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Greater regional brain atrophy rate in healthy elderly subjects with a history of cigarette smoking

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Abstract

Background: Little is known about the effects of cigarette smoking on longitudinal brain morphological changes in the elderly. This study investigated the effects of a history of cigarette smoking on changes in regional brain volumes over 2 years in healthy, cognitively intact elderly individuals. We predicted that individuals with a history of cigarette smoking, compared with never smokers, demonstrate greater rate of atrophy over 2 years in regions that manifest morphological abnormalities in the early stages of Alzheimer's disease (AD), as well as in the extended brain reward/executive oversight system (BREOS), which is implicated in the development and maintenance of substance use disorders.

Methods: Participants were healthy, cognitively normal elderly control subjects (75.9 ± 4.8 years of age) with any lifetime history of cigarette smoking (n = 68) or no history of smoking (n = 118). Data were obtained through the Alzheimer Disease Neuroimaging Initiative from 2005 to 2010. Participants completed four magnetic resonance scans over 2 years. A standardized protocol using high-resolution three-dimensional T1-weighted sequences at 1.5 T was used for structural imaging and regional brain volumetric analyses.

Results: Smokers demonstrated a significantly greater atrophy rate over 2 years than nonsmokers in multiple brain regions associated with the early stages of AD, as well as in the BREOS system. Groups did not differ on the rate of global cortical atrophy.

Conclusions: A history of cigarette smoking in this healthy elderly cohort was associated with decreased structural integrity of multiple brain regions, which manifested as a greater rate of atrophy over 2 years in regions specifically affected by incipient AD as well as chronic substance abuse.
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Keywords: Alzheimer's disease; MRI; Brain volumes; Brain reward system; Cigarette smoking; Brain atrophy

1. Introduction

The general health consequences associated with chronic cigarette smoking are well documented [1]. However, little research has specifically attended to the effects of smoking on human neurobiology over time [2]. Early computed tomography studies demonstrated that chronic cigarette smoking was associated with increased global brain atrophy with advancing age [3–6]. More recent cross-sectional magnetic resonance imaging (MRI) studies found that chronic cigarette smoking in nondemented middle-aged and elderly adults is associated with regionally specific abnormalities in

Data used in preparation of this article were obtained from the Alzheimer Disease Neuroimaging Initiative (ADNI) database (www.loni.ucla.edu/ADNI). As such, the investigators within the ADNI contributed to the design and implementation of ADNI and/or provided data but did not participate in analysis or writing of this report. A complete listing of ADNI investigators can be found at http://adni.loni.ucla.edu/wpcontent/uploads/how_to_apply/ADNI_Authorship_List.pdf.

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brain morphology and biochemistry. Specifically, smokers, as compared with nonsmokers, demonstrated smaller gray matter (GM) volumes and/or lower GM densities in the anterior cingulate cortex (ACC), dorsolateral cortex, orbitofrontal cortex (OFC), parahippocampal gyrus, and precuneus; smaller volumes in the left dorsal ACC; and lower GM densities in the right cerebellum [7–10]. Chronic smokers demonstrated lower cortical thickness in the medial OFC [11] and lower *N*-acetylaspartate concentration (a surrogate marker of neuronal integrity [12,13]) in the left hippocampus relative to nonsmokers [14].

Taken together, previous neuroimaging studies suggest that chronic cigarette smoking is associated with neurobiological abnormalities in regions that also exhibit morphological abnormalities in the incipient stages of Alzheimer's disease (AD) [15] (e.g., hippocampus, entorhinal cortex, parahippocampal gyrus, posterior cingulate region), as well as in components of the extended brain reward/executive oversight system (BREOS) in observed in substance use disorders [16]. Neurobiological abnormalities in the BREOS are implicated as major contributors to the development and persistence of all forms of addiction, including nicotine dependence [17]. Cortical components of the BREOS include the dorsolateral prefrontal cortex, OFC, insula, ACC, as well as the amygdala, hippocampus, and other regions/nuclei in the dorsal and ventral striata and basal forebrain [18–20].

To date, no study has specifically investigated the longitudinal effects of a history of cigarette smoking on brain volumes in regions that manifest abnormalities in the early stages of AD or in the BREOS in elderly adults free of clinically significant cardiovascular, cerebrovascular, and neurodegenerative diseases. Consequently, it is unknown whether a history of cigarette smoking is associated with regionally specific volume changes in healthy elderly individuals over time. The primary objective of this study was to compare volume changes in elderly individuals with a history of cigarette smoking (smokers) with those with no lifetime history of smoking (nonsmokers) for a period of 2 years with serial high-resolution MRI. We focused on regions that demonstrate morphological abnormalities during the incipient stages of AD as well as on BREOS regions that show morphological changes in those with substance use disorders. We predicted smokers, compared with nonsmokers, demonstrate significantly greater rate of atrophy over 2 years in (1) regions associated with morphological abnormalities in the early stages of AD, including the hippocampus, entorhinal cortex, parahippocampal gyrus, posterior cingulate region (posterior cingulate gyrus and isthmus), precuneus, and fusiform gyrus, and (2) in components of the BREOS, including the ACC (rostral and caudal divisions), dorsolateral prefrontal cortex (inferior frontal gyri, rostral and caudal middle frontal gyri, superior frontal gyri), insula, orbitofrontal region (medial and lateral regions, pars orbitalis), and amygdala.

2. Methods

2.1. Participants

The cohort examined consisted of a subsample of 186 participants (75.9 ± 4.8 years of age at baseline examination) who served as control subjects in the Alzheimer Disease Neuroimaging Initiative (ADNI) project. The following is a standard description of the ADNI project (see http://adni.loni.ucla.edu/about/): *ADNI is a multisite study supported by the National Institute on Aging (NIA), the National Institute of Biomedical Imaging and Bioengineering (NIBIB), the Food and Drug Administration (FDA), private pharmaceutical companies, and non-profit organizations, as a 5-year public-private partnership [21,22]. The primary goal of ADNI has been to test whether serial magnetic resonance imaging (MRI), positron emission tomography (PET), other biological markers and clinical and neuropsychological assessment can be combined to measure the progression of mild cognitive impairment (MCI) and early Alzheimer disease (AD). Determination of sensitive and specific markers of very early AD progression is intended to aid researchers and clinicians to develop new treatments and monitor their effectiveness, as well as lessen the time and cost of clinical trials. The initial goal of ADNI was to recruit 800 adults from approximately 58 sites in the United States and Canada, ages 55 to 90, to participate in the research –approximately 200 cognitively normal older individuals to be followed for 3 years, 400 people with MCI to be followed for 3 years and 200 people with early AD to be followed for 2 years.* Participants in this study were recruited from multiple sites in the United States from 2005 to 2010. Written informed consent was obtained from all participants before initiation of procedures. The study was conducted according to the Declaration of Helsinki, and U.S. 21 CFR Part 50—Protection of Human Subjects—and Part 56—Institutional Review Boards [23,24]. Participants who reported no tobacco use during lifetime were assigned to the nonsmoker group (n = 118), whereas those who reported any history of cigarette smoking during their lifetime were designated as smokers (n = 68). Three of the smokers also smoked pipes or cigars. Basic information regarding duration of smoking over lifetime and duration of abstinence from cigarettes was available for 58 smokers. At the time of the baseline study, 7.4% of the smokers were actively smoking. Table 1 presents demographic information on smokers and nonsmokers.

2.2. MRI image acquisition and processing

2.2.1. Acquisition

Participants completed four MR scanning sessions (discounting missed visits and subjects who had not yet reached 2 years) at baseline and 6, 12, and 24 months after baseline at multiple ADNI sites. A standardized protocol used high-resolution three-dimensional (3-D) T1-weighted sequences

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