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Smoking cessation behaviors three months following acute insular damage from stroke☆

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HIGHLIGHTS

- Insular damage is associated with increased odds of 3-month continuous abstinence.
- Insular damage increases the odds of complete cessation for any nicotine product.
- Time to smoking relapse is higher on average in those with insular lesions.
- Those abstinent by 3 months with insular damage did not smoke at all post-discharge.
- The insula may be a target for long-term smoking cessation therapies.

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ABSTRACT

Background: Recent evidence suggests that the insular cortex may play an important role in cognitive and emotional processes that facilitate drug use but it is unclear whether changes to the insula would result in sustained abstinence. To better understand the role of the insula in maintaining abstinence, we examined quitting patterns in smokers with acute damage to their insula relative to other regions.
Design: Prospective cohort study with 3 month follow-up, beginning June 2013 and ending May 2014.
Setting: Three acute care hospitals in Rochester, NY.
Participants: One-hundred-fifty-six current smokers hospitalized for acute ischemic stroke; 38 with insular infarctions and 118 with non-insular infarctions, assessed by 3 neuroradiologists.
Measurements: Self-reported smoking status (seven-day point prevalence and continuous abstinence), complete abstinence from any nicotine product, and disruption of smoking addiction (defined by criteria on smoking status, difficulty of quitting, and urge) were assessed at three months post-stroke. Time to relapse (in days) after discharge was also assessed.
Results: Insular damage was associated with increased odds of three-month continuous abstinence (OR = 3.71, 95% CI: 1.59, 8.65) and complete cessation from any nicotine product (OR = 2.72, 95% CI: 1.19, 6.22). Average time to relapse was longer in the insular-damaged group (17.50 days, SD = 19.82) relative to non-insular damage (10.42 days, SD = 18.49). Among quitters, insular damage was also associated with higher relative odds of experiencing a disruption of addiction compared to non-insular damage (adjusted OR = 5.60, 95% CI: 1.52, 20.56).
Conclusions: These findings support the potential role of the insular cortex in maintaining smoking and nicotine abstinence. Further research is needed to establish whether the insula may be a novel target for smoking cessation interventions.

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1. Introduction

Tobacco dependence is regarded as a chronic disease in which long-term management strategies are needed to address the strong neurophysiological reward response, physical withdrawal symptoms, and the psychological response to environmental, social, and cultural cues associated with its use (Fiore et al., 2008). Although many smokers are aware of the increased health risks and have a desire to quit, only one third make an attempt to quit annually (Rigotti, 2002). Even with current pharmacotherapies for tobacco dependence, 66.8 to 81.0% of smokers motivated to quit relapse by six months (Fiore et al., 2008; Rigotti, 2002). Given the high prevalence of relapse, it is important to further evaluate potential mechanisms responsible for this addictive behavior so that successful strategies for cessation intervention can be developed.

The craving for nicotine has been suggested to originate from long-term adaptations within specific neural systems that promote escalating drug use, difficulty quitting, and relapse (Naqvi & Bechara, 2010). Mesocorticolimbic regions of the brain, including the ventral tegmental area (VTA), amygdala, nucleus accumbens (NAc), prefrontal cortex (PFC), and hippocampus have observed molecular adaptations when exposed to addictive drugs in preclinical studies (Abdolahi, Acosta, Breslin, Hemby, & Lynch, 2010; Naqvi, Rudrauf, Damasio, & Bechara, 2007) and have been the primary target for smoking cessation pharmacotherapies such as bupropion (Mansvelder, Fagen, Chang, Mitchum, & McGehee, 2007) and varenicline (Nocente et al., 2013). Recently, the insular cortex – the cerebral cortex beneath the sylvian fissure surrounded by the temporal, frontal, and parietal opercula – has been of particular interest due to the potential role it plays in conscious urges, notably for cravings associated with addictive drugs (Naqvi & Bechara, 2010). The central and peripheral effects of nicotine are implicated in conscious pleasure induced by cigarette use. Smoking-related cues are believed to result in interconnections from the amygdala and orbitofrontal cortex/ventromedial PFC triggering a representation of the euphoria produced by nicotine in the insula and consequently insular projections to the NAc core, thus motivating nicotine dependent behaviors (Naqvi & Bechara, 2010).

To date, only two experimental animal studies have examined the effects of insular cortex inactivation on drug seeking and have demonstrated the integrity of the insula to facilitate motivation to take methamphetamine (Contreras, Ceric, & Torrealba, 2007) and nicotine (Forget, Pushparaj, & Le Foll, 2010). Human studies of stroke-induced lesions have also shown insular damage to be associated with higher odds of cessation (Gaznick, Tranel, McNutt, & Bechara, 2014; Naqvi et al., 2007; Suner-Soler et al., 2012), although one study obtained a null finding (Bienkowski, Zatorski, Baranowska, Ryglewicz, & Sienkiewicz-Jarosz, 2010). These studies, however, had relatively small sample sizes, only examined cessation of cigarette smoking and did not address subsequent use of other nicotine products such as nicotine replacement therapies (NRT) and other forms of tobacco.

We previously demonstrated that acute insular damage by ischemic stroke among current cigarette smokers resulted in reduced and less severe withdrawal symptoms during hospitalization, a period of forced abstinence (Abdolahi et al., in press). Although this may be the mechanism through which cessation occurs, it is important to characterize quitting patterns in these individuals to better understand the durability of the insula in maintaining abstinence. We hypothesized that, among current cigarette smokers admitted for acute ischemic stroke, those with damage to their insular cortex would be more likely to quit by three months post-stroke and experience complete cessation from all forms of nicotine, compared to smokers with non-insular damage. Three-month follow-up was chosen as a clinically-relevant timeframe in which the neuroadaptations that underlie addiction last after quitting (Dawkins, Powell, Pickering, Powell, & West, 2009). To account for the possibility that insular function may be restored with time, particularly for smaller lesions, we also hypothesized that smokers with insular damage would relapse later – after discharge – than those with non-insular damage. In an effort to confirm the findings reported by Naqvi et al. (2007), we also hypothesized that, among the quitters, those with insular damage would be more likely to experience a "disruption of addiction" relative to individuals with non-insular strokes.

2. Materials and methods

2.1. Study population

The Measuring the Impact on Nicotine Dependence after Stroke (MINDS) study population consisted of adults aged 18 years and older who were admitted to one of three acute care hospitals in Rochester, NY with a diagnosis of acute ischemic stroke (ICD-9 code 434.91) and were active cigarette smokers at the time of stroke onset. Patients were eligible to participate if they met all of the following inclusion criteria: (1) smoked at least one cigarette per day during the month prior to their stroke and at least 100 in their lifetime; (2) were able to understand and speak the English language; (3) were stable enough, as determined by a nurse practitioner, to give autonomous informed consent and respond to the survey questions verbally or on paper; and (4) were willing to respond to the follow-up survey three months after hospital admission. Participants were excluded from follow-up evaluation if they had a subsequent stroke of any kind or were maintained in an environment where access to smoking was restricted.

Fig. 1 illustrates a flow diagram of recruitment and follow-up figures. A total of 470 ever smokers with acute ischemic stroke were identified between June 2013 and February 2014. Among the 204 (43.4%) who met the definition of a current smoker, 48 (23.5%) were excluded for not meeting the eligibility criteria; thus, 156 (76.5%) patients were enrolled into the study. Due to exclusions and losses to follow-up, 134 (85.9%) enrolled participants completed the three month follow-up assessment.

All participants signed informed consent forms. The institutional review boards at all participating institutions approved the study protocols and procedures.

2.2. Exposure assessment

Acute infarctions to the insular cortex, the primary exposure region, and the aforementioned mesocorticolimbic structures were characterized by neuroradiologists (H.Z.W., E.M.S., B.E.S.) using standard of care computed tomography (CT) and magnetic resonance imaging (MRI) techniques in all participants. Participants with cerebral infarctions not in the insular cortex were considered unexposed. The neuroradiologists were blinded to information other than what is commonly clinically available. If evidence from CT and MRI were conflicting, findings from the MRI were more heavily taken into account than CT. In situations where MRI was contraindicated, only CT imaging was used. On MRI, diffusion weighted imaging was used to evaluate lesion location if completed within a few days from the stroke. If follow-up MRI scans were available, the fluid attenuated inversion recovery sequence was used to verify the initial MRI findings. The NAc and VTA were the two most difficult regions to evaluate as they are not routinely evaluated in practice and the standard MRI uses 5 mm slices which may be too thick to detect lesions in these regions (Sethi et al., 2012).

Exposure data were collected and managed using Research Electronic Data Capture (REDCap) (Harris et al., 2009) tools hosted at the University of Rochester. Lesions in 80 (51.3%) participants were identified using only MRI, 22 (14.1%) using only CT, and 54 (34.6%) using both MRI and CT. All participants were confirmed to have acute to subacute infarctions.

2.3. Ascertainment of end points

Participants were evaluated three months after the date of stroke – as a clinically relevant timeframe for neuroadaptations to occur after

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