Published in final edited form as:
Neurogenetics. 2014 March ; 15(1): 77–78. doi:10.1007/s10048-013-0379-4.

Evidence of a role for *SNCA* in impulse control in humans

Casey R. Guillot, Ph.D.[a], **Jennifer R. Fanning, Ph.D.**[b], **Tiebing Liang, Ph.D.**[c], and **Mitchell E. Berman, Ph.D.**[d]

Casey R. Guillot: cguillot@usc.edu

[a]University of Southern California Keck School of Medicine, 2250 Alcazar St, CSC 240, Los Angeles, CA 90033; Tel: 323-442-2598; Fax: 323-442-2359

[b]National Center for PTSD/Yale Department of Psychiatry, West Haven, CT

[c]Indiana University School of Medicine, Indianapolis, IN

[d]Department of Psychology, Mississippi State University, Mississippi State, MS

Alpha-synuclein has been implicated in impulsivity in mice [1] and in addictive behavior (suggestive of impaired impulse control) in both rodents and humans [1, 2]; therefore, the α-synuclein gene (*SNCA*) may influence the development of impulsivity in humans, although to date no study has examined this relationship. Based on the greater frequency of the C-allele of *SNCA* rs356195 in alcohol non-cravers relative to cravers in a prior study [2], we hypothesized that individuals with the CC genotype of *SNCA* rs356195 would display greater impulse control than T-allele carriers.

One hundred and thirty-nine healthy Caucasian men and women aged 21 to 55 (M = 25.95; SD = 7.45) were recruited using the same procedure as Guillot et al. [3] and were genotype tested for the single-nucleotide polymorphism (SNP) *SNCA* rs356195. Participants were administered the Barratt Impulsiveness Scale Version 11 (BIS-11) [4] and Self-Control Scale (SCS) [5] as self-report measures of impulsivity and the GoStop Impulsivity Paradigm (GoStop) [6] as a behavioral measure of impulsivity. GoStop analysis involved a smaller sample of 93 participants for the reason discussed in Guillot et al. [3].

The genotypic frequency distributions did not deviate significantly from expected Hardy-Weinberg equilibrium in the total sample (78 CC, 50 CT, and 11 TT; χ^2 = .55, p = .46) or GoStop sample (52 CC, 33 CT, and 8 TT; χ^2 = .68, p = .41). Given the small TT group sizes (8–11 per measure) and thus the low power for detecting differences between the TT group and other groups, we chose to test only the dominant model of genetic inheritance by comparing C homozygotes to T-allele carriers. Fisher's exact tests and one-way analyses of variance revealed that C homozygotes and T-allele carriers did not differ significantly in respect to gender, age, years of education, or marital status (all ps > .34).

In comparing C homozygotes to T-allele carriers, one-way analyses of variance revealed significantly lower BIS-11 scores ($F_{(1, 137)}$ = 4.74, p = .031, d = .37), higher SCS scores ($F_{(1, 137)}$ = 5.00, p = .027, d = .38), lower GoStop Ratios ($F_{(1,91)}$ = 9.74, p = .002, d = .65), and greater GoStop inhibition percentages ($F_{(1, 91)}$ = 6.37, p = .013, d = .52), mean latencies to respond on stop trials ($F_{(1,91)}$ = 13.34, p = .0004, d = .76), and mean latencies to respond on go trials ($F_{(1, 91)}$ = 12.90, p = .0005, d = .75), all of which indicate greater impulse control in C homozygotes relative to T-allele carriers (see Table 1).

Based on our results, it appears that variation in *SNCA* may confer risk for the development of impulsivity in humans. Of course, our findings should be considered preliminary until the relationship between *SNCA* and impulsivity is examined in larger samples using a range of SNPs, particularly samples large enough to test for multiple models of genetic inheritance.

Nonetheless, the current relationship between *SNCA* and impulsivity may have broad implications given the prominent role of impulsivity in addictive and aggressive behavior [5].

Acknowledgments

This study was supported by funding from the National Institute on Alcohol Abuse and Alcoholism to Mitchell E. Berman (Grant Number: AA14025).

References

1. Peña-Oliver Y, Buchman VL, Dalley JW, Robbins TW, Schumann G, Ripley TL, King SL, Stephens DN. Genes Brain Behav. 2012; 11:137–146. [PubMed: 22142176]
2. Foroud T, Wetherill LF, Liang T, Dick DM, Hesselbrock V, Kramer J, Nurnberger J, Schuckit M, Carr L, Porjesz B, Xuei X, Edenberg HJ. Alcohol Clin Exp Res. 2007; 31:537–545. [PubMed: 17374032]
3. Guillot CR, Fanning JR, Bullock JS, McCloskey MS, Berman ME. Exp Clin Psychopharmacol. 2010; 18:409–417. [PubMed: 20939644]
4. Patton JH, Stanford MS, Barratt ES. J Clin Psychol. 1995; 51:768–774. [PubMed: 8778124]
5. Tangney JP, Baumeister RF, Boone AL. J Pers. 2004; 72:271–324. [PubMed: 15016066]
6. Dougherty DM, Marsh-Richard DM, Hatzis ES, Nouvion SO, Mathias CW. Drug Alcohol Depend. 2008; 96:111–120. [PubMed: 18378098]

Table 1

Means and standard deviations on impulsivity measures for groups defined by the absence (CC) or presence (CT/TT) of the T-allele of SNCA rs356195

	CC	CT/TT
BIS-11	62.28 (9.97)*	66.39 (12.30)*
SCS	126.74 (19.08)*	119.28 (20.11)*
GoStop		
Inhibition %	44.76 (27.54)*	31.23 (23.05)*
ML-ST	418.55 (111.90)***	341.85 (83.90)***
ML-GT	522.96 (209.10)***	381.62 (158.12)***
Ratio	.69 (.34)**	.89 (.26)**

BIS-11 Barratt Impulsiveness Scale Version 11, *SCS* Self-Control Scale, *ML-ST* mean latency to respond on stop trials in seconds, *ML-GT* mean latency to respond on go trials in seconds.

* different at $p < .05$,

** different at $p < .01$,

*** different at $p < .001$