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Identifying Mediators of Intervention Effects Within a Randomized Controlled Trial to Motivate Cancer Genetic Risk Assessment Among Breast and Ovarian Cancer Survivors

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Abstract

Background A theory-guided Tailored Counseling and Navigation (TCN) intervention successfully increased cancer genetic risk assessment (CGRA) uptake among cancer survivors at increased risk of hereditary breast and ovarian cancer (HBOC). Understanding the pathways by which interventions motivate behavior change is important for identifying the intervention's active components.

Purpose We examined whether the TCN intervention exerted effects on CGRA uptake through hypothesized theoretical mediators.

Methods Cancer survivors at elevated risk for HBOC were recruited from three statewide cancer registries and were randomly assigned to three arms: TCN (n = 212), Targeted Print (TP, n = 216), and Usual Care (UC, n = 213). Theoretical mediators from the Extended Parallel Process Model, Health Action Planning Approach, and Ottawa Decision Support Framework were assessed at baseline and 1-month follow-up; CGRA uptake was assessed at 6 months. Generalized structural equation modeling was used for mediation analysis.

Results The TCN effects were most strongly mediated by behavioral intention alone (β = 0.49 and 0.31) and by serial mediation through self-efficacy and intention (β = 0.041 and 0.10) when compared with UC and TP, respectively. In addition, compared with UC, the TCN also increased CGRA through increased perceived susceptibility, knowledge of HBOC, and response efficacy.

Conclusions Risk communication and behavioral change interventions for hereditary cancer should stress a person's increased genetic risk and the potential benefits of genetic counseling and testing, as well as bolster efficacy beliefs by helping remove barriers to CGRA. System-level and policy interventions are needed to further expand access.

Lay summary

It is recommended that cancer survivors at increased risk for heredity seek cancer genetic risk assessment (CGRA), which includes cancer genetic counseling and genetic testing. A Tailored Counseling and Navigation (TCN) intervention successfully increased CGRA uptake among women with a history of cancer who enrolled in a randomized controlled trial. Understanding reasons for TCN's effectiveness can guide future interventions that use risk messages and behavior change techniques. We conducted mediation analyses, which enabled identification of the TCN's active components. Eligible breast and ovarian cancer survivors (n = 641) were recruited from three statewide cancer registries and were assigned to three groups: TCN, Targeted Print, and Usual Care. Mediator variables drawn from behavioral and risk communication theories were assessed at baseline and 1-month follow-up; CGRA uptake was assessed at 6 months. The strongest mediator was intention to obtain a CGRA, followed by self-efficacy, perceived risk, knowledge of hereditary breast and ovarian cancer, and perceived CGRA benefits. Risk communication and behavioral change interventions for hereditary cancer should stress a person's increased genetic risk and the potential benefits of genetic counseling and testing, as well as bolster efficacy beliefs by helping remove CGRA barriers. System-level and policy interventions are needed to further expand access.

Keywords Remote behavioral intervention · Cancer genetic risk assessment · Hereditary breast and ovarian cancer syndrome · Early detection of cancer · Extended Parallel Process Model · Genetic counseling

Introduction

Cancer survivors with certain pathogenic variants in genes, such as BRCA1 and BRCA2, are at higher risk of a second primary breast, ovarian, and possibly other types of cancer [1, 2]. These pathogenic variants also increase the risk of cancers in their relatives [1, 3, 4]. Identifying cancer survivors who are at increased risk of hereditary breast and ovarian cancer (HBOC) is crucial for informing cancer prevention, early detection, and treatment [5–7]. It is also critical to enable subsequent cancer prevention and early detection in their biological relatives [8, 9].

National guidelines recommend cancer genetic risk assessment (CGRA) for women diagnosed with epithelial ovarian, fallopian tube, primary peritoneal, and high-risk breast cancers [10]. CGRA is a consultation service that includes clinical assessment of hereditary cancer risks, genetic testing when appropriate, and risk management recommendations. Although the clinical utility of CGRA is well established, over half of breast and ovarian cancer survivors who meet national criteria have not had a CGRA [11, 12]. Moreover, fewer than one in five eligible breast and ovarian cancer survivors have undergone genetic testing [13]; thus, a large number of cancer survivors and families do not benefit from cancer genetic services. The underutilization of CGRA is more common in rural residents and racial and ethnic minorities [14–16]. Barriers to CGRA and genetic testing include low awareness of HBOC risks and genetic testing [14, 15, 17], anticipated negative emotional reactions [15, 17], lack of physician recommendation and referral [11, 16], logistical difficulties [17, 18], and concerns about insurance discrimination [15, 17] and out-of-pocket expenses [15, 17, 18].

The Genetic Risk Assessment for Cancer Education and Empowerment (GRACE) Project aimed to increase guideline-concordant CGRA in ethnically and geographically diverse cancer survivors who met the National Comprehensive Cancer Network's (NCCN) criteria for CGRA [10, 19]. The overarching goal of this three-arm population-based trial was to test the efficacy of two remote interventions to increase guideline-based CGRA for HBOC: a theory-guided, phone-based Tailored Counseling and Navigation (TCN) intervention, and a low-intensity mailed Targeted Print (TP) intervention, compared with Usual Care (UC). It also aimed to assess the underlying theoretical mechanisms of intervention effects. Specifically, the GRACE Project examined the extent to which threat perceptions, efficacy beliefs, emotions, decisional conflict, and knowledge might be affected by the TCN or TP, and whether these mediating factors would lead to increased rates of CGRA. We previously reported that more women in the TCN arm sought CGRA than those in the TP and UC, and that the effect of TP was not significantly different from UC [19]. Because TCN successfully improved CGRA uptake, it is critical to examine the mechanisms of this effect by eliciting the active components of the intervention, identifying areas for future intervention refinement, and enhancing the understanding of the theoretical underpinnings of TCN. For quality study reporting, we followed an international, Consensus-based Guideline for Reporting Mediation Analyses [20].

Theoretical Framework

The strongest behavioral interventions often integrate constructs from multiple theories [21]. We combined constructs and propositions in the Extended Parallel Process Model (EPPM), Health Action Planning Approach (HAPA), and the Ottawa Decision Support Framework (ODSF) to guide the intervention development, implementation, analysis, and evaluation [22–27].

The EPPM focuses on risk information communication [27, 28]. It emphasizes channeling fear in a positive direction to control the external risk (i.e., seeking CGRA) and reduce maladaptive responses (e.g., rejecting the risk message). As individuals receive risk messages about HBOC, they initiate threat appraisals, including perceived severity (the perceived seriousness of HBOC) and susceptibility (the perceived likelihood of having HBOC). When individuals believe HBOC is a life-threatening disease and that they and/or their family members are at increased risk for HBOC and/or a second cancer, fear is elicited, and individuals are then motivated to begin efficacy appraisals. Efficacy appraisals include response efficacy (perceived effectiveness of CGRA for reducing the threat of HBOC) and self-efficacy (beliefs in one's ability to perform the recommended response, seeking CGRA) [27]. When individuals believe that CGRA is effective in reducing HBOC risk and have high levels of confidence in their ability to obtain CGRA, they are more motivated to control the danger by seeking CGRA. The TP addressed threat and efficacy beliefs in the educational brochure, and the TCN provided personalized counseling and navigation to activate both threat and efficacy beliefs.

While the EPPM guides strategy development to promote the intention to engage in health behavior, the HAPA recognizes that many individuals are already motivated to engage in health behavior but fail to carry out those goals. The HAPA proposes that the nature of behavior change consists of two processes: forming an intention (motivation phase), followed by planning to act and action (volition phase) [26, 29]. TCN drew from the second phase to bridge the gap between intentions and CGRA uptake. Health coaches used an implementation-intention strategy to help patients formulate a plan to obtain CGRA by specifying when, where, and what steps would be taken. This strategy was effective in promoting intended behavior in previous studies [30, 31].

The ODSF was developed for assessing healthcare decisional needs, providing decision support, and evaluating decision outcomes [22, 25]. The ODSF provides a structure to facilitate informed decision-making and measure cognitive factors (in addition to those that comprise the EPPM), such as knowledge of HBOC and decisional conflict. Both TP and TCN addressed knowledge of HBOC, but only health coaches in the TCN arm provided decision support by addressing uncertainty about decisions. Knowledge deficits regarding HBOC are well documented [14, 17]. We assumed sufficient understanding of HBOC may be a prerequisite for women to understand the effectiveness of genetic counseling and testing (i.e., response efficacy). For example, CGRA is still applicable and beneficial if participants have sons but not daughters.

While the intervention content was guided by the aforementioned theories, the delivery style of coaching sessions in the TCN arm drew from motivational interviewing, a semi-directive counseling style that helps people resolve ambivalence and move toward healthy changes [23, 24]. Motivational interviewing helped health coaches to establish a respectful, collaborative relationship with participants and encourage them to engage and respond more positively to risk

information during conversations that were directed to the identified goal (i.e., to seek CGRA).

Purpose

We aimed to compare cognitive and emotional mediators across the three study arms (TCN, TP, and UC) and assess potential underlying theoretical mediating mechanisms. We hypothesized that TCN would activate perceived threat (which in turn aroused fear of HBOC), enhance perceived efficacy and knowledge of HBOC, and reduce decisional conflict; these changes on the putative mediators would then lead to increased CGRA intention (i.e., motivation), and ultimately, CGRA uptake.

Methods

Study Design

The GRACE was a three-arm randomized superiority trial [32]. The study was approved by the Institutional Review Boards of the Colorado Cancer Registry, Rutgers University, and the University of New Mexico. Women with a history of breast and ovarian cancer were recruited from November 2017 to July 2020, and all enrollees provided informed consent. After completion of the baseline survey, participants were randomized into one of the three arms using a computer-generated random number list with a size of 9 in each block. The TP materials (in English or Spanish) were mailed to both TP and TCN arms within 1 week of completing the baseline survey. The telephone psychoeducational sessions in the TCN arm occurred approximately 2 weeks after the completion of the baseline survey. Follow-up surveys were then administered 1 and 6 months following the interventions for the TP and TCN arms and the baseline survey for the UC arm. The primary outcome was CGRA uptake at the 6-month follow-up. High intervention fidelity was evidenced by 95% of TCN coaching sessions compliant with the fidelity checklist; key indicators of motivational interviewing fidelity (e.g., global scores, % complex reflections) were consistently in the good to excellent range [19, 33].

Participants and Procedures

Our study sample consisted of 641 women recruited from three statewide cancer registries: Colorado Cancer Registry, New Jersey State Cancer Registry, and New Mexico Tumor Registry. To be eligible, participants had to be biologically female, fluent in English, and meet at least one of the NCCN criteria for CGRA that could be obtained from cancer registry data (breast cancer diagnosed at age ≤50 years; triple-negative breast cancer diagnosed at age ≥60 years; ≥two primary breast cancers; or any epithelial ovarian, peritoneal, or fallopian tube cancer), and have not had CGRA.

No intervention was delivered to the UC arm in the 6 months after enrollment. Participants in the TP arm (targeted print-only arm) received a brochure designed for high-risk breast and ovarian cancer survivors that addressed important theoretical determinants of CGRA uptake: knowledge about HBOC risks, threat appraisal (to validate or raise perceived seriousness and risks of HBOC), response efficacy (benefits and expectations about CGRA), self-efficacy (CGRA resources, cost, and insurance reimbursement), and possible actions to take (e.g., to make an appointment with a cancer risk specialist).

Women in the TCN arm were mailed the same brochure and a sealed envelope containing visual aids. Subsequently, they were engaged in a 30- to 45-min tailored telephone call with a health coach. All coaches were trained in the theoretical aspects of the intervention and in motivational interviewing by an experienced motivational interviewing trainer who periodically reviewed session tapes and met with coaches to ensure intervention fidelity. The coaches incorporated risk communication and behavioral change techniques to impact participants' cognitive and emotional intervention targets. The coaching session was tailored according to participants' perceptions of threat, efficacy, fear of HBOC, knowledge about HBOC and CGRA, and barriers to CGRA, as well as their decisional conflict expressed during the TCN session. The session was also tailored according to other individual characteristics (e.g., logistical concerns, personal and family cancer history, cultural beliefs such as fatalism). Participants were navigated to a cancer genetic provider when appropriate. An action plan was developed during the session and women were reminded of their action plan in a follow-up tailored letter (sent immediately after the telephone call) and in a mailed Action Plan Reminder Card (6 weeks after the call). In addition, when participants gave permission, the researchers sent a letter that informed the patient's providers (primary care or oncology provider) that their patient met the criteria for a genetics referral. Finally, participants received another phone call approximately 7 weeks after the initial call to provide additional navigation if needed. The study enrollment, randomized assignment, and retention are shown in Fig. 1. Details about intervention development, subject randomization, masking, and implementation were reported elsewhere [19].

Measures

We used standardized measures to evaluate potential theoretical mediators. The four cognitive EPPM constructs were perceived susceptibility, perceived severity, self-efficacy, and response efficacy. They were measured with the adapted Risk Behavior Diagnosis Scale, which consists of four subscales measuring each of the four constructs [34]. Each subscale contains four items, and responses to each item range from 1 (strongly disagree) to 5 (strongly agree). For all subscales, higher scores indicate stronger threat or efficacy beliefs. The baseline and 1-month alphas ranged from 0.81 to 0.93.

An emotional construct of the EPPM, fear of HBOC, was measured with the Negative Affect in Risk subscale of the Cancer Risk Belief Scale [35]. The 6-item subscale measured perception of HBOC risk; item responses range from 1 (strongly disagree) to 4 (strongly agree). Higher scores indicate greater fear of HBOC. The baseline and 1-month alphas were both 0.94.

HBOC knowledge was measured with an adapted National Center for Human Genome Research Questionnaire [36, 37]. The 11-item scale examines knowledge of HBOC risk and prevention, cancer hereditary patterns, and cancer genetic testing; responses to items are "true" (1), "false" (0), and "don't know" (0). Higher sum scores indicate a higher knowledge level of HBOC. The baseline and 1-month alphas for the scale were 0.89 and 0.86.

Decisional conflict regarding CGRA was measured with the 4-item SURE scale (Sure of myself; Understand information; Risk-benefit ratio; Encouragement) [38]. Responses to

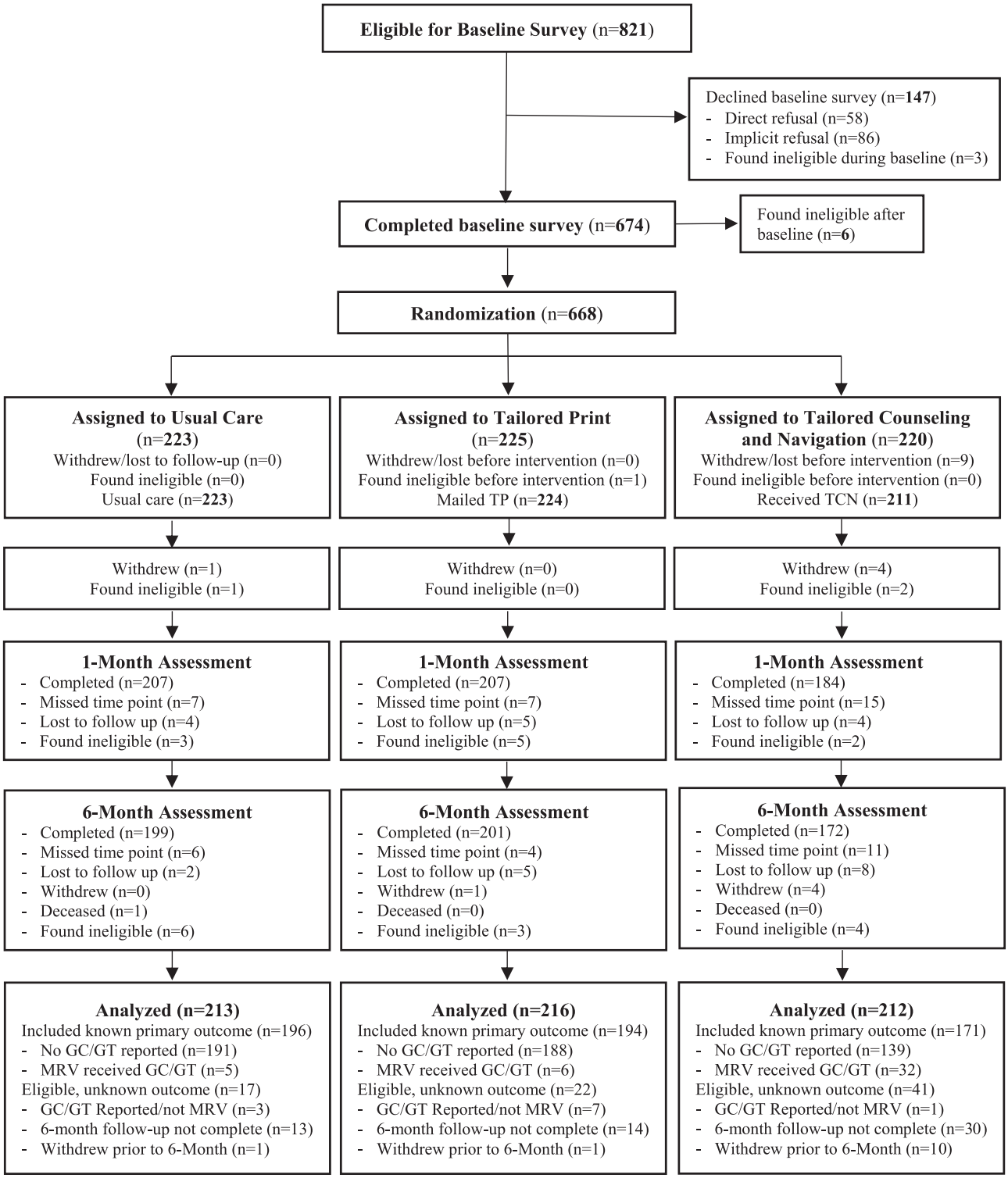

Fig. 1. GRACE study CONSORT diagram. *Note.* Some participants were found ineligible after randomization and were removed from all analyses. These participants had prior cancer genetic risk assessment, but they did not report it at recruitment due to recall errors.

items are "yes" (1) and "no" (0). Higher sum scores indicate lower decisional conflict. The 1-month alpha for the scale was 0.73.

CGRA intention at 1 month was assessed in terms of the self-reported likelihood that the participant would undergo CGRA for HBOC within the next 6 weeks. Responses for this item ranged from 1 (not at all) to 5 (extremely likely).

Most hypothesized mediators were measured in the baseline and 1-month surveys; decisional conflict was measured only in the 1-month survey because the decision (seeking CGRA or not) was not elicited prior to the intervention. The primary outcome was CGRA uptake as assessed in the 6-month survey and verified through medical record documentation.

Covariates included demographic and clinical characteristics, which were assessed with standard measures. Health literacy was assessed with a 3-item literacy screening questionnaire [39]. Responses to items ranged from "none of the time" (0) to "all of the time" (4). Higher sum scores indicate lower health literacy. The baseline alpha for the scale was 0.74.

Statistical Analysis

The demographic and clinical characteristics of participants were compared among three arms using ANOVA and chi-square analysis. Theoretical mediators at baseline and 1 month were summarized and their distribution was examined with histograms. The mediators at baseline and 1 month were compared (within arms and between arms) using paired *t*-tests and two-sample *t*-tests; CGRA uptake at 6 months was compared using a chi-square test.

Generalized structural equation modeling (GSEM) was used for mediation analysis because CGRA uptake is a binary outcome [40]. All 1-month cognitive and emotional mediators were predicted by preintervention corresponding measures to control for baseline levels. All scores were standardized prior to fitting the model to generate standardized coefficients. Maximum likelihood estimation was used for estimating regression coefficients/parameters [40]. Indirect effects were estimated, and a bootstrapping method (1,000 replications) was used to construct 95% confidence intervals.

Based on prior research, age, household income, education attainment, ethnicity, health insurance, urban/rural residence, cancer type, years since diagnosis, health literacy, and family history of cancer were controlled as covariates in all regressions in GSEM. In addition, we adopted negative imputation for the outcome variable, CGRA uptake—we assumed if there was no documented verification of CGRA, the outcome behavior did not occur. To determine if the results were robust, sensitivity analyses with multiple imputation for missing data was conducted, based on the assumption of missing at random [41]. Twenty imputations were performed using Multiple Imputation by Chained Equations [42]. Effect estimates were derived using Rubin's rule [41]. All analyses were performed using StataMP 17. The Monte Carlo simulation technique, similar to Tofighi and MacKinnon's method, was used to examine the power and estimate the minimal detectable indirect effects [43]. Results showed that our study has at least 80% power (alpha = 0.05, two-sided) to test a minimal indirect effect of 0.37 for a single mediator, and an indirect effect of 0.007–0.032 for serial mediation with two to three mediators.

Results

Participants' demographic and clinical characteristics are summarized in Table 1. A total of 641 women were randomly assigned to TCN (n = 212), UC (n =213), and TP (n = 216). Of the 43 women who had medical record verified CGRA by the 6-month follow-up, 32 (74.4%) were from the TCN arm, compared with only 5 (11.6%) and 6 (14.0%) women from UC and TP, respectively (Table 2).

Table 3 describes assessments of theoretical mediators at baseline and 1 month. At baseline, the cognitive and emotional measures were not statistically different between the three arms, except that response efficacy was slightly higher in TCN compared with TP (mean difference = −0.52, p = .049). Thus, these baseline measures were controlled to tease out the effects of the 1-month mediators and account for their variations over time. At 1 month after the intervention, women randomized to TCN had significantly higher perceived susceptibility of HBOC, higher self-efficacy and intention to seek CGRA, as well as lower decisional conflict, compared with women randomized to UC and TP. Also, at 1 month, none of the comparisons were statistically significant between UC and TP. When compared within study arms, perceived susceptibility and self-efficacy improved significantly in only the TCN arm, while knowledge of HBOC increased in both the TCN and TP arms.

We also examined the perceived severity of HBOC at baseline and 1 month. A negatively skewed distribution was observed (at the two time points: median = 18 and 17; range = 4–20). Participants commonly reported high perceived severity with little variation, which was consistent with previous research [44]. Thus, perceived severity was not included in GSEM.

Figure 2 shows standardized path coefficients and corresponding significance levels of the model comparing TCN and UC (Model 1). Table 4 shows the estimates of indirect effects of the (serial) mediation chains. We found that most hypothesized mediational chains from the TCN intervention to CGRA uptake were supported. Specifically, compared with UC, TCN directly increased perceived susceptibility (β_{DE} = 0.25), leading to higher intentions to seek CGRA (β_{DE} = 0.17), and ultimately increased CGRA uptake at 6 months (β_{DE} = 0.80). This serial mediation effect was significant in bootstrap estimation (β_{IE} = 0.034). Similarly, compared with UC, TCN increased self-efficacy (β_{DE} = 0.20), which was associated with higher intentions (β_{DE} = 0.24) that increased CGRA uptake. This serial mediation effect was 0.041. Additionally, TCN enhanced knowledge of HBOC (β_{DE} = 0.20), which improved response efficacy beliefs about CGRA (β_{DE} = 0.20) and then intentions (β_{DE} = 0.24) and, ultimately, CGRA uptake (β_{IE} = 0.0076). The mediation effect through intention alone was 0.49.

Figure 3 and Table 5 summarize potential mediating pathways for TCN effects compared with TP (Model 2). A similar pattern of direct coefficients was found. However, three differences were noted. First, the effect of TCN on perceived susceptibility at 1 month was marginally significant (β_{DE} = 0.16, p = .069), as was the association between perceived susceptibility and intention (β_{DE} = 0.11, p = .051). TCN did not significantly improve knowledge of HBOC (β_{DE} = 0.045, p = .45), compared with TP. Finally, higher levels of fear of HBOC were significantly associated with higher intention to seek CGRA in Model 2 (β_{DE} = 0.15, p = .008). Mediation effects were also tested (Table 5); compared with TP, an indirect effect of TCN on CGRA uptake passed through intention alone (β_{IE} = 0.31), as well as through both self-efficacy and intention (β_{IE} = 0.10). Other theoretical variables, such as perceived susceptibility and fear, did not significantly mediate intervention effects.

The response rate was 91.3% at the 1-month assessment, leading to about 10% of missing values in Models 1 and 2. Thus, sensitivity analyses with multiple imputation for missing data were conducted (results presented in Supplementary Material). The GSEM comparing TCN and UC yielded results close to Model 1, except that the intervention effects on self-efficacy became marginally significant (β_{DE} = 0.18, p = .077). Similarly, the GSEM comparing TCN and TP was close

Table 1 Sociodemographic and Clinical Characteristics of Subjects by Study Arm

Study arm	All (*N* = 641) *n* (%)	UC (*N* = 213) *n* (%)	TP (*N* = 216) *n* (%)	TCN (*N* = 212) *n* (%)	*p* value
Age (mean, *SD*)	61.1 (10.2)	61.0 (9.9)	61.1 (10.1)	61.2 (10.7)	.99
Years since diagnosis (mean, *SD*)	11.2 (7.7)	11.3 (7.7)	11.0 (7.6)	11.3 (7.7)	.85
Health literacy level (mean, *SD*)	4.16 (2.4)	4.15 (5.7)	4.18 (5.4)	4.16 (6.4)	.99
Self-reported race and ethnicity					.43
Hispanic	160 (25.4)	55 (26.4)	47 (22.1)	58 (27.9)	
Non-Hispanic White	379 (60.3)	129 (62.0)	131 (61.5)	119 (57.2)	
Non-Hispanic Black	37 (5.9)	7 (3.4)	17 (8.0)	13 (6.3)	
Other	53 (8.4)	17 (8.2)	18 (8.5)	18 (8.7)	
Missing	12	5	3	4	
Self-reported Ashkenazi Jewish ancestry					.60
No	597 (97.1)	198 (97.1)	203 (96.2)	196 (98.0)	
Yes	18 (2.9)	6 (2.9)	8 (3.8)	4 (2.0)	
Missing	26	9	5	12	
Marital status					.40
Single/divorced/separated/widowed	251 (39.2)	76 (35.7)	90 (41.9)	85 (40.1)	
Married/domestic partnership	389 (60.8)	137 (64.3)	125 (58.1)	127 (59.9)	
Missing	1	0	1	0	
Education level					.37
Less than high school/High school grad/GED	115 (18.1)	43 (20.3)	40 (18.7)	32 (15.3)	
Some college, Assoc Degree, or Vocational School	230 (36.2)	81 (38.2)	69 (32.2)	80 (38.3)	
Bachelor's degree or higher	290 (45.7)	88 (41.5)	105 (49.1)	97 (46.4)	
Missing	6	1	2	3	
Annual household income					.95
<$30,000	141 (24.8)	51 (27.0)	44 (22.9)	46 (24.5)	
$30,000–$49,999	101 (17.8)	35 (18.5)	35 (18.2)	31 (16.5)	
$50,000–$69,999	85 (14.9)	29 (15.3)	28 (14.6)	28 (14.9)	
$70,000 or more	242 (42.5)	74 (39.2)	85 (44.3)	83 (44.1)	
Missing	72	24	24	24	
Rural/urban residence					.12
Urban	529 (82.5)	185 (86.9)	175 (81.0)	169 (79.7)	
Rural	112 (17.5)	28 (13.1)	41 (19.0)	43 (20.3)	
Has health insurance					.31
No	35 (5.5)	15 (7.0)	8 (3.7)	12 (5.7)	
Yes	604 (94.5)	198 (93.0)	208 (96.3)	198 (94.3)	
Missing	2	0	0	2	
Has a personal health care provider					.95
No	26 (4.1)	9 (4.2)	8 (3.7)	9 (4.2)	
Yes	615 (95.9)	204 (95.8)	208 (96.3)	203 (95.8)	
Cancer site					.40
Ovarian	94 (14.7)	36 (16.9)	32 (14.8)	26 (12.3)	
Breast	547 (85.3)	177 (83.1)	184 (85.2)	186 (87.7)	
Number of first- (FDR) and second-degree relatives (SDR) with breast or ovarian cancer					.83
0 FDR and 0 SDR	409 (63.8)	132 (62.0)	142 (65.7)	135 (63.7)	
1 FDR or 1 SDR	131 (20.4)	49 (23.0)	40 (18.5)	42 (19.8)	
2 or more FDR/SDR	101 (15.8)	32 (15.0)	34 (15.7)	35 (16.5)	
Has ever heard about CGRA or genetic counseling prior to the study					.60
No	252 (39.4)	78 (36.6)	87 (40.5)	87 (41.0)	
Yes	388 (60.6)	135 (63.4)	128 (59.5)	125 (59.0)	
Missing	1	0	1	0	

Note. *CGRA* cancer genetic risk assessment; *SD* standard deviation; *TCN* Tailored Counseling and Navigation; *TP* Targeted Print; *UC* Usual Care. Rural or urban residence was based on Rural-Urban Commuting Area (RUCA) codes at the zip code level. The demographic and clinical characteristics of participants were compared among three arms using ANOVA and chi-square analysis.

Table 2 Self-reported and Medically Verified Cancer Genetic Risk Assessments by Study Arm

Study arm	All (*N* = 641) *n* (%)	UC (*N* = 213) *n* (%)	TP (*N* = 216) *n* (%)	TCN (*N* = 212) *n* (%)	*p* value[a]
Self-reported CGRA uptake at 6 months					<.05
Had CGRA	56 (9.8)	9 (4.5)	14 (7.0)	33 (19.3)	
No CGRA	514 (90.2)	190 (95.5)	186 (93.0)	138 (80.7)	
Missing	71	14	16	41	
Medical record verified CGRA uptake					<.01
Had CGRA	43 (7.5)	5 (2.5)	6 (3.0)	32 (18.7)	
No CGRA	527 (92.5)	194 (97.5)	194 (97.0)	139 (81.3)	
Missing	71	14	16	41	
Negatively imputed verified CGRA uptake					<.01
Had CGRA	43 (6.7)	5 (2.3)	6 (2.8)	32 (15.1)	
No CGRA	598 (93.3)	208 (97.7)	210 (97.2)	180 (84.9)	

Note. *CGRA* cancer genetic risk assessment; *TCN* Tailored Counseling and Navigation; *TP* Targeted Print; *UC* Usual Care.
[a]CGRA uptake at 6 months was compared across three arms using chi-square tests.

to Model 2. Our interpretation was thus based on Models 1 and 2.

Discussion

To understand the mechanisms underlying the intervention effects, we examined whether TCN exerted effects on CGRA uptake through hypothesized theoretical mediators (intervention targets) and the extent of TCN's influence. As expected, the TCN intervention effectively heightened perceived susceptibility, compared with both UC and TP. This was expected because health coaches specifically tailored TCN according to participants' personal and family history of cancer and addressed their misconceptions about HBOC risks (e.g., a woman with breast or ovarian cancer is not at risk if they do not have a family history of cancer). Based on the EPPM, the threat of HBOC was not perceived to be significant unless it became personally relevant [27, 45]; TP was thus not as effective as TCN even if it also addressed the increased risk of HBOC. Furthermore, the serial mediation effect of TCN on CGRA uptake passed through perceived susceptibility and then intention, supporting the important role threat perception played in promoting CGRA uptake. In addition, perceived susceptibility mediated the intervention effects on fear of HBOC, which aligned with EPPM and previous research that fear is an important immediate outcome when risks become threatening [27, 44, 46, 47].

Although we hypothesized that TCN would have a direct effect on response efficacy, the relationship was not significant; instead, TCN had indirect effects on response efficacy through enhanced knowledge of HBOC, compared with UC (Model 1). The positive association between knowledge of HBOC and response efficacy indicated that a basic understanding of hereditary cancers (e.g., preventive options are available following a positive test) may help individuals at increased risk appreciate the usefulness of CGRA in informing them of their personal and familial risk and possible risk reduction strategies. The serial mediation chain through knowledge of HBOC, response efficacy, and intention (serial mediation 3 in Table 4) requires further testing because our study measured the mediators at the same time point (i.e., 1-month follow-up). In addition, the association between TCN versus TP and knowledge of HBOC was not significant (Model 2). This suggests that the TP materials increased women's knowledge of HBOC to some extent, consistent with existing evidence of the usefulness of this low-cost approach to raise awareness [48, 49]. However, our study supported the conclusion that change in knowledge alone is not sufficient in inducing behavioral change.

TCN also increased self-efficacy compared with both UC and TP. This effect then strengthened CGRA intention and ultimately increased uptake. Efficacy appraisals play a critical role in determining whether a subsequent response is adaptive or maladaptive [27, 50]. To successfully employ fear appeals, strong evidence suggests that such appeals need to be accompanied by efficacy messages about effective and easily implemented action recommendations [45, 50]. Otherwise, with low efficacy beliefs, fear might be channeled toward maladaptive responses such as dismissing the message or messenger (the boomerang effect). Our findings suggest that TCN's personalized discussion of participants' ability to seek CGRA and strategies to overcome anticipated barriers (e.g., connecting participants to genetic counseling services if they did not know where to find one) can help avoid a fear-related "boomerang."

Intention as an immediate antecedent of behavior has been widely supported in empirical research [51, 52]. CGRA intention alone mediated 23.98% and 18.07% of the total intervention effects on CGRA uptake in Models 1 and 2, suggesting that intention is a crucial mediator and that our implementation-intention strategy successfully translated women's goals (seeking CGRA to learn more about their HBOC risks and risk management strategies) into action [29, 31].

Aligning with our hypothesis, fear of HBOC was induced by TCN in both Models 1 and 2. However, increased fear was associated with higher CGRA intention only when comparing TCN with TP. We cannot make definitive conclusions about fear's effect on our outcomes based on the discordant findings. The original EPPM postulated that fear does not directly affect people's adaptive responses [27, 44], but some research has shown that fear can be directly associated with certain protective behaviors (e.g., adopting cancer screening

Table 3 Theoretical Mediators at Pre- and 1-Month Postintervention in Three Arms

	Within study arms				Between study arm		
	Study arms	Baseline mean (*SD*)	One-month mean (*SD*)	Mean difference (*SE*)[a]	Study arms	Baseline mean difference (*SE*)[b]	One-month mean difference (*SE*)[b]
Perceived susceptibility	UC	13.32 (11.98)	13.31 (11.13)	0.03 (0.20)	TCN vs. UC	−0.06 (0.33)	0.70 (0.33)*
	TP	12.96 (12.51)	13.35 (10.12)	0.41 (0.22)	TCN vs. TP	0.30 (0.34)	0.67 (0.32)*
	TCN	13.25 (11.79)	14.02 (9.73)	0.81 (0.24)**	TP vs. UC	−0.36 (0.24)	−0.04 (0.32)
Response efficacy	UC	16.42 (8.76)	16.44 (9.16)	−0.03 (0.21)	TCN vs. UC	0.40 (0.26)	0.14 (0.29)
	TP	16.30 (8.74)	16.30 (6.53)	−0.04 (0.20)	TCN vs. TP	0.52 (0.26)*	0.28 (0.26)
	TCN	16.82 (6.13)	16.57 (6.81)	−0.31 (0.21)	TP vs. UC	−0.12 (0.29)	−0.14 (0.28)
Self-efficacy	UC	13.40 (10.18)	13.58 (9.43)	0.17 (0.23)	TCN vs. UC	−0.19 (0.31)	0.64 (0.30)*
	TP	13.07 (11.26)	13.24 (10.08)	0.16 (0.22)	TCN vs. TP	−0.53 (0.32)	0.98 (0.30)**
	TCN	13.59 (9.86)	14.22 (7.46)	0.59 (0.27)*	TP vs. UC	0.33 (0.32)	−0.34 (0.31)
Knowledge of HBOC	UC	5.93 (6.67)	5.85 (7.56)	−0.08 (0.18)	TCN vs. UC	0.25 (0.25)	0.41 (0.29)
	TP	5.61 (6.53)	5.99 (7.18)	0.38 (0.19)*	TCN vs. TP	−0.09 (0.25)	0.28 (0.28)
	TCN	5.69 (6.93)	6.26 (10.22)	0.58 (0.22)*	TP vs. UC	0.33 (0.25)	0.13 (0.26)
Fear of HBOC	UC	2.41 (0.79)	2.47 (0.68)	0.07 (0.05)	TCN vs. UC	−0.03 (0.09)	0.04 (0.08)
	TP	2.33 (0.82)	2.48 (0.72)	0.16 (0.04)**	TCN vs. TP	−0.10 (0.09)	0.03 (0.09)
	TCN	2.43 (0.81)	2.52 (0.70)	0.08 (0.05)	TP vs. UC	0.08 (0.09)	0.01 (0.08)
Decisional conflict	UC	–	2.44 (2.11)	–	TCN vs. UC	–	0.89 (0.13)**
	TP	–	2.55 (1.79)	–	TCN vs. TP	–	0.78 (0.13)**
	TCN	–	3.33 (1.16)	–	TP vs. UC	–	0.11 (0.14)
CGRA intention	UC	–	2.69 (1.27)	–	TCN vs. UC	–	0.75 (0.12)**
	TP	–	2.78 (1.29)	–	TCN vs. TP	–	0.66 (0.12)**
	TCN	–	3.44 (1.46)	–	TP vs. UC	–	0.09 (0.11)

Note. *CGRA* cancer genetic risk assessment; *HBOC* hereditary breast and ovarian cancer; *SD* standard deviation; *SE* standard error; *TCN* Tailored Counseling and Navigation; *TP* Targeted Print; *UC* Usual Care.
[a]Paired *t*-tests were used to compare baseline and 1-month differences within each arm (complete cases were included in the tests).
[b]Two-sample *t*-tests were used to compare between-arm differences at two time points (baseline and 1 month).
*$p < .05$.
**$p < .01$.

tests) [28, 46, 51]. The role played by emotional elements is more complex than what was tested in these studies. For example, anxiety, a construct not included in our theoretical framework, may have been triggered due to the uncertainty of developing HBOC during one's lifetime [9, 28]. Moreover, the relationship between emotions (fear and anxiety) and threat in a condition of high efficacy is described as bidirectional [27, 50]. That is, perceived threat arouses fear; fear, in turn, may upgrade perceived threat, which then motivates adaptive responses [27, 50]. With more comprehensive measures and relevant study design, future research could disentangle the relationship between emotional elements and perceived threat, as well as the emotions' effects on behavior outcomes [50].

Decisional conflict was significantly reduced by the TCN, as nearly half the participants (n = 258, 44.18%) expressed no decisional conflict on the measure assessing this concept. However, reduced decisional conflict did not directly increase CGRA uptake in our study. A possible explanation is that some barriers may have served as a moderator; barriers to CGRA uptake have reduced actual behavior control when a decision to seek CGRA has been determined [29, 52]. The top two barriers to genetic counseling reported by our participants were lack of provider referral (33.7%) and cost (26.5%) [19]. Another possible reason for this insignificant link was that this testing was not sufficiently powered due to our binary CGRA outcome.

We also observed a strong direct effect of TCN on CGRA uptake, compared with both UC and TP, indicating that other components of the intervention unrelated to the tested mediators may have been operating. For example, the tailored letter following the telephone session could serve as a cue to action for participants. The follow-up phone call to participants 7 weeks after the TCN phone session was designed to provide additional navigation to remove barriers. Further, motivational interviewing itself might have helped reduce resistance to change, but we did not measure this directly. The clinical letter sent to TCN participants' providers informed them about their patient's increased risk and increased providers' knowledge and thus led to a CGRA referral [53].

When compared with UC, TCN's effects on CGRA uptake through the serial mediation chain 1–3 in Model 1 (Table 4) was modest (ranging 0.008–0.041) and explained only 4.07% of the intervention effect. Similarly, compared with TP, the TCN effects mediated by self-efficacy and intention (Table 5) constituted 5.97% of total effects. This was, in part, because the direct intervention effect on CGRA uptake was very strong—the CGRA uptake rate in the TCN arm was seven times the rate in UC. This led to a large standardized direct effect (β_{DE} = 1.46, log odds) calculated from the logistic regression equation using CGRA uptake as the outcome. Although the serial mediation effects were modest in size, this is nonetheless noteworthy, because measurement accuracy of psychological variables is limited by the fact that salient beliefs

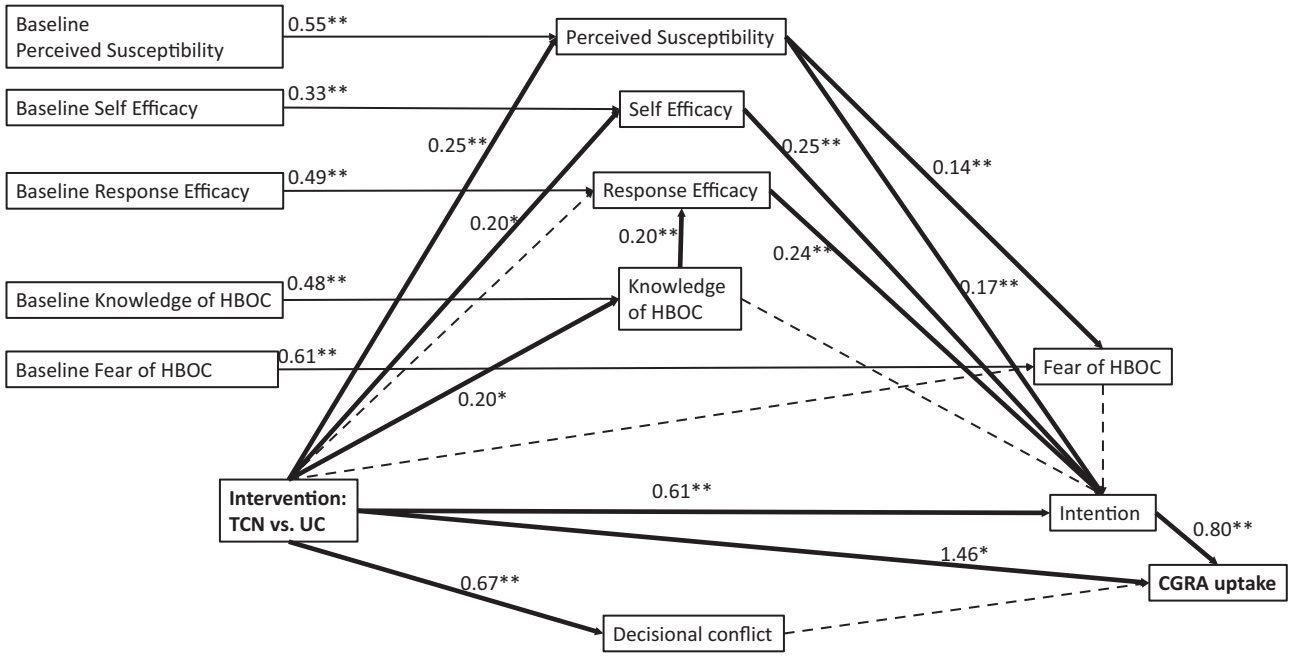

Fig. 2. A generalized structural equation model (Model 1) linking intervention (Tailored Counseling and Navigation vs. Usual Care), theoretical mediators, and cancer genetic risk assessment uptake. *Note. CGRA* cancer genetic risk assessment; *HBOC* hereditary breast and ovarian cancer; *TCN* Tailored Counseling and Navigation; *UC* Usual Care. All parameters are standardized effect sizes. All relationships were controlled for age, household income, education attainment, ethnicity, years since diagnosis, health insurance, urban/rural residence, breast or ovarian cancer, health literacy, and whether having at-risk first- or second-degree relatives. Theoretical mediators were assessed at baseline and then 1 month after the intervention; CGRA uptake was assessed 6 months after the intervention. *$p < .05$; **$p < .01$. Nonsignificant pathways are shown in dash arrows.

Table 4 Standardized Path Estimates of the Effects of TCN Versus UC on CGRA Uptake and Proportion-Mediated Measures

	Perceived susceptibility	Self-efficacy	Knowledge of HBOC	Response efficacy	Intention	CGRA uptake	Estimate of effect	Proportion mediated
Serial mediation (1)	0.25 [0.077, 0.43]				0.17 [0.05, 0.29]	0.80 [0.26, 1.34]	0.034 [0.0046, 0.11]	1.68%
Serial mediation (2)		0.20 [0.010, 0.40]			0.25 [0.14, 0.36]	0.80 [0.26, 1.34]	0.041 [0.0010, 0.14]	2.01%
Serial mediation (3)			0.20 [0.020, 0.38]	0.20 [0.066, 0.35]	0.24 [0.13–0.34]	0.80 [0.26, 1.34]	0.0076 [0.0004, 0.026]	0.38%
Mediation (4)					0.61 [0.42, 0.79]	0.80 [0.26, 1.34]	0.49 [0.20, 1.17]	23.98%
Direct effect						1.46 [0.27, 2.64]	1.46 [0.27, 2.64]	71.95%
Total effect							2.02	

Note. CGRA cancer genetic risk assessment; *TCN* Tailored Counseling and Navigation; *UC* Usual Care. Each mediation (Rows 1–4) represents a distinct mediational chain from the intervention (TCN vs. UC) to intention to CGRA uptake. The product of three path estimates in serial mediation 1 [0.25*0.17*0.80 = 0.034] is the estimate of the corresponding indirect effect. Row 5 is the direct effect of the intervention (TCN vs. UC) on CGRA uptake. The sum of the 5 estimates of effect is the total effect of intervention on CGRA uptake (Row 6). The proportion mediated is the estimate of the mediation effect divided by the total effect (e.g., 0.034/2.02 yielded 1.68% for Row 1). Original estimates (no rounding) were calculated, but rounded estimates were reported.

that determine behavior constantly change, varying by time and contexts [51]. The serial mediation supports that both threat and efficacy constructs of the EPPM play an important role in motivating adaptive behavior change, and future interventions should adequately address both elements.

Although a robust intervention effect was observed, we note that only 19% of the women in TCN received a CGRA. This rather low uptake rate suggests that non-individual-level barriers need to be addressed more effectively. The time and labor-intensive nature of genetic services, combined with a national shortage of genetic counselors, limits access to CGRA. Hence, training more genetic providers is warranted. Cost has been identified as another major barrier in our study and by others [19, 54]; while many insurance plans cover CGRA, the extent of coverage and out-of-pocket costs vary widely [55]. Also, obtaining CGRA can be complex, involving multiple steps and appointments. Navigating health systems may be difficult and can decrease patients' self-efficacy in seeking CGRA. Besides intention, our study found the strongest mediator was self-efficacy, emphasizing that eliminating access barriers is of profound importance. Therefore, policy interventions and more streamlined genomic care delivery models

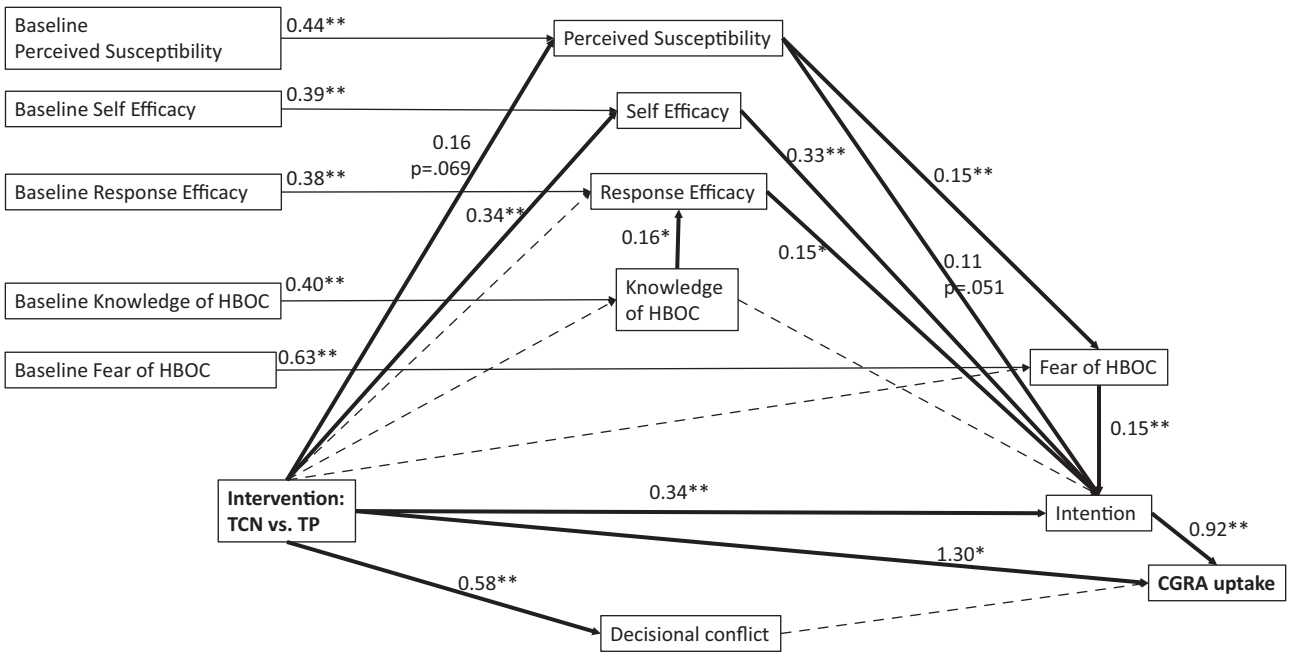

Fig. 3. A generalized structural equation model (Model 2) linking intervention (Tailored Counseling and Navigation vs. Targeted Print), theoretical mediators, and cancer genetic risk assessment uptake. *Note. CGRA* cancer genetic risk assessment; *HBOC* hereditary breast and ovarian cancer; *TCN* Tailored Counseling and Navigation; *TP* Targeted Print. All parameters are standardized effect sizes. All relationships were controlled for age, household income, education attainment, ethnicity, years since diagnosis, health insurance, urban/rural residence, breast or ovarian cancer, health literacy, and whether having at-risk first- or second-degree relatives. Theoretical mediators were assessed at baseline and then 1 month after the intervention; CGRA uptake was assessed 6 months after the intervention. *$p < .05$; **$p < .01$. Nonsignificant pathways are shown in dash arrows. *p* values are denoted for marginally significant pathways.

Table 5 Standardized Path Estimates of the Effects of TCN Versus TP on CGRA Uptake and Proportion-Mediated Measures

	Perceived susceptibility	Self-efficacy	Intention	CGRA uptake	Estimate of effect	Proportion mediated
Serial mediation (1)	*0.16 [–0.013, 0.034]*		*0.11 [–0.0006, 0.23]*	0.92 [0.36, 1.48]	*0.017 [–0.002, 0.075]*	–
Serial mediation (2)		0.34 [0.14, 0.54]	0.33 [0.23, 0.43]	0.92 [0.36, 1.48]	0.10 [0.032, 0.30]	5.97%
Mediation (3)			0.34 [0.13, 0.53]	0.92 [0.36, 1.48]	0.31 [0.11, 0.79]	18.07%
Direct effect				1.30 [0.74, 2.53]	1.30 [0.74, 2.53]	75.96%
Total effect					1.72	

Note. Nonsignificant pathways are italicized. *CGRA* cancer genetic risk assessment; *TCN* Tailored Counseling and Navigation; *TP* Targeted Print. Each mediation (Rows 1–3) represents a distinct mediational chain from the intervention (TCN vs. TP) to intention to CGRA uptake. The product of three path estimates in serial mediation 2 [0.34*0.33*0.92 = 0.10] is the estimate of the corresponding indirect effect. Row 4 is the direct effect of the intervention (TCN vs. TP) on CGRA uptake. The sum of the 4 estimates of effect is the total effect of intervention on CGRA uptake (Row 5). The proportion mediated is the estimate of the mediation effect divided by the total effect (e.g., 0.10/1.72 yielded 5.97% for Row 2). Original estimates (no rounding) were calculated, but rounded estimates were reported.

that effectively address social determinants and structural barriers for overcoming inequities and expanding access need to be developed [56], rather than solely focusing on changing individual-level barriers.

Our study's limitations should be acknowledged. A relatively small number of Black women were enrolled although we intended to recruit a more racially diverse sample. Research has shown Black patients experience unique challenges and barriers contributing to lower use of genetic counseling and testing, such as systemic racism, mistrust in medical systems, and lack of provider recommendations [53]. Thus, future research should evaluate such interventions in Black and other underserved or understudied populations. In addition, the 1-month cognitive mediators and fear were measured at the same time as intention, thereby limiting our ability to study causal relationships with CGRA intention. Moreover, although the measurement of putative mediators showed adequate internal consistency in our study, some of them contained only four to six items, which may not capture all important attributes of the construct; this measurement limitation could contribute to our modest indirect effects. Finally, structural equation modeling assumes all confounders are controlled for in analyses; unknown confounders may exist, and the assumption might not hold, leading to biased results.

Despite the limitations, our findings supported most hypothesized mediators, including perceived susceptibility, self-efficacy, response efficacy, knowledge of HBOC, and intention. Our conclusions are bolstered through the use of conservative, negative imputation for the primary outcome—CGRA

uptake. Future risk communication and behavior change interventions promoting CGRA uptake should stress the increased risk of having a pathogenic variant and the potential benefits of genetic counseling and testing, as well as bolster efficacy beliefs and motivation by helping remove barriers to obtaining CGRA, including structural and system-level barriers. Our findings also provide implications for future theory-based interventions that aim to enable people to make informed decisions and adopt health-related behaviors to mitigate the effects of chronic disease threats.

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Compliance with Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards Jinghua An, Shou-En Lu, Jean McDougall, Scott T. Walters, Yong Lin, Emily Heidt, Antoinette Stroup, Lisa Paddock, Sherry Grumet, Deborah Toppmeyer, and Anita Y. Kinney declare that they have no conflict of interest.

Authors' Contributions Jinghua An (Conceptualization [lead], Formal analysis [lead], Writing – original draft [lead], Writing – review & editing [lead]), Shou-En Lu (Formal analysis [equal], Supervision [equal], Writing – review & editing [equal]), Jean McDougall (Writing – review & editing [equal]), Scott T. Walters (Writing – review & editing [equal]), Yong Lin (Writing – review & editing [equal]), Emily Heidt (Data curation [equal], Project administration [lead], Writing – review & editing [equal]), Antoinette Stroup (Data curation [equal], Writing – review & editing [equal]), Lisa Paddock (Data curation [equal], Writing – review & editing [equal]), Sherry Grumet (Writing – review & editing [equal]), Deborah Toppmeyer (Writing – review & editing [equal]), and Anita Y. Kinney (Conceptualization [lead], Formal analysis [supporting], Funding acquisition [lead], Supervision [lead], Writing – original draft [equal], Writing – review & editing [equal])

Transparency Statements (1) Study registration: This study was preregistered at the NIH clinical trial registry (https://clinicaltrials.gov/ct2/show/NCT03326713). (2) Analytic plan preregistration: The analysis plan was not formally preregistered. (3) Analytic code availability: Analytic code from this study is not available in a public archive but will be made available by emailing the corresponding author. (4) Materials availability: Materials used to conduct the study are available online as supplementary materials in A. Kinney et al. Contemp. Clin. Trials. 2018, 73:123–135.

Data Availability

Deidentified data from this study are not available in a public archive but can be made available by emailing the corresponding author.

Supplementary Material

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

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