Published in final edited form as:
J Psychosoc Oncol. 2015 January 2; 33(1): 48–65. doi:10.1080/07347332.2014.977419.

What Women Think: Cancer Causal Attributions in a Diverse Sample of Women

Vivian M. Rodríguez, Ph.D.[1], **Maria E. Gyure, M.S.**[1], **Rosalie Corona, Ph.D.**[1], **Joann N. Bodurtha, M.D., M.P.H.**[2], **Deborah J. Bowen, Ph.D.**[3], and **John M. Quillin, Ph.D., M.P.H.**[1]

[1]University Virginia Commonwealth

[2]University Johns Hopkins

[3]University Boston

Abstract

Women hold diverse beliefs about cancer etiology, potentially affecting their use of cancer preventive behaviors. To date, research has greatly focused on the causal attributions cancer patients and survivors hold about cancer, and studies have been conducted primarily with White participants. Less is known about causal attributions held by women with and without a family history of cancer from a diverse community sample. This study sought to identify cancer causal attributions of women with and without a family history of cancer, and explore its relation to socio-cultural factors. Diverse women (60% African-American) recruited at an urban, safety-net women's health clinic (*N*=471) reported factors they believed cause cancer. Responses were coded into nine attributions and analyzed using chi-squares and logistic regressions. Lifestyle-choices (63%), genetics/heredity (34%), and environmental-exposures (19%) were the top causal attributions identified. Women without a family history of cancer were more likely to identify genetics/heredity as an attribution for cancer than women with a history of cancer in their families. Women who identified as White, who had a higher educational attainment, and had commercial insurance were more likely to report genetics/heredity as a causal attribution for cancer. These findings suggest that socio-cultural factors may play a role in the causal attributions individuals make about cancer, which can, in turn, inform cancer awareness and prevention messages.

Keywords

causal attributions; causal beliefs; family history; heredity; cancer

Introduction

Having strong beliefs about what causes cancer has the potential to increase coping and preventive strategies to reduce cancer risk (Kaphingst, Lachance, & Condit, 2009; Lykins et al., 2008). Prior literature suggests, however, that holding certain causal beliefs such as fatalistic views may also hinder the uptake of important health behaviors (Befort, Nazir,

Correspondence to: Vivian M. Rodríguez , PO Box 842018, Department of Psychology, Virginia Commonwealth University, Richmond, VA 23284, USA. rodriguezvm@vcu.edu..

Engelman, & Choi, 2013; Niederdeppe & Gurmankin Levy, 2007). Consequently, understanding the nature of cancer causal beliefs is of critical importance to develop effective cancer education programs and increase adherence to screening recommendations and health behaviors.

The causal attributions individuals make about cancer may be shaped by their experience with the disease. Having a family history of cancer, for instance, may predispose people to assign a critical role to genetics as a causal agent of cancer. For instance, Hay et al. (2011) investigated the causal beliefs held by patients diagnosed with melanoma, their first-degree relatives (parent, child, or sibling), and extended relatives. In their study, approximately 45% of participants attributed heredity/genetics as an important risk factor for melanoma. Interestingly, first-degree relatives and extended relatives were more likely than patients to attribute the cause of melanoma to heredity. Providing further support to this finding, evidence has shown that individuals with a family history of cancer are likely to disagree with the statement that cancer is caused by a person's behavior or lifestyle (Kowalkowski, Hart, Du, Baraniuk, & Latini, 2012). The causal role individuals attribute to heredity in cancer is yet to be fully understood. Growing evidence suggests, however, that individuals whose families have been affected by cancer consider genetics in the etiology of cancer.

The causal attributions made about cancer have the potential to help individuals affected by cancer maintain or improve their health by focusing on modifiable and preventive behaviors to reduce cancer recurrence and/or decrease the likelihood of cancer in other family members (Lykins et al., 2008). Costanzo, Lutgendorf, Bradley, Rose, and Anderson (2005) showed that women who had implemented positive changes in their diet since their breast cancer diagnosis were more likely to believe that their cancer was caused by stress or environmental toxins than women who did not make these changes. Among women with a first-degree relative with breast cancer, Lemon, Zapka, and Clemow (2004) found that 42% of women reported health behavior changes in the six months following their relative's diagnosis. It appears that participants were eager to change those health behaviors that could potentially reduce their lifetime risk of developing cancer. Positive behavior changes included increased physical activity, higher consumption of fruits and vegetables, and decreased fat and alcohol intake. Thus, a closer examination of the factors that shape and influence cancer attributions becomes an important adjunct to cancer prevention and control efforts.

Another consideration when discussing the cancer causal attributions that individuals hold pertains to the limited knowledge or misperceptions about the common risk factors for cancer. In a large national survey, fewer than 38% of women believed that not exercising and drinking alcohol are important risk factors for breast cancer (Wold, Byers, Crane, & Ahnen, 2005). A more recent study showed that one-third of women believed having prior colon surgery increases the risk for colon cancer, and 60% of women believed changes in one's immune system are a risk factor for the development of breast and colon cancers (Wang, Miller, Egleston, Hay, & Weinberg, 2010). The increasing complexity of the interactive and multi-factorial process of cancer, from normal cellular processes to its conception, contributes to the public's confusion about the etiology of cancer. Importantly,

lack of accurate information about cancer risk factors may potentially contribute to the underuse of preventive health strategies.

Causal attributions and subsequent preventive action may also be influenced by socio-cultural factors such as race/ethnicity and socio-economic status (Kohli & Dalal, 1998; Landrine & Klonoff, 1994; Murguia, Zea, Reisen & Peterson, 2000). Kwate and colleagues (2005) surveyed women with a family history of breast cancer about their causal attributions for breast cancer. In their study, White women were more likely to endorse 'environmental poisons' and heredity as causal factors for breast cancer than African-American women. Moreover, African-American women endorsed interpersonal-level factors including 'a blow to the breast', 'personal behavior', and 'other people' more often than White women. Furthermore, evidence shows that individuals from underserved communities are likely to hold fatalistic beliefs about cancer prevention efforts. For instance, one study found that rural residents endorsed more fatalistic beliefs about cancer prevention than urban residents (Befort, Nazir, Engelman, & Choi, 2013). The authors posit that limited access to health care and information make rural residents more likely to endorse such beliefs hindering their cancer screening uptake. Another study found that Latino and African-American participants held more fatalistic beliefs concerning colorectal cancer compared to White participants (Lumpkins et al., 2013). A more recent study with a sample of acculturated Latinas showed that one in five women believed they had little power to lower their risks of cancer (Ramirez, 2014). Lastly, Coups and colleagues (2014) found that holding greater fatalistic beliefs in a sample of Hispanic adults was associated with decreased shade-seeking behaviors in the context of melanoma prevention. Given the highly changing demographic landscape in the U.S., it is important to consider the cancer causal beliefs of underserved populations as this may have important implications for cancer education.

Common themes in causal beliefs have been identified throughout the cancer attribution literature providing a glimpse into the beliefs and knowledge individuals have about the causes of cancer. These themes include but are not limited to lifestyle factors, genetic or biological factors, and personal factors (Ferruci et al., 2011). The available research has greatly focused on cancer patients or survivors' attributions for cancer and has been conducted with predominately White samples (Ford & Kaphingst, 2009). Less is known about causal attributions held by women with and without a family history of cancer from a diverse community sample. Utilizing data from a randomized controlled trial (Kin Fact Study, R01-CA140959-01) aimed at increasing family communication about hereditary cancers, we sought to examine relations between causal attributions of cancer in a diverse sample of women with and without a family history of cancer. The relation between socio-demographic factors and cancer causal attributions was also explored.

Method

Participants

Four-hundred and ninety English-speaking women, ages 18 years and older, were recruited from July 2010 through January 2012 in the waiting areas of the Women's Health Clinic of Virginia Commonwealth University Health System (VCUHS). Women who had a personal

history of cancer (3.9%) were excluded from all analyses, yielding a final sample of 471 women.

Sixty percent of participants self-identified as African-American and the mean age of the sample was 32.9 years. Based on medical record review, 42.3% of women had managed care insurance while 30.4% had no insurance. Thirty-one percent of women reported graduating high school or obtaining a GED as their highest level of education completed. In addition, nearly three-fourths of the sample (73.5%) had a family history of cancer in a first or second degree relative (see Table 1 for demographic characteristics).

Procedure

The present manuscript utilizes baseline data from a National Cancer Institute funded study, the Kin Fact Study. Kin Fact is a longitudinal, randomized controlled trial testing the effects of a brief intervention on family communication about hereditary cancer risk. Participants were recruited following appointment registration at an outpatient women's health clinic. Once enrolled, recruiters obtained a three generation cancer focused pedigree through structured interviews with participants. Participants completed baseline measures and were then randomized to either the intervention or control group. The intervention, which consisted of a 15-20 minute PowerPoint presentation including personalized cancer risk information and communication skills training, occurred immediately following their appointments. Participants were followed over time, and asked to complete follow-up measures at one, six, and 14 months after baseline. Women received honoraria after completing each phase of this longitudinal study totaling approximately $40. This manuscript focuses exclusively on the baseline assessment.

Instruments

Demographic variables—Participants reported on their age, race/ethnicity, and education level. Insurance status was obtained through reviewing medical records for information on insurance held at the time of recruitment.

Family history of cancer—Family history of cancer in first and second degree relatives was identified through pedigrees taken by research assistants at baseline. Among women with a family history of cancer, the most common types of cancers reported were breast cancer (39.6%), colon cancer (19%), lung cancer (12.1%), and melanoma (11.3%).

Causal attributions—Following pedigree construction, participants were verbally asked the following open-ended question: “Please tell us the three most important factors that you believe caused cancer in your family. Please order your responses with “1” being the most important, “2” being the next most important, and so on.” Women who did not have a family history of cancer were asked “Please tell us the three most important factors that you believe cause cancer in general.” Although women were asked for three responses, they did not have to provide three responses, and few (5.7%) women provided more than three responses.

Data Preparation

Utilizing an inductive approach, the first and second author became familiar with the data by doing an extensive review of the causal attribution responses prior to drafting the coding book. An initial list of 16 codes was developed after this preliminary examination of the data. Following this initial phase of coding-construction, a theoretical phase ensued. Additional categories were included based on categories used in prior literature on causal attributions. The final coding book included 28 specific categories and an “other” response category (see Table 2). The aforementioned procedure for developing the coding book followed Braun and Clarke's (2006) guidelines on thematic analysis.

Two additional raters independently coded all data responses into 29 specific attributions, which were then collapsed into broad attributions modeled after Ferrucci et al. (2011). The broad attributions categories included (1) lifestyle choices, (2) environmental exposures, (3) aging, (4) genetics/heredity, (5) stress, (6) prior health condition, (7) chance, (8) self-care/knowledge, (9) other, and (10) no attribution identified. In the event that participants indicated multiple attributions, raters assigned a separate code to each attribution. A meeting among the raters and the first and second authors was held to resolve discrepancies and achieve a final code for each response. Raters achieved excellent agreement on the codes, with kappa's ranging from .89 to .92.

Results

The majority of women (88.7%) were able to identify at least one causal attribution for cancer. Overall, lifestyle choices was the most frequently reported causal attribution (63.1%), followed by genetics/heredity (34.4%) and environmental exposures (19.3%). Table 3 presents the frequency and percentage of participants who reported the 10 causal attributions, separately by women with and without a family history of cancer.

Family History versus No Family History

We conducted a series of chi-square tests of independence (see Table 3) to explore whether there were differences between women with and without a family history of cancer in identifying three causal attributions (lifestyle choices, genetics/heredity, and environmental exposures). Results showed no significant association between lifestyle choices and family history of cancer, χ^2 (1, N = 471) = 1.09, p =.30. Specifically, women with and without a family history of cancer were equally likely to identify lifestyle choices as causal factors for cancer, and did so at a higher rate than other attributions. Significant differences, however, were found for genetics/heredity, χ^2 (1, N = 471) = 3.92, p =.04, and environmental exposures, χ^2 (1, N = 471) = 4.64, p =.03. Women without a family history of cancer were 1.5 times more likely (OR = 1.53; 95% CI: 1.01, 2.33) to report genetics/heredity as a causal attribution than women with a family history of cancer. Furthermore, women with a family history of cancer were 1.9 times more likely (OR = 1.89; 95% CI: 1.40, 2.54) to report environmental exposures as a causal attribution of cancer than women without such history.

The Relation between Socio-demographic Factors and Causal Attributions

Logistic regressions were conducted to further examine the relationship between family history of cancer and the top three causal attributions for cancer. In particular, we were interested in investigating whether socio-demographic variables (race, age, education, and insurance level) moderated the relationships between family history of cancer and causal attributions.

Given that our sample consisted of mainly African-American (60%) and White (32%) women, we chose to examine differences between women of these two racial groups only (n = 431). We also chose to divide the sample into women who were less than 40 years of age, and women who were 40 years of age or above. The reason for this cutoff is pertaining to women reaching an age where cancer screening for certain types of cancer is encouraged (i.e. breast cancer) thus increasing awareness about cancer risk factors. Two indices were used to examine socio-economic status: education level and insurance status. In order to examine differences in education level, we dichotomized the variable into women who reported a high school/GED degree or less, and women who had completed some college or higher. Insurance status was measured using three categories (i.e. commercial, managed care, and no insurance). Only significant findings are reported.

Main effects—We first examined potential main effects for each socio-demographic factor and broad causal attribution independent of family history of cancer. Several main effects were found for genetics/heredity as a causal attribution for cancer. For instance, a significant main effect was found for race, in that White women were 1.9 times more likely to report genetics/heredity as a causal attribution for cancer than African-American women (OR=1.92; 95% CI: 1.24, 2.94). White women were also more than twice as likely to report environmental exposures as a causal attribution for cancer than African-American women (OR=2.22, 95% CI: 1.37, 3.70). Furthermore, a significant main effect was found for education in that women with a higher education level (some college or greater) were 2.7 times more likely to report genetics/heredity as a causal attribution for cancer than women with lower education attainment (OR=2.69; 95% CI: 1.73, 4.17). Women with a higher education level were also almost 3 times more likely to report environmental exposures than women with lower levels of education (OR=2.65, 95% CI: 1.54, 4.54). Lastly, a significant main effect was found for having commercial insurance (OR=1.92; 95% CI: 1.26, 2.93). Women who reported having commercial insurance were almost twice as likely to report genetics/heredity as a causal attribution for cancer compared to women with other forms of insurance or no insurance.

Moderation analyses—We then performed hierarchical logistic regressions with interaction terms to test for moderation. We explored the effect of socio-demographic variables and the presence or absence of a family history of cancer on each of the three broad causal attributions identified (lifestyle choices, genetics/heredity, and environmental exposures).

Lifestyle choices: No significant moderation was found for race, education, and insurance status on reporting of lifestyle choices, however, a significant effect was found for age. The

model which included the interaction term between age and family history of cancer was significant in predicting lifestyle choices, $\chi^2(1) = 4.84$, $p = .02$ (see Table 4). In order to break down this effect, separate chi-square tests on the reporting of lifestyle choices as an attribution were performed separately for the age and family history variables. For women without a family history of cancer, there was a marginal association between the women's age and whether or not they reported lifestyle choices, $\chi^2(1) = 3.05$, $p = .08$. Odds ratios indicated that the odds of reporting lifestyle choices were three times higher for women 40 years old or above than for women who were below 40 years of age (OR=3.1, 95% CI: 0.83, 11.59). For women below 40 years of age, there was a marginal association between family history of cancer and whether they reported lifestyle choices, $\chi^2(1) = 3.48$, $p = .06$. The odds of reporting lifestyle choices as a causal attribution for cancer were 1.5 higher for women with a family history of cancer than for women without this history (OR=1.55, 95% CI: 1.23, 1.96).

Genetics/heredity: We then explored whether race, age, education, and insurance level played a role in the reporting of genetics/heredity as a causal attribution of cancer for women who have a history of cancer in their family and for women who do not have such history. Hierarchical logistic regressions yielded no significant interactions, suggesting that race, age, education level, nor insurance status moderate the relationship between family history of cancer and genetics/heredity as a causal attribution for cancer (see Table 5).

Environmental exposures: Similar to lifestyles choices, results showed that age moderated the effect of family history of cancer on reporting of environmental exposures as a causal attribution (see Table 6). No other significant moderating interactions were found for race, education level, or insurance status.

The model which included the interaction term between age and family history of cancer was significant in predicting environmental exposures, $\chi^2(1) = 8.54$, $p = .003$. In order to break down this effect, separate chi-square tests on the reporting of environmental factors as attributions were performed separately for the age and family history variables. For women without a family history of cancer, there was a significant association between the women's age and whether or not they reported environmental exposures, $\chi^2(1) = 17.52$, $p = .000$. Odds ratios indicated that the odds of reporting environmental exposures were 9.85 (95% CI: 5.28, 18.42) higher for women 40 or older than for women who were below 40 years of age. For women below 40 years of age, there was a significant association between family history of cancer and whether they reported environmental exposures as attributions, $\chi^2(1) = 8.11$, $p = .004$. The odds of reporting environmental exposures were almost three times higher for women with a family history of cancer than for women without this history (OR=2.86, 95% CI: 1.95, 4.18). A marginally significant association was found between family history of cancer and environmental exposures for women 40 or above years of age, $\chi^2(1) = 3.05$, $p = .08$. Women who were 40 years or older and with no known family history of cancer were twice more likely to report environmental exposures than women with a family history of cancer (OR=2.28, 95% CI: 1.29, 4.02).

Discussion

Attribution theory suggests that people typically interpret behaviors in terms of its causes as a way to attribute meaning and understand their environment (Kelley & Michela, 1980). Cancer causal attributions are then developed as a way to understand and process the cancer experience (Lavery & Clarke, 1996). Prior literature suggests that cancer causal attributions have been associated with adaptive coping strategies to reduce cancer risk (Kaphingst, Lachance, & Condit, 2009; Lykins et al., 2008). Understanding the nature of cancer causal beliefs among individuals may help inform cancer preventive and risk-reducing interventions. Therefore, the primary objective of this study was to examine cancer causal attributions in a diverse sample of women who are not cancer patients. Exploratory analyses were performed between women with a family history of cancer and women without this history. Furthermore, we explored the role of socio-demographic factors in the causal attributions among these groups. To the best of our knowledge this is one of the first studies examining these relationships in a healthy, urban sample of diverse women.

Our results indicated that the vast majority of women (close to 90%) were able to identify at least one causal attribution for cancer. In support of prior literature, the three most reported attributions were lifestyle choices, genetics/heredity, and environmental exposures. This is also consistent with the American Cancer Society's primary causal attributions for cancer (ACS, 2012). Results indicated that lifestyle choices were reported by a large proportion of the women in our sample (60%) regardless of race, age, or socio-economic status. Women were most likely to believe cancer was caused by lifestyle choices, such as smoking, eating a poor diet, and drinking alcohol. In addition, close to a third of women also identified genetics/heredity as a causal attribution for cancer, and about 20% reported environmental exposures as an attribution.

One of the primary findings of this study is that women without a family history of cancer were more likely to report genetics/heredity as a causal attribution for cancer than women with cancer in their family history. This finding is counterintuitive as prior research has supported a link between having a family history of cancer and identifying heredity as a cause for cancer (Hay et al., 2011). It may be that women with a family history of cancer are less inclined to identify their family lineage (a factor out of their control) as a causal attribution for cancer, while women without such a family history may want to attribute cancer to factors that are unchanging as opposed to those that they have control over (health behaviors, for instance). Future research may want to better understand this finding through qualitative work that would allow the researcher to obtain a deeper understanding of the reasons for this phenomenon, including the influences of locus of control and family stigma on cancer causal attributions.

It appears that socio-demographic factors may play a significant role in the identification of genetics as a potential causal attribution for cancer. For example, we found that race, educational level, and insurance status were associated with the reporting of genetics/heredity as a causal attribution for cancer. White women, women with higher educational attainment, and those with commercial insurance were more likely to provide genetics/heredity causal attributions compared to African-American women, those with a high school

degree or less, and those with managed care or no health insurance. This finding is consistent with prior studies (Breslow, Sorkin, Frey, & Kessler, 1997; Kwate, Thompson, Valdimarsdottir, & Bovbjerg, 2005) which have found that White women are more likely to endorse family history or heredity as a causal attribution for breast cancer than minority women. Yet, given the relevant literature on fatalistic beliefs about cancer, it is surprising that African-American women, who may be more likely to hold these beliefs, were less likely to identify genetics/hereditary as a cause (Lumpkins et al., 2013). More research is needed to elucidate our understanding of minority women's beliefs about cancer; in particular, their view on the role of heredity in cancer risk and how genetic literacy affects these beliefs.

The findings from this study contribute to the discussion on health disparities and access to health-related information, and suggest that race, education, and socio-economic status are important factors in bringing awareness to the connection between genetics and cancer etiology (Wang, Miller, Egleston, Hay, & Weinberg, 2010). Educational campaigns may want to explore this link further as the beliefs women from underserved populations hold about cancer, particularly the role of genetics in cancer etiology, may inhibit the uptake of cancer screening recommendations based on family history.

Strengths and Limitations

This research enhances the existing literature on causal attributions for cancer by utilizing a diverse sample of women with and without a personal history of cancer, recruited within a large, urban, safety-net women's health clinic. Our sample included a high proportion of African-American women and participants from primarily lower socio-economic statuses. In addition, we focused our analysis on the attributions personally unaffected women made about the causes of cancer rather than exploring beliefs of cancer survivors. Lastly, we measured causal attributions by asking an open-ended question instead of providing a checklist of known beliefs based on prior literature. This method ensured that we got a variety of rich responses which were then quantitatively coded.

The present study is not without limitations. The item used to assess causal attributions differed according to whether or not the participant had a family history of cancer. It is important to note that no strong conclusions should be made regarding these exploratory analyses given the changes in the wording of the causal attributions question asked to women with a family history and women without a family history. In order to draw definitive conclusions, findings should be replicated. Regardless of this difference in methodology, it appears that women in both groups believe that lifestyle choices, genetics/heredity, and environmental exposures are the primary causes of cancers. One limitation to the external validity of the study is its focus on women. Examining gender differences in causal attributions may be an important consideration when developing educational campaigns and cancer prevention messages. For instance, in a sample of men with testicular cancer, the authors found that most men attributed cancer to psychological stress, an attribution lower in priority for the women in this sample (Fischer Pendersen, Rossen, Olesen, von der Maase, & Vedsted, 2012). In addition, this study examined attributions

about cancer in general. Examining causal attributions for specific cancer types could yield meaningful data that could be used to tailor prevention messages even further.

Implications and Future Directions

The present study contributes to the small but growing literature in cancer causal attributions by documenting the beliefs diverse women hold about cancer etiology. To date there has been a dearth of research examining the attributions women without a personal history of cancer but with a family history of cancer have about the cause of cancer. With the added availability of genetic information and health care decisions that are contingent on this information, assessing the public's understanding of genetic knowledge and how it may relate to cancer etiology becomes an important research goal. Authors in the field suggest that prior schemas or cognitive representations about cancer may influence the way individuals respond to cancer risk communication messages and risk factors for cancer (Walter, Emery, Braithwaite, & Marteau, 2004). Therefore, targeting previously held beliefs about the causes of cancer may be an important cost-effective cancer prevention tool.

The literature has overwhelmingly shown that women tend to be considered the “kin keepers” of family health history and as such, are seen as the responsible ones for disseminating health information within the family (Foster, Eeles, Ardern-Jones, Moynihan, & Watson, 2004; Koehly et al., 2009; Nycum, Avard, & Knoppers, 2009; Wiseman, Dancyger, & Michie, 2010). The findings from this study suggest that women's beliefs about cancer may be influenced by whether or not they have a family history of cancer. Understanding women's beliefs about what causes cancer and the factors that may place their family at risk for cancer becomes an important prevention goal. It is likely that these beliefs are passed on from family member to family member possibly affecting the family's views on cancer screening and prevention efforts. For instance, qualitative accounts have found that women create an atmosphere of “health talk”, especially among sisters and other female relatives, which supports open discussion about health within the family (Forrest et al., 2003; Lindenmeyer, Griffiths, & Hodson, 2010). Therefore, identifying the beliefs families adopt about the causes of cancer and how these beliefs translate into behavior is of utmost importance to advance the field of cancer prevention. Research can also examine whether beliefs about genetics/heredity as a cancer causal attribution may contribute to the development of fatalistic attitudes and beliefs, which in turn, play a role in the uptake of prevention practices (Niederdeppe & Gurmankin Levy, 2007). Alternatively, it could be that women endorsing heredity as a causal attribution for cancer may be more prone to surveillance behaviors and regular screening practices.

There is still much to be learned about the implications of cancer causal attributions, in particular, about the relationship between specific attributions and the adoption of cancer risk-reducing behaviors. More research is needed to elucidate the utility of cancer causal beliefs in prevention efforts. This study provides early support for a connection between family history of cancer, socio-demographic factors, and specific causal attributions for cancer, which sets the stage for future investigations in the area of causal beliefs.

Acknowledgements

This manuscript is supported by the National Institutes of Health - National Cancer Institute Grant (R01-CA140959-01) awarded to Dr. John M. Quillin and Research Supplement to Promote Health-Related Research (3R01CA140959-02S1) awarded to Vivian M. Rodríguez. We would like to recognize the work of two research assistants, Lauren DeLosSantosCoy and Diana Naidoo, who helped code qualitative responses.

References

American Cancer Society. What causes cancer?. 2012. Retrieved from http://www.cancer.org/cancer/cancercauses/index

Befort CA, Nazir N, Engelman K, Choi W. Fatalistic cancer beliefs and information sources among rural and urban adults in the USA. Journal of Cancer Education. 2013; 28:521–526. doi: 10.1007/s13187-013-0496-7. [PubMed: 23813489]

Braun V, Clarke V. Using thematic analysis in psychology. Qualitative Research in Psychology. 2006; 3(2):77–101. doi: 10.1191/1478088706qp063oa.

Breslow RA, Sorkin JD, Frey CM, Kessler LG. Americans' knowledge of cancer risk and survival. Preventive Medicine. 1997; 26(2):170–177. doi: 10.1006/pmed.1996.0136. [PubMed: 9085385]

Costanzo ES, Lutgendorf SK, Bradley SL, Rose SL, Anderson B. Cancer attributions, distress, and health practices among gynecologic cancer survivors. Psychosomatic Medicine. 2005; 67(6):972–980. doi: 10.1097/01.psy.0000188402.95398.c0. [PubMed: 16314603]

Coups EJ, Stapleton JL, Manne SL, Hudson SV, Medina-Forrester A, Rosenberg SA, Goydos JS. Psychosocial correlates of un protection behaviors among U.S. Hispanic adults. Journal of Behavioral Medicine. Advance online publication. 2014 doi: 10.1007/s10865-014-9558-5.

Ferrucci LM, Cartmel B, Turkman YE, Murphy ME, Smith T, Stein KD, McCorkle R. Causal attribution among cancer survivors of the 10 most common cancers. Journal of Psychosocial Oncology. 2011; 29(2):121–140. doi: 10.1080/07347332.2010.548445. [PubMed: 21391066]

Fischer Pendersen A, Rossen P, Olesen F, von der Maase H, Vedsted P. Fear of recurrence and causal attributions in long-term survivors of testicular cancer. Psycho-Oncology. 2012; 21:1222–1228. doi: 10.1002/pon.2030. [PubMed: 21919117]

Ford BM, Kaphingst KA. Lay interpersonal sources for health information related to beliefs about the modifiability of cancer risk. Cancer Causes & Control. 2009; 20(10):1975–1983. doi: 10.1007/s10552-009-9392-1. [PubMed: 19578935]

Forrest K, Simpson SA, Wilson BJ, van Teijlingen ER, McKee L, Haites N, Matthews E. To tell or not to tell: Barriers and facilitators in family communication about genetic risk. Clinical Genetics. 2003; 64:317–326. doi: 10.1034/j.1399-0004.2003.00142.x. [PubMed: 12974737]

Foster C, Eeles R, Ardern-Jones A, Moynihan C, Watson M. Juggling roles and expectations: Dilemmas faces by women talking to relatives about cancer and genetic testing. Psychology and Health. 2004; 19:439–455. doi: 10.1080/08870440410001684168.

Hay J, DiBonaventura M, Baser R, Press N, Shoveller J, Bowen D. Personal attributions for melanoma risk in melanoma-affected patients and family members. Journal of Behavioral Medicine. 2011; 34(1):53–63. doi: 10.1007/s10865-010-9286-4. [PubMed: 20809355]

Kelley HH, Michela JL. Attribution theory and research. Annual Review of Psychology. 1980; 31:457–501. doi: 10.1146/annurev.ps.31.020180.002325.

Koehly LM, Peters JA, Kenen R, Hoskins LM, Ersig AL, Kuhn NR, Greene MH. Characteristics of health information gatherers, disseminators, and blockers within families at risk of hereditary cancer: Implications for family health communication interventions. American Journal of Public Health. 2009; 99:2203–2209. doi: 10.2105/AJPH.2008.154096. [PubMed: 19833996]

Kohli N, Dalal AK. Culture as a factor in causal understanding of illness : A study of cancer patients. Psychology & Developing Societies. 1998; 10(2):115–129. doi: 10.1177/097133369801000202.

Kwate NO, Thompson HS, Valdimarsdottir HB, Bovbjerg DH. Brief report: Etiological attributions for breast cancer among healthy African American and European American women. Psycho-Oncology. 2005; 14(5):421–425. doi: 10.1002/pon.905. [PubMed: 15744781]

Landrine H, Klonoff EA. Cultural diversity in causal attributions for illness: The role of the supernatural. Journal of Behavioral Medicine. 1994; 17(2):181–193. [PubMed: 8035451]

Lavery JF, Clarke VA. Causal attributions, coping strategies, and adjustment to breast cancer. Cancer Nursing. 1996; 19(1):20–28. [PubMed: 8904383]

Lemon SC, Zapka JG, Clemow L. Health behavior change among women with recent familial diagnosis of breast cancer. Preventive Medicine. 2004; 39(2):253–262. doi: 10.1016/j.ypmed.2004.03.039. [PubMed: 15226033]

Lindenmeyer A, Griffiths F, Hodson J. 'The family is art of the treatment really': A qualitative exploration of collective health narratives in families. Health. 2010; 20:1–15. doi: 10.1177/1363459310384493.

Lumpkins CY, Cupertino P, Young K, Daley C, Yeh HW, Greiner KA. Racial/ethnic variations in colorectal cancer screening self-efficacy, fatalism, and risk perception in a safety-net clinic population: Implications for tailored interventions. Journal of Community Medicine & Health Education. 2013 Advance online publication.

Lykins EL, Graue LO, Brechting EH, Roach AR, Gochett CG, Andrykowski MA. Beliefs about cancer causation and prevention as a function of personal and FH of cancer: A national, population-based study. Psycho-Oncology. 2008; 17(10):967–974. doi: 10.1002/pon.1306. [PubMed: 18203236]

Murguia A, Zea MC, Reisen CA, Peterson RA. The development of the cultural health attributions questionnaire (CHAQ). Cultural Diversity & Ethnic Minority Psychology. 2000; 6(3):268–283. [PubMed: 10938635]

Niederdeppe J, Gurmankin Levy A. Fatalistic beliefs about cancer prevention and three prevention behaviors. Cancer Epidemiology, Biomarkers & Prevention. 2007; 16(5):998–1003. doi: 10.1158/1055-9965.EPI-06-0608.

Nycum G, Avard D, Knoppers BM. Factors influencing intrafamilial communication of hereditary breast and ovarian cancer genetic information. European Journal of Human Genetics. 2009; 17:872–880. doi: 10.1038/ejhg.2009.33. [PubMed: 19319160]

Ramirez AS. Fatalism and cancer risk knowledge among a sample of highly acculturated Latinas. Journal of Cancer Education. 2014; 29:50–55. doi: 10.1007/s13187-013-0541-6. [PubMed: 24078314]

Walter FM, Emery J. 'Coming down the line'-- patients' understanding of their FH of common chronic disease. Annals of Family Medicine. 2005; 3(5):405–414. doi: 10.1370/afm.368. [PubMed: 16189056]

Wang C, Miller SM, Egleston BL, Hay JL, Weinberg DS. Beliefs about the causes of breast and colorectal cancer among women in the general population. Cancer Causes & Control. 2010; 21(1):99–107. doi: 10.1007/s10552-009-9439-3. [PubMed: 19787437]

Wiseman M, Dancyger C, Michie S. Communicating genetic risk information within families: A review. Familial Cancer. 2010; 9:691–703. doi: 10.1007/s10689-010-9380-3. [PubMed: 20852947]

Wold KS, Byers T, Crane LA, Ahnen D. What do cancer survivors believe causes cancer? (United States). Cancer Causes & Control. 2005; 16(2):115–123. doi: 10.1007/s10552-004-2414-0. [PubMed: 15868453]

Table 1

Sample Characteristics (N = 471).

	N	M	SD
Age	471	32.9	11.7
< 40	356	27.2	5.5
≥ 40	115	50.4	7.5
Genetic Literacy[a]	469	5.8	2.3

	N	%
Ethnicity		
Hispanic/Latino Origin	17	3.6
Not Hispanic/Latino Origin	454	96.4
Race[b]		
American Indian/Alaska Native	4	0.9
Asian	6	1.3
Black/African-American	281	60.0
White	150	32.1
Other	8	1.7
Multi-racial	19	4.1
Insurance Status[c]		
Commercial Insurance	129	27.4
Managed Care Insurance	199	42.3
No Insurance	143	30.4
Education Level[d]		
Attended Some Grade School	9	2.3
Attended Some High School	59	15.0
Graduated High School or Earned a GED	123	31.3
Attended Some College, Vocational, or Trade School	87	22.1
Graduated from a Two-Year College, Vocational, or Trade School	42	10.7
Graduated from a Four-Year College	41	10.4
Attended Some Graduate School	13	3.3
Earned a Graduate Degree	19	4.8

Note.

[a] Genetic literacy was measured on a scale ranging from 1 through 8; higher scores indicate greater literacy. Two participants did not complete the genetic literacy test.

[b] Three participants did not report their race

[c] Managed care insurance included Medicaid and Medicare

[d] Seventy-eight participants did not have the opportunity to report their education level, as education level was added to the baseline survey after recruitment had begun.

Table 2

Coding Scheme.

Broad Causal Attribution	Specific Causal Attribution
Lifestyle Choices	Smoking/Tobacco
	Alcohol
	Poor Diet/Eating Behaviors
	Lack of Physical Activity/Exercise
	Sun Exposure/Tanning
	Sexual History/STDs
	Drugs
	Lifestyle Choices
Environmental Exposures	Second-hand Smoke
	Exposure to Asbestos
	Exposure to Radiation
	Coal-mining
	Air/Water Pollution
	Chemicals in Food
	Exposure to Chemicals/Toxins
	Occupational Hazards (i.e., war, military)
	Environmental Exposures
Aging	Aging
Genetics/Heredity	Genetics/Heredity
Stress	Stress
Prior Health Condition	Obesity
	Medical Illness (i.e., diabetes, GI problems, virus, bacteria)
	Poor Health
	Trauma/Injury
Chance	Bad Luck/Chance
Self-care/Knowledge	Poor Self-care
	Lack of Medical Follow-Up
	Lack of Information/Education about Cancer
Other	Other Type of Responses
No Attribution Identified	Don't Know / No Response

Table 3

Causal Attributions by Women with and without a History of Cancer in a Diverse Community Sample

Causal Attributions	Total Sample N = 471	No Family History N = 125	Family History N = 346	χ^2	OR [95% CI]
Lifestyle Choices	297 (63.1)	74 (59.2)	223 (64.5)	1.09	0.80 [0.53-1.21]
Genetics/Heredity	162 (34.4)	52 (41.6)	110 (31.8)	3.92*	1.53 [1.01-2.33]
Environmental Exposures	91 (19.3)	16 (12.8)	75 (21.7)	4.64*	1.89 [1.40-2.54]
Self-care/Knowledge	51 (10.8)	10 (8.0)	41 (11.8)	1.41	0.65 [0.32-1.34]
Other	30 (6.4)	11 (8.8)	19 (5.5)	1.67	1.66 [0.77-3.59]
Prior Health Condition	28 (5.9)	6 (4.8)	22 (6.4)	0.40	0.74 [0.29-1.87]
Stress	25 (5.3)	9 (7.2)	16 (4.6)	1.21	1.60 [1.12-2.30]
Chance	16 (3.4)	4 (3.2)	12 (3.5)	†	
Aging	7 (1.5)	2 (1.6)	5 (1.4)	†	

Note. Numbers represent frequencies. Percentages are in parenthesis.

*$p < .05$.

†Given the low number of women who endorsed the ‘Chance’ and ‘Aging’ attribution, the chi-square test was not performed as it did not meet the required assumptions.

Table 4

Logistic Regressions for Lifestyle Choices Controlling for Family History (FH) of Cancer

	B	SE	Wald χ^2 (1)	OR	95% CI	*p*
Main Effects[1]						
Race	.25	.22	1.39	1.29	[.85, 1.96]	.24
Age	−.12	.23	.28	0.89	[.57, 1.38]	.60
Education	−.08	.22	.13	0.93	[.61, 1.41]	.72
Health Insurance						
Commercial Insurance	−.23	.21	1.14	0.80	[.52, 1.21]	.29
Managed Care	.29	.20	2.08	1.33	[.90, 1.96]	.15
No Insurance	−.11	.21	.26	0.90	[.60, 1.35]	.61
Moderation						
Race × FH	.51	.56	.82	.60	[.20, 1.81]	.37
Age × FH	−1.46	.72	4.15	.23	[.06, .95]	.04
Education × FH	.41	.50	.69	1.51	[.57, 3.98]	.41
Commercial Insurance × FH	−.40	.53	.56	.67	[.24, 1.90]	.46
Managed Care × FH	−.38	.44	.77	.68	[.29, 1.60]	.38
No Insurance × FH	.89	.48	3.44	2.43	[.95, 6.19]	.06

Note.

[1] Socio-demographic variables were entered alongside the dichotomous family history variable in the first step of the logistic regression in order to calculate main effects.

[2] To examine moderating effects, an interaction term between each socio-demographic variable and family history of cancer was calculated and entered in the second step of the logistic regression.

Table 5

Logistic Regressions for Genetics/Heredity Controlling for Family History (FH) of Cancer

	B	SE	Wald χ^2 (1)	OR	95% CI	*p*
Main Effects[1]						
Race	−.65	.22	8.69	.52	[.34, .80]	.00
Age	−.00	.23	.00	1.00	[.63, 1.57]	.99
Education	.99	.22	19.52	2.69	[1.73, 4.17]	.00
Health Insurance						
Commercial Insurance	.65	.22	9.06	1.92	[1.26, 2.93]	.00
Managed Care	−.62	.21	8.90	.54	[.36, .81]	.00
No Insurance	.06	.21	.08	1.06	[.70, 1.61]	.78
Moderation						
Race × FH	−.09	.56	.03	.91	[.30, 2.75]	.87
Age × FH	.48	.63	.57	1.61	[.47, 5.58]	.45
Education × FH	−.10	.50	.04	.91	[.34, 2.41]	.85
Commercial Insurance × FH	.44	.52	.69	1.55	[.55, 4.32]	.41
Managed Care × FH	−.18	.44	.16	.84	[.35, 2.00]	.69
No Insurance × FH	−.44	.48	.85	.64	[.25, 1.64]	.36

Note.

[1] Socio-demographic variables were entered alongside the dichotomous family history variable in the first step of the logistic regression in order to calculate main effects.

[2] To examine moderating effects, an interaction term between each socio-demographic variable and family history of cancer was calculated and entered in the second step of the logistic regression.

Table 6

Logistic Regressions for Environmental Exposures Controlling for Family History (FH) of Cancer

	B	SE	Wald χ^2 (1)	OR	95% CI	*p*
Main Effects[1]						
Race	−.81	.25	10.14	.45	[.27, .73]	.00
Age	.58	.26	5.14	1.79	[1.08, 2.95]	.02
Education	.97	.28	12.49	2.65	[1.54, 4.54]	.00
Health Insurance						
Commercial Insurance	1.07	.25	19.09	2.92	[1.81, 4.72]	.00
Managed Care	−.42	.25	2.83	.66	[.40, 1.07]	.09
No Insurance	−.72	.29	6.38	.49	[.28, .85]	.01
Moderation						
Race × FH	−.582	.76	.59	.56	[.13, 2.47]	.44
Age × FH	−2.02	.68	8.71	.13	[.04, .51]	.00
Education × FH	.45	.66	.46	1.57	[.43, 5.71]	.50
Commercial Insurance × FH	−.53	.63	.71	.59	[.17, 2.03]	.40
Managed Care × FH	.51	.62	.69	1.67	[.50, 5.61]	.41
No Insurance × FH	−.27	.75	.13	.76	[.18, 3.29]	.72

Note.

[1] Socio-demographic variables were entered alongside the dichotomous family history variable in the first step of the logistic regression in order to calculate main effects.

[2] To examine moderating effects, an interaction term between each socio-demographic variable and family history of cancer was calculated and entered in the second step of the logistic regression.