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Epidemiological Dimensions of Social Anhedonia

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Abstract
Social anhedonia (SA)—reduced drive for and pleasure from social interaction—is associated with social/emotional dysfunction and risk for psychopathology. However, our understanding of the factors that contribute to variation in SA remains limited. Here, we investigate the epidemiology of SA in an international population-based sample of more than 19,000 individuals who completed the Revised Social Anhedonia Scale through TestMyBrain.org. We find that SA exhibits considerable variation over the life span and is higher in males versus females, people of lower socioeconomic status, those of African ethnicity, nonmigrants, and people living in ethnically dense locations and less urban environments. Gender, socioeconomic status, and urbanicity were the only factors that captured unique variance in SA. These findings provide a framework for understanding how variation in epidemiological factors contribute to variation in elemental building blocks of psychopathology, and demonstrate the utility of using big data approaches toward the study of risk and Research Domain Criteria dimensions.

Keywords
social anhedonia, social processes, schizophrenia, Research Domain Criteria, epidemiology, open data

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Our nature as social animals is to interact, bond, and form enduring connections with one another. Indeed, decades of research have shown that human beings exhibit a fundamental need to belong, that is, a constitutional motivation to be with others (Baumeister & Leary, 1995; Silvia & Kwapil, 2011). However, motivation for and enjoyment of social interaction vary from person to person. Given the importance of social connection for mental and physical health and well-being (Cacioppo, Hawkley, Norman, & Berntson, 2011), the consequences of this variation can be profound.

Consider the phenomenon of social anhedonia (SA)—the reduced drive for and pleasure from social interaction. Socially anhedonic people experience a genuine preference for solitude and disinterest in social interaction that cannot be attributed to social anxiety or social exclusion (Brown, Silvia, Myin-Germeys, & Kwapil, 2007). SA is associated with a variety of social and emotional difficulties including less daily and trait positive affect and more negative affect (Blanchard, Collins, Aghevli, Leung, & Cohen, 2011; Brown et al., 2007; Gooding, Davidson, Putnam, & Tallent, 2002; Kwapil, Brown, Silvia, Myin-Germeys, & Barrantes-Vidal, 2012); less social skill, contact, interest, and pleasure (Brown et al., 2007; Kwapil et al., 2009; Kwapil et al., 2012; Llerena, Park, Couture, & Blanchard, 2012); and less social support and social coping strategies (Blanchard et al., 2011; Horan, Brown, & Blanchard, 2007).

Beyond impaired social and emotional functioning, SA represents a major component of schizotypy, a multidimensional construct involving schizophrenia-like phenomena that is thought to index vulnerability for schizophrenia-spectrum disorders (Kwapil, Barrantes-Vidal, & Silvia, 2007). In line with this idea, high levels of SA and concomitant social withdrawal prospectively predict schizophrenia-spectrum disorders years later (Gooding, Tallent, & Matts, 2005; Kwapil, 1998; Tarbox

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& Pogue-Geile, 2008). SA also represents a major pathophysiological component of schizophrenia-spectrum disorders (Horan, Blanchard, Clark, & Green, 2008). In those diagnosed with a schizophrenia-spectrum disorder, SA is trait like, remaining stable over time despite fluctuations in symptoms (Blanchard, Horan, & Brown, 2001), and is associated with reduced positive reactions and willingness to interact with social partners (Blanchard, Park, Catalano, & Bennett, 2015; McCarthy et al., 2018), general social dysfunction (Blanchard et al., 2001; McCarthy et al., 2018), and neural abnormalities in social cognitive processes (Dodell-Feder, Tully, Lincoln, & Hooker, 2014).

Given SA's role in risk for schizophrenia-spectrum disorders, possibly through its association with schizotypy, and SA's contribution to social and emotional difficulties in those with and without these conditions, it is critical to understand risk factors for SA. However, our understanding of the variation in SA and the contexts in which it is most likely to manifest is limited. As our field moves toward a dimension-based framework of psychopathology as outlined by NIMH's Research Domain Criteria (RDoC), it is incumbent on us to understand the etiology of those dimensions, just as decades of prior research have endeavored to understand the etiology of categorical nosological entities. As a component of the RDoC Affiliation and Attachment subconstruct within Social Processes, SA is conceptually simpler than schizophrenia and schizotypy, and may more precisely reflect relevant dimensions underlying its heterogeneity. Greater understanding of how SA varies may in turn provide more precise etiological pathways leading to severe psychopathology, that is, *why* a disorder like schizophrenia occurs from disturbances in elemental perceptual, cognitive, and motivational systems.

Here, we evaluate a population-based sample of more than 19,000 international participants to uncover the epidemiological dimensions of SA: a phenomenon that may lie along the causal pathway from elemental social processes to the expression of psychopathology. Specifically, we investigate whether SA, as measured by the Revised Social Anhedonia Scale (Eckblad, Chapman, Chapman, & Mishlove, 1982), is associated with age, gender, socioeconomic status (i.e., education and median income of the city in which the participant completed the questionnaire; henceforth, "community income"), ethnicity, and, given the link among SA, schizotypy, and schizophrenia, other factors that have been shown to influence risk for schizophrenia, including migrant status, ethnic density (i.e., the proportion of residents in a participant's city that belong to the participant's ethnic group), and urbanicity.

Our data collection relied on web-based survey methods through the noncommercial research website TestMyBrain.org to reach a large and diverse sample that would not have been feasible with geographically restricted in-person testing. Prior work has demonstrated that data collected from such participants are highly reliable and comparable in quality to data collected in traditional lab or clinic settings (Germine et al., 2012; Soto, John, Gosling, & Potter, 2011), and mirror findings from nationally representative population-based samples (Hartshorne & Germine, 2015). Thus, our sample provides a reasonable starting point for estimates of the relation between epidemiological factors and SA at the population level.

Methods

Participants

Our sample was 19,432 international participants, 36.41% of whom were from the United States (54.79% were from predominantly English-speaking countries including the United States; Table 1). Additional information regarding our sample is included in the Supplemental Material available online. Participants provided informed consent by electronically signing a form prior to participation. The study was approved by the Harvard University Institutional Review Board.

Given the geographic diversity of our participants (Table 1), we evaluated the effect of location on SA and found a main effect of continent, $F(5, 16{,}316) = 11.03$, $p < .001$, $\eta^2 = .003$, such that individuals from the continents of Australia/Oceania, Europe, and North America reported less SA than individuals from the continent of Africa (ps < .002, d = .23–.30, common language effect size [CLES] = 41.56%–43.56%) and Asia (ps < .001, d = .12–.20, CLES = 44.33%–46.60%). The effect of continent did not interact with other variables that might affect the expression of SA, including ethnicity, $F(26, 13{,}414) = 1.44$, $p = .070$, and migrant status, $F(5, 15{,}236) = 1.89$, $p = .092$, suggesting that the effects of ethnicity and migrant status were relatively stable across geographic location. To further evaluate the possible moderating effect of participant location, for the ethnicity and migrant status analyses, we conducted additional analyses from predominantly English-speaking/European ethnicity countries described in the following sections.

Social anhedonia

SA was assessed with the 40-item Revised Social Anhedonia Scale (RSAS; Eckblad et al., 1982). The RSAS is a self-report measure designed to assess social amotivation/

Table 1. Participant Characteristics and Analysis Sample Sizes

Variable	Analysis *N*	*n* (%)	*M* (*SD*) [range]
Continent	16,322		
Africa		261 (1.60)	
Asia		2,612 (16.00)	
Australia/Oceania		744 (4.56)	
Europe		4,530 (27.75)	
North America		8,002 (49.03)	
South America		173 (1.06)	
Country			
U.S.[a]		7,075 (36.41)	
Non-U.S.[a]		9,606 (49.43)	
No data available[a]		2,751 (14.16)	
Majority English-speaking/European ethnicity country[b]		10,646 (54.79)	
Non–majority English-speaking/European ethnicity country[b]		8,786 (45.21)	
Age	19,432		26.18 (11.84) [9–72]
Gender	19,170		
Male		7,132 (37.20)	
Female		12,038 (62.80)	
Education	19,100		
None		825 (4.32)	
Middle school		1,065 (5.58)	
High school		5,034 (26.35)	
Some college		5,223 (27.35)	
College		3,740 (19.58)	
Graduate school		3,213 (16.82)	
Community income[c]	5,802		58,658 (23,888) [8,864–242,782]
Ethnicity	15,986		
African		611 (3.82)	
American Indian or Alaskan Native		118 (0.74)	
East Asian		1,251 (7.83)	
European		11,228 (70.24)	
Native Hawaiian or Pacific Islander		98 (0.61)	
South Asian		2,109 (13.19)	
Middle Eastern		571 (3.57)	
Migrant status	15,574		
Migrant		1,941 (12.46)	
Nonmigrant		13,633 (87.54)	
Ethnic density[d]	4,690		63.41 (27.58) [0–100]
African		286 (6.10)	22.14 (17.31) [0–83.93]
American Indian or Alaskan Native		58 (1.24)	1.95 (3.67) [0–18.85]
East Asian		188 (4.01)	11.74 (12.28) [0–60.67]
European		3,935 (83.90)	72.80 (17.69) [1.51–100]
Native Hawaiian or Pacific Islander		41 (0.87)	0.71 (1.90) [0–9.53]
South Asian		182 (3.88)	12.37 (13.20) [0–65.63]
Urbanicity[e]	5,809		471,795 (1,379,478) [63–8,426,743]

[a]Percentage calculated with respect to categories with shared superscripts. [b]Percentage calculated with respect to categories with shared superscripts. Majority English-speaking countries include the United States, Canada, Great Britain, Ireland, Australia, and New Zealand. [c]*M, SD*, and range expressed as median income in the city in which the participant completed the questionnaire. [d]*M, SD*, and range expressed as percentage. [e]*M, SD*, and range expressed as number of people in the city in which the participant completed the questionnaire.

disinterest and lack of pleasure from social interaction. It is widely used in the schizophrenia (Blanchard, Mueser, & Bellack, 1998) and psychosis risk literature (Gooding et al., 2005; Kwapil, 1998), as well as in nonclinical studies of social affiliation (Germine, Dunn, McLaughlin, & Smoller, 2015). The RSAS exhibits adequate psychometric properties (Kwapil et al., 2007). Elevated scores on the RSAS have been shown to

predict the onset of psychosis spectrum illnesses (Gooding et al., 2005; Kwapil, 1998) and are associated with a variety of neuropsychological (Gooding & Tallent, 2003), daily functioning (Blanchard et al., 2011), and neural measures (Dodell-Feder et al., 2014).

We note that despite the RSAS being used with participants as young as 12 years of age (Rosa et al., 2000), it has been validated only for participants aged 18 years and older. To confirm the validity of the scale with younger participants, we compared the psychometric properties of the scale when administered to older and younger participants and provide these data in the Supplemental Material.

One of two versions of the RSAS was administered in which participants either responded to each item with the original true/false response scale (n = 5,365) or with a 5-point Likert-type scale ranging from *strongly disagree* to *strongly agree* (n = 14,067). To our knowledge, the RSAS has not previously been validated with a Likert-type response scale. Thus, we similarly confirmed the validity of this approach by comparing the psychometric properties of the RSAS when administered with the original versus Likert-type scale, and provide these data in the Supplemental Material.

For analysis, the original scale and Likert-type scale data sets were separately z-scored and then combined. All analyses were performed and are plotted with these z-scores. We note that none of the findings change in terms of statistical significance or effect size when including scale type as a covariate.

Data analysis

Data were analyzed in R. For all analyses, scores are reported with 95% confidence intervals (CIs) and are accompanied by effect sizes for group differences (Cohen's d and CLES) and/or effect sizes denoting variance accounted for (adjusted R^2, η^2). Findings were considered statistically significant at $p < .05$, with correction for multiple comparisons where appropriate. We note that our massive sample size renders traditional null hypothesis testing less informative (Lin, Lucas, & Shmueli, 2013). Consequently, we largely focus our discussion and interpretation on effect sizes using standard guidelines (J. Cohen, 1988). Additional details regarding data analysis are provided in the Supplemental Material.

Results

Age and gender

Segmented regression demonstrated that the relation between age and SA was best fit by a three segment linear function, R^2 = .0022 (Fig. 1a; also see the Supplemental Material). Specifically, SA steadily increases from age 9, b = .04, 95% confidence interval (CI) = [.01, .07], reaching an initial transition point at age 15.13, 95% CI = [13.29, 16.97]. After this age, SA continues to increase, but at a lower/reduced rate, b = .004, 95% CI = [.002, .007], until peaking at age 43.33, 95% CI = [37.31, 49.34]. This peak is followed by a decline in SA into late age, b = –.01, 95% CI = [–.02, –.004]. Thus, SA exhibits two transition periods across the life span: one in adolescence, which is preceded by a steep increase and followed by a milder increase, and another in adulthood, which is followed by moderate decline.

Males reported significantly greater SA compared with females, $t(15{,}263) = 13.64$, $p < .001$ (Fig. 1b). This effect was comparable with gender differences in social anhedonia that have been reported in other studies (Chmielewski, Fernandes, Yee, & Miller, 1995), d = .20, 95% CI = [.17, .23], CLES = 55.7%.

Socioeconomic status

We observed a main effect of education on SA, $F(5, 19{,}094) = 13.19$, $p < .001$, $\eta^2 = .003$ (Fig. 1c). Compared with more highly educated participants, those who received less than a college education reported above average ($z > 0$) and higher levels of SA, with participants receiving no education reporting the highest levels of SA. Compared with less educated participants, those who received a college or graduate degree reported below average ($z < 0$) and lower levels of SA, with those receiving a graduate degree reporting the lowest levels. Post hoc tests revealed that the differences in SA between college or graduate school educated participants and all lower educated groups were statistically significant (ps < .05; the difference between college educated versus no education groups was significant at p = .053) except for participants reporting a middle school education. These differences were small in magnitude, ds = –.09, CLES = 47.46% to d = –.18, CLES = 44.8%, with the biggest difference existing between the least and most educated group, d = –.18, 95% CI = [–.26, –.10], CLES = 44.8%. The main effect of education remained when controlling for age and gender.

Community income exhibited a negative relation with SA, $\beta = -.0635$, $p < .001$, explaining a small but statistically significant amount of variance, R^2 = .0039, such that participants with higher community incomes reported less SA (Fig. 1d). The effect of income on SA did not change when controlling for age and gender.

Ethnicity

We observed a main effect of ethnicity on SA, $F(6, 15{,}979) = 6.53$, $p < .001$, $\eta^2 = .002$ (Fig. 1e). Participants of European and Native Hawaiian/Pacific Islander

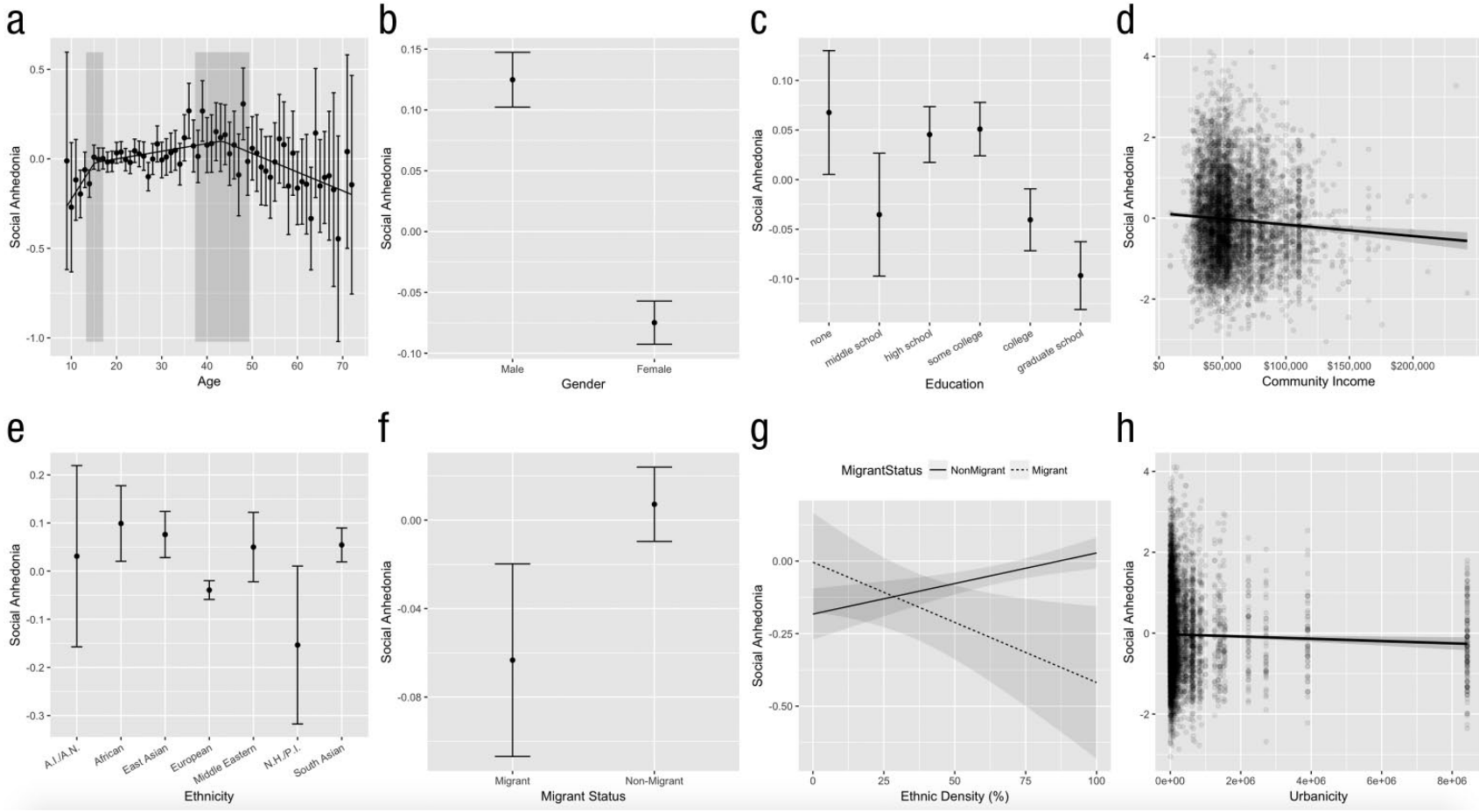

Fig. 1. The relation between the epidemiological factors and social anhedonia. (a) Mean social anhedonia *z*-score plotted by age with 95% confidence intervals (CIs). The solid line represents the three-segment function generated with segmented regression that best fit the data. Shaded regions represent 95% CIs around the two breakpoints in which the relation between social anhedonia and age changes. (b) Mean social anhedonia *z*-score by gender with 95% CIs. (c) Mean social anhedonia *z*-score as a function of education level with 95% CIs. (d) Scatterplot of the relation between social anhedonia *z*-score and community income. Line represents linear regression estimate with 95% CI band. (e) Mean social anhedonia *z*-scores as a function of ethnicity with 95% CIs. A.I. = American Indian; A.N. = Alaskan Native; N.H. = Native Hawaiian; P.I. = Pacific Islander. (f) Mean social anhedonia *z*-scores as a function of migrant status with 95% CIs. (g) Interaction of ethnic density and migrant status with the regression estimate for migrants depicted in the dashed line and nonmigrants depicted in the solid line. Shaded areas represent 95% CI band around the regression estimate. (h) Scatterplot of the relation between social anhedonia *z*-score and urbanicity (i.e., number of people living in the city in which the participant completed the questionnaire). Line represents linear regression estimate with 95% CI band.

descent reported lower than average levels of SA ($z < 0$), with Native Hawaiian/Pacific Islander reporting the lowest levels of SA. All other ethnic groups reported above average levels of SA ($z > 0$), with participants of African descent reporting the highest levels of SA. Post hoc tests revealed that participants of European descent reported significantly lower levels of SA compared with participants of African, $p = .015$, $d = -.13$, 95% CI = [–.22, –.05], CLES = 46.2%, East Asian, $p = .002$, $d = -.12$, 95% CI = [–.18, –.06], CLES = 46.7%, and South Asian descent, $p = .002$, $d = -.09$, 95% CI = [–.14, –.04], CLES = 47.5%, all of which were small effects. The main effect of ethnicity remained significant when controlling for age and gender.

Findings were relatively similar when analyzing data from predominantly English-speaking/European ethnicity countries in that we found a main effect of ethnicity, $F(6, 8{,}836) = 2.57$, $p = .018$, $\eta^2 = .002$, whereby participants reporting African ethnicity had the highest levels of SA with a similar magnitude of difference from European participants, $d = -.12$, 95% CI = [–.22, –.02], CLES = 46.56%, as when analyzing the entire sample, although the difference was not significant. Participants reporting Asian ethnicities reported the lowest levels of SA, with the only significant difference being between participants of South Asian versus African ethnicity, $d = -.26$, 95% CI = [–.40, –.12], CLES = 42.76%, $p = .033$. This pattern of findings (i.e., Asian < European < African), more closely resembles prior studies of psychotic symptoms by ethnicity in the United States (C. I. Cohen & Marino, 2013).

Migrant status

Migrants reported less SA than nonmigrants, $t(2{,}554) = 2.96$, $p = .003$, which was a small effect, $d = -.07$, 95% CI = [–.12, –.02], CLES = 48.0% (Fig. 1f). Given epidemiological findings on schizophrenia demonstrating that incidence is highest among non-White minority migrants (Kirkbride et al., 2012), we evaluated whether the effect of migrant status on SA differs as a function of ethnic minority status (i.e., participants in the United States reporting European versus non-European descent) and found no interaction between the terms (see the Supplemental Material). The main effect of migrant status remained when controlling for age and gender.

Findings were similar when evaluating the effect of migrant status on SA in predominantly English-speaking/European ethnicity countries. Specifically, migrants reported less SA than nonmigrants, $t(1{,}411) = 2.41$, $p = .016$, which was a small effect, $d = -.08$, 95% CI = [–.14, –.01], CLES = 47.85%.

Ethnic density

Ethnic density exhibited a positive relation to SA, $\beta = .0350$, $p = .017$, explaining a small, but statistically significant amount of variance, $R^2 = .001$, such that greater ethnic density (i.e., the greater proportion of the city's population that is of the same ethnicity of the participant) was associated with greater SA. Given reports in the epidemiological literature of ethnic density contributing to the incidence of psychotic disorders among immigrants and non-White minority groups specifically (Veling et al., 2008), we evaluated whether the effect of ethnic density on SA differs as a function of migrant and ethnic minority status. In line with this idea, we found a significant interaction between ethnic density and migrant status, $\beta = .1757$, $p = .001$. Simple slopes analysis revealed a small positive relation between ethnic density and SA for nonmigrants, $b = .0021$, $p = .001$ (Fig. 1g). In contrast, there was a small negative relation between ethnic density and SA for migrants, $b = -.0042$, $p = .025$. Said otherwise, ethnic density may confer a protective effect against SA for migrants but the opposite, albeit very small effect, for nonmigrants. The effect of ethnic density on SA was not moderated by minority status, $\beta = .0644$, $p = .418$. The relation between ethnic density and SA remained when controlling for age and gender.

Urbanicity

Urbanicity exhibited a negative relation with SA, $\beta = -.0362$, $p = .006$, explaining a small but statistically significant amount of variance, $R^2 = .0011$, such that participants from more populous regions reported less SA (Fig. 1h). The effect of urbanicity on SA did not change when controlling for age and gender.

The unique and total effect of the variables on SA

The epidemiological variables investigated here were all related (see the Supplemental Material) raising the question of whether any of these factors captured unique variance in SA. Furthermore, each variable on its own captured only a small amount of variance in SA. Using the data set from participants in the United States for which we had information for all variables ($N = 3{,}938$), we conducted a simultaneous regression including all of the variables (age [3-segment function], gender, education [coded as high/low education], community income, ethnicity [coded as minority/nonminority], migrant status, ethnic density, urbanicity) to address two questions: (a) Which factors capture unique variance in SA? and (b) How much variance in SA do these factors together explain? Four variables emerged as significant correlates of SA: education, such that more educated participants reported less SA than less educated participants, $\beta = -.0956$, $p < .001$, gender, such that females reported less SA than males, $\beta = -.0769$,

$p < .001$, community income, $\beta = -.0766$, $p < .001$, and urbanicity, $\beta = -.04946$, $p = .003$. Age, while not significant when controlling for all other factors, had the largest impact on SA, $\beta = 1.0046$. Together, all of the factors explained only a small amount of variance in SA, $R^2 = .0252$. The inclusion of relevant interaction terms (i.e., Age × Gender, Ethnic Density × Migrant Status) improved fit, $F(2, 3{,}923) = 9.76$, $p < .001$, but did not substantially increase the amount of variance explained, $R^2 = .0295$.

Discussion

Here, we report findings from the first population-based study of SA in an international sample of more than 19,000 individuals to better understand the epidemiology of variation in SA. We found multiple risk factors for increased SA with the most robust factors being socio-economic status, gender, and urbanicity. These findings reveal previously unknown associations between SA and demographic/social-environmental variables, as well as demonstrating the utility of using big data approaches toward studying the epidemiology of RDoC constructs.

Several of the associations we found are notable. First, our findings suggest that the expression of SA is associated with social and economic disadvantage. Though it is impossible to disentangle cause from effect here, the stress of social disadvantage may deleteriously impact one's capacity for enjoyment of social interaction and/or motivation to seek it out. SA may also deleteriously affect the size of and/or quality of one's social network, removing an important buffer from stress. This might explain, in part, the connection between SA, schizotypy, and psychopathology. It is also notable that the current findings largely mirror those from the schizophrenia literature and are generally consistent with etiological theories of schizophrenia positing a primarily role of social disadvantage in the development of the disorder (Morgan et al., 2008). Second, controlling for other factors, SA varies as a function of gender such that males report higher SA than females. This finding is consistent with reports of higher rates of negative symptoms in males versus females with schizophrenia (Gur, Petty, Turetsky, & Gur, 1996), higher incidence of schizophrenia in males versus females (Kirkbride et al., 2012), and poorer social functioning in diagnosed males (Hooley, 2010). Research has demonstrated a link between SA and social cognition (Germine & Hooker, 2011), and women tend to outperform men on social cognitive tasks (Baron-Cohen et al., 2015). Thus, our findings may reflect enhanced social skills in women, which may contribute to more enjoyment of social interaction, fulfilling social affiliation, more social support, and protection against SA. Third, controlling for other factors, less urbanicity was associated with greater SA. Here too, the causal direction is unclear: fewer opportunities to socialize and/or smaller social networks may make socializing difficult and contribute to less motivation to seek it out, or lack of interest in socializing may contribute to seeking out environments where social interaction is less likely to occur. Future work is needed to clarify the causal direction and mechanism underlying these associations.

It is important to note that the epidemiological factors we investigated here explained only a very small amount of the variance in SA. When examined separately, each factor explained less than 1% of the variance in SA, and group differences were all in the small range ($d < .20$). Said otherwise, for one of our largest effects—the gender effect ($d = .20$)—there is only approximately a 56% chance that a randomly selected male would report greater SA than a randomly selected female. When considered together, the epidemiological factors explained between 2% and 3% of the variance in SA. This is comparable with the degree to which these factors explain variance in risk for schizophrenia. As is the case with mental disorders, such epidemiological risk factors do not explain sufficient variance to allow us to *predict* who will develop or not develop a disorder (or, in this case, who will have higher versus lower social anhedonia), but they provide important clues regarding how the environment might shape or contribute to differences in risk in the population.

It would be interesting for future work to consider SA within the broader context of personality. For example, SA has been consistently linked to introversion (Gooding, Padrutt, & Pflum, 2017; Kwapil et al., 2007), suggesting that this and other related constructs may be affected by and expressed through similar mechanisms. Epidemiological data may also speak to the extent to which SA does or does not overlap with other aspects of personality (Martin, Cicero, Bailey, Karcher, & Kerns, 2016), an important consideration for conceptualizing the nature of personality and for the assessment and treatment of personality disorders.

Several limitations are notable. First, we evaluated SA in a nonrandom sample leaving open the possibility of self-selection effects. Second, these findings are cross-sectional, which preclude causal inferences regarding developmental changes in SA, and the effect of the variables on SA. Third, we used proxies for migrant status, urbanicity, income, and ethnic density, which may have resulted in classification inaccuracies. Fourth, we did not include an assessment of mental health leaving open the possibility that mental health issues may have contributed to the relations observed here.

To summarize, we find that SA varies as a function of several demographic and social-environmental variables. These data can help identify factors that may confer risk for SA and, in turn, additional phenomena (e.g., social isolation) that are associated with declines in well-being and the onset of psychopathology. We also demonstrate how modern methods for population-based assessment can provide inroads into understanding the epidemiology of RDoC dimensions like SA, investigations that will be a fundamental part of building a dimensional classification system for mental disorders. Given the similarity of findings between traditional lab and web-based methods of data collection, we see no reason why the field should not move toward similar methods especially concerning questions of the type addressed here.

Action Editor

Michael F. Pogue-Geile served as action editor for this article.

Author Contributions

Both authors developed the study concept. Data analysis was performed by D. Dodell-Feder under the supervision of L. Germine. D. Dodell-Feder drafted the manuscript, and L. Germine provided critical revisions. Both authors approved the final manuscript for submission.

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Declaration of Conflicting Interests

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

Supplemental Material

Additional supporting information can be found at http://journals.sagepub.com/doi/suppl/10.1177/2167702618773740

Open Practices

All data have been made publicly available via Open Science Framework and can be accessed at http://osf.io/5bpr7. The complete Open Practices Disclosure for this article can be found at http://journals.sagepub.com/doi/suppl/10.1177/2167702618773740. This article has received the badge for Open Data. More information about the Open Practices badges can be found at https://www.psychologicalscience.org/publications/badges.

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