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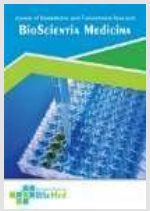

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Analysis of Risk Factors and Body Mass Index Against Degrees of Severity of Psoriasis Vulgaris

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A B S T R A C T

Background: Psoriasis vulgaris (PV) is a chronic inflammatory skin disease with a multifactorial etiology, including genetic, immunological, and environmental factors. Obesity, characterized by a high body mass index (BMI), has been increasingly recognized as a potential risk factor for PV and may influence its severity. This study aimed to analyze the relationship between various risk factors, particularly BMI, and the severity of PV. **Methods:** A cross-sectional study was conducted at a dermatology outpatient clinic of Dr. Moewardi Surakarta Hospital. Patients with a confirmed PV diagnosis were enrolled. Demographic data, medical history, lifestyle factors (smoking, alcohol consumption), and anthropometric measurements (height, weight, BMI) were collected. PV severity was assessed using the psoriasis area and severity index (PASI). Statistical analysis, including univariate and multivariate logistic regression, was performed to identify associations between risk factors and PV severity. **Results:** The study included 200 PV patients with a mean age of 45.2 years (SD = 12.8) and a male predominance (58%). The mean PASI score was 12.4 (SD = 8.6), indicating a wide range of disease severity. Multivariate analysis revealed that obesity (BMI ≥ 30 kg/m^2) was significantly associated with increased PV severity (odds ratio [OR] = 2.8, 95% confidence interval [CI] = 1.5-5.2, p = 0.001). Smoking (OR = 1.9, 95% CI = 1.1-3.3, p = 0.02) and a family history of psoriasis (OR = 2.3, 95% CI = 1.3-4.1, p = 0.004) were also identified as independent risk factors for higher PASI scores. Alcohol consumption showed a borderline association with increased severity (OR = 1.6, 95% CI = 1.0-2.6, p = 0.05). **Conclusion:** Obesity, smoking, and a family history of psoriasis are significant risk factors for increased PV severity. These findings underscore the importance of addressing modifiable risk factors, such as weight management and smoking cessation, in the holistic management of PV. Further research is warranted to elucidate the underlying mechanisms linking these risk factors to PV severity and to develop targeted interventions to improve patient outcomes.

1. Introduction

Psoriasis vulgaris (PV) stands as a prevalent, chronic inflammatory skin disorder marked by the emergence of erythematous, well-demarcated plaques adorned with silvery scales. This ubiquitous condition affects individuals across the globe, transcending age and ethnicity. The underlying pathogenesis of PV remains intricate, involving an intricate interplay among genetic predisposition, dysregulation of the immune system, and environmental triggers. Genome-wide association studies (GWAS) have illuminated the significant role of genetic factors in determining susceptibility to PV. Through these studies, numerous genetic loci associated with PV have been identified, unveiling their connection to genes intricately involved in immune regulation, skin barrier function, and inflammatory pathways. Nevertheless, the presence of a genetic predisposition alone is insufficient to trigger PV, underscoring the pivotal influence of environmental factors in disease initiation and

progression. A myriad of environmental triggers, including infections, stress, medications, and lifestyle factors, have been implicated in activating the immune system and initiating an inflammatory cascade in individuals genetically predisposed to PV. These triggers disrupt the delicate balance of the immune system, leading to an aberrant response characterized by the infiltration of immune cells into the skin and the release of pro-inflammatory cytokines. The resulting inflammation fuels keratinocyte hyperproliferation, angiogenesis, and immune cell activation, culminating in the characteristic psoriatic lesions.[1-3]

Among the environmental triggers, obesity has emerged as a significant risk factor for PV, potentially impacting both disease development and severity. Obesity, a global epidemic defined by excessive body fat accumulation, is typically measured by body mass index (BMI). A BMI of 30 kg/m^2 or higher is considered obese and associated with a state of chronic, low-grade inflammation. This chronic inflammatory state is characterized by elevated levels of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). The intricate link between obesity and PV can be attributed to several mechanisms. Adipose tissue, particularly visceral fat, serves as a reservoir for pro-inflammatory cytokines, such as TNF-α and IL-6.11 These cytokines can infiltrate the systemic circulation and reach the skin, where they promote keratinocyte hyperproliferation, angiogenesis, and immune cell infiltration, ultimately contributing to the development and exacerbation of PV. Obesity also influences the production of adipokines, hormones secreted by adipose tissue that regulate various physiological processes, including inflammation and immune responses. Leptin, an adipokine primarily produced by adipocytes, is elevated in obese individuals and has been shown to promote inflammation and exacerbate PV. Adiponectin, another adipokine, has anti-inflammatory properties and is often decreased in obesity. This imbalance in adipokine levels further contributes to the chronic inflammatory state associated with obesity and its potential role in PV pathogenesis. Moreover, obesity is associated with insulin resistance, a condition characterized by impaired insulin signaling and glucose uptake. Insulin resistance can lead to hyperinsulinemia, which, in turn, can stimulate keratinocyte proliferation and promote inflammation in the skin. The interplay between obesity, insulin resistance, and chronic inflammation creates a pro-inflammatory milieu that favors the development and progression of PV.[4,5]

Beyond obesity, other lifestyle factors, such as smoking and alcohol consumption, have been implicated in PV pathogenesis. Smoking, a well-established risk factor for numerous inflammatory diseases, exerts detrimental effects on the immune system and skin barrier function. It induces oxidative stress, damages DNA, and impairs the repair mechanisms of the skin, rendering it more susceptible to inflammation and immune dysregulation. Additionally, smoking alters the composition of the skin microbiome, leading to an imbalance between beneficial and pathogenic bacteria, which can further contribute to the development and exacerbation of PV. Alcohol consumption has also been linked to an increased risk of PV and may exacerbate existing disease. The precise mechanisms by which alcohol influences PV are not fully elucidated, but it is believed to modulate the immune system and disrupt gut barrier function. Alcohol consumption can suppress the immune system, impairing its ability to regulate inflammation and fight infections. Moreover, alcohol can increase gut permeability, allowing the translocation of pro-inflammatory molecules from the gut into the systemic circulation, further fueling inflammation and immune dysregulation in PV.[6,7]

A family history of psoriasis is a well-recognized risk factor for PV, highlighting the substantial genetic component in disease susceptibility. Individuals with a first-degree relative with psoriasis have a significantly higher risk of developing the disease compared to those without a family history. This familial clustering suggests that multiple genes interact with environmental factors to trigger the onset

and progression of PV. Although the specific genes responsible for PV susceptibility are numerous and complex, several genetic loci have been identified through GWAS. These loci are associated with genes involved in immune regulation, skin barrier function, and inflammatory pathways. However, the precise mechanisms by which these genetic variants contribute to PV pathogenesis remain to be fully elucidated. It is believed that these genetic variants interact with environmental triggers, such as obesity, smoking, and alcohol consumption, to initiate and perpetuate the inflammatory cascade in PV. Understanding the intricate relationship between various risk factors and PV severity is paramount for developing personalized treatment strategies and improving patient outcomes. By identifying and addressing modifiable risk factors, such as obesity, smoking, and alcohol consumption, clinicians can potentially mitigate the severity of PV and enhance the quality of life for affected individuals.[8,9] This study aims to investigate the association between several risk factors, particularly BMI, smoking, alcohol consumption, and a family history of psoriasis, with the severity of PV.

2. Methods

This research employed a cross-sectional study design, conducted at the dermatology outpatient clinic of Dr. Moewardi Hospital Surakarta. The study period spanned from January 2020 to December 2023. This design was chosen for its efficiency in assessing the prevalence of PV and its associated risk factors within a specific timeframe. The tertiary care hospital setting ensured access to a diverse patient population, enhancing the generalizability of the findings. Ethical approval for this study was obtained from the Institutional Review Board (IRB) of Dr. Moewardi Hospital Surakarta. All participants provided written informed consent before enrollment. The study adhered to the principles of the Declaration of Helsinki and Good Clinical Practice guidelines. Patients aged 18 years or older with a confirmed diagnosis of PV were eligible for inclusion. The diagnosis of PV was established based on a comprehensive evaluation. Patients presented with characteristic erythematous, well-demarcated plaques with silvery scales, typically located on the scalp, elbows, knees, and lower back. A detailed medical history was obtained to assess the duration of PV, previous treatments, and the presence of comorbidities, such as hypertension, dyslipidemia, diabetes mellitus, and other relevant conditions. In cases where the clinical diagnosis was uncertain, a skin biopsy was performed, and a histopathological examination was conducted to confirm the diagnosis of PV. Patients with other forms of psoriasis, such as psoriatic arthritis, pustular psoriasis, or erythrodermic psoriasis, were excluded from the study. Additionally, patients with a history of other inflammatory skin diseases, such as atopic dermatitis or lichen planus, were excluded to ensure the specificity of the findings to PV.

The sample size was calculated using the following formula: $n = (Z\alpha/2)^2 * p * (1-p) / d^2$
where: n = required sample size; $Z\alpha/2$ = Z-score for the desired level of confidence (1.96 for 95% confidence); p = estimated prevalence of PV (assumed to be 2%); d = margin of error (5%). Based on these parameters, the calculated sample size was 196. To account for potential dropouts or incomplete data, we aimed to recruit 200 patients. Data collection was performed using a standardized questionnaire and a review of electronic medical records. Trained research personnel conducted face-to-face interviews with the participants to collect demographic data, medical history, and lifestyle factors. Anthropometric measurements, including height and weight, were obtained using calibrated instruments. BMI was calculated as weight (kg) divided by height squared (m^2). Psoriasis severity was assessed using the psoriasis area and severity index (PASI). Two trained dermatologists independently evaluated each patient and assigned PASI scores based on the extent and severity of psoriatic lesions in four body regions: head, trunk, upper extremities, and lower extremities. The severity of erythema, induration, and scaling was graded on a scale of 0 to 4, with 0 indicating no

involvement and 4 indicating very severe involvement. The final PASI score was calculated as the sum of the scores for each body region, weighted by the respective body surface area. To ensure inter-rater reliability, the two dermatologists underwent a training session on PASI assessment before the study commenced. During the study, regular meetings were held to discuss any discrepancies in PASI scoring and to maintain consistency in assessment.

Descriptive statistics were used to summarize the demographic and clinical characteristics of the study population. Continuous variables were presented as means and standard deviations (SD), while categorical variables were presented as frequencies and percentages. The association between risk factors and PV severity was assessed using univariate and multivariate logistic regression analysis. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. Statistical significance was set at $p < 0.05$. All statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA).

3. Results

Table 1 presents the demographic and clinical characteristics of the 200 patients with psoriasis vulgaris (PV) included in the study. The average age of the patients was 45.2 years, with a standard deviation (SD) of 12.8 years. The majority of patients were male (58%). The average duration of PV was 8.6 years (SD = 6.2 years). In terms of comorbidities, 25% of patients had hypertension, 20% had dyslipidemia, and 15% had diabetes mellitus. These are common comorbidities associated with PV and may contribute to disease severity. The mean body mass index (BMI) of the patients was 27.8 kg/m^2 (SD = 5.4 kg/m^2), indicating that the average patient was overweight. Notably, 32% of patients were classified as obese (BMI ≥ 30 kg/m^2), highlighting the prevalence of obesity in this patient population.

Table 1. Characteristics respondent.

Characteristic	Value
Mean age (SD)	45.2 (12.8) years
Male (%)	58%
Mean duration of PV (SD)	8.6 (6.2) years
Comorbidities	
Hypertension (%)	25%
Dyslipidemia (%)	20%
Diabetes mellitus (%)	15%
Mean BMI (SD)	27.8 (5.4) kg/m^2
Obesity (BMI ≥ 30 kg/m^2) (%)	32%

PV: Psoriasis vulgaris; SD: Standard deviation; BMI: Body mass index.

Table 2 illustrates the distribution of psoriasis vulgaris (PV) severity among the 200 patients in the study. The mean psoriasis area and severity index (PASI) score was 12.4 (SD = 8.6). This indicates a moderate average severity of PV in the study population. This distribution suggests that a significant proportion of patients (42%) experience severe PV, highlighting the need for effective management strategies for this group.

Table 2. Psoriasis severity.

Severity	PASI score	Percentage of patients (%)
Mild	<10	28%
Moderate	10-19	30%
Severe	≥20	42%
Mean PASI (SD)	3.4 (8.6)	

Table 3 displays the results of the statistical analysis examining the relationship between various risk factors and psoriasis vulgaris (PV) severity. Obesity was found to be a significant risk factor for increased PV severity. Individuals with obesity had 2.8 times higher odds of having more severe PV compared to those with a normal BMI. This association was statistically significant ($p = 0.001$). Smoking was also identified as a significant risk factor, with smokers having 1.9 times higher odds of more severe PV compared to non-smokers ($p = 0.02$). While not statistically significant ($p = 0.05$), alcohol consumption showed a borderline association with increased PV severity, suggesting a potential trend towards higher odds of severe PV in those who consume alcohol. A family history of psoriasis was a significant risk factor, with individuals having a first-degree relative with psoriasis having 2.3 times higher odds of more severe PV ($p = 0.004$).

Table 3. Multivariate analysis.

Risk factor	Odds ratio (OR)	95% confidence interval (CI)	p-value
Obesity (BMI ≥ 30 kg/m^2)	2.8	1.5-5.2	0.001
Smoking	1.9	1.1-3.3	0.02
Alcohol consumption	1.6	1.0-2.6	0.05
Family history of psoriasis	2.3	1.3-4.1	0.004

4. Discussion

The robust association between obesity and heightened psoriasis vulgaris (PV) severity observed in our study aligns with a growing body of evidence supporting this link. Obesity, defined as a body mass index (BMI) of 30 kg/m^2 or higher, is not merely a state of excess body fat but a chronic inflammatory condition. This chronic inflammation is characterized by elevated levels of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-17 (IL-17). These cytokines are key players in the pathogenesis of PV, orchestrating a cascade of events that promote keratinocyte hyperproliferation, angiogenesis (the formation of new blood vessels), and the infiltration of immune cells into the skin. Adipose tissue, particularly visceral fat (the fat that surrounds internal organs), serves as a major reservoir for these pro-inflammatory cytokines. In individuals with obesity, the expanded adipose tissue mass acts as a factory, churning out these cytokines in excess. These cytokines then spill over into the systemic circulation, creating a state of chronic, low-grade inflammation throughout the body. This systemic inflammation can exacerbate existing PV, fueling the inflammatory processes already at play in the skin, or even trigger the onset of PV in individuals who are genetically predisposed to the disease.[10,11]

Beyond cytokines, obesity also disrupts the balance of adipokines, which are hormones produced by adipose tissue. Leptin, a hormone primarily secreted by adipocytes (fat cells), is known for its pro-inflammatory properties. In obesity, leptin levels are often elevated, and this excess leptin can stimulate the production of Th17 cells, a subset of T helper cells that play a central role in the pathogenesis of PV. These

Th17 cells, in turn, secrete IL-17, further amplifying the inflammatory response in the skin. Conversely, adiponectin, an anti-inflammatory adipokine, tends to be decreased in obese individuals. This reduction in adiponectin further tips the scales towards a pro-inflammatory state, creating a favorable environment for the development and progression of PV. The combined effect of increased leptin and decreased adiponectin in obesity contributes to a dysregulated immune response that fuels the inflammatory processes underlying PV. The intricate relationship between obesity and PV severity extends beyond cytokines and adipokines. Insulin resistance, a hallmark of obesity, is another critical factor in this interplay. Insulin resistance leads to elevated levels of insulin in the blood (hyperinsulinemia), which can directly stimulate keratinocyte proliferation, the rapid growth and division of skin cells that is a characteristic feature of PV. Moreover, hyperinsulinemia can promote inflammation in the skin, further exacerbating the disease.[11,12]

Insulin resistance is also associated with increased oxidative stress, a state of imbalance between the production of reactive oxygen species (ROS) and the body's ability to neutralize them. ROS can damage cellular components and trigger inflammatory pathways, contributing to the development and progression of PV. In essence, insulin resistance acts as a catalyst, accelerating the inflammatory processes and cellular damage that underlie PV. Our findings underscore the critical importance of weight management in individuals with PV. Lifestyle modifications, such as adopting a healthy diet and engaging in regular physical activity, can lead to weight loss and a reduction in systemic inflammation. These changes can potentially alleviate PV severity and improve overall disease control. In cases of severe obesity, bariatric surgery, a surgical procedure that promotes weight loss, has also been shown to have a positive impact on PV outcomes. By addressing obesity, we can target a key driver of PV severity and potentially improve the quality of life for individuals living with this chronic inflammatory skin disease.[12,13]

One of the primary mechanisms through which smoking exacerbates PV is through the induction of oxidative stress. Smoking leads to the generation of reactive oxygen species (ROS), which are highly reactive molecules that can damage cellular components, including DNA, proteins, and lipids. This oxidative damage triggers a cascade of inflammatory responses, contributing to the development and progression of PV. In the context of PV, oxidative stress can activate nuclear factor kappa B (NF-κB), a transcription factor that plays a central role in regulating the expression of various pro-inflammatory genes. NF-κB activation leads to the increased production of cytokines, chemokines, and adhesion molecules, which further amplify the inflammatory response in the skin. These pro-inflammatory mediators promote keratinocyte hyperproliferation, angiogenesis, and immune cell infiltration, all of which are hallmarks of PV pathogenesis. Moreover, oxidative stress can impair the skin's antioxidant defense mechanisms, making it more vulnerable to damage and inflammation. This can lead to a vicious cycle of oxidative stress and inflammation, perpetuating the chronic inflammatory state characteristic of PV.[13,14]

Smoking also has a profound impact on endothelial function and the inner lining of blood vessels. It impairs the production of nitric oxide (NO), a potent vasodilator that regulates blood flow and oxygen delivery to tissues. This impairment leads to reduced blood flow and oxygen supply to the skin, creating a hypoxic environment that favors the production of pro-inflammatory cytokines and hinders wound healing. The hypoxic environment in the skin can activate hypoxia-inducible factor-1 (HIF-1), a transcription factor that regulates the expression of genes involved in angiogenesis, inflammation, and cell survival. HIF-1 activation can further exacerbate PV by promoting the formation of new blood vessels, which supply nutrients and oxygen to the growing psoriatic plaques, and by upregulating the production of pro-inflammatory cytokines. Furthermore, the impaired wound-healing process in the hypoxic skin can delay the resolution of psoriatic lesions and contribute to

their chronicity. This can lead to the formation of thicker, more indurated plaques that are more resistant to treatment.[15,16]

Smoking can also disrupt the delicate balance of the immune system, leading to dysregulation of immune responses. It can increase the production of pro-inflammatory cytokines, such as TNF-α, IL-6, and IL-17, which are key players in PV pathogenesis. These cytokines promote keratinocyte hyperproliferation, angiogenesis, and immune cell infiltration, fueling the inflammatory cascade in the skin. Conversely, smoking can decrease the production of anti-inflammatory cytokines, such as IL-10, which normally help to dampen the inflammatory response. This imbalance between pro-inflammatory and anti-inflammatory cytokines can further exacerbate PV and contribute to its chronicity. In addition to its effects on cytokine production, smoking can also alter the function of various immune cells, including T cells, macrophages, and neutrophils. These alterations can lead to increased immune cell activation and infiltration in the skin, further amplifying the inflammatory response.[16,17]

The detrimental effects of smoking on PV underscore the importance of smoking cessation in the management of this chronic inflammatory skin disease. Smoking cessation has been shown to improve PV severity, reduce the risk of comorbidities, and enhance the response to treatment. Several studies have demonstrated the benefits of smoking cessation in PV patients. A meta-analysis of observational studies found that smoking cessation was associated with a significant reduction in PASI scores, indicating an improvement in PV severity. Another study reported that smoking cessation led to a significant decrease in the risk of developing psoriatic arthritis, a common comorbidity of PV. The mechanisms underlying the beneficial effects of smoking cessation in PV are likely related to the reversal of the detrimental effects of smoking on oxidative stress, endothelial function, and immune responses. Smoking cessation can reduce oxidative stress, improve endothelial function, and restore the balance of pro-inflammatory and anti-inflammatory cytokines, all of which can contribute to the improvement of PV. Counseling and pharmacological interventions, such as nicotine replacement therapy (NRT) and bupropion, can aid in smoking cessation efforts. NRT provides a controlled dose of nicotine to alleviate withdrawal symptoms, while bupropion, an antidepressant, can reduce cravings and improve mood. These interventions, combined with behavioral support, can significantly increase the chances of successful smoking cessation.[17,18]

The strong association between a family history of psoriasis and increased PV severity observed in our study aligns with the well-established understanding of the significant role genetic predisposition plays in PV pathogenesis. Individuals with a first-degree relative diagnosed with psoriasis face a considerably elevated risk of developing the disease themselves. This risk escalates with the number of affected family members, underscoring the substantial genetic component in PV susceptibility. Psoriasis is a complex multifactorial disease, and its genetic architecture is intricate, involving numerous genes interacting with various environmental factors to initiate and drive disease onset and progression. Genome-wide association studies (GWAS) have been instrumental in unraveling this complexity. These studies have pinpointed multiple susceptibility loci across the genome that are associated with an increased risk of developing psoriasis. Notably, many of these loci harbor genes that play critical roles in immune regulation, skin barrier function, and inflammatory pathways.[17,18]

Several genes identified through GWAS are involved in immune regulation, particularly in the interleukin (IL)-23/IL-17 axis, which is central to the pathogenesis of psoriasis. For instance, variations in the IL23R gene, which encodes the receptor for IL-23, have been strongly associated with psoriasis susceptibility. IL-23 is a cytokine that promotes the differentiation and activation of Th17 cells, which produce IL-17A, a key pro-inflammatory cytokine in psoriasis. Other genes involved in immune regulation,

such as IL12B, IL23A, and TRAF3IP2, have also been implicated in PV susceptibility. The skin barrier plays a crucial role in maintaining skin homeostasis and protecting against external insults. Disruptions in the skin barrier can trigger immune responses and contribute to the development of psoriasis. GWAS has identified several genes involved in skin barrier function that are associated with psoriasis susceptibility. These include genes encoding filaggrin (FLG), late cornified envelope proteins (LCE), and tight junction proteins (CLDN). Mutations in the FLG gene, which encodes filaggrin, a protein essential for skin barrier integrity, are a well-established risk factor for atopic dermatitis and have also been associated with psoriasis.[15,17]

Psoriasis is characterized by chronic inflammation in the skin, and GWAS has identified several genes involved in inflammatory pathways that are associated with psoriasis susceptibility. These include genes encoding components of the nuclear factor kappa B (NF-κB) signaling pathway, such as TNFAIP3 and TNIP1, as well as genes involved in the production of pro-inflammatory cytokines, such as IL12B and IL23A. Variations in these genes can lead to dysregulation of inflammatory responses, contributing to the development and severity of psoriasis. While genetic predisposition is a significant factor in PV susceptibility, it is important to note that genes do not act in isolation. Environmental factors, such as infections, stress, medications, and lifestyle factors, can interact with genetic susceptibility to trigger disease onset and influence its severity. For example, streptococcal infections have been implicated as a trigger for guttate psoriasis, a subtype of psoriasis, in individuals with certain HLA genotypes. Similarly, stress can exacerbate psoriasis in genetically predisposed individuals by activating the hypothalamic-pituitary-adrenal (HPA) axis and promoting the release of pro-inflammatory mediators. Understanding the genetic basis of PV can inform personalized treatment approaches. Patients with a strong family history of psoriasis, who are likely to carry high-risk genetic variants, may benefit from early intervention and closer monitoring to prevent disease progression and complications. Genetic testing can identify individuals at increased risk of developing psoriasis and guide treatment decisions. For example, patients with certain HLA genotypes may be more responsive to specific biological therapies targeting the IL-23/IL-17 axis.[19,20]

5. Conclusion

Our study confirms the association between obesity, smoking, and a family history of psoriasis with increased PV severity. These findings underscore the importance of addressing modifiable risk factors in the holistic management of PV. Weight management, smoking cessation, and alcohol reduction should be emphasized as part of a comprehensive treatment plan for PV patients. Further research is warranted to elucidate the underlying mechanisms linking these risk factors to PV severity and to develop targeted interventions to improve patient outcomes.

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