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Altered patterns of brain activity during transient anger among young males with alcohol use disorders: A preliminary study

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Abstract

The aim of the study was to investigate the neural substrates associated with processing anger among young males with alcohol use disorders (AUDs) using functional magnetic resonance imaging (fMRI). Eighteen individuals with AUD and 15 demographically similar non-abusers participated in the study. Participants were scanned on their brain functioning while they viewed an audio-visual film clip that was previously designed specifically to induce anger emotion, followed by anpsychological assessment. Greater brain activities were detected in the left inferior frontal gyrus (IFG) and dorsal anterior cingulate cortex (dACC) among subjects with AUD compared to the controls during the exposure to anger-provoking stimuli. Despite the same level of subjective anger during anger induction, the greater activations both in the IFG and dACC regions may suggestthat individuals with AUD have a greater propensity to undergo cognitive control and self-regulation while experiencing anger.

Key words: fMRI; Alcohol use disorders; Anger; Brain activation

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1. Introduction

It is well known and widely accepted that alcoholics are characterized abnormal emotional processing. For example, alcoholics revealed the hyperactivity to negative stimuli and hypoactivity to positive stimuli (Deither & Blairy, 2012). In another study, alcoholics showed abnormal emotional reactivity displaying exceedingly high or low emotional response and less heart rate changes (Kornreich et al., 1998). These emotional impairments appear to influence on the difficulties in alcoholics' interpersonal relationships (Uekermann & Daum, 2008).

Also with their emotional deficits, cerebral impairment is one of the well-known characteristic among alcoholics (Oscar-Berman & Marinkovic, 2003). Findings from a number of neuroimaging studies indicate that deficits in emotional processing among alcoholics is associated with altered brain activity in the regions such as the anterior cingulate cortex (ACC; Salloum et al., 2007), amygdala and hippocampus (Marvinkovic et al., 2009), or parahipocampal gyrus and hippocampus (Gilman & Hommer, 2008). Such deficit in neural circuitry may underlie the emotional impairment.

However, to my knowledge, brain function among alcoholics during emotional experience has never been studied. The above studies on alcoholics' emotional processing are related emotion perception regarding emotional facial expression or static pictures such as International Affective Picture System (IAPS). In this study, we are to examine the behavioral and neural responses during transient anger among alcoholics. We chose anger because anger is the most commonly expressed and ill successfully handled among alcoholics (Han et al., 1996), not to mention experiencing of higher anger level than non-alcoholics (Potter-Efron & Potter-Efron, 1991; Walfish et al., 1990). For example, the average State-Trait Anger expression Inventory (STAXI; Spielberger et al., 1988) score of male AUD patients were higher than that of the average population (Potter-Efron & Potter-Efron, 1991; Tivis et al., 1998).

In sum, we hypothesized that alcoholics may show different levels of subjective experience of anger or altered brain activity in the orbitofrontal cortex (OFC), ACC, and the insular regions that are implicated in anger experience or regulation (Dougherty et al., 1999; Denson et al., 2009; Linquist et al., in press). To pursue the study purpose, we measured neural activation during a mood induction task which consists of neutral and anger film extracts validated in our previous study to induce temporary anger.

2. Method

2.1. Participants

The sample of this study composed of thirty-three right-handed college students aged between 21 and 35 at the time of study entry. Eighteen subjects in the Alcohol Use Disorder (AUD) group were diagnosed with AUD based on DSM-IV (American Psychiatric Association, 1994) criteria for alcohol abuse or dependence. Demographically identical (i.e., age range, education level, and smoking status) fifteen social drinkers who did not meet the DSV-IV AUD diagnosis composed the normal control (or comparison) group. Participant characteristics, including demographic profiles are provided in Table 1.

Various screening tools were adopted in this study to identify individuals with or without AUD. Those included the Korean version of Alcohol Use Disorders Identification Test (AUDIT-K) (Lee, 2000), National Alcoholism Screening Test (NAST) (Kim et al., 1991), and a Structured Clinical Interview based on DSM-IV (Hahn et al., 2000). Scores greater than 15 points on the AUDIT-K is considered sufficient to meet the DSM-IV

criteria for AUDs (Park et al., 2000). A positive response of more than one item on the NAST is considered sufficient to meet the DSM-IV criteria for alcohol use disorder (Park et al., 2000). Although subjects with AUD reported no incidences of receiving any type of alcohol abuse treatment for their alcohol problems within the past 12 months at the time of the study. Both the AUD group and the normal control group reported no lifetime use of any type of drugs. Detailed alcohol and drug use characteristics of the study participants are presented in Table 1.

None of the study participants experienced any history of psychiatric or co-occurring mental health disorders, as prospective study participants were initially screened for any history of a serious head injury, serious medical problem(s), neurological disorder(s), Axis I psychiatric disorders other than AUD, or current use of psychotropic medication using self-reports and mental health history.

Table 1. Participant characteristics

	AUD group [n = 18; % or Mean (SD)]	Control group [n = 15; % or Mean (SD)]
Male	100%	100%
Age (years)	23.90 (2.39)	23.70 (0.95)
Drug use in a lifetime	0%	0%
Number of cigarettes smokedper day	1.55 (3.21)	3.00 (6.75)
Number of drinking days per month	9.73 (2.97)**	1.50 (1.29)**
Number of drinks per drinking day	9.35 (1.04)**	3.50 (1.80)**
Alcohol withdrawal symptoms within (at least one) past 12 months	45 %	0 %
Number of blackouts in a month	1.64 (1.53)*	0*
AUDIT-K	25.36 (4.48)**	6.10 (1.10)**
NAST	3.91 (1.70)**	0 (0)**

**$p < 0.001$.* $p < 0.01$.

2.3. Procedures

Study participants were individually instructed on the experimental procedure prior to the experiment. Emotion provoking film clips from movies and websites that were excerpted and validated via our previous study were used in the current study. The audio-visual film clips were used for the purpose of inducing dynamic and realistic emotion and the validation and standardization procedures are described in Sohn et al.'s (2005) study in details.

Six basic emotions were included in the study. The standardized six stimuli were employed from the Sohn et al.'s (2005) study. The fMRI experimental task consisted of 6 emotion blocks (i.e., anger, fear, disgust, sadness, happiness, and surprise) that lasted 120-s per emotion. Each emotion block was preceded with the 30-s fixation block. In the 120-s emotion block, the block was composed of 30-s neutral condition and 90-s emotion condition. The neutral condition was chosen from the same audio-visual clips selected for each emotional stimuli so as to match the contents, color, and hue with the emotional condition. Total scanning session took approximately 15 minutes. As the focus was the anger emotion in the study, only anger condition was explored hereafter. The anger stimuli that was fMRI experimental task was the an audio-visual film clip where a male adult battering a bus driver.

After the scanning session, psychological assessment was administered; subjects were asked to label what emotion they had felt while viewing the clip among 11 disrecte emotions (i.e., happiness, sadness, anger, contempt, disgust, fear, surprise, bored interested, neutral, and other). They also were asked to rate the intensity of the emotion on a 7 point Likert scale (1: being least angry, and 7: being most angry) and report the part that they experience most intense emotion. The stimulus was created using an audio-visual software file which then were projected onto a screen using a mirror with

subjects each wearing a headphone set to experience both visual and acoustic stimuli. Subjects' written consent was obtained for this clinical study after the provision of a thorough explanation of the study purposes and demonstration of the procedures. The study was comprehensively reviewed and conducted in accordance with our Institutional Review Committee.

2.4. Imaging Parameters

Imaging was conducted on a 3.0 T whole-body ISOL Technology FORTE scanner (ISOL Technology, Korea) equipped with whole-body gradients and a quadrature head coil. Single-shot echo-planar fMRI scans were acquired in 35 continuous slices, parallel to the anterior commissure-posterior commissure line. The parameters for fMRI include the followings: the repetition time/echo time [TR/TE] were 3000/30 ms, respectively, flip angle 80, field of view 240 mm, matrix 64×64, slice thickness 4 mm, and in-plane resolution 3.75 mm. Three dummy scans from the beginning of the run were excluded to decrease the effect of non-steady state longitudinal magnetization.

2.5. Data analysis

As the focus was the anger emotion in the study, only anger condition was analyzed in the data analysis. In the behavioral data analysis, independent *t*-test was conducted by using SPSS 18.0 to examine the group difference for the level of subjective anger experience. For the fMRI data analysis, The 30-s emotional part that each participant experienced the most intensive anger feelings were selected based on self-reprot in the psychological assessment. As a results, 30-s neutral condition and 30-s anger condition were used in the fMRI data analysis.

The fMRI data were processed with SPM 2 (Wellcome Department of Cognitive Neurology, London, UK). At the beginning of preprocessing, all functional images were realigned with the images taken proximate to the anatomical study by using affine transformation routines implemented in SPM2. Motion correction was done by using Sinc interpolation. In order to remove artifacts resulted from cardio-respiratory and other cyclical influences, time series data were filtered with 240-s high-pass filter. In the next coregistration step, the realigned scans were coregistered to the participant's anatomical images obtained within a session. In the normalization step, the coregistered data were normalized to SPM2's template image that uses the space defined by the Montreal Neurologic Institute, which is similar to the Talairach and Tournoux's (1988) atlas. In the final step of preprocessing, with a 8-mm isotropic Gaussian kernel, the functional map was smoothened prior to statistical analysis.

In the step of postprocessing, by using the general linear model and the theory of Gaussian random fields implemented in SPM2. Statistical analysis was done individually and also with a group. Activated areas in the brain during anger condition compared to neutral condition were color-coded by *t*-score using the subtraction procedure. To test the effect of difference between two groups (i.e., the AUD group vs control group), the double subtraction method was utilized.

3. RESULTS

3.1. Psychological assessment

The mean (SD) average level of anger experienced was 5.22(1.96) for the AUD group and 5.47(1.64) for the control group. As intended, viewing the anger film excerpts evoked only anger emotion and no other mutually exclusive emotion. The independent *t*-test result

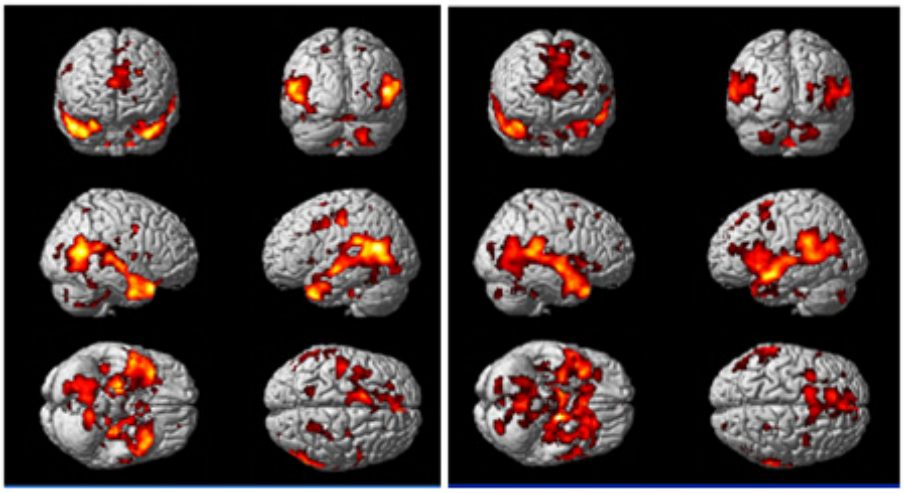
did not yield a statistically significant difference between two groups in the level of anger experienced in the scanner (t = -0.38, df = 31, p = 0.703).

3.2. Brain activation areas of each group

Figure 1 illustrates the brain areas that exhibited significant activities during anger emotion in the control and the AUD groups (uncorrected p <0.001, k (cluster size) = 20). In the control group, significant activations were observed in the bilateral middle temporal gyrus (Brodmann's areas; BA 39), left middle occipital gyrus (BA 37), bilateral superior temporal gyrus (BA 38), left inferior temporal gyrus (BA 20), bilateral parahippocampal gyrus, left middle frontal gyrus (BA 9), left superior frontal gyrus, bilateral Medial frontal gyrus (BA 6), bilateral posterior cingulate (BA 31/23), right claustrum, left postcentral gyrus (BA 3). In the AUD group, they were the bilateral middle temporal gyrus, left superior frontal gyrus (BA 6/8), right medial frontal gyrus, right cerebellar tonsil, left superior temporal gyrus (BA 38), right parahippocampal gyrus (BA 36), right fusiform gyrus, left middle frontal gyrus, left pyramis, left cerebellar tonsil, left cingulate gyrus (BA 24).

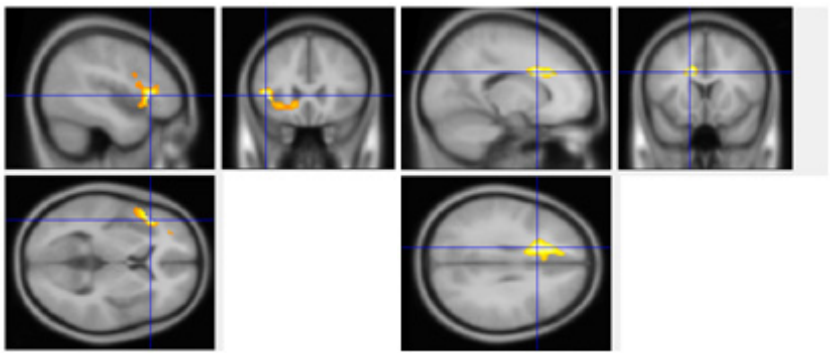
3.3. Group difference in brain activation

Figure 2 reveals the contrasting effects between the



Control group AUD group

Figure 1. Significant brain activation areas during anger condtion compared to neutral condtion (uncorrected p < 0.001, k (cluster size) = 20).



Yellow clusters each represents inferior frontal gyrus (BA 45) (left panel) and dorsal part of anterior cingulate gyrus (BA 24) (right panel).

Figure.2. Contrasting brain activations in the AUD group relative to control group during anger condtion compared to neutral condtion (uncorrected p < 0.001, k (cluster size) = 20).

Table 2. Talairach coordinates and *t*-values of activated brain areas

Region	Side	X	Y	Z	Brodmann's areas (BA)	*t* value
Anger condtion-neutral condtion comparison						
The control group						
Middle temporal gyrus	Left	-54	-60	16	-	4.76
Middle occipital gyrus	Left	-48	-68	4	BA 37	4.27
Superior temporal gyrus	Left	-44	14	-30	BA 38	4.75
Inferior temporal gyrus	Left	-50	-2	-30	BA 20	3.77
Parahippocampalgyrus	Right	22	-16	-16	-	4.73
Middle temporal gyrus	Right	50	-62	26	BA 39	4.47
Superior temporal gyrus	Right	38	20	-30	BA 38	4.43
Middle temporal gyrus	Right	50	-72	10	BA 39	4.29
Middle frontal gyrus	Left	-28	32	32	BA 9	4.29
Middle temporal gyrus	Left	-58	-14	-6	-	4.28
Superior temporal gyrus	Left	-58	-22	-2	-	3.62
Superior frontal gyrus	Left	-8	46	32	-	4.20
Medial frontal gyrus	Right	8	44	22	-	3.56
Parahippocampalgyrus	Left	-28	-20	-16	-	4.17
Posterior cingulate	Left	-6	-52	22	BA 31	4.12
Posterior cingulate	Right	6	-58	18	BA 23	3.44
Medial frontal gyrus	Left	-12	8	54	BA 6	3.56
Claustrum	Right	30	-4	14	-	3.54
Parahippocampalgyrus	Left	-16	-46	4	-	3.13
Middle frontal gyrus	Left	-36	6	40	-	3.00
Postcentral gyrus	Left	-44	-18	56	BA 3	3.00
Postcentral gyrus	Left	-46	-18	46	BA 3	2.94
The AUD group						
Middle temporal gyrus	Left	-48	0	-16	-	5.93
Middle temporal gyrus	Right	50	-38	2	-	5.77
Superior frontal gyrus	Left	-12	30	54	BA 6	6.45
Superior frontal gyrus	Left	-4	38	46	BA 8	4.87
Medial frontal gyrus	Right	10	50	18	-	3.91
Cerebellar tonsil	Right	30	-60	-32	-	3.75
Superior temporal gyrus	Left	-28	12	-24	BA 38	2.99
Superior temporal gyrus	Left	-36	16	-24	-	2.90
Parahippocampalgyrus	Right	40	-32	-20	BA 36	3.25
Fusiform gyrus	Right	44	-42	-16	-	2.91

Region	Side	X	Y	Z	Brodmann's areas (BA)	*t* value
Middle frontal gyrus	Left	-16	8	62	-	2.92
Pyramis	Left	-14	-68	-28	-	3.23
Cerebellar tonsil	Left	-2	-50	-42	-	2.75
Cingulate gyrus	Left	-16	-2	44	BA 24	2.78
The AUD group > The control group						
Inferior frontal gyrus	Left	-44	24	4	BA 45	3.80
Anterior cingulate gyrus	Left	-16	14	28	BA 24	3.08
Cingulate gyrus	Left	-14	28	28	BA 32	2.78

Brain activation comparisons between the anger condition and the neutral condition in each group and contrasting effects between two groups (i.e.,AUD group vs. control group) (uncorrected $p < 0.001$, k (cluster size) = 20).

control and the AUD groups using the double subtraction method (uncorrected $p < 0.001$, k (cluster size) = 20). Greater activation in the left inferior frontal gyrus (BA 45) and anterior cingulate gyrus (BA 24/32) were observed in the AUD group compared to the control group. There was no significantly activated region in the control group to support exhibition of any greater activation than the AUD group. The talairach coordinates and *t*-scores of activated brain areas are shown in Table 2.

4. Discussion

The aim of this study was to investigate the neural substrates associated with processing anger among young males with alcohol use disorders (AUDs) using functional magnetic resonance imaging (fMRI). Areas that were significantly activated in the normal controls in the study were consistent with the previous research outcome that support the findings on brain regions responsible for underlying anger processing in healthy men. They are the left superior temporal gyrus (BA 38) (Dougherty et al., 1999; Kimbrell et al., 1999), left middle temporal gyrus, left middle frontal gyrus (BA 9) (Denson et al., 2009; Dougherty et al., 1999; Kimbrell et al., 1999), left superior frontal gyrus, left middle occipital gyrus, left postcentral gyrus (BA 3) (Damasio et al., 2000). The medial frontal activation (BA 9), specifically, is suggested to be related to the general experience of emotion, as this region was also activated not only during anger state, but also happiness and sadness, pleasant and unpleasant emotions in healthy individuals (Drexler et al., 2000).

In this study, brain regions prominently found to be associated with anger processing was occipitotemporal cortex (OTC). The bilateral activations in the OTC were associated with an unpleasant emotion in a previous study by Lane et al. (1997). However, it seems that the changes in the OTC reflect the intensity, not the valence of emotion (Lane et al., 1997), as there is a relationship between arousal rating and occipito-temporal response as well as arousal rating predicts signal changes in the OTC (Wright et al., 2004). The OTC plays an important role in processing general affective arousal; viewing pictures of mutilation may cause OTC activation that reflects the high affective arousal ratings (Wright et al., 2004). Consistent with this notion, a possible explanation for showing a significant activation in the OTC in this study could be explained by the participants' affective arousal induced by the violent scene of the film clip.

Regarding the group comparison, the AUD and control participants did not differ in the intensity level of

experienced anger: However, functional abnormalities in the brain activity were found in the AUD group during transient anger. Specifically, the AUD group exhibited greater activation in the left inferior frontal gyrus (IFG; BA 45) and dorsal part of anterior cingulate gyrus (dACC; BA 24) compared to the controls during anger. The IFG is hypothesized to be involved response inhibition (Fu et al., 2008). The dACC is known to be responsible for effortful cognitive control or emotion regulation (Gasquoine, 2013), consistent with the notion that the region is a ‘‘neural alarm system’’ that associated with processing incongruent stimuli and goals (Eisenberger & Lieberman, 2004; Kross et al., 2007). As the AUD and control groups showed the same level of subjective anger after watching the film, the greater activations both in the IFG and dACC regions implicated in response inhibition and emotion regulation may suggest that alcoholics could have exerted greater self-control in an effort to tolerate anger as much as the controls.

Despite the findings, this study has a few limitations. First, because our study used anger provoking stimuli that had been rated 5 or higher on a 7 point scale, the variance of our subjects' ratings were not as large as we hoped, which made it difficult to observe a significant correlation between IFG or dACC and anger emotion ratings. Second, because we did not obtain information on psychological factors that relates to or measure emotion control during anger provoking condition, it is limited to suggest any direct association between greater activation in the IFG or dACC among AUD group and possible greater effort to control emotion.

Despite some of the aforementioned limitations, this study is the first to provide preliminary findings on brain functional differences in emotion processing between AUD young adults and those without drinking issue.

References

Damasio, A. R., Grabowski, T. J., Bechara, A, Damasio, H., Ponto, L. L., Parvizi, J., & Hichwa, R. D. (2000). Subcortical and cortical brain activity during the feeling of self-generated emotions, *Nature Neuroscience*, *3(10)*, 1049-1056.

Denson, T. F., Pedersen, W. C., Ronquillo, J., & Nandy, A. S. (2009). The angry brain: Neural correlates of anger, angry rumination, and aggressive personality, *Journal of Cognitive Neuroscience, 21(4),* 734-744.

Dethier, M., & Blairy, S. (2012). Capacity for cognitive and emotional empathy in alcohol-dependent patients. Psychology of Addictive Behaviors*, 26(3),* 371 – 383.

Dougherty, D. D., Shin, L. M., Alpert, N. M., Pitman, R. K., Orr, S. P., Lasko, M., Macklin, M. L., Fischman, A. J., & Rauch, S. L. (1999). Anger in healthy men: A PET study using script-driven imagery, *Biological Psychiatry, 46*, 466 – 472.

Drexler, K., Schweitzer, J. B., Quinn, C. K., Gross, R., Ely, T. D., Muhammad, F., & Kilts, C. D. (2000). Neural activity related to anger in cocaine-dependent men: A Possible Link to Violence and Relapse, *The American Journal on Addictions, 9*, 331 – 339.

Eisenberger, N., & Lieberman, M. D. (2004). Why rejection hurts: A common neural alarm system for physical and social pain, *Trends in Cognitive Sciences, 8,* 294 – 300.

Fu, L. P., Bi, G. H., Zou, Z. T., Wang, Y., Ye, E. M., Ma, L., Ming-Fan & Yang, Z. (2008). Impaired response inhibition function in abstinent heroin dependents: an fMRI study. *Neuroscience Letters, 438(3)*, 322-326.

Gasquoine, P. G. (2013). Localization of function in anterior cingulate cortex: from psychosurgery to functional neuroimaging. *Neuroscience & Biobehavioral Reviews*, *37*, 340 – 348.

Gilman, J. M., & Hommer, D. W., (2008). Modulation of brain response to emotional images by alcohol cues in alcohol-dependent patients. *Addiction Biology, 13(3–4)*, 423–434.

Han, K.-W., Kim, M.-J., Kim, S.-G., & Byun, W.-T. (1996) Study of the conditioned stimuli provoking alcohol craving in the patients with alcohol dependence. *Journal of Korean Neuropsychiatric Association, 35(4)*, 809-819.

Hahn, O. S., Ahn, J. H., Song, S. H., Cho, M. J., Kim, J. K., Bae, J. N., Cho, S. J., Jeong, B. S., Suh, D. W., Hahm, B. J., Lee, D. W., Park, J. I., & Hong, J. P. (2000). Development of the Korean version of structured clinical interview schedule for DSM-IV Axis I Disorder: Interrater reliability, *Journal of Korean Neuropsychiatry Association, 39,* 362-372.

Kim, K. B., Hahn, K. S., Lee, J. K., Rhee, M. K., Kim, Y. K., & Kim, C. K. (1991). The preliminary study of the Korean alcoholism screening test (III)-Alcoholism Screening Test of Seoul National Mental Hospital (I): NAST (I). *Journal of Korean Neuropsychiatry Association, 30*, 569-581.

Kimbrell, T. A., George, M. S., Parekh, P. I., Ketter, T. A., Podell, D. M., Danielson, A. L., Repella, J. D., Benson, B. E., Willis, M. W., Herscovitch, P., & Post, R. M. (1999). Regional brain activity during transient self-induced anxiety and anger in healthy adults, *Biological Psychiatry, 46*, 454–465.

Kornreich, C., Philippot, P., Verpoorten, C., Dan, B., Baert, I., Le Bon, O., Verbanck, P., & Pelc, I. (1998). More heterogeneous film-induced emotional response in newly detoxified alcoholics compared to controls-A preliminary sudy. *Addictive Behaviors, 23(3)*, 413-418.

Kross, E., Egner, T., Ochsner, K., Hirsch, J., & Downey, G. (2007). Neural dynamics of rejection sensitivity. *Journal Cognitive Neuroscience, 19*, 945–956.

Lane, R. D., Reiman, E. M., Bradley, M.M., Lang, P. J., Ahem, G. L., Davidson, R. J. & Schwartz, G. E. (1997). Neuroanatomical correlates of pleasant and unpleasant emotion, *Neuropsychologia, 35 (11)*, 1437–1444.

Lee, B. W. (2000). Development of the Korean version of Alcohol Use Disorders Identification Test (AUDIT-K). *Korean Academy of Addiction Psychiatry, 4*, 83-92.

Lindquist, K. A., Wager, T. D., Kober, H., Bliss-Moreau, E., & Barrett, L. F. (in press). The brain basis of emotion: A meta-analytic review. Behavioral and Brain Sciences.

Marinkovic, K., Oscar-Berman, M., Urban, T., O'Reilly, C. E., Howard, J. A., Sawyer, K., & Harris, G. J. (2009). Alcoholism and dampened temporal limbic activation to emotional faces, *Alcoholism: Clinical and Experimental Research, 33(11)*, 1880–1892.

Oscar-Berman, M., & Marinkovic, K. (2003). Alcoholism and the brain: an overview. *Alcohol Research & Health, 27(2),* 125–133.

Park, B. K., Kim, J. S., & Lee, D. B. (2000). Comparison of four alcoholism screening tools based on sensitivity and specificity for DSM-IV criteria, *Journal of the Korean Academy of Family Medicine, 21*, 1427-1435.

Potter-Efron, P. S., & Potter-Efron, R. T. (1991). Anger as a treatment concern with alcoholics and affected family members, *Alcoholism Treatment Quarterly, 8,* 31-46.

Salloum, J. B., Ramchandani, V. A., Bodurka, J., Rawlings, R., Momenan, R., George, D., & Hommer, D. W. (2007). Blunted rostral anterior cingulate response during a simplified decoding task of negative emotional facial expressions in alcoholic patients. *Alcoholism: Clinical and Experimental Research, 31(9)*, 1490–1504.

Sohn, J. H., Lee, O. Y., Suk, J. A, Park, M. S. (2005). *Development and validation of emotion-inducing film stimuli*. Seoul: Korean Federation of Science and Technology Societies Press.

Spielberger, C. D., Krasner, S. S., & Soloman, E. P. (1988). The experience, expression and con-trol of anger. In Janisse MP (ed), *Health Psychology: Individual Differences and Stress*, pp. 89-108. New York: Springer-Verlag.

Talairach, J., & Tournoux, P. (1988). *Co-planar stereotaxic atlas of the human brain*. New York: George Thieme Verlag.

Tivis, L. J., Parsons, O. A., & Nixon, S. J. (1998). Anger in an inpatient treatment sample of chronic alcoholics. *Alcohol Alcoholism: Clinical and Experimental Research, 22,* 902–907.

Uekermann, J., & Daum Fitzpatrick, I. (2008). Social cognition in alcoholism: a link to prefrontal cortex dysfunction? *Addiction, 103,* 726–735.

Walfish, S., Massey, R., & Krone, A. (1990). MMPI profiles of adolescent substance abusers in treatment. *Adolescence, 25*, 567-572.

Wright P., He G., Shapira N. A., Goodman W. K., & Liu Y. (2004). Disgust and the insula: fMRI responses to pictures of mutilation and contamination, *Neuroreport, 15*, 2347.

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