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COGNITIVE IMPAIRMENT PROFILES IN CHRONIC SMOKERS ACROSS BRINKMAN INDEX DEGREES

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Abstract

Background: Smoking is a changeable risk factor for cognitive decline, working through harmful effects on the brain and inflammation. Although many people in Indonesia still smoke, there is little detailed information on how long-term tobacco use, measured by the Brinkman Index, relates to specific cognitive impairments. **Methods:** A descriptive study was done at the Neurology Outpatient Clinic of Dr Saiful Anwar General Hospital, Malang, from July to August 2025. Eighty-eight adult current and former smokers were included sequentially. Cognitive function was assessed using the Indonesian Montreal Cognitive Assessment (MoCA-INA; impairment defined as a score below 26), and total tobacco use was measured with the Brinkman Index, categorised as mild (<200), moderate (200–600), or severe (>600). **Results:** All participants were men, with an average age of 52.9 years. More than half (71.6%) were former smokers. On average, they started smoking at 18.7 years old, smoked for about 29.3 years, and smoked 12.2 cigarettes daily. Most (72.3%) had moderate-to-high Brinkman Index scores. Cognitive problems were found in 65 people (73.9%), with an average MoCA-INA score of 20.7; all with problems had smoked before. Memory was the most affected area (98.4%), followed by thinking skills (75.4%), visual and planning skills (61.5%), attention (53.8%), language (50.1%), and awareness of surroundings (18.5%). **Conclusion:** Long-term heavy tobacco use is linked to lower cognitive function, especially in memory, thinking, visual skills, and attention. These results support early cognitive testing for people who have smoked for a long time.

Keywords: Brinkman Index; chronic smoker; cognitive impairment; MoCA-INA

Abstrak

Latar Belakang : *Merokok merupakan faktor risiko yang dapat dimodifikasi dan terbukti berkontribusi terhadap penurunan fungsi kognitif melalui mekanisme neurotoksik, neuroinflamasi, dan stres oksidatif. Di Indonesia, prevalensi perokok aktif masih sangat tinggi, namun data mengenai profil gangguan kognitif pada populasi perokok khususnya yang dinilai berdasarkan akumulasi paparan tembakau melalui Indeks Brinkman masih sangat terbatas.* ***Metode:*** *Penelitian deskriptif potong-lintang (cross-sectional) yang dilakukan di Poliklinik Neurologi RSUD Dr. Saiful Anwar Malang dari Juli hingga Agustus 2025. Sebanyak 88 perokok aktif dan mantan perokok laki-laki diikutsertakan secara berurutan (consecutive sampling). Fungsi kognitif dinilai menggunakan Indonesian Montreal Cognitive Assessment (MoCA-INA; gangguan didefinisikan sebagai skor <26). Akumulasi paparan rokok diukur menggunakan Indeks Brinkman dan dikategorikan menjadi ringan (<200), sedang (200–600), atau berat (>600).* ***Hasil:*** *Seluruh subjek (100%) berjenis kelamin laki-laki dengan rerata usia 52,9 ± 14,1 tahun. Mayoritas subjek merupakan mantan perokok (71,6%) dengan rerata usia mulai merokok 18,7 tahun, rerata durasi merokok 29,3 tahun, dan intensitas konsumsi rata-rata 12,2 batang per hari. Sebanyak 72,3% subjek termasuk kategori perokok sedang hingga berat berdasarkan Indeks Brinkman. Dari total 88 subjek, sebanyak 65 orang (73,9%) mengalami gangguan kognitif dengan rerata skor MoCA-INA 20,7. Domain kognitif yang paling banyak terganggu adalah memori (98,4%), diikuti abstraksi (75,4%), visuospasial/eksekutif (61,5%), atensi (53,8%), bahasa (50,1%), dan orientasi (18,5%).* ***Kesimpulan:*** *Paparan rokok jangka panjang dengan dosis kumulatif tinggi menurunkan fungsi kognitif (MoCA-INA), terutama domain memori, abstraksi, visuospasial/eksekutif, dan atensi. Tingginya Indeks Brinkman mencerminkan paparan berat, menegaskan pentingnya evaluasi fungsi kognitif dini pada perokok kronis guna mendeteksi penurunan neurokognitif.*

Kata kunci*: Indeks Brinkman; perokok kronis; gangguan kognitif; MoCA-INA*

INTRODUCTION

Smoking continues to be among the most important public health problems worldwide, with nearly 1.3 billion active smokers living globally[1]. Indonesia stands out as a country with both a high and consistently rising prevalence of smoking[2]. Which in turn, increases the burden of substantial chronic disease[3]. While smoking is well known for its cardiovascular and respiratory effects[4], growing evidence also points to neurological consequences: smoking compounds can cross the blood-brain barrier and interfere with the central nervous system[5].

Smoking works as an adjustable risk factor for cognitive impairment[6]. It tends to coincide with poorer cognitive ability plus a greater risk of Alzheimer's disease[5]. The primary processes revolve around oxidative stress, vascular inflammation, and neurotoxicity[7], with mitochondrial dysfunction stemming from benzo(a)pyrene[8]. How long and how hard people smoke, measured by the Brinkman Index, correlates closely with the severity of cognitive problems. Furthermore, individuals who smoke display more rapid brain atrophy compared to non-smokers[5].

The Montreal Cognitive Assessment (MoCA) is a validated tool for screening cognitive function across several domains, with an Indonesian-language variant, MoCA-INA, developed for community use[9]. The instrument evaluates eight cognitive domains, including executive function, memory, and attention[9]. It demonstrates good sensitivity and specificity in identifying Mild Cognitive Impairment, which is often overlooked in routine assessments[10].

Although analytic studies have supported the relationship between smoking and cognitive decline, detailed descriptive information about smoking profiles among people with cognitive impairment in Indonesia remains scarce[9]. It is therefore necessary to clarify how smoking patterns, particularly smoking duration and Brinkman Index values, relate to clinical characteristics in groups with low MoCA-INA scores[6]. Establishing this is important for determining the most vulnerable group 5 and for supporting the design of cognitive screening strategies, health policy, and secondary prevention efforts [6,9].

This work is designed to explain the profile and key characteristics of individuals with cognitive impairment with a history of chronic smoking[6]. Specifically, it assesses demographic characteristics, the duration and intensity of smoking 4, the distribution of the Brinkman Index 6, as well as MoCA-INA scores and the cognitive domains most affected [9]. The

findings are intended to optimize clinical management for smoking-related patients [5].

MATERIALS AND METHODS

a. Study Design and Ethical Approval

This study characterizes tobacco exposure patterns and the clinical features of smokers with cognitive impairment. The Research Ethics Committee of Dr. Saiful Anwar Hospital in Malang, East Java, Indonesia, approved the study. Before data collection began, researchers explained the study to each participant in detail and obtained written informed consent.

b. Study Setting and Period

Data were collected at the Neurology Outpatient Clinic, Saiful Anwar Hospital, a type-A referral center that provides neurology services across East Java, from July through August 2025.

c. Population and sampling

The study enrolled adult smokers (aged > 18 years) who attended the neurology outpatient clinic during the study period and met the inclusion criteria. The study's cross-sectional design meant that total sampling was applied. We recruited all eligible patients consecutively and then assessed cognitive function using the MoCA-INA.

- Inclusion criteria: age greater than 18 years; current or former smoker status; attendance at the neurology outpatient clinic during the study period; provision of written informed consent and sufficient alertness and cooperation to complete the interview and the MoCA-INA assessment
- Exclusion criteria: inability to complete cognitive testing due to impaired consciousness, severe aphasia, or other major neurological deficits; lack of cooperation during the interview or assessment; and a history of severe neurological or psychiatric illness that could independently affect cognition (for example, severe dementia, uncontrolled epilepsy, or acute psychosis)
- Sampling technique and sample size: A consecutive method of guided sampling was used, and recruitment continued until the required sample size requirement was reached. In total, 88 individuals from the eligible population available throughout the study interval were included.

d. Study Variables

The study variables were grouped into three domains: (1) demographic characteristics (age, and educational level, categorized as elementary/junior high school, senior high school, diploma,

or university); (2) smoking characteristics (duration of smoking, mean daily cigarette consumption, and Brinkman Index); and (3) cognitive status (the total MoCA-INA score, individual domain scores, and the classification of cognitive impairment).

e. Operational Definitions

Smoking status was established through a structured interview that assessed current and past smoking behaviours. Exposure duration was calculated as the gap between the year daily smoking began and the year of data collection. Intensity was the typical number of cigarettes smoked per day during that period. Multiplying the two, cigarettes per day by years smoked, gave the Brinkman Index: mild under 200, moderate 200 to 600, heavy above 600. Cognitive impairment meant a total MoCA-INA score below 26 out of 30, the cutoff validated for the Indonesian population.[9]

f. Data Collection Instruments and Procedure

Two instruments were used. A structured questionnaire captured demographic data and smoking history; it was pilot-tested on 10 respondents to check clarity. Cognitive performance was measured with the MoCA-INA, the Indonesian-adapted Montreal Cognitive Assessment, which scores eight domains (visuospatial/executive function, naming, attention, language, abstraction, delayed recall, orientation, and calculation) for a maximum of 30 points. Respondents with fewer than 12 years of formal schooling receive a 1-point adjustment.

Eligibility rested on medical records and a brief interview. After patients gave informed consent, researchers conducted a structured interview, 10 to 15 minutes, covering demographics and smoking history. A face-to-face MoCA-INA assessment came next: 15 to 20 minutes in a quiet room to limit distraction. Researchers calculated Brinkman Index values by hand from the interview responses. Data went onto case report forms, checked for completeness before each participant left the study site.

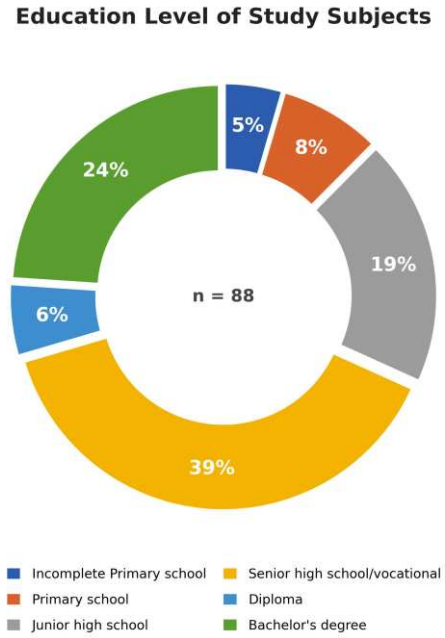
RESULTS

a. Demographic Characteristics

Table 1 summarizes the demographic characteristics of the study population, including age and educational attainment.

Table 1. Demographic Characteristics

Characteristic		n / mean ± SD	Proportion (%)
Total subjects	n = 88 (100%)		
Age	Mean ± SD	52.9 ± 14.1 years	
Education Level	Incomplete Primary school	4	4.5
	Primary school	7	7.9
	Junior high school	17	19.3
	Senior high school/vocational	34	38.6
	Diploma	5	5.6
	Bachelor's degree	21	23.9



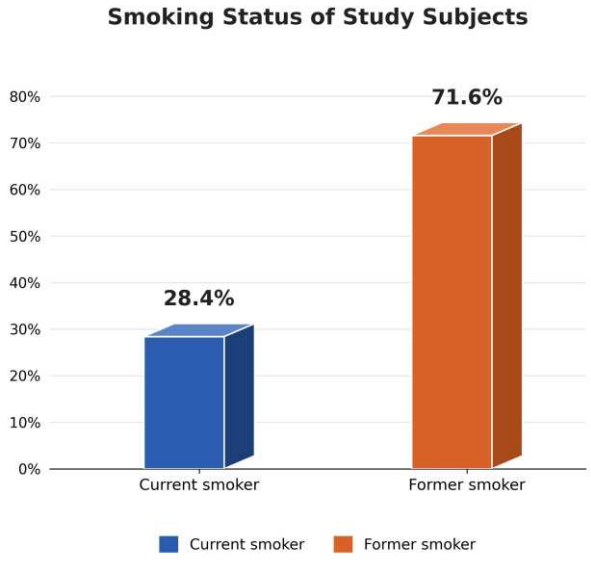
Graph 2. Proportion of education level data

b. Smoking Exposure Profile and Brinkman Index

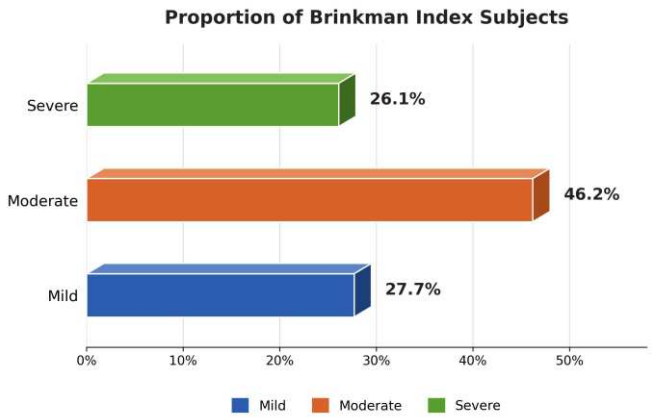
The distribution of data regarding the history, duration, intensity of smoking, and the category of the subject's Brinkman Index can be seen in Table 2, Graph 3, and Graph 4 in the following data:

Table 2. Smoking Exposure Profile and Brinkman Index

Characteristic		n/ mean ± SD	Proportion (%)
Total Subjects	n = 88 (100%)		
Smoking Status	Current smoker	25	28.4
	Former smoker	63	71.6
Age at smoking onset	Mean ± SD	18.7 ± 3.7 years	
Cigarettes per day	Mean ± SD	12.2 ± 6.3 cigarettes/day	
Smoking Duration	Mean ± SD	29.3 ± 14.9 years	
Brinkman Index	Mild	18	27.7
	Moderate	30	46.2
	Severe	17	26.1



Graph 3. Proportion of smoking status of research



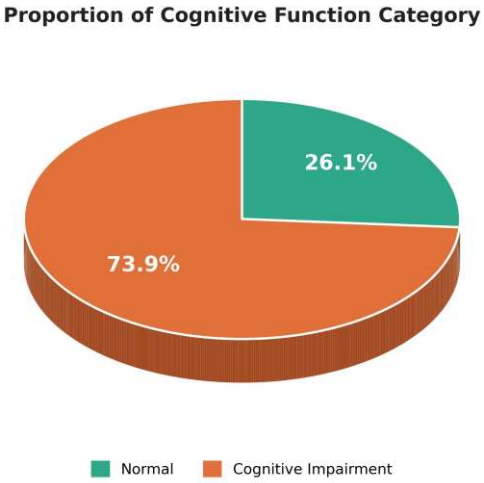
Graph 4. Proportion of Brinkman Index subjects

c. Cognitive Impairment Profile (MoCA-INA)

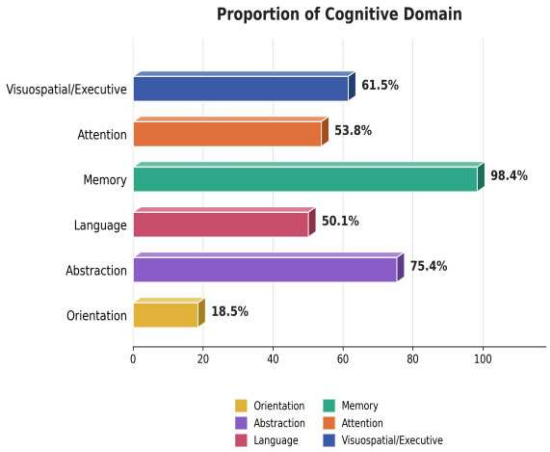
Details on total scores, cognitive impairment status, and data from each domain on the subject MoCA-INA can be seen in Table 3, Graph 5, and Graph 6 below:

Table 3. MoCA-INA Score Profile and Impaired Cognitive Domains

Cognitive Function Variable		**n/ mean ± SD**	**Proportion (%)**
Total MoCA-INA score	Mean ± SD	20.7 ± 6.0	
Cognitive Function Category	Normal	23	26.1
	Cognitive Impairment	65	73.9
MoCA-INA Domains	Visuospatial/Executive	40	61.5
	Attention	35	53.8
	Memory	64	98.4
	Language	33	50.1
	Abstraction	49	75.4
	Orientation	12	18.5



Graph 5. Proportion of Cognitive Function Category



Graph 6. Proportion of Cognitive Domain

DISCUSSION

Each participant was a man, in step with global and local tobacco-use patterns showing males are generally more likely to

smoke than females. A sizable South Korean cohort reflected this pattern, with 95.8% of over 700,000 participants being male[11]. Similarly, Indonesian data corroborate this trend, showing a decline in female smoking prevalence from 6.9% in 2000 to 3.3% in 2020[12]. A Sudanese study recruited only males because of sociocultural barriers to identifying female smokers[13].

The majority of participants had completed secondary education (38.6%) or held a bachelor's degree (23.9%). This is consistent with demographic evidence that smoking prevalence is higher among individuals with secondary or vocational education[4]. Educational attainment may also serve as an indicator of cognitive reserve, with lower levels of education correlating with heightened susceptibility to smoking[11]. For this reason, it is important to include individuals across the full educational range, given the increased likelihood of cognitive decline at the lower end of the range[14]. Accordingly, educational level should be included as a covariate in future analyses aimed at isolating the effect of smoking on cognition, consistent with approaches used in other large studies[15,16].

In this sample, the smoking profile mainly consisted of former smokers (71.6%) with a mean onset age of 18.7 years, which underscores neurobiological risks tied to early nicotine exposure, which has been associated with cognitive decline and dementia[17]. Both current and former smokers showed cognitive impairment in 65 subjects (73.9%), with an average smoking history of 29 years, reflecting chronic-exposure patterns seen in other large cohorts in which prolonged smoking is associated with neurodegenerative outcomes[11]. Many participants had already quit; however, the literature indicates that structural brain changes associated with smoking can persist after cessation, although quitting remains beneficial by slowing the rate of brain atrophy and cognitive decline compared with continued smoking[18,19].

The high prevalence of moderate-to-heavy smokers in this cohort, accounting for 72.3%, suggests a substantial cumulative toxic exposure associated with structural cerebral damage[18,19].

Neuroimaging studies demonstrate that increased pack-years are associated with decreased grey matter volume in memory-related regions[15]. Local research in Indonesia similarly reported an increased risk of cognitive impairment among individuals with ≥20 pack-years, supporting smoking duration as a predictor of neurocognitive deficits[12].

Cognitive impairment was found in 65 of 88 subjects (73.9%), and all were smokers. This indicates smoking may

adversely affect cognitive function, as reflected by a low mean MoCA-INA score (20.7 ± 6.0), well below the normal cutoff of 26. The high prevalence is consistent with a rural Indonesian study reporting a dose-response pattern: when ≥20 pack-years are reached, the risk of cognitive problems rises substantially[12]. The reduction in overall scores suggests a systemic impact of chronic tobacco exposure, aligning with decreased grey matter volume observed in the UK Biobank study[19]. The absence of typical scores across subjects suggests that smoking is an important changeable risk factor linked to quicker cognitive worsening and dementia [11].

Memory was the most affected domain (98.4%), consistent with neuroimaging findings linking smoking to hippocampal atrophy, a structure critical for episodic memory[17]. A national Chinese study further confirmed this, showing that active smokers performed markedly worse on episodic memory than non-smokers, particularly after longer smoking duration [20]. This memory impairment is likely worsened by neuroinflammatory processes and oxidative stress stemming from smoking, which then harms hippocampal neurogenesis[13]. Through the lung-brain axis, smoking-induced neuroinflammation and oxidative stress influence hippocampal neurogenesis: lung injury releases pro-inflammatory cytokines that activate hippocampal microglia[7]. Excessive microglial activation, together with the accumulation of reactive oxygen species accumulation, generates a neurotoxic milieu that impairs mitochondrial function and reduces neural progenitor cell proliferation, ultimately resulting in hippocampal atrophy and cognitive deficits [17, 21].

Deficits in abstraction (75.4%) and visuospatial/executive function (61.5%) suggest frontal lobe dysfunction, supported by resting-state fMRI findings of reduced connectivity in the dorsal attention network and grey matter loss in the frontal gyrus, a region linked to cognitive control[22]. This finding provides further support for the notion that smoking causes cortical thinning in the frontal areas, which in turn undermines planning and problem solving [19]. Findings reveal impairments in abstraction, visuospatial abilities, and executive functioning.

Moreover, 53.8% of participants reported attention deficits, while memory issues were more frequently observed. Although nicotine can temporarily enhance attention, chronic exposure slows processing speed. NHANES findings connect smoking with reduced processing speed, likely reflecting neurotoxicity[16]. Reduced attention may result from thalamic volume loss, since heavy smokers show

thalamic shrinkage[15], which could underlie attention deficits.

Cognitive decline is promoted by cerebrovascular injury, oxidative stress, and neuroinflammation[11]. These findings reinforce earlier work indicating that long-term, high-intensity smoking, as measured by the Brinkman Index, increases the risk of neurocognitive impairment

CONCLUSION

The study included only men. This follows the broader pattern of smoking-related disease falling more heavily on males, and it likely means participants here carried heavier cumulative exposure and a higher risk of cognitive impairment. Many also had limited schooling. Lower education has been linked in prior studies to reduced cognitive reserve, which may compound the effects of long-term smoking and hasten decline.

Smoking in this cohort began early and continued for years, consistent with sustained accumulation of nicotine and other toxic compounds, which is generally associated with lasting structural and functional brain changes. Cognitive impairment persisted even in participants who had quit smoking. The neurocognitive effects of sustained smoking, then, may not fully reverse, particularly for those with substantial cumulative exposure.

By the Brinkman Index, a substantial share of participants qualified as moderate-to-heavy smokers, indicating high cumulative exposure. Decline tracked both intensity and duration: heavier, longer smoking meant worse cognitive performance. Memory took the biggest hit at the domain level, followed by abstraction, then visuospatial/executive function and attention. Rather than one isolated domain, cumulative smoking exposure appears to affect the neural circuits underlying memory and executive control more broadly.

Chronic smokers scored worse on the MoCA-INA overall, particularly in memory, abstraction, visuospatial/executive function, and attention. Higher Brinkman Index scores, marking greater cumulative exposure, likely contributed to this impairment. Chronic smokers may benefit from routine cognitive screening to catch neurocognitive decline early.

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