ann. behav. med. (2023) 57:86–92
https://doi.org/10.1093/abm/kaac008

BRIEF REPORT

First-generation College Students Have Greater Systemic Inflammation than Continuing-Generation College Students Following the Initial College Transition: A Brief Report

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Published online: 21 April 2022

Abstract

Background First-generation college students ("first-gens") are often at a disadvantage socially and academically; whether they are at risk physiologically is unknown despite the well-established link between greater education and better long-term health.

Purpose To examine whether first-gens have higher levels of cardiovascular disease (CVD) risk markers relative to continuing-generation college students ("continuing-gens").

Methods A panel of CVD risk markers was assessed among 87 emerging adults (41 first-gens) twice over their first year of college.

Results Compared to continuing-gens, first-gens had greater systemic inflammation (composite of averaged *z*-scores for C-reactive protein and interleukin-6; $B = 0.515$, SE = 0.171, $p = .003$) during the fall but not spring semester ($p > .05$). Associations were independent of family home ownership and childhood adversity, even though first-gens were more likely to live in rental homes and reported riskier home environments. Lower childhood subjective social status (SSS) accounted for greater systemic inflammation among first-gens as evidenced by an indirect effect of college generation status on systemic inflammation through childhood SSS ($a_1b_1 = 0.261$, bootstrapped SE = 0.103, 95% boot CI [0.078, 0.482]). There were no differences in metabolic risk and latent virus regulation by college generation status in either semester ($p > .10$).

Conclusions This is the first study to find that first-gens have higher levels of systemic inflammation than continuing-gens following the college transition and that childhood SSS may be one explanatory pathway. First-gens may benefit from university resources that address social class differences, which should be provided early on so that first-gens can reap the health-relevant benefits of higher education, at least in the short term.

Keywords First-generation college students · Systemic inflammation · Subjective social status

Introduction

A quarter of emerging adults at 4-year institutions are first-generation college students ("first-gens"), meaning neither of their parents has a 4-year degree [1]. Compared to continuing-generation college students ("continuing-gens"), first-gens are often at a disadvantage academically and socially. They are more likely to come from working-class families who may have access to fewer resources [2], report greater psychosocial stress [3] and are more likely to withdraw from college early [4]. Fortunately, a wealth of research has subsequently focused on creating interventions to better support first-gens' academic pursuits and well-being [5, 6].

Despite this needed research, first-gens' physical health has been largely overlooked. This is concerning given the established link between more years of education and better long-term health, including fewer proximal biological risk factors (e.g., systemic inflammation [7]) and lower rates of chronic diseases (e.g., cardiovascular disease; CVD [8]). Indeed, only two studies have considered the role of college generation status on physical health outcomes. Stephens et al. [9] found that first-gens had greater physiological stress reactivity (i.e.,

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greater salivary cortisol production) to a social stressor that evoked little to no physiological stress reactivity among continuing-gens. Importantly, differences were eliminated after a brief intervention in which first-gens were encouraged to view their social class backgrounds as sources of strength [6]. We extend Stephens et al.'s findings by comparing first- and continuing-gens on a panel of metabolic (i.e., resting systolic (SBP) and diastolic blood pressure (DBP), heart rate (HR), waist-to-hip ratio (WHR), total/HDL cholesterol, glycated hemoglobin (HbA1c)), and inflammatory markers (i.e., C-reactive protein (CRP), interleukin-6 (IL-6)) as well as exposure to and regulation of Epstein–Barr virus (EBV) assessed twice over the first year of college. When elevated, these measures are associated with greater CVD risk [10–12]. We also examine whether broader group differences in SES and psychosocial factors (childhood adversity [13]), might explain differences in CVD risk between first- and continuing-gens.

Why Might First-gens have Elevated CVD Risk Markers?

Compared to continuing-gens' parents, first-gens' parents have completed fewer years of education. Lower parent educational attainment has been cross-sectionally associated with greater viral load among adolescents [14], higher CRP [15] and greater relative risk for metabolic syndrome, a cluster of elevated metabolic risk markers including high BP, cholesterol, and poorer blood glucose regulation, among young adults [16].

Additionally, parent educational attainment is often used as a proxy of childhood socioeconomic status (SES) either alone or along with other measures, such as income or home ownership. Meta-analytic evidence supports a link between lower SES and poorer physical health outcomes including higher CRP and IL-6 [7], incidence of metabolic syndrome [17], and greater exposure to and poorer regulation of EBV [18], the virus that causes mononucleosis. Lower *childhood* SES in particular has been associated with central adiposity [19], systemic inflammation [20], and the development of CVD among adults [21]. Socioeconomic disadvantage in childhood and across the lifespan is thought to confer risk for poorer health outcomes in part through health behaviors, more limited access to financial and social resources, and exposure to uncontrollable psychosocial stressors and environmental toxins [8, 22]. Similarly, exposure to childhood adversity, which may co-occur with lower childhood SES [13], has been tied to CVD risk [23].

First- and continuing-gens may also differ in *subjective* social status (SSS), meaning how people perceive their place in society relative to others. These beliefs are often informed by overt and covert societal messages that communicate who, because of various sociocultural characteristics (e.g., education, employment, race) has more or less value, power, or control [24, 25]. Because first-gens' parents do not have 4-year degrees, they may be more likely to work in positions that, albeit essential, may be viewed as less prestigious relative to positions that require a college degree. Meta-analytic evidence links lower SSS to poorer self-rated health, health symptoms and to certain biological CVD risk factors [24], such as inflammation [26, 27]. It is thought that lower SSS may contribute to poorer health by engendering feelings of inadequacy and helplessness, potentially promoting a state of physiological and psychological arousal and distress [22, 25].

Research Aims and Hypotheses

Here, we compare first- and continuing-gens on a panel of metabolic and inflammatory markers as well as exposure to and regulation of EBV to examine whether CVD risk differs as a function of college generation status and whether socioeconomic (i.e., home ownership, childhood SSS) and related psychosocial factors (i.e., childhood adversity) may underlie potential group differences. Based on prior work, we hypothesize that first-gens will have elevated CVD risk markers compared to continuing-gens, and that these differences may be reflective of broader group differences in subjective and objective SES and adversity exposure.

Methods

Participants

At a public university, 87 emerging adults were followed over their first two semesters. Participants needed to be first-year college students at the main campus, free of serious physical illnesses and between 18 and 21 years of age, to make the sample more representative of full-time students at 4-year institutions and reduce the likelihood that potential differences in CVD risk would be confounded by developmental differences [28]. Students were ineligible if they participated in a 6-week on-campus summer program. Please see Table 1.

Procedure

Eligibility and demographic information were collected via an online survey. Participants attended visits at the university's Clinical Research Center in October 2019 and February 2020. Visits were held prior to semester breaks and finals, which could have confounded study findings [29]. All visits took place before the transition to remote learning due to Covid-19. Participants were asked to abstain from eating, drinking (except water),

Table 1. Sample Descriptives.

	First-generation college students	Continuing-generation college student
	(*n* = 41; 47.1%)	(*n* = 46; 52.9%)
	n (%) or *M* (SD)	*n* (%) or *M* (SD)
Sociodemographic characteristics		
Racial/ethnic minority	19 (46.3)	13 (28.3)
Women	**38 (92.7)**	**31 (67.4)**
Age	18.15 (0.42)	18.09 (0.29)
Body mass index (fall/spring)	23.72 (4.67)/ 24.41 (4.80)	24.02 (3.41)/ 24.51 (3.45)
Hours since awaking (fall/spring)	4.24 (2.71)/ 4.40 (2.43)	4.69 (2.40)/ 4.48 (1.99)
Family currently renting home	**9 (22.0)**	**2 (4.3)**
Childhood SSS	**5.73 (1.90)**	**7.37 (1.18)**
Risky family environment	**2.12 (0.79)**	**1.82 (0.57)**
CVD risk markers (fall/spring)		
Metabolic composite	-0.029 (0.516)/0.006 (0.474)	.025 (0.505)/ -0.001 (0.545)
Inflammatory composite	.289 (0.885)/0.177 (0.845)	-0.245 (0.712)/ -0.138 (0.778)
EBV seropositivity (≥22 RU/mL)	25 (61.0)/ 23 (56.1) §	22 (47.8)/ 24 (52.2)
Among seropositive participants, number of EBV-CA IgG (RU/mL)	Log: 2.11 (0.33); Raw: 195.40 (299.03)/ Log: 2.19 (0.36); Raw: 241.33 (317.50)	Log: 2.22 (0.29); Raw: 213.80 (202.21)/ Log: 2.23 (0.29); Raw: 222.02 (208.51)

Note: First-generation college students = participant whose parents do not have 4-year degrees; continuing-generation college student = participant who has at least one parent with a 4-year degree; *SSS* subjective social status; *CVD* cardiovascular disease; *EBV-CA IgG* Epstein Barr Virus capsid antigen immunoglobin G. A metabolic composite was created by taking the mean of *z*-scores for SBP, DBP, HR, WHR, Total/HDL cholesterol ratio, and HbA1c. Similarly, an inflammatory composite was created by taking the mean of *z*-scores for log-transformed CRP and IL-6 in the fall semester**. Significant group differences ($p < .05$) in sociodemographic characteristics are bolded**. Based on independent-sample *t*-test and chi-square tests, first-generation college students were more likely to be women ($\chi^2(1, N = 87) = 8.451$, $p = .004$), live in rented homes ($\chi^2(1, N = 87) = 6.082$, $p = .014$), and report lower childhood SSS ($t(65.43) = -0.4.765$, $p < .001$) and riskier family environments ($t(71.88) = 1.999$, $p = .049$) compared to continuing-generation college students in the sample.

§ 37 first-generation college students and 45 continuing-generation college students were retained for the spring visit. The 5 participants (4 first-gen) who were unable to participate in the spring did not significantly differ from participants who completed both visits on any study variables. The reason for fewer seropositive first-gen participants in the spring is due to two seropositive participants withdrawing from the study.

or exercising within 1 h of their visit and were rescheduled if sick or injured [30]. Visits were 70–90 min and followed the same format. After consent, anthropometric measurements, BP and HR were taken, a nurse drew participants' blood via antecubital venipuncture, and participants completed questionnaires and a semi-structured interview about their background and college experiences. Both fall and spring semester visits took place between 8:30 AM and 5:45 PM. Participants were compensated up to $60. The Pennsylvania State University's Institutional Review Board approved the study.

Measures

College generation status

As part of the eligibility survey, participants indicated their parent(s)' highest level of educational attainment. Participants were categorized as first-gen if neither parent had a 4-year degree and continuing-gen if at least one parent had a 4-year degree [1]. Given the study focus, first-gens were oversampled to represent nearly half the sample (47.1%).

CVD risk markers

Metabolic markers. After a 5-min resting period, BP and HR were taken 4 times over a 6-min period using a Dinamap V100 automatic BP monitor and occluding cuff (GE Healthcare; Chicago, IL, USA). Mean scores were calculated from the last three readings to estimate resting SBP, DBP, and HR, respectively. Waist circumference was measured at the midpoint between the hipbone and lowest rib and hip circumference at the widest part of the buttocks. WHR was calculated by dividing waist by hip circumference. Lipids were measured in serum using spectrophotometry and total cholesterol was divided by HDL cholesterol to calculate the total/HDL cholesterol ratio. HbA1c was measured in whole blood using immunoturbidimetry.

Immune markers. Blood samples were centrifuged at 3200 rpm for 15 min (Quest Diagnostics Horizon Centrifuge Model 642E) 30–60 min after the blood draw. Serum was then immediately stored at −20°C before being transferred to a −80°C freezer at the end of each day for long-term storage. Samples were analyzed within 13 months and run in duplicate for each analyte using high-sensitivity enzyme-linked immunosorbent assay kits (see Supplemental file). CRP and IL-6 were used to measure systemic inflammation. We also measured EBV exposure and regulation, as indexed by the number of EBV capsid antigen (CA) immunoglobulin G (IgG), which are present upon exposure and maintained at detectable doses for life [31].

Covariates

Participants reported their age, race, ethnicity, gender, whether they regularly smoked cigarettes and when they woke up on the day of their visit. Their temperature was taken and their height and weight were measured to determine body mass index (BMI, kg/m^2).

Explanatory pathways

During the fall visit, participants indicated whether their parent(s) owned or rented their home. They also completed the MacArthur Scale of Subjective Social Status that was developed for use with youth who are not financially independent from their parents and has strong convergent and divergent validity [32]. On a 10-rung ladder, participants marked where they felt their family stood in relation to other families in their community, with higher rungs representing higher SSS. Participants also completed the 13-item Risky Families Questionnaire [33], which measures exposure to psychosocial adversities at home on a 5-point scale. higher mean scores reflected riskier family environments. The scale had strong internal consistency ($\alpha = 0.87$).

Statistical Analyses

CRP, IL-6, and EBV-CA IgGs were log-transformed to normalize their distributions. An inflammatory composite for each semester was created by taking the mean of *z*-scores for log-transformed CRP and IL-6 (Pearson's $r = 0.39$ and0.34 for fall and spring, respectively). Similarly, a metabolic composite for each semester was created by taking the mean of *z*-scores for SBP, DBP, HR, WHR, total/HDL cholesterol ratio, and HbA1c (Cronbach's $\alpha = 0.43$ for both fall and spring). Exposure to and regulation of EBV were examined separately. Multiple linear regression was used to assess associations between college generation status and the metabolic and inflammatory composites for each semester. Logistic regression was used to model odds of being EBV seropositive. Associations between college generation status and EBV-CA IgG levels were only modeled among seropositive participants using linear regression. For significant effects only, we separately added family home ownership, childhood SSS and risky family environment to examine whether SES proxies and childhood adversity explained associations between college generation status and CVD risk. To assess whether there was an indirect effect of being first-gen on inflammation through childhood SSS, we used PROCESS [36] to fit mediation models using 10,000 bootstrapped samples and estimating 95% bias-corrected confidence intervals (CI) around indirect effects (see Supplemental file for nonsignificant results of mediation models through family home ownership and risky family environment). Models

adjusted for race/ethnicity (1 = racial/ethnic minority, 0 = white nonHispanic) and gender (1 = female, 0 = male). Models for inflammatory composites also adjusted for BMI and hours since awaking [34, 35]. No participants were excluded for fevers. See Supplemental file for post hoc sensitivity analyses based on CRP levels as well as results for individual metabolic and immune markers.

Results

Eighty-seven participants completed the fall semester visit and 82 were retained in the spring. The five participants who were unable to attend spring visits did not differ from the remaining 82 participants on relevant study variables (all *p*s > .10; see Supplemental file). Across all participants, first-gens were more likely to be women ($p = .004$), live in rental homes ($p = .014$), and report lower childhood SSS ($p < .001$) and riskier family environments ($p = .049$; see Table 1).

Main Effects

Being first-gen was associated with greater systemic inflammation ($B = 0.515$, SE = 0.171, $p = .003$) in the fall, but not spring ($p > .05$). College generation status was not associated with the metabolic composite, EBV seropositivity, or number of EBV-CA IgG antibody titers among seropositive participants in either semester (*p*s > .10; see Supplemental file).

Explanatory Pathways

First-gens had higher systemic inflammation independent of family home ownership ($B = 0.536$, SE = 0.176, $p = .003$) and childhood adversity ($B = 0.501$, SE = 0.174, $p = .005$), but not childhood SSS ($p > .10$). When fitting mediation models using PROCESS [36], there was support for an indirect effect of college generation status on systemic inflammation in the fall through childhood SSS, such that first-gens reported lower childhood SSS and lower childhood SSS was in turn associated with greater systemic inflammation ($a_1b_1 = 0.261$, bootstrapped SE = 0.103, 95% boot CI [0.078,0.482]).

Discussion

We sought to extend prior research on first-gens' physical health by comparing first- and continuing-gens on a panel of biological CVD risk markers collected twice over the first year of college. First-gens had higher levels of systemic inflammation in the fall compared to continuing-gens, similar to Stephens et al.'s [9] finding of first-gens' greater cortisol production in response to a laboratory stressor. Although different outcomes, systemic inflammation and greater physiological stress reactivity (especially to frequent stressors) are generally considered detrimental for physical health and may reflect physiological wear and tear [37]. Differences in inflammation were explained by lower childhood SSS ratings among first-gens. Although no studies link college generation status to SSS specifically, our results align with work connecting first-gen status to lower *objective* SES, such as working-class status and fewer family assets [2, 4], and complement recent findings of lower SSS associating with higher CRP [27] and greater IL-6 production following laboratory stressors among US adults [26].

Importantly, differences in systemic inflammation were limited to the fall suggesting that the physiological impact of the college transition on first-gens' immune system may be short-lived. Compared to continuing-gens from more socially advantaged backgrounds, the college transition may be more stressful for first-gens. For example, adults who rated themselves lower in SSS had greater IL-6 production in response to a social stressor partly because they rated the stressor as more threatening [26]. Coupled with Stephens et al.'s findings [6, 9], it may be that the college transition, which includes interacting with peers from more educated families and navigating new university cultural norms, may make first-gens' childhood SSS more salient [2]. This in turn could contribute to greater psychosocial stress and systemic inflammation [22], highlighting the need for interventions focused on social class early on in the college transition.

Nonetheless, observed differences in systemic inflammation may have been present prior to college, perhaps due to differing childhood experiences or lifestyle factors between first- and continuing-gens that we did not consider here. For example, first-gens may have entered college with greater systemic inflammation and benefitted from leaving a potentially more stressful home environment, thus resulting in first-gens having similar levels of systemic inflammation to continuing-gens by spring. Baseline levels of systemic inflammation and information on overall well-being and health behaviors prior to college is needed to discern when, and why, differences in systemic inflammation emerge between first- and continuing-gens.

Notably, first- and continuing-gens did not differ on metabolic risk markers, odds of EBV exposure, and number of EBV antibody titers. This could mean that college generation status may matter more for systemic inflammation or perhaps that differences in inflammation emerge prior to differences in metabolic indicators [38]. Although the weak internal consistency for metabolic composites could partly explain the null effects, this is unlikely as there were null findings regardless of whether metabolic markers were examined in a composite or individually (see Supplemental file). Nonetheless, future studies should follow students over a longer time to assess whether group differences in metabolic indicators and

latent virus regulation emerge later on given the graded association between SES and long-term health [8].

A primary strength of this study is the assessment of a panel of metabolic and systemic inflammation markers and exposure to and regulation of EBV, all of which are associated with CVD risk. By restricting the sample to first-year students only, results are less susceptible to survival bias as may have been the case had we recruited emerging adults who were further along in their college careers. Ninety-four percent of participants were retained for the spring semester visit and all fall and spring visits were conducted within several weeks of each other, reducing the likelihood that differences in systemic inflammation reflect differences in assessment timing. Finally, home ownership and childhood SSS may be more accurately recalled by emerging adults compared to other SES proxies (e.g., family income).

This study is not without its limitations. The small sample size prevented the assessment of differential effects of college generation status on CVD risk depending on whether first-gens identified with racial or ethnic minority groups or had parents with no versus some post-secondary education. Given the composition of the sample and that all participants came from the same university, findings may not generalize to first-gens who are men, nonbinary, older than 21, or who attend different institutions, such as community colleges or universities where first-gens make up a larger percentage of the population. This is an observational study so we cannot infer causality. As such, the significant indirect effects of college generation status on differences in systemic inflammation through childhood SSS should be considered preliminary given their cross-sectional nature. Additionally, we do not have information on health and health-relevant factors (e.g., diet, sleep) prior to and during the college transition to eliminate alternative pathways. Similarly, certain CVD risk factors (e.g., HbA1c) may reflect health both prior to and during the college transition. These limitations speak to assessing emerging adults' physical health over a longer time to disentangle timing and underlying mechanisms. Composite scores were created to reduce risk for type 1 error; however, replication studies are needed given the small sample size. Beyond SES, there may be other factors (e.g., access to informational support, stereotype threat) that might explain differences in systemic inflammation between first- and continuing-gens, which should be explored in future studies. Finally, spring visits occurred 6 months after college began so we may have missed meaningful differences in CVD risk that may unfold over years and may depend upon whether students remain in college or withdraw after the first year.

Despite these limitations, this study is the first to examine whether first-gens have elevated CVD risk markers compared to their continuing-gen peers and the first to find that first-gens have greater systemic inflammation following the initial college transition. Findings suggest that the benefits of interventions focused on addressing social class inequities in higher education, e.g., those focused on reframing social class differences as sources of strength, may extend to benefits for systemic inflammation among first-gens [5, 6]. Moreover, findings highlight that the term "first-gen" represents much more than parent educational attainment alone. Even though effects of being first-gen on systemic inflammation were independent of home ownership and adversity, it is important to acknowledge that along with lower childhood SSS, first-gens were more likely to be raised in homes their parents rented and to report riskier family environments. Knowing what the term "first-gen" may represent for their students, universities could leverage questions about parent educational attainment (which are often included when enrolling in college) as a means to connect with first-gens and provide them with resources early on in the college transition, which could potentially benefit their physical health.

Supplementary Material

Supplementary material is available at *Annals of Behavioral Medicine* online.

Acknowledgements Research reported in this manuscript was supported by a grant from the National Heart, Lung, and Blood Institute of the National Institutes of Health under award number (F31HL149179 (PI: EJJ; Sponsor: HMCS); 5T32HL007560-37 (PIs: Peter Gianaros & Rebecca Thurston). The content of the manuscript is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Compliance with Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards Authors Emily J. Jones and Hannah M. C. Schreier declare that they have no conflict of interest. All procedures, including the informed consent process, were conducted in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000.

Primary Data XXXXX.

Authors' Contributions Dr. Jones designed the study and collected data under the supervision of Dr. Schreier. Dr. Jones analyzed the data and wrote the original draft of the manuscript and Dr. Schreier reviewed and edited the manuscript. Both authors read and approved the final manuscript.

Ethical Approval All procedures performed in this study were in accordance with the ethical standards of our institutional research ethics committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Consent was obtained from all individual participants included in the study.

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