

The Influence of Physical Activity on Inflammatory Biomarkers and Body Composition: A Literature Review of NLR, PLR, and Anthropometric Correlates in Young Adults

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ABSTRACT

Physical inactivity, sedentary behaviour, and excess adiposity are increasingly recognized as important contributors to low-grade systemic inflammation in young adults. This literature review examines the influence of regular physical activity on hematological inflammatory biomarkers, particularly the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR), and evaluates their relationships with body composition indicators such as body mass index (BMI), waist circumference (WC), and waist-to-hip ratio (WHR). NLR and PLR are inexpensive, accessible, complete blood count-derived indices that reflect interactions among innate immunity, adaptive immunity, platelet activity, and physiological stress. Their potential usefulness in apparently healthy populations has attracted attention because subclinical inflammation may develop before overt metabolic or cardiovascular disease appears. The review synthesizes current evidence concerning reference ranges of NLR and PLR in healthy young adults, with particular consideration of regional and ethnic variation. It also explores the biological pathways through which physical activity may influence inflammatory regulation, including improved insulin sensitivity, reduced visceral adiposity, enhanced skeletal-muscle signalling, and better metabolic control. The limitations of BMI as an isolated measure of adiposity are discussed, while WC and WHR are considered complementary markers of central fat distribution and cardiometabolic risk. Particular attention is given to the interaction between physical activity and adiposity, as exercise may exert anti-inflammatory effects independently of weight loss, although body composition may also mediate part of this relationship. The review further considers the sensitivity and practical value of NLR and PLR compared with established inflammatory markers such as high-sensitivity C-reactive protein and interleukin-6. Important confounding factors, including age, sex, academic stress, sleep quality, smoking, diet, medication use, recent infection, and acute exercise, are also examined. Methodological inconsistencies in defining active and sedentary behaviour, blood-sampling conditions, and anthropometric assessment remain major challenges. Overall, NLR and PLR may serve as useful supportive indicators of low-grade inflammation when interpreted alongside anthropometric and lifestyle variables. However, standardized population-specific research, particularly among Indian medical students, is required before these ratios can be applied confidently in preventive screening or early risk assessment.

Keywords: Physical activity; Neutrophil-to-lymphocyte ratio; Platelet-to-lymphocyte ratio; Body composition; Young adults

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INTRODUCTION

Low-grade systemic inflammation has become an important concern in preventive medicine because it may

develop silently before the appearance of clinically evident metabolic or cardiovascular disease. Young adulthood is generally regarded as a period of good health; however,

increasing sedentary behaviour, reduced structured exercise, academic pressure, irregular sleep, unhealthy dietary patterns, and progressive central adiposity may initiate inflammatory alterations at an early age. These alterations may not produce obvious symptoms or abnormal conventional clinical findings. Consequently, there is growing interest in accessible biomarkers that can identify subtle disturbances in immune and metabolic homeostasis among apparently healthy young adults. The complete blood count is one of the most commonly performed and economically accessible laboratory investigations. In addition to providing absolute numbers of neutrophils, lymphocytes, platelets, monocytes, and other blood cells, it permits the calculation of several composite inflammatory indices. Two indices that have received considerable attention are the neutrophil-to-lymphocyte ratio and the platelet-to-lymphocyte ratio. The neutrophil-to-lymphocyte ratio, commonly abbreviated as NLR, is calculated by dividing the absolute neutrophil count by the absolute lymphocyte count. The platelet-to-lymphocyte ratio, or PLR, is obtained by dividing the platelet count by the absolute lymphocyte count. Both measures combine information from different components of the immune and haematological systems and may therefore provide broader information than an isolated cell count [1]. Neutrophils are essential components of innate immunity and respond rapidly to infection, tissue injury, physiological stress, and inflammatory signals. Lymphocytes are central to adaptive immune regulation and may decrease temporarily in response to stress-related hormonal and immune changes. An increase in neutrophils accompanied by a relative decrease in lymphocytes can therefore raise NLR and suggest a shift towards an inflammatory or stress-responsive state. Platelets also have functions extending beyond haemostasis. They participate in inflammatory signalling, endothelial interactions, immune-cell recruitment, and thrombo-inflammatory processes. PLR may consequently reflect the balance between platelet-associated inflammatory activity and lymphocyte-mediated immune regulation [2]. Although NLR and PLR were initially investigated primarily in patients with infections, malignant diseases, cardiovascular disorders, and other clinical conditions, their potential relevance in healthy populations is increasingly recognised. Their interpretation in young adults, however, requires caution. A value considered elevated in one population may fall within the expected distribution in another because blood-cell counts are influenced by age, sex, ethnicity, genetic background, altitude, nutritional status, laboratory instrumentation, medication use, menstrual status, smoking, recent infection, sleep, psychological stress, and physical exertion. Reference intervals established in one geographical region cannot automatically be applied to all populations [3]. Large population-based investigations have attempted to establish reference intervals for NLR, PLR, and related systemic inflammatory indices. These investigations demonstrate the importance of population-specific reference values and appropriate partitioning according to demographic characteristics. They also show why a universal threshold should not be used without considering

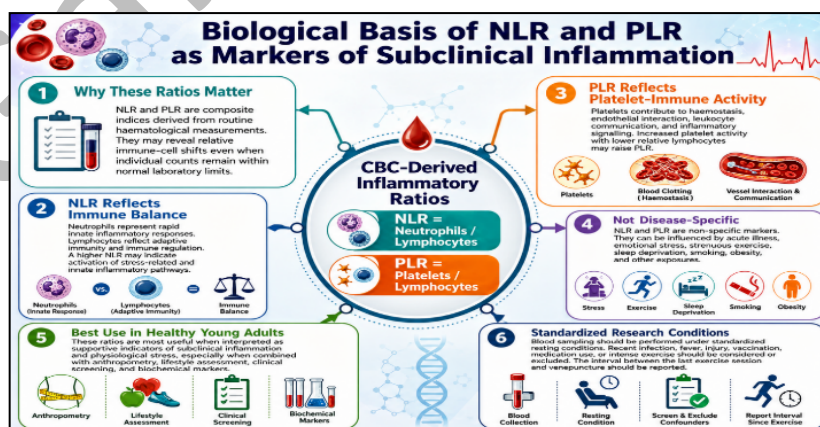
the health status and background of the population being examined. For research involving young Indian adults, particularly medical students, the absence of a widely accepted local reference framework represents a significant methodological challenge [4]. Physical activity is another major determinant of immune and metabolic health. Regular moderate activity supports cardiovascular fitness, glucose regulation, endothelial function, skeletal-muscle metabolism, and healthy body composition. It may also regulate chronic inflammatory signalling through mechanisms involving reduced visceral fat, improved insulin sensitivity, altered adipokine secretion, antioxidant adaptation, and repeated anti-inflammatory responses generated by contracting skeletal muscle. Nevertheless, exercise is not uniformly anti-inflammatory under all circumstances. A single strenuous session can temporarily modify circulating leukocyte distribution, platelet activity, and inflammatory mediators. The timing of blood collection in relation to exercise is therefore essential when NLR and PLR are assessed. The distinction between habitual physical activity and an acute exercise response is particularly important. Habitual activity refers to the usual pattern of movement accumulated through transportation, recreation, household tasks, occupation, and planned exercise. Acute exercise refers to a specific bout of activity that may produce temporary physiological stress. Blood collected immediately after intense exercise may reflect short-term leukocyte mobilisation rather than the participant's usual inflammatory state. Research designed to compare active and sedentary young adults should therefore standardise pre-analytical conditions, including the period of rest before blood sampling, the timing of the most recent exercise session, fasting status, hydration, and time of day [5]. Body composition may mediate or modify the association between physical activity and inflammation. Adipose tissue is metabolically active and communicates with immune, vascular, endocrine, and skeletal-muscle systems. Excess adiposity, particularly abdominal or visceral fat accumulation, may promote persistent inflammatory signalling even in individuals who have not yet developed diabetes, hypertension, or another diagnosed metabolic disorder. Body mass index is widely used to classify weight status, but it does not distinguish fat mass from lean mass or identify the anatomical distribution of adipose tissue. Waist circumference and waist-to-hip ratio can provide additional information regarding central fat accumulation and cardiometabolic risk. In young adults, reliance on BMI alone may lead to incomplete interpretation. A physically trained individual may have an elevated BMI because of greater muscle mass, whereas an individual with a BMI within the conventional normal range may still have excessive central or visceral adiposity. Therefore, BMI, waist circumference, and waist-to-hip ratio should be considered complementary rather than interchangeable measurements. Evaluating these indicators together may provide a more meaningful assessment of how overall and abdominal body composition relate to NLR and PLR [6]. The relationship between physical activity, body composition, and haematological inflammation is unlikely to be linear or governed by a single mechanism. Physical

activity may influence inflammatory ratios directly through immune regulation and indirectly through reductions in adiposity. Conversely, excess body fat may reduce mobility, discourage exercise participation, disturb sleep, and contribute to metabolic dysfunction, thereby intensifying inflammation. Sex hormones, age, diet, smoking, alcohol use, recent illness, medication, menstrual-cycle phase, sleep quality, and psychological stress may further influence these relationships. Medical students constitute a particularly relevant young-adult population. They may experience prolonged sitting, examination stress, irregular sleep, restricted opportunities for exercise, and inconsistent dietary habits. These exposures can coexist even among individuals without diagnosed disease. Academic stress can activate neuroendocrine pathways that influence leukocyte trafficking and immune regulation, while sleep disruption may affect metabolic and inflammatory processes. Failure to measure these factors could produce misleading associations between physical activity, anthropometry, NLR, and PLR. Another important question concerns the potential use of NLR and PLR as low-cost screening measures. These ratios are attractive because they can be derived without additional specialised laboratory tests when a complete blood count is already available. However, accessibility does not automatically establish diagnostic validity. NLR and PLR are nonspecific indices and may change because of many temporary or unrelated conditions. Their most appropriate role in healthy young adults may therefore be as exploratory markers used alongside clinical, anthropometric, behavioural, and biochemical information rather than as independent diagnostic tests [7].

Biological Basis of NLR and PLR as Markers of Subclinical Inflammation

NLR and PLR are composite indices derived from routine haematological measurements. Their potential value arises from their ability to represent interactions among innate immunity, adaptive immunity, physiological stress, haemostasis, and inflammatory regulation. Unlike a single leukocyte or platelet count, a ratio may reveal a relative shift between cell populations even when both individual counts remain within conventional laboratory limits. This

feature makes these ratios potentially relevant to the investigation of subclinical inflammation in apparently healthy young adults. Neutrophils respond rapidly to tissue injury, microbial signals, stress hormones, and inflammatory mediators. They migrate to affected tissues, participate in phagocytosis, release antimicrobial substances, and contribute to cytokine signalling. Lymphocytes regulate antigen-specific responses, immune memory, antibody production, and cellular immune defence. A relative increase in neutrophils or decrease in lymphocytes may indicate activation of stress-responsive and innate immune pathways. NLR therefore provides an integrated representation of these opposing or complementary changes [8]. PLR adds information about platelet activity. Platelets participate in coagulation but also communicate with leukocytes and endothelial cells. They release mediators capable of influencing vascular permeability, leukocyte recruitment, and immune signalling. Increased platelet activity combined with relative lymphocyte reduction may raise PLR. However, platelet numbers are influenced by iron status, menstrual blood loss, acute infection, medication, hydration, and several haematological conditions. These influences must be considered when PLR is used in non-clinical populations [9]. Neither NLR nor PLR is specific to a single inflammatory pathway. Both can respond to acute illness, emotional stress, strenuous exercise, sleep deprivation, smoking, obesity, and other exposures. Their nonspecificity is a limitation for diagnosis but may be useful when the purpose is to describe overall physiological stress or systemic inflammatory balance. Their interpretation is strongest when measurements are repeated or integrated with anthropometry, lifestyle assessment, clinical screening, and established biochemical markers. For young-adult research, blood sampling should be performed under standardised resting conditions. Participants with recent infection, fever, injury, vaccination, medication use, or intense exercise should be excluded or assessed separately. The interval between the last exercise session and venepuncture must also be reported. Such controls help distinguish habitual low-grade inflammatory status from temporary leukocyte redistribution caused by acute physiological stress [10].



Reference Ranges for NLR and PLR in Healthy Young Adults

Establishing reference ranges is essential before NLR and PLR can be interpreted in healthy populations. A clinical threshold derived from patients with severe disease is unsuitable for identifying subtle variation among young adults. Reference intervals should ideally be developed using carefully screened participants without acute infection, chronic inflammatory disease, haematological abnormalities, major medication use, pregnancy, or other conditions capable of modifying circulating blood cells. Reference values are influenced by demographic and laboratory characteristics. Age-related immune changes may alter neutrophil, lymphocyte, and platelet distributions. Sex-related differences can result from hormonal influences, menstruation, iron balance, contraceptive use, and body-fat distribution. Ethnicity, geographical region, altitude, environmental exposure, nutrition, and genetic background may also influence haematological profiles. Therefore, reference intervals should not be transferred uncritically between countries or ethnic groups [11]. Laboratory methodology is another important source of variation. Different analysers may apply distinct technologies, calibration systems, sample-processing procedures, and quality-control practices. Pre-analytical factors such as venous sampling technique, anticoagulant type, storage duration, temperature, fasting status, hydration, and time of day can also affect measurements. Studies should report these details so that findings can be reproduced and compared. Young-adult reference intervals should ideally be partitioned by sex and evaluated across narrower age groups. Participants classified as healthy should undergo more than a brief symptom check because undiagnosed obesity, smoking, anaemia, metabolic dysfunction, or recent infection may influence the distribution. Statistical outliers should be examined carefully rather than removed automatically, since an extreme ratio may reflect an unrecognised biological or methodological factor [12]. The lack of representative South Asian reference data limits the interpretation of NLR and PLR in Indian young adults. Regional investigations should include participants from diverse social, nutritional, and geographical backgrounds. Until robust local intervals are available, studies should present the full distribution of observed ratios and avoid assigning participants to disease-oriented categories. Population-specific percentiles may be more informative than externally selected cut-offs. Reference-range research should also recognise that a statistically defined interval is not necessarily a clinical decision threshold. A reference interval describes the distribution among selected healthy individuals, whereas a decision threshold identifies a level associated with a particular clinical outcome. These concepts should remain separate when NLR and PLR are investigated as early indicators of inflammation [13].

Physical Activity, Sedentary Behaviour, and Immune Regulation

Physical activity influences immune function through repeated interactions among skeletal muscle, adipose tissue,

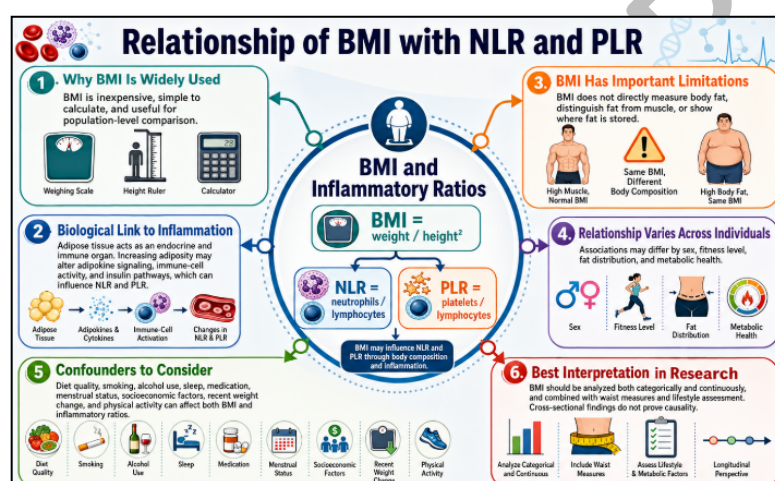
the cardiovascular system, neuroendocrine pathways, and circulating immune cells. Regular moderate activity may improve immune surveillance, metabolic regulation, vascular function, and resilience to physiological stress. Contracting skeletal muscle also acts as a signalling organ by releasing mediators that communicate with the liver, adipose tissue, and immune system. The anti-inflammatory influence of habitual activity may operate through several pathways. Physical activity can improve insulin sensitivity, support mitochondrial function, reduce oxidative stress, and limit the accumulation of visceral adipose tissue. It may also reduce the chronic inflammatory signals released by dysfunctional adipose tissue. These mechanisms suggest that active individuals could demonstrate a more balanced circulating neutrophil, lymphocyte, and platelet profile than individuals who remain sedentary for prolonged periods [14]. Sedentary behaviour is not simply the absence of planned exercise. It refers to low-energy waking behaviour performed while sitting, reclining, or lying down. An individual may meet weekly exercise recommendations but still spend most of the day sitting. Consequently, exercise participation and sedentary time should be measured separately. In medical students, classroom attendance, study sessions, screen exposure, and examination preparation may generate prolonged sitting even among those who report occasional exercise. The immune response to an acute exercise session differs from the adaptation associated with habitual activity. Strenuous or prolonged exercise can temporarily mobilise neutrophils, redistribute lymphocytes, and affect platelet function. This short-term response is part of normal physiological adaptation and should not automatically be interpreted as chronic inflammation. Blood samples taken soon after exercise may therefore show altered NLR or PLR despite an overall healthy lifestyle [15]. A well-designed investigation should assess the frequency, intensity, duration, and type of activity and should document the time elapsed between the last exercise session and blood collection. Objective measures such as accelerometers provide detailed movement information, whereas validated questionnaires offer a more economical approach in large cohorts. Combining both methods in a subsample can improve classification accuracy. The concept of a protective exercise threshold should be treated as a dose-response question rather than a single universal cut-off. Benefits may depend on baseline fitness, body composition, exercise intensity, recovery, sleep, and nutritional status. Research should therefore examine activity as a continuous exposure in addition to comparing active and sedentary categories [16].

Relationship of BMI with NLR and PLR

Body mass index is widely used because it requires only body weight and height, is inexpensive, and enables comparisons across large populations. It is calculated as weight in kilograms divided by height in metres squared. BMI is useful for broad classification, but it does not directly measure adipose tissue, distinguish fat from

skeletal muscle, or identify where fat is stored. These limitations are important when BMI is related to inflammatory biomarkers in young adults. Adipose tissue functions as an active endocrine and immune organ. Progressive adipose expansion can alter adipokine secretion, attract immune cells, influence insulin signalling, and contribute to chronic low-grade inflammatory activity. These processes provide a biological basis for examining whether increasing BMI is accompanied by changes in NLR or PLR. Nevertheless, the relationship may vary according to sex, fitness, fat distribution, and metabolic health [17]. A muscular individual may have a relatively high BMI without excessive adiposity, while a person with low muscle mass and central fat accumulation may have a BMI within the conventional normal category. This misclassification may weaken or obscure associations with inflammatory ratios. Research among physically active young adults should therefore avoid interpreting BMI as an exact measure of body fat. BMI should preferably be analysed both categorically and continuously. Categories permit comparison with established weight classifications,

while continuous analysis preserves information and can reveal gradual or nonlinear relationships. Researchers should also consider whether the association differs between men and women because body-fat percentage and fat distribution can differ at the same BMI. Potential confounders include diet quality, smoking, alcohol use, sleep duration, medication, menstrual status, socioeconomic background, and recent weight change. These factors may influence either BMI, inflammatory ratios, or both. Adjustment for physical activity is also essential because two individuals with the same BMI may have substantially different levels of fitness and metabolic health [18]. BMI alone cannot establish whether adiposity causes an increase in NLR or PLR. Cross-sectional relationships may reflect reverse causality, residual confounding, or temporary physiological variation. Longitudinal observations and interventions are needed to clarify the direction of association. For young-adult screening, BMI should be combined with waist measurements and lifestyle assessment rather than used as an isolated indicator of inflammatory risk [19].



Waist Circumference and Waist-to-Hip Ratio as Anthropometric Correlates

Waist circumference and waist-to-hip ratio are used to assess abdominal body-fat distribution. They may provide information that BMI cannot capture because central adiposity is metabolically different from fat stored in peripheral regions. Visceral adipose tissue is located around internal abdominal organs and is associated with altered lipid metabolism, insulin resistance, endothelial dysfunction, and inflammatory signalling. Waist circumference is measured at a defined anatomical site using a non-stretchable tape. However, studies differ in whether the measurement is taken at the midpoint between the lower rib and iliac crest, directly above the iliac crest, or at the narrowest part of the torso. These methodological differences can affect classification and make comparisons difficult. Investigators should state the anatomical landmark, participant posture, respiratory phase, clothing conditions, tape tension, and number of measurements [20]. Waist-to-hip ratio is calculated by dividing waist circumference by hip circumference. It reflects the relative

distribution of abdominal and gluteofemoral tissue. Because both measurements contribute to the ratio, error in either measurement affects the final value. Hip circumference should be recorded at the widest region of the buttocks with the tape maintained horizontally. Central adiposity may be more closely associated with inflammatory activity than overall body weight. Adipose tissue expansion can alter communication between adipocytes and immune cells, contributing to persistent immune activation. This mechanism supports investigation of NLR and PLR across progressively higher waist measurements. However, population-specific body proportions must be considered, particularly in South Asian adults, who may develop metabolic risk at comparatively lower BMI values [21]. Sex-specific analysis is essential because men and women differ in typical fat distribution and hormonal regulation. Waist measures may also vary according to age, ethnicity, posture, abdominal muscularity, recent food intake, and investigator technique. Standardised

training and duplicate measurements can reduce observer error. Waist circumference, waist-to-hip ratio, and BMI should be evaluated together. Concordant elevation across all measures may suggest general and central adiposity, whereas discordant findings may identify individuals whose risk would be missed by BMI alone. Examining these patterns alongside NLR and PLR may clarify whether abdominal distribution is more relevant to subclinical inflammation than body size itself [22].

Interaction Between Physical Activity and Adiposity

Physical activity and adiposity are closely related but biologically distinct exposures. Regular activity can influence inflammatory regulation even when body weight remains stable, while weight reduction can modify metabolic health independently of changes in formal exercise participation. Separating these pathways is necessary to determine whether activity has a direct anti-inflammatory influence or whether its apparent benefit is mainly mediated through lower adiposity. Exercise may affect immune balance through improved insulin sensitivity, vascular function, antioxidant capacity, autonomic regulation, and skeletal-muscle signalling. These adaptations can occur before substantial changes in body mass. Consequently, an active individual with overweight may have a different inflammatory and metabolic profile from a sedentary individual with a similar BMI. Conversely, excessive or poorly recovered training may temporarily increase physiological stress and alter circulating cell ratios [23]. Adiposity may mediate part of the relationship between activity and NLR or PLR. Active behaviour can limit visceral-fat accumulation, while reduced visceral adiposity may decrease inflammatory signalling. Statistical adjustment for BMI or waist circumference can help assess whether the activity-biomarker association remains after accounting for body composition. However, simple adjustment does not fully establish mediation and may remove part of the biological pathway through which activity operates. Stratified analysis can provide additional insight. Participants may be grouped as active with lower adiposity, active with higher adiposity, sedentary with lower adiposity, and sedentary with higher adiposity. This approach can reveal whether physical activity is associated with more favourable inflammatory ratios within similar anthropometric categories. Interaction terms can also test whether the association between adiposity and inflammatory markers differs according to activity level. Fitness should not be treated as identical to self-reported activity. Cardiorespiratory fitness reflects physiological capacity and is influenced by activity, genetics, health status, and training history. Including a practical fitness measure could strengthen interpretation, although this may not always be feasible in field-based student research [24]. Longitudinal studies are needed to distinguish direct, indirect, and bidirectional pathways. Repeated measures of activity, body composition, and blood indices would clarify whether changes in exercise behaviour precede changes in NLR or PLR and whether these changes occur independently of weight loss [25].

Sensitivity of NLR and PLR Compared with hs-CRP and IL-6

High-sensitivity C-reactive protein and interleukin-6 are frequently used to investigate systemic inflammation. hs-CRP is produced mainly by the liver in response to inflammatory signalling and is widely used in cardiovascular and metabolic research. IL-6 is a cytokine involved in immune regulation, hepatic acute-phase responses, metabolism, and communication between skeletal muscle and other tissues. NLR and PLR measure different biological dimensions and should not be considered direct substitutes for these markers. The principal advantage of NLR and PLR is accessibility. Both can be calculated from a routine complete blood count without specialised assays. This makes them attractive in resource-limited settings and in exploratory screening of large groups. They may also be available retrospectively when stored laboratory records include differential leukocyte and platelet counts [26]. Their main limitation is nonspecificity. An altered NLR or PLR can result from infection, acute stress, medication, smoking, sleep disruption, menstrual factors, nutritional deficiency, strenuous exercise, or haematological variation. hs-CRP is also nonspecific but represents an acute-phase protein rather than a relative distribution of circulating cells. IL-6 provides closer insight into cytokine signalling but can vary rapidly and requires more specialised processing and assay procedures. Biomarker sensitivity should be evaluated according to the purpose of measurement. A marker suitable for identifying acute clinical deterioration may not be ideal for characterising subtle metabolic inflammation in healthy young adults. Agreement between NLR, PLR, hs-CRP, and IL-6 should not be assumed because each represents a different component of the inflammatory response [27]. A multimarker approach may provide the strongest interpretation. NLR and PLR can describe haematological immune balance, while hs-CRP can indicate hepatic acute-phase activity. Where resources permit, IL-6 or additional metabolic measures can provide mechanistic context. Repeated testing can help distinguish persistent low-grade inflammation from a temporary response. NLR and PLR should therefore be regarded as complementary and hypothesis-generating measures. Before they are recommended as screening tools, studies must establish reproducibility, population-specific reference distributions, associations with recognised risk factors, and incremental value beyond basic anthropometry and clinical history [28].

Inflammatory Break Points and Anthropometric Thresholds

An inflammatory break point may be described as an anthropometric level beyond which an inflammatory marker begins to increase more rapidly. Identifying such a point could be useful for early prevention because it may indicate that biological risk begins before the conventional threshold for obesity is reached. However, break-point analysis is methodologically complex and should not be based on arbitrary visual inspection. The association between adiposity and inflammation may be linear,

nonlinear, threshold-based, or modified by sex and activity level. Categorising BMI or waist circumference into broad groups can conceal important variation near category boundaries. Continuous modelling using restricted cubic splines, fractional polynomials, or segmented regression may identify whether the slope changes across the anthropometric distribution [29]. A statistically detected break point should not automatically be interpreted as a clinical threshold. Its reliability depends on sample size, measurement precision, population characteristics, and the number of observations around the estimated change point. Internal validation, confidence intervals, and replication in an independent cohort are necessary. A threshold identified in one ethnic or age group may not apply to another. South Asian populations require particular attention because BMI may not fully represent body-fat percentage or central adiposity. Waist-based thresholds may therefore offer additional information. Nevertheless, waist circumference must be measured using a standard protocol, and sex-specific models should be considered. The possible influence of height may also be evaluated through measures such as waist-to-height ratio, although this measure falls outside the primary anthropometric indices of the present review [30]. Physical activity may shift or soften an apparent inflammatory break point. For example, active participants may tolerate a higher anthropometric level before marked inflammatory alteration becomes apparent, or activity may reduce the slope across the entire range without producing a distinct threshold. These possibilities can be examined through interaction models. Because cross-sectional data cannot prove temporal progression, an observed break point should be described as an analytical feature rather than evidence that inflammation begins at a precise body measurement. Longitudinal research with repeated anthropometric and haematological measurements is required before a clinically actionable threshold can be proposed [31].

Confounding Effects of Age, Sex, Stress, Sleep, and Other Lifestyle Factors

NLR and PLR are influenced by numerous factors that may also be associated with physical activity or body composition. Failure to measure these variables can produce confounding, in which an observed association is partly or entirely explained by another exposure. Even within a relatively narrow young-adult age range, demographic and behavioural differences require careful assessment. Sex may influence leukocyte and platelet distributions, body-fat percentage, abdominal fat patterning, hormonal status, and activity behaviour. Menstrual-cycle phase, menstrual blood loss, hormonal contraception, and iron status may affect platelet and lymphocyte measures. Sex-stratified descriptive and multivariable analyses are therefore preferable to simple statistical adjustment alone [32]. Psychological stress is especially relevant among medical students. Examination pressure, clinical responsibilities, competitive academic environments, and concerns about future performance may activate neuroendocrine pathways that influence immune-cell trafficking. Acute stress near the day of blood collection

may produce a temporary response, while chronic perceived stress may contribute to longer-term behavioural and physiological changes. Sleep duration and quality should also be considered. Medical students may experience late-night study, irregular schedules, screen exposure, and early academic commitments. Sleep disruption can affect appetite regulation, glucose metabolism, activity participation, stress responses, and immune function. A questionnaire covering recent and habitual sleep could help interpret unexpected biomarker values. Smoking, alcohol intake, diet, caffeine use, hydration, and medication are additional influences. Recent infection, dental inflammation, allergy, injury, vaccination, and strenuous exercise should be specifically screened because participants may otherwise consider themselves healthy. Female participants should be assessed for pregnancy where appropriate, and individuals with known haematological or inflammatory disorders should be excluded. Confounder selection should be based on biological reasoning rather than automated statistical procedures alone. Overadjustment can occur when variables lying on the causal pathway, such as waist circumference in the relationship between activity and inflammation, are treated only as confounders. A predefined conceptual model can help distinguish confounders, mediators, and effect modifiers [33].

CONCLUSION

Regular physical activity may help maintain a more balanced inflammatory profile in young adults by influencing NLR, PLR, and body composition. Anthropometric measures, particularly waist circumference and waist-to-hip ratio, may provide additional insight beyond BMI when assessing low-grade systemic inflammation. However, NLR and PLR should be interpreted alongside lifestyle, demographic, and clinical factors because they are nonspecific markers. Standardized studies in Indian medical students are needed to establish population-specific reference values and clarify the independent effects of physical activity and adiposity.

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