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A community survey on knowledge of the impact of environmental and epigenetic factors on health and disease

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Abstract

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CONFLICT OF INTEREST

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

ETHICAL APPROVAL

The survey was approved by the University of Cincinnati Institutional Review Board and adhered to ethical guidelines for research with human subjects. Pink Ribbon Girls (PRG) and the Department of Environmental Health were allotted 92 days (1 March to 31 May 2011) for data collection. Minimal demographic information was requested.

Aim—An outreach effort was designed to survey breast cancer survivors, supporters and their families and friends with respect to their interest in, and knowledge of, the potential impact of the environment and epigenetics on health.

Methods—Two nearly identical questionnaires (one for adolescents and one for adults) were designed to gauge the perception of this community as to whether the environment impacts health and cancer risk through processes other than genetics. The questionnaires were filled out at casual social gatherings, fundraisers and wellness campaigns as well as in schools (730 participants). The differences among correct (scientific consensus) versus other responses (incorrect and not known) were evaluated. Each answer was first analysed individually and then grouped into one of three categories (diet, inheritance and environment) with age, race and gender. Differences for each response, question or group were compared by repeated measures analysis of variance.

Results—Respondents generally acknowledged that many factors could be associated with breast cancer although answers to key questions related to epigenetics based on diet, inheritance and environment were often incorrect or not known. The adult participants tended to answer more questions correctly than adolescents did. The majority of participants preferred the Internet as a major source for obtaining further information.

Conclusion—The growing awareness and educational needs for adolescents may bring new paradigm-related environmental risk factors, which may minimise negative epigenetic outcome in subsequent generations. There is an educational opportunity, especially using electronic media, for public education concerning the impact of the environment on human health.

Keywords

breast cancer; diet; environment; epigenetic; outreach; education

INTRODUCTION

The Developmental Origins of Health and Disease (DOHaD) theory describes how during early life the environment induces changes in development that have a long-term impact on later health and disease risk.[1,2] Parental lifestyle, for example, diet, smoking, drinking, obesity and exposure to endocrine disruptor chemicals, has been shown to modulate disease risk.[3–9] It is thought that some of these developmental alterations come about through changes in the activity of genes through epigenetic processes.[10]

The epigenetic code literally means ‘above’ the genome and does not affect the information recorded in DNA sequence. Epigenetics can modify the genome and ensure which genetic information (genotype) can be accurately translated into biological function (phenotype) in the right place (tissues or organs) at the right time (stages of development). The sequence of the DNA stores all the data necessary to build a living cell or organism. Epigenetics, like the operating system of a computer, decodes the information and determines when, how and where a given set of instructions must be executed.[11,12] The epigenetic code can be altered by environmental exposures, such as chemicals, nutrition and stress, at different life stages from conception to puberty and from adulthood to ageing. Such exposures can have profound and long-lasting impacts on gene expression across multiple generations.[13,14] Accumulated evidence suggests that epigenetics is the key to understanding such trans-

generational influences. A classic example was the study on diethylstilbestrol (DES). Gestational exposure to DES resulted in elevated incidence of clear cell carcinoma of the vagina and breast cancer in the offspring.[15–17] These studies were the milestone to open an avenue in the concept for reprogramming (epi)genetic memories after early exposure, which may eventually augment risk of disease development. These studies also identified that *in utero* was the most critical exposure window in each individual life, and these reprogrammed memories can be transmitted along different life stages. Disease development may be expedited with the second hit (e.g. lifestyle change and excess UV exposure), especially during the critical *disease susceptibility window*. The prevailing hypothesis is that prenatal exposure to dietary compounds or any environmental factors can cause prolonged changes in gene expression through methylation of DNA bases (i.e. 5' methylation of cytosine in CpG dinucleotides) or modifications of specific amino acid residues in histone proteins (e.g. acetylation or methylation at fourth lysine of histone 3 protein). Some of these epigenetic changes can be transmitted from generations to generations in experimental models.

The extent of awareness and understanding regarding epigenetics, the implications for epigenetic changes to affect disease risk factors and the possibilities for preventive measures can be tailored for each population. Spector *et al.*[18] and Romani *et al.*[19] established the studies on epigenetics and the breast cancer risk perception on public health perspectives. Based on these studies, an understanding of a woman's breast cancer risk perception has been described as complex and multifaceted. A better understanding of the aetiology of breast cancer and personal risk assessment is greatly needed. Therefore, an accurate assessment of genetic and epigenetic knowledge is critically important for educators to tailor policy or educational programmes for helping the population at large as well as women with a family history of breast cancer.

In this study, we collaborated with Pink Ribbon Girls (PRG, Cincinnati, Ohio chapter) as the outreach community partner. PRG is a non-profit breast cancer advocacy organisation whose mission is to provide personalised assistance to young patients throughout all phases of the breast cancer journey – including education, awareness and one-on-one support for survivors and their families. PRG's primary outreach seeks to raise awareness of breast cancer in young women at health fairs, social gatherings and classroom presentations.

The aim of this pilot outreach project was to identify areas and age groups for targeting educational materials about epigenetics and modifiable disease risk and to provide researchers with specific topics of greatest educational need and interest to the respondents. PRG utilised its existing community events scheduled for the spring of 2011 to enrol participants in this study. Respondents filled out a single-page survey that contained questions aimed at assessing the breadth of their understanding of epigenetic effects on health and disease and the extent to which they perceived those events could modify their disease risk. Questions also sought to ascertain whether respondents recognised epigenetics as a mechanism for multi-generational effects of environmental exposure on disease, particularly on breast cancer.

MATERIALS AND METHODS

Population

The questionnaires requested age, gender and race as identifiers. Age groups 14–19, 20–24, 25–34, 35–44, 45–54, 55–64, 65–74 and 75+ years were later collapsed as follows: 14–19, 20–24, 25–44, 45–54 and 55+ years. Initial race categories were Caucasian, African American, Hispanic and Other but were rearranged to the following: Caucasian, African American, Other and Missing race (the latter being respondents who did not provide race information on the survey). The socioeconomic background of respondents ranged from working class to upper middle class. The educational level for adult participants in general was above high school level. Among respondents, 40 described themselves as in health-care work.

Questionnaires

The purpose of the study was to assess the PRG community about the influence of several environmental factors on general health and specifically breast cancer risk. It also probed the respondents about their understanding of developmental stages with heightened vulnerability to environmental exposure, that is, ‘windows of susceptibility’, such as during pregnancy, foetal growth and puberty. Perceptions were queried about non-DNA-transmitted inherited traits and paternal impact on health. The questions were designed by representatives from the PRG (B.B. and T.M.) and breast cancer researchers (V.G., Y.-K.L., M.M., L.L., S.M.P., S.-M.H.) at the University of Cincinnati to determine which audience was more appropriate to benefit from educational events and targeted media than another as a way to increase their awareness that their environment can influence their epigenome in a complex and multi-generational way. Questions were designed to be brief, to target relevant but common beliefs and presuppositions, as well as to stimulate thought.

Two nearly identical surveys were used except for minor word changes of some questions – one for adolescents and one for adults. Most adolescents (n = 309, ages 14–19) and some very young adults (n = 28, ages 20–24) chose to complete the adolescent survey (n = 337). The choice of adult versus adolescent survey was optional, and due to the similarity in the responses among 20- to 24-year-olds, the data were pooled by age into 14- to 19-year-olds and 20- to 24-year-olds regardless of which survey was chosen.

Questions 1–10 are shown in Table 1 along with the (correct) percentage of the responses from the participants. (Correct was defined as the answer that best fits current scientific consensus (correct (C)) highlighted.) These 10 questions focused on the individual and multi-generational effects of environmental exposures on disease but were also loosely grouped into three broader categories: dietary environment (Q2 – My diet affects my child only while I am pregnant with her; Q5 – If I drink during pregnancy, I can only harm my child but not my grandchild; Q7 – My diet does not affect my daughter and my granddaughter; Q8 – A low-fat diet during pregnancy helps lower my son’s breast cancer risk), genetic inheritance and environment (Q9 – If I don’t get breast cancer, my daughter won’t get it, either; Q10 – Identical twins have the same risk for disease) and toxins and environment, specifically cigarette smoke (Q1 – Damage from the environment to my

mother was not transmitted to my child; Q6 – My smoking affects the birth weight of my son but not his DNA). One of three responses could be offered for each question: ‘Agree’, ‘Disagree’ and ‘Don’t Know (DK)’. The percentage of correct responses for each category was calculated overall (Table 1) and also by age, race and gender (Table 2).

In addition, boxes were provided for respondents to check whether they were affiliated with health-care professions, whether they were breast cancer survivors and whether they had ever heard the words ‘epigenetics’ or ‘epigenome’ before. Also, respondents were asked their preferences for receipt of educational media whether by phone/ email, group presentation, video, internet or brochure, as well as an indication of their interest in learning more about the questionnaire topics. The latter was presented using a visual scale of 1–5, where 1 = not interested, 3 = somewhat interested and 5 = very interested.

The events

The surveys were distributed *ad-lib* at fundraisers, wellness campaigns – including the ‘Looking Upstream for Environmental Causes of Breast Cancer’ event held in 2011 and funded by the Breast Cancer and the Environment research programme – high school class events, casual social venues and family gatherings. The administration of Mother of Mercy High School in Cincinnati, Ohio, handed out the surveys to students as well as teachers and assisted with the collection of forms from 50 students apiece from grades 9, 10, 11 and 12. Surveys were also completed voluntarily at several University of Cincinnati fraternity and sorority meetings.

Data analysis

Adolescent and adult surveys numbered 368 each. Hard copies of the surveys were reviewed with respect to completeness and consistency; questionnaires with more than five unanswered questions were removed (6 out of 736). A quality control check of the accuracy of data entry was completed by re-entering a 10% random sample of the hard copies from each type of survey, confirming 100% agreement between the original and randomly chosen samples.

The differences in percentages of correct (agreement with scientific consensus) responses versus other responses (DK and Agree) across all questions were evaluated by Pearson’s chi-square statistics testing equality of percentages between two independent multinomial samples. In order to include all response categories (‘Agree’, ‘Disagree’ and ‘DK’) in measuring differences among responses to questions across age, race and gender, response choices were coded (non-consensus Agree/Disagree = 0; DK = 0.5; or consensus Agree/Disagree = 1). The mean value was obtained for each of the 10 questions separately based on ‘Agree’, ‘Disagree’ and ‘DK’. This category was further analysed based on age, race and gender. Differences among questions for each group and average response levels across questions were compared by repeated measures analysis of variance.

RESULTS AND DISCUSSION

The primary goal was to determine respondents’ level of understanding of the impact of environment and epigenetics/epigenome on health in generations. The research and

community partners sought to determine the most appropriate way to disseminate educational outreach materials for this specific purpose. Some questions sought to assess whether subjects recognised heightened periods of environmental-exposure vulnerability, such as the foetal period, which represents one of several ‘windows of susceptibility’ to epigenetic change, bringing to light the respondents’ basic understanding of epigenetics.

Questions 1–10 are shown in Table 1 along with the percentage of those who ‘Agree’ with each statement or ‘Disagree’ or ‘DK’. The scientific consensus was indicated as ‘C’ and lightly shaded. Table 2 illustrates the analysis of consensus answers to each question by age, race and gender; Table 3 represents the percentage of responses to all questions (Q1–Q10) and questions categorised as disease inheritance (Q9 and Q10), diet (Q2, Q3, Q4, Q5, Q7 and Q8) or the environment (Q1 and Q6). Responses were stratified by age, race and gender.

The majority of participants answered questions 1, 2, 4, 7 and 9 according to consensus (54.9%–91.9%), but over 60% of participants incorrectly answered questions Q3 and Q8. (Q3 stated, ‘My daughter is not affected by her father’s eating habits’, and Question 8 stated, ‘A low-fat diet during pregnancy helps lower my son’s breast cancer risk’.) This suggests a perception that a father’s eating habits have little effect on his daughter’s health and suggests a general lack of understanding that (1) men can get breast cancer and/or (2) dietary-modified epigenome from males can be transmitted to their daughter. These perceptions were not statistically differently by age, race or gender. A plausible explanation is that the impact of paternal diet on female offspring’s disease risk and understanding of maternal dietary impact on male breast cancer risk may not be clearly understood by this community. A significant gap in public understanding exists about paternal and maternal dietary impact on disease risk of offspring.

Q9 and Q10 also showed wide disparity in the percentage of correct responses. Q9 and Q10 were designed to discern whether environmental impact on health was perceived to be mostly dependent on strict DNA inheritance (Q9 stated, ‘If I don’t get breast cancer, my daughter won’t get it, either’) or whether environment could alter the course of disease by other processes (other than inherited predisposition and DNA mutations), such as epigenetics. Over 90% of participants answered Q9 correctly, understanding that environmental factors could bring about breast cancer that was solely attributable to DNA (Table 2). Differences in the percentage of correct responses between Q9 and Q10 are significant (90.9 + 1.5 (Q9) versus 39.2 + 2.5 (Q10), $p = .048$, (paired t-test), not dependent on age, gender and race (Table 3)). It seemed to be clear to most respondents that other factors specifically impact breast cancer risk. Table 3 shows some difference between females (66.9% correct) and males (55.1% correct) in terms of recognising the role of inheritance in breast cancer risk (Q9 and Q10). Although there was no significant difference, this observation points to a possible area for future intervention for educating young men in particular about epigenetics and the multi-generational effects of environmental impacts on health and disease. When questions were grouped (as inheritance, diet and environment), responses from young adults showed a trend towards increasing the ‘correct’ percentage in the adult group (25–55+ years) versus adolescents (14–24 years) for overall questions (Table 3). Interestingly, a higher percentage of people from the adult group correctly answered two environment-related questions (Q1 and Q6) than did the adolescents, suggesting the

adolescent population was less aware of the environmental impact on their health. There was no significant difference in percentage of correct answers across race and gender (Table 3).

The number of respondents who left race unchecked was considerably higher than the number who left age and gender unchecked. One possible explanation is that privacy was deliberately sought rather than being inadvertently missed. In data analyses, however, it also was found that the incidence of 'missing values' in surveys of those respondents who did not indicate race was higher than for those who did check race.

Whether participants were interested in receiving more information about epigenetics and what type of educational material they would prefer to receive, according to the survey (Table 4), were almost unanimously neutral with the average score of 3.4 out of 5 showing only modest desire for further information on the topic. Based on the chi-square test for independence, there is a significant difference between survivors and non-survivors in accuracy in answering the survey questions ($p = .0407$). More survivors answered 'correctly' (~28.1%), and fewer answered 'DK' (8.8%), where the reverse pattern was observed in the non-survivor group (16.0% and 22.1%, respectively). This suggests that survivors were more aware of the topic of 'epigenetics' and other factors that influence breast cancer and disease risk. Similarly, there was a significant difference between survivors and non-survivors in terms of 'interest' in learning more about the survey topics. When data were stratified into 'high' (score ≥ 4) versus 'low' (score < 4) interest, survivors ($p = .0254$, based on Fisher's exact test) tended to show more interest in knowing more about epigenetics when compared with the non-survivor group.

However, definite preferences emerged for certain types of electronic media as a way to receive information on breast cancer, epigenetics and health. Younger individuals preferred videos (39.9%) and websites (49.8%); older individuals (aged 55+ years) were only slightly less enthusiastic about electronic formats in general (Table 5). Differences between adolescents and adults on this question were not extreme with the exception that 14- to 24-year-olds were more receptive to the use of educational videos. Overall, electronic media was a preferred source for their interest in learning more about the questionnaire topics and therefore is more likely successful as an educational outreach method.

Survey data reflected different levels of knowledge about the impact of environment on disease risks, with adults being slightly (but significantly) better able than adolescents to correctly answer questions about diet, environmental exposures and their impact on disease. The growing awareness and educational needs for adolescents may bring new paradigm-related environmental risk factors, which may minimise poor epigenetic outcome in subsequent generations. With respect to gender and race, there are no conclusive data. The survey illuminated particular knowledge gaps and areas of 'low information' (e.g. paternal contributions to cancer risk, breast cancer risks as they relate to males and male offspring and the role of epigenetics among persons with identical DNA) and affirmed the use of educational materials produced specifically for electronic media, including social media (e.g. YouTube, Facebook and Twitter). The youngest respondents indicated a preference for video presentations as opposed to static formats. Fullenkamp *et al.*[20] observed that among Appalachian subjects, they preferred paper/brochures for receiving information about

genetics and the environment, so each community should be assessed individually. Nonetheless, the largely urban and suburban young adult population surveyed here in this study (southwestern Ohio) clearly preferred electronic media. This might reflect disparate levels of access to personal computers, ‘iPhones’ and other devices for viewing and/or interacting with online resources.

This pilot study has several limitations. It was conceived and executed within a time frame assigned by the institutional review board at the beginning of an outreach activity to guide the production of educational media. The sample population for this study contained some biases in the adolescent population, which would have mirrored the distribution of schools and functions at which surveys were collected. It was weighted towards young adult Caucasian females based on the latter and on member-composition of the PRG organisation itself but represented a random sample. From this pilot project and the non-representation of certain racial and ethnic groups, the results are applicable to a select group overall. Nevertheless, the survey illuminated important topic areas and vehicles for future educational outreach efforts within this same community. A single educational outreach rarely receives a broad audience, but results could be extrapolated for similar communities. Despite several limitations, this study provided the public with an understanding of the connection between the epigenome and health and how lifestyle changes may modify gene expression and alter disease risk both for the individual and for future generations.

CONCLUSION

It is apparent that there are large gaps in knowledge of environmentally modifiable disease risk and that no single educational medium will effectively serve all. The challenge is (1) to create heightened interest in the role of the environment in disease risk, (2) to generate a thirst for knowledge of health issues and (3) to inspire participation in changing one’s epigenetic impact on future generations for the better. Advocacy groups like PRG often represent willing and supportive communities from which to draw out information and provide education.

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Table 1

Percentage of 730 participants who checked ‘Agree’, ‘disagree’ or ‘don’t Know (dK)’ to each question.

	Question	Agree (%)	disagree (%)	dK (%)
1	Damage from the environment to my mother was not transmitted to my child	19.5	54.9 **(c)**	25.6
2	My diet affects my child only while I am pregnant with her	24.7	61.5 **(c)**	13.8
3	My daughter is not affected by her father’s eating habits	60.1	12.6 **(c)**	27.3
4	My daughter’s DNA is not affected by her father’s drinking habits	18.5	63.6 **(c)**	18.0
5	If I drink during pregnancy, I can only harm my child but not my Grandchild	14.8	67.1 **(c)**	18.1
6	My smoking affects the birth weight of my son but not his DNA	15.5	61.8 **(c)**	22.7
7	My diet does affect my daughter and my granddaughter	61.4 **(c)**	19.3	19.3
8	A low-fat diet during pregnancy helps lower my son’s breast cancer Risk	26.7 **(c)**	12.7	60.6
9	If I don’t get breast cancer, my daughter won’t get it either	1.6	91.9 **(c)**	6.4
10	Identical twins have the same risk for disease	27.7	39.0 **(c)**	33.3

The correct response is indicated by **(c)** and shading.

Table 2

Analysis of correct answer in each individual question by age, race and gender

category	Questions 1–10[a]										
		Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10	All
Age group[b] (years)	*N*	Mean										
14–19	273	0.56	0.63	0.35 min	0.63	0.79	0.76	0.69	0.56	0.94 max	0.59	0.65
20–24	118	0.63	0.66	0.43 min	0.63	0.67	0.66	0.65	0.51	0.92 max	0.52	0.63
25–44	114	0.81	0.78	0.12 min	0.88	0.76	0.76	0.74	0.58	0.97 max	0.63	0.70
45–54	91	0.79	0.74	0.10 min	0.86	0.82	0.75	0.77	0.63	0.97 max	0.46	0.69
55+	129	0.77	0.71	0.17 min	0.78	0.73	0.70	0.74	0.60	0.96 max	0.52	0.67
Race[b]	Mean											
white	506	0.68	0.70	0.27 min	0.72	0.70.958	0.75	0.71	0.56	0.97 max	0.56	0.67
AA + Other	95	0.68	0.62	0.28 min	0.76	0.70.562	0.62	0.72	0.56	0.92 max	0.51	0.64
Missing	129	0.66	0.66	0.21 min	0.74	0.72	0.72	0.72	0.61	0.92 max	0.58	0.66
Gender	Mean											
Male	79	0.65	0.68	0.35 min	0.68	0.72	0.70	0.69	0.60	0.85 max	0.50	0.64
Female	635	0.68	0.69	0.25 min	0.73	0.77	0.74	0.71	0.56	0.96 max	0.56	0.67
All	730	0.68	0.68	0.27 min	0.73	0.76	0.73	0.71	0.57	0.95 max	0.56	0.66

AA: African American.

Mean values: 0 = Incorrect; 0.5 = Don’t Know; 1 = Correct.

[a]Means differed across questions for all rows ($p < .01$).

[b]Means of all questions differed among age and race groups ($p < .01$). Gender means were not significantly different ($p = .11$).

Table 3

Percentages of correct responses to all questions and to questions grouped as disease inheritance, diet and the environment and stratified by age, race and gender

		All Q1–Q10	**Inheritance Q9, Q10[a]**	**diet Q2, Q3,[b] Q4, Q5, Q7, Q8**	**Environment Q1, Q6**
Age (years)	No. of participants	% Correct, mean (minimum, maximum)			
14–19	273	51.2 (13.2, 90.5)	67.4 (44.3, 90.5)	45.5 (13.2, 72.9)	52.5 (39.9, 64.8)
20–24	118	46.4 (21.2, 87.3)	60.2 (33.1, 87.3)	41.9 (21.2, 55.9)	46.2 (42.4, 50.0)
25–44	114	61.3 (7.9. 95.6)	69.7 (43.9, 95.6)	54.7 (7.9, 82.5)	72.8 (70.2, 75.4)
45–54	91	59.1 (6.6, 94.5)	61.0 (27.5, 94.5)	55.9 (6.6, 80.2)	67.0 (62.6, 71.4)
55+	129	56.8 (10.9, 93.8)	65.1 (36.5, 93.8)	51.9 (10.9, 71.3)	63.2 (58.1, 68.2)
Race					
white	506	54.2 (12.1, 94.1)	66.9 (39.7, 94.1)	48.2 (12.1, 69.)	59.5 (55.3, 63.8)
AA + Other	95	52.3 (17.9, 86.3)	60.0 (33.7, 86.3)	49.6 (17.9, 67.4)	52.6 (49.5, 55.8)
Missing	129	54.7 (10.9, 87.6)	64.0 (40.3, 87.6)	50.5 (10.9, 66.7)	58.1 (52.7, 63.6)
Gender					
Male	79	51.5 (24.1, 75.9)	55.1 (34.2, 75.9)	49.2 (24.1, 62.0)	55.1 (51.9, 58.2)
Female	635	54.4 (10.7, 94.0)	66.9 (39.7, 94.0)	48.7 (10.7, 67.9)	59.2 (55.7, 62.7)

AA: African American.

Age was not reported by five participants. Race was not reported by 129 participants. Gender was not reported by 16 participants.

[a]Difference in the percent correct response between Q9 and Q10 is significant (90.9 + 1.5 (Q9) versus 39.2 + 2.5 (Q10), $p = .048$, (paired t-test).

[b]Q3 (all ages together) indicates that maternal diet is deemed to be unimportant in male offspring's risk for breast cancer. There was no difference in this perception when analysed by gender.

Table 4

Analysis of ‘Interest to learn more about epigenetics’ by age, race and gender

Category	*N*	Mean	Se
Age[a] (years)			
14–19	273	3.2	.06
20–24	118	3.1	.10
25–44	114	3.7	.11
45–54	91	3.8	.11
55+	129	3.8	.11
Race[a]			
White	506	3.3	.05
AA + Other	95	3.7	.11
Missing	129	3.4	.12
Gender[a]			
Male	79	3.1	.13
Female	635	3.4	.04
All participants	729	3.4	.04

SE: standard error; *N*: number of participants; AA: African American.

Interest was reported on an increasing scale from 1 to 5.

Interest was not reported by one participant.

[a]Means differed significantly across age, race and gender groups ($p = .001$).

Table 5

Media source preference by age, race and gender

Category	*N*	Brochure	Website	Group presentation	electronic/ email	Video
Age group (years), % Yes						
14–19	273	30.5	49.8	25.7	18.0	39.9
20–24	118	28.8	41.5	23.7	30.5	11.0
25–44	114	38.6	52.6	17.5	34.2	7.9
45–54	91	36.3	42.9	17.6	28.6	8.8
55+	129	34.9	32.6	18.6	21.7	7.0
Race, % Yes						
White	506	33.9	48.6	23.6	26.1	24.3
AA + Other	95	30.5	35.8	13.0	26.3	14.7
Missing	129	31.0	35.7	17.1	17.8	8.5
Gender, % Yes						
Male	79	30.4	39.2	21.8	22.8	22.2
Female	635	33.8	46.3	22.8	25.2	8.9
All participants	730	32.9	44.7	21.7	24.7	20.3

N: number of participants; AA: African American.

Age was not reported by five participants. Race was not reported by 129 participants. Gender was not reported by 16 participants.