# Iron Deficiency Anemia Symptom's, Causes, Diagnosis & Management of Anemia

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## Abstract.

Iron is essential to all living organisms and is basic to multiple metabolic functions. The most important function of Iron is oxygen transport in hemoglobin. Iron deficiency anemia in Animals is usually caused by chronic blood loss and can be exposed parenthetically as animals may have modified to the Iron Deficiency anemia. Severe iron deficiency is characterized by a microcytic, hypochromic. Which turn to potentially severe anemia with adjustable reformative response. Iron metabolism and homeostasis will be reviewed, followed by a discussion of diagnostic testing and therapeutic recommendations for dogs and cats with iron deficiency anemia. Iron deficiency anemia arises when the balance of iron intake, iron stores, and the body's loss of iron are inadequate to fully support production of erythrocytes. Iron deficiency anemia rarely causes death, but the impact on human health directly. In the European countries, this disease is easily identified and treated. In contrast, it is a health problem that affects major portions of the population in under developed countries viz India, Nepal, and Bangladesh. The prevention and successful treatment for iron deficiency anemia remains sadly insufficient worldwide, especially among underprivileged women and children.

**Key Words:** Iron deficiency anemia.

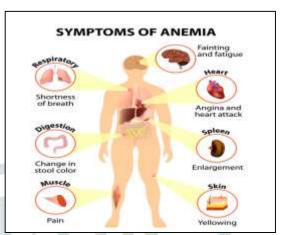
## A. Introduction:

Anemia happens when we do not have enough red blood cells. The cells travel with iron and hemoglobin, which is a protein that helps carry oxygen through the bloodstream to your organs all through the body. When someone develops anemia, they are said to be "anemic." Being anemic might mean that you feel more tired or cold than you usually do, or if your skin seems too pale. This is due to your organs not receiving the oxygen they need to do their jobs. Some people find out they are low in iron when they go to donate blood.

Iron deficiency anemia in Animal most commonly occurs secondary to chronic external blood loss. Treatment consists of addressing the underlying syndrome causing blood

- 1. loss and restoring iron stores.
- 2. Iron metabolism and homeostasis





## **Symptoms of Anemia**

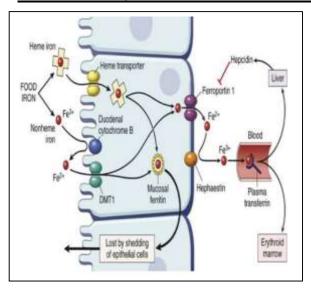
#### **Symptoms of Anemia**

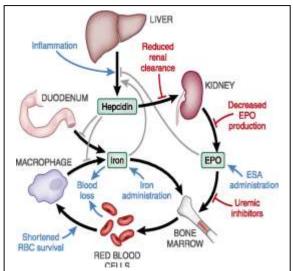
Iron in the form of heme is vital to many metabolic purposes including oxygen transportation in hemoglobin. Iron is also a constituent of multiple enzymes, including cytochromes, necessary for energy generation and drug metabolism. Through the donation or acceptance of an electron, iron exists in either a reduced ferrous (Fe<sup>21</sup>) or an oxidative ferric (Fe<sup>31</sup>) state.

The liver, which is the place of production of iron transport proteins, contains the major non-functional iron stores either as ferritin or hemosiderin. Ferritin is both diffuse and soluble, and is the primary iron storage protein. Hemosiderin is similar in structure, but has more iron relative to protein and is insoluble. Iron is also stored in reticulo endothelial cells of the bone marrow and spleen, but is not commonly stored in the bone marrow of cats.

Dietary iron is absorbed in the duodenum, but ferrous iron is absorbed, and it is transported across the apical membrane of the enterocyte by divalent metal transporter 1. It is then transferred across the enterocyte to the baso-lateral membrane by an unknown mechanism. Iron is transferred across the baso lateral membrane of enterocytes by ferroportin, then bound to transferrin in the plasma and trans- ported for use in target organs and/or storage.

Body stores of iron are tightly regulated to provide adequate iron for cellular needs without evolving toxicity from excess. Body excrete excessive iron, homeostasis is tightly controlled by limiting enteric iron uptake through impaired efflux from enterocytes. Iron efflux is regulated by hepcidin, a recently exposed Hormone produced by hepatocytes. When iron stores are adequate or high, hepcidin is released and binds to intestinal ferroportin causing internalization and demolition of ferroportin. The reduction in ferroportin causes absorbed dietary iron to remain in the enterocyte.





Factor affecting on iron absorption

Mechanism of Anemia

**To whom most likely develop anemia?** Anyone can develop anemia, although the following groups have a higher risk:

- Women: Blood loss during monthly periods and childbirth can lead to anemia. This is especially true if you have heavy periods or a condition like fibroids.
- Children, ages 1 to 2: The body needs more iron during growth spurts.
- **Infants:** Infants may get less iron when they are weaned from breast milk or formula to solid food. Iron from solid food is not as easily taken up by the body.
- **People over 65:** People over 65 are more likely to have iron-poor diets and certain chronic diseases.
- People on blood thinners medication: These medications include drugs include aspirin, clopidogrel, warfarin, heparin products, apixaban, betrixaban, dabigatran, edoxaban and rivaroxaban.

Age/Age Group (years)	Iron (mg/day)
Man	28
Pregnant Woman	38
Lactating Woman	30
Non Pregnant Woman	30
Children	3040
1-3 y	12
4-6 y	18
7-9 y	26
13-15 y	1)
Boys	41
Girls	28
16-18 y	
Boys	50
Girls	30

Age group	Dosage schedule	Duration
Children 6-60 months	20 mg elemental iron + 100 µg folic acid (one tablet of pediatric IFA or 5 ml of IFA syrup or 1 ml of IFA drops)	100 days if the child is clinicall found to be anemic.
School children 6-10 years	30 mg elemental iron + 250 μg folic acid	100 days.
Adolescents 11- 18 years	100 mg elemental iron + 500 µg folic acid	100 days
Pregnant women	One tablet of 100 mg elemental iron + 500 µg folic acid prophylactically	Daily
	If clinically anemic	2 such tablets to be given daily for 100 days
Nursing mothers	One tablet OF 100 mg elemental iron + 500 µg folic acid	100 days

Daily intake of Iron for men women and Childrens

Recommended dose of Iron in Anemia for Men Women children

## B. What are the signs and symptoms of anemia?

Several signs and symptoms occur in all types of anemia, such as fatigue, shortness of breath and feeling cold. Others include:

- Dizziness or weakness.
- Headache.
- Sore tongue.
- Pale skin, dry skin, or easily bruised skin.
- Fast heartbeat.
- Unintended movement in the lower leg
- restless legs syndrome



Comparison of normal and anemic patient

## C. Causes of iron deficiency anemia.

The most common cause of anemia is low levels of iron in the body. This type of anemia is called irondeficiency anemia. Your body needs a certain amount of iron to make hemoglobin, the substance that moves oxygen throughout your body. However, iron-deficiency anemia is just one type. Other types are caused by:

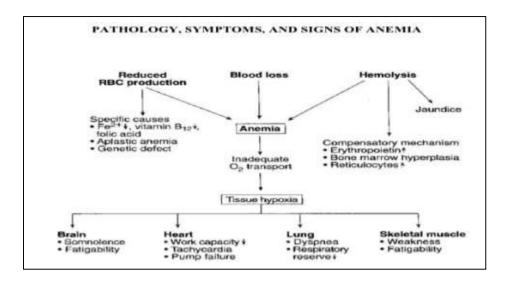
- Diets lacking in vitamin B12, or you can't use or absorb Vitamin B12 Diets lacking in folic acid, also called folate, or your body can't use folic acid correctly
- Inherited blood disorders
- Conditions that cause red blood cells to break down too fast
- Chronic conditions causing your body to not have enough hormones to create red blood cells. These include hyperthyroidism, hypothyroidism, advanced kidney disease, lupus and other long-term diseases.
- Blood loss related to other conditions such as ulcers, hemorrhoids or gastritis.

Since it mostly affects women and children, the impact of anemia is best understood by looking at maternal deaths and school dropout rates. It caused 20% of maternal deaths in India and was the associate cause in 50% of maternal deaths. Anemia during pregnancy also increases the chances of foetal deaths, abnormalities, pre-term and underweight babies. Last year, India reported anemia among 45% of its pregnant women—the highest in the world—even though there has been a fall of 12% in the last ten years.

In children, iron-deficiency anemia severely affects cognitive performance. It also impacts language skills, motor skills and coordination among infants and young children, and a deficit of five to 10 points in intelligence quotient. But these effects of iron deficiency in infancy cannot be correct by subsequent iron therapy. Anemia also impacts the immune system and increases chances of infections and inflammatory disease, further affecting individual productivity. The leading causes of anemia in India are poverty, caste factors and poor sanitation. But frequent occurrences of malaria and worm infestations too result in high incidence of anemia.

## D. Pathophysiology of Anemia in children.

Hemoglobin absorptions are normally higher at birth than at any other time of life. In addition, the neonatal reserves of storage iron are relatively generous. Most newborn infants are well supplied with iron. Between birth and four months of age, there is almost no change in the total body iron in the term infant. The need for exogenous iron is therefore modest during this era. The plentiful iron stores present at birth help to provide for synthesis of hemoglobin, myoglobin, and enzyme iron during the first four months. Additional iron from the hemoglobin breakdown is also made available to meet the iron needs because the concentration of hemoglobin declines from a mean of 17.0 g/dl at birth



Pathophysiology, symptoms, sign of Anemia

to a low of 11.0 g/dl at two months of age. This low point used to be called the early anemia of infancy, and was distinguished from the 'late anemia of infancy,' because it was unemotional to iron management.

After four months of age, a steady shift occurs from an profusion of iron to the marginal iron reserves that characterize the period of continued rapid growth. This window of vulnerability to iron deficiency is the major focus of concern. The change from feast to want with respect to iron is primarily due to the large amount of iron required to maintain a near constant mean hemoglobin concentration of 12.5 g/dl within a

rapidly increasing blood volume between four and twelve months. A large quantity of iron, about 0.8 mg/day, must be absorbed from the diet during this period. The rate and extent to which storage iron becomes depleted can be estimated from the changes in the concentration of serum ferritin and depends both on the scale of iron storage at birth and on the post-delivery diet.

In the first two months of life, there is minimal dietary iron absorption and stores are mobilized to meet the iron requirement. Thereafter, dietary iron absorption becomes important and by four to six months of age, the iron stores have usually been depleted and diet becomes the vital source for iron. A low birth weight baby would have less iron stores and therefore would need extra iron as well as iron at an early age from the diet source.

The main reason for iron-deficiency anemia in infants and children is the inadequate supply of iron in the food. Iron is a mineral the body needs in order to make red blood cells. Children go through periods of rapid growth and the diet should supply enough to enable the increased need for more red blood cells.

The three main reasons for Iron Deficiency Anemia in children are:

- a. Poor bioavailability of iron consumed, related to the low consumption of absorption enhancers and a high consumption of absorption inhibitors in the second year of life
- b. Insufficient intake of iron as compared to the need
- c. Increased requirement during the rapid growth stage of infancy and early childhood, between six and twenty-three months.

# E. Major Causes of Iron Deficiency Anemia in Human Being

## I. Blood Loss

Each ml of RBC contains 1.0 mg of iron. Each day, 1.0 mg of iron is absorbed from the diet and 20 mg of iron from erythrocytes are available to support erythropoiesis. Once iron stores are depleted, dietary and recycled erythrocyte iron are not usually sufficient to compensate for acute blood loss. In all cases of iron deficiency anemia, blood loss should be major concern. Hemorrhage itself is by far the most common mechanism for acute iron loss and anemia. Hemorrhage decreases the host's red cell mass, decreases the supply of iron for erythropoiesis, and increases the iron demand for erythropoiesis. Chronic blood loss from menstruation or hookworm infection has the greatest impact worldwide. Less than 2 mL of blood is lost daily in the stool of healthy. Detection of occult blood losses of up to 60 mL/d may be problematic without particular stool test. Bleeding may occur from multiple sites along the intestinal tract, with an increased incidence of bleeding from the colon. Sometimes overlooked causes of blood loss include blood donation and nosebleeds due to change of temperature in summer season.

## **II.** Pregnancy, Lactation, Bleeding and Menstruation Cycle:

Requirements for iron are greatest around the time of birth. Iron demand is high in menstruating as well as pregnant females. During pregnancy, it is estimated that 1200 mg of iron are required from conception through delivery. Iron intake and stores in the mother must satisfy fetal development, and blood loss at delivery. Additionally, the maternal erythrocyte mass should increase from 350 to 450 ml. By comparison, pregnant women without iron supplements only increase their red cell mass by 180 – 250 ml. One interpretation of this difference is that fetal iron demands are prioritized over the red cell mass of the mother. Those losses are balanced by the absence of menstruation in the lactating female.

Maternal iron deficiency anemia during pregnancy and the perinatal period have Shocking effects on both the mother and child. In addition to the direct effects of anemia, reduced fetal brain maturation, pediatric cognitive defects, and maternal depression are associated with iron deficiency anemia. The reversibility of cognitive defects caused at an early age by iron deficiency is unclear. Importantly, untreated iron deficiency in pregnant females will be passed to the infant. If left un-treated during infancy, childhood, and adolescence, anemia and iron-associated cognitive defects may conceivably be passed between generations much like genetic traits. Unless the iron deficiency is treated at some stage of life, the cycle of iron deficiency from mother to child may remain unbroken for several generations. In the fetus and during infancy, iron is required for the growth and growth of all tissues.

#### III. Hookworm

Like iron deficiency anemia, hookworm infection affects several hundred million humans worldwide. A recent study reported that there is a considerable overlap between malaria and hook- worm in sub Saharan Africa. Worldwide, there are t hook worm species that infect humans. Both are found in tropical regions based on the requirement of moist soil for survival. The worm is introduced to the soil by fecal matter in regions where sanitation is not present. From the soil, the parasite accesses the duodenum of a new human host directly by mouth, or indirectly via the skin. Once in the gut, the worm may be retained for several years as it releases eggs in the stool. Owing to their location in the small bowel, capsule endoscopy is helpful for diagnosis if eggs are not present in the stool. Hookworms live on 0.3–0.5 mL of blood daily from the intestinal mucosa.

# IV. Diet and Mal absorption of Iron

Local economics generally directive the level of nutrition among the world. The diet, by itself, infrequently causes iron deficiency anemia in the absence of severe undernourishment. A vegetarian diet is usually sufficient to prevent anemia even though the iron stores of the host may be low. The diet becomes far more relevant when the iron stores are lost, or anemia has already developed, and the host requires additional iron absorption from the gut for recovery. This occurs in the multiple settings described earlier in this work:

blood loss, rapid growth during infancy, malaria, and hookworm. In these settings, the diet and iron supplements become critical for maintaining iron availability. Supplemental dietary iron may be needed, because the average Western diet is not sufficient to meet the needs of pregnancy In addition to the iron content itself, the bioavailability of iron for absorption de-pends largely on the dietary components. Iron in the form of heme is especially bioavailable, and meat-containing diets are also beneficial Vitamin C improves dietary availability of iron as well as avoidance of tea or other iron-chelating substances.

## F. Efforts to control Anemia in India.

The goals of the Government of India's tenth five-year plan for anemia control for children include:

- 1. Screening of children for anemia wherever required and appropriate treatment for those found to be anemic
- 2. Reducing the prevalence of anemia by 25% and moderate and severe anemia in children by 50%

As anemia has continued to be highly prevalent among children, the program has been re-designated as the National Nutritional Anemia Control Program in 1991. This program aims at decreasing the incidence of anemia among the vulnerable sections of the population, namely pregnant and lactating women, intrauterine device (IUD) users, and children in the one-to-five year age group. For children between 12 and 59 months the program prescribes one tablet of 20 mg elemental iron and to treat children found clinically anemic 100  $^{\circ}$ 9 of folate for 100 days in a year.

Therefore, according to the new policy the recommendations are as follows:

- 1. Children from six to sixty months will be given one milliliter of IFA syrup for 100 days in a year.

  One milliliter of syrup will contain 20 mg elemental iron and 100 micrograms of folic acid
- 2. For safety reasons, the bottle should be dispensed in a way that only one milliliter can be dispensed at a time

## G. Formulations for treatment of anemia

## (1) In case of Infants and Children.

Two studies were focused on pediatric anemia. In these two studies, three different drugs have been studied for their clinical efficacy against anemia among pediatric age group; Punarnavadi Mandura, Dadimadi Ghrita *and* Trikatrayadi Lauha. In addition to the significant efficacy of these drugs, the probable mode of action has also been delineated. Punarnavadi Mandura is preferably administered with buttermilk which is having acidic pH and contains lactic acid. Iron absorption is aided by decreased pH. Furthermore, it might be possible that iron combines with lactic acid to form ferrous lactate before

absorption which is used by modern allopathic medicine for the management of Anemia. Alternatively, it may also happen that the proteins of buttermilk or the amino acids released after digestion may combine with iron before the absorption takes place. This point of view is also supported by the World Health Organisation as it supports the use of meat and other proteins for the absorption of iron

## (2) In case of Pregnancy:

For Management of Anemia, two different drugs have been studied for their clinical efficacy against anemia among pregnant women which includes Dhatrilauha Vati and Pandughni Vati. Anemia is one of the most common complications of pregnancy and contributes directly or indirectly to 20% of deaths in third world countries. Anemia during pregnancy is termed *as* Garbhini Pandu and is primarily caused due to vitiation *Rasa* Dhatu. According to Ayurveda, during pregnancy, Rasa Dhatu has to shoulder three times more responsibilities than in normal individuals. It becomes responsible for the nourishment of three factors; fetus, breast and the pregnant woman. Due to this stress *on* Rasa Dhatu during pregnancy, there are more chances that the pregnant woman gets affected with Garbhini Pandu. Dhatrilauha Vati is composed of Lauha Bhasma, which is an iron supplement and has Deepana (Stomachic) property that leads to proper metabolism and Dhatu Poshana (tissue nourishment). Amalaki (Emblica officinalis) Amruta help in the nourishment of basic structural body constituents and are supportive for the absorption of iron. Yastimadhu (Glycyrrhiza glabra) has Shonita Sthapana (prevent bleeding tendency and normalize hematopoiesis) property. The cumulative effects of all the ingredients leads to the correction of metabolism, iron absorption and improved blood formation, thereby correction of anemia.

## (3). Home Remedies for Anemia:

- 1) **Apple.** Stick to the quotation of "an apple a day, keeps the doctor away". Apple is a rich—source of iron and various other nutrients, and not only one but 2 or 3 apples in a day can recover iron deficiency for a day.
- **2) Honey.** Honey is an excellent natural remedy for anemia. Make a concoction of a tablespoon of honey, lemon juice, and apple cider
- **3) Vinegar.** Eat this daily to cure anemia. Honey increases hemoglobin in the blood and it is full of iron and copper elements.
- **4) Blackcurrant.** Soak 8-10 blackcurrants for overnight in water and for 3 to 4 weeks prepare a health routine of having this as the first thing in the morning. Do not forget to remove blackcurrant seeds before consuming it.
- **5) Beetroot.** Beetroot is one of the iron rich vegetables on the earth. You may eat it with salad and can use its juicy extracts. Beetroot juice can be mixed with spinach juice, apple juice, or carrot juice.

- **6) Iron pots.** Cook your food in iron containers and pots on a daily basis as it can combat the problem of iron deficiency by increasing the iron in your food.
- 7) Massage. Getting a whole body massage with oils, like mustard, olive or tender oil can help improving the blood flow in the body.
- **8) Figs.** Figs are also a great source of iron. Usage of 3to 4 figs everyday can enhance iron in your body. Figs may also be consumed after soaking in water during the night and consuming early in the morning, daily.
- 9) Tomato Juice. Imbibe the juice of tomato mixed with apple juice, daily, to alleviate anemia.
- **10) Banana.** Consume a ripe banana with two tablespoons honey, every day. It is a natural remedy for overcoming anemia.
- 11) Almonds. Soak about 6-7 almonds in water for a night and eat it the next morning, after peeling its skin.
- **12) Amla.** Mix 60ml of fresh Amla juice with 25ml of honey and drink it every morning to alleviate anemia.
- **13) Spinach.** Spinach, as a vegetable, is an excellent remedy to overcome anemia. It also works great when its juice is mixed with honey.
- 14) Sun Bath. Sunbathing is good for those people, who have less red blood cell count.it has vitamin D.

## **H. Conclusion:**

In this review article of Anemia, we studies that the main cuses, sign symptoms of Anemia. In our countries Anemia are generally caused due to lack of nutritional food and diet supplement. Pregnancy, Lactation, Bleeding, Period, and Poverty, Diseases like Malaria and worm infection is the important factor which effect of health are causes Anemia. Unlike other prevalent anemias and hemoglobinopathies, the diagnosis and treatment of iron deficiency anemia is achievable in most, if not all individuals. However, consideration of iron deficiency anemia must include the possible convergence of several causative factors. If resources are adequate, care must be given toward modified approaches to therapy. In the under developed Asian countries, more communal approaches are being taken to overcome the overlapping causes of iron deficiency anemia that affect hundreds of millions worldwide. It is predicted that increased research and understanding of fundamental iron biology will assist in devising new strategies aimed to- ward the global elimination of this disease.

#### References

- I.Crichton R. Iron Metabolism: From Molecular Mechanisms to ClinicalConsequences. 3rd ed. West Sussex, UK: John Wiley and Sons, 2009; 17–56:141–325.
- II.Guyton. A.C. & Hall, JE. 2008. Textbook of Medical Physiology, 11<sup>th</sup> edition, Elsevier, New Delhi.
- III.Harvey JW. Iron metabolism and its disorders. In: Kaneko JJ, Harvey JW, Bruss ML, eds. Clinical Biochemistry of Domestic Animals. 6th ed. Burlington, Massachusetts: Elsevier, 2008:259–285.
- IV.Harvey JW. Microcytic anemia. In: Feldman BF, Zinkl JG, Jain MC, eds. Schalm's Veterinary Hematology. 5th ed. Philadephia, Pennsylvania: Lippincott, Williams and Wilkins, 2000:200–204.
- V.Knovich MA, Storey JA, Coffman LG, et al. Ferritin for the clinician. Blood Rev 2009;23:95–104.
- VI.Weiss DJ. Iron and copper deficiencies and disorders of iron metabolism. In: Weiss DJ, Wardrop KJ, eds. Schalm's Veterinary Hematology. 6th ed. Ames: Blackwell Publishing, 2010:167–171.
- VII.Dzanis DA. The Association of American Feed Control Officials dog and cat food nutrient profiles: Substantiation of nutritional adequacy of complete and balanced pet foods in the United States. J Nutr 1994; 124(12 Suppl):2535S–2539S.
- VIII.Gross Kl, Wedekind KJ, Cowell CS, et al. Nutrients. In: Hand MS, Thatcher CD, Remillard RL, et al. eds. Small Animal Clinical Nutrition. 4th ed. Marceline, Missouri: Walsworth Publishing Company, 2000: 75–77.
- IX.Michel KE. Unconventional diets for dogs and cats. Vet Clin North Am Sm Anim Practice 2006;36:1269–1281.
- X.Giger U. Regenerative anemias caused by blood loss or hemolysis. In: Ettinger SJ, Feldman EC, eds. Textbook of Veterinary Internal Medicine. 6th ed. St. Louis, Missouri: Elsevier Saunders, 2005:1886–1908.
- XI.Chulilla JAM, Colas MSR, Martin MG. Classification of anemia for gastroenterologists. World J Gastroenterol 2009;15:4627–4637.
- XII.White C, Reine N. Feline nonregenerative anemia: Pathophysiology and etiologies. Compend Contin Educ Vet 2009;31:E1–E19.
- XIII.Stockholm SL, Scott MA. Fundamentals of Veterinary Clinical Pathology. 2nd ed. Ames, Iowa: Blackwell Publishing, 2002:105–150.
- XIV.Abrams-Ogg T. Nonregenerative anemia. In: Ettinger SJ, Feldman EC, eds. Textbook of Veterinary Internal Medicine. 7th ed. St. Louis, Missouri: Elsevier Saunders, 2010:788–797.
- XV.Waldrop JE, Rozanski EA, Freeman LM, et al. Packed red blood cell transfusions in dogs with gastrointestinal hemorrhage: 55 cases (1999–2001). J Anim Hosp Assoc 2003;39:523–527.
- XVI.Klein SC, Peterson ME. Canine hypoadrenocorticism: Part I. Can Vet J 2010;51:63–69.
- XVII.Gelens HC, Moreau RE, Stalis IH, et al. Arteriovenous fistula of the jejunum associated with gastrointestinal hemorrhage in a dog. J Am Vet Med Assoc 1993;202:1867–8.
- XVIII.Anderson C, Aronson I, Jacobs P. Erythropoiesis: Erythrocyte deform- ability is reduced and fragility increased by iron deficiency. Hematol 2000;4:457–460.
  - XIX.Moritz A, Becker M. Automated hematology systems. In: Weiss DJ, Wardrop KJ, eds. Schalm's Veterinary Hematology. 6th ed. Ames, Iowa: Blackwell Publ, 2010:1054–1066.
  - XX.Polzin DJ. Chronic kidney disease. In: Ettinger SJ, Feldman EC, eds. Textbook of Veterinary Internal Medicine. 7th ed. St. Louis, Missouri: Elsevier Saunders, 2010:1990–2021.
  - XXI.Simpson KW, Meyer DJ, Boswood A, et al. Iron status and erythrocyte volume in dogs with congenital portosystemic vascular anomalities. J Vet Intern Med 1997;11:14–19.
- XXII.Lima CS, Paula EV, Takahasi T, et al. Causes of incidental neutropenia in adulthood. Ann Hematol 2006;85:705–709.

- XXIII.Cook AK, Gilson SD, Fischer WD, Kass PH. Effect of diet on results obtained by use of two commercial test kits for detection of occult blood in feces of dogs. Am J Vet Res 1992;53:1749–1751.
- XXIV.Duncan JR, Prasse KW, Mahaffey EA. Veterinary Laboratory Medicine Clinical Pathology. 3rd ed. Ames, Iowa: Iowa State Univ Press, 1994: 160.
- XXV.Harvey JW, French TW, Meyer DJ. Chronic iron deficiency anemia in dogs. J Am Anim Hosp Assoc 1982;18:946–960.
- XXVI.Tortora.G.J.2014.Principles of Anatomy And Physiology, 14th edition
- XXVII.Harshmohan Textbook of Pathology, 6<sup>th</sup> Edition,2010.
- XXVIII.Kapur, D., Agarwal, K.N., Sharma, S., Kela, K., Kaur, I., 2002. Iron status of children aged 9-36 months in an urban slum Integrated Child Development Services project in Delhi. Indian Pediatr. 139:136-44.
- XXIX.Kilbridge, J., Bakea, T.G., Parapia, L.A., Khoury, S.A., Shugaidef, S.W., Jerwood, D., 1999. Anaemia during pregnancy as a risk factor for iron-deficiency anaemia in infancy: a case-control study in Jordan. Int. J. Epidemiol 28:461-8.
- XXX.Ministry of Family and Health Welfare. 2002/2003. *District-Level Household Survey*. New Delhi, Government of India.
- XXXI.Mukherjee, K.L., Ghosh, S., 2012. Medical laboratory Technology. Procedure Manual for Routine Diagnostic Tests. Vol I (Second edition), 263-266.
- XXXII.Natasha, S., Yasmin G., 2010. Inkosi Albert Luthuli Central Hospital. Proceedings of South African Thalassaemia Association.
- XXXIII.Pasricha SR, Flecknoe-Brown SC, Allen KJ, et al; Diagnosis and management of iron deficiency anaemia: a clinical update. Med J Aust. 2010 Nov 1;193(9):525-32.
- XXXIV.WHO (2004). Micronutrient deficiency: Battling iron deficiency anaemia: the challenge. Available from: http://www.who.int/nut/ida.htm, accessed on April 24, 2008
- XXXV.WHO, 2005. Worldwide prevalence of anaemia 1993–2005: WHO global database on anaemia / Edited by Bruno de Benoist, Erin McLean, Ines Egli and Mary Cogswell.