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**Research Article**

# Effect of a School-Delivered "Tobacco-Free Home" Campaign on Adolescent Smoking and Periodontal Risk: An Analysis of Psychosocial Pathways

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## KEYWORDS

Adolescent, Health Behavior, Periodontal Disease, Smoking Cessation, Social Support

## ABSTRACT

**Introduction:** Adolescent smoking contributes to early-onset periodontal disease, while institutional prevention programs often lack reinforcement at home. This study evaluated an 8-week psychosocial intervention integrating school-based Cognitive Behavioral Therapy (CBT) with a family-engaged Tobacco-Free Home (TFH) campaign to improve smoking behavior, risk attitudes, and social support.
**Materials and Methods:** A quasi-experimental non-equivalent control group design involved 259 adolescent smokers in Bali, Indonesia. Intact classrooms were clustered into intervention (n = 139) and control (n = 120) groups. Psychosocial variables were measured at baseline and week 8. Data were analyzed using t-tests, Pearson correlation, and ANCOVA adjusted for baseline covariates.
**Result and Discussion:** The intervention group showed significantly greater improvements in all outcomes compared with controls. Smoking behavior improved significantly ($\Delta = 0.76$, $p < 0.001$), while ANCOVA confirmed strong group effects ($F = 62.14$). Positive correlations ($r > 0.49$) indicated that smoke-free home norms reinforced cognitive self-regulation, supporting social support as a key mediator.
**Conclusion:** The CBT–TFH model effectively modified determinants of adolescent smoking behavior, with social support as an important catalyst for change. By extending intervention effects into the home environment, this strategy may help reduce long-term periodontal disease risk.

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## INTRODUCTION

The global tobacco epidemic remains a major cause of preventable morbidity and mortality, accounting for more than 8 million deaths annually.[1] In Indonesia, adolescent smoking continues to pose a significant public health challenge, with initiation frequently occurring in early adolescence and strongly influenced by the stages of behavioral change.[2] At this stage, susceptibility to social influence is heightened.[3] Beyond systemic effects, tobacco exposure contributes to early-onset periodontal disease and bruxism through nicotine-induced vasoconstriction that disrupts gingival microcirculation and suppresses local immune response.[4–6] However, awareness of tobacco-related oral manifestations remains limited among adolescents, reflecting a persistent gap between disease burden and health literacy, which can be improved through targeted educational methods.[7–9]

Current interventions largely rely on school-based education to improve knowledge and risk perception.[10] Although effective in increasing awareness, such approaches are insufficient to sustain long-term behavioral change. Adolescent smoking behavior is shaped by cognitive factors, perceived social norms, and environmental reinforcement.[11] Existing dental public health strategies remain institution-focused with limited integration of domestic environments, undermining the sustainability of school-based behaviors. Furthermore, key psychosocial drivers of sustained change remain insufficiently understood, highlighting a critical gap in integrated interventions addressing social and environmental determinants of adolescent smoking.[12–14]

To address these gaps, this study applies the Theory of Planned Behavior, which conceptualizes behavioral intention through attitudes, subjective norms, and perceived behavioral control.[11] We propose an integrated dual-setting intervention combining school-based Cognitive Behavioral Therapy with family engagement through a Tobacco-Free Home contract. This approach extends intervention into the adolescent's primary social environment while introducing a mechanistic framework that positions social support as a central mediator of sustained behavioral change, particularly within collectivist contexts.[15]

The clinical relevance of this approach lies in its potential to influence biological pathways of oral disease. Tobacco-induced dysbiosis promotes colonization by periodontopathogens such as *Porphyromonas gingivalis* and *Fusobacterium nucleatum*, leading to increased Interleukin-6 production and periodontal destruction.[16] By modifying smoking behavior through integrated psychosocial mechanisms, this intervention aims to interrupt these pathogenic processes at an early stage. Therefore, this study evaluates the effectiveness of the integrated CBT–TFH model on smoking determinants and explores social support as a pivotal catalyst for mitigating long-term periodontal risk in youth populations.

## MATERIAL AND METHODS

Adhering to standard reporting guidelines for nonrandomized evaluations,[17] this study employed a quasi-experimental non-equivalent control group pre-test–post-test design. The study was conducted at SMAN 1 Ubud, Bali, Indonesia, from May to December 2025. Cluster-level allocation was implemented by assigning intact classrooms into an intervention group (n=139) and a control group (n=120), minimizing cross-

contamination within the shared school environment.[18]

Participants were recruited using purposive sampling from a baseline population of 1,052 students. A total of 259 adolescent smokers were included based on self-reported smoking within the past 30 days and enrollment in grades X–XII. Students participating in other cessation programs or with cognitive limitations affecting questionnaire completion were excluded. Although purposive sampling may limit generalizability, it enabled targeted recruitment of a high-risk population aligned with the study objectives. Baseline equivalence across psychosocial variables was assessed and subsequently controlled in the analysis. Sample size was determined a priori to achieve adequate statistical power ($\beta = 0.80$) for detecting moderate-to-large effects, with an additional 10% oversampling to account for potential attrition. The calculation incorporated the design effect associated with cluster sampling to ensure sufficient power despite intra-cluster correlation.

The 8-week dual-setting psychosocial intervention integrated school-based Cognitive Behavioral Therapy (CBT) with a family-engaged Tobacco-Free Home (TFH) campaign. The CBT component included cognitive restructuring, behavioral rehearsal, and self-monitoring, while the TFH component involved active family participation culminating in a negotiated smoke-free home contract to modify domestic social norms.[12] The intervention was delivered by calibrated dental public health professionals and school counselors. Intervention fidelity was monitored using structured observation checklists to ensure adherence to the theoretical framework.[19] The control group received a standardized, non-interactive health education curriculum.

Study outcomes included smoking behavior (frequency and self-regulation), attitudes toward smoking hazards (perceived severity and susceptibility), and perceived social support, assessed at baseline and week 8. Measurement instruments were adapted from established Theory of Planned Behavior constructs[11] and demonstrated strong psychometric properties, with high internal consistency (Cronbach's $\alpha > 0.85$) across Likert-scale domains. To reduce self-report and social desirability bias, questionnaires were culturally adapted and pre-tested for clarity and relevance within the target population. Although participant blinding was not feasible, outcome assessors and data analysts were blinded to group allocation to minimize detection bias.

Data analysis was conducted using IBM SPSS Statistics version 31. Missing data were handled using listwise deletion due to the low attrition rate (<5%), minimizing the risk of bias. Analysis of Covariance (ANCOVA) was applied as the primary inferential method, with adjustment for baseline covariates and clustering effects at the classroom level to strengthen causal inference.[20] Results were reported as mean differences ($\Delta$) and effect sizes (Cohen's d and $\eta^2$), with statistical significance set at $p < 0.05$. Ethical approval was obtained from the Universitas Mahasaraswati Denpasar Ethics Committee (No. 03.0050/KEP-Unmas/V/2025), and informed consent was secured from all participants with parental and institutional permission.[21]

## RESULTS AND DISCUSSIONS

Table 1. Baseline Demographic and Psychosocial Equivalence of Participants

| Variables | Intervention (n = 139) | Control (n = 120) | p-value | SMD |
|---|---|---|---|---|
| Demographic characteristics | | | | |
| Sex, n (%) | | | 0.82[a] | 0.03 |
| Male | 133 (95.7) | 114 (95.0) | | |
| Female | 6 (4.3) | 6 (5.0) | | |
| Grade level, n (%) | | | 0.92[a] | 0.02 |
| Grade X | 30 (21.6) | 24 (20.0) | | |
| Grade XI | 43 (30.9) | 38 (31.7) | | |
| Grade XII | 66 (47.5) | 58 (48.3) | | |
| Baseline measures (mean ± SD) | | | | |
| Smoking Behaviour | 3.21 ± 0.63 | 3.10 ± 0.61 | 0.16[b] | 0.18 |
| Attitudes (Hazards) | 3.14 ± 0.49 | 3.08 ± 0.49 | 0.33[b] | 0.12 |
| Social Support | 3.12 ± 0.56 | 3.14 ± 0.58 | 0.79[b] | 0.03 |

Notes:
1. [a] Chi-square test.
2. [b] Independent samples t-test.
3. Data are presented as mean ± SD or n (%).
4. Baseline refers to pre-test values.
5. No statistically significant differences were observed between groups ($p > 0.05$).
6. Grade level was used as a proxy for age due to the relatively homogeneous age distribution of participants.
7. SMD: standardized mean difference; values < 0.1 indicate small

Table 2. ANCOVA and Mean Differences for Post-Test Psychosocial Outcomes

| Variable / Analysis | Δ / MD (95% CI) | F | Effect Size (d / $\eta^2$) | p-value |
|---|---|---|---|---|
| Smoking Behavior | | | | |
| Intervention[3] | 0.76 (0.71–0.81) | | | <0.001 |
| Control[3] | 0.31 (0.27–0.35) | | | <0.001 |
| Between-group[4] | 0.56 (0.42–0.70) | | d = 0.98 | <0.001 |
| ANCOVA (Group effect) | | 62.14 | $\eta^2$ = 0.195 | <0.001 |
| Attitudes (Hazards) | | | | |
| Intervention[3] | 0.75 (0.71–0.79) | | | <0.001 |
| Control[3] | 0.31 (0.28–0.34) | | | <0.001 |
| Between-group[4] | 0.50 (0.39–0.61) | | d = 1.08 | <0.001 |
| ANCOVA (Group effect) | | 71.45 | $\eta^2$ = 0.217 | <0.001 |
| Social Support | | | | |
| Intervention[3] | 0.77 (0.73–0.81) | | | <0.001 |
| Control[3] | 0.31 (0.27–0.35) | | | <0.001 |
| Between-group[4] | 0.44 (0.30–0.58) | | d = 0.79 | <0.001 |
| ANCOVA (Group effect) | | 49.22 | $\eta^2$ = 0.162 | <0.001 |

Notes:
1. Δ = mean change (post–pre).
2. MD = between-group mean difference.
3. [3] Within-group comparisons were performed using paired t-tests.
4. [4] Between-group comparisons were conducted using independent samples t-tests.
5. ANCOVA results represent post-test outcomes adjusted for baseline (pre-test) values.
6. Effect sizes are reported as Cohen's d for between-group differences and partial eta squared ($\eta^2$) for ANCOVA.
7. All tests were two-tailed with a significance level of $p < 0.05$.
8. Higher scores indicate more favorable outcomes.

Statistical analysis of baseline parameters (Table 1) indicated that the intervention and control groups were comparable across all demographic characteristics and primary outcome variables ($p > 0.05$). The observed Standardized Mean Difference (SMD) values ranged from 0.02 to 0.18, reflecting negligible to small differences between groups at baseline and supporting the assumption of initial comparability. This baseline similarity reduces the likelihood that subsequent post-intervention differences are attributable to pre-existing group disparities, thereby strengthening the internal validity of the study. Such findings are consistent with quasi-experimental approaches in educational

research, where careful group allocation and baseline assessment are essential to minimize selection bias and support causal inference.[22]

Furthermore, previous structural equation modeling studies and pediatric awareness research have demonstrated that behavioral and knowledge distributions within school-based populations tend to be relatively homogeneous, reinforcing the suitability of the present quasi-experimental design.[10,23] Collectively, these results provide a robust foundation for evaluating intervention effects and are aligned with the theoretical assumptions of the theory of planned behavior.[24]

The magnitude and consistency of the intervention effects observed across psychosocial outcomes (table 2) indicate that the integrated approach substantially enhances behavior-related determinants beyond what is typically achieved through single-setting interventions. The large effect sizes across variables ($d = 0.79–1.08$; $\eta^2 = 0.162–0.217$) demonstrate strong practical relevance and reinforce the importance of addressing both cognitive and environmental dimensions of adolescent smoking behavior. This is consistent with evidence that adolescent smoking is shaped by interacting individual, social, and contextual determinants, including peer influence and relational dynamics.[3,14,15]

Consistent with the Theory of Planned Behavior, which highlights the roles of subjective norms and perceived behavioral control,[11,24,25] the observed effects also reflect the importance of social context in shaping behavioral intentions. The magnitude of effects aligns with findings from multicomponent and school-based interventions demonstrating the effectiveness of integrated behavioral strategies.[26,27] In contrast, studies examining social and behavioral determinants suggest that insufficient environmental and social reinforcement may limit behavioral outcomes,[15] whereas systematic reviews emphasize that parental engagement and home-based reinforcement are critical for sustaining behavioral change.[10,28]

Mechanistically, the findings suggest that the intervention operates through a synergistic pathway in which cognitive restructuring is reinforced by consistent social norms within the home environment, in line with evidence on social norm transformation in tobacco-related behaviors.[19] The integration of family-based reinforcement may enhance the durability of cognitive changes by embedding them within the adolescent's immediate social context, supported by studies linking social support and household environments to behavioral outcomes.[12,15,29,30] Furthermore, the strong effect observed for attitudes ($\eta^2 = 0.217$) suggests that parental modeling may contribute to attenuating pro-smoking influences and facilitating risk-averse behavioral trajectories, consistent with evidence on family-based and parental influence mechanisms.[27,28,31]

Table 3. Pearson Correlation Matrix Among Post-Test Variables

| Variable | Smoking Behavior | Attitude toward Smoking Hazards | Social Support |
|---|---|---|---|
| Smoking Behavior | 1.00 | | |
| Attitude toward Smoking Hazards | 0.52[1] | 1.00 | |
| Social Support | 0.58[1] | 0.49[1] | 1.00 |

Notes:

1. Pearson correlation coefficients (r) are reported.
2. [1]All correlations were statistically significant ($p < 0.001$).
3. Correlations were calculated based on post-test scores.
4. Higher scores indicate more favorable outcomes.

Statistical modeling (Table 3) demonstrated moderate-to-strong positive correlations among all psychosocial variables ($p < 0.001$), with smoking behavior most strongly associated with social support ($r = 0.58$), followed by attitudes toward smoking hazards ($r = 0.52$). Social support was also moderately correlated with attitudes ($r = 0.49$), indicating a coherent psychosocial structure in which behavioral outcomes are linked to both cognitive perceptions and social context. These findings support the view that adolescent smoking-related behaviors are shaped by interacting individual and social determinants, including peer relationships and perceived support systems.[3,14,15]

These interrelationships are consistent with evidence showing that parental motivation and behaviors influence adolescent health outcomes and behavioral intentions,[31] while social support contributes to psychological resilience and adaptive behavioral regulation.[29] In addition, environmental factors such as smoke-free home environments have been shown to influence smoking-related attitudes and reduce tobacco exposure.[13] Collectively, these findings suggest that social support functions as a key contextual factor interacting with cognitive processes to influence smoking-related behaviors, although the observed associations should be interpreted as non-causal given the correlational design.

**Clinical Implications for Dentistry**

Tobacco use disrupts the oral microbiome and promotes dysbiosis characterized by increased periodontal pathogens such as *Porphyromonas gingivalis* and *Fusobacterium nucleatum*,[16] while nicotine-induced vasoconstriction may mask early inflammatory signs such as gingival bleeding, delaying clinical detection.[4] Within this biological context, the CBT–Tobacco-Free Home (CBT-TFH) model represents a clinically relevant strategy by targeting both cognitive and environmental determinants of smoking behavior during adolescence. By intervening at this critical developmental stage, CBT-TFH may help mitigate early pathogenic processes and enhance the effectiveness of preventive and non-surgical dental care. This integrative approach is supported by evidence indicating that reducing tobacco exposure lowers the risk of periodontal destruction and other smoking-related oral complications,[32] positioning CBT-TFH as a promising adjunct in preventive dentistry.

**Methodological Limitations**

While this study provides robust evidence, several limitations should be noted. The quasi-experimental design using intact classroom clusters may introduce cluster-level dependency; although ANCOVA was applied, future studies should incorporate multilevel modeling to account for hierarchical structures and intraclass correlation. [17,33]

Self-reported measures may also be subject to social desirability bias, warranting the inclusion of objective biomarkers such as salivary cotinine. In addition, the short intervention period (8 weeks) limits assessment of long-term sustainability, highlighting the need for longitudinal follow-up of home-based smoke-free norms.[34] Nevertheless, the consistently large effect sizes support the dual-setting CBT–Tobacco-Free Home (CBT-TFH) model as a promising and scalable strategy for preventive interventions in adolescent oral health.

## CONCLUSION

The integrated CBT–Tobacco-Free Home (CBT–TFH) model effectively modifies key determinants of adolescent smoking, with social support emerging as a central driver of behavioral

change. By targeting both cognitive and environmental factors, this approach may disrupt habitual tobacco use and help prevent downstream biological consequences, including periodontal destruction and oral dysbiosis. From a clinical perspective, CBT–TFH offers a practical upstream strategy for dentistry to reduce long-term oral health risks in vulnerable youth populations. By extending intervention beyond school settings into the home environment, this model provides a meaningful and scalable contribution to preventive oral health and public health practice.

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