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Research Article

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Study of Correlation Between Rbc and It's Indices In Students of Osmania Medical College With Different Body Mass Indices

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ABSTRACT

Currently nutritional changes are a public health problem in all over the world, affecting all age groups including young adults. While the under nutrition is affecting one side, over nutrition is also affecting the people on the other side. This leads to obesity. Levels of overweight and obesity together with other cardiovascular risk factors increase with age even within this age span[1]. The Study aims the Correlation between RBC and it's indices in Students of Osmania Medical College with different body mass indices and look for correlation between BMI and RBC and it's indices. A cross-sectional study was conducted in 100 healthy individuals with different body mass indices in the age group of 18-35yrs. Subjects were divided into 4 categories on BMI. 2ml venous blood collected in EDTA containers. It is analysed in automatic Haemocytometer. Pearson correlation was done to find the correlation between BMI & RBC and it's indices. Pearson correlation was done to find the correlation between BMI & RBC and it's indices. In the study there was significant positive correlation between BMI and RBC, negative correlation between BMI and MCV, MCH, MCHC. Individuals with high BMI have increased RBC count & low RBC indices.

Key Words: *Body mass index, Obesity, Red blood corpuscles, Indices of RBC*

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INTRODUCTION

Human body needs a proper nutrition through well balanced diet to fulfil body requirements and to maintain basic body physiology. Improper nutrition leads to consumption of either insufficient supply of nutrients or consumption of excess calories.

While the under nutrition is affecting one side, over nutrition is also affecting the people on the other side. This leads to obesity. At present this problem is raising world wide due to change in food habits & sedentary life style.

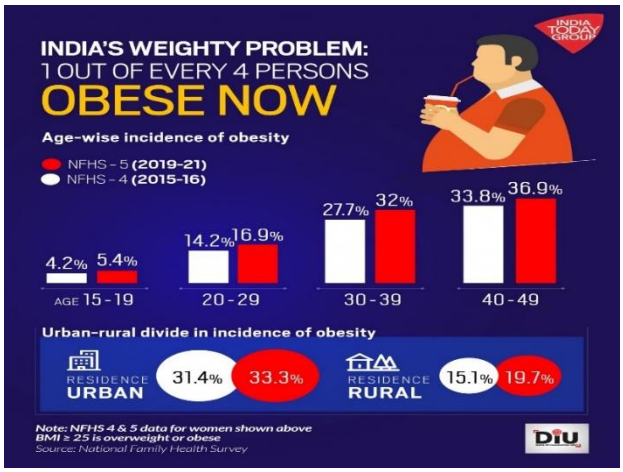
According to a study done in developing countries on young adults, young adults are prone to overweight and obesity during the transition from adolescence to adult in developing countries as much as in developed countries. Levels of overweight and obesity together with other cardiovascular risk factors increase with age even within this age span[1].

WHO classified obesity based on BMI as follows:

WHO CLASSIFICATION OF WEIGHT STATUS	
WEIGHT STATUS	**BODY MASS INDEX (BMI), kg/m^2**
Underweight	<18.5
Normal range	18.5 – 24.9
Overweight	25.0 – 29.9
Obese	≥ 30
Obese class I	30.0 – 34.9
Obese class II	35.0 – 39.9
Obese class III	≥ 40

WHO CLASSIFICATION OF OBESITY

According to obesity survey in India, done by NFHS (National Family Health Survey) 4&5 , it is showing that both in urban & rural areas the obesity problem is increasing in all age groups, this can be analysed by data collected in urban and rural areas in 2015-16 & 2019-21.



AGE WISE DISTRIBUTION OF OBESITY IN INDIA

Increase in BMI leads to increased risk of comorbidities. The table below is adapted from WHO, 2004. It is showing that as BMI increases risk of comorbidities increases.

Table 1. Classification of adult underweight, overweight and obesity according to BMI.

Classification	BMI	Risk of comorbidities
Under weight	<18.50	Low (but risk of other clinical problems increased)
Normal range	18.50 – 24.99	Average
Overweight:	≥ 25.00	
Pre-obese	25.00 – 29.99	Increased
Obese class I	30.00 – 34.99	Moderate
Obese class II	35.00 – 39.99	Severe
Obese class III	≥ 40.00	Very severe

Adapted from the WHO, 2004.

BMI AND RISK OF CO-MORBIDITIES BY WHO,2004

MATERIALS AND METHODS

A cross- sectional study was conducted in 100 healthy individuals with different body mass indices in the age group of 18-35 years. The study was conducted at Osmania Medical College, Hyderabad, Telangana and Institutional ethics committee approval was obtained (vide ECR/300/Inst/AP/2013/RR-16 Regd No.20116001004D).

Subjects were enrolled with informed consent. Detailed procedure was explained to the subject and data was obtained after approval from the individual. Weight was measured using a digital weighing machine. Height was measured using a stadiometer and BMI was calculated for each subject by using QUETELET formula.

$$BMI = \frac{Weight\ (kg)}{[Height(m)]^2}$$

QUETELET formula

BMI categorized according to WHO criteria, 2ml venous blood collected in EDTA containers. It is analysed in automatic Haemocytometer.

INCLUSION CRITERIA were
1. The study population comprises students of all grades of age 18-35yrs.
2. Students who have given consent.

EXCLUSION CRITERIA were
1. Students on iron supplementation
2. Any acute infection during the study period
3. Pregnant and Lactating women
4. Chronic disorders – Diabetics, Hypertensives, Asthma, and other Comorbidities

STATISTICAL ANALYSIS

Data was collected in a prestructured proforma and tabulated in Microsoft Excel.
The data was entered in Microsoft excel 2007 and was analysed by SPSS for Windows, Version 22 (IBM SPSS Statistics, Somers NY, USA) was used to analyze data. Data has been presented as mean, standard deviation or 95% confidence intervals. p-value calculated.

Correlation coefficient (r)	Interpretation
0 - 0.3	Positive Weak correlation
0.3-0.6	Positive Moderate correlation
0.6-1.0	Positive Strong correlation
0 to (-0.3)	Negative Weak correlation
(-0.3) to (-0.6)	Negative Moderate Correlation
(-0.6) to – (1)	Negative Strong Correlation

Graphical representation of data was done using MS Excel and MS word and various types of graphs such as bar diagram and Pie diagram were obtained. p valueof<0.05 was considered as statistically significant.

RESULTS

Table 1 : Distribution Of Anthropometric Parameters Among Study Subjects

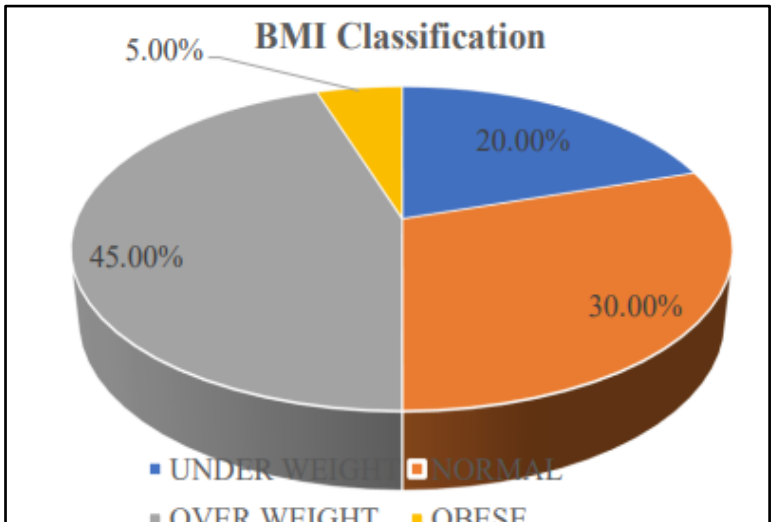
	Mean	Median	Standard deviation	Minimum	Maximum
Height (cm)	161.28	159	10.167	142	188
Weight (kg)	59.7	64	10.156	40	75
BMI (kg/m^2)	23.382	24.64	4.30116	15.6	30.08

Table 2 : Haematological Parameters Distribution Among Study Subjects

PARAMETER	Mean	Median	Standard deviation	Minimum	Maximum
RBC 10^{12}/L	4.9416	4.885	0.38271	4.56	6.1
MCV fl	84.15	85.05	0.0633	68	93.9
MCH pg	27.188	28.45	3.5315	17.9	32
MCHC g/dl	32.856	33.25	2.4218	26.3	38

Table 3 :Bmi Classification Distribution

BODY MASS INDEX	FREQUENCY	PERCENT
Under weight	20	20.0%
Normal	30	30.0%
Over weight	45	45.5%
Obese	5	5.0%
Total	100	100%



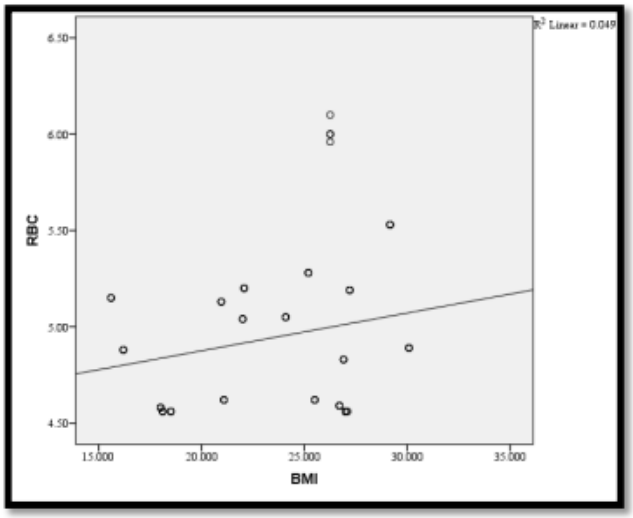
PIE DIAGRAM SHOWING BMI CLASSIFICATION DISTRIBUTION

Table 4 : Correlation Between Bmi And Rbc

		RBC
BMI (kg/m^2)	Pearson Correlation	0.221
	P Value	0.027*
	N	100

*Significant

In the study there was significant positive correlation between BMI and RBC. I.e. With increase in BMI there was increase in RBC count.



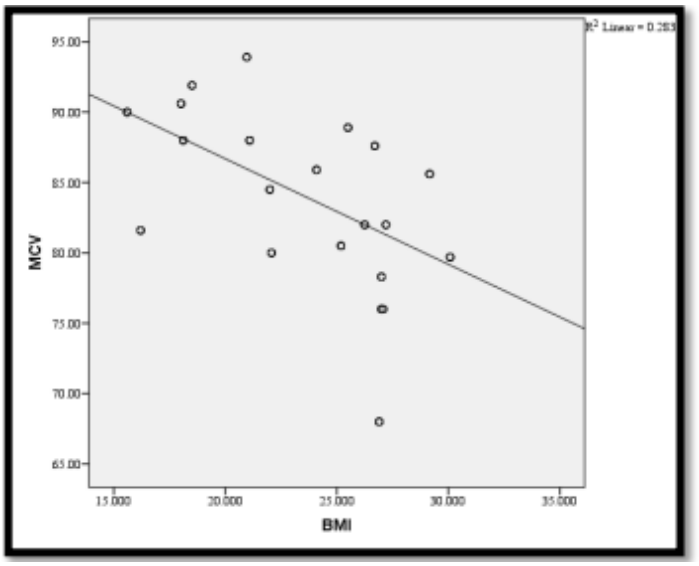
SCATTER PLOT SHOWING POSITIVE CORRELATION BETWEEN BMI AND RBC

Table 5 : Correlation Between Bmi And Mcv

		MCV
BMI (kg/m^2)	Pearson Correlation	-0.532
	P value	0.001*
	N	100

*Significant

In the study there was significant negative correlation between BMI and MCV and vice versa. I.e. with increase in BMI there was decrease in MCV.



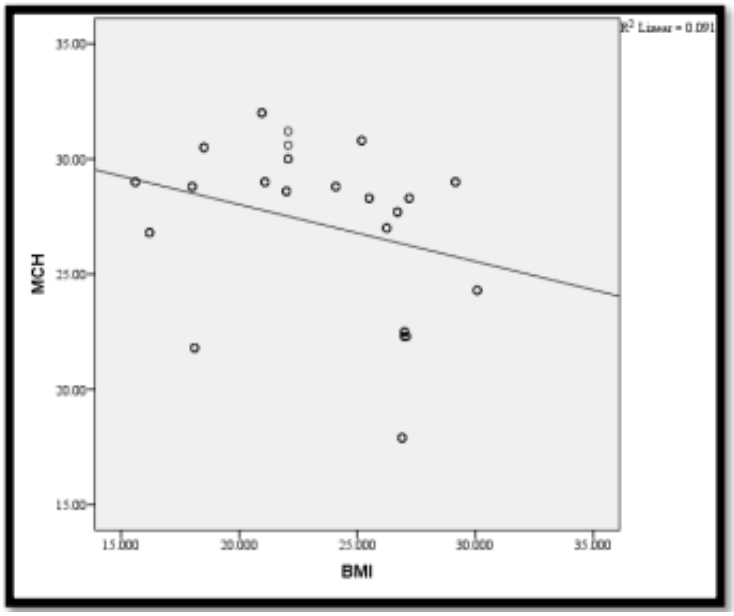
SCATTER PLOT SHOWING NEGATIVE CORRELATION BETWEEN CORRELATION BETWEEN BMI AND MCV

Table 6 : Correlation Between Bmi And Mch

		MCH
BMI (kg/m^2)	Pearson Correlation	-0.301
	P Value	0.002*
	N	100

*Significant

In the study there was significant negative correlation between BMI and MCH. I.e. with increase in BMI there was decrease in MCH.

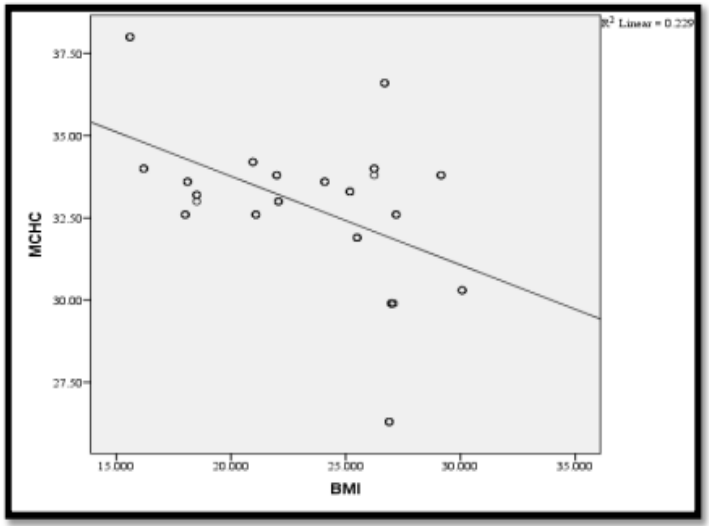


SCATTER SHOWING NEGATIVE CORRELATION BETWEEN BMI AND MCH

Table 7 : Correlation Between Bmi And Mchc

		MCHC
BMI (kg/m^2)	Pearson Correlation	-0.479
	P Value	0.001*
	N	100

*Significant



SCATTER PLOT DIAGRAM SHOWING NEGATIVE CORRELATION BETWEEN BMI AND MCHC

In this study it was observed that there was significant positive correlation between Body mass index(BMI) & RBC. There is a significant negative correlation between BMI and Mean corpuscular volume(MCV), Mean corpuscular haemoglobin(MCH)& Mean corpuscular haemoglobin concentration(MCHC).

DISCUSSION

In the present study our demographic analysis revealed mean age of subjects was 24.11 ± 5.174 years, median age was 23 years. 55% were males and 45 were females and 20% were underweight, 30% had normal BMI, 45% were overweight and 5% were obese.

Mean MCV was 84.15 ± 6.06 fl, mean MCH was 27.188 ± 3.53 pg, mean MCHC was 32.85 ± 2.42 g/dl. In this study a Significant positive correlation between BMI and RBC (r = 0.221) was observed, this is in accordance with an European study showed a positive correlation of BMI with RBC by Barazzoni et al., 2014 [2].

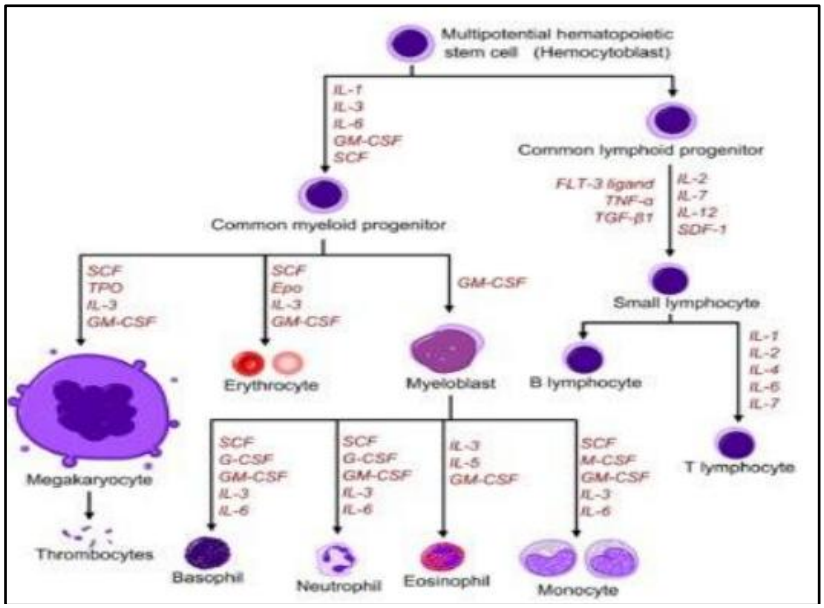
And there is a Significant negative correlation between BMI and MCV (r = -0.532), MCH (r = -0.301), MCHC (r = -0.479) I.e. with increase in BMI there was decrease in MCV, MCH, MCHC, it is accordance with a study by Gebrie A et al[3].

In the bone marrow from multi potent haematopoietic stem cells under the influence of IL-1, IL- 3,IL-6, GM-CSF, SCF common myeloid progenitor cells are formed . From common myeloid progenitor cells under the influence of Epo, IL-3, GM-CSF,SCF Erythrocytes are formed[4].

In obese patients, adipokines which are released by adipocytes affects health conditions, through an abnormal production of adipokines[5]. Adipose tissue releases interleukin-1, interleukin-6 and interleukin-10, and chemokines CCL2,CCL5,CXCL8 &CXCL10.

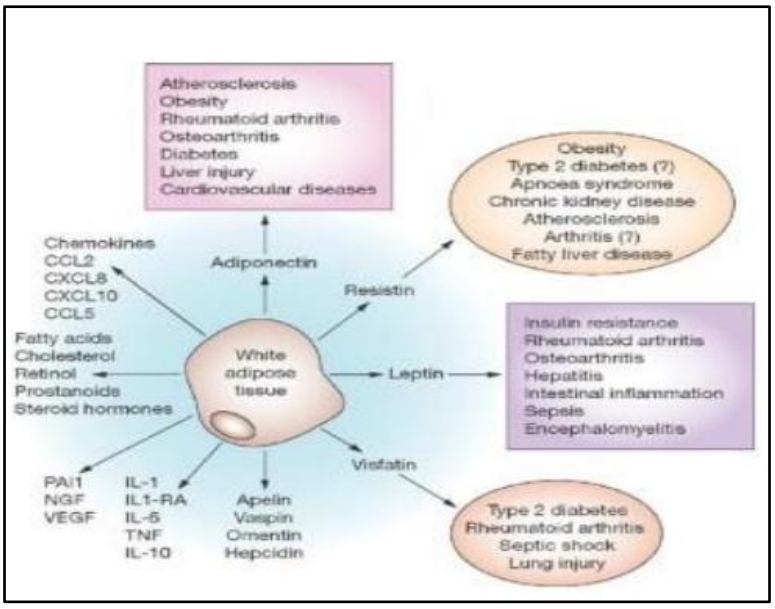
It also releases leptin, resistin, visfatin, hepcidin, apelin, vaspin, omentin, tumour necrosis factor, vascular endothelial growth factor,nerve growth factor, plasminogen activator inhibitor 1. In these adipokines inteleukins, leptin will affect bonemarrow architecture[6].

The growth factors required for haematopoiesis are shown in the below diagram.



HAEMATOPOIESIS AND REQUIRED GROWTH FACTORS

Adipokines which are released from adipose tissue in obese people are as follows

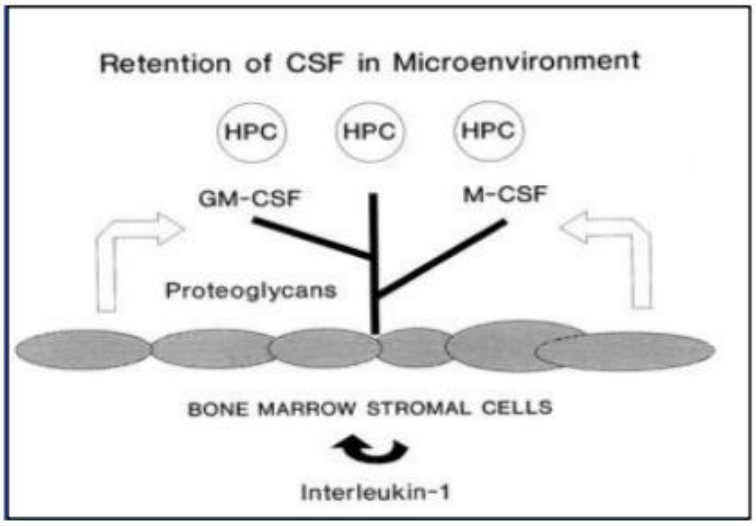


ADIPOKINES IN OBESITY

The polypeptide interleukin 1 (IL-1) is the primary mediator of the acute phase reactant & obesity is considered as chronic systemic low grade inflammation. It has profound endocrinologic, metabolic and hematologic effects.

Recently, IL-1 has been recognized as a molecule that is important in the regulation of hematopoiesis. IL-1 induces the production of several different hematopoietic growth factors including granulocyte-macrophage, granulocyte and macrophage colony-stimulating factors and interleukin 6 by a variety of accessory cells. In addition, IL-1 acts synergistically with colony-stimulating factors in the proliferation of primitive hematopoietic progenitor cells[7].

IL-1 in the bonemarrow stroma acts paracrinally and helps in retainment of colony stimulating factors by binding them to proteoglycans and these factors will be presented to hematopoietic progenitor cells[7].



ROLE OF INTERLEUKIN-1 IN HAEMATOPOIESIS

Interleukin 6 (IL-6) plays critical roles in the immune response and hematopoiesis. It enhances interleukin 3-induced proliferation of hematopoietic stem cells. A study by Ikebuchi et al[8] and further studies by Toshio Himno et al[9] in vitro on murine bonemarrow proved combination of IL-6 and IL-3 is useful for expanding hematopoietic stem cells.

Ikebuchi et al[8] first demonstrated that IL-6 and IL-3 acted synergistically in support of the proliferation of murine multipotential hematopoietic progenitors in vitro in semisolid culture. They suggested that the effect of IL-6 was the shortening of the Go period of stem cells. Subsequent studies by Toshio Himno et al61 demonstrated that the number of colony-forming units (CFU-s) in murine bone marrow cells was increased 5-fold when they were cultured in the presence of both IL-6 and IL-3 in liquid culture. The data indicate that the combination of IL-6 and IL-3 is useful for expanding hematopoietic stem cells[9-10].

A work done by Ana Cardoso et al[11] on IL-10 in mice revealed effect of IL-10 in vivo as a trigger of Myelopoiesis in bonemarrow. The study the depot injections of IL-10 were given to mice. The study showed elevated levels of IL-10 drive the expansion of hematopoietic stem cells in the Bone marrow and promoted the differentiation of myeloid cells at the expense of other lineages.

In consequence, IL-10-induced animals accumulate monocytes and granulocytes in the periphery, the enhanced myelopoiesis is rapidly reversed once IL-10 levels return to normal, as is also the case of the hematologic alterations detected in humans receiving IL10[11]. IL-10 can induce extramedullary myelopoiesis revealed by a study conducted in mice by Ana Cardoso et al[11] especially in spleen.

A study done by Brian D. Bennett et al[12] in both human and murine hematopoietic stem cell populations, about role of leptin in haematopoiesis, the study revealed that like other cytokine receptors, the leptin receptor seems to be expressed on progenitor and more mature hematopoietic cells. However, the proliferative effect of leptin on hematopoietic stem cell populations in myeloid, lymphoid and erythroid colony assays demonstrates that leptin can act on very early cells of the hematopoietic lineage.

To characterize the potential hematopoietic role of leptin, researchers used antisense oligonucleotides corresponding to several regions adjacent to the initiation methionine of the leptin receptor & performed methylcellulose colony assays in the presence of complete conditioned media containing serum, granulocyte/macrophage colony stimulating factor (GM-CSF), G-CSF, interleukin-3 (IL-3), IL-6, erythropoietin (EPO) and c-Kit ligand (KL).

Under these conditions, the ability of human CD34+ stem cells to form colonies was not inhibited. leptin augmented myeloid colony formation, and dramatically increased the formation of lymphoid and erythroid colonies and the leptin showed synergistic effect with KL, GM-CSF and IL-3 [12].

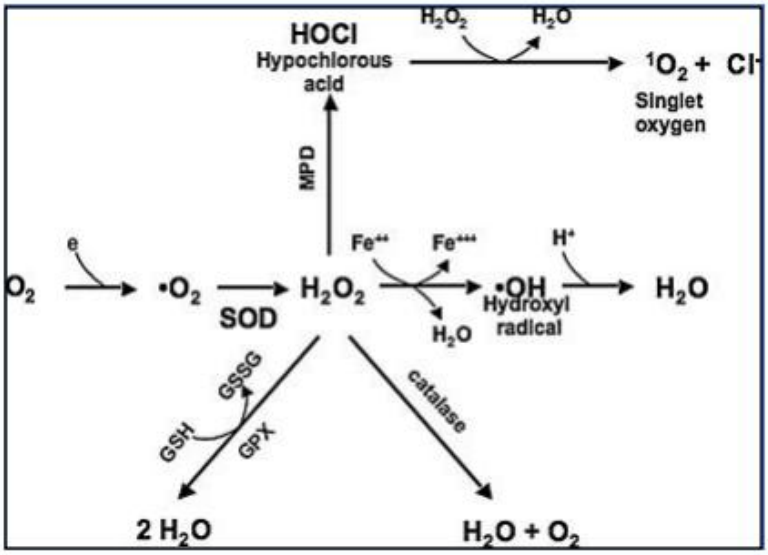
From this it is evident that although the formation of lymphoid series is decresed because of presence of factors which are converting multipotent hematopoietic stem cells into the myeloid series, leptin is causing formation of all lineages of haematopoietic system there by as the BMI is increased, lymphocytes are decreased but due to the action of leptin, they are not decreased much, in this study also it came that as BMI is increased lymphocytes are decreased in number but not statistically significant.

Accumulated adipose tissue induces the synthesis of pro-inflammatory cytokines, including TNF-α, IL-1, and IL-6, which promote increased generation of Reactive oxygen species (ROS) by macrophages and monocytes; therefore, a rise in concentration could be responsible for increased ROS.

TNF-α favours the systemic acute-phase response, via the release of IL-6, another pro-inflammatory molecule, and via the reduction of systemic anti-inflammatory cytokines, like adiponectin. TNF-α also increases the interaction of electrons with oxygen to generate superoxide anions.

ROS production can be induced by TNF-α by promoting NADPH oxidase pathway . Serum TNF-α levels are increased in obesity and it is decreased with weight loss. NADPH oxidase pathway transfers electrons from NADPH to oxygen and represents a major source of ROS synthesis in adipocytes. Generated O2 radicals are further converted into hydrogen peroxide (H2O2), longer-lived membrane-permeable ROS. The hydrogen peroxide damages RBC cell membrane thereby increasing their fragility.

Recent evidence suggests that oxidative stress contributes significantly to the regulation of hematopoietic cell homeostasis. In particular, red blood cells and hematopoietic stem cells are highly sensitive to deregulated accumulation of reactive oxygen species (ROS)



REACTIVE OXYGEN SPECIES IN OBESITY

When the red blood cells accumulates increasing amounts of ROS, this leads to abnormal oxidation of hemoglobin in the form of methemoglobin and increased osmotic fragility in red blood cells. Specifically, deformability of red blood cells is abolished in the presence of hydrogen peroxide[13].

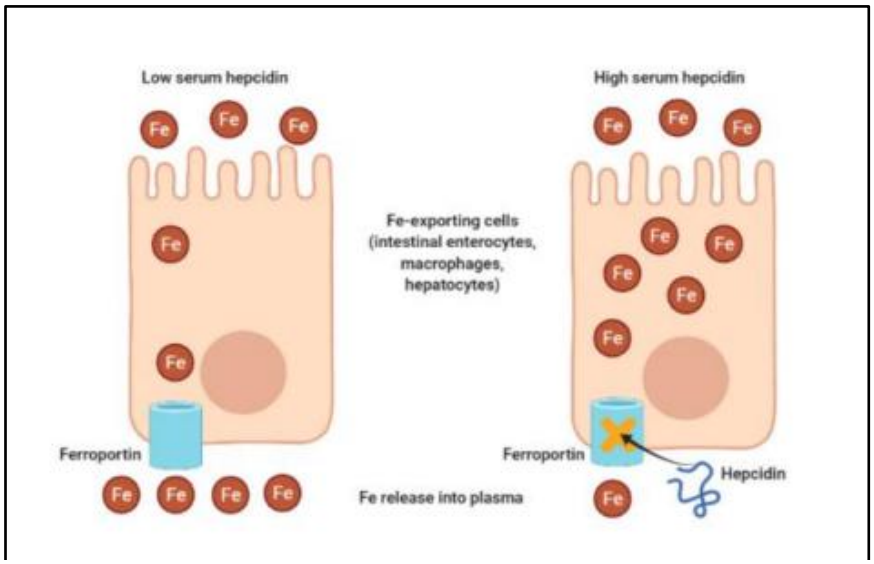
It induces stress erythropoiesis because the tissue oxygenation gets affected resulting in enhanced proliferation of erythroid progenitors. This is the another reason for increased RBC count in higher BMI people as the adipokines will cause enhanced release of reactive oxygen species.

As a result of all the above factors, Red blood corpuscle production is increased in obese individuals.

In obese people Mean corpuscular volume (MCV), Mean corpuscular haemoglobin (MCH) and Mean corpuscular haemoglobin concentration (MCHC) decreased because Iron absorption in the proximal small intestine mucosa of the gut requires transport across the apical and basolateral membranes of duodenal enterocytes.

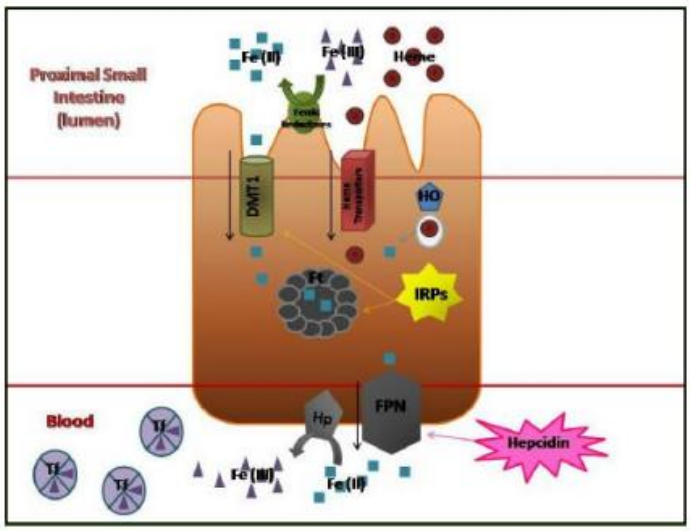
The dietary non-heme iron in the duodenal lumen is reduced by a ferric reductases and thus made available for divalent metal transporter 1 (DMT1), which transports ferrous iron across the apical brush border membrane and heme iron is transported by heme transporters.

The amount of iron not retained by the cell inside the iron storage protein ferritin (Ft) is transferred to the bloodstream. The basolateral release of non- heme iron (which is also derived from heme catabolized by heme oxygenase [HO]) is mediated by ferroportin (FPN) which transports the metal across the membrane and hephaestin (Hp), which re-oxidizes iron as a necessary step for binding to the plasma carrier protein transferrin (Tf). The hepcidin causes ferroportin internalization and degradation, decreasing the transfer of iron to the body[14].



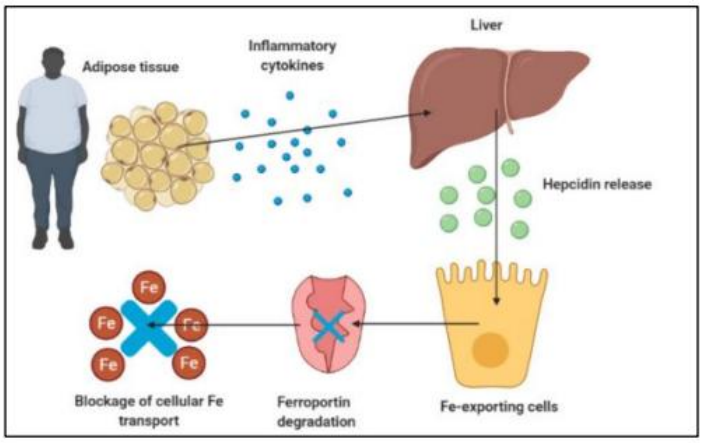
IRON METABOLISM IN CONDITIONS OF NORMAL AND HIGH SERUM HEPCIDIN

Adipocyte hepcidin expression has a positive correlation with BMI, with a negative association with transferrin saturation. Therefore, lower bioavailability of iron among obese adults might be potentially related to the greater adipose hepcidin. Although hepcidin expression is more than 100-fold higher in hepatocytes than in adipocytes, secreted hepcidin from both tissues may have relevance for humans because in obesity, adipose tissue mass may be 20-fold greater than liver mass[14].



INTESTINAL IRON ABSORPTION AND ROLE OF FERROPORTIN

In high serum levels of hepcidin, there is decrease in serum iron because the hepcidin causes ferroportin internalization and degradation which is essential for iron transport from intestinal cell to blood stream.



HIGH HEPCIDIN LEVELS IN OBESITY

Obesity is characterized by a low grade systemic inflammation. Increased levels of acute phase protein (e.g. c-reactive protein (CRP)), pro-inflammatory cytokines (e.g. interleukin-6 , IL-1β and TNFα) and adipose tissue derived mediators (e.g. leptin) are known to trigger the transcriptional activation of hepcidin[15].

This leads to iron deficiency anaemia in obese people, resulting in decrease in haemoglobin level, decrease in RBC size and decrease in MCH, MCHC & MCV. This is evident from this current study that the values of MCV (r= -0.532, p= 0.001), MCH (r= -0.301,p=0.002), MCHC (r= -0.479, p=0.001), indicating that as BMI is increasing, all these values are decreasing due to iron deficiency causing iron deficiency microcytic anemia.

CONCLUSIONS

In this study it was observed that there was significant positive correlation between Body mass index(BMI) & RBC, because growth factors like interleukin-1, interleukin-6, & GM-CSF required by multipotent stem cells to convert them into myeloid progenitor cells are secreted by adipose tissue in high quantity.

There is a significant negative correlation between BMI and Mean corpuscular volume(MCV), Mean corpuscular haemoglobin(MCH)& Mean corpuscular haemoglobin concentration(MCHC) is noted, because of hepcidin release by adipocytes and also increased release of hepcidin by liver due to it's transcriptional activation. Hepcidin blocks ferroportin which is essential for the transfer of iron from enterocyte into bloodstream. Now because of increased hepcidin it is causing less iron available for haemoglobinization, resulting in low values of MCV, MCH & MCHC. From this study it is suggesed that increase in BMI plays a significant role in alteration of normal haematological parameters and can alter physiological functioning in the individuals.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

A cross- sectional study was conducted in 100 healthy individuals with different body mass indices in the age group of 18-35 years. The study was conducted at Osmania Medical College, Hyderabad, Telangana and Institutional ethics committee approval was obtained (vide ECR/300/Inst/AP/2013/RR-16 Regd No.20116001004D).

LIST OF ABBREVIATIONS

BMI – Body Mass Index
RBC –Red Blood Corpuscles
MCV – Mean Corpuscular Volume
MCH – Mean corpuscular Haemoglobin
MCHC – Mean corpuscular Haemoglobin Concentration

DATA AVAILABILITY

Readers can access through
https://docs.google.com/spreadsheets/d/1fNvLXiuIfCY7B1d4IYM4BdF4P6gg-bObP8mlvoM0OU0/edit?usp=sharing
or
https://1drv.ms/x/s!AiR5MLM_t91oiH3ZtoON4f7L6bmM?e=pdsIIv

CONFLICTS OF INTEREST

"The author(s) declare that there is no conflict of interest regarding the publication of this paper".

FUNDING STATEMENT

No funding provided

AUTHOR'S CONTRIBUTIONS

Dr. Anitha. Ranalyzed the data and interpreted the student's data regarding body mass index and indices of red blood corpuscles. Dr. Geetha shavali was contributed in writing the manuscript. All authors read and approved the final manuscript.

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Professor Dr.A.Santa Kumari,
My associate & assistant professors,
Fellow postgraduate students,
Lab & technical personnel,
Study subjects from Osmania medical college.

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