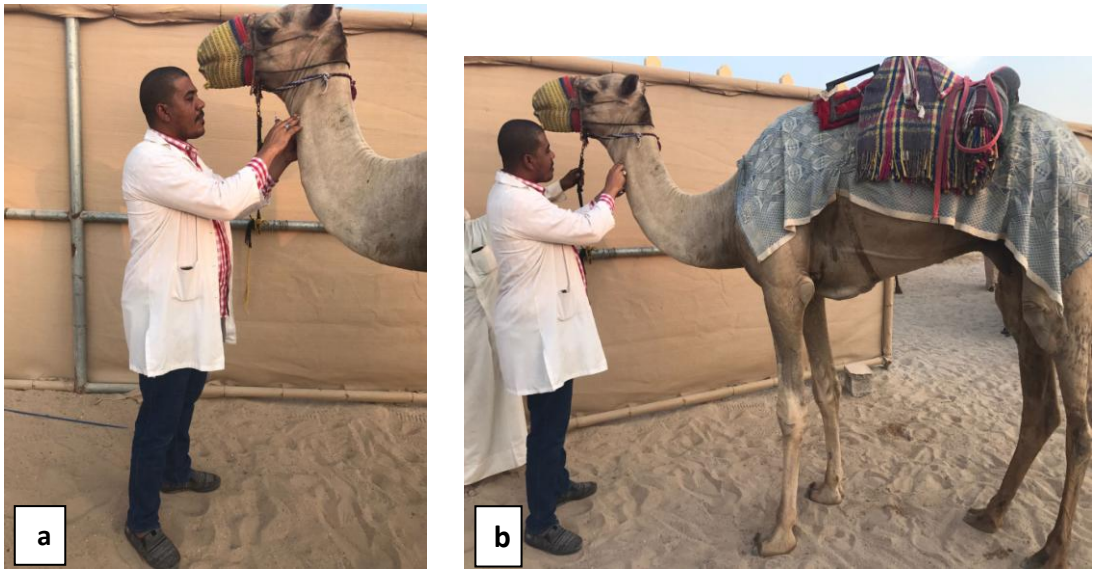

Fig. 2 Samples of blood from jugular vein

3.4. Camel racing

Camel racing is a popular amusement attraction and sport in Qatar. There is a regular flow of camels going to and from the track during the race season. The race distances vary by tournament, but each race not often exceeds five kilometres. However, races in the main festival run over distances of 6, 8 and 10 kilometres.

The first set of camel races begins in October in Al shahaniya, the launching point for a racing season that continues with local tournaments every Friday till February and the big events occur in March and April.

Professional camel racing is a big deal in Qatar and these camels can run at speeds of up to 40 mph in short sprints and can carry on to 30 mph for an hour.

https://www.onlineqatar.com/attractions/Al-Shahaniya-Camel-Racetrack.aspx

In this study the race distance was about 4-5 km in the race tract (Fig. 3a,b).

Fig. 3a.The racing camels at the racing track

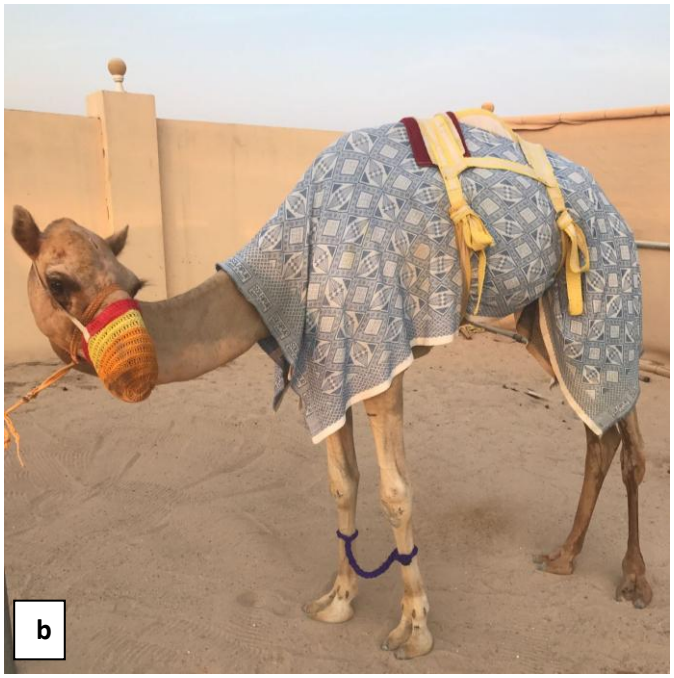

Fig. 3b. A racing camel

3.5 Biochemical analysis of serum samples:

Serum samples were subjected to 11 biochemical tests to assess the effect if any, on the liver and kidneys functions, and effect on minerals level.

The serum was used for determination of serum parameters including total proteins, enzymes and minerals. These serum constituents were analyzed spectrophotometrically by (Alara Chemistry Analyzer, USA) (Fig .4) using commercial reagent kits. The activities of lactate dehydrogenase (LDH), aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP) and gamma-glutamyltransferase (GGT) enzymes were measured. The concentrations of Total protein (TP), creatinine, and urea were measured. Also the serum was used for the determination of some minerals (calcium (Ca), iron (Fe) and copper (Cu)) were measured.

3.6 Statistical analysis

The difference between mean values of data collected were tested by the t-test, the comparisons were made between mean treatment values and baseline values within the same group to eliminate individual variation, differences were considered significant at $P<0.05$ level.

Fig. 4. Alara Chemistry Analyzer, USA

CHAPTER FOUR
RESULTS

Among the whole sample of the camels under the study (n=264), the mean age of the studied camels was 3.3±0.93 years. Total protein, creatinine and urea were 6.2±0.42, 1.4±0.19 and 16.8±2.94 respectively. Liver enzymes ALT, AST, ALP and LDH mean values were 33.9±15.60, 96.8±58.02, 188.9±40.49 and 437.5±103.52 respectively. Mean values of serum minerals; Iron, Calcium and Copper were 91.4±12.93, 10.4±0.41 and 76.1±8.97 (Table 4.1).

Among the male sample of the camels under the study (n=120), the mean age of the studied male camels was 3.6±0.94 years. Total protein, creatine and urea were 6.1±0.45, 1.4±0.19 and 16.8±3.11 respectively. Liver enzymes ALT, AST, ALP and LDH mean values were 32.2±10.65, 92.8±36.45, 189.8±43.62 and 430.6±96.5 respectively. Mean values of serum minerals; Iron, Calcium and Copper were 90.2±13.54, 10.4±0.41 and 75.0±9.14 (Table 4.2).

Among the female sample of the camels under the study (n=144), the mean age of the studied male camels was 3.4±0.93 years. Total protein, creatine and urea were 6.2±0.40, 1.4±0.19 and 16.8±2.81 respectively. Liver enzymes ALT, AST, ALP and LDH mean values were 35.3±18.68, 100.0±71.44, 188.2±37.44 and 443.3±109.27 respectively. Mean values of serum minerals; Iron, Calcium and Copper were 90.2±13.54, 10.4±0.41 and 75.0±9.14 (Table 4.3).

Among the whole sample of the studied camels the positive correlations (indicate significant direct relationship between each two variables) 0.22 and 0.20 were reported between age and total protein and urea respectively.

Negative correlations (indicate significant indirect i.e reverse relationship) -0.19, -0.19, -0.31 and -0.51were reported between age and ALT, AST, ALP and LDH respectively. No significant correlation reported between age of the camel and serum minerals (Fe, Ca and Cu) (Table 4.4).

Among sample of the studied male camels negative correlations (indicate significant indirect i.e reverse relationship) -0.40, -0.28, -0.31 and -0.43 were reported between age and ALT, AST, ALP and LDH respectively. No significant correlation reported between age of the camel and total protein, ceratine, urea and serum minerals (Fe, Ca and Cu) (Table 4.5).

Among the sample of the studied female camels the positive correlations (indicate significant direct relationship between each two variables) 0.30 and 0.31 were reported between age and total protein and urea respectively. Negative correlations (indicate significant indirect i.e reverse relationship) -0.31, and -0.56 were reported between age and ALP and LDH respectively. No significant correlation reported between age of the camel and, creatine, ALT, AST serum minerals (Fe, Ca and Cu) (Table 4.6).

With regard to the total sample, significant differences in total protein, creatine, urea, ALT, ALP, AST and LDH were reported in relation to age of the camel (P value < 0.05), while iron, calcium and copper reported no significant differences with regard to age of the camel (P value > 0.05) (Table 4.7).

Among the control sample of the camels under the study (n=52), the mean age of the studied camels was 2.9±0.59 years. Total protein, creatinine and urea were 6.1±0.41, 1.4±0.16 and 17.0±4.92 respectively. Liver enzymes ALT, AST, ALP and LDH mean values were 38.6±9.87, 80.0±21.11, 177.0±62.23 and 371.9±83.03 respectively. Mean values of serum

minerals; Iron, Calcium and Copper were 105.5±24.41, 10.1±0.49 and 73.1±7.57 respectively (Table 4.8).

Among the control sample of the studied camels the positive correlations (indicate significant direct relationship between each two variables) 0.46 and 0.30 were reported between age and urea and ALT respectively. No significant correlation reported between age of the camel and TP, creatine, AST, ALP and LDH and serum minerals (Fe, Ca and Cu) (Table 4.9).

Among the control sample (males n= 20) of the studied camels the positive correlations (indicate significant direct relationship between each two variables) 36, 0.46 and 0.30 were reported between age and creatine urea and ALT respectively. No significant correlation reported between age of the camel and TP, AST, ALP and LDH and serum minerals (Fe, Ca and Cu) (Table 4.10).

Among the control sample (females n= 32) of the studied camels the positive correlations (indicate significant direct relationship between each two variables) 20, 42, 0.27, 47 and 0.26 were reported between age and TP, creatine urea, ALT, AST and LDH respectively. No significant correlation reported between age of the camel and ALP and serum minerals (Fe, Ca and Cu) (Table 4.11).

Considering the status (before race/after race) significant differences were found the mean values of total protein, iron, creatine, urea, LDH and copper among the control group of camels under the study, and when considering the sex of the camel differences were reported in mean values of iron and copper. No significant differences observed with reference to status (before and after race) and sex of the camel in all blood parameters (Table 4.12).

Table 4.1: Overall mean values of blood parameters the studied camels

	Valid N	Mean	Std. Dev.	Coef. Var.
AGE	264	3.5	0.93	26.9
TP	264	6.2	0.42	6.9
IRON	264	91.4	12.93	14.1
CREATINE	264	1.4	0.19	13.4
UREA	264	16.8	2.94	17.5
ALT	264	33.9	15.60	46.0
AST	264	96.8	58.02	59.9
ALP	264	188.9	40.29	21.3
LDH	264	437.5	103.52	23.7
CALCIUM/ppm	264	10.4	0.41	3.9
COPPER/ Ca mg/dl	264	76.1	8.97	11.8

Table 4.2: Mean values of blood parameters the studied camels (males)

	Valid N	Mean	Std. Dev.	Coef. Var.
AGE	120	3.6	0.94	26.5
TP	120	6.1	0.45	7.3
IRON	120	90.2	13.54	15.0
CREATINE	120	1.4	0.19	13.3
UREA	120	16.8	3.11	18.4
ALT	120	32.2	10.65	33.1
AST	120	92.9	36.45	39.2
ALP	120	189.8	43.62	23.0
LDH	120	430.6	96.15	22.3
CALCIUM	120	10.4	0.41	4.0
COPPER	120	75.0	9.14	12.19

Table 4.3: Mean values of blood parameters the studied camels (females)

	Valid N	Mean	Std. Dev.	Coef. Var.
AGE	144	3.4	0.93	27.2
TP	144	6.2	0.40	6.4
IRON	144	92.4	12.34	13.4
CREATINE	144	1.4	0.19	13.5
UREA	144	16.8	2.81	16.7
ALT	144	35.3	18.68	52.9
AST	144	100.0	71.15	71.2
ALP	144	188.2	37.44	19.9
LDH	144	443.3	109.27	24.7
CALCIUM	144	10.5	0.40	3.8
COPPER	144	77.0	8.75	11.37

Table (4.4) The Overall correlation between mean values of the blood parameters and age of the camel

	AGE	TP	IRON	CREATINE	UREA	ALT	AST	ALP	LDH	CALCIUM	COPPER
AGE	1.00										
TP	0.22	1.00									
IRON	0.02	0.10	1.00								
CREATINE	0.08	0.21	-0.11	1.00							
UREA	0.20	0.21	-0.05	0.54	1.00						
ALT	-0.19	-0.03	0.04	0.02	-0.07	1.00					
AST	-0.19	-0.10	0.04	-0.05	-0.02	0.69	1.00				
ALP	-0.31	-0.29	0.09	-0.05	-0.13	0.16	0.24	1.00			
LDH	-0.51	-0.28	0.01	0.01	-0.09	0.55	0.60	0.41	1.00		
CALCIUM	-0.08	0.19	0.11	0.19	0.02	-0.09	-0.01	0.08	0.02	1.00	
COPPER	0.01	0.19	0.20	-0.06	-0.04	0.10	0.02	0.00	-0.05	0.02	1.00

Table (4.5) The correlation between mean values of the blood parameters and age of the camel (males)

	AGE	TP	IRON	CREATINE	UREA	ALT	AST	ALP	LDH	CALCIUM	COPPER
AGE	1.00										
TP	0.15	1.00									
IRON	-0.01	0.00	1.00								
CREATINE	0.02	0.16	-0.15	1.00							
UREA	0.09	0.16	-0.18	0.57	1.00						
ALT	-0.40	-0.11	0.25	0.20	0.01	1.00					
AST	-0.28	-0.16	0.00	0.16	0.08	0.70	1.00				
ALP	-0.31	-0.30	0.07	0.01	-0.15	0.31	0.32	1.00			
LDH	-0.43	-0.29	0.02	0.03	-0.03	0.60	0.68	0.50	1.00		
CALCIUM	-0.14	0.16	0.07	0.08	0.05	-0.10	-0.09	0.17	0.05	1.00	
COPPER	-0.02	0.25	0.21	-0.02	0.02	-0.12	-0.15	-0.10	-0.16	0.01	1.00

Table (4.6) The correlation between mean values of the blood parameters and age of the camel Females

	AGE	TP	IRON	CREATINE	UREA	ALT	AST	ALP	LDH	CALCIUM	COPPER
AGE	1.00										
TP	0.30	1.00									
IRON	0.07	0.19	1.00								
CREATINE	0.12	0.27	-0.05	1.00							
UREA	0.31	0.27	0.09	0.52	1.00						
ALT	-0.09	-0.02	-0.08	-0.06	-0.11	1.00					
AST	-0.16	-0.10	0.06	-0.15	-0.07	0.69	1.00				
ALP	-0.31	-0.29	0.11	-0.12	-0.11	0.11	0.23	1.00			
LDH	-0.56	-0.30	-0.01	0.00	-0.15	0.54	0.59	0.34	1.00		
CALCIUM	-0.02	0.20	0.13	0.30	-0.02	-0.11	0.01	-0.01	-0.01	1.00	
COPPER	0.05	0.10	0.18	-0.08	-0.09	0.19	0.09	0.11	0.03	0.00	1.00

Table (4.7) Differences in blood parameters of the camels with regard to sex and age (overall)

SEX	F	M	SE	p sex	p age	P interaction						
AGE	2	3	4	5	2	3	4	5				
No.	22	63	37	22	16	43	39	22				
TP	6.19 bc	6.09 c	6.42 a	6.43 a	6.12 bc	6.09 c	6.10 c	6.34 ab	0.077	0.031	0.000	0.069
IRON	93.32 ab	91.49 ab	90.73 ab	97.05 a	87.19 b	92.49 ab	89.36 ab	89.32 ab	2.456	0.042	0.536	0.212
CREATINE	1.41 ab	1.33 b	1.41 ab	1.43 ab	1.48 a	1.36 b	1.41 ab	1.43 ab	0.035	0.360	0.008	0.771
UREA	16.59 bc	15.97 c	16.76 bc	19.27 a	17.38 bc	16.26 bc	16.72 bc	17.82 ab	0.540	0.783	0.000	0.279
ALT	44.86 a	30.95 cd	39.43 abc	31.41 cd	41.06 ab	33.70 bcd	30.05 d	26.50 d	2.844	0.058	0.000	0.061
AST	149.55 a	85.56 c	101.76 bc	88.77 c	124.31 ab	90.60 c	88.38 c	82.68 c	10.608	0.188	0.000	0.487
ALP	207.41 ab	193.24 bc	178.41 cd	170.77 cd	219.81 a	191.09 bc	188.41 bcd	167.82 d	7.360	0.406	0.000	0.598
LDH	599.09 a	435.57 c	411.14 cd	363.59 e	536.56 b	430.74 c	418.62 cd	374.59 de	16.149	0.286	0.000	0.171
CALCIUM	10.50	10.46	10.54	10.41	10.39	10.50	10.40	10.26	0.076	0.100	0.245	0.358
COPPER	78.21	74.98	79.87	76.46	76.46	74.29	75.25	74.79	1.694	0.070	0.133	0.530

Table 4.8: Over all mean values of blood parameters the studied camels control sample

	Valid N	Mean	Std. Dev.	Coef. Var.
AGE	52	2.9	0.59	20.5
TP	52	6.1	0.41	6.8
IRON	52	105.5	24.41	23.1
CREATINE	52	1.4	0.16	11.5
UREA	52	17.0	4.92	29.0
ALT	52	38.6	9.87	25.6
AST	52	80.0	21.11	26.4
ALP	52	177.0	62.23	35.2
LDH	52	371.9	83.03	22.3
CALCIUM	52	10.1	0.49	4.8
COPPER	52	73.1	7.57	10.4

Table (4.9) The correlation between mean values of the blood parameters and age of the camel
Control group n = 52

	AGE	TP	IRON	CREATINE	UREA	ALT	AST	ALP	LDH	CALCIUM	COPPER
AGE	1.00										
TP	0.08	1.00									
IRON	-0.21	0.24	1.00								
CREATINE	0.23	-0.28	-0.18	1.00							
UREA	0.46	-0.26	-0.09	0.55	1.00						
ALT	0.30	-0.16	-0.16	0.46	0.55	1.00					
AST	0.16	-0.09	-0.08	0.52	0.54	0.64	1.00				
ALP	-0.07	-0.17	0.06	0.50	0.36	0.65	0.70	1.00			
LDH	0.09	0.27	0.36	0.11	0.39	0.29	0.42	0.19	1.00		
CALCIUM	-0.06	0.27	0.36	-0.18	0.01	0.05	0.11	-0.13	0.41	1.00	
COPPER	0.01	0.39	0.11	-0.29	0.01	-0.13	0.01	-0.13	0.17	0.23	1.00

Table (4.10) The correlation between mean values of the blood parameters and age of the camel

Control group Males, No = 20

	AGE	TP	IRON	CREATINE	UREA	ALT	AST	ALP	LDH	CALCIUM	COPPER
AGE	1.00										
TP	0.09	1.00									
IRON	-0.05	0.21	1.00								
CREATINE	0.36	-0.51	-0.02	1.00							
UREA	0.62	-0.28	-0.02	0.61	1.00						
ALT	0.30	-0.32	-0.05	0.72	0.76	1.00					
AST	0.07	-0.17	-0.02	0.62	0.53	0.83	1.00				
ALP	-0.08	-0.24	0.05	0.59	0.36	0.72	0.80	1.00			
LDH	0.09	0.36	0.18	-0.07	0.26	0.31	0.36	-0.08	1.00		
CALCIUM	0.00	0.46	0.10	-0.04	-0.08	0.16	0.13	-0.11	0.61	1.00	
COPPER	0.28	0.60	0.00	-0.32	0.11	0.02	-0.01	-0.06	0.22	0.14	1.00

Table (4.11) The correlation between mean values of the blood parameters and age of the camel
Control group Females, No = 32

	AGE	TP	IRON	CREATINE	UREA	ALT	AST	ALP	LDH	CALCIUM	COPPER
AGE	1.00										
TP	0.20	1.00									
IRON	-0.35	0.19	1.00								
CREATINE	0.13	-0.18	-0.34	1.00							
UREA	0.42	-0.32	-0.22	0.49	1.00						
ALT	0.27	0.02	-0.20	0.20	0.41	1.00					
AST	0.47	-0.06	-0.20	0.42	0.56	0.36	1.00				
ALP	-0.01	-0.15	0.06	0.41	0.37	0.57	0.49	1.00			
LDH	0.26	0.17	0.36	0.18	0.43	0.40	0.52	0.47	1.00		
CALCIUM	-0.14	0.13	0.51	-0.32	0.06	-0.08	0.08	-0.18	0.31	1.00	
COPPER	-0.15	0.22	0.02	-0.38	-0.14	-0.21	-0.03	-0.29	0.05	0.27	1.00

Table (4.12) Differences in blood parameters of the camels with regard to sex and age
Control group

	Before race	After race	SE	p status				
	M	F	M	F	p SEX	p status*SEX		
	10	16	10	16				
AGE	3.05	2.81	3.05	2.81	0.171	1.000	0.172	1.000
TP	6.25 a	6.27 a	5.76 b	6.07 a	0.108	0.003	0.128	0.173
IRON	104.20 b	122.25 a	88.70 b	100.00 b	6.181	0.004	0.022	0.588
CREATINE	1.24 b	1.28 b	1.42 a	1.45 a	0.038	0.000	0.380	0.744
UREA	15.90	16.94	16.20	18.25	1.421	0.573	0.283	0.723
ALT	37.90	37.38	42.60	37.75	2.840	0.376	0.349	0.450
AST	73.90	78.56	81.70	84.25	6.103	0.275	0.557	0.863
ALP	153.40	171.19	193.00	187.44	17.796	0.123	0.733	0.515
LDH	377.30 ab	411.88 a	315.90 b	363.63 ab	22.281	0.018	0.071	0.769
CALCIUM	10.19	10.29	9.97	10.03	0.138	0.090	0.567	0.896
COPPER	73.92 a	76.72 a	65.79 b	73.58 a	1.916	0.005	0.008	0.199

CHAPTER FIVE
DISCUSSION

In this study 264 camels were investigated to assess the liver enzymes activities and other blood parameters (iron, copper and calcium, total protein, urea and creatinine) as diagnostic measures in race camels. Over all sample means of liver enzymes ALT, AST, ALP and LDH mean value were 33.9±15.60, 96.8±58.02, 188.9±40.49 and 437.5±103.52 respectively. These values comparable with previous studies, *i.e.* According to Tapasya and Chosdol (2007), the activity of ALT and AST in serum at any moment reflects the relative rate at which they enter and leave circulation. In cases of camel hepatitis there is significant increase in the activity of ALT and AST. Significant decrease in A/G ratio due to decrease in serum albumin and increase in serum globulin concentrations with insignificant change in BUN are observed in all cases of hepatic affection (Salem and Hassan, 2011). Hepatic sinusoids are primary site for clearance aminotransferases, so they are absent in urine or bile.

Considering the control group, liver enzymes ALT, AST, ALP and LDH mean values were 38.6±9.87, 80.0±21.11, 177.0±62.23 and 371.9±83.03 respectively. In normal condition average values of ALP vary with age and are relatively high in neonatal and at puberty but lower in middle age and higher again in old age in small animals. However, Serum ALP concentration is higher in lactating sows than their suckling and weaning calves though some controversy exists between different scholars (Omer *et al.*, 2007).

Considering the status (before race/after race) significant differences were found the mean values of LDH the control group of camels under the study, while other liver enzymes were not significantly different. When

considering the sex of the in addition to status (before and after race) and sex camel, no significant differences observed in all liver enzymes. According to Belina, *et al.* (2015) its tentative clinical diagnosis should be confirmed by special techniques and diagnostic approaches such as necropsy, histopathology, serum liver enzyme (ALT, AST, GGT and ALP) tests, molecular pathology tests like IHC, PCR and in situ hybridization, and diagnostic cytology. Molecular pathology tests are employed in specific antigen detection, nucleic acid amplification and localization of cells containing specific nucleic acid sequences**.**

Total protein, creatinine and urea in the whole sample were 6.2±0.42, 1.4±0.19 and 16.8±2.94 respectively. On the other hand among the control sample of the camels under the study (n=52), total protein, creatinine and urea were 6.1±0.41, 1.4±0.16 and 17.0±4.92 respectively.

In the control group, total protein, creatinine and urea significantly between the measurements before and after the race, but not significantly different when the status (before and after race) and sex of the camel. Studies showed that total protein, creatinine and urea varied according to physiological status of the camel. Bhargava *et al.* (2004) reported 3.0 ± 0.07, 6.6 ± 0.18, 6.2 ± 0.16, 5.9 ± 0.25 and 6.1 ± 0.24 g/dl total protein in camel calves of two years age, adult males in rut, adult non-rut males, non-pregnant and pregnant females, respectively. Koudier *et al.* (2008) also estimated total protein in the blood plasma of camels over one calendar year and reported it to be 7.8±0.61 g/dl during breeding season (October - January) and 6.53 ± 0.44 g/dl in quiescent period (February - September). Chiericato *et al.,* (2013) reported urea concentration as 39.9 and 36.6 mg/dl in males and females, respectively. They did not observe any sex related difference in urea concentration. The effect of the breeding season on urea concentration was studied by Koudier *et al.*, (1988). Blood urea during

breeding season was 4.89 ± 0.67 mmol/l while during the non-breeding season it was 6.84 ± 0.87 mmol/l. Azwai *et al.*, (1990) analysed 142 blood samples from dromedaries aged 3 months to 25 years belonging to both sexes and reported overall mean concentration of blood urea as 31.72 mg/dl (range 21.82 - 78.12 mg/dl).

In the whole sample (n=264) Mean values of serum minerals; Iron, Calcium and Copper were 91.4±12.93, 10.4±0.41 and 76.1±8.97. On the other hand, among the control sample (n=52) the mean values of serum minerals; Iron, Calcium and Copper were 105.5±24.41, 10.1±0.49 and 73.1±7.57 respectively. Moreover, except iron, no significant differences reported in the serum minerals regarding the status and the sex of the camels before and after the race. Wernery *et al*., (1999) reported a range of 87-135 μg dL^{-1} in 2-12 years old racing camels in the UAE. Osman and Al-Busadah (2003) reported a Fe level of 80 μg dL^{-1} in Saudi camels. Lower values of Fe (46.2 μg dL^{-1}) were reported in Iranian 5 years old female camels (Badiei *et al*., 2006). Mohamed (2004) reported value of 57.6-72.4 μg/100 mL in Sudanese adult camels. Serum Cu levels could be quite variable as reported in temperate camels (7-114 μg 100 mL) by Faye *et al*., (1995). Calcium values of (3.39-8.9 mEq L^{-1}) were reported by Osman and Al-Busadah (2003) for Saudi camels (9.0 mEq L^{-1}) and Wernery *et al*., (1999) for UAE camels (9.5-11.5 mg dL^{-1}) but higher than the 2.8 mmol L^{-1} reported for Iranian camels (Mohri *et al*., 2008).

CONCLUSION AND RECOMMENDATIONS

Conclusion

This research study concluded that

Often every parameter is affected by age not sex in the racing camel. Whenever, the greater the lifetime, the lower the creatine and the more urea. Whenever the higher the age, the higher the protein in the sexes.Whenever the greater the age, the lower the enzymes of the liver (AST-ALT-ALP) as well as the muscles. Calcium, iron, muscle enzyme (LDH), protein, creatine are affected by the physiological activity of the race camel. The race reduces the protein, iron, calcium, copper, muscle enzyme (LDH) while increasing the level of keratin.No interaction effect of sex and activity on the examined traits.

Recommendations

- The measurements of liver (AST-ALP-ALT), muscle (LDH), protein and kidney enzymes (creatine) in the racing camel can be used as an indicator of age.
- As for the physiological activity of the race camel, it is explained by the readings of calcium, copper, iron.
- Further lessons are required to document of blood profile to provide information that is considered to be the base information for the correct race camel in the field.

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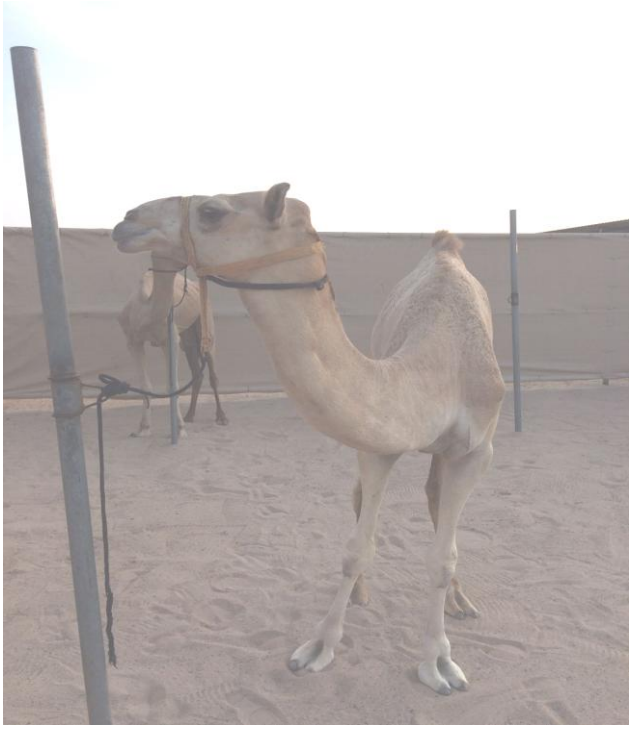
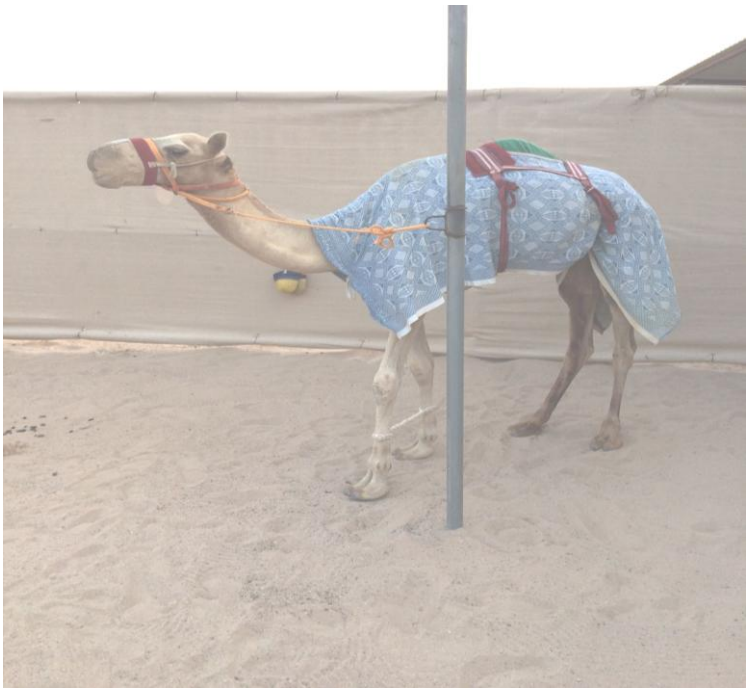
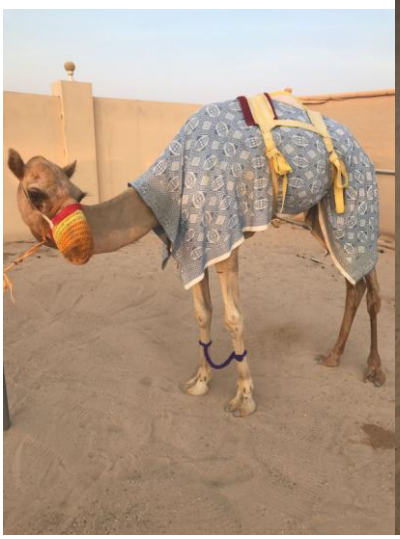
Appendix

https://en.wikipedia.org/wiki/Al-Shahaniya#/media/File:Camel_Race_Track_Qatar.jpg





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