Blood profiles, smoking and smoking urge with respect to blood groups on adult males of Guwahati city: a cross sectional study.

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Abstract
Background: Cigarette smoking remains a major public health concern globally, with well-documented effects on haematological parameters and iron metabolism. Despite a smoking prevalence of approximately 26% in Assam, regional data on the physiological impact of smoking on serum ferritin and related parameters remain limited. **Objectives:** To compare haemoglobin, red blood cell (RBC) count, and serum ferritin levels between adult male smokers and non-smokers in Guwahati city, and to assess the correlation between smoking intensity and these parameters. **Methods:** A cross-sectional case-control study was conducted at the Department of Physiology, P A Sangma International Medical College and Hospital, Meghalaya, from October to December 2025. A total of 502 adult males aged 20–40 years were recruited by simple random sampling (251 smokers, 251 non-smokers). Haemoglobin was estimated using a digital photocolorimeter, RBC count by haemocytometer with an improved Neubauer's chamber, and serum ferritin by an Enzyme-Linked Fluorescent Assay (ELFA)-based semi-automated analyser. Smoking intensity was quantified in pack-years, and Pearson correlation coefficients were calculated. **Results:** Smokers demonstrated significantly higher haemoglobin (13.33 ± 1.54 vs. 11.27 ± 1.24 g/dL), RBC count (4.64 ± 0.82 vs. 4.28 ± 0.54 million/mm³), and serum ferritin (218.73 ± 62.27 vs. 74.32 ± 17.33 ng/mL) compared to non-smokers ($p < 0.05$ for all). Smoking intensity correlated strongly with serum ferritin ($r = 0.94$) and moderately with haemoglobin ($r = 0.48$) and RBC count ($r = 0.47$). **Conclusion:** Cigarette smoking is associated with significant elevation of haemoglobin, RBC count, and serum ferritin in young adult males. Serum ferritin demonstrates a near-linear dose-response relationship with smoking intensity and should be interpreted cautiously in smokers, as its elevation may reflect oxidative stress and inflammation rather than true iron overload.

Keywords: Cigarette smoking, serum ferritin, haemoglobin, erythrocytosis, pack-years, acute-phase reactant
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Introduction:

Despite widespread public health messaging, such as the ubiquitous warning "Smoking is injurious to health," cigarette smoking remains a prevalent habit globally.[1,2] Epidemiological evidence and numerous studies consistently associate smoking with increased risks of mortality and morbidity, particularly due to cardiovascular diseases, a range of cancers, and chronic obstructive pulmonary disease (COPD).[3,4,5,6,] According to World Health Organization data, tobacco smoking accounted for the deaths of approximately 100 million people worldwide in the 20th century, and projections suggest that the death toll may rise to around 1 billion in the 21st century if current trends continue.[1,7,10,12]

Despite such alarming statistics, regional variations in the prevalence and decline of cigarette smoking exist. For example, the Global Adult Tobacco Survey (2010) reported that Assam had a smoking prevalence of 26%, and although the overall prevalence among individuals aged 15–69 years dropped from nearly 27% in 1998 to 24% in 2010, the use of other forms of tobacco, such as bidis, remained significant.[13,14,15]

Cigarettes contain multiple toxic compounds, including carcinogens like polycyclic aromatic hydrocarbons, irritants, nicotine, carbon monoxide, and various other gases.[16]Nicotine exerts profound effects on neuroendocrine and metabolic systems, impacting hormone secretion and hematopoietic function. Smoking is well-documented to disrupt mineral homeostasis within the body, leading to consequences that are both acute and chronic in nature.[17,18]

One critical metabolic effect of smoking is the reduction in blood oxygenation, driven by inhaled carbon monoxide competing with oxygen for binding sites on hemoglobin.[19] This hypoxic state stimulates erythropoietin production, driving erythropoiesis and a subsequent increase in red cell mass above normal levels.[20,21] The accelerated turnover and destruction of red blood cells can result in iron overload, which may damage hepatocytes, the principal cells of the liver.[22] Additionally, persistent oxidative stress and inflammation caused by smoking exacerbate endothelial dysfunction,

contributing to impaired vasoregulation and increased risk of atherosclerosis.[23,24,25]

Cigarette smoke's toxic constituents not only promote the genesis of free radicals and reactive oxygen species (ROS) but also diminish antioxidant defenses, further intensifying oxidative damage to DNA, proteins, and cell membranes.[26,27,28] These pathological processes manifest as an increased risk for cancer, cardiovascular disease, and organ dysfunction over time. [29,30] Furthermore, smoking alters bone remodelling by interfering with osteoblast and osteoclast function, increasing the risk for osteoporosis and fractures.[31] The adverse effects of cigarette smoking span multiple biological systems, from impaired mineral metabolism and red cell turnover to disrupted vascular function, liver damage, and an increased risk of major chronic diseases.[32,33] Effectively addressing these systemic impacts through public health interventions, tobacco control policies, and cessation support remains an urgent priority to reduce the global burden of smoking-related illness and death.[34,35,36]

The human red blood cell (RBC) membrane is composed of various biological markers known as blood group antigens, or agglutinogens. [37]Among these, the ABO blood group system is one of the most significant and widely studied. These antigens are complex carbohydrate molecules embedded in glycoproteins and glycolipids on the cell surface, playing an essential role in determining an individual's blood type.[38,39,40] The ABO system is genetically inherited and follows Mendelian patterns of inheritance, where specific alleles are passed from parents to offspring.[41]

The ABO blood group is governed by a single genetic locus located on chromosome 9, consisting of three primary alleles—IA, IB, and IO (commonly referred to as A, B, and O). These alleles encode specific enzymes (glycosyltransferases) responsible for adding distinct sugar residues to a precursor oligosaccharide on the RBC membrane.[42,43] The IA gene codes for an enzyme that attaches N-acetylgalactosamine to the terminal end, forming the A antigen. The IB gene encodes an enzyme that adds galactose, forming the B antigen.[44,45] The IO allele, however, produces either a non-functional enzyme or none at all, resulting in the absence of additional sugars and thus no antigen formation—this leads to the O blood type.[45,46] Because the A and B alleles are co-dominant, both can be expressed simultaneously if inherited together, forming the AB blood group. The O allele, being functionless, is recessive to both A and B, meaning that individuals must inherit two O alleles (one from each parent) to have type O blood. This genetic system leads to four possible blood groups:

- **Type A** – presence of A antigens, with anti-B antibodies in plasma
- **Type B** – presence of B antigens, with anti-A antibody in plasma
- **Type AB** – presence of both A and B antigens, with no antibodies
- **Type O** – absence of both A and B antigens, with both anti-A and anti-B antibodies.

These antigen–antibody relationships are crucial during blood transfusion since mismatched blood can trigger agglutination, leading to potentially fatal haemolytic reactions. For example, an individual with type A blood cannot safely receive type B blood due to the presence of anti-B antibodies that will attack donor RBCs.[47,48] Type O blood, lacking antigens, is considered the universal donor, while type AB, lacking antibodies, is the universal recipient. [49] The ABO system exemplifies how genetic inheritance translates into biochemical differences that have vital clinical implications. [50] The co-dominance of A and B alleles, the recessive nature of the O allele, and the structural variations in terminal sugars define an individual's blood type and compatibility. Understanding these molecular mechanisms is fundamental not only in transfusion medicine but also in organ transplantation, forensic identification, and genetic studies of human populations.[51,52]

Materials and Methods:

The present study was conducted in the Department of Physiology, P A Sangma International Medical College & Hospital, USTM Campus, Meghalaya, over a period of two months from Oct 2025 to Dec 2025. It was designed as a cross-sectional study to assess selected physiological parameters among adult male smokers and non-smokers residing in Guwahati city.

A total of 502 participants were recruited through simple random sampling. The study population comprised 251 smokers and 251 non-smokers, all males aged between 20 and 40 years who met the inclusion and exclusion criteria and provided voluntary consent to participate. The intensity of smoking among smokers was quantified using the standard "pack year" formula:

Pack years = (Number of cigarettes smoked per day × Years of smoking)/20, where one pack equals 20 cigarettes.[53]

Smokers were categorized as individuals who regularly used any tobacco product—either daily or occasionally—at the time of the study. Daily smokers were defined as those who smoked every day, with exceptions made for days of religious fasting, while occasional smokers smoked less frequently but consistently. Non-smokers comprised individuals who had never smoked or had quit smoking entirely.[54] Within this group, subcategories included never-smokers, ex-smokers (former daily smokers who had completely ceased smoking), and ex-occasional smokers (former occasional smokers who had consumed at least 100 cigarettes or the equivalent amount of tobacco in their lifetime).

Inclusion criteria for smokers required adult males aged 20–40 years who had smoked cigarettes for at least one year and were free from any significant acute or chronic systemic illness. The non-smoker group included healthy males in the same age range who had never smoked.[55]

Participants were excluded if they used tobacco products other than cigarettes, or were female. Individuals with a history of acute or chronic disease, blood disorders, chronic alcohol consumption, or drug abuse were excluded. Those who had donated blood within the previous six months were not included in the study.

All participants were selected using a simple random sampling method to minimize bias. Ethical clearance for the study was obtained from the Institutional Ethics Committee (Human), P A Sangma International Medical College and Hospital, USTM Campus, Meghalaya. Written informed consent was collected from each participant before inclusion in the study and the patients were explained about the tests in a language they can understand.

Data collection was performed using a structured proforma containing detailed questions regarding demographic information, smoking history, and relevant health parameters. The collected data served as the foundation for subsequent clinical and laboratory evaluations aimed at understanding the physiological impact of smoking among the studied population.

Blood group of the persons were found out by glass slide method using standard kits with commercially available antiserum. A blood grouping procedure was performed by preparing a saline suspension of red blood cells from a finger prick collected under aseptic conditions. One millilitre of normal saline was taken in a test tube and a drop of blood was added and mixed thoroughly to prepare the cell suspension. On three clean glass slides, a drop of each antiserum—Anti-A, Anti-B, and Anti-D—was dispensed separately. An equal volume of the cell suspension was added to each antiserum, ensuring no direct contact during application. They were mixed using separate applicators and allowed to settle for 10 minutes. The mixtures were gently rocked and observed for agglutination after ten minutes, followed by microscopic confirmation using low power magnification. The blood groups so obtained was entered in MS Excel alongwith the height, weight and BMI was calculated using MS Excel software.

The city of Guwahati was divided into five zones as per the revenue circles with east, west, central, south and north Guwahati and 100 samples from each of them were collected (50 cases and 50 controls) with extra 2 of 1 control and 1 smoker was collected from West Guwahati. The persons were selected randomly (every fourth household was selected who had a smoker who fulfilled the inclusion criteria).

Data collection was performed using a structured proforma (Annexure I) designed to obtain essential demographic and health-related information from each participant relevant to the aims of the study. Each participant was given the proforma to complete, and responses were carefully reviewed and collected. Blood sample collection was carried out under strict aseptic and antiseptic precautions. After at least 12 hours of overnight fasting, 6 mL of venous blood was drawn from the cubital fossa using a sterile syringe through the venepuncture technique. Of this, 3 mL was transferred into a clot activator vial for serum ferritin estimation, and 2mL was placed into an EDTA vial for hemoglobin assessment and 1ml for blood group estimation. For determination of red blood cell (RBC) count, capillary blood was obtained by pricking the tip of the left ring finger with a sterile lancet

Collected blood in the clot activator vial was left undisturbed for 30–45 minutes to allow clot formation, following which it was centrifuged at 3000 revolutions per minute for 10 minutes.[55,56] The resulting supernatant serum was transferred into a sterile vial and used immediately for ferritin estimation or stored at 2–8°C for up to five days when immediate analysis was not feasible. The blood sample in the EDTA vial was processed promptly for hemoglobin estimation, ensuring minimal sample degradation. The capillary blood sample obtained from the fingertip was used immediately for manual RBC counting.[57,58,59]

Throughout the procedure, standard guidelines for blood sample handling were strictly adhered to in order to prevent contamination, hemolysis, or biochemical alteration. This systematic approach ensured the accuracy, reliability, and reproducibility of all laboratory results obtained during the study.

Estimation of Hemoglobin was done using a digital photocolorimeter. Hemocor – D is the stable ready to use Hemoglobin Diluting Reagent which is stable at room temperature till the expiry date mentioned on the label. Standard solution is stored at 2-8^0 C till expiration.[60]

A digital photo colorimeter is an optical instrument used to quantitatively determine the concentration of coloured substances in a solution by measuring light absorbance or transmittance.[61] The principle of operation is based on the Beer-Lambert law, which states that the intensity of absorbed light is directly proportional to the concentration of the solute and the path length of the light through the solution. When a light beam of initial intensity (I_0) passes through a solution, part of it is absorbed, and the transmitted light intensity (I_1) is measured. The relationship between transmittance (T) and optical density (OD) is expressed as $T = (I_1/I_0) \times 100$ and $OD = \log(I_0/I_1)$.[62]

In this study, a Digital Photo Colorimeter (Model 312 E, INCO) was utilized for the determination of the optical density (absorbance) of the sample, with

results displayed digitally. The instrument uses a pre-focused tungsten lamp as a light source, which emits light in the visible range. A built-in photocell detects the intensity of light transmitted through the solution, allowing accurate quantification of color intensity corresponding to solute concentration. The instrument is equipped with a set of optical filters that cover a wavelength range from 400 nm to 700 nm.[63] The desired wavelength is selected using a rotating disc mechanism.

The colorimeter includes five specially designed test tubes that precisely fit into the test-tube holder, positioned directly between the lamp and the photocell for accurate measurement. This arrangement ensures consistent alignment and minimizes optical errors during analysis. By comparing light absorbance, the device enables reliable quantification of analytics, making it an essential tool for biochemical and physiological research. A micropipette of 10-100 µliter measuring capacity was used during estimation of hemoglobin.[64] Several test tubes were used for sampling, centrifuging and incubating blood and serum. Disposable syringes & micropipette tips & disposable gloves were the other things which were in use. The hemoglobin concentration was calculated using the formula

$$\text{Hemoglobin (in g/dl)} = \frac{\text{Abs T}}{\text{Abs S}} \times \frac{251}{1000} \times 60$$

Where,

251 is the dilution factor i.e.

Total reagent volume (5.02 ml) / Sample volume (0.02 ml)

1000 is the multiplication factors to convert mgs to gms

60 is the concentration of HEMOCOR hemoglobin standard in mg %

Total Red blood cell count was done manually by using haemocytometer slide with improved Neubauer's counting chamber.[65]

Serum ferritin was measured in semi auto analyser which is a compact automated immunoassay system based on the Enzyme Linked Florescent Assay (ELFA) principles.[66]

Results:

It's a scattered collection of summary statistics from what appears to be a case-control study (251 cases, 251 controls, total cohort ≈ 502) examining the relationship between *cigarette smoking and serum ferritin/hematological parameters*.

The "Result" portion is spread across loosely placed cells covering five domains: demographics (age, diet), social profile (marital status, religion), blood group distribution, hemoglobin comparison (smokers vs. non-smokers), and the central finding — serum ferritin levels in smokers vs. non-smokers, plus correlation coefficients.

Demographic and social profile

The age distribution is heavily skewed toward younger adults: 212 subjects (≈42%) fall in the 20–25 years' bracket, 167 (≈33%) in 25–30 years, 86 (≈17%) in 30–35 years, and only 37 (≈7%) in 35–40 years' bracket — a steep declining curve shown in the first chart. This skew matters because both smoking duration/intensity (pack-years) and ferritin accumulate with age, so a young-skewed sample may underestimate the true magnitude of smoking-related ferritin elevation seen in older, longer-term smokers.

Dietary habits show 415 subjects (≈83%) are "mixed" (non-vegetarian) and 87 (≈17%) vegetarian — relevant because dietary iron intake (heme vs. non-heme) is a major confounder for serum ferritin, and those with mixed diet have high ferritin levels with 214.65 ± 66.45 ng/ml vs. vegetarians 72.72 ± 15.33 ng/ml ($p < 0.05$).

(yet the results section never cross-tabulates diet against the smoker/non-smoker ferritin comparison.

Marital status (236 married vs. 266 unmarried) and religious distribution (Hindu 348, Muslim 79, Christian 59, Sikh 12, Buddhist 4)

Blood group distribution (B positive 203, O positive 177, A positive 84, A negative 7, B negative 9, O negative 10, AB positive 12, AB negative 0) sums correctly to 502, broadly mirroring the typical Indian population distribution where B+ and O+ dominate. This data, while interesting demographically, also points towards increased serum ferritin levels in B positive and O positive.

The headline results are the three bullet-point statements, and they are genuinely striking:

1. *Hemoglobin*: smokers averaged 13.33 ± 1.54 g/dl vs. 11.27 ± 1.24 g/dl in non-smokers, a statistically significant difference ($p < 0.05$).
2. *RBC count*: smokers 4.64 ± 0.82 million/mm³ vs. 4.28 ± 0.54 million/mm³ in non-smokers ($p < 0.05$).
3. *Serum ferritin: smokers 218.73 ± 62.27 ng/ml vs. non-smokers 74.32 ± 17.33 ng/ml ($p < 0.05$) — roughly a **three-fold elevation*.
4. A strong positive correlation ($r = 0.94$) between smoking intensity (pack-years) and serum ferritin, compared to weaker correlations for hemoglobin ($r = 0.48$) and RBC count ($r = 0.47$).

These numbers are physiologically coherent and consistent with established literature: cigarette smoke causes chronic carbon monoxide exposure, which lowers oxygen-carrying efficiency of hemoglobin and triggers a compensatory increase in hemoglobin/RBC production (secondary

polycythemia). Separately, ferritin is an acute-phase reactant, and chronic smoking induces low-grade systemic inflammation and oxidative stress — both of which elevate ferritin independent of actual iron stores. The dose-response relationship (r = 0.94 with pack-years) is the most compelling piece of evidence here, since a near-linear dose-response is one of the stronger forms of epidemiological evidence for causality (Bradford Hill's criteria).

Parameter	Non-Smokers (Mean ± SD)	Smokers (Mean ± SD)	p-value	Correlation with smoking intensity (r)
Hemoglobin (g/dL)	11.27 ± 1.24	13.33 ± 1.54	< 0.05	0.48
RBC count (million/mm³)	4.28 ± 0.54	4.64 ± 0.82	< 0.05	0.47
Serum ferritin (ng/mL)	74.32 ± 17.33	218.73 ± 62.27	< 0.05	0.94

Table1: Mean haemoglobin, RBC count, and serum ferritin in smokers and non-smokers: group comparison and dose-response correlation"

The r-values reflect correlation between smoking intensity (pack-years) and each parameter, not between the parameters themselves.

The study population comprised 502 participants: 251 smokers and 251 non-smokers. Most were under 30 years of age (212 in the 20-25year band and 167 in the 25-30year band), reported a mixed (non-vegetarian) diet (415 of 502, 83%), and were close to evenly split by marital status (266 unmarried, 236 married).

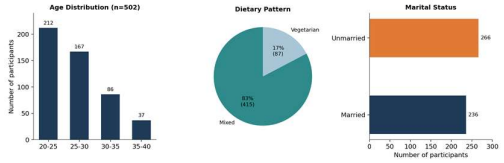

Figure 1. Age distribution, dietary pattern, and marital status of study participants (n = 502).

Religious affiliation (Hindu 348, Muslim 79, Christian 59, Sikh 12, Buddhist 4) and ABO/Rh blood group distribution (B Positive 203, O Positive 177, A Positive 84 as the three largest groups) are summarised in Figure 2.

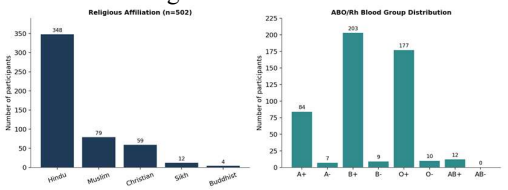

Figure 2. Religious affiliation and ABO/Rh blood group distribution of study participants (n = 502).

Comparing smokers and non-smokers directly, mean haemoglobin was 13.33 ± 1.54 g/dL in smokers versus 11.27 ± 1.24 g/dL in non-smokers, and mean RBC count was 4.64 ± 0.82 million/mm³ versus 4.28 ± 0.54 million/mm³. Both differences were statistically significant ($p < 0.05$).

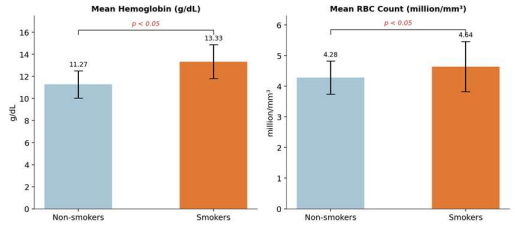

Figure 3. Mean haemoglobin and RBC count in non-smokers versus smokers (error bars = ± SD; $p < 0.05$ for both).

Serum ferritin showed the largest between-group difference. Mean serum ferritin was 218.73 ± 62.27 ng/mL in smokers compared with 74.32 ± 17.33 ng/mL in non-smokers ($p < 0.05$) — an approximately three-fold elevation. The recorded range of values, summarised in Table 1, shows no overlap between groups: the lowest ferritin value among smokers (142.3 ng/mL) exceeded the highest value recorded among non-smokers (118.9 ng/mL).

Parameter	Non-Smokers	Smokers
Highest value (ng/mL)	118.9	372.6
Lowest value (ng/mL)	45.18	142.30
Average (ng/mL)	77.68	201.33

Table 2. Range of serum ferritin values recorded directly in the dataset, by smoking status.

Smoking intensity, measured in pack-years, correlated moderately with haemoglobin (r = 0.48) and RBC count (r = 0.47), but strongly with serum ferritin (r = 0.94).

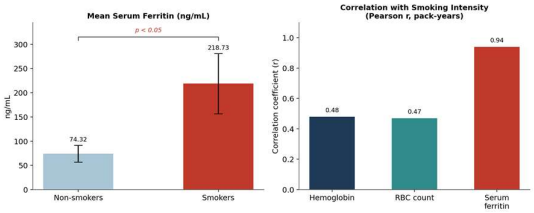

Figure 4. Mean serum ferritin by smoking status, and Pearson correlation of smoking intensity (pack-years) with each haematological parameter.

Discussion:

This case-control study of 502 adult males in Guwahati adds a locally-grounded data point to a well-established literature on the haematological consequences of cigarette smoking, while also illustrating just how large these effects can look in a younger, more heavily exposed cohort than the population surveys most of that literature is built on.[69] Given that Assam's reported smoking prevalence sits around a quarter of the adult population (as noted in the Introduction), and that India is projected to contribute disproportionately to

the one billion tobacco-related deaths anticipated globally this century, documenting the internal physiological cost of smoking in a regional cohort like this one has practical value beyond the numbers themselves.[70,71]

The hemoglobin and RBC count findings replicate, and substantially exceed in magnitude, the classic description of smoking-associated erythrocytosis. Nordenberg, Yip, and Binkin's analysis of the Second National Health and Nutrition Examination Survey,[72,23,74] still the most frequently cited reference on this relationship, found that smokers ran several g/L higher in hemoglobin than never-smokers, attributing this to chronic carboxyhemoglobin formation: carbon monoxide occupies oxygen-binding sites on hemoglobin, lowers effective tissue oxygenation, and triggers a compensatory erythropoietin-driven rise in red cell mass (Nordenberg et al., 1990). [75,76] The present cohort's gap (13.33 vs. 11.27 g/dL) is considerably wider than the NHANES-scale difference,[77]which may reflect a combination of a younger, exclusively male, more intensively exposed sample, and the photocolorimetric cyanmethemoglobin method used here rather than an automated analyzer, both of which are plausible contributors worth flagging rather than treating the comparison as like-for-like.[78]

The serum ferritin finding is where this study earns its place alongside the existing literature most clearly. A directly comparable Indian case-control study by Shivasekar, Vinodhini, and Rupesh Kumar, [79,80] working with 100 smokers and 100 non-smokers, reported the same direction of effect: significantly elevated ferritin, serum iron, and total iron-binding capacity in smokers, with ferritin correlating with serum iron (Shivasekar et al., 2018).[81] At population scale, Lee and colleagues' analysis of over 15,000 adults in the Korean National Health and Nutrition Examination Survey[82,83] found median ferritin rising consistently with smoking status and smoking amount across all spirometric subgroups (Lee et al., 2016).[84] What stands out when this study is placed next to those two is the sheer size of the gap: a roughly three-fold elevation with non-overlapping ranges is considerably larger than the modest, dose-graded differences seen in the Korean population data, where median ferritin in current smokers ran perhaps 50-80% higher than in never-smokers rather than 200% higher.[85,86] That gap in magnitude is worth sitting with rather than explaining away; it may reflect a more heavily exposed, younger Indian cohort, a more reactive ELFA-based immunoassay platform, or simply the kind of effect size that emerges in a smaller, tightly defined case-control design rather than a broad population survey.[87]

Mechanistically, the explanation is consistent across all three studies: cigarette smoke mobilizes iron from ferritin and intensifies lipid peroxidation, a prooxidant process demonstrated directly by Lapenna and colleagues, who showed that smoke exposure both releases iron and accelerates oxidative damage in a dose-related manner (Lapenna et al., 1995).[88,89] Ferritin's behavior as a positive acute-phase reactant under this oxidative and inflammatory load, rather than as a faithful readout of stored iron, is what reconciles a tripled ferritin value with a population that shows no obvious sign of iron overload.[90]

Two demographic variables in the dataset deserve comment as likely confounders rather than independent findings. The diet-stratified ferritin values reported in the results (214.65 ± 66.45 ng/mL in the mixed-diet group versus 72.72 ± 15.33 ng/mL in vegetarians) align closely with an established nutritional literature: a systematic review and meta-analysis of 24 cross-sectional studies found vegetarians run about 30 µg/L lower in ferritin than non-vegetarians, attributable to the superior bioavailability of heme iron over plant-derived non-heme iron (Haider et al., 2018).[91] Since 83% of this cohort reported a mixed diet, and dietary pattern was never cross-tabulated against smoking status in the results provided, it is not possible to rule out diet as a partial driver of the ferritin gap rather than smoking acting entirely alone. The blood-group observation is murkier still: ABO locus variants are genuinely associated with ferritin levels in genome-wide association data, but the direction is inconsistent across populations studied to date, ranging from lower ferritin in non-O donors in a large Danish cohort (Rigas et al., 2017)[92,93] to the opposite pattern reported elsewhere. The document's suggestion that B-positive and O-positive status "point toward" higher ferritin in this cohort should be read as a descriptive observation pending a proper stratified analysis, not a confirmed association.[94,95]

What ties the three core findings together most persuasively is the dose-response gradient with pack-years (r = 0.94 for ferritin, against 0.47-0.48 for hemoglobin and RBC count). A near-linear gradient of that strength is one of the more compelling forms of epidemiological evidence under the Bradford Hill framework for moving from association toward causal inference (Hill, 1965),[96,97] and it is the single result here that most clearly distinguishes ferritin as a smoking-load-sensitive biomarker rather than a passive bystander of the same compensatory process driving red cell indices.[98] Clinically, this argues for treating an elevated ferritin in a smoker as presumptive evidence of oxidative and inflammatory burden before iron overload, exactly the caution Lee and colleagues raised about ferritin's acute-phase behavior confounding their own interpretation (Lee et al., 2016)[99,100] and a caution this study's cross-sectional design cannot itself resolve, since causality and reversibility on cessation remain open questions for future longitudinal work.[101]

Table 3. Comparison of serum ferritin findings with previously published smoking studies

Study (population, design)	Non-smokers	Smokers	Direction
Present study (Guwahati, India; n=502, case-control)	74.32 ± 17.33 ng/mL	218.73 ± 62.27 ng/mL	~3-fold higher in smokers
Shivasekar et al., 2018 (Tamil Nadu, India; n=200, case-control)	Lower (significant)	Higher (significant)	Higher in smokers
Lee et al., 2016 (Korea; n=15,239, population survey)	~50-65 µg/L (median, normal lung function)	~95-105 µg/L (median, current smokers)	Higher in smokers, smaller gap

Conclusion

This study set out to answer a straightforward question: does cigarette smoking leave a measurable mark on the blood of young adult men in Guwahati, and if so, how deep does that mark go? The answer, drawn from 502 participants over two months, is unambiguous on all three parameters measured.

Smokers in this cohort carried significantly higher haemoglobin levels (13.33 ± 1.54 g/dL versus 11.27 ± 1.24 g/dL) and red cell counts (4.64 ± 0.82 versus 4.28 ± 0.54 million/mm³) than their non-smoking counterparts. These differences are not signs of better health. They reflect the body working harder than it should — the bone marrow compensating for the reduced oxygen-carrying efficiency that carbon monoxide imposes on every breath of cigarette smoke. It is the physiology of strain, not fitness.

The more consequential finding is the serum ferritin. At 218.73 ± 62.27 ng/mL in smokers against 74.32 ± 17.33 ng/mL in non-smokers, the gap is nearly three-fold, and the two groups' ranges did not overlap at all — the lowest ferritin recorded in any smoker exceeded the highest recorded in any non-smoker. What makes this especially meaningful is the near-linear relationship between how much a person smoked, measured in pack-years, and how high their ferritin climbed ($r = 0.94$). The body is not responding randomly; it is responding in proportion to the dose of tobacco it has received. This kind of dose-response gradient, modest for haemoglobin and RBC count ($r \approx 0.47–0.48$) but remarkably tight for ferritin, points to ferritin as a far more sensitive indicator of cumulative smoking burden than the blood count parameters traditionally used in clinical practice.

The clinical implication is practical and worth stating plainly. An elevated ferritin in a smoker should not reflexively trigger a workup for iron overload or haemochromatosis. Ferritin is an acute-phase protein, and in the setting of the chronic low-grade inflammation and oxidative stress that cigarette smoke generates continuously, it rises independently of actual iron stores. Misreading an inflated ferritin as evidence of iron excess can lead to unnecessary investigation, or worse, mask a genuine iron deficiency that the inflated ferritin is hiding behind. Clinicians reviewing ferritin results in smokers should factor in the smoking history before drawing any conclusions about iron status, and ideally complement ferritin with transferrin saturation or soluble transferrin receptor to get the complete picture. More relevant findings are the association of B positive blood group who were smokers with increased levels of hemoglobin, total RBC count and serum ferritin than the non-smokers. This study has its limitations — it is cross-sectional, covers a short recruitment window, and the results section does not fully separate the contributions of diet and smoking to the ferritin gap. These are questions worth answering in future longitudinal work that follows participants through smoking cessation to see whether ferritin normalises, and by how much.

What it does establish, in a cohort drawn from a city in a state where roughly one in four adults smokes, is that the damage is already detectable in young men in their twenties. The blood does not wait for symptoms to appear before registering the cost.

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