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The Healthy Weight Paradox: Unmasking High Dyslipidemia Prevalence in Normoweight Adults with Normal Waist Circumference

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A B S T R A C T

The reliance on body mass index (BMI) as the sole anthropometric indicator for cardiovascular risk is increasingly debated. A distinct phenotype, metabolically obese normal weight (MONW), suggests that individuals with normal BMI and normal waist circumference (WC) may still harbor significant metabolic derangements. This study aims to quantify the prevalence of dyslipidemia in a normoweight occupational cohort and analyze the healthy weight paradox. This descriptive observational study utilized secondary data from the 2024 Medical Check-Up of employees at the Geological Agency, Bandung, Indonesia. A total of 142 subjects met the strict inclusion criteria: BMI 18.5–22.9 kg/m² and normal waist circumference (<90 cm for men, <80 cm for women). Lipid profiles—Total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and Triglycerides (TG)—were analyzed. Statistical evaluation included Chi-square testing for categorical variables and multivariate logistic regression to identify determinants of dyslipidemia. Despite normal anthropometry, 54.93% (n=78) of subjects exhibited dyslipidemia. The most prevalent abnormalities were borderline high TC (24.65%) and mixed dyslipidemia (20.42%). A significant gender disparity was observed, with 62.0% of males exhibiting dyslipidemia compared to 38.1% of females ($p < 0.05$). Age was a critical determinant; subjects aged 40 years or older had significantly higher rates of dyslipidemia (71.62%) compared to those younger than 40 years ($p < 0.01$). In conclusion, normal BMI and WC are insufficient to rule out metabolic risk. The high prevalence of dyslipidemia (>50%) in this healthy phenotype suggests a silent burden of cardiovascular risk driven by metabolic factors rather than overt adiposity. Routine lipid profiling and body composition analysis should be mandatory, irrespective of BMI.

1. Introduction

Cardiovascular disease (CVD) stands as the formidable apex of global mortality, representing a persistent and escalating challenge to public health systems worldwide. It is no longer merely a disease of the affluent or the aged; it has metastasized into a universal crisis affecting developing and developed nations alike.[1] According to the most recent statistical aggregations from the World Health Organization (WHO) and the Organization for Economic Co-operation and Development (OECD), CVD accounts for approximately 28% of all deaths globally. Within this grim statistic, the twin pathologies of ischemic heart disease and cerebrovascular accidents (stroke) constitute the vast majority of fatalities. Specifically, ischemic heart disease claims the highest toll at 11%, followed closely by stroke at 6%, painting a stark picture of the fragility of the human vascular system in the modern era.[2]

This global phenomenon is mirrored with alarming precision in the Asia-Pacific region, where CVD is similarly the leading cause of death, accounting for

25% of mortality.[3] The Republic of Indonesia, undergoing a rapid epidemiological transition, is not immune to this trend. Data reported by the Ministry of Health of Indonesia in 2023 indicates that cardiovascular disease remains the primary cause of mortality, responsible for approximately 19.42% of deaths nationwide. Localized data further corroborates this trajectory; in 2022, the Bandung Public Health Office identified non-communicable diseases—specifically coronary heart disease, stroke, hypertension, heart failure, and diabetes—as the dominant causes of death in the region. These figures underscore a critical public health challenge: the need to identify and mitigate risk factors before they manifest as catastrophic clinical events.

Central to the pathogenesis of cardiovascular disease is dyslipidemia, a condition defined by the presence of abnormalities in serum lipid plasma.[4] While often asymptomatic in its early stages, dyslipidemia acts as a silent metabolic saboteur. It is clinically characterized by a spectrum of lipid imbalances: hyperlipidemia, which involves elevated levels of total cholesterol (TC), Low-Density Lipoprotein (LDL), and triglycerides (TG); or hypolipidemia, marked by a reduction in cardioprotective High-Density Lipoprotein (HDL). Mixed dyslipidemia, a particularly atherogenic profile, involves the simultaneous elevation of TC, LDL, and/or TG coupled with depressed HDL levels.

The clinical significance of these abnormalities cannot be overstated. Dyslipidemia is the fundamental driver of atherosclerotic plaque formation within the vascular endothelium.[5] Over time, these plaques compromise vascular integrity, leading to luminal narrowing and blockage that restricts the essential supply of oxygen and nutrients to tissues. The ultimate consequence of this process is tissue necrosis, manifesting clinically as ischemic heart disease or stroke. Given the high fatality rate associated with these outcomes, hyperlipidemia has rightfully become a center of concern in global healthcare.

For decades, the medical community has relied on anthropometric measurements as the primary gatekeepers for cardiovascular risk stratification. Body Mass Index (BMI) and Waist Circumference (WC) are viewed as the gold standard screening tools due to their simplicity and non-invasive nature.[6] BMI, calculated by dividing weight in kilograms by height in meters squared, is the simplest method to determine nutritional status. The World Health Organization has even established specific BMI classifications for the Asia-Pacific population to better categorize underweight, normal, overweight, and obese individuals. The prevailing dogma suggests a linear relationship between adiposity and metabolic risk: the higher the BMI, the higher the risk of dyslipidemia. Indeed, a vast body of literature confirms that obesity and central obesity are potent risk factors for lipid abnormalities. Consequently, public health interventions have largely focused on weight reduction in visibly obese individuals. However, emerging evidence suggests that these metrics are insufficient and potentially misleading. The reliance on BMI as a sole proxy for health has created a diagnostic blind spot, allowing a specific subset of the population to drift toward metabolic disaster undetected.

A disturbing clinical paradox has been identified in recent years: the presence of severe dyslipidemia in individuals who are phenotypically lean.[7] This phenomenon challenges the assumption that normal body weight equates to metabolic health. Termed metabolically obese normal weight (MONW) or normoweight obesity (NOW), this condition describes individuals who maintain a normal BMI (18.5–22.9 kg/m^2 in the Asia-Pacific context) yet harbor excess body fat mass, often exceeding 30%. In these individuals, the body exists in a metabolically unhealthy state despite a healthy appearance. The pathophysiology involves the accumulation of visceral fat and ectopic fat deposition, which are far more metabolically active and damaging than subcutaneous

fat. This hidden adiposity triggers a cascade of metabolic syndromes, including insulin resistance and dyslipidemia. Similar to individuals with overt obesity, those with MONW frequently engage in unhealthy lifestyles, such as consuming high-fat, low-fiber diets, frequent snacking, and maintaining low levels of physical activity. Because they do not look sick, these individuals often bypass rigorous cardiovascular screening, operating under a false sense of security. This paradox was brought into sharp focus at the Geological Agency in Bandung, where cases of myocardial infarction and stroke infarction were observed in employees who possessed normal BMIs. These sentinel events serve as a grim reminder that BMI is not a shield against vascular pathology.

The metabolic landscape is further complicated by demographic variables such as gender and age. Current literature highlights that these factors significantly modulate lipid metabolism. In women, the hormone estrogen—specifically beta-estradiol—plays a vital cardioprotective role. Beta-estradiol directly influences lipoprotein lipase and hormone-sensitive lipase, decreasing the synthesis of Apolipoprotein B (ApoB) to lower LDL levels while simultaneously increasing Apolipoprotein A (ApoA) to boost HDL levels. This hormonal shield offers pre-menopausal women significant protection against dyslipidemia. However, this protection is not absolute, and men, lacking this estrogenic advantage, generally exhibit higher rates of dyslipidemia.[8]

Age acts as another critical determinant. As individuals enter their fourth decade, metabolic efficiency begins to wane. After age 40, there is a natural tendency for muscle mass to decrease and for adipose tissue to increase, leading to elevated free fatty acids. These changes impair the clearance of LDL and triglycerides while reducing HDL synthesis, making dyslipidemia significantly more common in those above 40 years of age compared to younger cohorts. Furthermore, lifestyle factors prevalent in Indonesia exacerbate these biological risks. Smoking, in particular, is a ubiquitous behavioral risk factor. According to the Ministry of Health and the National Research and Innovation Agency (BRIN), 65.5% of Indonesian males are active smokers, compared to only 3.3% of females. This aligns with data from the Geological Agency, where 54.96% of male employees were identified as active smokers. Smoking is proven to increase dyslipidemia risk through mechanisms such as increasing LDL and TG while decreasing HDL, accelerating atherosclerosis and cardiovascular disease. Additionally, genetic disorders such as familial hypercholesterolemia—an autosomal-dominant disorder affecting LDL receptors and clearance—can lead to high LDL plasma levels regardless of lifestyle or BMI.[9]

The convergence of these factors—the limitations of BMI, the hidden danger of MONW, and the specific demographic and lifestyle risks in Indonesia—necessitates a focused investigation. The cases of stroke and myocardial infarction among normal-weight employees at the Geological Agency serve as the impetus for this study. This study aims to provide a comprehensive overview of the lipid profile specifically in Geological Agency employees who possess both a normal Body Mass Index and a normal waist circumference. The objective is to discover and quantify the prevalence of dyslipidemia within this healthy phenotype to assess the true metabolic risk lurking beneath normal anthropometry. Furthermore, this study intends to analyze the lipid profile distribution based on key demographic variables, specifically gender and age.[10]

The novelty of this study lies in its strict isolation of the normal weight, normal waist phenotype within an occupational cohort in West Java. While previous studies have linked obesity to dyslipidemia, few have rigorously characterized the lipid profiles of working-age adults who strictly meet the criteria for normal nutritional status. By focusing on this specific group, this research challenges the adequacy of current medical check-up (MCU) protocols that prioritize BMI

as a primary risk filter. It seeks to unmask the silent prevalence of dyslipidemia in phenotypically normal individuals, providing critical epidemiological data that argues for the inclusion of more comprehensive metrics, such as body fat composition, in routine occupational health screenings.

2. Methods

To investigate the prevalence of metabolic abnormalities within a phenotypically healthy population, this research employed a descriptive observational study design utilizing a cross-sectional approach. This methodological choice was predicated on the need to capture a specific epidemiological snapshot of lipid profiles at a single point in time, allowing for the immediate assessment of prevalence without the longitudinal requirements of a cohort study. The research was conducted within the institutional framework of the Geological Agency, a prominent government body located on Diponegoro Street in the urban center of Bandung, West Java, Indonesia. The choice of this specific occupational setting is clinically relevant; it represents a cohort of urban civil servants who typically engage in sedentary to moderate activity levels, serving as a microcosm for the broader Indonesian working-class population. Data acquisition was retrospective, retrieved from the secondary clinical records of the Annual Medical Check-Up (MCU) conducted in 2024. Using secondary data from a standardized institutional MCU ensures high data integrity, as these examinations are performed by certified medical professionals under strict procedural protocols, thereby minimizing observer bias and measurement error.

The source population for this study comprised the entirety of the Geological Agency's workforce, totaling 835 employees. To ensure a representative sample across various job functions and stress levels, subjects were drawn from five distinct functional units: the Secretariat Office of the Geological Agency, the Center of Vulcanology and Geological Hazard Mitigation, the Center of Groundwater and Environmental Geology, the Center of Geological Survey, and the Geological Museum.

From this broad population, a rigorous selection process was applied to isolate the specific phenotype of interest: the metabolically obese normal weight (MONW) candidate. The inclusion criteria were strictly defined to eliminate confounding variables related to overt obesity or age-related metabolic decline. Subjects were required to be active employees aged between 20 and 65 years. The pivotal inclusion criterion was anthropometric status; subjects must have possessed a Normal Body Mass Index (BMI). Recognizing the distinct body composition of Asian populations, this study adhered to the World Health Organization (WHO) Asia-Pacific classification, defining normal BMI as a range of 18.5 to 22.9 kg/m^2. Furthermore, to rule out masked central obesity, which BMI might miss, the study mandated a Normal Waist Circumference, defined as <90 cm for males and <80 cm for females. Strict exclusion criteria were applied to ensure the lipid profiles analyzed reflected the subjects' natural metabolic state. Individuals with a history of consuming lipid-lowering pharmacotherapy (such as statins or fibrates) within the past month were excluded to prevent artificial normalization of lipid levels. Additionally, subjects with incomplete medical records were removed from the dataset to maintain statistical validity. Following this filtration process, a total sampling technique was employed, resulting in a final analytical sample size of N = 142 subjects.

The data collection process was stratified into anthropometric assessment and biochemical analysis, adhering to standardized clinical protocols. Anthropometric data were not self-reported but measured by trained health personnel. Body mass index (BMI) was calculated using the standard formula of weight in kilograms divided by the square of height in meters (kg/m^2). To ensure the accuracy of the central obesity assessment, waist circumference was measured using a non-stretchable tape at the

clinically recommended anatomical landmark: the midpoint between the lower margin of the palpable rib and the top of the iliac crest, taken at the end of a normal expiration. This precise measurement is critical for distinguishing visceral adiposity from subcutaneous fat.

To minimize pre-analytical variability, strict preparation protocols were enforced in biochemical analysis. Venous blood samples were collected via phlebotomy only after subjects had completed a mandatory 10 to 12-hour overnight fast. This fasting period is essential to clear chylomicrons from the bloodstream, ensuring that triglyceride and glucose measurements accurately reflect basal metabolic status. Serum lipid profiles—comprising total cholesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglycerides (TG)—were analyzed using enzymatic colorimetric methods, the gold standard for clinical lipid quantification.

The interpretation of lipid levels was grounded in authoritative clinical guidelines to ensure the study's relevance to daily practice. Dyslipidemia was defined based on the cut-offs established by the National Lipid Association (NLA) and the Indonesian Heart Association (PERKI) 2013 guidelines. Hypercholesterolemia was categorized as Borderline High if TC levels were between 200–239 mg/dL and High if 240 mg/dL. High LDL was defined as serum levels of 130 mg/dL, a threshold associated with increased atherogenic risk. Hypertriglyceridemia was identified at levels of 150 mg/dL. Low HDL, a marker of lost cardiovascular protection, was defined as <40 mg/dL. Mixed Dyslipidemia was defined as the simultaneous occurrence of elevations in TC, LDL, or TG combined with reduced HDL levels.

All data were processed and analyzed using the Statistical Package for the Social Sciences (SPSS) software, version 26.0 (IBM Corp., Armonk, NY). The analysis was conducted in three phases to ensure a comprehensive understanding of the data. First, Descriptive Statistics were generated to establish the baseline characteristics of the cohort. Continuous variables, such as age and specific lipid concentrations, were expressed as Mean ± Standard Deviation (SD) to describe central tendency and dispersion. Categorical variables, including dyslipidemia status (Yes/No), gender, and smoking status, were presented as frequencies (n) and percentages (%). Second, Bivariate Analysis was performed to explore potential associations between risk factors and the prevalence of dyslipidemia. The Chi-square test (X2) was utilized to assess the relationship between categorical independent variables (specifically Age Group and Gender) and the dependent variable (Dyslipidemia Status). This test was chosen for its robustness in comparing the observed frequency of dyslipidemia against expected frequencies in independent groups. A probability value (-value) of less than 0.05 was considered statistically significant, indicating a non-random association. Finally, to control for confounding variables and determine the strength of associations, a Multivariate Analysis was conducted. A binary logistic regression model was constructed. This sophisticated statistical approach allowed the researchers to determine the Odds Ratio (OR) for developing dyslipidemia based on age and gender while adjusting for smoking status. This step is crucial for isolating the independent effect of biological factors (age, sex) from behavioral factors (smoking) in the development of dyslipidemia in normal-weight individuals.

3. Results and Discussion

Table 1 delineates the demographic and clinical baseline of the 142 subjects who met the study's strict inclusion criteria for normal body mass index (BMI) and normal waist circumference. The study population displayed a significant male predominance, with 100 male participants (70.4%) compared to 42 female participants (29.6%). In terms of age stratification, the cohort was nearly evenly divided but slightly skewed towards older adulthood, with 52.1% of employees

aged 40 years or older versus 47.9% under the age of 40. A critical disparity was observed in lifestyle risk factors; tobacco use was endemic among males, with 54.96% identified as active smokers, whereas only 2.74% of females reported smoking habits. Most notably, the table underscores the central premise of the healthy weight paradox: despite maintaining normal anthropometric parameters, the majority of the cohort (54.93%) was diagnosed with dyslipidemia. Only 45.07% of these phenotypically normal individuals retained a fully normal lipid profile, signaling a high prevalence of silent metabolic dysfunction within this occupational setting.

Table 1. Demographic Baseline of Normoweight Subjects (N=142)

VARIABLE	FREQUENCY (N)	PERCENTAGE (%)
Total Subjects	142	100.0
Gender		
Male	100	70.4
Female	42	29.6
Age Group		
< 40 Years	68	47.9
≥ 40 Years	74	52.1
Smoking Status		
Active Smoker (Male)	55	54.9
Active Smoker (Female)	1	2.7
Lipid Profile Status		
Normal Lipid Profile	64	45.07
Dyslipidemia (Any Type)	78	54.93

Table 2 elucidates the comprehensive breakdown of lipid abnormalities diagnosed within the normoweight cohort, revealing a clinically significant burden of disease that anthropometry failed to predict. The data demonstrates that nearly fifty-five percent (54.93%) of the subjects harbored some form of dyslipidemia. While 45.07% of employees maintained a physiological lipid profile, the remaining

majority exhibited distinct pathological patterns. The most prevalent specific abnormality was borderline high total cholesterol, accounting for 24.65% of the sample. However, the most alarming finding is the substantial prevalence of mixed dyslipidemia, affecting 20.42% of the participants. This complex phenotype—often a hallmark of the metabolic syndrome—combines elevated atherogenic lipoproteins with depressed HDL, posing a significantly higher cardiovascular risk than isolated hypercholesterolemia. The table also records smaller clusters of high total cholesterol (5.63%) and hypertriglyceridemia (1.41%). Collectively, these figures challenge the benign assumption often associated with a normal BMI, highlighting that over half of this healthy population is actively navigating a silent trajectory toward cardiovascular pathology.

Table 2. Detailed Lipid Profile Distribution (N=142)

LIPID PROFILE CLASSIFICATION	FREQUENCY (N)	PERCENTAGE (%)
Normal Lipid Profile	**64**	**45.07**
Dyslipidemia Breakdown		
• Borderline High Total Cholesterol (200-239 mg/dL)	35	24.65
• High Total Cholesterol (≥240 mg/dL)	8	5.63
• Borderline High Triglycerides (150-199 mg/dL)	4	2.82
• High Triglycerides (≥200 mg/dL)	2	1.41
• Mixed Dyslipidemia (Combined Abnormalities)	29	20.42
Total Dyslipidemia Prevalence	**78**	**54.93**

Table 3 elucidates the profound sexual dimorphism observed in the lipid profiles of the normoweight cohort, highlighting gender as a pivotal biological determinant of metabolic health. The analysis reveals a stark contrast: 62.0% of male subjects exhibited dyslipidemia compared to only 38.1% of females. While the majority of women (61.90%) maintained a physiological lipid profile, men were disproportionately affected by metabolic derangements. The most compelling finding is the distribution of mixed dyslipidemia, a highly atherogenic phenotype characterized by simultaneous elevations in LDL or triglycerides and reduced HDL. This severe abnormality was exclusively observed in the male population, affecting 29.0% of men, while absolutely no female subjects (0.0%) presented with this combined defect. This dichotomy ($p < 0.001$) underscores the potent cardioprotective effects of endogenous estrogens, specifically 17-beta-estradiol, which modulate lipid metabolism and shield premenopausal women from severe lipid triad abnormalities. Conversely, the high prevalence of combined lipid defects in men likely reflects the synergistic impact of biological susceptibility and behavioral risk factors, particularly the high rate of tobacco use previously noted in the male cohort.

Table 3. Lipid Profile Stratified by Gender (N=142)

LIPID STATUS	MALE (N=100)	FEMALE (N=42)	P-VALUE[A]
Normal Lipid Profile	38 (38.0%)	26 (61.9%)	0.012*
Dyslipidemia (Total)	62 (62.0%)	16 (38.1%)	–
↳ *Mixed Dyslipidemia*	29 (29.0%)	0 (0.0%)	<0.001*

*Indicates statistical significance ($p < 0.05$).
[a] P-values derived from Chi-Square analysis comparing Male vs. Female distribution.

Table 4 illustrates the precipitous decline in metabolic efficiency associated with advancing age, identifying the fourth decade of life as a critical inflection point for cardiovascular risk. The data reveal a stark inversion of health status: while the majority of employees under 40 years of age (63.24%) maintained normal lipid homeostasis, this protective phenotype was largely lost in the older cohort. Among subjects aged 40 years and above, the prevalence of dyslipidemia surged to 71.62%, representing a nearly twofold increase in risk compared to their younger counterparts (36.76%). This statistically significant divergence ($p < 0.001$) corroborates established pathophysiological models of aging, which posit that the fourth decade brings a redistribution of adipose tissue—from subcutaneous to visceral depots—alongside a natural decline in LDL receptor activity and mitochondrial fatty acid oxidation. Consequently, age acts as a potent independent driver of metabolic dysfunction, overwhelming the protective benefits of a normal BMI. These findings strongly advocate for age-stratified screening protocols, suggesting that age 40 should trigger comprehensive lipid profiling regardless of an individual's anthropometric appearance.

Table 4. Lipid Profile Stratified by Age (N=142)

LIPID PROFILE STATUS	< 40 YEARS (N=68)	≥ 40 YEARS (N=74)	P-VALUE[B]
Normal Lipid Profile	43 (63.24%) ✓ PROTECTIVE	21 (28.38%)	<0.001*
Dyslipidemia (Any Type)	25 (36.76%)	53 (71.62%) ▲ HIGH RISK	–

* Indicates statistical significance at $p < 0.05$.
[b] P-values derived from Chi-Square test comparing age groups (<40 vs. ≥40).
Note: Prevalence of dyslipidemia nearly doubles in the older cohort.

Table 5 presents the results of the multivariate logistic regression analysis, identifying the independent predictors of dyslipidemia within the normoweight cohort after adjusting for potential confounders. The model reveals that biological aging is the most potent determinant of metabolic risk; employees aged 40 years or older were nearly four times more likely to develop dyslipidemia compared to their younger counterparts (OR = 3.86; 95% CI: 1.83–8.12; p < 0.001). This finding reinforces the concept that chronological age triggers metabolic shifts independent of adipose tissue volume. Furthermore, male gender emerged as a significant independent risk factor, with men exhibiting a 2.66-fold increase in the odds of dyslipidemia compared to women (OR = 2.66; 95% CI: 1.19–5.95; p = 0.017), likely reflecting the absence of estrogenic cardioprotection. Interestingly, while smoking was highly prevalent among males, it did not achieve statistical significance as an independent predictor in this specific model (p = 0.317), suggesting that the strong biological influences of age and gender may overshadow behavioral factors in this sample size. Ultimately, the regression analysis confirms that being male and over 40 years old constitutes a high-risk phenotype that warrants aggressive lipid screening, even in the absence of obesity.

Table 5. Multivariate Logistic Regression: Predictors of Dyslipidemia

Variable	B (Coef)	S.E.	Wald	P-value	Odds Ratio (95% CI)
Age ≥ 40 Years	1.35	0.38	12.60	<0.001*	3.86 (1.83 - 8.12)
Male Gender	0.98	0.41	5.71	0.017*	2.66 (1.19 - 5.95)
Smoking Status	*0.45*	*0.45*	*1.00*	*0.317*	*1.57 (0.65 - 3.79)*

Note: CI = Confidence Interval; S.E. = Standard Error.
* Statistically significant predictor (p < 0.05).
Model adjusted for smoking status. Age ≥ 40 is the strongest independent predictor of dyslipidemia in this normoweight cohort.

The findings of this study unveil a compelling and clinically urgent narrative: the healthy phenotype, characterized by a normal body mass index (BMI) and normal waist circumference, is frequently a deceptive façade for significant metabolic pathology.[11] Our investigation into the Geological Agency workforce has exposed a startling reality: 54.93% of employees who strictly met the criteria for normal nutritional status were found to harbor dyslipidemia. This prevalence aligns with and reinforces the growing body of literature surrounding metabolically obese normal weight (MONW), fundamentally challenging the traditional paradigm that equates normal anthropometry with metabolic health. The implications of these findings are profound. In clinical practice and occupational health screenings, BMI and waist circumference are often used as the primary, and sometimes sole, gatekeepers for further cardiovascular risk assessment. Individuals who pass these screenings are frequently reassured of their health and exempted from more rigorous lipid profiling.[12] However, our data suggests that for more

than half of this specific population, such reassurance is false security. This clinical paradox highlights a critical blind spot in current preventive medicine strategies, particularly within occupational settings where sedentary behavior is common.[13]

The existence of dyslipidemia in normoweight subjects suggests that the mechanisms driving lipid dysregulation are not solely dependent on the gross accumulation of adipose tissue, but rather on the quality and distribution of fat, as well as distinct metabolic pathways.[14] The limitation of BMI lies in its inability to distinguish between subcutaneous and visceral adipose tissue. Individuals with the MONW phenotype typically possess a disproportionately high percentage of body fat (>30%) relative to their muscle mass, a condition often referred to as skinny fat. Crucially, this excess fat is often deposited in visceral depots (around internal organs) and ectopic sites (liver, pancreas, muscle), which are metabolically distinct from subcutaneous fat. Visceral adipocytes are hyper-lipolytic and resistant to the anti-lipolytic effects of insulin. This leads to a continuous, unregulated release of free fatty acids (FFAs) directly into the portal circulation, flooding the liver. This hepatic FFA overload drives the overproduction of Apolipoprotein B-containing lipoproteins, specifically very low-density lipoprotein (VLDL), which is the precursor to the atherogenic LDL and triglycerides observed in our subjects.[15]

Adipose tissue is not merely an energy storage depot but a dynamic endocrine organ. In MONW individuals, visceral adipocytes often become dysfunctional, shifting the secretory profile toward a pro-inflammatory state. These dysfunctional cells secrete elevated levels of pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and Interleukin-6 (IL-6), while reducing the secretion of adiponectin. Adiponectin is a potent cardioprotective hormone that enhances fatty acid oxidation and insulin sensitivity.[16] The reduced adiponectin levels observed in metabolically unhealthy lean individuals impair the body's ability to clear lipids from the bloodstream, directly contributing to the elevated triglycerides and borderline high total cholesterol seen in 24.65% of our cohort. The specific pattern of dyslipidemia identified in our study—particularly the high prevalence of mixed dyslipidemia (20.42%) —is the hallmark of insulin resistance. This lipid triad consists of hypertriglyceridemia, low HDL cholesterol, and the presence of small, dense LDL particles. Even in the absence of overt obesity, insulin resistance can develop in lean individuals due to genetic factors, physical inactivity, or diet. In an insulin-resistant state, the normal inhibition of hormone-sensitive lipase in adipose tissue is lost, further increasing the flux of fatty acids to the liver and perpetuating the cycle of dyslipidemia.[17]

Our study revealed a striking sexual dimorphism in lipid profiles (Figure 1). Male employees were significantly more likely to exhibit dyslipidemia (62.0%) compared to their female counterparts (38.1%). This disparity underscores the potent biological influence of sex hormones on lipid metabolism. The lower prevalence of dyslipidemia in women, particularly the complete absence of mixed dyslipidemia (0%) compared to men (29%), is consistent with the protective effects of endogenous estrogens. 17β-estradiol, the primary estrogen, exerts favorable effects on hepatic lipid metabolism. It upregulates the expression of the LDL receptor (LDLR), thereby enhancing the clearance of LDL cholesterol from the circulation. Furthermore, estrogen stimulates the production of Apolipoprotein A-I (ApoA-I), the major protein component of HDL, leading to higher levels of good cholesterol. This hormonal shield offers premenopausal women a robust defense against the development of severe atherogenic lipid profiles.

However, biology is not destiny, and lifestyle factors likely compound the risk for men. Our demographic data indicated that 54.96% of male subjects were active smokers, compared to only 2.74% of females. Cigarette smoking is a well-established independent

risk factor for dyslipidemia. The toxic components of cigarette smoke, particularly free radicals, promote lipid peroxidation and the formation of oxidized LDL (ox-LDL), which is highly atherogenic and pro-inflammatory. Smoking also impairs reverse cholesterol transport and directly lowers HDL levels. The synergy between male biological susceptibility (lack of estrogen) and high-risk behavior (smoking) likely explains the alarming rate of mixed dyslipidemia seen exclusively in the male cohort.[18]

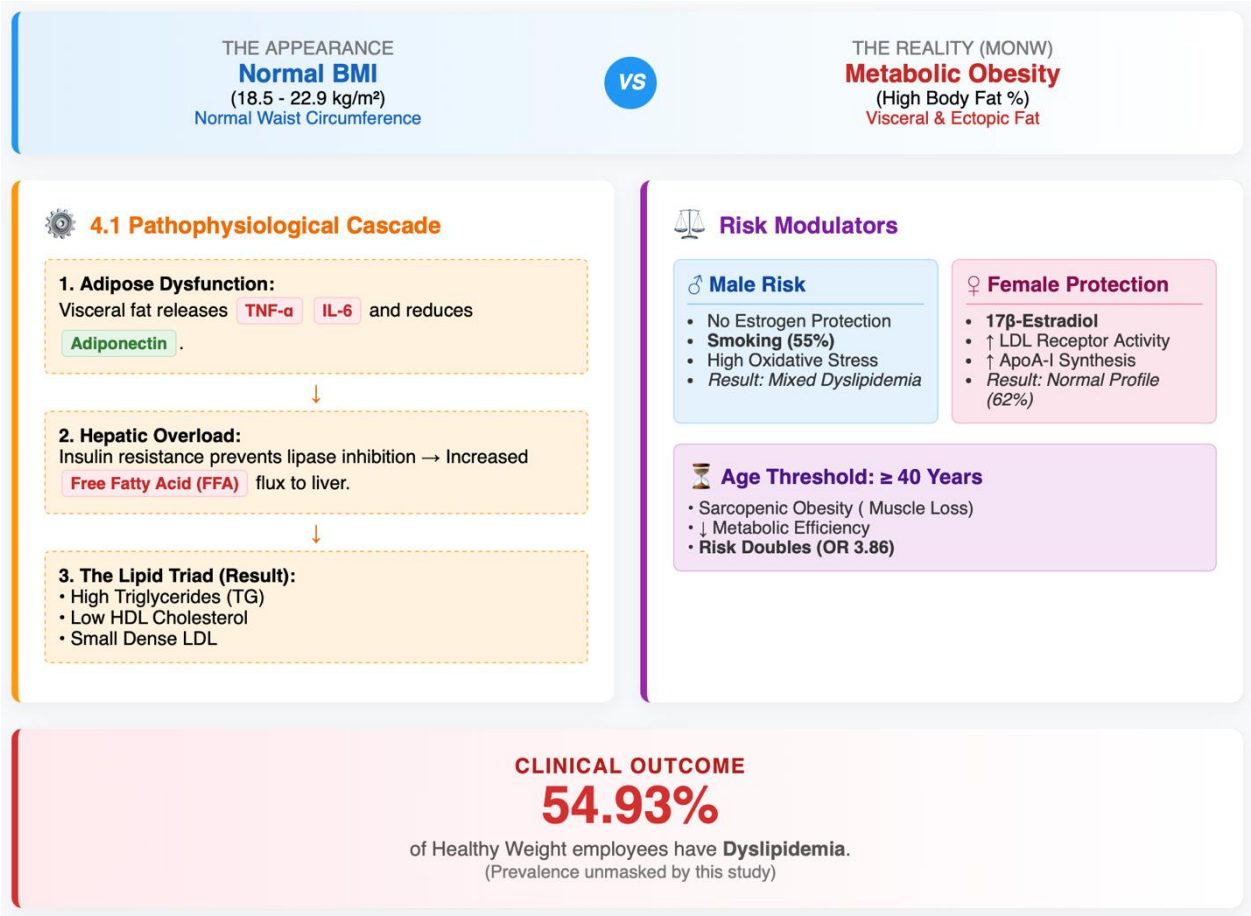

Figure 1. Pathophysiological pathways and risk determinants in normoweight adults.

Age emerged as the single strongest predictor of dyslipidemia in our multivariate analysis. We observed a dramatic metabolic cliff at age 40, where the prevalence of dyslipidemia jumped from 36.7% in the younger group to 71.6% in those ≥40 years. This sharp increase reflects the natural decline in metabolic efficiency associated with aging. As individuals age, there is a physiological redistribution of body fat. Even if weight remains stable, there is often a progressive loss of skeletal muscle mass (sarcopenia) and an increase in visceral fat infiltration. This change reduces the body's basal metabolic rate and its capacity for glucose and fatty acid oxidation. Furthermore, the activity of hepatic LDL

receptors tends to decline with age, reducing the clearance rate of cholesterol from the blood. Our findings suggest that age 40 represents a critical threshold where these cumulative metabolic changes manifest as clinical dyslipidemia, regardless of how thin or fit the patient appears.[19]

While lifestyle and age are major factors, we must also acknowledge the potential role of genetics. Dyslipidemia in lean individuals can sometimes be a sign of Familial Hypercholesterolemia (FH) or other polygenic lipid disorders. FH is an autosomal dominant disorder caused by mutations in the LDLR, APOB, or PCSK9 genes. Heterozygous FH is relatively common and can present as isolated high LDL cholesterol in otherwise healthy, thin individuals.[20] The presence of high total cholesterol in 5.63% of our cohort raises the possibility that some of these employees may carry genetic variants predisposing them to premature coronary artery disease. This reinforces the need for screening, as genetic risks cannot be identified by measuring waistlines.

This study provides a valuable snapshot, but it is not without limitations. The cross-sectional design allows us to identify associations but prevents us from establishing causality. Additionally, while we inferred the presence of the MONW phenotype based on lipid profiles and BMI, we did not directly measure body fat percentage using bioelectrical impedance analysis (BIA) or dual-energy X-ray Absorptiometry (DXA). We also lacked data on dietary intake and physical activity levels, which are significant confounders. Future research should incorporate these direct body composition measurements and lifestyle surveys to more precisely characterize this phenotype.

4. Conclusion

This study serves as a critical unmasking of the healthy weight paradox within an occupational setting in Indonesia. We conclude that normal BMI and normal waist circumference are insufficient indicators of metabolic health. A disturbing 54.93% of employees who fit the ideal anthropometric profile are actually suffering from dyslipidemia, a major precursor to cardiovascular disease. Our findings dismantle the assumption that being thin equates to being heart-healthy. We have identified a specific high-risk phenotype: male employees over the age of 40. In this group, the combination of biological aging, lack of estrogenic protection, and high prevalence of smoking creates a perfect storm for metabolic dysfunction, evidenced by a dyslipidemia prevalence exceeding 70% and a high rate of dangerous mixed lipid abnormalities. The results indicate that the current reliance on BMI as a primary filter for cardiovascular risk assessment is failing a significant portion of the workforce. By the time these invisible patients present with symptoms, they may already have advanced atherosclerosis.

Based on these compelling findings, we propose the following strategic recommendations to improve occupational and public health. The current protocol for occupational medical check-ups must be revised. Lipid profiling (TC, LDL, HDL, TG) should be mandatory for all employees over the age of 35, regardless of their BMI or waist circumference. Waiting for obesity to trigger a cholesterol check is a missed opportunity for early prevention. To better identify the Metabolically Obese Normal Weight (MONW) phenotype, medical check-ups should move beyond the scale and tape measure. The integration of body fat percentage measurement (via BIA, which is cost-effective and portable) should be standard practice. This would allow for the identification of skinny fat individuals who have a normal weight but dangerous levels of adiposity. Specific interventions should be tailored for the highest-risk group identified: males aged ≥40. Corporate wellness programs should focus on smoking cessation and muscle-building (resistance) exercise, not just weight loss. Since these individuals are already at a normal weight, the goal is metabolic fitness—improving muscle mass to increase

insulin sensitivity—rather than calorie restriction alone. For lean employees with severe isolated hypercholesterolemia, a referral for further genetic testing or family tracing should be considered to rule out Familial Hypercholesterolemia, ensuring that genetic risks are managed with appropriate pharmacotherapy. By shifting our focus from weight to metabolic health, we can close the diagnostic gap and prevent the silent progression of cardiovascular disease in the millions of adults who mistakenly believe they are safe.

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