

Article

Smoking, Central Obesity, and Periodontitis Among Iraqi Dental Patients: Exploring Metabolic-Behavioral Risk Clustering in a Cross-Sectional Study

Mohamed Saeed M. Ali , Omar Husham Ali  and Hadeel Mazin Akram * 

College of Dentistry, University of Baghdad, Baghdad 5421, Iraq; mohammed.s@codental.uobaghdad.edu.iq (M.S.M.A.); omar.husham@codental.uobaghdad.edu.iq (O.H.A.)

* Correspondence: hadeel.mazin@codental.uobaghdad.edu.iq

Abstract

Smoking and central obesity have both been linked to periodontitis, but their combined relationship with periodontal disease may be influenced by demographic and behavioral factors. This cross-sectional study analyzed records of 420 adult dental patients attending the College of Dentistry at the University of Baghdad. Data included demographic characteristics, smoking status, periodontal clinical findings, body mass index (BMI), and waist-to-height ratio (WHtR). Periodontitis was defined according to the 2018 classification framework, and logistic regression models were used to examine the associations of smoking and obesity-related indicators with periodontitis. The overall prevalence of periodontitis was 36.4%. Participants with periodontitis were significantly older than those without periodontitis (46.0 vs. 28.9 years; $p < 0.0001$). In the fully adjusted model, age remained the strongest factor associated with periodontitis (OR = 1.15 per year; 95% CI: 1.11–1.18; $p < 0.001$). The apparent association between smoking and periodontitis was substantially influenced by age, as current smoking was more common among younger participants in this sample. The association between smoking status and periodontitis appeared to differ according to WHtR category (interaction term $p = 0.016$); however, this finding should be interpreted cautiously because of the cross-sectional design and age imbalance across exposure groups. Overall, the findings suggest that age was the dominant factor associated with periodontitis in this dental patient sample, while the relationship between smoking, central obesity, and periodontitis requires further investigation in longitudinal studies with detailed smoking and metabolic data.

Keywords: periodontitis; smoking; central obesity; waist-to-height ratio; cross-sectional study; age



Academic Editors: Marwan El Ghoch and Adelia Bovell-Benjamin

Received: 20 May 2026

Revised: 22 June 2026

Accepted: 23 June 2026

Published: 25 June 2026

Copyright: © 2026 by the authors.

Licensee MDPI, Basel, Switzerland.

This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC BY\)](https://creativecommons.org/licenses/by/4.0/) license.

1. Introduction

Periodontitis is a chronic inflammatory disease affecting the supporting tissues of the teeth, leading to periodontal ligament destruction and alveolar bone loss [1–3]. If left untreated, periodontitis can result in tooth loss, affecting aesthetics, mastication, and quality of life. Worldwide, the prevalence of severe periodontitis is thought to be 10–15% of the global population; many more are affected by milder degrees of the disease [2,4]. The pathogenesis of periodontitis is largely attributed to an intricate interplay between dysbiosis in the subgingival microbiome and the host's immune-inflammatory response [5]. Although early dental biofilm formation is largely initiated by facultative Gram-positive

bacteria, progression to periodontitis is associated with a dysbiotic subgingival biofilm enriched with Gram-negative anaerobes. Disease progression and severity are further influenced by host immune-inflammatory responses and environmental and behavioral risk factors [6].

Of these modifiable risk factors, tobacco smoking is the most important one [7–9]. A wealth of evidence, including many systematic reviews and meta-analyses, has unequivocally demonstrated that smokers have a significantly increased burden of prevalence and severity of periodontitis when compared with non-smokers [10,11]. The prevalence of periodontitis is thought to increase by 85% in smokers [7,8]. The pathophysiological mechanisms are multifaceted. Tobacco use induces a more pathogenic subgingival microbial profile, depresses the activity of neutrophils (which are the first line of defense against periodontal pathogens), and damages gingival microvasculature [11–13]. This vascular dysfunction results in decreased blood flow and can cover up an important clinical signal of inflammation, which is the bleeding on probing (BOP), typically providing the wrong impression that smokers are periodontally healthy [13]. In addition, smoking can negatively influence the results of non-surgical and surgical periodontal treatment, creating difficulties in managing diseases in these patients [9].

Obesity has also been increasingly recognized as a major risk factor for several chronic non-communicable diseases, including periodontitis [14,15]. Obesity is currently regarded as a chronic condition with low-grade systemic inflammation [16]. Adipose tissue, visceral fat in particular, is not only a passive reservoir of energy storage but an active endocrine tissue that releases several pro-inflammatory cytokines (adipokines) like tumor necrosis factor- α (TNF- α) and interleukin 6 (IL-6) [17,18].

In addition to classical inflammatory cytokines like TNF- α and IL-6, central obesity is linked to changes in adipokine profiles. Leptin, adiponectin, resistin, and omentin-1 are mediators from adipose tissue that might connect visceral fat with periodontal inflammation [19]. Leptin and resistin are usually linked to pro-inflammatory activity, while adiponectin and omentin-1 are often seen as having anti-inflammatory or metabolically protective roles. Changes in these adipokines can affect systemic inflammation, immune responses, periodontal tissue breakdown, and healing after periodontal therapy [20].

These circulating inflammatory mediators can prime the host for an exaggerated inflammatory response to bacterial challenge in the periodontium, thereby amplifying tissue destruction [16]. While BMI is a convenient and widely used measure of general obesity, it does not differentiate between fat and muscle mass or describe fat distribution. Measures of central or abdominal obesity, such as Waist Circumference (WC) and Waist-to-Height Ratio (WHtR), are considered better indicators of visceral adiposity and are more strongly correlated with metabolic disease and systemic inflammation [21,22]. Several studies have now demonstrated a stronger association between central obesity metrics like WHtR and periodontitis compared to BMI, highlighting the importance of fat distribution in the obesity-periodontitis axis [21,23].

In real clinical settings, behavioral and metabolic factors often coexist rather than acting in isolation. Smoking and obesity may cluster within the same patient profile, creating a complex metabolic-behavioral context that could influence periodontal health [24,25]. Nevertheless, the interaction of smoking, central obesity, and periodontitis is complex and still difficult to interpret due to the possibility of unmeasured confounding factors driven by age and lifestyle. Despite the evidence suggesting that both smoking and obesity have negative effects on periodontal health, most previous studies investigated smoking and obesity separately, and fewer have investigated whether their coexistence impacts their associations with periodontitis, especially when central obesity is assessed using waist-to-height ratio instead of only BMI.

This question is relevant in dental patient populations, where behavioral and metabolic risk indicators may coexist. Thus, the current cross-sectional study was conducted to assess the associations between smoking status, obesity indicators (BMI and WHtR) and periodontitis among adult patients attending a university dental clinic in Baghdad/Iraq. The study also explored whether smoking and central obesity show evidence of metabolic-behavioral risk clustering in relation to periodontitis. We hypothesized that smoking and central obesity may interact in their association with periodontitis; however, given the cross-sectional design, the study was intended to assess associations rather than causal effects.

2. Materials and Methods

2.1. Study Design and Population

This analytical cross-sectional study was based on a secondary analysis of existing clinical records from 420 adult patients who attended the Periodontology Clinic, College of Dentistry, University of Baghdad, Baghdad, Iraq. The dataset included demographic information, smoking status, periodontal clinical findings, body weight, height, and waist circumference.

Eligible participants were adults aged 18 years or older who had complete records for periodontal diagnosis, age, sex, smoking status, height, weight, and waist circumference. Patients were excluded if they were edentulous or if the variables required for the present analysis were incomplete. The sample therefore represents a university dental clinic sample and should not be interpreted as a population-based sample of Iraqi adults.

The dataset included participants classified as periodontally healthy, having gingivitis, or having periodontitis; therefore, the study population should be understood as dental patients with complete periodontal records, not exclusively patients with periodontitis.

The study was approved by the Ethics Committee of the College of Dentistry, University of Baghdad, Iraq (Ref. number: 1249, approved on 7 January 2025). The study was conducted in accordance with the Declaration of Helsinki, and informed consent was obtained from all participants during the original data collection.

2.2. Clinical Examination

All included records contained full-mouth periodontal examination data. Periodontal measurements were recorded at six sites per tooth, excluding third molars: mesiobuccal, mid-buccal, distobuccal, mesiolingual, mid-lingual, and distolingual sites. The recorded clinical parameters included probing pocket depth (PPD), clinical attachment loss (CAL), bleeding on probing (BOP), and plaque index (PI). PPD was measured in millimeters from the gingival margin to the bottom of the periodontal pocket. CAL was measured in millimeters from the cemento-enamel junction to the bottom of the periodontal pocket. BOP was recorded as present or absent after probing, and PI was used as a clinical indicator of oral hygiene and biofilm accumulation.

The original dataset operationalized periodontal diagnosis according to the 2018 classification framework using full-mouth periodontal charting. Participants were classified as periodontally healthy, having gingivitis, or having periodontitis on the basis of recorded PPD, CAL, BOP, and plaque index findings. Periodontitis was identified when destructive periodontal attachment loss was consistent with the 2018 case-definition framework [26], including interdental CAL at two or more non-adjacent teeth or buccal/oral CAL of at least 3 mm with PPD greater than 3 mm at two or more teeth, when the clinical record did not indicate a non-periodontal cause of attachment loss. Participants without periodontitis-level attachment loss were classified as having gingivitis when gingival inflammation was present without destructive periodontal attachment loss, or as periodontally healthy when destructive attachment loss and relevant inflammatory findings were absent. Data on

periodontitis stage, grade, extent, and localization were not consistently available for all participants; therefore, these variables were excluded from the present regression models.

2.3. Variable Definitions

The primary outcome was the presence of periodontitis. For logistic regression analysis, the diagnostic variable was converted into a binary outcome. Participants diagnosed with periodontitis were coded as 1, while participants classified as periodontally healthy or having gingivitis were combined into a no-periodontitis group and coded as 0. This grouping was used to enable binary logistic regression.

Smoking status was analyzed as a binary exposure variable. Participants recorded as current smokers were coded as 1 (current smokers were defined as participants who reported smoking at the time of examination), while participants recorded as non-smokers were coded as 0. Information on former smoking, smoking duration, pack-years, smoking intensity, and type of tobacco product was not available in the dataset.

General obesity was assessed using body mass index (BMI), calculated as weight in kilograms divided by height in meters squared. BMI was categorized according to World Health Organization criteria as normal weight ($<25 \text{ kg/m}^2$), overweight ($25\text{--}29.9 \text{ kg/m}^2$), and obese ($\geq 30 \text{ kg/m}^2$). For regression modeling, BMI was also converted into a binary variable: normal weight ($<25 \text{ kg/m}^2$) and overweight/obese ($\geq 25 \text{ kg/m}^2$) [17].

Central obesity was assessed using waist-to-height ratio (WHtR), calculated by dividing waist circumference by height. WHtR was categorized as normal (<0.5) or high risk (≥ 0.5) based on commonly used clinical thresholds for central adiposity [21].

The main covariates included in the adjusted analyses were age, sex, education level, and brushing frequency. Brushing frequency was recoded into three categories for regression analysis: $0\times/\text{day}$, $1\times/\text{day}$, and $\geq 2\times/\text{day}$. This grouping was used because the higher brushing-frequency categories included very few observations and no periodontitis cases in some categories.

Plaque index was described clinically but was not included in the primary regression models. Age was analyzed as a continuous variable. Sex was coded as male or female. Education level and brushing frequency were treated as categorical variables.

2.4. Statistical Analysis

All statistical analyses were performed using Python version 3.11 with the pandas, scipy, and statsmodels libraries. Statistical significance was set at $p < 0.05$.

Descriptive statistics were calculated for the total sample and according to periodontitis status. Continuous variables were presented as mean $\pm$ standard deviation, while categorical variables were presented as frequencies and percentages. Continuous variables were compared between groups using independent t -tests or Mann–Whitney U tests, depending on data distribution. Categorical variables were compared using the Chi-square test or Fisher's exact test when appropriate.

Logistic regression models were used to examine the associations of smoking status, BMI, and WHtR with the odds of periodontitis. The adjusted models included age, sex, education level, and brushing frequency. Plaque index was described clinically but was not included in the primary adjusted models because it may function as both a local etiological factor and a potential mediator within the periodontal disease pathway.

Three regression approaches were specified. Model 1 examined smoking status, BMI category, and their interaction term. Model 2 examined smoking status, WHtR category, and their interaction term. Model 3 used a combined exposure variable based on smoking status and WHtR category, producing four mutually exclusive groups: non-smoker with normal WHtR, non-smoker with high WHtR, smoker with normal WHtR, and smoker with

high WHtR. The non-smoker with normal WHtR group was used as the reference category. The full output of the regression models is provided in Supplementary Table S1.

Adjusted odds ratios (ORs), 95% confidence intervals (CIs), and *p*-values were reported. Interaction terms were interpreted as statistical interactions or possible effect modifications, not as proof of biological synergy. Because some combined exposure groups included few periodontitis cases, these subgroup estimates were interpreted with caution.

3. Results

3.1. Sample Characteristics

The final study sample consisted of 420 participants. The mean age of the cohort was 35.2 ± 13.4 years, and 52.1% of participants were male. A high proportion of participants had central obesity, with 74.3% showing a WHtR value of 0.5 or greater. Current smokers represented 51.9% of the sample. The overall prevalence of periodontitis was 36.4% ($n = 153$).

Table 1 and Figure 1 compare baseline demographic, behavioral, anthropometric, and periodontal characteristics by periodontitis status. Participants with periodontitis were significantly older than those without periodontitis (46.0 ± 12.7 vs. 28.9 ± 9.3 years; $p < 0.0001$). The proportion of participants with high WHtR was also higher in the periodontitis group than in the no-periodontitis group (85.0% vs. 68.2%; $p = 0.0002$). As expected, periodontal clinical parameters were worse among participants with periodontitis. The mean PPD was 5.11 ± 0.66 mm in the periodontitis group compared with 2.53 ± 0.31 mm in the no-periodontitis group, while the mean CAL was 3.96 ± 3.34 mm compared with 0.00 ± 0.00 mm, respectively.

Current smoking was less frequent among participants with periodontitis than among those without periodontitis (27.5% vs. 65.9%; $p < 0.0001$). This finding should be interpreted cautiously because smoking was more common among younger participants in this sample, whereas periodontitis was strongly associated with older age.

Table 1. Baseline Characteristics of Study Participants.

Characteristic	Total ($n = 420$)	No Periodontitis ($n = 267$)	Periodontitis ($n = 153$)
Age (years), mean $\pm$ SD	35.2 ± 13.4	28.9 ± 9.3	46.0 ± 12.7
Male gender, n (%)	219 (52.1)	131 (49.1)	88 (57.5)
Current smoker, n (%)	218 (51.9)	176 (65.9)	42 (27.5)
BMI—Normal, n (%)	175 (41.7)	126 (47.2)	49 (32.0)
BMI—Overweight, n (%)	155 (36.9)	89 (33.3)	66 (43.1)
BMI—Obese, n (%)	90 (21.4)	52 (19.5)	38 (24.8)
WHtR ≥ 0.5 (high risk), n (%)	312 (74.3)	182 (68.2)	130 (85.0)
Plaque Index, mean $\pm$ SD	0.57 ± 0.24	0.49 ± 0.25	0.71 ± 0.14
BOP proportion, mean $\pm$ SD	0.37 ± 0.23	0.29 ± 0.24	0.51 ± 0.15
PPD (mm), mean $\pm$ SD	3.47 ± 1.33	2.53 ± 0.31	5.11 ± 0.66
CAL (mm), mean $\pm$ SD	1.44 ± 2.77	0.00 ± 0.00	3.96 ± 3.34

3.2. Combined Exposure Groups and Periodontitis Prevalence

Participants were classified into four combined exposure groups according to smoking status and WHtR category: non-smoker with normal WHtR, non-smoker with high WHtR, smoker with normal WHtR, and smoker with high WHtR. The unadjusted prevalence of periodontitis differed across these groups, as shown in Table 2 and Figure 2.

Demographic and clinical characteristics by periodontitis status

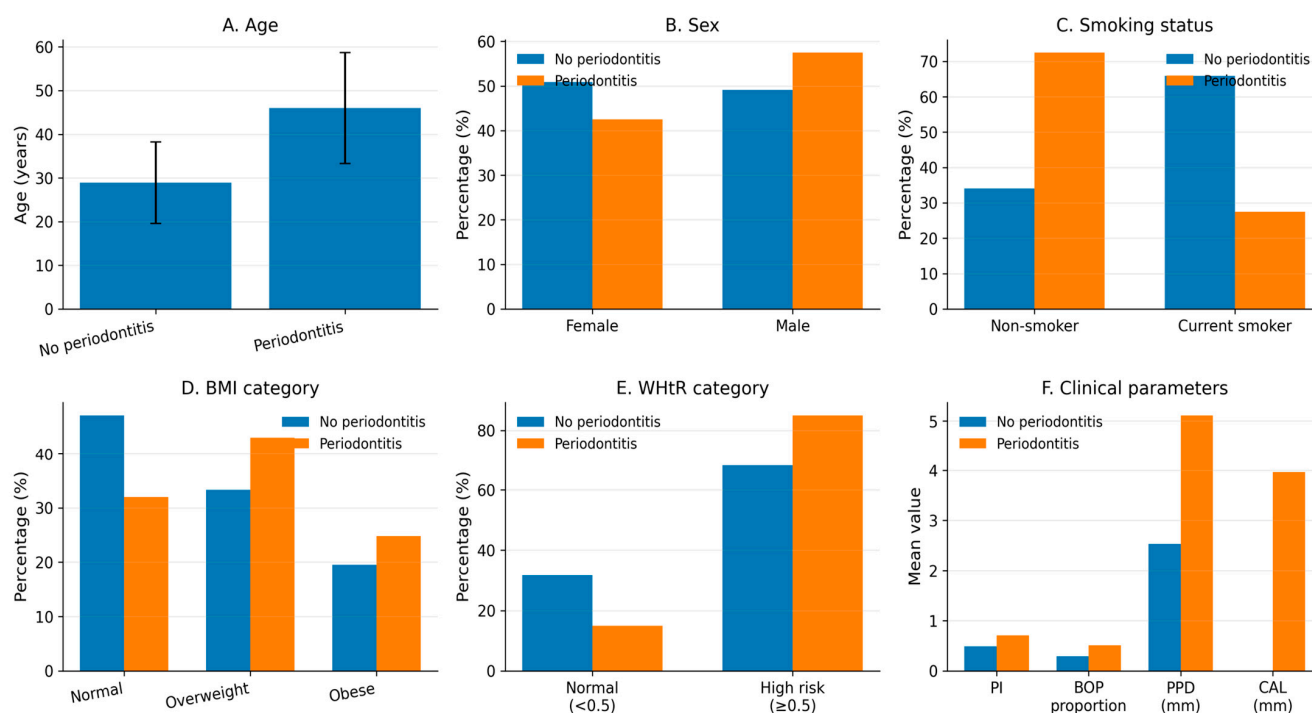


Figure 1. The figure summarizes differences in age, sex, smoking status, BMI category, WHtR category, and periodontal clinical parameters between participants with and without periodontitis.

Table 2. Periodontitis Prevalence by Combined Exposure Group.

Exposure Group	Total (n)	Periodontitis Cases (n)	Prevalence (%)
Non-smoker + Normal WHtR	39	19	48.7%
Non-smoker + High WHtR	163	92	56.4%
Smoker + Normal WHtR	69	4	5.8%
Smoker + High WHtR	149	38	25.5%

Combined exposure groups and periodontitis prevalence

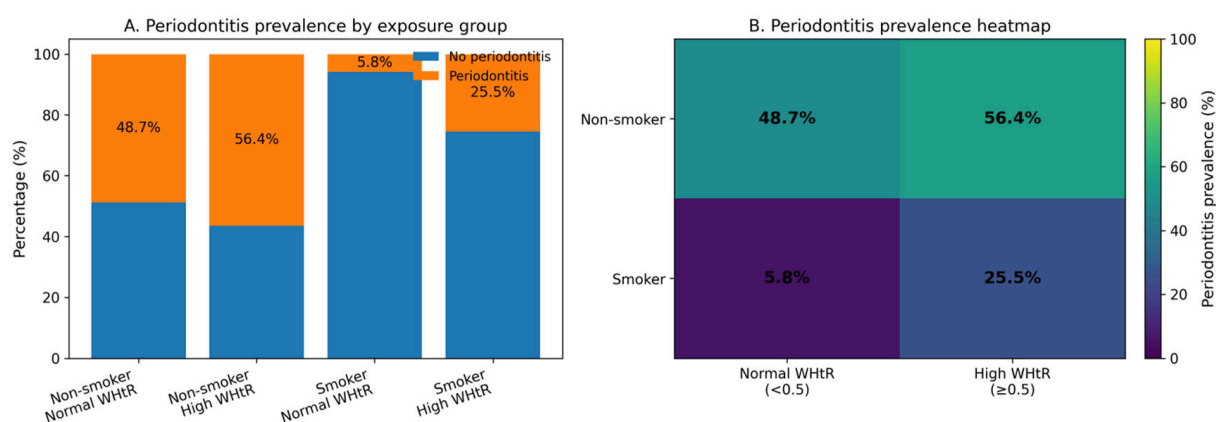


Figure 2. The stacked bar chart and heatmap show crude prevalence values and should be interpreted considering the age imbalance across exposure groups.

The highest unadjusted prevalence of periodontitis was observed among non-smokers with high WHtR, followed by non-smokers with normal WHtR. Lower unadjusted prevalence was observed among smokers with high WHtR, followed by smokers with normal WHtR.

lence values were observed among the two smoking groups. This pattern appears to be strongly influenced by the sample's age distribution, as non-smokers, particularly those with high WHtR, were generally older than smokers. Therefore, these unadjusted subgroup differences should not be interpreted as evidence that smoking is protective against periodontitis, as shown in Table 2 and Figure 2.

3.3. Multivariable Logistic Regression Analysis

The results of the multivariable logistic regression analyses are summarized in Table 3 and Figure 3, with the full model outputs provided in Supplementary Table S1. Table 3 presents the adjusted combined-exposure model (Model 3). The adjusted models included age, sex, education level, and brushing frequency. Plaque index was assessed clinically but was not included in the primary regression models.

Table 3. Adjusted Odds Ratios for Periodontitis from Logistic Regression (Model 3).

Variable	Adjusted OR (95% CI)	p-Value	Significance
Non-smoker + Normal WHtR	Reference	Reference	—
Non-smoker + High WHtR	0.24 (0.09–0.64)	0.005	<0.01
Smoker + Normal WHtR	0.07 (0.02–0.31)	<0.001	<0.001
Smoker + High WHtR	0.12 (0.04–0.35)	<0.001	<0.001
Age (per year)	1.15 (1.11–1.18)	<0.001	<0.001
Male gender	2.23 (1.14–4.36)	0.019	<0.05

OR, odds ratio; CI, confidence interval; WHtR, waist-to-height ratio. Model adjusted for age, sex, education level, and brushing frequency. Brushing frequency was modeled as 0×/day, 1×/day, and ≥2×/day. Full outputs from Models 1–3 are provided in Supplementary Table S1.

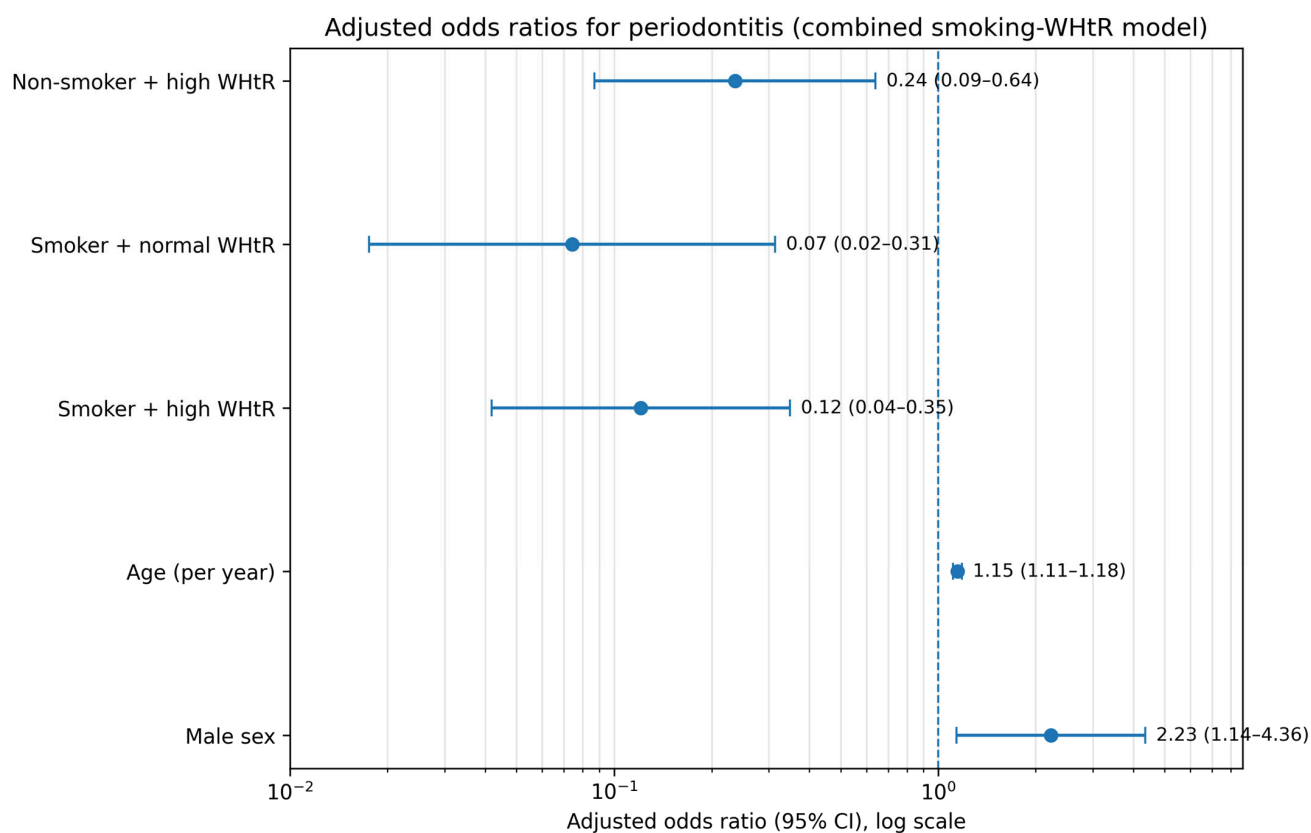


Figure 3. Forest plot of adjusted odds ratios for periodontitis from the combined smoking-WHtR exposure model. The model was adjusted for age, sex, education level, and brushing frequency. Brushing frequency was modeled as 0×/day, 1×/day, and ≥2×/day. Odds ratios are presented with 95% confidence intervals on a logarithmic scale.

Across the adjusted models, age was the strongest factor associated with periodontitis. Each additional year of age was associated with higher odds of periodontitis (adjusted OR = 1.15; 95% CI: 1.11–1.18; $p < 0.001$). Male sex was also associated with higher odds of periodontitis in the adjusted combined-exposure model (adjusted OR = 2.23; 95% CI: 1.14–4.36; $p = 0.019$).

In the model examining smoking and WHtR, the interaction term was statistically significant (adjusted OR = 6.89; 95% CI: 1.44–33.05; $p = 0.016$), suggesting possible effect modification. However, this finding should be interpreted cautiously because of the cross-sectional design, age imbalance across exposure groups, and the small number of periodontitis cases in some combined categories.

The adjusted odds ratios (ORs) for the combined exposure groups were lower than 1 when compared with the reference group (non-smoker with normal WHtR). This pattern differs from the unadjusted prevalence values and indicates the extent to which age and other covariates influenced the observed association. For this reason, the present dataset does not support the assertion that smoking combined with high WHtR increased the odds of periodontitis in this sample.

Figure 4 presents box-and-whisker plots for age, PPD, and CAL according to periodontitis status. Participants with periodontitis showed higher median values for age, PPD, and CAL than those without periodontitis. The plots provide a visual summary of data variability and support the descriptive findings reported in Table 1.

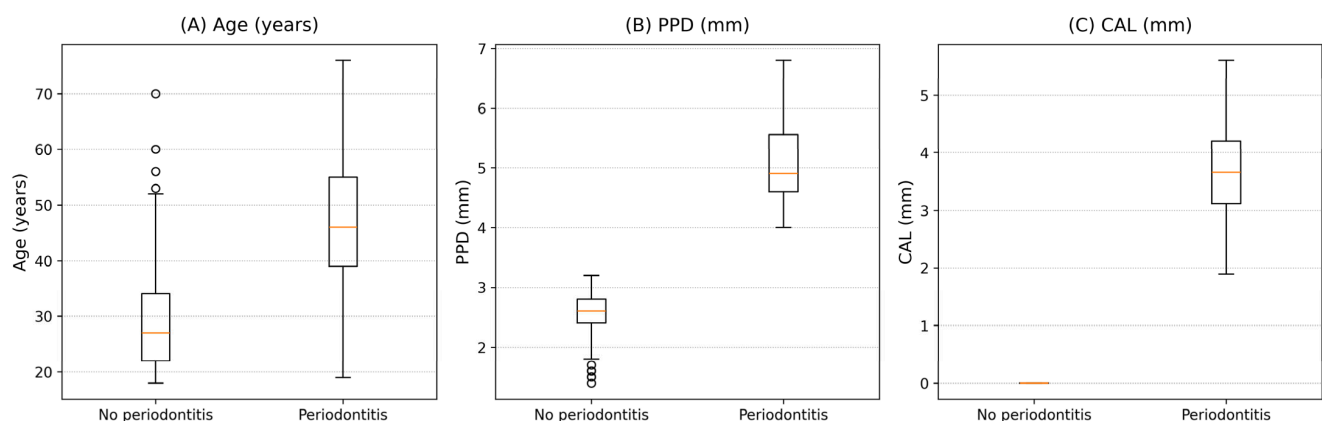


Figure 4. Box-and-whisker plots showing the distribution of (A) age, (B) probing pocket depth (PPD), and (C) clinical attachment loss (CAL) according to periodontitis status.

4. Discussion

The objective of this study was to evaluate how smoking and obesity-related indicators are associated with the presence of periodontitis among Iraqi dental patients. The findings indicate that this relationship was strongly influenced by age. Age was the dominant factor associated with periodontitis in this dataset and shaped the interpretation of the observed smoking- and obesity-related associations. The strong association between increasing age and periodontitis is clinically plausible, as periodontitis reflects cumulative periodontal tissue destruction over time. The 15% increase in the odds of periodontitis for each additional year of age is consistent with global epidemiological evidence identifying age as a major non-modifiable factor associated with periodontitis [27]. Periodontal status in the present dataset was based on full-mouth periodontal examination, including PPD, CAL, BOP, and plaque index, with diagnosis categorized according to the 2018 classification framework.

Perhaps the most difficult finding to interpret was the appearance of an apparently protective association between smoking and periodontitis in the fully adjusted regression models. This finding contrasts with the large body of evidence showing that smoking is a

major risk factor for periodontitis [7–9,28]. It is therefore more likely to reflect a statistical artifact than a biological protective effect. In this sample, smoking was more prevalent among younger participants, who had a lower prevalence of accumulated periodontal destruction. Although age was included in the adjusted models, the strong imbalance between age and smoking status appears to have reversed the expected direction of the association. This pattern is compatible with the logic of Simpson's paradox, where aggregated or imbalanced data can lead to misleading associations. The present study should not be interpreted as showing that smoking is protective. Rather, the observed smoking-periodontitis association was strongly influenced by the age distribution of this particular dental patient sample.

A statistically significant interaction was observed between smoking and central obesity measured by WHtR. This finding suggests possible effect modification between smoking status and central obesity in their association with periodontitis. However, it should be interpreted with caution, as a statistical interaction does not necessarily indicate biological synergy, particularly in a cross-sectional dataset with a marked age imbalance between exposure groups. Smoking may contribute to a local inflammatory microenvironment and impaired host defense, while visceral adiposity may contribute to low-grade systemic inflammation through adipokine release. These mechanisms may help explain why smoking and central obesity are clinically relevant in periodontal assessment, but the present cross-sectional data do not establish a combined causal effect.

The observation that WHtR was informative in the interaction model is consistent with the view that fat distribution may be metabolically relevant in periodontal research. Central obesity is one component of metabolic syndrome and is linked to systemic inflammatory and cardiometabolic risk factors [29,30].

Another point that should be considered is that smoking status may also be related to changes in appetite, eating behavior, and body weight, particularly among individuals who quit smoking. Post-cessation weight gain has been reported in the literature [31,32] and may influence BMI, WHtR, and other obesity-related indicators. In the present study, however, data on former smoking status, time since smoking cessation, dietary intake, and weight change were not available. Therefore, the possible contribution of smoking cessation-related behavioral changes could not be evaluated. The present findings suggest that WHtR may be a useful central adiposity indicator in periodontal research; however, its independent role in periodontitis should be interpreted cautiously and confirmed in longitudinal studies. These findings are similar to research that has demonstrated a direct association between WHtR and the presence of periodontal pockets, even in non-smoking populations [22,23].

4.1. Clinical Implications

The present findings have some clinical implications, although they should be interpreted cautiously. Age was the dominant factor associated with periodontitis in this sample, reinforcing the importance of regular periodontal screening and maintenance among older adults. From a clinical perspective, smoking status and central obesity may still be useful components of a broader periodontal risk assessment. However, the present findings do not directly support recommending more intensive periodontal follow-up based only on the combination of smoking and high WHtR. Maintenance planning should instead be based on the overall clinical profile, including age, periodontal diagnosis, disease severity, plaque control, systemic health, smoking history, and patient-specific risk factors. WHtR may still be a simple and non-invasive measure that can help clinicians discuss general metabolic health with patients, but its periodontal relevance should be confirmed in future longitudinal studies.

4.2. Limitations

This study has several limitations that should be considered when interpreting the findings. First, the cross-sectional design does not allow causal relationships or temporal sequence to be established between smoking, central obesity, and periodontitis. The results should therefore be interpreted as associations rather than evidence of risk or causation. This limitation is particularly important because age was strongly associated with periodontitis and was not equally distributed across the smoking and WHtR groups, which may have influenced the adjusted estimates and the interpretation of the interaction term.

Second, the study was based on a convenience sample of patients attending a university periodontal clinic. Therefore, the findings may not be generalizable to the wider Iraqi adult population. The sample size was based on the available eligible records and was not determined by an a priori sample size calculation. This is especially relevant for the interaction analysis, since interaction terms and combined exposure groups usually require larger and more balanced samples.

Third, several potentially important confounding variables were not available in the dataset, including diabetes status, detailed socioeconomic status, dietary intake, medication use, access to dental care, and other systemic health factors. Smoking status was also self-reported and analyzed only as current smoker or non-smoker. Information on former smoking status, time since smoking cessation, smoking duration, pack-years, smoking intensity, type of tobacco product, and smoking cessation-related weight change was not available. These missing details may have limited the ability to evaluate the true relationship between smoking, obesity-related indicators, and periodontal status.

Fourth, periodontitis was analyzed as a binary outcome. Although this approach was necessary for the logistic regression models used in the present analysis, it simplifies a complex and heterogeneous disease. Complete information on periodontitis stage, grade, extent, and localization was not consistently available for all participants; therefore, these variables could not be incorporated into the models. In addition, the no-periodontitis group combined periodontally healthy participants and participants with gingivitis, which may have introduced clinical heterogeneity and classification imprecision and the present analysis did not model individual periodontal clinical parameters, such as PPD, CAL, BOP, or plaque index, as separate continuous outcomes in relation to WHtR.

Fifth, examiner calibration data were not available in the secondary dataset. Since periodontal measurements such as probing pocket depth, clinical attachment loss, and bleeding on probing may be influenced by examiner variability, this should be considered when interpreting the clinical findings. Finally, although plaque index was recorded and described as a clinical parameter, it was not included in the primary adjusted models. This is another relevant limitation, as plaque accumulation is a central local etiological factor in periodontitis and may also confound or mediate the relationship between behavioral factors and periodontal disease.

5. Conclusions

In this cross-sectional sample of Iraqi dental patients, age was the main factor associated with periodontitis. Although smoking and central obesity are recognized as clinically relevant factors in periodontal and systemic health, their associations with periodontitis in the present study were strongly influenced by the age structure of the sample. A statistically significant interaction was observed between smoking status and WHtR; however, this finding should be interpreted cautiously and should not be considered evidence of biological synergy or causality.

The findings suggest that smoking status and central obesity may form part of a broader metabolic-behavioral profile relevant to periodontal assessment, but they do not

establish an independent combined-risk effect in this dataset. Future longitudinal studies with more detailed data on smoking history, diet, metabolic variables, and complete periodontal staging and grading are needed to clarify the relationships among smoking, central obesity, and periodontitis progression.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/obesities6040044/s1>, Table S1: Full multivariable logistic regression outputs for Models 1–3. The table reports adjusted ORs, 95% CIs, and *p*-values for all exposure variables, interaction terms, and adjustment covariates.

Author Contributions: Conceptualization, M.S.M.A. and H.M.A.; methodology, M.S.M.A., O.H.A. and H.M.A.; software, O.H.A.; validation, M.S.M.A., O.H.A. and H.M.A.; formal analysis, O.H.A.; investigation, M.S.M.A.; resources, M.S.M.A. and H.M.A.; data curation, M.S.M.A. and O.H.A.; writing—original draft preparation, M.S.M.A.; writing—review and editing, O.H.A. and H.M.A.; visualization, O.H.A.; supervision, H.M.A.; project administration, H.M.A. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the College of Dentistry, University of Baghdad, Iraq (Ref. number: 1249, approved on 7 January 2025).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The raw data supporting the conclusions of this article will be made available by the authors on request.

Acknowledgments: During the preparation of this study, the authors used Paperpal to paraphrase and improve readability. The authors have reviewed and edited the output and take full responsibility for the content of this publication.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

BMI	Body Mass Index
BOP	Bleeding on Probing
CAL	Clinical Attachment Loss
CEJ	Cemento-Enamel Junction
CI	Confidence Interval
OR	Odds Ratio
PI	Plaque Index
PPD	Probing Pocket Depth
SD	Standard Deviation
WHO	World Health Organization
WHtR	Waist-to-Height Ratio

References

1. Tonetti, M.S.; Jepsen, S.; Jin, L.; Otomo-Corgel, J. Impact of the global burden of periodontal diseases on health, nutrition and wellbeing of mankind: A call for global action. *J. Clin. Periodontol.* **2017**, *44*, 456–462. [[CrossRef](#)] [[PubMed](#)]
2. Kassebaum, N.; Bernabé, E.; Dahiya, M.; Bhandari, B.; Murray, C.; Marcenes, W. Global burden of severe periodontitis in 1990–2010: A systematic review and meta-regression. *J. Dent. Res.* **2014**, *93*, 1045–1053. [[CrossRef](#)] [[PubMed](#)]
3. Fadhil, R.; Akram, H.M.; Najah, A.; Gul, S.S. Association of matrix metalloproteinase-1-1607 1G/2G single nucleotide polymorphism genotypes with periodontitis in Iraqi population. *Braz. Dent. Sci.* **2022**, *25*, e3283. [[CrossRef](#)]

4. Chatzopoulos, G.S.; Jiang, Z.; Marka, N.; Wolff, L.F. Association between periodontitis extent, severity, and progression rate with systemic diseases and smoking: A retrospective study. *J. Pers. Med.* **2023**, *13*, 814. [\[CrossRef\]](#) [\[PubMed\]](#)
5. Lamont, R.J.; Koo, H.; Hajishengallis, G. The oral microbiota: Dynamic communities and host interactions. *Nat. Rev. Microbiol.* **2018**, *16*, 745–759. [\[CrossRef\]](#) [\[PubMed\]](#)
6. Darby, I. Risk factors for periodontitis & peri-implantitis. *Periodontology 2000* **2022**, *90*, 9–12. [\[CrossRef\]](#)
7. Leite, F.R.; Nascimento, G.G.; Scheutz, F.; Lopez, R. Effect of smoking on periodontitis: A systematic review and meta-regression. *Am. J. Prev. Med.* **2018**, *54*, 831–841. [\[CrossRef\]](#) [\[PubMed\]](#)
8. Tonetti, M.S. Cigarette smoking and periodontal diseases: Etiology and management of disease. *Ann. Periodontol.* **1998**, *3*, 88–101. [\[CrossRef\]](#) [\[PubMed\]](#)
9. Zhang, Y.; He, J.; He, B.; Huang, R.; Li, M. Effect of tobacco on periodontal disease and oral cancer. *Tob. Induc. Dis.* **2019**, *17*, 40. [\[CrossRef\]](#) [\[PubMed\]](#)
10. Alwithanani, N. Periodontal Disease and Smoking: Systematic review. *J. Pharm. Bioallied Sci.* **2023**, *15*, S64–S71. [\[CrossRef\]](#) [\[PubMed\]](#)
11. Jiang, Y.; Zhou, X.; Cheng, L.; Li, M. The impact of smoking on subgingival microflora: From periodontal health to disease. *Front. Microbiol.* **2020**, *11*, 66. [\[CrossRef\]](#) [\[PubMed\]](#)
12. Silva, H. Tobacco use and periodontal disease—The role of microvascular dysfunction. *Biology* **2021**, *10*, 441. [\[CrossRef\]](#) [\[PubMed\]](#)
13. Gautam, D.; Jindal, V.; Gupta, S.; Tuli, A.; Kotwal, B.; Thakur, R. Effect of cigarette smoking on the periodontal health status: A comparative, cross sectional study. *J. Indian Soc. Periodontol.* **2011**, *15*, 383–387. [\[CrossRef\]](#) [\[PubMed\]](#)
14. Kim, C.M.; Lee, S.; Hwang, W.; Son, E.; Kim, T.W.; Kim, K.; Kim, Y.H. Obesity and periodontitis: A systematic review and updated meta-analysis. *Front. Endocrinol.* **2022**, *13*, 999455. [\[CrossRef\]](#)
15. Moura-Grec, P.G.d.; Marsicano, J.A.; Carvalho, C.A.P.d.; Sales-Peres, S.H.d.C. Obesity and periodontitis: Systematic review and meta-analysis. *Cienc. Saude Coletiva* **2014**, *19*, 1763–1772. [\[CrossRef\]](#)
16. Reytor-González, C.; Parise-Vasco, J.M.; González, N.; Simancas-Racines, A.; Zambrano-Villacres, R.; Zambrano, A.K.; Simancas-Racines, D. Obesity and periodontitis: A comprehensive review of their interconnected pathophysiology and clinical implications. *Front. Nutr.* **2024**, *11*, 1440216. [\[CrossRef\]](#) [\[PubMed\]](#)
17. Suvan, J.; Petrie, A.; Moles, D.; Nibali, L.; Patel, K.; Darbar, U.; Donos, N.; Tonetti, M.; D’Aiuto, F. Body mass index as a predictive factor of periodontal therapy outcomes. *J. Dent. Res.* **2014**, *93*, 49–54. [\[PubMed\]](#)
18. Abu-Shawish, G.; Betsy, J.; Anil, S. Is obesity a risk factor for periodontal disease in adults? A systematic review. *Int. J. Environ. Res. Public Health* **2022**, *19*, 12684. [\[CrossRef\]](#) [\[PubMed\]](#)
19. Checa-Ros, A.; Hsueh, W.-C.; Merck, B.; González-Torres, H.; Bermúdez, V.; D’Marco, L. Obesity and oral health: The link between adipokines and periodontitis. *touchREVIEWS Endocrinol.* **2024**, *20*, 25. [\[CrossRef\]](#)
20. Clemente-Suárez, V.J.; Redondo-Flórez, L.; Beltrán-Velasco, A.I.; Martín-Rodríguez, A.; Martínez-Guardado, I.; Navarro-Jiménez, E.; Laborde-Cárdenas, C.C.; Tornero-Aguilera, J.F. The role of adipokines in health and disease. *Biomedicines* **2023**, *11*, 1290. [\[CrossRef\]](#) [\[PubMed\]](#)
21. Kangas, S.; Timonen, P.; Knuuttila, M.; Julia, A.; Ylöstalo, P.; Syrjälä, A.-M.H. Waist circumference and waist-to-height ratio are associated with periodontal pocketing—Results of the Health 2000 Survey. *BMC Oral Health* **2017**, *17*, 48. [\[CrossRef\]](#) [\[PubMed\]](#)
22. Chen, X.; Zeng, C.; Chen, X.; Sun, J.; Jin, F.; Chen, Z.; Song, J. Relationship between three body obesity indicators, WWI, BMI, WtHR, and periodontitis. *Sci. Rep.* **2025**, *15*, 697. [\[CrossRef\]](#) [\[PubMed\]](#)
23. Xu, L.; Liu, Y.; Zhao, C.; Wang, J.; Zhou, H. Association between weight-adjusted waist index and periodontitis: A population-based study. *PLoS ONE* **2024**, *19*, e0305010. [\[CrossRef\]](#) [\[PubMed\]](#)
24. Huang, Y.Q.; Xu, J.N.; Huang, Y.; Xu, Y.D.; Wang, H.L.; Shi, W.T.; Wang, J.; Wang, H. Independent and combined effects of smoking, drinking and depression on periodontal disease. *BMC Oral Health* **2024**, *24*, 535. [\[CrossRef\]](#) [\[PubMed\]](#)
25. Ganesan, S.M.; Joshi, V.; Fellows, M.; Dabdoub, S.M.; Nagaraja, H.N.; O’Donnell, B.; Deshpande, N.R.; Kumar, P.S. A tale of two risks: Smoking, diabetes and the subgingival microbiome. *ISME J.* **2017**, *11*, 2075–2089. [\[CrossRef\]](#) [\[PubMed\]](#)
26. Tonetti, M.S.; Greenwell, H.; Kornman, K.S. Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. *J. Periodontol.* **2018**, *89*, S159–S172. [\[CrossRef\]](#) [\[PubMed\]](#)
27. Nazir, M.; Al-Ansari, A.; Al-Khalifa, K.; Alhareky, M.; Gaffar, B.; Almas, K. Global prevalence of periodontal disease and lack of its surveillance. *Sci. World J.* **2020**, *2020*, 2146160. [\[CrossRef\]](#)
28. Albuslutan, A.A.; Saliem, S.S.; Shibli, J. Impact of oral hygiene instructions on cytokines in smokers and vapers with gingivitis. *J. Baghdad Coll. Dent.* **2025**, *37*, 78–89. [\[CrossRef\]](#)
29. Una, E.-S.; Menaka, S. Actions of Adjunctive Nutritional Antioxidants in Periodontitis and Prevalent Systemic Inflammatory Diseases. *Endocr., Metab. Immune Disord.-Drug Targets* **2015**, *15*, 261–276. [\[CrossRef\]](#)
30. Koo, H.S.; Hong, S.M. Prevalence and risk factors for periodontitis among patients with metabolic syndrome. *Metab. Syndr. Relat. Disord.* **2018**, *16*, 375–381. [\[CrossRef\]](#) [\[PubMed\]](#)

31. Tian, J.; Venn, A.; Otahal, P.; Gall, S. The association between quitting smoking and weight gain: A systemic review and meta-analysis of prospective cohort studies. *Obes. Rev.* **2015**, *16*, 883–901. [[CrossRef](#)] [[PubMed](#)]
32. Wang, X.; Qin, L.-Q.; Arafa, A.; Eshak, E.S.; Hu, Y.; Dong, J.-Y. Smoking cessation, weight gain, cardiovascular risk, and all-cause mortality: A meta-analysis. *Nicotine Tob. Res.* **2021**, *23*, 1987–1994. [[CrossRef](#)] [[PubMed](#)]

Disclaimer/Publisher’s Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.