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Mechanisms of Persistent Smoking in Occupational Lung Disease: A Qualitative Case Study

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Abstract

Background Smoking cessation remains challenging in chronic respiratory diseases, particularly among patients with occupational lung disease (OLD). However, the mechanisms sustaining smoking after diagnosis in occupationally exposed populations—where structural and sociocultural factors shape health behavior—remain poorly understood.

Methods We conducted a qualitative multiple case study using purposive sampling and semi-structured interviews with five male patients with OLD who continued smoking in Türkiye (April–December 2025). Data were analyzed using a reflexively informed, inductive case-based approach involving iterative coding and cross-case comparison. The analysis focused on identifying shared mechanisms rather than generating multiple discrete themes. Trustworthiness was supported through audit trail documentation, reflexivity, selective member checking, and Consolidated criteria for reporting qualitative research (COREQ)-guided reporting.

Results Two interrelated mechanisms explained persistent smoking. First, external causal attribution led participants to attribute disease primarily to occupational exposure rather than smoking, reducing perceived smoking-related risk. Second, structurally constrained behavioral persistence reflected the embedding of smoking within occupational stress, economic dependency, and normalized workplace practices. Across cases, these mechanisms interacted: altered illness representations reduced cessation motivation, while structural conditions reinforced continued smoking and relapse despite medical advice.

Conclusions Smoking persistence in patients with OLD is shaped by the interaction between illness attribution and structural constraints rather than individual choice alone. Interventions should move beyond individual-level counseling to incorporate attribution-focused communication and workplace-integrated cessation strategies. These findings provide a mechanism-based framework for addressing smoking in an underexplored occupational health context.

BACKGROUND

Occupational lung diseases (OLD) remain a substantial yet under-recognized contributor to global respiratory morbidity and mortality, disproportionately affecting low- and middle-income countries where regulatory enforcement and workplace protections are often limited (1). Conditions such as silicosis, occupational chronic obstructive pulmonary disease (COPD), and other dust-related lung diseases continue to impose a persistent and largely preventable disease burden (2–4). Chronic exposure to respirable crystalline silica and airborne particulates triggers sustained inflammatory responses, leading to progressive fibrosis and irreversible impairment of pulmonary function (5).

In parallel, cigarette smoking remains the most important modifiable risk factor for chronic respiratory disease worldwide (6). A growing body of evidence suggests that smoking and occupational exposures interact synergistically, resulting in a more-than-additive increase in the risk of COPD, lung cancer, and respiratory infections (7). Despite this well-established interaction, smoking cessation rates among individuals diagnosed with occupational lung disease remain persistently low (8), indicating a critical gap between biomedical knowledge and sustained behavioral change.

Traditional biomedical and individual-level behavioral models offer limited explanatory power for this phenomenon. While nicotine dependence plays a central role, smoking persistence is increasingly understood as embedded within broader social, economic, and occupational contexts, including precarious working conditions, financial dependency, and chronic psychosocial stress (9). In this context, smoking may function not only as an addictive behavior but also as a socially and structurally reinforced coping practice (11).

Importantly, emerging qualitative evidence from chronic respiratory disease populations, particularly among patients with COPD, demonstrates that continued smoking is shaped by complex processes of illness perception, identity, and meaning-making rather than addiction alone (12–14). Patients may normalize smoking, integrate it into their daily routines, and reinterpret illness in ways that sustain existing behaviors. However, these findings largely derive from general clinical populations and do not adequately capture the specific dynamics of occupational disease contexts.

In occupational settings, the attribution of disease causality is structurally and culturally distinct. Illness is frequently attributed to workplace exposures—such as dust, chemicals, or environmental hazards—rather than to individual behaviors like smoking. This externally oriented causal attribution may reconfigure risk perception, diffuse personal responsibility, and attenuate motivation for smoking cessation. Despite its potential importance, this mechanism has received limited empirical attention, particularly in middle-income countries where occupational exposures and smoking prevalence remain high and are embedded within specific sociocultural and labor contexts.

Accordingly, this study aims to examine the underlying mechanisms that sustain smoking behavior among patients diagnosed with occupational lung disease. Adopting a qualitative, case-based analytic approach, the study focuses on how illness attribution processes and structural conditions interact to shape persistent smoking. By moving beyond individual-level explanations and situating behavior within its sociocultural and occupational context, this research seeks to provide a more nuanced, mechanism-oriented understanding of smoking persistence and to inform the development of context-sensitive cessation strategies in occupational health settings.

METHODS

Study Design and Setting

This study employed a qualitative multiple case study design to examine the mechanisms underlying persistent smoking among patients diagnosed with OLD (15). A case-based approach was selected to enable in-depth exploration of individual experiences while allowing cross-case comparison of shared patterns.

The study was conducted at Ankara Ataturk Sanatoryum Research and Education Hospital in tertiary occupational health and pulmonary clinics in Türkiye between April 2025 and December 2025.

Participants and Sampling Strategy

A purposive sampling strategy was used to recruit information-rich participants who met the following inclusion criteria:

(1) confirmed diagnosis of occupational lung disease (e.g., silicosis, pneumoconiosis, occupational chronic obstructive pulmonary disease),

(2) current active smoking at the time of recruitment,

(3) age ≥ 18 years, and

(4) ability to provide informed consent.

Exclusion criteria included cognitive impairment, severe psychiatric illness, or inability to communicate in Turkish.

Sampling focused on workers from male-dominated occupational sectors in Türkiye, including construction, mining, and metal processing industries, where occupational exposure burden is high. All participants were male, reflecting the gender distribution in these sectors. The absence of female participants is acknowledged as a limitation and an important direction for future research.

Participants were recruited through occupational health clinics and patient registries. Written informed consent was obtained from all participants. Demographic and occupational characteristics—including age, occupation, exposure duration, smoking history (pack-years), and comorbidities—were collected as shown in Table 1.

Table 1
Participant Characteristics and Occupational Exposure Profile

Patient	Age	Sex	Occupation	Exposure Type	Exposure Duration (years)	Diagnosis	Smoking (pack-years)	Packs/day	Years Smoking	Years Since Diagnosis	Prior Quit Attempts
P1	57	Male	Marble worker	Silica dust	20	Silicosis	30	1	35	5	Yes (≥ 2)
P2	52	Male	Foundry worker	Metal dust/fumes	18	Occupational COPD	35	1.5	30	4	Yes (failed)
P3	38	Male	Sandblasting	High-intensity silica	10	Rapid silicosis	20	1	40	3	No
P4	50	Male	Construction	Mixed dust exposure	25	Fibrotic lung disease	28	1	25	6	Yes
P5	41	Male	Agricultural worker	Organic dust	15	Hypersensitivity-related lung disease	22	1	25	2	No

Note:: All participants had prolonged occupational exposure and sustained smoking behavior despite diagnosis, reflecting a high-risk, information-rich sample suitable for mechanism-oriented qualitative analysis.

Participant Flow

Fifteen patients were approached for participation. Six declined due to time constraints or lack of interest, and four were excluded due to ineligibility or clinical instability. Five participants completed the study. No withdrawals occurred after enrollment.

Sample Size Justification

Sample size was guided by the concept of information power, whereby adequacy is determined by the specificity of the research aim, sample relevance, and analytic depth rather than numerical thresholds. A sample of five participants was considered appropriate due to:

- a narrow and focused research aim,
- inclusion of information-rich cases with substantial occupational exposure histories, and
- a case-oriented analytic strategy prioritizing depth over breadth.

Rather than aiming for thematic saturation, the analysis focused on generating mechanism-based insights through detailed within-case and cross-case interpretation.

Data Collection

Data were collected through in-depth, semi-structured interviews using an interview guide developed specifically for this study (Supplementary Material S1). The interview guide was not previously published in any form and was designed based on existing literature and clinical expertise in occupational lung disease and smoking behavior research. The guide explored occupational exposure history, illness perceptions, smoking trajectories, cessation attempts, and structural and psychosocial influences.

Interviews were conducted in a private clinical setting, lasted 45–75 minutes, and were audio-recorded with participant consent. Field notes were used to capture contextual and non-verbal information. All interviews were conducted in Turkish and transcribed verbatim. Selected quotations were translated into English by a bilingual occupational health specialist and back-translated by an independent bilingual researcher to ensure semantic equivalence.The interview guide was developed based on prior literature and clinical expertise and explored:

- perceptions of disease causality and occupational exposure
- smoking behavior before and after diagnosis
- prior cessation attempts and barriers
- psychosocial, economic, and workplace influences

Field notes were recorded to capture contextual details, non-verbal cues, and preliminary analytic impressions.

All interviews were conducted in Turkish, transcribed verbatim, and anonymized prior to analysis.

Translation Procedure

Selected quotations were translated into English by a bilingual occupational health specialist. Back-translation was performed by an independent bilingual researcher to ensure semantic accuracy. Translations were cross-checked against original transcripts during analysis to preserve meaning.

Data Analysis

Data were analyzed using a reflexively informed, inductive case-based analytic approach.

Analysis proceeded in three iterative stages:

1. Within-case analysis: Each interview was coded inductively to identify key experiential elements.
2. Cross-case comparison: Codes were compared across cases to identify recurring patterns and divergences (16**)**.
3. Mechanism development: Patterns were interpreted at a higher level of abstraction to construct underlying mechanisms.
The analysis prioritized conceptual integration over the generation of multiple discrete themes, in order to avoid over-fragmentation of a small dataset(Figue 1).

Figure 1. Coding tree and process model of smoking persistence among patients with occupational lung disease.

Codes were inductively derived and organized into two higher-order themes to address concerns regarding over-fragmentation. Rather than representing discrete categories, themes form part of a dynamic process, in which external attribution and structural normalization shape constrained agency, sustaining smoking behavior over time. This processual interpretation provides a conceptual response to the temporal dimension of smoking persistence.

Data extraction and coding were performed by a single reviewer. To enhance internal consistency, a blinded re-assessment was conducted after a 2-week washout period, and discrepancies were resolved through structured self-review. N Vivo 12 software was used to support data organization and retrieval. The codebook underlying this analysis is presented to enhance analytic transparency, in a detailed codebook is provided in Supplementary Table 2.

Reflexivity

The research team consisted of clinicians experienced in occupational lung diseases. The principal investigator, a cardiologist working in an occupational diseases hospital with formal training in medical education and qualitative research, conducted all interviews.

A constructivist epistemological stance was adopted, recognizing that meanings are co-constructed between participants and researchers(17**)**. The researchers' clinical experience may have sensitized them to patterns such as external attribution of disease and normalization of smoking behavior.

To address this, reflexive journals were maintained throughout the study, documenting assumptions, analytic decisions, and potential biases. Regular team discussions were held to critically examine how these perspectives influenced interpretation.

Trustworthiness

Rigor was ensured through established qualitative criteria (18):

- **Credibility:** Selective member checking was conducted; two participants reviewed summary interpretations and confirmed their accuracy.
- **Dependability:** A detailed audit trail documented coding, analytic decisions, and mechanism development.

- **Confirmability:** Reflexive documentation and team-based analysis minimized bias.
- **Transferability:** Rich contextual descriptions were provided.

Reporting was guided by the COREQ checklist (19), provided as Supplementary Table S3.

Ethical Considerations

The study was approved by the Institutional Review Board of Ankara Bilkent City Hospital (Approval No: 2341/2024). Written informed consent was obtained from all participants. Clinical trial number: not applicable

Participants were informed that participation was voluntary and would not affect their clinical care. Interviews were conducted by a researcher without direct clinical responsibility to minimize social desirability bias.

Given the sensitive nature of discussing ongoing smoking after diagnosis, participants were allowed to decline questions or withdraw at any time. Emotional support resources were made available if needed.

RESULTS

Participant Characteristics

Five male participants with a confirmed diagnosis of OLD and ongoing active smoking were included. Participants worked in marble processing, foundry work, sandblasting, construction, and agriculture—occupations characterized by prolonged exposure to dust and airborne particulates. All participants had long-term smoking histories and continued smoking despite medical diagnosis and cessation counseling.

Overview of Analytic Findings

Analysis revealed two interrelated mechanisms explaining persistent smoking behavior across cases. The detailed thematic structure underpinning these mechanisms is presented in SupplementaryTable 4:

- External causal attribution and risk reconfiguration
- Structurally constrained smoking persistence

Rather than functioning as independent categories, these mechanisms operated in dynamic interaction, with cognitive interpretations of illness shaping behavior, and structural conditions reinforcing continued smoking.

1. External Causal Attribution and Risk Reconfiguration

Across all cases, participants primarily attributed their illness to occupational exposure rather than smoking:

“Bu hastalık sigaradan değil işten.” *(This disease is from the job, not cigarettes.)* (P2)

This externally oriented attribution was associated with a reconfiguration of perceived risk, in which smoking was cognitively decoupled from disease causation. Even when smoking was acknowledged as harmful, it was consistently positioned as secondary:

“Sigara zararlı ama asıl sebep iş.” *(Smoking is harmful but the main cause is the job.)* (P3)

Participants further minimized the potential benefits of smoking cessation, particularly after prolonged occupational exposure:

“Şimdi bırakmanın ne faydası var?” *(What difference would quitting now make?)* (P1)

A small number of accounts reflected partial internal attribution; however, this recognition did not translate into behavioral change:

“Sigara da etkili, tamamen masum değil.” *(Smoking also plays a role, it’s not completely innocent.)* (P3)

Taken together, these accounts indicate that external causal attribution functioned not merely as an explanatory belief, but as a cognitive mechanism that reduced the perceived urgency and relevance of smoking cessation.

2. Structurally Constrained Smoking Persistence

Smoking behavior was embedded within a set of structural conditions that constrained participants’ ability to initiate or sustain cessation. Occupational exposure itself was normalized as an inevitable aspect of work:

“Toz işin parçası.” *(Dust is part of the job.)* (P1)

Smoking was also socially normalized within workplace environments:

“Herkes sigara içiyor.” *(Everyone smokes.)* (P2)

Participants described smoking as a functional coping mechanism for managing stress and fatigue:

“Sigara stresimi azaltıyor.” *(Smoking reduces my stress.)* (P3)

Despite attempts to quit, participants frequently reported relapse linked to ongoing occupational and psychological demands:

“Hep geri başlıyorum.” *(I always start again.)* (P3)

Socioeconomic constraints further limited cessation capacity:

“İşi kaybetmek her şey demek.” *(Losing the job means everything.)* (P3)

Within this context, participants expressed reduced perceived control over their health behaviors:

“Hiçbir şey değişmez.” *(Nothing will change.)* (P4)

These findings suggest that smoking persistence was not solely a matter of individual choice, but was sustained by structurally embedded constraints including economic necessity, workplace culture, and stress cycles.

Cross-Case Integration

Cross-case analysis demonstrated that these two mechanisms operated in dynamic interaction. External causal attribution reduced the perceived role of smoking in disease causation, thereby weakening motivation for cessation. Simultaneously, structural constraints limited participants’ capacity to act on cessation intentions.

Together, these mechanisms produced a self-reinforcing pattern of behavioral persistence, in which reduced perceived risk and constrained agency mutually sustained continued smoking.

This interaction can be conceptualized as a two-level explanatory model:

- **Cognitive mechanism:** external causal attribution and risk reconfiguration
- **Structural mechanism:** constrained smoking persistence

Negative Case Analysis

A small number of accounts diverged partially from the dominant pattern. Some participants acknowledged the contributory role of smoking:

“Sigara da etkili, tamamen masum değil.” *(Smoking also plays a role, it’s not completely innocent.)* (P3)

However, this awareness did not result in sustained cessation, indicating that knowledge alone was insufficient to overcome structural constraints.

Analytic Transparency

The detailed codebook underlying the analysis is provided.

DISCUSSION

Principal Findings

This study provides an in-depth qualitative exploration of persistent smoking behavior among patients with OLD, revealing that smoking continuation is shaped by a dynamic interaction between illness attribution processes, occupational normalization of risk, addiction, and socioeconomic constraints. Rather than representing an individual behavioral choice, smoking emerged as a contextually embedded practice shaped by structural and cognitive mechanisms.

The methodological reporting of this study was guided by established qualitative standards, including COREQ reporting criteria (20), ensuring transparency in data collection and analysis procedures.

Positioning Within the Literature

Smoking behavior in chronic respiratory disease populations has been extensively studied, particularly in COPD cohorts, where illness perception and coping strategies have been shown to significantly influence smoking persistence (21). These studies demonstrate that patients often reinterpret illness in ways that preserve existing behaviors, including normalization of smoking and cognitive minimization of risk.

However, qualitative research also indicates that such illness representations are not uniform and are strongly shaped by context-specific factors such as occupational exposure narratives and social meaning-making (22). In occupational populations, this interpretive process may be systematically biased toward external causation, as demonstrated in risk perception studies among smokers and lung cancer screening participants (23).

Workplace-based qualitative studies further show that organizational culture and structural conditions influence smoking behavior and cessation feasibility (24). However, these studies primarily focus on prevention or workplace cessation programs rather than individuals already diagnosed with occupational disease.

In addition, systematic reviews highlight that job-related stress is consistently associated with tobacco use, although much of this evidence remains quantitative and limited in capturing lived experience (25). Occupational lung disease itself has been widely documented as a consequence of hazardous exposures in high-risk industries (26), yet little attention has been given to how affected individuals interpret causality between occupational exposures and modifiable risk behaviors such as smoking.

To contextualize sampling adequacy, this study followed the principle of information power, which suggests that sample size in qualitative research should be determined by study aim, specificity, and analytic strategy rather than numerical thresholds (27). In parallel, thematic analytic procedures were informed by established methodological guidance for qualitative analysis (28).

Collectively, this study extends the literature by integrating occupational attribution, structural constraints, and coping processes into a unified mechanism-based model of smoking persistence.

Risk Perception and Causal Attribution

A central finding is the dominance of external causal attribution, whereby participants attributed disease primarily to occupational exposure rather than smoking. This pattern aligns with illness representation theory, which posits that causal beliefs directly shape coping behavior (29).

Participants' accounts suggest that occupational exposure was not merely identified as a risk factor but functioned as a dominant explanatory framework that reduced perceived relevance of smoking cessation. Similar patterns have been observed in studies of lung cancer risk perception, where smokers selectively emphasize environmental over behavioral causes of disease (30).

Within this framework, external attribution appears to operate as a cognitive mechanism that reorganizes risk perception, weakening the perceived link between smoking and disease progression.

Normalization of Occupational Exposure

Participants consistently described hazardous exposures as an unavoidable component of working life, reflecting structural normalization of risk. This aligns with evidence from occupational health studies showing that workers in high-exposure industries often internalize environmental hazards as "part of the job" (31).

Such normalization contributes not only to delayed risk recognition but also reinforces fatalistic interpretations of disease causation and progression.

Smoking as a Coping Mechanism

Smoking was frequently described as a strategy for managing occupational stress and fatigue, consistent with broader evidence linking psychological distress and tobacco use (32). Meta-analytic findings confirm that smoking is often maintained as a form of affect regulation, particularly in high-stress environments (33).

Our findings extend this literature by demonstrating that coping functions of smoking are embedded within occupational and socioeconomic structures rather than solely individual psychological processes.

Addiction and Failed Cessation Attempts

Despite awareness of health risks, participants reported repeated unsuccessful cessation attempts. Existing evidence highlights that nicotine dependence interacts with psychological distress to sustain smoking behavior (34). Similarly, smoking cessation research demonstrates that relapse is strongly influenced by environmental stressors and lack of structural support (35).

Our findings suggest that addiction in this context is not an isolated driver but is reinforced by occupational stress and structural constraints.

Socioeconomic Constraints and Low Perceived Control

Economic necessity emerged as a key constraint shaping continued smoking. Prior workplace studies have identified socioeconomic pressure as a major barrier to behavior change in occupational populations. Consistent with this, participants expressed a diminished sense of control over both health behavior and future outcomes.

This aligns with broader evidence that tobacco use is socially patterned and closely linked to socioeconomic disadvantage at both national and global levels .

Integration of Findings

Taken together, the findings suggest that persistent smoking in occupational lung disease is sustained through the interaction of:

- Cognitive mechanisms (external attribution and risk reconfiguration)
- Behavioral mechanisms (coping and addiction reinforcement)
- Structural mechanisms (economic dependency and occupational normalization)

Rather than independent themes, these elements form an integrated explanatory model of smoking persistence in occupationally exposed populations.

Implications for Practice

Smoking cessation interventions in occupational lung disease should move beyond individual-level counseling and incorporate:

- Attribution-focused communication strategies targeting illness beliefs
- Stress and coping interventions embedded in occupational contexts
- Structural sensitivity to economic and workplace constraints
- Integration of workplace-based preventive approaches with clinical care systems

Workplace intervention research supports the feasibility of such integrated approaches in high-risk occupational settings (36).

Strengths and Limitations

This study provides novel qualitative insight into a highly specific and underexplored population. The use of a case-based design and in-depth interviews allowed detailed exploration of mechanisms underlying smoking persistence.

However, limitations include the small sample size, single-country recruitment, and male-only composition, which may limit transferability. Nevertheless, qualitative rigor was strengthened through transparent reporting guided by COREQ standards and structured analytic procedures

Conclusion

Persistent smoking among patients with occupational lung disease reflects a complex interplay of illness attribution, occupational normalization, addiction, and socioeconomic constraints. This study extends existing literature by proposing a mechanism-based model that integrates cognitive and structural determinants of behavior. These findings highlight the need for multidimensional, context-sensitive smoking cessation interventions tailored to occupationally exposed populations.

Abbreviations

OLD
Occupational lung disease
COPD
Chronic obstructive pulmonary disease
COREQ
Consolidated criteria for reporting qualitative research.

Declarations

Ethics approval and consent to participate

The study was approved by the Institutional Review Board of Ankara Bilkent City Hospital (Approval No: 2341/2024). Written informed consent was obtained from all participants prior to data collection. Participation was voluntary, and participants were informed that they could withdraw at any time without any effect on their clinical care.

Consent for publication

Not applicable. No identifiable personal data are included in this manuscript.

Availability of data and materials

The datasets generated and/or analyzed during the current study are not publicly available due to participant confidentiality but may be obtained from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

CM: Conceptualization, methodology, data collection, formal analysis, writing–original draft preparation.
[Add co-authors here if present]: contributed to data analysis, interpretation, and critical revision of the manuscript. All authors read and approved the final manuscript.

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Figures

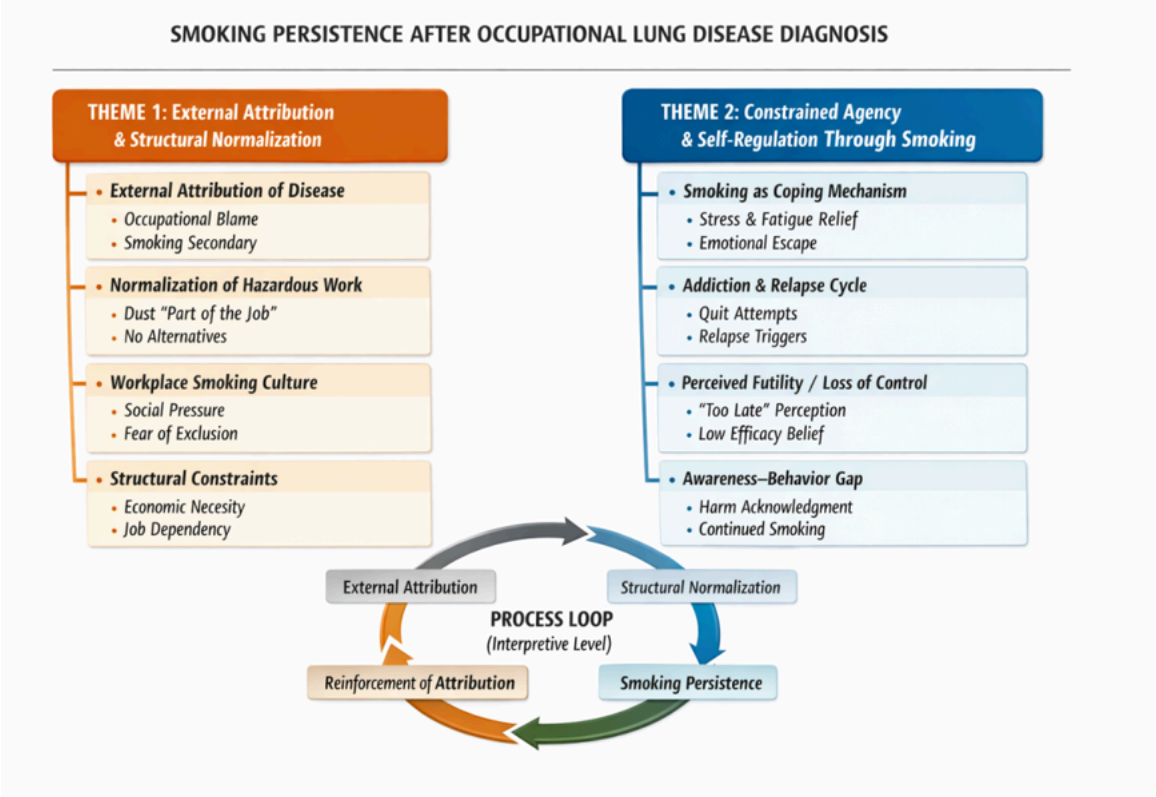


Figure 1

Coding tree and process model of smoking persistence among patients with occupational lung disease.

Codes were inductively derived and organized into two higher-order themes to address concerns regarding over-fragmentation. Rather than representing discrete categories, themes form part of a dynamic process, in which external attribution and structural normalization shape constrained agency, sustaining smoking behavior over time. This processual interpretation provides a conceptual response to the temporal dimension of smoking persistence.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryMaterialS1.docx
- SupplementaryTables.docx