

## Original Article



# Facial Emotion Recognition in Older Adults With Cognitive Complaints

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### Conflict of Interest

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## ABSTRACT

**Background and Purpose:** Facial emotion recognition deficits impact the daily life, particularly of Alzheimer's disease patients. We aimed to assess these deficits in the following three groups: subjective cognitive decline (SCD), mild cognitive impairment (MCI), and mild Alzheimer's dementia (AD). Additionally, we explored the associations between facial emotion recognition and cognitive performance.

**Methods:** We used the Korean version of the Florida Facial Affect Battery (K-FAB) in 72 SCD, 76 MCI, and 76 mild AD subjects. The comparison was conducted using the analysis of covariance (ANCOVA), with adjustments being made for age and sex. The Mini-Mental State Examination (MMSE) was utilized to gauge the overall cognitive status, while the Seoul Neuropsychological Screening Battery (SNSB) was employed to evaluate the performance in the following five cognitive domains: attention, language, visuospatial abilities, memory, and frontal executive functions.

**Results:** The ANCOVA results showed significant differences in K-FAB subtests 3, 4, and 5 ( $p=0.001$ ,  $p=0.003$ , and  $p=0.004$ , respectively), especially for anger and fearful emotions. Recognition of 'anger' in the FAB subtest 5 declined from SCD to MCI to mild AD.

Correlations were observed with age and education, and after controlling for these factors, MMSE and frontal executive function were associated with FAB tests, particularly in the FAB subtest 5 ( $r=0.507$ ,  $p<0.001$  and  $r=-0.288$ ,  $p=0.026$ , respectively).

**Conclusions:** Emotion recognition deficits worsened from SCD to MCI to mild AD, especially for negative emotions. Complex tasks, such as matching, selection, and naming, showed greater deficits, with a connection to cognitive impairment, especially frontal executive dysfunction.

**Keywords:** Emotions; Facial Recognition; Alzheimer Disease; Cognitive Dysfunction

## INTRODUCTION

Facial emotion recognition refers to the capacity to perceive, process, and identify emotions conveyed through facial expressions, and it has long been regarded as a fundamental human skill. Deficits have been observed in individuals with various neurodegenerative conditions and the impairment in facial emotion recognition skills is often attributed to global cognitive impairments resulting from pathological processes, rather than direct functional deficits.<sup>1</sup>

However, several studies have provided evidence suggesting the pivotal role of the prefrontal cortex in processing emotional stimuli; thus, making this brain region a likely candidate for potential impairments.<sup>2</sup> Some studies have indicated that Alzheimer's dementia (AD) patients exhibit difficulties in processing facial expressions of emotions.<sup>3-6</sup> Specifically, there is evidence in AD patients that impaired facial emotion recognition has an impact on interpersonal behavior, independent of general cognitive decline.<sup>7</sup> Therefore, adopting a social cognition approach could contribute to a deeper understanding of this intricate disorder.<sup>8</sup> Notably, the Diagnostic and Statistical Manual of Mental Disorders V (DSM-V) has incorporated social cognition as one of the cognitive domains affected by and evaluated in Alzheimer's disease.<sup>9</sup>

Nevertheless, the relationship between impairment in facial emotion recognition and cognitive deficits in AD patients remains somewhat enigmatic. Some studies have suggested that facial emotion recognition abilities are distinct from cognitive performance related to face processing, underscoring the involvement of separate systems in emotional and visuospatial processing.<sup>3</sup> Conversely, some authors have proposed a connection between deficits in facial emotion recognition and cognitive impairment,<sup>10</sup> particularly in terms of visual perceptual abilities.<sup>4</sup> A potential explanation for these conflicting findings may be the variations in emotion recognition tests, types of cognitive tasks, and patient samples with varying degrees of illness severity.<sup>5</sup> Facial emotion recognition is a complex process involving different cerebral structures, such as the occipitotemporal cortex, amygdala, orbitofrontal cortex, and right parietal cortex,<sup>11</sup> and as such, it engages in a range of cognitive abilities that are differentially affected during the progression of dementia. Therefore, it would be intriguing to elucidate the extent to which facial emotion recognition impairment is associated with memory decline, a hallmark of Alzheimer's disease from its preclinical stages.

Given the fact that facial emotion recognition impairment tends to escalate with the advancement of dementia,<sup>6</sup> it raises the question whether it is also present in individuals with the spectrum of Alzheimer's disease before the occurrence of symptoms of dementia, such as mild cognitive impairment (MCI) and subjective cognitive decline (SCD). Although limited information is available, recent studies have suggested that facial emotion recognition may be impaired in individuals with MCI.<sup>12</sup> Thus, research has indicated that facial emotion recognition impairment is also present in individuals with MCI and mild AD,<sup>3-5,12</sup> although authors have enquired whether the small sample size influenced these results.

No research has been conducted in Korea to date that evaluates facial emotion recognition among the elderly population and individuals with neurodegenerative diseases. With the recently validated measures, specifically the Korean version of the Florida Facial Affect Battery (K-FAB),<sup>13</sup> this study was designed to explore the facial emotion recognition impairments throughout the Alzheimer's disease spectrum, given that regions of the medial temporal lobe play crucial roles in both the memory and emotional processes and are affected early in MCI and mild AD patients.<sup>14,18</sup>

## METHODS

### Patients

Following comprehensive clinical evaluation, neurological examinations, laboratory tests, neuropsychological assessments, and magnetic resonance imaging (MRI), our study enrolled

a total of 76 individuals with probable AD as per the National Institute on Aging-Alzheimer's Association criteria.<sup>19</sup> Additionally, 76 participants with MCI, diagnosed according to the National Institute on Aging-Alzheimer's Association criteria,<sup>20</sup> and 72 individuals with SCD, as per the guidelines outlined by Jessen et al.,<sup>21</sup> were included. The assessment of dementia severity and general cognitive function was conducted using the clinical dementia rating (CDR)<sup>22</sup> and the Korean Mini-Mental State Examination, 2nd edition (K-MMSE-2),<sup>23,24</sup> respectively. Furthermore, we assessed depressive symptoms using the geriatric depression scale (GDS),<sup>25</sup> and cognitive performance across 5 domains (attention, language, visuospatial abilities, memory, and frontal executive functions) using the Seoul Neuropsychological Screening Battery (SNSB), the 2nd edition of SNSB (SNSB-II).<sup>26</sup> Participants with conditions that could potentially lead to cognitive impairment, such as hypothyroidism, vitamin B12 deficiency, syphilis, or a history of major psychiatric illness (e.g., major depression, bipolar disorder, or schizophrenia), were excluded. We also excluded patients with MRI abnormalities, such as brain tumors, radiation injury, hippocampal sclerosis, and multiple sclerosis. Subjects with severe white matter hyperintensities or multiple (more than 5) lacunar infarctions, consistent with Erkinjuntti's brain imaging criteria for subcortical ischemic vascular dementia,<sup>27</sup> were also excluded. None of the participants had a history of previous stroke or focal neurological symptoms.

The Institutional Review Board of the Catholic University of Korea, Eunpyeong St. Mary's Hospital approved the study (PC23RISI0170), and informed consent was obtained from all participants or their caregivers. This study was conducted in accordance with the Declaration of Helsinki.

### Facial emotion recognition examination

To evaluate the facial emotion recognition ability, we administered a standardized and validated instrument known as the K-FAB,<sup>13</sup> which consists of 5 subtests. The original version of the Florida Affect Battery (FAB)<sup>28</sup> was designed to assess the perception of facial and prosodic affect across various task demands, and it included 10 different subtests (comprising 5 facial, 3 prosodic, and 2 cross-modal subtests). However, the Korean version focused exclusively on facial affect tasks, including subtest 1 (facial identity discrimination), subtest 2 (facial affect discrimination), subtest 3 (facial affect naming), subtest 4 (facial affect selection), and subtest 5 (facial affect matching). These tasks involved the recognition of five different emotions (happiness, sadness, anger, fear, and neutrality).

In subtest 1, the facial identity discrimination task, subjects were shown pairs of unfamiliar faces and they had to determine whether the faces are the same or of a different person. The stimuli were photographs with a neutral facial expression. Their hair was covered by a surgical cap to reduce cues for identification. This identity discrimination task could serve as a perceptual "control" for the facial affect tasks. In subtest 2, the facial affect discrimination task, subjects had to determine whether two faces depicted the same or different emotional expressions. Subtest 3, the facial affect naming task required the subjects to verbally label facial expressions. Individual faces were shown as stimuli, and the subject was asked to name the emotion depicted by a particular face (i.e., happy, sad, angry, frightened, neutral). Subtest 4, the facial affect selection task assessed the ability to select target facial expressions named by the examiner. In each trial, subjects were shown five pictures of different people, each expressing different facial emotions. The subjects were asked identify the picture of the face that corresponded to the emotion named by the examiner (i.e., "point to the angry face"). In subtest 5, the facial affect matching task, subjects were asked to match the picture

of an emotional face to another face with the same emotional expression. The subjects were shown a stimulus slide consisting of multiple photographs. On the left side of the slide, there was a single photograph of a target emotional face. To the right of the slide, there were pictures of five people, expressing different emotional expressions. Their task was to match the target expression with its counterpart on the right of the slide.

### Statistical analysis

Demographic characteristics and results of all K-FAB tests were compared among the three diagnostic groups using the analysis of covariance (ANCOVA), with adjustments for age and sex. Bonferroni corrections for multiple comparisons were applied to all post-hoc tests, as deemed appropriate. Normality was assessed using the Shapiro-Wilk test. Pearson's correlation analysis was employed to evaluate the relationships between the results of all K-FAB tests and demographic variables, such as age, sex, and education in the entire sample. Subsequently, partial correlations, adjusted for age and education, were computed to estimate correlation coefficients and assess their significance for the relationships among the K-FAB test results, K-MMSE-2 scores, and the results of SNSB-II cognitive domains. Linear regression analysis was also conducted, examining the subtests of K-FAB and the cognitive domains of SNSB-II in relation to K-MMSE-2. Especially, in SCD subjects, who did not have any objective cognitive impairment, we investigated the domains of SNSB that will affect K-FAB, even if they had not yet deteriorated significantly.

A significance level of  $p < 0.05$  (two-tailed) was considered statistically significant, and all statistical analyses were performed using the Statistical Package for the Social Sciences version 28.0 software program (IBM Corp., Armonk, NY, USA).

## RESULTS

### Demographic comparisons

Sociodemographic characteristics of SCD, MCI, and mild AD are presented in **Table 1**. The three groups differed significantly in terms of age and sex. Therefore, subsequent group comparisons were performed after controlling for age and sex. Although the global cognitive level assessed by K-MMSE-2 differed, the GDS score was not different among groups. *Post hoc* comparisons revealed that K-MMSE-2 scores increased continuously from SCD to MCI and from MCI to mild AD.

### Facial emotion recognition comparisons

**Table 2** shows the differences among SCD, MCI, and mild AD groups for facial emotion recognition abilities. The ANOVA results indicated that starting from subtest 3, facial affect naming, there were differences among the 3 groups. More complex tasks, such as subtest 4, facial affect selection, and subtest 5, facial affect matching, also differed. Subtest 1, the facial identity discrimination task, served as a perceptual "control" for the facial affect tasks, and subtest 2, facial affect discrimination, was not different among the three groups.

Among the 5 target emotions, anger and fear were different among the 3 groups, although neutral emotion also showed a difference only in subtest 5. *Post hoc* comparisons revealed that deficits of anger recognition in subtest 5 increased continuously from SCD to MCI patients and from MCI to mild AD subjects (**Fig. 1**). Only fear emotion recognition was different, even after additionally controlling for the MMSE score ( $p = 0.013$ ).

**Table 1.** Clinical characteristics of study participants

Characteristics	SCD (n=72)	MCI (n=76)	AD (n=76)	p-value	Post hoc
Age (yr)	72.86±8.04	73.92±7.03	78.92±6.92	<0.001	1 vs. 3, 2 vs. 3
Sex (male: female)	15:57	33:43:00	18:48	0.009	1 vs. 2
Education (yr)	8.81±4.73	10.00±4.55	8.71±5.07	0.195	
APOE ε4 genotype	31 (29.03%)	41 (39.02%)	65 (44.62%)	<0.001	
K-MMSE-2	27.22±2.68	24.54±3.36	19.89±4.26	<0.001*	1 vs. 2 vs. 3
CDR	0.49±0.08	0.50±0.00	0.87±0.38	<0.001*	1 vs. 3, 2 vs. 3
SOB	1.24±0.74	1.60±0.83	5.30±2.23	<0.001*	1 vs. 3, 2 vs. 3
GDS	9.75±7.49	8.96±7.27	9.59±8.09	0.801	
SNSB-II (domains)	n=63	n=69	n=50		
Attention	54.40±25.56	37.72±27.12	40.32±28.27	0.001*	1 vs. 2, 1 vs. 3
Language	66.09±25.28	44.48±28.26	32.03±26.62	<0.001*	1 vs. 2 vs. 3
Visuospatial	58.23±21.07	40.43±27.70	35.34±31.41	<0.001*	1 vs. 2, 1 vs. 3
Memory	62.83±23.77	15.46±15.60	7.21±14.98	<0.001*	1 vs. 2, 1 vs. 3
Frontal executive	65.31±24.93	48.57±86.82	15.74±20.53	<0.001*	1 vs. 3, 2 vs. 3

SCD: subjective cognitive decline, MCI: mild cognitive impairment, AD: Alzheimer’s dementia, APOE: apolipoprotein E, K-MMSE-2: Korean mini-mental state examination, 2nd edition, CDR: clinical dementia rating, SOB: sum of boxes, GDS: geriatric depression scale, SNSB-II: Seoul Neuropsychological Screening Assessment, 2nd edition.

Values are presented as means ± standard deviations or raw numbers of participants. Analyses were performed using the analysis of variance and \*the analysis of covariance, adjusted for age and sex.

**Table 2.** Comparison of the K-FAB results in the 3 participant groups

Code	SCD (n=72)	MCI (n=76)	AD (n=76)	p-value*	Post hoc	p-value†
FAB1	18.81±1.91	18.55±1.92	17.68±2.12	0.146		
FAB1_S	0.64±1.28	0.79±1.27	1.75±0.22	0.484		
FAB1_D	0.61±0.99	0.66±1.04	1.14±1.36	0.988		
FAB2	17.46±1.89	17.07±1.86	16.65±2.20	0.244		
FAB2_S	1.14±1.69	1.75±1.95	2.18±1.90	0.153		
FAB2_D	1.43±0.99	1.18±1.22	1.20±1.37	0.358		
FAB3	16.96±2.42	15.38±2.79	14.76±2.27	0.001	1 vs. 2, 1 vs. 3	0.076
FAB3_H	0.00±0.00	0.03±0.16	0.05±0.21	0.502		
FAB3_S	1.19±1.04	1.54±1.04	1.44±1.25	0.235		
FAB3_A	1.17±1.11	1.49±1.11	1.94±1.05	0.017	1 vs. 3	0.969
FAB3_F	0.17±0.44	0.54±1.00	0.74±1.00	0.005	1 vs. 2, 1 vs. 3	0.288
FAB3_N	0.72±1.94	1.03±1.19	1.11±1.19	0.484		
FAB4	17.54±2.47	16.60±2.36	15.82±2.40	0.003	1 vs. 3	0.206
FAB4_H	0.11±0.46	0.09±0.34	0.26±0.54	0.093		0.481
FAB4_S	0.61±1.06	0.61±0.90	0.49±0.62	0.474		
FAB4_A	0.72±1.07	1.07±1.03	1.55±1.19	0.004	1 vs. 3, 2 vs. 3	0.733
FAB4_F	0.21±0.67	0.55±1.06	0.51±0.92	0.045		0.099
FAB4_N	0.89±1.02	1.08±1.06	1.40±1.07	0.167	1 vs. 3	
FAB5	14.94±4.39	12.77±4.30	11.48±4.31	0.004	1 vs. 2, 1 vs. 3	0.009
FAB5_H	0.55±1.00	0.85±1.11	1.00±1.29	0.286		
FAB5_S	1.85±1.21	2.12±1.09	2.08±1.14	0.432		
FAB5_A	1.01±1.11	1.51±1.14	1.98±1.14	0.003	1 vs. 2 vs. 3	0.528
FAB5_F	0.58±0.97	1.04±1.17	1.37±1.17	0.019	1 vs. 2, 1 vs. 3	0.013
FAB5_N	1.01±1.18	1.67±1.42	2.09±1.34	0.001	1 vs. 2, 1 vs. 3	0.031

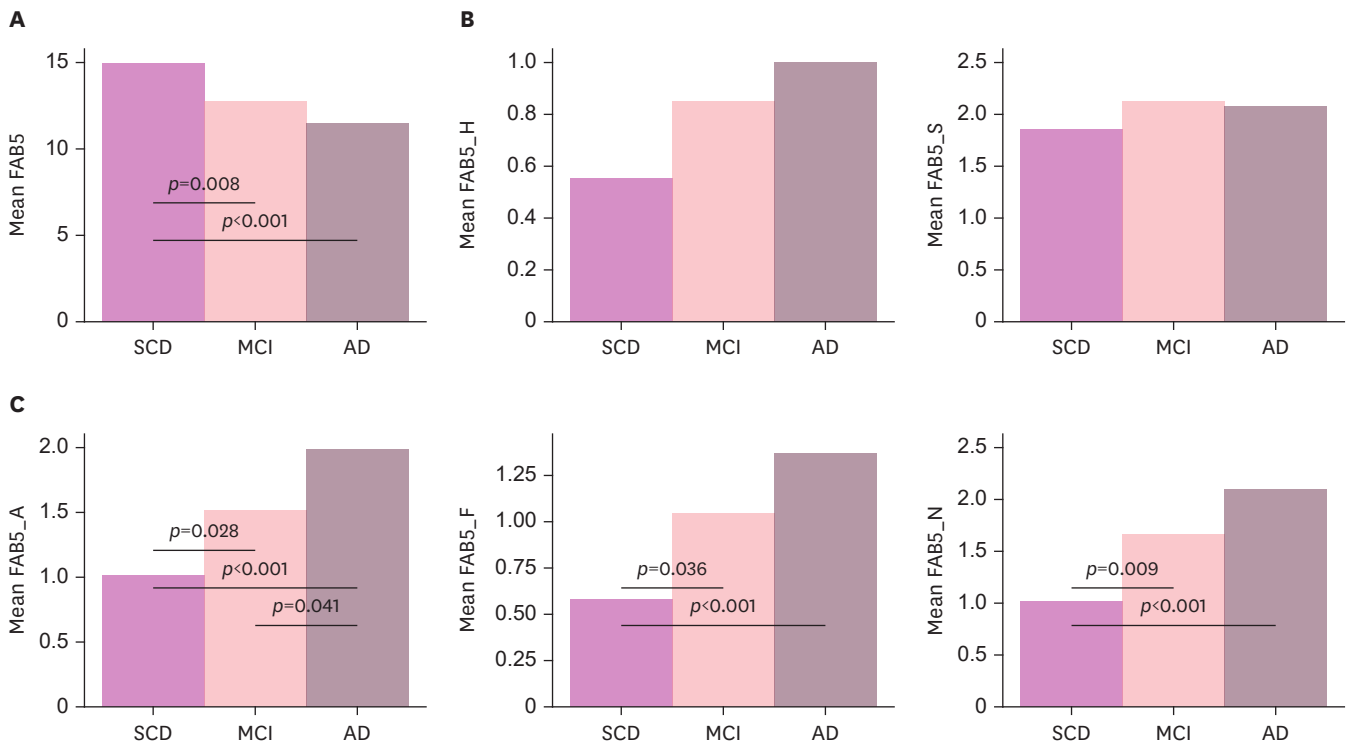
K-FAB includes subtest 1 (facial identity discrimination) and subtest 2 (facial affect discrimination), subtest 3 (facial affect naming), subtest 4 (facial affect selection), and subtest 5 (facial affect matching). Five different emotions (happiness [H], sadness [S], anger [A], fear [F], and neutral [N]) were used across these subtests.

K-FAB: Korean version of the Florida Facial Affect Battery, SCD: subjective cognitive decline, MCI: mild cognitive impairment, AD: Alzheimer’s dementia.

Values are presented as means ± standard deviations and analyses were performed using the analysis of covariance, \*adjusted for age and sex and †adjusted for age, sex, and MMSE score.

### Correlation of FAB with cognitive impairment

As the age increased (with subtest 1,  $r=-0.329$ ,  $p<0.001$ ; with subtest 2,  $r=-0.339$ ,  $p<0.001$ ; with subtest 3,  $r=-0.378$ ,  $p<0.001$ ; subtest 4,  $r=-0.255$ ,  $p<0.001$ ; subtest 5,  $r=-0.429$ ,  $p<0.001$ ) and educational attainment decreased (with subtest 1,  $r=0.178$ ,  $p=0.009$ ; with subtest 2,  $r=0.306$ ,  $p<0.001$ ; with subtest 3,  $r=0.229$ ,  $p=0.001$ ; subtest 4,  $r=0.369$ ,  $p<0.001$ ; subtest 5,  $r=0.392$ ,  $p<0.001$ ), facial emotion recognition impairments were observed.

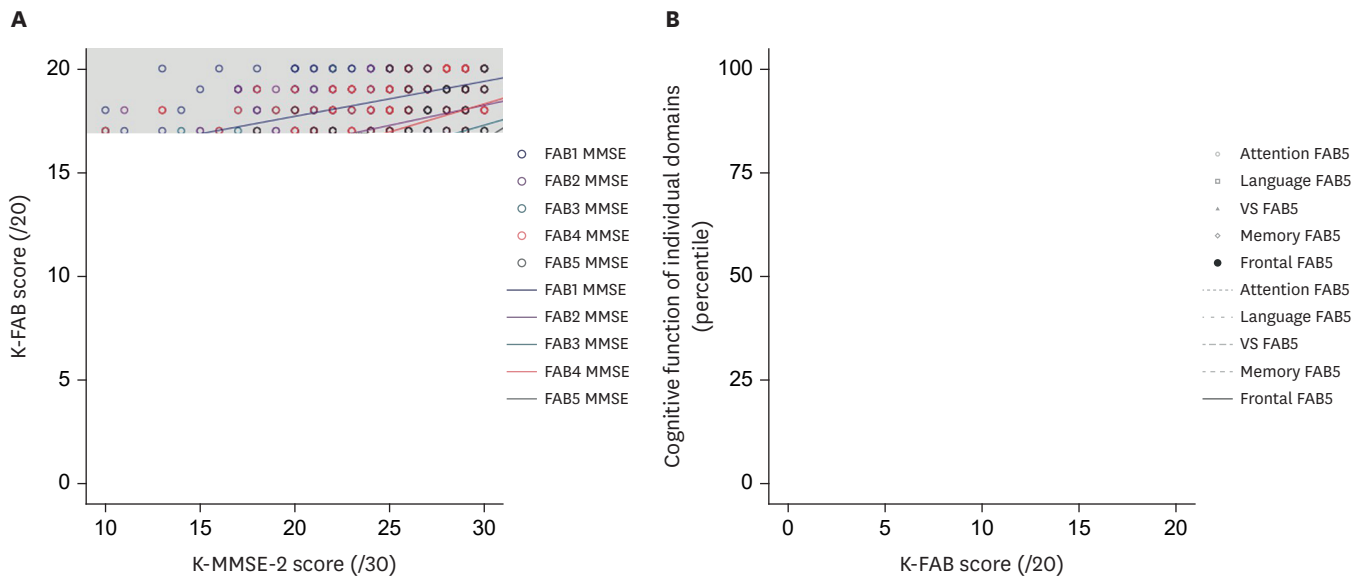


**Fig. 1.** The K-FAB scores in 3 groups. The FAB subtest 5 (facial affect matching) total score (A) and in contrast to happy and sad emotions (B), especially angry, fearful and neutral emotions (C) of the FAB subtest 5 showed a significant difference among the 3 groups, SCD, MCI, and mild AD. The *p*-values indicate significant differences after Bonferroni correction for multiple comparisons, as *post hoc* tests. K-FAB: Korean version of the Florida Facial Affect Battery, FAB5: K-FAB subtest 5, SCD: subjective cognitive decline, MCI: mild cognitive impairment, AD: Alzheimer’s dementia, H: happy, S: sad, A: angry, F: fearful, N: neutral.

After adjustments for age and education, partial correlation analysis showed that K-FAB tests correlated with the K-MMSE-2 score. The K-MMSE-2 score correlated with subtest 1 ( $r=0.237$ ,  $p=0.002$ ), subtest 2 ( $r=0.280$ ,  $p<0.01$ ), subtest 3 ( $r=0.363$ ,  $p<0.001$ ), subtest 4 ( $r=0.363$ ,  $p<0.001$ ), and subtest 5 ( $r=0.507$ ,  $p<0.001$ ). Linear regression analysis showed significant relationships of subtests of K-FAB with K-MMSE-2, subtest 1 (unstandardized coefficients  $B=0.333$ , standardized coefficients  $\beta=0.147$ ,  $p=0.015$ ), subtest 4 (unstandardized coefficients  $B=0.283$ , standardized coefficients  $\beta=0.154$ ,  $p=0.039$ ), and subtest 5 (unstandardized coefficients  $B=0.351$ , standardized coefficients  $\beta=0.349$ ,  $p<0.001$ ): constant 2.567 and R square 0.408. Only in SCD subjects, who had no objective impairments of cognition, frontal executive dysfunction among cognitive subdomains in SNSB-II was correlated with the K-FAB subtest 5 ( $r=0.338$ ,  $p=0.009$ ) and K-FAB subtest 2 ( $r=0.274$ ,  $p=0.034$ ). Otherwise, memory did not correlate with K-FAB tests, although visuospatial function was correlated with the K-FAB subtest 5 ( $r=0.324$ ,  $p=0.012$ ) (**Fig. 2**). Linear regression analysis showed a significant relationship between the K-FAB subtest 5 and frontal executive domain of SNSB-II (unstandardized coefficients  $B=0.044$ , standardized coefficients  $\beta=0.366$ ,  $p=0.007$ ); constant 27.636 and R square 0.162.

## DISCUSSION

In this study, we explored the capacity for facial emotion recognition among individuals diagnosed with SCD, MCI, and mild AD. Our findings demonstrated a significant impairment in the recognition of anger and fearful emotions in individuals with MCI and mild AD when



**Fig. 2.** Partial correlation analyses of K-FAB and K-MMSE-2, and of K-FAB and cognitive domains. (A) The K-MMSE-2 score correlated with subtest 1 ( $r=0.237, p=0.002$ ), subtest 2 ( $r=0.280, p<0.01$ ), subtest 3 ( $r=0.363, p<0.001$ ), subtest 4 ( $r=0.363, p<0.001$ ), and subtest 5 ( $r=0.507, p<0.001$ ), adjusted for age and education. Linear regression analysis showed significant relationships of subtests of K-FAB to K-MMSE-2, subtest 1 (constant  $B=0.333$ , standardized coefficients  $\beta=0.147, r^2=0.394, p=0.015$ ), subtest 4 (constant  $B=0.283$ , standardized coefficients  $\beta=0.154, r^2=0.394, p=0.039$ ), and subtest 5 (constant  $B=0.351$ , standardized coefficients  $\beta=0.349, r^2=0.394, p<0.001$ ). (B) Only in SCD subjects, among the cognitive subdomains in SNSB-II, frontal executive dysfunction was correlated with the K-FAB subtest 5 ( $r=0.338, p=0.009$ ). Other cognitive domains did not show significant associations. Linear regression analysis showed a significant relationship between the K-FAB subtest 5 and frontal executive domain of SNSB-II (constant  $B=0.044$ , standardized coefficients  $\beta=0.366, r^2=0.087, p=0.007$ ). K-FAB: Korean version of Florida Facial Affect Battery, K-MMSE-2, Korean Mini-Mental State Examination, 2nd edition, SCD: subjective cognitive decline, SNSB-II: Seoul Neuropsychological Screening Battery, 2nd edition.

compared to those with SCD. It is worth highlighting that even after adjusting for MMSE scores, differences in fear emotion recognition persisted across the 3 groups. Additionally, our analysis revealed that deficits in facial emotion recognition were associated with advancing age and lower levels of education. Furthermore, we observed a correlation between impaired facial emotion recognition and overall cognitive decline, with a particular emphasis on frontal executive dysfunction.

These findings indicate potential progression of facial emotion recognition impairment in individuals with dementia, beginning with a difficulty in recognizing fearful facial expressions during the MCI phase and subsequently extending to those with mild AD. This progression is likely to be a consequence of the documented degeneration of brain structures responsible for emotion processing, including the amygdala and other limbic and paralimbic structures.<sup>28,29</sup> While the amygdala has been found to respond to various facial emotion expressions,<sup>29</sup> it appears to play a crucial role in perceiving fear,<sup>30,31</sup> particularly in recognizing ambiguous or subliminal fearful cues.<sup>32</sup> Importantly, deterioration of the amygdala can account for a mild deficit in recognizing fearful expressions and the early onset of memory problems in MCI patients. This is due to connections of the amygdala with the prefrontal cortex and the hippocampal complex, which together form the neural network for emotional memory.<sup>33,34</sup> Indeed, the amygdala is well known for its role in consolidating the memory of fearful cues,<sup>35</sup> which aligns with studies supporting the cholinergic model in Alzheimer's disease.<sup>36</sup> It is noteworthy that the amygdala receives a significant cholinergic projection from the nucleus basalis of Meynert, and the acetylcholinesterase activity in the amygdala is notably reduced in Alzheimer's disease patients, with this reduction being positively correlated with the extent of global cognitive decline.<sup>37</sup>

The amygdala is a component of an intricate network that links the neocortex, hypothalamus, thalamus, basal forebrain, and brainstem, enabling bidirectional communication between the cortex and the autonomic nervous system.<sup>38</sup> Damage to the amygdala, in particular, has been associated with the loss of an autonomic fear response to aversive events,<sup>39</sup> as well as difficulties in recognizing fear and other negative emotions conveyed through facial expressions<sup>40,41</sup> and vocal cues.<sup>42</sup> Researchers have previously assigned distinct brain structures to different emotions, designating the amygdala for processing fear, the insula for processing disgust, and the prefrontal cortex for processing anger and highly arousing emotions. Nevertheless, whether there are specialized brain systems for mediating various types of emotions remains a subject of ongoing debate. It appears that certain structures may play a role in attributes relevant to specific emotions rather than the emotions themselves. As such, the amygdala may not be exclusively responsible for perceiving fear but might have a pivotal role in triggering the orienting response to novel and potentially stimulating events.<sup>43</sup>

Although research studies have consistently indicated difficulties in recognizing both positive and negative emotions, it is noteworthy that positive emotions, particularly happiness, tend to be affected to a lesser extent.<sup>44-47</sup> This difference may be due to the asymmetry in the representation of negative and positive emotional categories, which can be attributed to the conventional approach to assessing emotion perception. Most test batteries focus on six basic emotions, with four being negative (sadness, fear, anger, and disgust) and only two being positive (happy and surprised). The recognition of negative emotions typically requires more subtle discrimination compared to the recognition of positive emotions. Among them, 'happy' is often the most readily recognizable.<sup>48</sup>

The amygdala is widely recognized as a crucial component of the “social brain”<sup>8</sup> due to its role in modulating the neural system responsible for social cognition. Social cognition, in turn, underpins our capacity to interpret nonverbal communication, including facial expressions of emotions<sup>49</sup> and respond to emotional cues with appropriate interpersonal behaviors.<sup>8</sup> Thus, we interpret the result that the facial emotion recognition deficit in MCI patients is specific to anger and fear by hypothesizing that mild emotions are subtler and more challenging to recognize, requiring higher social cognition abilities than high-intensity emotions. However, low-intensity facial expressions are more frequently encountered in daily life. As a result, this type of impairment can have practical consequences in social interactions during the MCI phase, potentially affecting nonverbal communication, interpersonal relationships, and, consequently, the quality of life. Future studies should aim to confirm this hypothesis.

This study has some limitations. First, in terms of social cognition, we only examined social perception using the K-FAB. Additional assessments, including the theory of mind and empathy, could enrich the information on social cognition in elderly people and neurodegenerative disease.<sup>50</sup> Next, it is important to note that our study had a cross-sectional design, and longitudinal studies should be conducted to elucidate the progression and predictors of facial emotion recognition deficits in individuals, ranging from normal elderly individuals to those with SCD, MCI, and AD. Considering these factors, future research should strive to better understand the deficits in emotional processing in SCD, MCI, and AD. Detecting facial emotion recognition impairment early in the pre-dementia stage of Alzheimer’s disease may have clinical significance, as it could enable preventive interventions aimed at mitigating the decline in cognitive performance, interpersonal behavior, and social abilities, thereby improving the quality of life of both patients and caregivers.

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