

# Modifying Attributions of Colorectal Cancer Risk

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## Abstract

We report how a four-group risk communication intervention targeted to individuals in the carpentry trade affected their perceived causes (*i.e.*, attributions) for increased colorectal cancer (CRC) risk. The intervention varied the amount of information presented on CRC risk factors and whether participants received tailored feedback on their risk factors. In baseline and 3-month follow-up telephone surveys, carpenters ( $N = 860$ ) reported their perceived absolute and comparative CRC risks, perceived causes for increased CRC risk, and knowledge of CRC risk factors. At follow-up, neither the type or amount of information

provided, nor the use of tailoring, appreciably and consistently affected whether participants mentioned their specific risk factor (*e.g.*, lifestyle, occupational) emphasized in their intervention information. Furthermore, attributions did not affect CRC risk perceptions. These results suggest that participants do not integrate sufficiently CRC risk factor information into their conceptualizations of CRC risk, and that more effective methods are needed to contextualize risk factors information to achieve the goal of modifying CRC risk perceptions. (Cancer Epidemiol Biomarkers Prev 2004;13(4):560–566)

## Attributions of Colorectal Cancer Risk among Individuals in the Carpentry Trade

Several studies show that the effects of perceived colorectal cancer (CRC) risk on motivating people to screen vary by screening modality (1). The lack of consistent findings between risk perceptions and CRC screening behaviors may be due to people's risk attributions; that is, their perceived causes of CRC risk. A person's knowledge of having elevated CRC risk factors can increase his or her perceived CRC risk which, in turn, should motivate screening. In this study, we explicitly tested whether CRC risk attributions among members of an occupational group (carpenters) were influenced by tailoring and imparting knowledge of CRC risk factors. We also examined how attributing CRC risk to different causes (*e.g.*, genetic, physiological) affected CRC risk perceptions.

**Relationships between Risk Attributions and Format of Delivering Risk Factor Information.** What methods of communicating CRC risk factors can affect changes in CRC risk attributions? We hypothesized that enhancing the personal relevance of messages about CRC risk factors would increase the amount of information attended to, retained, and subsequently used in attributions of CRC risk to affect perceived CRC risk (2, 3). To enhance relevancy, we manipulated whether participants received tailored CRC risk factor information. Tailoring is "any combination of information or change strategies intended to reach one specific person

and based on characteristics that are unique to that person, related to the outcome of interest, and have been derived from an individual assessment" (4, 5). Because tailored information is customized to individuals' risk factors, it can enhance personal relevancy and overcome people's tendency to deny that CRC risk factor information applies to them.

In addition, people must be knowledgeable of a risk factor before believing it can affect their risk. Hence, it is important to test whether increasing CRC risk factor knowledge can influence risk attributions. Therefore, we varied in the intervention the amount and type (*e.g.*, lifestyle factors) of CRC risk factor information. In theory, individuals informed about a larger number of risk factors should be more likely to mention these factors as attributions than individuals informed of fewer risk factors; this should be especially true if the messages are tailored.

**Relationships between Types of CRC Risk Attributions and Perceptions of CRC Risk.** To what extent are CRC risk perceptions affected by attributions to different sources (*e.g.*, genetic, environmental)? To address this question, we used Weinstein's method to categorize risk attributions into the following areas: (a) personal actions (*e.g.*, diet, exercise, smoking, alcohol use, getting check-ups); (b) heredity (*e.g.*, family history of CRC or other cancers); (c) physiological (*e.g.*, age, current perceived health, having polyps, health problems); (d) environmental (*e.g.*, occupational exposures, pesticides); (e) psychological attributes (*e.g.*, stress, being an optimist/pessimist); (f) chance; (g) other; and (h) don't know (6). Because genetic (*e.g.*, family history) and physiological causes (having polyps, perceptions of poor health) have been associated with above-average CRC risk perceptions (7, 8), we expected participants who mentioned heredity and physiological CRC causes would report

Received 11/11/02; revised 12/1/03; accepted 12/11/03.

Grant support: National Cancer Institute grants CA63782 and CA72099.

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higher perceived CRC risk. Furthermore, consistent with the extant literature showing greater perceived control is related to lower perceived risk (9), we expected participants who mentioned causes seen as more controllable (e.g., personal actions) would report lower perceived CRC risk.

**Linking Intervention Design with Study Hypotheses.** The intervention reported here varied two dimensions of risk factor information: (a) type of risk factor—one set of interventions emphasized only three basic CRC risk factors (age, family history, and polyps—termed basic information), whereas the other set emphasized a more comprehensive set of risk factors including lifestyle and occupational factors—termed comprehensive information); and (b) tailoring of risk factor information (no/yes; see Table 1).

Data are reported from baseline and 3-month post-baseline follow-up telephone surveys assessing CRC risk increasing attributions, knowledge of CRC risk factors, and perceived CRC risks. We tested explicitly the following predictions:

**H1:** Individuals who received tailored risk factor information will be more likely than those who received non-tailored risk information to attribute their CRC risk to the specific risk factors mentioned in their intervention materials (tailored basic + tailored comprehensive > non-tailored basic + non-tailored comprehensive).

**H2:** Individuals with correct rather incorrect knowledge about CRC risk factors will be more likely to mention these risk factors as personal risk attributions (comprehensive tailored + non-tailored > non-tailored and tailored basic). As a corollary, because individuals who received comprehensive information were expected to have greater knowledge about CRC risk factors than those who received basic risk factors information only, we hypothesized the former groups will be more likely to mention occupational and personal action causes (comprehensive tailored + non-tailored > non-tailored and tailored basic).

**H3:** Higher perceived CRC risk will be associated more strongly among individuals who mention hereditary and physiology factors than personal action causes (e.g., smoking, drinking; 42).

## Methods

**Participants and Eligibility Criteria.** Study participants were identified and recruited from a sampling

**Table 1. Study design**

Risk factor domain	Not tailored; print only	Tailored; print plus phone
Basic risk factors	Non-tailored basic	Tailored basic
Basic, lifestyle, and occupational	Non-tailored comprehensive	Tailored comprehensive

Note: Basic risk factors consisted of age, family history, and polyps. Lifestyle risk factors consisted of consumption of fruits and vegetables, meat consumption, exercise, smoking, and alcohol use. Occupational risk factors consisted of exposure to wood dust, solvents, and asbestos.

frame of 4292 employed and retired carpenters receiving health care benefits through the New Jersey Carpenters Funds from 1996 to 1998. Eligible participants were men and women in the carpentry trade who: (a) were between ages 50 and 75; (b) were not currently in treatment or awaiting any cancer treatment; (c) had no history of CRC; (d) had never had a sigmoidoscopy or colonoscopy as part of routine screening;<sup>3</sup> and (e) had not had a fecal occult blood test (FOBT) within 15 months before baseline. Before the baseline interview (discussed below), we mailed letters including a consent form, study description, and notification of an upcoming telephone call by a professional survey firm.

**Baseline and 3-Month Surveys.** Interviewers telephoned members of the Carpenters' Fund 1–2 weeks after they received the pre-notification letter. Those who agreed to participate and met eligibility criteria completed a 25-min baseline and 3-month post-baseline follow-up telephone survey. Measures included the following:

**Perceptions of Absolute Risk.** These were assessed via verbal and numerical response formats. We used both because there is no "gold standard" for assessing perceived risks (10). Using verbal anchors, participants were asked what they thought was their chance of getting CRC in their lifetime. Response options were no chance, very unlikely, unlikely, likely, very likely, and certain to happen (scored 1–6). Participants then used numerical anchors to answer the same question on a 0% to 100% point scale, where 0% = no chance and 100% = certain to happen.

**Perceptions of Comparative Risk.** We asked participants to compare their lifetime risks to other people of their same sex, age, and race. Responses were much below average, below average, same average risk as a woman/man your age and race, above average, and much above average (scored 1–5, respectively).

**Attributions of CRC Risk.** As an open-ended question, participants were asked, "Overall, what do you think puts you at higher risk for getting CRC? Answer this question as it pertains to you only." We initially categorized responses using the scheme developed by Weinstein (6) and described previously and subsequently expanded by including occupational risk factors—classification scheme is available from the first author on request. Responses were coded independently by two project members (I.L. and S.G.); discrepancies were discussed with the larger group of investigators until consensus was reached.

**Knowledge of CRC Risk Factors.** We asked participants whether the following risk factors increased, decreased, or did not affect CRC risk: (a) being  $\geq 50$ ; (b) having a family history of CRC; (c) having a polyp; (d) stress (filler item); (e) eating more than three servings of red meat a week; (f) engaging in at least 30 min of moderate/heavy physical activity most days of the week (examples of moderate and heavy physical activity were provided); (g)

<sup>3</sup>Because results of a screening sigmoidoscopy or colonoscopy do have implications for affecting CRC risk (i.e., findings of no polyps, removal of polyps), and our intervention most powerfully aimed at affecting risk via our intervention messages rather than external influences, we did not include individuals who had screening sigmoidoscopy or colonoscopy.

eating less than five servings of fruits and vegetables a day; (h) smoking; (i) drinking more than one serving of alcohol each day; (j) being exposed to asbestos for several years; (k) being exposed to solvents (e.g., paint thinner) for several years; and (l) being exposed to wood dust for several years. A correct answer was scored as 1; incorrect responses and missing data were scored 0. Knowledge of risk factors was assessed after risk perceptions and risk attributions.

**Basic, Lifestyle, and Occupational Risk Factors.** For basic risk factors, in addition to specifying age, participants were asked if they had one or more first-degree relatives ever diagnosed with CRC (no/yes) and whether they have ever been told they had one or more polyps (no/yes, description of polyp provided).

For lifestyle risk factors, participants were asked: current smoking status (no, yes); number of servings of alcohol on average per day (a serving defined as a can or bottle of beer, a 4-oz. glass of wine, or one cocktail containing 1 oz. of liquor); whether, on average they got at least 30 min or more of moderate physical activity most days of the week, including work-related physical activity (no/yes; examples, taking brisk walks, swimming, cycling, home care such as general cleaning),<sup>4</sup> 11 and daily vegetable and fruit consumption. The latter was assessed by a seven-item food frequency index designed after a similar measure used by the Centers for Disease Control in their Behavioral Risk Factors Surveillance System and validated subsequently by Serdula *et al.* (11). Participants were asked how frequently (*i.e.*, number of times per day, week, or month) they ate or drank the following: 100% orange or grapefruit juice; other 100% fruit juice, green salad; french fries or fried potatoes; baked, broiled, or mashed potatoes; vegetables other than salad or potatoes; and fruit, not counting juice. After omitting the item pertaining to french fries and fried potatoes, the remaining items were summed to create a measure of servings per day ( $\alpha = 0.48$ , 12).

For occupational risk factors, participants were asked if they were ever exposed to organic, petroleum, or coal-based solvents, wood dust, and asbestos (no/yes). If yes, they were asked what year they were first and last exposed and average frequency of exposure during the entire time of exposure (1 = exposed less than one time per month to 4 = exposed more than three times per week). Those exposed to asbestos were asked if they "ever received a physician's diagnosis of asbestosis or asbestos-related pleural changes based on a chest X-ray?" (no/yes)—a yes response indicates a substantial lifetime exposure to asbestos. Risk factors were assessed after risk perceptions and risk attributions.

**Intervention Groups.** After completing the baseline survey, participants were stratified into two age groups, 50–64 and  $\geq 65$ . Participants in each strata were randomly assigned to one of four intervention groups that varied on risk factor information type and whether risk factors were tailored. Intervention occurred approximately 2 weeks after the baseline interview.

**The Non-Tailored Basic Information Group ( $n = 216$ ).** This group received a four-page brochure discussing three basic CRC risk factors—age, family history, and polyps. The brochure also included basic information regarding function of the colon and rectum, average lifetime risk of getting CRC (*i.e.*, 6% or 1 in 18), CRC being the second leading cause of cancer death in the U.S., and information on three forms of screening (FOBT, sigmoidoscopy, and colonoscopy).

**The Non-Tailored Comprehensive Information Group ( $n = 212$ ).** This group was identical to the non-tailored basic information group in all respects except one: in addition to receiving information on the "basic" (age, family history, and polyps) risk factors, they also received information on lifestyle (*i.e.*, diet, exercise, smoking, alcohol use) and occupational risk factors (*i.e.*, exposure to asbestos, wood dust, solvents).

**The Tailored Basic Information Group ( $n = 218$ ).** This group differed from the non-tailored basic group in two respects. First, in addition to discussions of the three basic risk factors, their printed materials also included a tailored section highlighting which of these three risk factors increased *their personal* CRC risk. For example, a 56-year-old man who had never been checked for polyps was informed that being 56 and the possibility of having undetected polyp(s), due to lack of screening, elevated his CRC risk.

Second, approximately 2 weeks after receiving the print materials, participants received a 5- to 10-min telephone counseling call from a trained counselor at the Duke Risk Communication Laboratory. The counselor discussed the three "basic" risk factors, which of these risk factors were relevant to the participant's personal CRC risk, and the importance of getting screened. The counselor addressed any questions. Ninety-four percent of participants were counseled.

**The Tailored Comprehensive Information Group ( $n = 214$ ).** This group was identical to the non-tailored comprehensive group in all respects except two: In addition to print information on basic, lifestyle, and occupational risk factors, their materials included a tailored section highlighting which risk factors possibly increased their CRC risk (see below). Second, the telephone counselor reviewed the same information as the tailored basic group, with additional discussions concerning how lifestyle and occupational risks affected their CRC risk.

Participants received a tailored message about lifestyle risk factors if they: (a) ate less than five servings of fruits or vegetables per day, (b) ate more than three servings of red meat a week, (c) smoked, (d) had more than one alcoholic drink per day, and/or (e) engaged in less than 30 min of moderate to intense exercise every day most days of the week. For occupational risk factors, they received a tailored message informing them of possible elevated risk if they had been exposed to: (a) asbestos for 5 or more years while in the carpentry trade and continued to be exposed at least once a month; (b) wood dusts and/or solvents for more than 10 years. Those diagnosed with asbestos-related disease by chest X-ray were informed of having increased CRC risk. Average number of tailored messages received in this group was 5.8 (SD = 1.4). Ninety-two percent of participants were counseled.

<sup>4</sup>We assessed both moderate and heavy levels of physical activity. Because results pertaining to heavy level of physical activity replicated the results with moderate activity, we only report results with moderate levels of activity.

**Statistical Methods.** H1 and H2 were first analyzed using  $\chi^2$  tests of association between risk attributions and receipt of tailoring (no/yes) and knowledge (correct yes/no) based on a priori contrasts as specified in the hypotheses. Significant associations were then reanalyzed controlling for baseline attributions and knowledge, as appropriate, using logistic regression analyses. The effects of attributions on risk perceptions were assessed via ANCOVA adjusting for baseline risk values. Due to the number of tests performed, a result was deemed significant at  $P < 0.01$ . In no cases did whether the risk factor was tailored (no/yes) interact significantly with whether the information was basic or comprehensive to affect risk attributions.

Using standard approaches to power calculations (13), the power for testing hypotheses 1 and 2 was estimated using methods for  $2 \times 2$  tables. For example, in Table 2, one dimension of the  $2 \times 2$  table is whether or not the participant received tailored feedback on the specific risk factor (*i.e.*, the predictor); the other dimension is whether or not the subject mentioned the specific risk factor. The power depends on the effective sample size, which in turn depends on the distribution of the predictor variable. When this distribution was relatively balanced (*e.g.*, 20–80% of subjects received tailored feedback), at an  $\alpha$  of 0.01, we had approximately 80% power to detect a small-to-moderate difference in the response proportions (effect size = 0.3; *e.g.*, 35% of those receiving tailored feedback mentioning the risk factor *versus* 20% of those not receiving tailored feedback mentioning the risk factor). When this distribution was unbalanced, we had 80% power to detect a moderate-to-large difference in proportions (effect size = 0.7). The power for testing

hypothesis 3 was estimated using methods for the analysis of covariance. We had 80% power to detect predictor variables (attributions) that explained 2–3% of the variation in the (continuous) response pertaining to perceived risks.

## Results

**Recruitment Outcomes and Final Sample Characteristics.** Eight hundred sixty and 704 eligible participants completed baseline and 3-month follow-up surveys, respectively. Reasons for nonparticipation at baseline and in the follow-up survey are available from the first author on request. Because we were not able to collect any demographic or other information on those we could not reach, refused, or were deemed ineligible, we were unable to make meaningful comparisons on the representativeness of the final study sample at baseline in relation to the total sample.

Participants' baseline and 3-month follow-up demographic and occupational information is as follows. Mean age at baseline was 59.6 (SD = 7.6) and 60.0 (SD = 7.6) at follow-up. The sample was 97% male at both time points. At baseline, racial composition was 95% White and 2.7% African-American; at follow-up, it was 95% White and 1.7% African-American. At baseline and follow-up, 11% reported education less than high school, 38% were high school graduates, 44% had some college, and 7% were college graduates. Mean years in the carpentry trade was 36 at both baseline and follow-up. The proportion of retirees was 41% at baseline and 44% at follow-up. Those who did and did not complete follow-up did not differ in race, education, or years in the carpentry

**Table 2. Proportion of participants who mentioned specific CRC risk factors as attributions at follow-up as a function of whether they received tailored feedback**

Risk factor	% of sample that received tailored feedback on specific risk factor	% who got tailored feedback and mentioned risk factor	% who did not get tailored feedback and mentioned risk factor
<b>Basic risk factors</b>			
Age	49.0	5.2	3.9
Family history	3.1	36.4	2.4 <sup>b</sup>
Polyps	49.0	0.6	0.3
<b>Lifestyle risk factors</b>			
Meat	14.5	5.9	3.0
Vegetables and fruits	18.8	1.5	1.0
Diet (meat)	14.5	20.6	18.8
Diet (vegetable and fruits)	18.8	21.2	18.5
Exercise	1.8	15.4	1.2 <sup>b</sup>
Smoking	3.4	41.7	4.7 <sup>a</sup>
Alcohol use	10.6	5.3	3.2
<b>Occupational risk factors</b>			
Solvents	13.4	1.1	0.0 <sup>a</sup>
Wood dust	21.9	1.3	1.3
Asbestos	3.0	4.8	0.6 <sup>a</sup>
Occupation (solvents)	13.4	21.3	8.8 <sup>b</sup>
Occupation (wood dust)	21.9	18.2	8.4 <sup>b</sup>
Occupation (asbestos)	3.0	14.3	10.4
Occupation (X-ray)	1.1	0.0	0.7

Note:  $N = 704$ . The middle and third columns represent the percentage of participants who mentioned a risk factor as a function of getting tailored information for that specific risk factor. For example, among the 49% who received a tailored information on age, 5.2% mentioned age as a risk attribution while among those who did not get tailored feedback on age, 3.9% mentioned age as a risk attribution. For the attributional domains of diet and occupation, the risk factors in parentheses represent the extent to which that risk factor was related to mentioning the category of diet or occupation.

<sup>a</sup> $P < 0.05$ .

<sup>b</sup> $P < 0.001$ .

trade; perceived absolute and comparative risk; or risk attributions. Those who did not complete the follow-up were significantly younger than those who completed follow-up ( $M = 56.8$  versus  $M = 59.5$ ,  $t_{(858)} = 3.1$ ,  $P < 0.002$ ) and hence less likely to be retired (32% versus 44%,  $P < 0.01$ ). Among respondents, the CRC risk factor profiles did not differ between intervention groups at baseline or at follow-up.

**Effects of Tailored Intervention on Specific CRC Risk Attributions.** According to the first hypothesis, individuals who received tailored information on specific risk factors should be more likely than participants who did not receive tailored information to mention these factors at follow-up. For example, those who received non-tailored information on smoking should be less likely than those who received a tailored message regarding their *own smoking* to mention smoking as a risk-increasing factor at follow-up. We examined whether the following specific risk factors emphasized in the intervention were mentioned as attributions at follow-up: older age; family history of CRC; possibility of having polyps or recurrence of polyps; alcohol intake; smoking; eating fruits or vegetables and meat; diet in general; exercise; being exposed to wood dust, solvents, and asbestos; and occupation more generally. Table 2 presents percentages of participants mentioning each specific risk factor as an attribution by whether they received a tailored message on that specific risk.

Compared to those in the non-tailored groups, those who received tailored information were more likely to attribute risk to family history (2.4% versus 36.4%,  $P < 0.001$ ), exercise (1.2% versus 15.4%,  $P < 0.001$ ), and smoking (4.7% versus 41.7%,  $P < 0.001$ )—exposures to solvents (0% versus 1.1%) and asbestos (0.6% versus 4.8%) were significant at the conventional 0.05  $\alpha$  level. Furthermore, those who received tailored messages about solvents and wood dust (tailored comprehensive group) were more likely to attribute some occupational factor as contributing to their higher CRC risk than those who did not receive tailored messages. These results were not substantially altered in reanalyses controlling for whether the participant mentioned the specific risk attribution at baseline. Thus, there was partial support for H1.

**Effects of Knowledge on Attributions for Specific CRC Risk Factors.** According to the second hypothesis, individuals with knowledge about CRC risk factors should be more likely to mention these as risk factors than individuals who lack knowledge on CRC risk factors. We first examined whether being correct or incorrect about a specific risk factor at follow-up was associated with attributing personal risk to that factor (see Table 3).

Overall, the prediction was supported for only four risk factors: smoking, alcohol use, meat consumption and exposure to solvent as contributors to diet and occupation as personal risk factors, respectively. Follow-up results were not significantly altered controlling for baseline knowledge for a specific risk factor and whether the specific risk factor was mentioned at baseline as an attribution (data not shown).

In general, individuals who received comprehensive information about CRC risk factors were not more likely to mention lifestyle and occupational risk factors than individuals who received only basic information about

age, family history, and polyps. These results occurred despite improvement in knowledge of CRC risk factors overall and as a function of intervention group. On the basis of McNemar's tests, improvement in knowledge from baseline occurred for several specific risk factors: polyps (12.8% improvement,  $P < 0.001$ ), exercise (18.9% improvement,  $P$ s  $< 0.001$ ), smoking (17.3% improvement,  $P < 0.001$ ), and all occupational risk factors (19.2%, 22.7%, and 21.6% improvement for asbestos, wood dust, and solvents, respectively,  $P < 0.001$ ). In addition, the greatest overall knowledge and knowledge of occupational risk factors occurred among participants who received comprehensive tailored information [ $(F(1,699) = 16.2$ ,  $P < 0.0001$ , data not shown)].

**Effects of Risk Attributions on Perceived CRC Risk Perceptions.** According to the third hypothesis, we expected participants who at follow-up mentioned genetic or physiological attributions would report greater perceived CRC absolute and comparative risk than individuals who did not mention any genetic or physiological causes. We further expected those who mentioned personal causes would report lower perceived CRC absolute and comparative risk than individuals who did not mention personal action causes.<sup>5</sup> These predictions were not supported. Rather, lower perceived absolute numerical risk was found among those who reported *not having* any risk factor (8.4% of sample) than among those who mentioned having at least one risk factor [ $M = 22.1$  versus  $M = 33.6$ ;  $F(1,487) = 13.99$ ,  $P < 0.001$ ]. Furthermore, individuals who responded to the attribution question by stating they were not at higher risk (7.0% of sample) reported greater perceived comparative risk than those who never made such a statement [ $M = 3.9$  versus  $M = 3.3$ ;  $F(1,667) = 10.8$ ,  $P < 0.002$ ]. No other effects were found.<sup>6</sup>

## Discussion

We assessed how formats of varying intensities of communicating CRC risk factors to members of a higher risk occupational group affected their risk attributions and, ultimately, whether these attributions affected perceived CRC risks. There are two "take home" messages. First, tailoring and varying the type and amount of CRC risk factors information did not appreciably and consistently affect risk attributions; nor did the attributions significantly affect risk perceptions. Second, because of these primarily null findings, it may be premature to

<sup>5</sup>At follow-up, 30.3%, 10.5%, 8.7%, 7.9%, 3.8%, and 0.8% mentioned personal action, occupational, physiological, genetic, environmental, and psychological causes, respectively. 8.4% and 7.0% mentioned that they had no factor that could elevate their risk or stated explicitly they were not at higher risk, respectively. 27.9% mentioned they did not know of a risk factor that could elevate their CRC risk.

<sup>6</sup>We assessed if overall knowledge at follow-up was related to perceptions of absolute and comparative risks at follow-up. Overall, having greater overall knowledge was associated with greater perceived absolute numerical risk ( $r = 0.17$ ,  $P < 0.0001$ ) but was unrelated to verbal absolute risk ( $r = 0.00$ ) or comparative risk ( $r = -0.04$ ). We also examined whether the total number of tailored risk factor messages a participant received affected perceptions of absolute and comparative risks at follow-up. Overall, participants who received more tailored risk factors reported greater absolute numerical risk ( $r = 0.16$ ,  $P < 0.0001$ ), but was unrelated to absolute verbal risk ( $r = -0.02$ ) or comparative risk ( $r = 0.00$ ).

design interventions that try to affect risk perception via CRC risk attributions without a more comprehensive understanding of these processes.

With respect to our first hypothesis, the receipt of tailored feedback had weak effects on participants' attributing CRC risk to the *specific* risk factors mentioned in their interventions. Of the 11 risk factors featured in the intervention, receiving tailored feedback increased the likelihood of mentioning only 3. The latter results are somewhat promising but need to be tempered by findings that, even among those who received tailored risk factor information, few mentioned a *specific risk factor* highlighted in their intervention as an attribution. For example, although 49% of the sample received tailored feedback on age, only 5% mentioned age as a risk factor. Knowledge of CRC risk factors was similarly weakly related with attributions of risk, independent of the tailoring and despite general improvements in knowledge.

There were very few notable changes from baseline to follow-up in perceptions of absolute and comparative risk as a function of attributional domain. We found lower perceived absolute numerical risk among those who reported *not having* any risk factor relative to those who mentioned having at least one risk factor. While the proportion of individuals who mentioned not having anything that could elevate their risk was small (8%), such attributions may ultimately result in poor screening rates. In exploratory analyses, we ruled out the possibility that differences reflected poor knowledge of risk factors. An alternative possibility is that individuals who report having no risk factors may feel that if they have not had any symptoms of CRC by their age, they are unlikely to get CRC—prompting the perception of not having any risk factors. Alternatively, they may believe

they have risk factors, but their ability to appreciably affect CRC risk is so low as to essentially remove the risk factors as deserving of attention. Whatever the reason, more research is needed to understand what factors affect risk attributions, especially among those who claim to have none.

What are the practical implications of these findings? Three themes emerge. First, greater efforts are needed to understand how people form and use attributions of CRC risk and how attributions affect risk perceptions and ultimately behavior change. For example, how do people weigh risk factors for CRC risk? Are risk attributions and resulting risk perceptions affected more by the *ease* with which individuals can retrieve risk factors from memory or by the total number of risk factors they retrieved from memory (14, 15)?

Second, greater efforts are needed to help individuals integrate CRC risk factor information into their personal risk attributions. Contextualizing risk factor information may help; recipients could be given social comparison information about their risk factor standing (*i.e.*, how their risk factors and hence overall risk compare to others). The importance of social comparison information stems from the fact that people need a benchmark to evaluate the meaning and hence potential threat of CRC risk factor information, and is predicated on findings that social comparisons powerfully affect risk beliefs and perceptions (16). If people learn they have more risk factors than others, this may increase the saliency of these risk factors and hence risk perceptions.

Third, if future interventions are designed to affect CRC risk attributions in order to affect risk perceptions, which attributional domain(s) (*e.g.*, physiological, genetic) might be most effective for doing so? Our results show

**Table 3. Proportion of participants who mentioned at follow-up specific risk factors in their attributions of risk as a function of correctly or incorrectly knowing how a risk factor is related to CRC risk**

Risk factor	% who knew correctly how risk factor was related to CRC risk	% who had correct knowledge and mentioned risk factor	% who had incorrect knowledge and mentioned risk factor
<b>Basic risk factors</b>			
Age	86.5	5.2	0.0 <sup>a</sup>
Family history	91.2	3.6	1.5
Polyps	90.3	0.3	1.6
<b>Lifestyle risk factors</b>			
Meat	60.5	4.7	1.4 <sup>a</sup>
Vegetables and fruits	36.9	1.9	0.7
Diet (meat)	60.5	23.2	12.6 <sup>b</sup>
Diet (vegetable and fruits)	36.9	18.1	19.6
Exercise	67.5	1.9	0.4
Smoking	72.9	8.0	0.5 <sup>b</sup>
Alcohol use	33.5	7.6	1.3 <sup>b</sup>
<b>Occupational risk factors</b>			
Solvents	63.4	0.2	0.0
Wood dust	50.4	2.2	0.3 <sup>a</sup>
Asbestos	68.5	0.9	0.4
Occupation (solvents)	63.4	13.7	3.6 <sup>b</sup>
Occupation (wood dust)	50.4	14.6	3.5 <sup>a</sup>
Occupation (asbestos)	68.5	5.2	0.0 <sup>a</sup>

Note:  $N = 704$ . The second and third columns represent percentages of participants who at follow-up mentioned the risk factor as an attribution based on their knowledge of the risk factor in relation to CRC risk. For example, among the 86% of participants who knew age increased CRC risk, 5.2% mentioned age as a risk attribution; among those with incorrect knowledge, 0% mentioned age as a risk attribution. For the attributional domains of diet and occupation, percentages represent how often these domains were mentioned as a function of having correct or incorrect knowledge between the risk factor mentioned in parentheses and CRC risk.

<sup>a</sup> $P < 0.05$ .

<sup>b</sup> $P < 0.001$ .

there is a need to target individuals who report having no risk factors. We expected personal action causes (*e.g.*, smoking, drinking, exercise, diet) would be related to perceived risk, but this was not supported. One possibility is that participants acknowledge these as risk factors common to several health outcomes, not just CRC. A possible consequence is that the magnitude by which these risk factors (*e.g.*, diet, exercise) are perceived to affect CRC risk specifically is downplayed, contrary to CRC specific risk factors (*e.g.*, family history). Although individuals who mentioned family history as a risk attribution did not report greater risk than those who did not mention family history, family history as a risk factor may more powerfully affect the emotional aspect of risk (*e.g.*, fear) than the cognitive dimension (estimation of probability).

There are several caveats to our findings. First, our results are limited primarily to white men in the carpentry trade. We selected individuals in the carpentry because they engage in detrimental lifestyle habits (smoke, poor diet) and have occupational exposures (solvents, wood dust, asbestos) that have been associated with CRC and gastrointestinal disorders (17–28); hence, as a group, they may benefit from interventions that highlight these risk factors via tailoring and education. Second, our ability to determine how powerfully our tailoring affected specific attributions was limited in that only a relative few participants received feedback pertaining to each risk factor. The exception to this was age and polyps. Future research could more powerfully assess these effects by targeting specific groups in which certain risk factors are more prevalent (*e.g.*, all smokers, those with a family history). This would also help determine how providing tailored information about a specific risk factor may affect risk attributions and risk perceptions. Third, we did not assess immediately after the intervention participants' reflections of which risk factors applied to them and whether they believed the risk factors information. This is critical to assess, especially among those who received tailored intervention. The reasons fewer participants mentioned the risk factors reported in their interventions may have been due to forgetting or not being persuaded by the information. Fourth, we used measures that resulted in questionable reliability in this population (*e.g.*, fruits and vegetables) as well as assessing several risk factors using single-item measures (*e.g.*, exercise). A potential consequence is that we may have underestimated the number of individuals who had elevated risk profiles. Nonetheless, our results suggest future efforts be devoted to improving knowledge of CRC risk factors and to increase our scant understanding about how CRC risk attributions are formed and used. Understanding these mechanisms may result in risk communication interventions that are more effective at promoting screening across various modalities.

### Acknowledgments

We thank the New Jersey Carpenter Fund for their help in providing access to their enrollees. We thank David Farrell, from People Design, for helping to create the intervention materials.

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