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Longitudinal Investigation of Behavior Change Across Multiple Cancer Risk

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LONGITUDINAL INVESTIGATION OF BEHAVIOR
CHANGE ACROSS MULTIPLE CANCER RISK

BEHAVIORS

BY

HUI-QING YIN

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE
REQUIREMENTS FOR THE DEGREE OF

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ABSTRACT

There is accumulated evidence to support the efficacy of population-based behavioral interventions, however, our understanding of how and why effective interventions promote behavior change is still lacking. The goal of these two studies was to investigate mechanisms of single and multiple behavior change with a focus on cancer risk behaviors, so as to further our understanding of how effective behavioral interventions can promote successful behavior change; improving public health while reducing healthcare costs.

These studies pooled primary data from three large population-based randomized intervention trials that included important cancer-related risk behaviors, including smoking, unhealthy eating, and sun exposure. A total of $N=9522$ adults across the three samples reported at least one baseline behavioral risk, and were assessed at baseline, 12- and 24-months. Two alternative latent variable modeling techniques were applied to examine behavior change within and jointly across the three cancer risk behaviors.

Latent growth curve (LGC) modeling approaches were employed in the first study to systematically examine 2-year growth trajectories of observed behavioral outcomes within each risk behavior individually and jointly across pairs of co-occurring behavioral risks. Smoking behavior decreased over time across all participants, with treatment predicting a slightly steeper decrease in the number of cigarettes smoked. Conditional LGC models also supported significant intervention effects on increasing healthy eating and sun protection behaviors over time. Parallel-

process LGC models revealed that growth trajectories were associated across behaviors within pairs of co-occurring risks.

The second study applied latent transition analysis techniques to examine transitions through the discrete stages for changing individual cancer-related risk behaviors and to compare stage transition patterns across risk behaviors. Stage transition models supported the stability, progression and regression in behavioral stages over time across all three cancer risks. Conditional stage transition models also provided evidence for intervention efficacy for all three behaviors, in terms of moving at-risk participants to reach behavioral criteria, promoting stage progress among those who did not reach criteria, and in maintaining successful behavior change during the follow-up interval. In addition, findings from the second study revealed the stability of precontemplation stage membership across all three behaviors; stage progress from the precontemplation stage was even less likely among control participants.

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CHAPTER 1

INTRODUCTION

Healthcare costs in the U.S. have increased dramatically over the last two decades. In 2012, \$2.8 trillion was spent on healthcare, or about \$8,915 per person, and approximately 17.9% of GDP (Centers for Medicare and Medicaid Services [CMS], 2014). NIH estimated the total annual costs of cancer were \$201.5 billion in 2008, including \$77.4 billion in health expenditures and \$124 billion in lost productivity due to premature death (American Cancer Society [ACS], 2013). Close to 75% of U.S. annual health care costs were expended on preventable chronic diseases, including cancer, cardiovascular disease, and diabetes (Fisher et al, 2011). The primary risks for these diseases are common modifiable health risk behaviors. Cancer, cardiovascular disease and diabetes are strongly linked to four health risk behaviors: tobacco use, unhealthy eating, physical inactivity and alcohol use (Fisher et al, 2011). Improving health behaviors play a central role in disease prevention and health promotion efforts, and effective health behavior change interventions can help to prevent many diseases, promote well-being and reduce healthcare costs.

Behavioral interventions have primary and secondary prevention effects on both physiological and psychological health and well-being (Fisher et al, 2011; Krebs, Prochaska & Rossi, 2010; Kreuter, Stretcher & Glassman, 1999; Noar, Benac & Harris, 2007). Tailored interventions based on the Transtheoretical Model (TTM) have been developed for more than 20 different health behaviors (Prochaska, Redding &

Evers, 2008), and demonstrated efficacy in a series of clinical trials (e.g., Prochaska, DiClemente, Velicer & Rossi, 1993; Prochaska, Velicer, Fava, Rossi & Tsoh, 2001a; Prochaska et al, 2001b; Velicer, Prochaska, Fava, Laforge & Rossi, 1999; Velicer et al, 2006a; Velicer, Prochaska & Redding, 2006b). More recently, TTM-based computer tailored interventions (CTI) have been applied to changing multiple behaviors. Interventions have simultaneously and sequentially targeted multiple behaviors to prevent cancer and other chronic diseases, and have produced significant impacts (Blissmer et al, 2010; Johnson et al, 2008; Prochaska et al, 2004, 2005, 2008, 2011, 2012; Velicer et al, 2004).

Although tailored communications are effective, their efficacy can still be improved. For example, the TTM-tailored interventions for smoking cessation have consistently produced 22 to 25% point prevalence abstinence at long term follow-up (Prochaska et al, 1993, 2001a, 2001b; Velicer et al, 1999, 2006a, 2006b). While these were good results, this also means that almost 75% of treated smokers did not successfully quit. In addition, CTIs targeting risky sun exposure and unhealthy diet behavior have demonstrated efficacy: the proportion of treated participants who had taken effective action at long term follow-up was about 23 to 31% for adopting sun protective behaviors, or 29 to 34% who reached behavioral criteria for reduced dietary fat intake (Prochaska et al., 2004, 2005; Weinstock, Rossi, Redding & Maddock, 2002). Given that close to 70% of treated at-risk participants had not successfully reduced their behavioral risks for sun exposure or unhealthy eating, there is similar potential and need to improve on the efficacy of these interventions. Empirically based

enhancement of these benchmark programs represents a major challenge and an opportunity in tailored health communications research.

One of the most significant barriers to enhancing the efficiency and effectiveness of behavioral interventions is insufficient knowledge about the underlying mechanisms of behavior change. Many intervention trials have targeted a wide variety of behaviors, with numerous studies showing intervention efficacy. We have accumulated convincing evidence that we can change behaviors. However, there are gaps in our understanding of how and why behaviors changed. The mechanisms of behavior change have basically been regarded as a black box containing unknown processes. In-depth understanding of these mechanisms would inform and enhance behavioral intervention design. Conducting empirical research using a variety of new analytical methods to target this 'black box' and elucidate its contents is critical to advancing the development of the next generation of interventions. Behavioral and health science strongly needs studies that generate more evidence and build the knowledge base. Such studies must have an emphasis on comparing longitudinal models and results across different and multiple behaviors in different populations.

There are several general analytic approaches that are well-suited to investigate the underlying mechanisms of behavior change, including: 1) latent growth curve modeling (LGCM) (MacCullum, Kim, Malarkey & Kiecolt-Glaser, 1997; McArdle & Epstein, 1987; Meredith & Tisak, 1990), and 2) latent transition analysis (LTA)/latent class analysis (LCA) (Collins & Lanza, 2010; Goodman, 1974). These approaches have the capability to analyze multiple latent variables in longitudinal research designs. They also have the potential to extend to multiple group designs that

investigate model invariance across different populations. These analytical capabilities are essential for examining underlying mechanisms.

A number of studies have employed LGCM or LCA/LTA to model the complex trajectories and/or mechanisms of behavior change (e.g. Adams et al, 2009; Brick, 2015; Brick, Babbitt & Velicer, 2014; deRuiter, Cairney, Leatherdale & Faulkner, 2014; Evers, Harlow, Redding & LaForge, 1998; Kobayashi, Yin, Redding & Rossi, 2014; Lanza & Collins, 2008; Lanza, Collins, Lemmon & Schafer, 2007; Lanza, Patrick & Maggs, 2010; Richert, Schüz & Schüz, 2013; Roesch et al, 2009; Schumann, John, Rumpf, Hapke & Meyer, 2006; Yin, Rossi, Kobayashi & Redding, 2014a). For example, Martin and colleagues (1996) examined longitudinal stage transitions for smoking cessation over a six month interval using data for 545 current and former smokers. Their best-fitting model suggested progression and regression between adjacent stages as well as two-stage progression. They concluded that movement through the stages was not always linear, that forward movement was more likely to occur than backward movement, and that over the six month interval, moving to adjacent stages was more likely to occur than two-stage progression. In another study, Roesch and colleagues (2009) used latent growth curve modeling to evaluate 12-month growth trajectories of adolescent physical activity, and found that increases in physical activity over time were significantly associated with increases in several psychosocial variables, including self-efficacy, family and peer support and behavior change strategies. More recently, Kobayashi (2012) demonstrated the application of LCA to simultaneously analyze more than two behavioral outcomes and identified two latent subgroups for stage of change progression for three behaviors. Although these

studies provided useful suggestions about the mechanisms of behavior change, the amount of accumulated knowledge is still too limited to develop a general description of change mechanisms.

The current research focused on both LGCM and LTA to model behavior change over time in three cancer-related risk behaviors of smoking, unhealthy eating, and sun exposure. Study 1 applied LGCM to model latent characteristics of trajectories for behavioral outcomes, and also investigated possible predictors of change across the different behaviors. Because multiple potential mediators can be added to LGC models as simple time-invariant covariates/predictors, these can serve as useful exploratory modeling approaches. The proposed analyses used LGCM to examine some of the mechanisms underlying change in health behaviors over time. In Study 2, LTA was employed to model latent characteristics of transitions across discrete behavioral stages, and to investigate the intervention effect on stage transitions over time. The stage transition models were then compared across three different health risk behaviors under the TTM framework.

The proposed research has the potential to advance the science of health behavior change from multiple perspectives. First, smoking cessation, healthy eating and sun protection are particularly important behaviors in order to prevent cancer. Second, this study investigated the mechanisms of behavior change focusing on both single behavior and multiple behavior paradigms. Third, LGCM and LTA are among the more flexible analytical approaches that can be applied to investigating change over time within and across behaviors. Fourth, conducting integrative data analyses using pooled, large-scale, datasets with increased heterogeneity in the samples could

yield more comprehensive knowledge and will result in greater generalizability of study findings. Fifth, since all data were from population-based randomized trials, the data included large proportions of individuals who had no intention to change their behavior. This study characteristic improves the generalizability of findings to large populations.

This is one of the first studies to systematically examine and compare latent growth trajectories and latent stage transitions jointly across three of the most important behaviors for cancer prevention. These research findings can help produce the empirical foundation for even more effective, low cost, tailored interventions for multiple health behaviors and demonstrate the potential that LGCM and LTA have as alternative analytical approaches for examining behavior change and advancing cancer prevention.

The aim of the current research was to investigate mechanisms of single and multiple behavior change across three cancer-related risk behaviors of smoking, unhealthy eating, and sun exposure using two alternative latent variable modeling approaches. In Study 1, we employed LGC modeling approach to examine 2-year growth trajectories for quantitative behavioral outcomes within each individual risk behavior. We tested the effects of TTM-tailored intervention on the rate (slope) of behavior change, as previously reported outcomes include significant increases over time in treatment relative to controls on sun protection and diet behavior (Prochaska et al., 2004, 2005; Weinstock, Rossi, Redding & Maddock, 2002). We also modeled growth trajectories jointly within pairs of co-occurring risk behaviors to understand whether the trajectories were associated across behaviors in the pair. In Study 2, we

explored mechanisms of stage progression and behavior change across the three cancer risk behaviors. This study applied LTA techniques in order to: 1. Describe the pattern of stage transitions over two years for each of the three cancer-related risk behaviors; 2. Examine the effect of TTM-tailored intervention on stage transition probabilities for each behavior; and 3. Compare models of stage transition/progression across the three behaviors.

CHAPTER 2

METHOD

Secondary analyses were conducted using primary data pooled from three separate population-based randomized controlled trials conducted from 1995–2000 by the Cancer Prevention Research Center at the University of Rhode Island. Intervention design and outcomes for each of the three primary trials have been reported previously (Linnan et al., 2002; Prochaska et al., 2004; 2005; Velicer et al., 2004). All three randomized trials targeted smoking, unhealthy diet and sun exposure. All trials used common TTM-tailored interventions and no-treatment, assessment-only control groups. Participants in all three trials completed assessments at baseline, 12-, and 24-months follow-up. The main effects of stage of change on observed behavioral outcomes were estimated using available data from the baseline assessment, and compared across samples. Examination of the longitudinal changes in behaviors were conducted using all available data from the baseline, 12-, and 24-months assessments combined across intervention and control groups for all three trials.

Participants and Procedure

This study pooled data from three separate population-based intervention trials with adult participants comprising (a) one sample of parents of adolescents ($N = 2,460$), (b) one sample of patients from an insurance provider list ($N = 5,382$), and (c) worksite employees ($N = 1,906$). These samples were population-based and reflect the demographics of the New England region. The samples included slightly more than

50% female, 2-4% Black/African Americans and 2-5% Hispanic, providing adequate demographic heterogeneity for the planned analyses. The subpopulations that were at risk (i.e. that were in the TTM pre-action stages of precontemplation, contemplation, or preparation) on the target behaviors (smoking, unhealthy diet, sun exposure) at baseline were included in the analyses.

Participants were adults who were proactively recruited for each intervention trial as described below. Eligibility included being at risk for at least one of the health risk behaviors targeted for intervention. *At-risk status* for each individual behavior was defined as being in the precontemplation, contemplation, or preparation stage of change. In each trial, participants were randomized to intervention or control conditions after providing informed consent. Participants randomized to the intervention group received TTM-tailored intervention materials mailed to their homes at baseline, 6-, and 12-months for each risk behavior that they were at risk for (e.g., nonsmokers did not receive any intervention for smoking). They were also provided with a multiple behavior self-help manual based on TTM strategies. Details of the intervention have been reported previously (Linnan et al., 2002; Prochaska et al., 2004; 2005; Velicer et al., 2004; Yin et al., 2013). All original trial procedures were approved by the Institutional Review Board at the University of Rhode Island.

Parent Sample. The first sample consisted of parents of 9th-grade students who participated in a school-based study. The 22 participating schools in the North Eastern US provided a list of parents. From this list, 2,460 eligible parents agreed to participate and completed the baseline survey. Eligible parents had to be at risk for at least one of the three risk behaviors: smoking, unhealthy diet and sun exposure.

Eighty-four percent (83.6%) of eligible participants were recruited with one parent recruited from each eligible household. Assessments were administered for all participants at baseline, 12, and 24 months. The original study outcomes were reported previously (Prochaska et al, 2004).

Patient Sample. A health insurance provider provided a list of patient names for a TTM-tailored intervention study that targeted smoking, unhealthy diet, sun exposure and mammography. Initial screening identified a total of 12,978 potential households, which were contacted by phone. Across the 8,539 patients who agreed to participate, 5,382 were eligible and were enrolled in the trial. One patient was recruited from each eligible household. Assessments were administered for all participants at baseline, 12, and 24 months. The original study outcomes were reported previously (Prochaska et al, 2005).

Employee Sample. The employee sample was part of a multiple risk behavior study that targeted smoking, unhealthy diet, sun exposure and physical inactivity. Participants were recruited from a total of 22 worksites (Linnan et al., 2002). Across the 2,224 eligible employees, 1,906 individuals agreed to participate, and were then randomized at the individual level. Assessments were administered for all participants at baseline, 12, and 24 months. The original study outcomes were reported previously (Velicer et al, 2004).

Measures

Background measures were assessed during baseline. The measures included demographics, problem behavior history, screening questions and health history.

Demographic data consisted of age, gender, racial and ethnic group status, marital status, education, and employment status.

Stages of Change. Common measures exist across risk factors because the randomized trials employed measures based on TTM constructs. The Stages of Change (SOC) are the central organizing construct in the TTM and provide the temporal dimension that accounts for the most variance in outcomes. The SOC reflects an individual's readiness to change from not meeting behavioral criteria to meeting behavioral criteria for a specific health risk (e.g. to quit smoking). The SOC is typically assessed based on an algorithm that assigns an individual to one of five ordered levels: 1. Precontemplation, not intending to meet behavioral criteria in the next 6 months; 2. Contemplation, intending to change in the next 6 months; 3. Preparation, intending to change behavior to meet criteria within the next 30 days; 4. Action, currently meeting behavioral criteria, but for less than 6 months; and 5. Maintenance, has met behavioral criteria for 6 months or more. To account for seasonal variations in sun exposure, the SOC algorithm for sun protection behavior uses 12 months instead of 6 months as the threshold separating (a) the precontemplation from the contemplation SOC, and (b) the action from the maintenance SOC. Generally, individuals in the pre-Action stages (precontemplation, contemplation, preparation) are considered "at-risk" because they have not yet taken effective action to meet behavioral criteria for reducing the specific health risk. The SOC criteria are unique for each behavior and as much as possible consensus criteria were used (e.g., abstinence for smoking). In measurement development studies, the behavior criteria for stage were always compared against standard measures of the

problem behavior. The reliability, utility, and predictive validity of the SOC algorithm have been demonstrated for various behaviors, including smoking cessation, healthy diet, and sun protection (DiClemente et al., 1991; Greene et al., 1999; Hall & Rossi, 2008; Prochaska & DiClemente, 1983; Velicer et al., 2007; Weinstock, Rossi, Redding, Maddock, & Cottrill, 2000). These stages of change have also demonstrated predictable relationships with other important TTM constructs, including Decisional Balance and Self Efficacy (Blissmer et al., 2010; DiClemente et al., 1991; Fava, Velicer & Prochaska, 1995; Hall & Rossi, 2008; Prochaska & Velicer, 1997). Velicer, Martin and Collins (1996) suggested that using SOC as an outcome measure has the advantage of being sensitive to all stage transitions, may increase precision and statistical power, and improve theoretical meaningfulness and interpretability. In the first study, SOC was used as a grouping variable instead of the primary behavior change outcome. Study 2 examined stage transitions in three cancer risk behaviors of smoking, unhealthy eating, and sun exposure, and focused on the SOC as the primary indicator of behavior change.

Quantitative Behavioral Measures. Besides the discrete stage measures, each trial also assessed quantitative measures for each target behavior. These quantitative behavioral outcome measures were common across the three trials, and are the dependent variables modeled in the longitudinal analyses for Study 1. Smoking behavior was assessed by the number of cigarettes participants reported smoking on a typical day, with a lower cigarette count representing lower smoking severity, and a count of zero indicating smoking abstinence or cessation. Healthy eating behavior was measured with the Dietary Behavioral Questionnaire (DBQ), which consists of 22-

items assessing food preparation and consumption on four subscales: 1) Substituting lower-fat foods for high-fat foods, 2) Avoiding high-fat foods, 3) Modifying food preparation methods to reduce fat consumption, and 4) Increasing consumption of fruits, vegetables, or higher-fiber foods. The DBQ has been validated against the NCI/Block Food Frequency Questionnaire (Greene et al, 2013; Kobayashi, 2011). Mean DBQ scores were computed based on responses to at least 20 out of the 22 items, with higher scores indicating more healthy eating behavior. Sun protective behavior was measured by the Sun Behavior Protection Index (SBPI; Rossi, Redding & Weinstock, 1998), which assesses how frequently participants limited sun exposure with subscales for Sun Avoidance and Sunscreen Use. Mean SBPI scores were computed based on responses to all 7 items of the measure, with higher scores indicating more sun protective behaviors.

Decisional Balance. Decision making constructs are represented by a Decisional Balance Inventory (DBI; Greene et al., 1999; Rossi et al., 1994; Velicer DiClemente, Prochaska & Brandenburg, 1985; Yin et al., 2014b) developed for each of the behaviors. The DBI measures the relative importance of the positives, benefits, or advantages (pros) of changing and the negatives, costs, or disadvantages (cons) of changing a specific behavior. The DBI assesses the pros and cons of smoking; higher endorsement of the pros of smoking indicates that the perceived benefits of smoking are considered to be more important. For diet and sun behaviors, the DBI assesses the pros and cons of reducing dietary fat and adopting sun protective behavior respectively. A comprehensive meta-analysis of 120 studies including 48 health behaviors found predictable, replicable relationships, named the strong and weak

principles of change, between the Pros and the Cons across the stages of change (Hall & Rossi, 2008; Prochaska et al, 1994).

Situational Temptations/Self-efficacy. Situational Temptations/Self-efficacy represents a variation of the self-efficacy construct (Bandura, 1977; 1982) and reflects how confident people are that they can maintain the behavior change in challenging situations. Instruments developed to assess situational temptations for smoking and dietary fat reduction and self-efficacy for sun protection behaviors have demonstrated measurement validity and reliability (Babbin et al., 2015; DiClemente, Prochaska & Gibertini, 1985; Velicer, DiClemente, Rossi & Prochaska, 1990; Rossi & Rossi, 1994). The temptations measures for smoking and diet behaviors assess how tempted a person feels to smoke or eat higher-fat foods across different situations, with higher endorsement reflecting a greater degree of temptation. For the self-efficacy scale for sun protection, higher mean scores indicate greater confidence in the ability to protect oneself from sun exposure.

Data Analysis

Behavior change was examined using two complementary longitudinal latent variable modeling techniques. Latent Growth Curve (LGC) modeling was the main analytical procedure employed in Study 1 to examine growth trajectories of quantitative behavioral measures. LGC models can be fitted as restricted factor models within the structural modeling framework, and are used to estimate within-person change and determinants of between-person differences in key change parameters (McArdle & Epstein, 1987; Meredith & Tisak, 1990). Each set of measured (manifest) indicators was sequentially examined in single behavior analyses, and multiple sets of

indicators were then simultaneously examined across co-occurring risk behavior pairs. In Study 2, Latent Transition Analysis (LTA) was the primary analytical approach employed to examine behavior change modeled as stage transitions for each of the three cancer-related risk behaviors of smoking, unhealthy eating and sun exposure. LTA are multivariate statistical models in a family of finite mixture models that allow unobserved underlying heterogeneity in outcomes to be modeled as discrete/categorical latent variables (i.e., a latent status/class variable) that are allowed to change over time. These are powerful and flexible analytical tools particularly suited for making large contingency tables interpretable (Goodman, 1974).

Preliminary analyses. The analyses examined the “functional relationships” between measured behavioral outcomes and the stages of change, an approach recommended for measure development and validation (Redding, Maddock & Rossi, 2006). Two-way factorial ANOVA was used to assess any differences in the behavioral scores (e.g. DBQ, SPBI) assessed at baseline across (i) the three baseline stages of precontemplation, contemplation, and preparation, (ii) the samples from different randomized trials, and (iii) any potential interaction between stage and sample. Behavioral scores were expected to show significant main effects for stage and nonsignificant or negligible stage by sample interactions effects.

Study 1: Latent Growth Trajectories. In Study 1, latent growth curve (LGC) models were developed sequentially to examine the two year trajectories of measured smoking, sun protective, and healthy eating behavior over time. Quantitative behavioral outcomes assessed at baseline, 12- and 24-months served as indicators for the growth trajectories for each risk behavior. A series of growth curve models were

fitted to estimate the rate of behavior change (slope) and initial level (intercept) for each risk behavior separately. With just three waves of data, only linear models could be estimated, and indicator residual variances were constrained to be equal over time in the growth models. Full-information maximum-likelihood estimation using the lavaan software package (Rosseel, 2012) in the R statistical computing environment was employed for all LGC models, allowing all available data from each participant to be used under the assumption that data was missing at random.

First, unconditional single behavior LGC models (Figure 1) were developed beginning with fewer estimated parameters, then sequentially increasing model complexity. The fit of the growth curve models to the data was evaluated using multiple fit indices, including the Comparative Fit Index (CFI; Bentler, 1990), the root mean square error of approximation (RMSEA; Browne & Cudeck, 1993), and the non-normed fit index or Tucker-Lewis index (NNFI/TLI; Tucker & Lewis, 1973). Better model fit is indicated by higher values (closer to 1) for CFI and NNFI, and RMSEA values less than .06 (Bentler, 1990; Hu & Bentler, 1999). The χ^2 is also reported for completeness, although it is known to be very sensitive to sample size (Kline, 2011).

Next, conditional LGC models that included intervention condition as a time-invariant covariate were evaluated to estimate the effect of treatment on the rate of behavior change (see Figure 2). Effect size d (with 95% confidence intervals) representing the standardized difference in behavioral outcomes were computed to estimate the magnitude of the intervention effect (Feingold, 2009; 2015; Raudenbush & Liu, 2001). The TTM constructs Decisional Balance (Pros, Cons) and Situational Temptations/Self-efficacy were then examined as predictors of behavior trajectories.

Baseline mean levels of behavior-specific Pros, Cons, or Situational Temptations/Self-efficacy were included as additional time-invariant covariates, and the coefficients were estimated when the slope factor was regressed on multiple covariates (intervention plus TTM constructs) simultaneously.

Finally, unconditional parallel-process LGC models (see Figure 3) were also developed to estimate trajectories for two behaviors simultaneously within pairs of co-occurring risk behaviors (MacCallum et al, 1997; deRuiter et al, 2014). The parallel-process growth models were estimated using data drawn from participants at baseline risk for both behaviors in the risk pair. The main parameter of interest in these unconditional parallel-process LGC models was the association (ψ) between the parallel behavior trajectories, especially the covariance between slope factors for each behavior pair. This allowed us to examine multiple behavior change, specifically whether rate of change in one behavior was associated with change in the second behavior within each behavior pair. Next, the stability of the unconditional parallel-process LGC models for each behavior pair were examined across subsamples defined by intervention condition using multiple sample invariance testing procedures (Hancock, Kuo & Lawrence, 2001; Yin, Rossi, Kobayashi & Redding, 2014). Parameters of interest were sequentially restricted to be equal across subsamples, starting from the least restrictive model and progressing to more restrictive models. Model invariance was assessed by examining the deterioration in model fit as additional cross-sample equality constraints were imposed, based on the χ^2 -difference test for nested models. Because the χ^2 statistic is very powerful when sample sizes are large (Kline, 2011), differences in practical fit indices such as the CFI, which is not

affected by sample size, were also assessed. Difference values for ΔCFI less than 0.01 have been suggested to be indicative of factorial invariance (Chen, 2007; Cheung & Rensvold, 2002).

Study 2: Behavioral Stage Transitions. In Study 2, latent transition analysis (LTA) was applied to examine patterns of stage transitions between latent status (i.e. behavior stage) subgroups over the two intervals between baseline, 1-, and 2-years. Because only those participants considered “at-risk” (i.e., baseline SOC was precontemplation, contemplation or preparation) for each behavior were selected, only three behavioral stages are represented in the stage transition models at baseline, although four different stage levels (precontemplation, contemplation, preparation, and action/maintenance) are represented in the models at years 1 and 2 (see Figure 4). Responses to behavior-specific SOC indicators were simultaneously assessed across the three time points to estimate (i) an individual’s behavior status at each time point (latent status membership probabilities δ), and (ii) the likelihood of change in status over time conditional on the estimated status at the previous time point (latent status transition probabilities τ). Measured SOC at each time point served as indicators of latent status for each risk behavior. However, rather than assume perfect reliability of the observed categorical responses, threshold levels for the measured binary SOC indicators were specified to allow the same low level of measurement error over time in the latent transition models (Kaplan, 2008). Full-information maximum-likelihood estimation with robust standard errors via the expectation-maximization algorithm (Dempster, Laird, & Rubin, 1977) in Mplus (Muthén & Muthén, 2012) was employed for all latent transition models. This maximum-likelihood procedure accounted for

data missing at random due to attrition, allowing all available data from each participant to be used to estimate parameters, standard errors, and fit statistics that are robust to non-normality and non-independence of observations. The best fitting model to the data, including the number of transition paths, was determined by comparing alternative nested models (see Figure 4) using several fit criteria and statistics. The Akaike Information Criterion (AIC; Akaike, 1981), Bayesian Information Criterion (BIC; Schwarz, 1978), and the likelihood ratio statistic G^2 that is distributed asymptotically as χ^2 (Agresti & Yang, 1987) are commonly used for this purpose. Better model fit is indicated by smaller values of AIC, BIC, and G^2 , and by G^2 values smaller than the model degrees of freedom (Lanza, Flaherty & Collins, 2003). Nested models were also evaluated using the scaled difference likelihood ratio test (ΔG^2) based on loglikelihood values and scaling correction factors obtained with the robust maximum-likelihood estimator (Asparouhov & Muthen, 2010; Satorra & Bentler, 2001; 2010). All models were estimated several times using random start values to minimize the risk of misspecification due to local maxima. Consistency in estimated parameters indicates that the estimation procedure correctly specified the global maximum.

Once the stage transition model with the appropriate number of transition paths that best fit the data was determined for each risk behavior, the stability of the transition parameters over time was assessed. The corresponding stationary model, in which stage transition probability parameters (τ) were constrained to be equal across time intervals, was then estimated for each risk behavior. Because only three behavioral stages (precontemplation, contemplation, and preparation) are represented

in the first wave, the equality across time constraints were only specified for stage transition parameters conditioned on those three stages; the transition parameters conditioned on action/maintenance stage from the second to third wave were freely estimated in the stationarity model. The scaled difference likelihood ratio (ΔG^2) test was used to compare the nested stationary and nonstationary models for each risk behavior.

Next, TTM-intervention condition was included as a time-invariant covariate (TICV) in the best fitting single behavior stage transition model to estimate the effect of treatment on stage transition probabilities (Muthén & Asparouhov, 2011). Finally, if the stage transition models for individual risk behaviors showed similar patterns in terms of the number of stage transition paths, the models were compared across risk behaviors. Comparison of results across different behaviors was generally conducted on an absolute basis.

CHAPTER 3

RESULTS

Participants

The final analytic sample included 9522 participants pooled across three population-based intervention trials who had valid baseline stage of change responses for each of the three cancer risk behaviors of smoking, unhealthy diet, and sun exposure. All available data from each participant was used for model estimation; full information maximum likelihood estimation procedures accounted for data missing at random due to attrition. The characteristics of these participants are presented in Table 1. The majority of participants included in this study were non-Hispanic White women, with mean age 44.6 years ($SD = 11.2$). Almost half of participants (47.3%) perceived their general health to be “Very good” or “Excellent.”

Table 2 summarizes the distribution of baseline stage of change for each cancer-related behavioral risk among the participants. A total of 2164 participants (23% of overall sample) reported being current smokers at baseline and were included in the smoking dataset. There were 6729 participants, more than two-thirds (71%) of the overall sample, who were at risk for unhealthy eating at baseline. There were 7065 participants (74%) who were at risk due to sun exposure.

Preliminary Analyses

Means and SDs for three quantitative behavioral measures (number of cigarettes smoked/day, DBQ, and SPBI) assessed at baseline are presented in Tables 3, 4, and 5 respectively. Two-way factorial ANOVAs revealed large effect sizes for stage

of change on the quantitative measures of behavior assessed at baseline, with small effects of sample, and negligible Stage X Sample interactions, confirming that expected stage of change effects on measured behavioral outcomes were consistent across samples. This suggests that it was reasonable to pool the data across sample for analyses as in several previous studies (e.g. Paiva et al, 2012; Kobayashi, 2013; Yin et al., 2013). Table 3 shows that the number of cigarettes smoked per day at baseline were significantly higher among participants in the precontemplation and contemplation stages of change, compared to those in preparation to quit smoking, $F(2, 2147) = 18.08, p < .001, \eta^2 = 0.017$. Sample was found to have a small and significant effect on the number of cigarettes smoked per day, $F(2, 2147) = 5.20, p < .01, \eta^2 = 0.005$; participants in the employee sample reported smoking fewer cigarettes per day at baseline compared to those in the parent sample. The effect of stage of change on smoking behavior was consistent across samples, $F(4, 2147) = 0.38, p = .821$.

Significant between-stage differences were also found for the DBQ mean score at baseline, $F(2, 6642) = 55.36, p < .001, \eta^2 = 0.016$. Follow-up Tukey tests revealed that participants in preparation reported significantly higher DBQ mean scores when compared to those in precontemplation or contemplation at baseline (Table 4). DBQ mean scores were also significantly different across sample, $F(2, 6642) = 8.16, p < .001, \eta^2 = 0.002$, with slightly higher mean DBQ scores in the patient sample compared to the employee sample. The effect of stage of change on DBQ mean scores was consistent across samples, $F(4, 6642) = 0.39, p = .814$.

The SPBI mean scores were significantly different across stage at baseline, $F(2,7012) = 2125.05, p < .001, \eta^2 = 0.377$. Table 5 shows that baseline SPBI mean scores were significantly higher in later stages compared to earlier stages. The SPBI mean scores were also slightly higher for the patient sample compared to the employee sample, $F(2,7012) = 10.44, p < .001, \eta^2 = 0.003$. A small but significant effect of Stage X Sample interaction was detected, $F(4, 7012) = 4.83, p < .01, \eta^2 = 0.003$, although this was more likely an artifact of the large sample size.

Study 1: Behavioral Growth Trajectories

Descriptive statistics for the quantitative behavioral outcome measures at each time point were computed for the LGCM analytic samples for each risk behavior. For smoking, participants were included in the analytic sample if they were in the precontemplation, contemplation or preparation stages, and reported non-zero cigarette counts at baseline. Out of 2164 smokers, 8 had missing baseline cigarette count data, and another 39 reported smoking zero cigarettes, producing an analytic sample of 2117 smokers for LGCM. The distribution of cigarette count data was found to be positively skewed with high kurtosis at each time point (Table 6), so a square root transformation was applied to the data to bring it closer to a normal distribution. The square root transformed cigarette counts at each time point were then used as indicators in all LGC models for smoking behavior. Table 6 also reveals that the standard deviation for cigarette counts was much larger at 1- and 2-year compared to baseline.

There were 78 participants out of 6729 at risk for unhealthy eating at baseline with insufficient data to compute the baseline DBQ mean score, the remaining 6651

participants comprised the LGCM analytic sample for diet behavior. Table 7 shows that mean DBQ scores appear to be slightly higher at 1- and 2-years compared to baseline, the standard deviations were similar across time points, and skewness and kurtosis were acceptable.

For sun exposure, 44 of the 7065 at risk participants did not have sufficient baseline SPBI data to be included in the LGCM analytic sample ($N = 7021$). Table 8 shows that SPBI mean scores also appear to be slightly higher at year 1 and 2 compared to baseline, the standard deviations were similar across time points, and skewness and kurtosis were acceptable.

Unconditional LGC Models. Table 9 shows the model fit statistics for nested unconditional 2-year LGC models developed sequentially for smoking, diet, and sun exposure separately. In order for the linear growth model for smoking behavior to converge, the constraint of equality over time for indicator residual variances was released for the first time point, with only 12- and 24-month residuals set to be equal. The unconditional linear growth model for smoking with random intercept and slope factors (Model 1A.5) fit the data in the full sample well: $\chi^2(2, N=2117) = 24.218, p < .001$; CFI = .973; RMSEA = .072, 90% CI [.048, .100]. Examination of the unstandardized parameter estimates revealed that smoking behavior decreased significantly over time, mean slope $\hat{\alpha}_S = -0.26$, SE = 0.028, $p < .001$. The estimated slope factor variance of 0.47, SE = 0.06, $p < .001$, indicated that there was significant inter-individual variation in the rate of behavior change. The intercept factor estimated mean $\hat{\alpha}_I = 4.01$, SE = 0.03, $p < .001$ (factor variance 0.93, SE = 0.11, $p < .001$), showed an initial starting level of approximately 16.06 (i.e. 4.01^2) cigarettes/day

expressed in the original outcome metric, with significant variation between individuals in baseline smoking behavior. In addition, the estimated covariance between both intercept and slope factors $\hat{\psi}_{IS} = 0.21$, $SE = 0.07$, was positive and significant based on $\Delta\chi^2(1) = 8.259$, $p < .01$, suggesting that lower initial smoking levels were associated with steeper decreases (more negative slope) in smoking behavior. The unconditional growth trajectory model for smoking behavior and standardized parameter estimates are shown in Figure 7.

The unconditional linear LGC model for eating behavior with random intercept and slope factors (Model 1A.5) fit the data in the full sample well: $\chi^2(3, N=6651) = 169.793$, $p < .001$; CFI = .980; RMSEA = .091, 90% CI [.080, .103]. An alternative linear growth model with uncorrelated intercept and slope factors (Model 1A.6) was more parsimonious and also provided a good fit to the data: $\chi^2(4, N=6651) = 169.992$, $p < .001$; CFI = .980; RMSEA = .079, 90% CI [.069, .089]. Examination of the unstandardized parameter estimates revealed that healthy eating behavior increased slightly but significantly over time, mean slope $\hat{\alpha}_S = 0.06$, $SE = 0.003$, $p < .001$. The estimated slope factor variance was 0.01, $SE = 0.001$, $p < .001$, indicating significant variation between individuals variation in rate of behavior change. The intercept factor estimated mean $\hat{\alpha}_I = 3.30$, $SE = 0.007$, $p < .001$, represented a mean score around 3.30 (on a scale of 1 to 5) on the DBQ. The intercept factor estimated variance was 0.23, $SE = 0.005$, $p < .001$, and indicated significant variation among individuals in diet behavior. The estimated covariance between intercept and slope factors was negligible and nonsignificant, $\hat{\psi}_{IS} = 0.001$, $SE = 0.002$, $\Delta\chi^2(1) = 0.199$, $p = .655$, suggesting that the rate of change in diet behavior was unrelated to initial DBQ scores.

The unconditional growth trajectory model for diet behavior with uncorrelated intercept and slope latent factors is shown in Figure 8 with standardized parameter estimates.

The unconditional LGC model with random intercept and slope factors (Model 1A.5) for sun protective behavior fit the data in the full sample reasonably well: $\chi^2(3, N=7021) = 268.225, p < .001$; CFI = .963; RMSEA = .112, 90% CI [.101, .124]. Examination of the unstandardized parameter estimates found that sun protective behavior increased significantly over time, mean slope $\hat{\alpha}_S = 0.14, SE = 0.005, p < .001$. The estimated slope factor variance was 0.026, $SE = 0.003, p < .001$, indicating significant between-individual variation in the rate of behavior change. The model estimated intercept factor mean $\hat{\alpha}_I = 3.01, SE = 0.009, p < .001$ (variance 0.39, $SE = 0.01, p < .001$), indicated that initial mean scores were above the theoretical midpoint of 2.50 (on 5 point scale) on the SPBI, and that there was significant variation between individuals in baseline sun protective behavior. In addition, the estimated covariance between both intercept and slope factors was positive and significant $\hat{\psi}_{IS} = 0.01, SE = 0.004, \Delta\chi^2(1) = 7.591, p < .01$. Further probing of the standardized parameter estimates revealed that the estimated correlation between the intercept and slope factors was 0.113, suggesting that higher initial SPB mean scores were weakly associated with steeper increases in sun protective behavior. The unconditional growth trajectory model for sun protection behavior with standardized parameter estimates are shown in Figure 9.

Conditional LGC Models. Table 10 shows the model fit statistics for nested conditional 2-year LGC models for each behavior, when TTM-intervention condition

was included as a fixed, time-invariant covariate. The fit of the conditional LGC model with random intercept and slope factors (Model 1B.5) for smoking was excellent: $\chi^2(3, N=2117) = 24.078, p < .001$; CFI = .975; RMSEA = .058, 90% CI [.038, .080]. Examination of the unstandardized parameter estimates found that smoking behavior decreased significantly over time in the control condition, $\hat{\alpha}_S = -0.24, SE = 0.04, p < .001$ from an initial level of smoking $\hat{\alpha}_I = 4.08, SE = 0.04, p < .001$. Slope and intercept factor variance estimates of $\hat{\psi}_{SS} = 0.46, SE = 0.06, p < .001$, and $\hat{\psi}_{II} = 0.92, SE = 0.11, p < .001$ respectively, indicated significant variation between control group participants in the rate of smoking decrease and initial smoking behavior. As observed in the unconditional LGCM for smoking (Model 1A.5), this conditional model (Model 1B.5) estimated covariance $\hat{\psi}_{IS} = 0.21, SE = 0.07, p < .01$, suggested that initial smoking level was associated with the rate of decrease in smoking behavior. The estimated regression coefficient $\hat{\gamma}_I = -0.15, SE = 0.056, p < .01$, revealed that the initial smoking level was significantly lower in the intervention condition compared to controls. Finally, the estimated decrease in smoking was slightly steeper in the intervention group, $\hat{\gamma}_S = -0.06, SE = 0.06, p = .316$, although this intervention effect was very small and not significant due to the large estimated standard error. The estimated standardized difference (with 95% confidence intervals) $\hat{d} = -0.09$ [95% CI: -0.26, 0.09], suggests that smoking behavior at Year 2 was slightly lower in treatment versus control groups. However, the magnitude of this outcome difference due to the intervention effect on decreasing smoking behavior was small and also not significant, with 95% CI that contain zero (no difference in

outcome). Figure 10 shows the conditional growth trajectory model for smoking behavior with standardized parameter estimates.

The conditional LGCM for eating behavior with uncorrelated random slope and intercept factors (Model 1B.6) provided an excellent fit to the data: $\chi^2(5, N=6651) = 177.131, p < .001$; CFI = .980; RMSEA = .072, 90% CI [.0631, .081]. Examination of the unstandardized parameter estimates found that healthy eating behavior increased significantly over time in the control condition, $\hat{\alpha}_S = 0.03, SE = 0.004, p < .001$ from an initial level DBQ scores $\hat{\alpha}_I = 3.29, SE = 0.009, p < .001$. Slope and intercept factor variance estimates of $\hat{\psi}_{SS} = 0.013, SE = 0.001, p < .001$, and $\hat{\psi}_{II} = 0.223, SE = 0.005, p < .001$ respectively, indicated significant variation among control group participants in the rate of healthy eating behavior increase and initial diet behavior levels. The rate of change in diet behavior was unrelated to initial DBQ scores, $\hat{\psi}_{IS} = 0, SE = 0.002, \Delta\chi^2(1) = 0.005, p = .944$, replicating the finding from the previous unconditional growth model for diet (Model 1A.6). In all subsequent LGC models, the growth trajectories for diet were specified with intercept and slope factors uncorrelated within diet behavior. The estimated regression coefficient $\hat{\gamma}_I = 0.03, SE = 0.013, p = .021$, revealed that the initial DBQ level was just significantly higher in the intervention condition compared to controls. Finally, the estimated regression coefficient $\hat{\gamma}_S = 0.06, SE = 0.006, p < .001$, suggests that intervention condition predicted a significantly steeper increase in healthy eating behavior. The estimated standardized difference $\hat{d} = 0.23$ [95% CI: 0.19, 0.28], indicates that healthy eating behavior (DBQ mean scores) at Year 2 was significantly higher in treatment versus control groups, due to the intervention effect on increasing healthy diet behavior. The

unconditional growth trajectory model for healthy diet behavior with uncorrelated intercept and slope factors is shown in Figure 11 with standardized parameter estimates.

The conditional LGCM for sun protective behavior with random slope and intercept factors (Model 1B.5) fit the data well: $\chi^2(4, N=7021) = 270.621, p < .001$; CFI = .963; RMSEA = .097, 90% CI [.088, .170]. Examination of the unstandardized parameter estimates found that sun protective behavior increased significantly over time in the control condition, $\hat{\alpha}_S = 0.10, SE = 0.006, p < .001$ from an initial SPBI scores $\hat{\alpha}_I = 2.98, SE = 0.012, p < .001$. Slope and intercept factor variance estimates of $\hat{\psi}_{SS} = 0.02, SE = 0.003, p < .001$, and $\hat{\psi}_{II} = 0.387, SE = 0.010, p < .001$ respectively, indicated significant variation among control group participants in the rate of sun protective behavior change and initial SPBI levels. An alternative conditional LGCM for sun protective behavior with uncorrelated random slope and intercept factors (Model 1B.6) also fit the data well: $\chi^2(5, N=7021) = 276.346, p < .001$; CFI = .965; RMSEA = .088, 90% CI [.079, .097]. Although the estimated covariance between intercept and slope factors was just significant, $\hat{\psi}_{IS} = 0.01, SE = 0.002, \Delta\chi^2(1) = 5.725, p = .017$, the magnitude of the parameter estimate was fairly small (standardized parameter estimate was 0.10) and suggest that increases in sun protective behaviors were only weakly associated with initial SPBI scores. The estimated regression coefficient $\hat{\gamma}_I = 0.05, SE = 0.018, p = .007$, revealed that the initial SPBI level was just significantly higher in the intervention condition compared to controls. Finally, the estimated regression coefficient $\hat{\gamma}_S = 0.09, SE = 0.009, p < .001$, suggests that intervention condition predicted a significantly steeper increase in

sun protective behavior as measured by SPBI mean scores. The estimated standardized difference $\hat{d}=0.25$ [95% CI: 0.20, 0.30], indicates that sun protective behavior (SPBI mean scores) was significantly higher in treatment versus control groups at Year 2 due to the intervention effect on increasing sun protection behavior. The conditional growth trajectory model for sun protection behavior (Model 1B.5) is shown in Figure 12 with standardized parameter estimates.

Conditional LGC Models including baseline Decisional Balance and Situational Temptations/Self-efficacy as covariates. Descriptive statistics for baseline mean scores for behavior specific Pro, Cons, and Situational Temptations/Self-efficacy are presented in Table 11. Participants' endorsement of the Pros and Cons of unhealthy (high-fat) diet behavior was low for both constructs, with mean scores for both Pros (M=2.28, SD=1.08) and Cons (M=2.26, SD=0.97) below the theoretical midpoint (2.50) for the scales. Three additional conditional LGC models were examined for each behavior separately that included intervention condition as a time-invariant covariate, and either baseline mean Pros, Cons, or Situational Temptations/Self-efficacy as a second time-invariant covariate. A final conditional LGCM for each behavior was assessed that included four covariates simultaneously (Model 1C.5): intervention condition together with all three baseline mean scores for Pros, Cons, and Situational Temptations/Self-efficacy. Model fit statistics for these nested conditional single-behavior LGC models with multiple time-invariant covariates are presented in Table 12. Table 13 lists the unstandardized regression coefficient estimates from Model 1C.5 for each of the four time-invariant covariates (intervention condition, baseline mean Pros, Cons, and Situational Temptations/Self-

efficacy) for each risk behavior. The regression coefficients for intervention condition from Model 1C.1 are also presented in Table 13 for comparison.

The conditional LGCM for smoking with intervention condition, Pros, Cons, and Temptations included as time-invariant covariates fit the data well, $\chi^2(6, N=2117) = 24.355, p < .001$; CFI = .987; RMSEA = .038; 90% CI [.023, .054]. Examining the unstandardized parameter estimates revealed that after controlling for baseline Pros, Cons and Temptations, smoking behavior did not change significantly over time in the control condition, $\hat{\alpha}_S = 0.04, SE = 0.175, p = .825$, from an initial level of smoking, $\hat{\alpha}_I = 1.37, SE = 0.148, p < .001$. The estimated regression coefficient $\hat{\gamma}_{I_{Trt}} = -0.09, SE = 0.049, p = .07$, revealed that the initial smoking level was slightly lower in the intervention condition compared to control, although this difference was not significant. Baseline Pros and Temptations were both significant predictors of initial smoking behavior, $\hat{\gamma}_{I_{Pro}} = 0.13, SE = 0.034, p < .001$, and $\hat{\gamma}_{I_{Tmpt}} = 0.74, SE = 0.040, p < .001$, suggesting that participants who endorsed the Pros of smoking more highly, and those reporting higher Temptations, did smoke more. Baseline Cons was not found to be a significant predictor of initial smoking behavior, $\hat{\gamma}_{I_{Con}} = -0.05, SE = 0.028, p = .097$. None of the covariates were significant predictors of change in smoking over time (slope). Higher baseline Pros predicted a slight increase in smoking over time, $\hat{\gamma}_{S_{Pro}} = 0.02, SE = 0.040, p = .687$. Higher baseline Cons and Temptations predicted a slight decrease in smoking over time, $\hat{\gamma}_{S_{Con}} = -0.02, SE = 0.032, p = .592$, and $\hat{\gamma}_{S_{Tmpt}} = -0.08, SE = 0.049, p = .120$, respectively. After controlling for baseline Pros, Cons and Temptations, intervention condition predicted a slight but nonsignificant decrease in smoking behavior over time, $\hat{\gamma}_{S_{Trt}} = -0.06, SE = 0.057, p =$

.285. The conditional growth curve model for smoking behavior with four time-invariant covariates is presented in Figure 13 with standardized parameter estimates.

The conditional LGCM for diet behavior with intervention condition, Pros, Cons, and Temptations included as time-invariant covariates fit the data well, $\chi^2(8, N=6651) = 179.158, p < .001$; CFI = .981; RMSEA = .057, 90% CI [.050, .064].

Examining the unstandardized parameter estimates revealed that after controlling for baseline Pros, Cons and Temptations, healthy eating behavior did not change significantly over time in the control condition, $\hat{\alpha}_S = 0.02, SE = 0.013, p = .144$, from an initial DBQ mean score of $\hat{\alpha}_I = 3.35, SE = 0.025, p < .001$. The estimated regression coefficient revealed that the initial DBQ level was slightly higher in the intervention condition compared to control $\hat{\gamma}_{I_{T_{rt}}} = 0.03, SE = 0.013, p < .05$. Baseline Pros and Temptations were significant predictors of initial healthy eating behavior, $\hat{\gamma}_{I_{Pro}} = 0.05, SE = 0.007, p < .001$, and $\hat{\gamma}_{I_{T_{mpt}}} = 0.07, SE = 0.009, p < .001$, suggesting that higher endorsement of the Pros of reducing dietary fat, and higher levels of Temptations reported at baseline, were associated with higher initial DBQ scores. Baseline Cons was also found to be a significant predictor of initial diet behavior, $\hat{\gamma}_{I_{Con}} = -0.16, SE = 0.008, p < .001$, suggesting that higher endorsement of the Cons of reducing dietary fat was predictive of lower baseline DBQ scores. Baseline Pros was not a significant predictor of eating behavior change over time, $\hat{\gamma}_{S_{Pro}} = 0.001, SE = 0.003, p = .750$. Higher baseline Cons predicted a very slight increase in healthy eating over time, $\hat{\gamma}_{S_{Con}} = 0.01, SE = 0.004, p < .01$. Baseline Temptations was not a significant predictor of the slope for DBQ scores, $\hat{\gamma}_{S_{T_{mpt}}} = -0.006, SE = 0.005, p = .168$. After controlling for baseline Pros, Cons and Temptations, healthy eating behavior increased more

steeply in the intervention condition compared to controls, $\hat{\gamma}_{S_{T_{rt}}} = 0.06$, $SE = 0.006$, $p < .001$. The conditional growth trajectory model for diet behavior with four time-invariant covariates is presented in Figure 14 with standardized parameter estimates; latent intercept and slope factors for diet are uncorrelated.

The conditional LGCM for sun protection behavior with intervention condition, Pros, Cons, and Self-efficacy included as time-invariant covariates fit the data well, $\chi^2(7, N=7021) = 335.447$, $p < .001$; CFI = .973; RMSEA = .082, 90% CI [.074, .089]. Examining the unstandardized parameter estimates revealed that after controlling for baseline Pros, Cons and Self-efficacy, sun protection behavior increased significantly over time in the control condition, $\hat{\alpha}_5 = 0.10$, $SE = 0.025$, $p < .001$, from an initial SPBQ mean level $\hat{\alpha}_I = 1.60$, $SE = 0.033$, $p < .001$. After controlling for baseline Pros, Cons and Self-efficacy, the initial SPBI level in the intervention condition was not significantly higher compared to control $\hat{\gamma}_{I_{T_{rt}}} = 0.02$, $SE = 0.013$, $p = .871$. Baseline Pros and Self-efficacy were significant predictors of initial sun protection behavior, $\hat{\gamma}_{I_{Pro}} = 0.20$, $SE = 0.009$, $p < .001$, and $\hat{\gamma}_{I_{SEff}} = 0.40$, $SE = 0.009$, $p < .001$, suggesting that participants who endorsed the Pros of sun protection more highly, and those reporting higher baseline Self-efficacy, had higher initial SPBI scores. Baseline Cons was also found to be a significant predictor of initial sun protection behavior, $\hat{\gamma}_{I_{Con}} = -0.15$, $SE = 0.007$, $p < .001$, suggesting that higher endorsement of the Cons of sun protection was predictive of lower baseline SPBI scores. Baseline Pros was not a significant predictor of sun protection behavior change over time, $\hat{\gamma}_{S_{Pro}} = -0.01$, $SE = 0.006$, $p = .057$. Higher baseline Cons predicted a slight increase in SPBI scores over time, $\hat{\gamma}_{S_{Con}} = 0.02$, $SE = 0.005$, $p < .001$. Baseline Self-

efficacy was not a significant predictor of the slope for SPBI scores, $\hat{\gamma}_{SEff} = -0.001$, $SE = 0.007$, $p = .876$. After controlling for baseline Pros, Cons and Self-efficacy, intervention condition predicted as steeper increase in sun protection behavior, $\hat{\gamma}_{Srtt} = 0.10$, $SE = 0.009$, $p < .001$. The conditional growth curve model for sun protection behavior with four time-invariant covariates is presented in Figure 15 with standardized parameter estimates.

Unconditional parallel-process LGCM of co-occurring risk behavior dyads.

Parallel-process LGC models were developed to estimate 2-year growth trajectories jointly across behaviors within co-occurring risk pairs of 1) Smoking and unhealthy diet, 2) Unhealthy diet and sun exposure, and 3) Smoking and sun exposure. Data was drawn from participants who were at risk at baseline for both behaviors in each risk pair. There were 1496 participants at risk for both smoking and unhealthy diet at baseline. There were 4681 participants at risk for unhealthy diet and sun exposure at baseline. For smoking and sun exposure, 1499 participants had both behavioral risks at baseline. Model fit statistics for nested unconditional parallel-process LGC models developed sequentially for each behavior pair are presented in Table 14. Two alternative unconditional parallel-process LCGMs with random intercepts and slopes were assessed for each risk behavior dyad: covariance between all growth factors across behaviors were estimated in Model 1D.5, and in Model 1D.6, independence was assumed between intercept-slope factors across behaviors, and only the covariance's between intercept-intercept and between slope-slope were estimated across behaviors in the pair (see Figure 3).

The unconditional parallel-process LCGMs with random intercepts and slopes for smoking and diet behavior dyad fit well. χ^2 -difference test of the nested models 1D.5 and 1D.6 found that estimating the two additional parameters did not significantly improve model fit: $\Delta\chi^2(2, N=1496) = 1.921, p = .383$, so the more parsimonious Model 1D.6 was retained. Examining the unstandardized parameter estimates revealed that smoking behavior decreased significantly over time, $\hat{\alpha}_{sm} = -0.27, SE = 0.034, p < .001$, from an initial level of smoking, $\hat{\alpha}_{ism} = 4.13, SE = 0.033, p < .001$, while healthy eating behavior increased slightly over the same interval, $\hat{\alpha}_{dat} = 0.05, SE = 0.008, p < .001$, from an initial level of $\hat{\alpha}_{idt} = 3.12, SE = 0.015, p < .001$. The estimated covariance between smoking and diet intercept factors was negative and significant, $\hat{\psi}_{ismidt} = -0.17, SE = 0.019, p < .001$, suggesting that higher initial levels of smoking were associated with lower initial DBQ mean scores (i.e., less healthy diet behavior). The estimated covariance between smoking and diet slope factors was also negative but not significant, $\hat{\psi}_{smst} = -0.02, SE = 0.008, p = .067$, suggesting that the rate of decrease in smoking behavior was not strongly related to the rate of increase in healthy eating. The parallel-process LGC model for smoking and diet behaviors (Model 1D.6) are shown in Figure 16 with standardized parameter estimates.

The unconditional parallel-process LCGMs with random intercepts and slopes for diet and sun protection behavior dyad fit the data well: $\chi^2(14, N=4681) = 303.242, p < .001$; CFI = 0.974; RMSEA = .066, 90% CI [.060, .073] for the more parsimonious model 1D.6. Examination of the unstandardized parameter estimates found that healthy eating behavior increased significantly over time, $\hat{\alpha}_{dat} = 0.06, SE =$

0.004, $p < .001$, from an initial level of $\hat{\alpha}_{I_{dt}} = 3.25$, $SE = 0.008$, $p < .001$. Sun protection behavior also increased significantly over the same interval, $\hat{\alpha}_{S_{su}} = 0.13$, $SE = 0.006$, $p < .001$, from an initial level of $\hat{\alpha}_{I_{su}} = 2.96$, $SE = 0.011$, $p < .001$. The estimated covariance between diet and sun protection intercept factors, $\hat{\psi}_{I_{dt}I_{su}} = 0.09$, $SE = 0.006$, $p < .001$, indicates that higher initial levels of healthy eating were associated with higher initial levels of sun protection behavior. The estimated covariance between sun protection and diet slope factors was also positive, $\hat{\psi}_{S_{dt}S_{su}} = 0.01$, $SE = 0.001$, $p < .001$, suggesting that the rate of increase in healthy eating behavior was significantly associated with the rate of increase in sun protection behavior. The parallel-process LGC model (Model 1D.6) for sun protection and healthy eating behaviors are shown in Figure 17 with standardized parameter estimates.

The unconditional parallel-process LCGMs with random intercepts and slopes for the smoking and sun protection behavior pair provided a good fit to the data: $\chi^2(12, N=1499) = 77.913$, $p < .001$; CFI = 0.967; RMSEA = .061, 90% CI [.048, .074] for the more parsimonious model 1D.6. Examining the unstandardized parameter estimates revealed that smoking behavior decreased significantly over time, $\hat{\alpha}_{S_{sm}} = -0.23$, $SE = 0.033$, $p < .001$, from an initial level of smoking, $\hat{\alpha}_{I_{sm}} = 4.03$, $SE = 0.033$, $p < .001$, while sun protection behavior also increased slightly over the same interval, $\hat{\alpha}_{S_{su}} = 0.14$, $SE = 0.012$, $p < .001$, from an initial level of $\hat{\alpha}_{I_{su}} = 2.79$, $SE = 0.021$, $p < .001$. The estimated covariance between smoking and sun intercept factors was negative and significant, $\hat{\psi}_{I_{sm}I_{su}} = -0.15$, $SE = 0.026$, $p < .001$, suggesting that higher initial levels of smoking were associated with lower initial levels of sun protection behavior (i.e.,

lower SPBI mean scores). The estimated covariance between smoking and sun protection slope factors was also negative but not significant, $\hat{\psi}_{s_{sm}s_{su}} = -0.01$, $SE = 0.013$, $p = .387$, suggesting that the rate of decrease in smoking behavior was not consistently related to the rate of increase in sun protection behavior. The parallel-process LGC model (Model 1D.6) for smoking and sun protection behaviors are shown in Figure 18 with standardized parameter estimates.

Stability of parallel-process LGCM of co-occurring risk behavior dyads across intervention condition. Finally, multiple-sample invariance analyses were conducted to assess the stability of the slope parameters across intervention condition. The parallel-process growth model (Model 1D.6) was fitted simultaneously to intervention and control group data for each risk behavior dyad. Four invariance models were tested sequentially for each health behavior pair: Equal form, Equal slope factor means, Equal slope factor means and covariance, and Equal slope factor means and factor covariances. Model fit statistics are presented in Table 15 for each invariance model by behavior dyad. Overall model fit, and the χ^2 -difference and ΔCFI for nested model comparisons, were both considered when examining the invariance models. Cohen's q was also computed to estimate the magnitude of the difference in slope factor correlations between intervention conditions (Cohen, 1988).

The model with equal slope factor means and factor covariances (Model 1E.4) for the smoking and unhealthy diet risk dyad fit the data well, $\chi^2(27, N=1496) = 62.037$, $p < .001$; $CFI = 0.985$; $RMSEA = .042$, 90% CI [.028, .055]. Examination of the parameter estimates from the equal forms model found that in smokers who were also at baseline risk for unhealthy diet, smoking decreased over time in the

intervention group, $\hat{\alpha}_{sm(1)} = -0.27$, $SE = 0.057$, $p < .001$, and among controls, $\hat{\alpha}_{sm(0)} = -0.27$, $SE = 0.042$, $p < .001$. Healthy eating behavior also increased significantly over the same interval in the treatment group, $\hat{\alpha}_{dt(1)} = 0.08$, $SE = 0.012$, $p < .001$, and in the control group, $\hat{\alpha}_{dt(0)} = 0.02$, $SE = 0.010$, $p < .001$. Comparing the nested models when slope factor means were constrained to equality across intervention condition showed that healthy eating behavior increased at a significantly different rate (slope) across intervention condition, $\Delta\chi^2(2, N=1496) = 14.947$, $p < .001$. It should be noted that imposing the equality constraint did not cause the model fit to deteriorate too badly, based on $\Delta CFI = 0.006$ that was within the criteria for factorial invariance, suggesting that the large sample size might have influenced the $\Delta\chi^2$ achieving significance. The estimated covariance between smoking and diet slope factors was negative and significant in the control group, $\hat{\psi}_{sm dt(0)} = -0.026$, $SE = 0.010$, $p = .011$, suggesting that the rate of decrease in smoking behavior was associated with the rate of increase in healthy eating in untreated participants. However, no such relationship was found between the slopes for smoking and healthy eating in the intervention group, $\hat{\psi}_{sm dt(1)} = 0.003$, $SE = 0.014$, $p = .807$. The magnitude of the difference in the slope-slope correlation between the control ($\hat{r}_{(0)} = -.37$) and treatment ($\hat{r}_{(1)} = .02$) groups was estimated by $\hat{q} = 0.41$. However, comparing the nested models when $\hat{\psi}_{sm dt}$ was constrained to equality suggests that this association was not significantly different across intervention groups, $\Delta\chi^2(1, N=1496) = 2.711$, $p = .100$. Figure 19 shows the standardized parameter estimates across intervention condition for the parallel-process growth curve model for smoking and diet behaviors.

The model with equal slope factor means and factor covariances (Model 1E.4) for the sun exposure and unhealthy diet risk dyad fit the data well, $\chi^2(28, N=4681) = 343.751, p < .001$; CFI = 0.971, RMSEA = .069, 90% CI [.063, .076]. Examination of the parameter estimates from the equal forms model found that in participants with both behavioral risks at baseline, sun protection behavior increased over time in the intervention group, $\hat{\alpha}_{su(1)} = 0.17, SE = 0.009, p < .001$, and also among controls, $\hat{\alpha}_{su(0)} = 0.09, SE = 0.008, p < .001$. Healthy eating behavior also increased significantly over the same interval in both the treatment group, $\hat{\alpha}_{dt(1)} = 0.08, SE = 0.006, p < .001$, and in the control group, $\hat{\alpha}_{dt(0)} = 0.03, SE = 0.005, p < .001$.

Constraining slope factor means to equality across intervention condition yielded $\Delta\chi^2(2, N=4681) = 85.949, p < .001$ for the nested model comparison, suggesting that the rate of increase for each behavior occurred at significantly different rates (slopes) across intervention condition, although $\Delta CFI = 0.008$ indicated acceptable deterioration in model fit. The estimated covariance between sun protection and diet slope factors was positive and significant in the control group, $\hat{\psi}_{su,dt(0)} = 0.008, SE = 0.002, p < .001$, and in the intervention group, $\hat{\psi}_{su,dt(1)} = 0.01, SE = 0.002, p < .001$, suggesting that the rates of increase in sun protection and healthy eating behaviors were consistently related. The magnitude of the difference in the slope-slope correlation between the control ($\hat{r}_{(0)} = .52$) and treatment ($\hat{r}_{(1)} = .28$) groups was estimated by $\hat{q} = 0.24$. However, comparing the nested models when $\hat{\psi}_{su,dt}$ was constrained to equality supports the invariance of this parameter across intervention groups, $\Delta\chi^2(1, N=4681) = 0.97, p = .324$, and $\Delta CFI = 0$. The estimated covariance between diet and sun protection intercept factors indicates that higher initial levels of

healthy eating were associated with higher initial levels of sun protection behavior in the control group, $\hat{\psi}_{I_{su}I_{dt}(0)} = 0.07$, $SE = 0.007$, $p < .001$, and also in the intervention group, $\hat{\psi}_{I_{su}I_{dt}(1)} = 0.11$, $SE = 0.008$, $p < .001$. Constraining the covariance between intercept factors to equality across intervention condition yielded $\Delta\chi^2(1, N=4681) = 13.131$, $p < .001$, but $\Delta CFI = 0.001$ for the nested model comparison, supporting invariance of this parameter across intervention condition. The parallel-process model for sun protection and healthy eating behavior trajectories is shown in Figure 20 with standardized parameter estimates across intervention condition.

The model with equal slope factor means and factor covariances (Model 1E.4) for the sun exposure and smoking risk behavior dyad provided a good fit to the data, $\chi^2(25, N=1499) = 74.396$, $p < .001$; $CFI = 0.975$, $RMSEA = .051$, 90% CI [.038, .065]. Examination of the parameter estimates from the equal forms model found that in smokers who were also at baseline risk for sun exposure, sun protection behavior increased over time in the intervention group, $\hat{\alpha}_{S_{su}(1)} = 0.18$, $SE = 0.018$, $p < .001$, and among controls, $\hat{\alpha}_{S_{su}(0)} = 0.10$, $SE = 0.016$, $p < .001$. Smoking also decreased significantly over the same interval in both the treatment group, $\hat{\alpha}_{S_{sm}(1)} = -0.25$, $SE = 0.054$, $p < .001$, and in the control group, $\hat{\alpha}_{S_{sm}(0)} = -0.20$, $SE = 0.042$, $p < .001$. Constraining slope factor means to equality across intervention condition yielded $\Delta\chi^2(2, N=1499) = 9.824$, $p = .007$, and $\Delta CFI = 0.004$, for the nested model comparison, suggesting that the rate of change (slopes) for each behavior may not be too dissimilar across intervention condition. The estimated covariance between sun protection and smoking slope factors was negative but not significant in the intervention group, $\hat{\psi}_{S_{su}S_{sm}(1)} = -0.03$, $SE = 0.019$, $p = .176$, and close to zero in the

control group, $\hat{\psi}_{S_{su}S_{sm}(0)} = 0.004$, $SE = 0.016$, $p = .786$, indicating that the rates of increase in sun protection and decrease in smoking were not consistently related. The magnitude of the difference in the slope-slope correlation between the control ($\hat{r}_{(0)} = .03$) and treatment ($\hat{r}_{(1)} = -.08$) groups was estimated by $\hat{q} = 0.11$. Comparing the nested models when $\hat{\psi}_{S_{su}S_{sm}}$ was constrained to equality supports the invariance of this parameter across intervention groups, $\Delta\chi^2(1, N=4681) = 1.561$, $p = .211$, $\Delta CFI = 0$. The parallel-process model for smoking and sun protection behavior trajectories is shown in Figure 21 with standardized parameter estimates across intervention condition.

Study 2: Behavioral Stage Transitions

Latent Transition Analysis (LTA) was applied to examine patterns of stage transitions over time between mutually exclusive latent status subgroups (i.e. behavior stage). Stage transition models were developed to investigate progression over the 2-year interval through the discrete stages for changing individual cancer-related risk behaviors. For all models, behavior-specific SOC measured at baseline, 1- and 2-years served as indicators of stage membership at each time point. At baseline, three stage categories were represented, corresponding to the precontemplation, contemplation and preparation stages. Four discrete behavioral stages were represented in the models at 1- and 2-years, corresponding to precontemplation, contemplation, preparation, and action/maintenance stages. Measurement error for stage membership was assumed to be invariant over time, so threshold levels for SOC binary indicators were fixed at the same value to simulate a low level of measurement error.

Stage Membership Probabilities. Table 16 shows the stage membership probabilities at baseline, Year 1 and 2, estimated in baseline stage transition models (Figure 4) for each cancer-related risk behavior. Out of 2164 participants who were smokers at baseline, approximately a third (0.357) were in the precontemplation stage for smoking cessation, less than half (0.440) were in contemplation, and one-fifth (0.203) were prepared to quit smoking. After one year, approximately 13.5% of participants were estimated to have quit smoking based on membership in the action or maintenance stages. By the 2-year follow-up, the proportion of smokers who had reached the action or maintenance stage was 0.204.

Model estimated stage membership probabilities for unhealthy diet behavior show that at baseline, more than half (0.523) of the 6729 “at-risk” participants were in the precontemplation stage for changing eating behavior, 14.5% were in the contemplation stage, and approximately one third (0.331) were in the preparation stage. After one year, close to one quarter (0.234) of participants were estimated to have reached behavioral criteria for reducing dietary fat, based on membership in the action or maintenance stages. By the 2-year follow-up, the proportion who reached action or maintenance had increased slightly to 0.259.

For the 7065 participants who were at risk for sun exposure at baseline, model estimated stage membership probabilities show that approximately one-third (0.324) of participants were estimated to be in the precontemplation stage for sun protection at baseline, 23.5% were in the contemplation stage, with another 44.1% in the preparation stage. Less than one-fifth (0.169) of participants were estimated to have reached behavioral criteria for reducing sun exposure after one year, based on

membership in the action or maintenance stages. By the 2-year follow-up, the proportion who reported reaching behavioral criteria for reducing sun exposure was 0.311.

Two-year Stage Transition Patterns. For each risk behavior, the unconditional saturated baseline model (2A.1) of 2-year stage transition pattern was fitted in which all possible transition parameters were estimated. Three alternative models (2A.2, 2A.3, 2A.4) of possible 2-year stage transition patterns were also assessed. The models differed on the number of estimated stage transition parameters (paths); other transition paths were fixed to zero. The best-fitting unconditional stage transition model to the data was determined based on several fit indices, including lower values for the likelihood ratio G^2 , AIC, and BIC. The nested models were also evaluated using the scaled difference likelihood ratio test (ΔG^2). Model fit statistics for the baseline unconditional stage transition model and nested models representing alternative stage transition patterns for each risk behavior are presented in Table 17.

For smoking, the baseline unconditional stage transition model (Model 2A.1) which included all possible transition paths, provided the best fit to the data, $G^2(2024, N=2164) = 1095.539, p = 1.00$; AIC = 11876.95; BIC = 12007.59. Out of the four stage transition patterns assessed, the baseline model had the lowest likelihood ratio G^2 , AIC and BIC values, and G^2 less than model degrees of freedom (DF). Nested model comparisons found that restricting transition paths to only two stages forward and two stages backward (Model 2A.2) resulted in a significantly worse fitting model, $\Delta G^2(3, N=2164) = 460.929, p < .001$.

For unhealthy diet risk, the baseline unconditional stage transition model (Model 2A.1) was also confirmed to be the best-fitting model to the data, $G^2(2024, N=6729) = 3192.298, p < .001$; AIC = 41187.81; BIC = 41344.54. None of the unconditional stage transition models for diet behavior had G^2 values that were lower than model DF because of the large sample size. Nested model comparisons found that restricting transition paths to only two stages forward and two stages backward (Model 2A.2) for diet behavior resulted in a significantly worse fitting model, $\Delta G^2(3, N=6729) = 7540.782, p < .001$.

For sun exposure risk, the baseline unconditional stage transition model (Model 2A.1) was also confirmed to be the best-fitting model to the data, $G^2(2024, N=7062) = 3236.122, p < .001$; AIC = 42235.06; BIC = 42392.91. Due to the large sample size for sun exposure, none of the unconditional stage transition models had G^2 values that were lower than model DF. Nested model comparisons showed that restricting transition paths to only two stages forward and two stages backward (Model 2A.2) for sun exposure resulted significantly worse fitting model, $\Delta G^2(3, N=7065) = 1075.357, p < .001$.

Stationarity of Stage Transition Models. The stability over time of stage transition parameters estimated by the baseline unconditional stage transition model (Model 2A.1) was then assessed. For each cancer risk behavior, the corresponding stationary model was fitted, in which stage transition probability parameters (τ) were constrained to be equal across time intervals. Because only three behavioral stages (precontemplation, contemplation, and preparation) are represented in the first wave, the equality across time constraints were only specified for stage transition parameters

conditioned on those three stages; the transition parameters conditioned on action/maintenance stage from the second to third wave were freely estimated in the stationary model (Model 2A.1S).

Model fit statistics for the baseline (nonstationary) and stationary unconditional models for 2-year stage transitions are presented in Table 18 for each cancer risk behavior. The stationary stage transition model (Model 2A.1S) for smoking was more parsimonious, and provided a better fit to the data, $G^2(2033, N=2164) = 1105.618, p = 1.00$; AIC = 11869.033; BIC = 11948.549. Nested model comparisons found that constraining transition paths from precontemplation, contemplation and preparation to be equal over time did not negatively impact model fit, $\Delta G^2(9, N=2164) = 10.367, p = .081$.

For unhealthy diet risk, the more parsimonious stationary unconditional stage transition model (Model 2A.1S) was found to have higher AIC, but lower values of G^2 and BIC compared to the saturated baseline model, $G^2(2033, N=2164) = 3176.270, p < .001$; AIC = 41231.155; BIC = 41326.553. Nested model comparisons found that constraining transition paths for precontemplation, contemplation and preparation to be equal over time yielded $\Delta G^2(9, N=6729) = 63.102, p < .001$, which did not support the hypothesis of stationary stage transition parameters.

Compared to the unconditional baseline stage transition model for sun exposure risk, the stationary model (Model 2A.1S) was revealed to have lower values of G^2 , AIC and BIC: $G^2(2033, N=2164) = 3176.270, p < .001$; AIC = 41231.155; BIC = 41326.553. However, nested model comparisons found that constraining transition paths for precontemplation, contemplation and preparation to be equal over time

yielded scaled $\Delta G^2(9, N=7065) = 54.962, p < .001$, which may have achieved significance because of the large sample size. Therefore, the more parsimonious stationary model (Model 2A.1S) was retained for sun exposure.

Two-year Stage Transition Parameter Estimates. Stage transition parameters representing the probability of membership in stage B at time t , conditioned upon estimated membership in stage A at time $t-1$, were estimated for the two 1-year intervals for each cancer-related risk behavior. Table 19 shows the transition parameters estimated by the stationary unconditional stage transition model (Model 2A.1S) for smoking and sun exposure risks, and from the nonstationary unconditional model (Model 2A.1) for diet behavior. Values on the diagonal represent the probability of remaining in the same stage (stagnant) over one year, values to the right of the diagonal represent positive progress toward behavioral criteria for reduced risk over the same interval, and values to the left of the diagonal represent backward regression in readiness to reduce behavioral risk.

For smoking, transition parameter estimates suggest that across participants in precontemplation were more likely to remain in the same stage after one year (.65) than to make stage progress (.35). Participants in contemplation were also more likely to remain in the same stage after one year (.53) than to make stage progress (.29) or to regress in to precontemplation (.18). Participants in the preparation stage were estimated to be more likely to regress to the contemplation stage (.36) than to progress to action/maintenance (.23). Participants who reached the action/maintenance stage (quit smoking) by the first year had a high probability (.76) of staying quit.

For unhealthy diet risk behavior, participants in precontemplation at baseline were almost more likely to remain in the same stage (.54) than to make stage progress (.46). Participants in precontemplation after a year were even more likely to be stuck in that stage (.63). Similarly, stage transition parameters estimated in the stationary model for sun exposure risk indicate that participants in precontemplation were also much more likely to remain in the same stage (.67) over a year than to make progress (.33) in their readiness to adopt behavioral criteria. Finally, participants who reached the action or maintenance stage after the first year had a higher probability of maintaining their behavior change (.63).

Intervention Effect on Two-year Stage Transitions. Next, conditional stage transition models that included intervention condition as a time-invariant covariate (TICV) were fitted to the data for each risk behavior. The conditional stage transition models were nonstationary with all transition paths freely estimated. Model fit statistics for the conditional 2-year stage transition models for smoking, unhealthy diet, and sun exposure risk behaviors are presented in Table 20.

Model 2C.1 estimated baseline stage membership probabilities across intervention condition are shown in Table 21 for each behavior. For each risk behavior, the estimated stage membership probabilities at baseline were quite similar across intervention condition. Nested model comparisons found that constraining baseline stage distribution to be the same across intervention condition did not negatively impact stage transition model fit for each risk behavior. The effectiveness of baseline randomization of participants was confirmed for smoking, $\Delta G^2(2, N=2164) = 3.987, p = .136$; unhealthy diet, $\Delta G^2(2, N=6729) = 1.729, p = .421$; and sun

exposure, $\Delta G^2(2, N=7065) = 4.582, p = .101$. Comparing across risk behaviors reveals that participants at risk for unhealthy diet were much more likely to be in precontemplation (.52) at baseline, compared to baseline precontemplation membership probability of around .34 for smoking and .32 for sun exposure risks.

The conditional 2-year stage transition model for smoking with intervention condition included as a TICV fit the data well, $G^2(4048, N=2164) = 1119.882, p = 1.00, AIC = 14886.44, BIC = 15153.39$. Model estimated two year stage transition parameters for smoking risk are presented in Table 22 for the intervention condition relative to the assessment-only comparison condition. Model estimated stage transition probabilities for smoking cessation reveal that during the intervention period (first year), treated smokers had slightly higher probabilities of stage progress (.40|PC; .34|C; .30|PR) compared to those in the control group (.35|PC; .25|C; .20|PR). Over the second year, transition parameters representing stage progress were actually lower for treated smokers in the precontemplation (.26) and contemplation (.26) stages at Year 1 compared to controls (.35|PC; .30|C); although the probability of making progress from the preparation stage was higher in the intervention group (.25) than in the control group (.19). Smokers in the intervention group who quit smoking by the first year also had a higher probability of staying quit (.82) versus those in the comparison group (.69).

The conditional 2-year stage transition model for unhealthy diet risk with intervention condition included as a TICV fit the data well, $G^2(4048, N=6729) = 3424.337, p = 1.00; AIC = 50461.52; BIC = 50781.78$. Model estimated two year stage transition probabilities for unhealthy diet risk are presented in Table 23 for the

intervention condition relative to the assessment-only comparison condition. Model estimated transition probabilities indicate that compared to controls, treated participants generally had slightly higher probabilities of stage progress during the intervention period (first year), and in the second year follow-up period. Stage transition parameter estimates also reveal that intervention group participants who reached behavioral criteria for reduced dietary risk (action/maintenance stage) had slightly higher probability of maintaining (.53) the behavior change compared to controls (.50).

The conditional 2-year stage transition model for sun exposure risk with intervention condition included as a TICV also provided a good fit to the data:

$$G^2(4048, N=7065) = 3861.821, p = 1.00; AIC = 51877.87.52; BIC = 52200.43.$$

Examination of the stage transition parameter estimates for sun protection behavior (Table 24) revealed that stage progress probabilities were higher for the intervention group over both the first and second years. In addition, for those participants who reached the action/maintenance stage for sun protection by Year 1, the probability of maintaining behavioral criteria was higher in the intervention (.72) than control group (.51).

Comparison across cancer-risk behaviors. The two-year stage transition model estimating up to three stages forward and backward provided the best fit to the data across smoking, unhealthy eating and sun exposure risk behaviors. Model estimated baseline stage membership probabilities revealed some differences across behaviors. Almost one third of at-risk participants were in the precontemplation stage at baseline for smoking (.36) or sun exposure (.32), however, more than half of participants (.52)

at risk for unhealthy diet were in the same stage at baseline. Smokers were least prepared (.20) to change their behavior compared to participants at risk for unhealthy diet (.33) or sun exposure (.44).

Unconditional two year stage transition models supported the stationarity of transition paths for smoking cessation and sun protection behavioral stages, but not for diet behavior. For each risk behavior, the highest estimated stage transition probabilities for the first interval were for staying in the precontemplation stage (>.50) instead of making stage progress. Participants still in the precontemplation stage after the first year were again more likely (> .62) to remain in precontemplation over the second interval.

Across three risk behaviors, TTM-tailored intervention demonstrated positive effects during the first year in terms of moving at-risk participants to reach behavioral criteria (reaching action/maintenance stage), and also in increasing readiness to reduce behavioral risk (making forward stage progress) even if they had not yet taken effective action. Over the second (post-intervention follow-up) interval, transition parameters representing positive stage progress, including reaching the action/maintenance stage, were also higher in the intervention condition for diet and sun protection behaviors. During the post-intervention interval, estimated stage progress probabilities were higher in the control group smokers who were still in precontemplation or contemplation at Year 1. Finally, intervention group participants who had reached behavioral criteria for reducing health risk by Year 1 had higher probabilities of maintaining their behavior change compared to controls.

CHAPTER 4

DISCUSSION

The current research applied two alternative latent variable modeling approaches to investigate mechanisms of single and multiple behavior change across three of the most important behaviors for cancer prevention: smoking, unhealthy diet, and risky sun exposure. In Study 1, LGC modeling approaches were employed to systematically examine and compare 2-year growth trajectories of behavioral outcomes for each individual risk, and jointly across pairs of co-occurring behavioral risks. Study 2 applied LTA techniques to model 2-year stage transition patterns for each of the three cancer-related risk behaviors. Both studies also examined TTM-tailored intervention effects on behavior change over time, in growth trajectories and stage transitions within each behavior.

Study 1 results supported linear trends in 2-year growth trajectories of quantitative outcomes for all three behaviors. Smoking behavior was found to decrease slightly over time across all participants; however, the large estimated variance in the slope indicated large variability between individuals. The positive covariance between the intercept and slope factors for smoking suggested that individuals who initially smoked more (greater problem severity) had more difficulty reducing smoking behavior, whereas those who smoked less at baseline were able to achieve a greater reduction in their number of cigarettes smoked, which is consistent with previous research findings that behavior change at 24-months was related to

problem severity (Blissmer et al., 2010). Healthy eating behavior and sun protective behavior were also found to increase slightly over time across all participants, with significant inter-individual variation in both the initial levels on the behavioral measures (DBQ or SPBI) as well as the rate of increase (slope) of the behaviors over time. Interestingly, the covariance between the intercept and slope factors for diet behavior was not significant, suggesting that the rate of change in diet behavior was unrelated to initial DBQ scores. For sun protection behavior, the significant but small estimated covariance between intercept and slope factors indicated that higher baseline levels on the SPBI were only weakly associated with steeper increases over time in sun protective behavior.

TTM-tailored intervention had a significant and positive effect on increasing both healthy eating behavior and sun protective behavior over time, consistent with previously reported outcomes (Prochaska et al., 2004, 2005; Weinstock, Rossi, Redding & Maddock, 2002). Healthy eating and sun protection behavioral outcomes at the 2-year follow-up were approximately 0.25 of a standard deviation higher ($\hat{d} = 0.23$ for diet and $\hat{d} = 0.25$ for sun protection) in the treatment group. This could be interpreted as medium-to-large intervention effects on the rate of increase in healthy eating and sun protection (Rossi, 2013). However in contrast, the intervention did not appear to have a significant effect on the mean slope for smoking behavior. This is not what was expected based on numerous studies that found significant intervention effects on point prevalence abstinence for smoking cessation (Prochaska et al, 1993, 2001a, 2001b; Velicer et al, 1999, 2006a, 2006b). One key consideration to keep in mind is that the behavioral outcome measure for smoking examined in the current

study is based on the number of cigarettes smoked per day, whereas previous studies assessed intervention efficacy in terms of cessation, a dichotomous outcome that does not differentiate between smoking two or 20 cigarettes/day in smokers who were not unsuccessful in quitting. Another possible explanation may be that individuals who smoked fewer cigarettes daily (lower problem severity) were more likely to be able to quit smoking, whereas those who smoked more were less likely to change their smoking behavior (Blissmer et al., 2010).

The TTM constructs of Pros, Cons, and Situational Temptations/Self-efficacy were also assessed as baseline predictors of growth trajectories. Baseline Temptations and Pros were found to significantly predict initial smoking behavior, which was not surprising as Temptations can also serve as an indicator of smoking problem severity. When assessed simultaneously in LGCM with multiple time-invariant covariates, baseline Cons were also found to be significant predictors of mean slope for healthy eating and sun protective behaviors.

Parallel-process LGC models were developed to examine inter-relationships between growth trajectories within pairs of co-occurring risk behaviors. For the series of parallel-process LGC models, we were primarily interested in the covariance parameters estimated between the parallel behavior trajectories for each risk dyad. Previous multiple behavior change research described the phenomenon of “co-action,” observed within the context of co-occurring behavioral risk pairs, in which taking action on one behavior was associated with increased odds of successful action on the second behavior (Paiva et al., 2012; Yin et al., 2013). In the interest of understanding the process of multiple behavior change, examining the association between parallel

behavior trajectories could provide some insight into whether and how co-occurring risk behaviors change together over time.

Unconditional parallel-process LGCM for the smoking and diet risk dyad fitted to data from the full sample found that higher initial levels of smoking were significantly associated with lower initial DBQ mean scores (i.e., less healthy diet behavior), but that the rate of decrease in smoking behavior was not consistently related to the rate of increase in healthy eating. For the smoking and sun protection behavior dyad, higher initial levels of smoking were significantly associated with lower initial levels of sun protection behavior (lower SPBI mean scores), although the rate of decrease in smoking behavior was weakly and not significantly related to the rate of increase in sun protection behavior. Sun protection and diet was the only behavior pair shown to have significant covariances between parallel behavior trajectories. In the full sample, higher initial levels of healthy eating were associated with higher initial levels of sun protection behavior and the rates of increase in sun protection and healthy eating behaviors were also consistently related. Multiple-sample analyses of the parallel-process LGC model for diet and sun protection across intervention condition revealed significantly steeper increases (slope) over time in each behavior in the treatment group. Although the initial levels for both behaviors were more strongly related in the treatment group compared to controls, no significant intervention group difference was found for the association between slope factors for diet and sun protection behaviors. Therefore, previous research findings of significant co-action in the treatment group for the sun protection-diet behavior pair (Paiva et al., 2013) are more likely explained by the effect of TTM-tailored interventions on

increasing the rate of change (slope) for each treated behavior, which we also observed in the single behavior conditional LGC models.

Study 2 employed LTA techniques to examine behavioral stage transitions in three cancer-related risks over time, intervention condition, and across behaviors. For all three behaviors, the saturated stage movement model that freely estimated all possible transition paths was preferred over more restrictive models that constrained movement patterns to two or fewer stages. These findings indicate that a lot of stage movement is possible, and does occur. Given that the duration between assessment time points was one year in the data, finding this amount of stage movement over such a long interval was not unexpected, and also consistent with previous research that found movement of up to four stages in smokers when assessments were taken at intervals of one year (Schumann, John, Rumpf, Hapke & Meyer, 2006). Longitudinal data that include more frequent assessment time points (e.g. intervals of 3-6 months instead of 12-month intervals) would provide better resolution to study the stage transition process, and may even support a more parsimonious model of stage movement patterns. For example, other research with smokers assessed at shorter intervals of 6-months favored a more restricted transition model with a two-stage forward, one-stage back movement pattern (Martin et al., 1996). In the present study, examination of the transition parameters revealed that the transition paths on or closest to the diagonal (of the parameter estimate matrix) had the highest probabilities, while those paths furthest from the diagonal generally had very much lower (although non-zero) probabilities. This tells us that over a one year interval, either no stage movement or one-stage movement was much more likely to occur compared to greater

movement over multiple stages within the same interval.

Based on data for the full analytical sample combining intervention and control group participants, the stationary unconditional stage transition model was supported for smoking cessation, and was also preferred over the nonstationary model for sun protection behavior based on better overall model fit. For healthy diet behavior, the nonstationary model was shown to fit significantly better (based on significant scaled difference ΔG^2), although the overall G^2 and AIC both favored the more parsimonious stationary model. It is possible that the power of the scaled likelihood ratio difference test was amplified due to the large sample size. These findings suggest that stage movement patterns may be reasonably stable over time for the different behaviors.

In the matrix of transition parameter estimates, values along the diagonal represent stage membership stability (the probability of remaining stagnant in the same stage over one year), values to the right of the diagonal represent stage progression (toward behavioral criteria for reduced risk) over the same interval, and values to the left of the diagonal represent backward regression in readiness to reduce behavioral risk. From a health promotion and cancer prevention perspective, we would hope to see higher transition probabilities for stage progression, along with low to zero probabilities for stage regression. We would also prefer to see lower values along the diagonal, except for the bottom-right cell along the diagonal, where higher values (of $\tau_{AM|AM}$) indicate successfully maintaining behavior change at follow-up for those who took effective action during the first year. For all three behaviors, inspection of the unconditional model estimated transition parameters revealed that precontemplation stage membership was the most stable: participants in precontemplation were more

likely to remain in that stage than to make any progress in readiness to reduce their behavioral risk.

For all three behaviors, conditional stage transition models that included intervention condition as a time-invariant covariate supported the efficacy of TTM-tailored intervention. Transition parameter estimates indicated that treated at-risk participants were more likely to reach behavioral criteria (move to action/maintenance stage), and to maintain their behavior change during follow-up, which was consistent with previous study outcomes (Prochaska et al., 2004, 2005; Velicer et al., 2004; Weinstock, Rossi, Redding & Maddock, 2002). In addition, treated participants were more likely to increase their readiness to reduce behavioral risk (make forward stage progress) even if they had not taken effective action. These effects were observed across all stages and both time intervals for both diet and sun protection behaviors, however, the pattern of effects looked slightly different for smoking cessation. Although treatment effects were observed during the first year (intervention period) on overall stage progress and especially on quitting smoking, during the follow-up interval, transition parameters indicated more stage progress among controls in precontemplation and contemplation. During the follow-up, transition parameter estimates showed that treated smokers were more likely to move from preparation to action/maintenance stage (point prevalence abstinence), and also to maintain their quit status. Probing of the transition parameter estimates reveals an interesting pattern of effects that suggests that treatment may accelerate smoking cessation stage progress, with some possible “catching-up” by controls during follow-up, but the net outcome is still higher smoking cessation rates at follow-up in favor of treatment.

Limitations

One of the strengths of the current research is that the findings are based on data for participants pooled across multiple large randomized trials, and the data also include three of the most important behaviors for cancer prevention. However, one major limitation of this study was that the data only included three common assessment time points, at intervals of one year between each wave, thus restricting the growth trajectory or stage transition patterns that could be modeled. Data that include more frequent and shorter intervals between assessments would most likely provide a richer framework for investigating the process of behavior change. A second limitation of this study was the restricted range in the data because the sample consisted entirely of individuals identified to be “at-risk” for one or more cancer risk behavior at baseline, and individuals identified to be in action or maintenance stages at baseline were not assessed for the specific behavior at any follow-up time point. Although a sample of participants in all stages of change at baseline would provide greater variance in responses on outcome measures, it may not be representative of intervention populations and thus difficult to justify from a cost perspective. Another limitation of the current sample relates to the racial and ethnic demographics. A sample that is more diverse in terms of racial identity, with adequate numbers of other racial groups besides white and black, would support assessment of stability for growth trajectory and/or stage transition models across racial/ethnic identity subgroups, potentially improving the generalizability of these findings.

Perhaps one current limitation specific to the mixture modeling (LTA) approach lies with the number and type of fit criteria available for assessing model fit.

The likelihood ratio G^2 (based on the log likelihood) and information criteria such as the AIC and BIC are more commonly used to assess model fit, however, interpretation of these indices are relative as they do not have any absolute or theoretical limits for “perfect” fit, preventing comparison of non-nested models.

Additional avenues for future research could include evaluating plausible covariates such as number of co-occurring behavioral risks as predictors of growth trajectories or stage transitions. In addition, indicators for different behaviors could be used to identify a multiple behavior risk status, in order to model stage transitions over time in multiple behaviors jointly.

Summary

The current research employed two alternative latent variable modeling approaches to investigate mechanisms of single and multiple behavior change across three cancer-related risk behaviors of smoking, unhealthy eating, and sun exposure. Study 1 applied LGC modeling approaches to examine 2-year growth trajectories of quantitative outcomes for all three behaviors. Conditional LGC models supported significant TTM-intervention effects on increasing the rates of change over time for healthy eating and sun protection behaviors. Parallel-process LGC models developed to estimate 2-year growth trajectories jointly across behaviors within co-occurring risk pairs provided evidence that initials levels were significantly correlated between both behaviors in each risk dyad. In addition, for the sun protection and diet behavior pair, the rate of increase over time was shown to be associated between both behaviors in the pair.

Study 2 demonstrated the applicability and usefulness of using LTA to elucidate how individuals change in their behavioral risks under intervention, by describing transition patterns in discrete stages over time. Unlike typical approaches to assessing intervention effectiveness that compare final study outcomes based on the number or proportion of participants that reach criteria (e.g. quit smoking) at a single point in time, the longitudinal nature of latent transition models allow us to study the process of behavior change. Results of this study supported the viability of the baseline stage movement pattern model (Figure 5, Model 1) across the three cancer risk behaviors, in which stage movements over each one year interval could be described by stability and progression and regression of one-to-three stages. Study 2 results also provided evidence for TTM-tailored intervention efficacy across smoking cessation, dietary fat reduction, and sun protection behavior, in terms of moving at-risk participants to reach behavioral criteria, promoting stage progress among those who did not reach criteria, and in maintaining successful behavior change during the follow-up interval. In addition, our findings revealed the stability of precontemplation stage membership across all three behaviors; stage progress from the precontemplation stage was even less likely among control participants.

TABLES

Table 1. Characteristics of participants across three samples of adults.

	<u>Sample Identification</u>			
	Parents (<i>N</i> = 2402)	Patients (<i>N</i> = 5284)	Employees (<i>N</i> = 1836)	Combined (<i>N</i> = 9522)
Characteristic	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Age (years)	42.5 (5.5)	46.0 (13.0)	43.3 (10.2)	44.6 (11.2)
Body Mass Index (kg/m ²)	25.2 (4.7)	25.8 (5.1)	26.2 (4.8)	25.7 (4.9)
Education (years)	14.0 (3.2)	14.5 (3.2)	14.0 (3.3)	14.3 (3.29)
	% with characteristic	% with characteristic	% with characteristic	% with characteristic
Female	75.3	69.8	47.3	66.8
White	92.2	96.8	92.8	94.8
Hispanic	3.4	1.3	3.1	2.2
Employed	83.9	76.8	99.0	70.9
Married	76.9	68.0	71.2	47.3
Perceived general health as “Very good” or “Excellent”	75.3	69.8	47.3	47.3

Table 2. Stage of change distribution for smoking cessation, healthy eating, and sun protection at baseline.

Behavior	Stage of change	<u>Sample Identification</u>							
		Parents (1 st)		Patients		Worksite		Combined	
		(N = 2402)		(N = 5284)		(N = 1836)		(N = 9522)	
		N	%	N	%	N	%	N	%
<i>Smoking</i>	PC	281	11.7	355	6.7	136	7.4	772	8.1
	C	278	11.6	537	10.2	136	7.4	951	10.0
	PR	118	4.9	266	5.0	57	3.1	441	4.6
	A	72	3.0	176	3.3	51	2.8	299	3.1
	M	718	29.9	1727	32.7	594	32.4	3039	31.9
	Nonsmoker	935	38.9	2223	42.1	862	46.9	4020	42.2
<i>Diet</i>	PC	970	40.4	1921	36.4	626	34.1	3517	36.9
	C	257	10.7	498	9.4	227	12.4	982	10.3
	PR	557	23.2	1213	23.0	460	25.1	2230	23.4
	A	43	1.8	96	1.8	39	2.1	178	1.9
	M	575	23.9	1556	29.4	484	26.4	2615	27.5
<i>Sun</i>	PC	650	27.1	1222	23.1	420	22.9	2292	24.1
	C	363	15.1	918	17.4	379	20.6	1660	17.4
	PR	789	32.8	1689	32.0	635	34.6	3113	32.7
	A	14	0.6	27	0.5	8	0.4	49	0.5
	M	586	24.4	1428	27.0	394	21.5	2408	25.3

Note: PC = Precontemplation, C = Contemplation, PR = Preparation, A = Action, M = Maintenance.

Table 3. Mean & SD number of cigarettes smoked per day across stage of change at baseline.

Timepoint	Stage of change	N	Mean	SD	ANOVA & Tukey test results	η^2
<i>Baseline</i>	PC	768	18.67	11.47	$F(2, 2147) = 18.08^{***}$	.017
	C	948	17.85	11.59	PC, C > PR	
	PR	440	14.18	10.22		

Note: PC = Precontemplation, C = Contemplation, PR = Preparation, A = Action, M = Maintenance;
 $^{***} p < .001$.

Table 4. Mean & SD Diet Behavioral Questionnaire scores across stage of change at baseline.

Timepoint	Stage of change	N	Mean	SD	ANOVA & Tukey test results	η^2
<i>Baseline</i>	PC	3472	3.23	0.56	$F(2, 6642) = 55.36^{***}$ PR > PC, C	.016
	C	973	3.27	0.51		
	PR	2206	3.40	0.50		

Note: PC = Precontemplation, C = Contemplation, PR = Preparation, A = Action, M = Maintenance;
 $^{***} p < .001$.

Table 5. Mean & SD Sun Protection Behavior Index scores across stage of change at baseline.

Timepoint	Stage of change	<i>N</i>	Mean	SD	ANOVA & Tukey test results	η^2
<i>Baseline</i>	PC	2284	2.46	0.76	$F(2, 7012) = 2125.05^{***}$ PR > C > PC	.377
	C	1645	2.72	0.62		
	PR	3092	3.51	0.33		

Note: PC = Precontemplation, C = Contemplation, PR = Preparation, A = Action, M = Maintenance;
 $^{***} p < .001$.

Table 6. Descriptive statistics for number of cigarettes/day at baseline, 1- and 2-years.

Cigarettes/day	Timepoint	N	Mean	SD	Min	Max	Skewness	Kurtosis
<i>Count</i>	Baseline	2117	17.72	11.25	1	99	2.07	9.87
	Year 1	1395	18.30	18.02	0	99	2.62	8.88
	Year 2	1259	17.49	18.59	0	99	2.51	8.25
<i>Square-root transformed count</i>	Baseline	2117	4.01	1.28	1.00	9.95	0.35	1.40
	Year 1	1395	3.75	2.05	0.00	9.95	0.24	1.16
	Year 2	1259	3.51	2.27	0.00	9.95	0.17	0.37

Table 7. Descriptive statistics for Diet Behavioral Questionnaire mean score at baseline, 1- and 2-years.

Timepoint	<i>N</i>	Mean	SD	Min	Max	Skewness	Kurtosis
Baseline	6651	3.30	0.54	1.27	4.82	−0.35	−0.13
Year 1	5076	3.41	0.57	1.32	4.95	−0.31	−0.07
Year 2	4722	3.43	0.58	1.32	4.95	−0.27	−0.14

Note: Diet Behavioral Questionnaire mean score was computed for each participant based a minimum of 20 non-missing item responses.

Table 8. Descriptive statistics for Sun Protection Behavior Index mean score at baseline, 1- and 2-years.

Timepoint	<i>N</i>	Mean	SD	Min	Max	Skewness	Kurtosis
Baseline	7021	2.98	0.75	1.00	5.00	−0.58	−0.18
Year 1	5233	3.23	0.80	1.00	5.00	−0.32	−0.17
Year 2	4882	3.28	0.81	1.00	5.00	−0.36	−0.17

Table 9. Model fit statistics for unconditional 2-year latent growth trajectories by cancer risk behavior.

Behavior	Model 1A	Latent Growth Curve Model	DF	χ^2	CFI	NNFI	RMSEA	90% CI
<i>Smoking</i>	1	Null model	6	911.205	0.000	0.456	0.267	[0.252 , 0.282]
	2	Random intercept	5	323.708	0.617	0.770	0.174	[0.158 , 0.190]
	3	Fixed intercept, fixed slope	5	849.119	0.000	0.392	0.282	[0.267 , 0.299]
	4	Random intercept, fixed slope	4	215.976	0.745	0.809	0.158	[0.141 , 0.177]
	5	Random intercept and slope	2	24.218	0.973	0.960	0.072	[0.048 , 0.100]
	6	Random intercept and slope, uncorrelated	3	32.477	0.965	0.965	0.068	[0.048 , 0.090]
<i>Unhealthy diet</i>	1	Null model	7	8542.992	0.000	0.561	0.428	[0.421 , 0.436]
	2	Random intercept	6	727.666	0.913	0.957	0.134	[0.126 , 0.143]
	3	Fixed intercept, fixed slope	6	8371.581	0.000	0.498	0.458	[0.450 , 0.466]
	4	Random intercept, fixed slope	5	363.132	0.957	0.974	0.104	[0.095 , 0.113]
	5	Random intercept and slope	3	169.793	0.980	0.980	0.091	[0.080 , 0.103]
	6	Random intercept and slope, uncorrelated	4	169.992	0.980	0.985	0.079	[0.069 , 0.089]
<i>Sun exposure</i>	1	Null model	7	7683.913	0.000	0.539	0.395	[0.388 , 0.403]
	2	Random intercept	6	1445.106	0.798	0.899	0.185	[0.177 , 0.193]
	3	Fixed intercept, fixed slope	6	7252.473	0.000	0.493	0.415	[0.407 , 0.423]
	4	Random intercept, fixed slope	5	467.938	0.935	0.961	0.115	[0.106 , 0.124]
	5	Random intercept and slope	3	268.225	0.963	0.963	0.112	[0.101 , 0.124]
	6	Random intercept and slope, uncorrelated	4	275.816	0.962	0.971	0.098	[0.089 , 0.108]

Note: DF = Degrees of freedom; CFI = Comparative Fit Index; NNFI = Non-Normed Fit Index; RMSEA = Root mean square error of approximation.

Table 10. Model fit statistics for conditional 2-year latent growth trajectories by cancer risk behavior with intervention condition included as a time-invariant covariate.

Behavior	Model 1B	Latent Growth Curve Model	DF	χ^2	CFI	NNFI	RMSEA	90% CI
<i>Smoking</i>	1	Null model	9	920.689	0.000	0.276	0.219	[0.207 , 0.231]
	2	Random intercept	7	324.769	0.621	0.675	0.146	[0.133 , 0.160]
	3	Fixed intercept, fixed slope	8	858.603	0.000	0.240	0.224	[0.212 , 0.237]
	4	Random intercept, fixed slope	6	216.047	0.750	0.750	0.129	[0.114 , 0.144]
	5	Random intercept and slope	3	24.078	0.975	0.950	0.058	[0.038 , 0.080]
<i>Unhealthy diet</i>	1	Null model	10	8677.717	0.000	0.385	0.361	[0.355 , 0.367]
	2	Random intercept	8	827.024	0.903	0.927	0.124	[0.117 , 0.131]
	3	Fixed intercept, fixed slope	9	8506.305	0.000	0.330	0.377	[0.370 , 0.384]
	4	Random intercept, fixed slope	7	458.37	0.947	0.954	0.098	[0.091 , 0.106]
	5	Random intercept and slope	4	177.126	0.980	0.969	0.081	[0.071 , 0.091]
	6	Random intercept and slope, uncorrelated	5	177.131	0.980	0.976	0.072	[0.063 , 0.081]
<i>Sun exposure</i>	1	Null model	10	7822.194	0.000	0.356	0.334	[0.327 , 0.340]
	2	Random intercept	8	1540.561	0.789	0.842	0.165	[0.158 , 0.172]
	3	Fixed intercept, fixed slope	9	7390.755	0.000	0.324	0.342	[0.335 , 0.348]
	4	Random intercept, fixed slope	7	555.426	0.925	0.935	0.106	[0.098 , 0.113]
	5	Random intercept and slope	4	270.621	0.963	0.945	0.097	[0.088 , 0.170]
	6	Random intercept and slope, uncorrelated	5	276.346	0.963	0.955	0.088	[0.079 , 0.097]

Note: DF = Degrees of freedom; CFI = Comparative Fit Index; NNFI = Non-Normed Fit Index; RMSEA = Root mean square error of approximation.

Table 11. Descriptive statistics for baseline Pros, Cons, and Situational Temptations/Self-efficacy across cancer risk behaviors.

Behavior	TTM Construct	Mean	SD	Skewness	Kurtosis
<i>Smoking</i>	Pros	2.72	0.87	0.30	-0.21
	Cons	3.69	0.90	-0.54	-0.26
	Temptations	3.36	0.73	-0.09	-0.20
<i>Unhealthy eating</i>	Pros	2.28	1.08	0.61	-0.46
	Cons	2.26	0.97	0.55	-0.29
	Temptations	2.66	0.78	0.00	-0.38
<i>Sun exposure</i>	Pros	3.64	0.91	-0.66	0.07
	Cons	2.78	0.99	0.06	-0.66
	Self-efficacy	2.68	0.86	-0.07	-0.37

Table 12. Model fit statistics for conditional 2-year latent growth trajectories by cancer risk behavior including intervention condition and baseline TTM constructs as time-invariant covariates.

Behavior	Model 1C	Time-Invariant Covariate(s)	DF	χ^2	CFI	NNFI	AIC	RMSEA	90% CI
<i>Smoking</i>	1	TRT	12	554.334	0.601	0.502	35593.083	0.146	[0.136 , 0.157]
	2	TRT, Pros	10	342.080	0.756	0.634	35384.828	0.125	[0.114 , 0.137]
	3	TRT, Cons	10	549.955	0.603	0.405	35592.703	0.160	[0.148 , 0.171]
	4	TRT, Temptations	10	42.816	0.976	0.964	35085.565	0.039	[0.028 , 0.052]
	5	TRT, Pros, Cons, Temptations	6	24.355	0.987	0.966	35075.104	0.038	[0.023 , 0.054]
<i>Unhealthy diet</i>	1	TRT	14	595.954	0.934	0.930	79077.914	0.079	[0.074 , 0.085]
	2	TRT, Pros	12	594.041	0.934	0.918	79080.001	0.085	[0.080 , 0.091]
	3	TRT, Cons	12	311.384	0.966	0.958	78797.345	0.061	[0.055 , 0.067]
	4	TRT, Temptations	12	595.921	0.934	0.918	79081.882	0.086	[0.080 , 0.091]
	5	TRT, Pros, Cons, Temptations	8	179.158	0.981	0.964	78673.118	0.057	[0.050 , 0.064]
<i>Sun exposure</i>	1	TRT	13	5262.511	0.572	0.506	95327.181	0.240	[0.234 , 0.245]
	2	TRT, Pros	11	3211.920	0.739	0.644	93280.590	0.204	[0.198 , 0.210]
	3	TRT, Cons	11	4713.203	0.616	0.477	94781.874	0.247	[0.241 , 0.253]
	4	TRT, Self-efficacy	11	1165.510	0.906	0.872	91234.180	0.122	[0.116 , 0.128]
	5	TRT, Pros, Cons, Self-efficacy	7	335.447	0.973	0.973	90412.118	0.082	[0.074 , 0.089]

Note: DF = Degrees of freedom; TRT = Intervention condition; CFI = Comparative Fit Index; NNFI = Non-Normed Fit Index; RMSEA = Root mean square error of approximation; Intercept and slope factors are uncorrelated within diet behavior.

Table 13. Prediction of 2-year latent growth trajectories by cancer risk behavior

Behavior	Model	Time-Invariant Covariate(s)	Intercept			Slope		
			$\hat{\gamma}_I$	SE		$\hat{\gamma}_S$	SE	
<i>Smoking</i>	1C.1	TRT	−0.154	0.056	**	−0.057	0.057	
	1C.5	TRT	−0.088	0.049		−0.061	0.057	
		Pros	0.130	0.034	***	0.016	0.040	
		Cons	−0.046	0.028		−0.017	0.032	
		Temptations	0.744	0.040	***	−0.076	0.049	
<i>Unhealthy diet</i>	1C.1	TRT	0.030	0.013	*	0.063	0.006	***
	1C.5	TRT	0.030	0.013	*	0.064	0.006	***
		Pros	0.048	0.007	***	0.001	0.003	
		Cons	−0.162	0.008	***	0.010	0.004	*
		Temptations	0.074	0.009	***	−0.006	0.005	
<i>Sun exposure</i>	1C.1	TRT	0.047	0.018	**	0.094	0.009	***
	1C.5	TRT	0.002	0.013		0.095	0.009	***
		Pros	0.203	0.009	***	−0.012	0.006	
		Cons	−0.146	0.007	***	0.018	0.005	***
		Self-efficacy	0.401	0.009	***	−0.001	0.007	

Note: TRT = Intervention condition; $\hat{\gamma}_I$ and $\hat{\gamma}_S$ are unstandardized regression coefficients;
 * $p < .05$; ** $p < .01$; *** $p < .001$.

Table 14. Model fit statistics for unconditional parallel-process 2-year latent growth trajectories in risk behavior dyads.

Behavior dyad	Model 1D	Latent Growth Curve Model	DF	χ^2	CFI	NNFI	RMSEA	90% CI
<i>Smoking & Unhealthy diet</i>	1	Null model	22	2413.572	0.000	0.295	0.270	[0.261 , 0.279]
	2	Random intercepts	19	336.2	0.863	0.892	0.106	[0.096 , 0.116]
	3	Fixed intercepts, fixed slopes	18	1910.794	0.181	0.318	0.265	[0.255 , 0.275]
	4	Random intercepts, fixed slopes	17	212.517	0.915	0.925	0.088	[0.077 , 0.098]
	5	Random intercepts and slopes	11	40.789	0.987	0.982	0.043	[0.029 , 0.057]
	6	Random intercepts and slopes, intercepts and slopes uncorrelated across behaviors	13	42.710	0.987	0.985	0.039	[0.026 , 0.052]
<i>Unhealthy diet & Sun exposure</i>	1	Null model	23	11486.459	0.000	0.321	0.326	[0.321 , 0.331]
	2	Random intercepts	20	1392.752	0.875	0.906	0.121	[0.116 , 0.127]
	3	Fixed intercepts, fixed slopes	19	9482.018	0.141	0.321	0.326	[0.321 , 0.332]
	4	Random intercepts, fixed slopes	18	615.073	0.946	0.955	0.084	[0.079 , 0.090]
	5	Random intercepts and slopes	12	299.665	0.974	0.967	0.072	[0.065 , 0.079]
	6	Random intercepts and slopes, intercepts and slopes uncorrelated across behaviors	14	303.242	0.974	0.972	0.066	[0.060 , 0.073]
<i>Sun exposure & Smoking</i>	1	Null model	22	2156.555	0.000	0.275	0.254	[0.245 , 0.264]
	2	Random intercepts	19	475.519	0.772	0.820	0.127	[0.117 , 0.137]
	3	Fixed intercepts, fixed slopes	18	1597.658	0.213	0.344	0.242	[0.232 , 0.252]
	4	Random intercepts, fixed slopes	17	267.228	0.875	0.890	0.099	[0.089 , 0.110]
	5	Random intercepts and slopes	10	71.649	0.969	0.954	0.064	[0.051 , 0.078]
	6	Random intercepts and slopes, intercepts and slopes uncorrelated across behaviors	12	77.913	0.967	0.959	0.061	[0.048 , 0.074]

Note: DF = Degrees of freedom; CFI = Comparative Fit Index; NNFI = Non-Normed Fit Index; RMSEA = Root mean square error of approximation; Intercept and slope factors are uncorrelated within diet behavior.

Table 15. Invariance model fit statistics for unconditional parallel-process 2-year latent growth trajectories in risk behavior dyads assessed across intervention condition.

Behavior dyad	Model 1E	Invariance Model	DF	χ^2	$\Delta\chi^2$	CFI	Δ CFI	NNFI	RMSEA	90% CI
<i>Smoking & Unhealthy diet</i>	1	Equal forms	23	44.342	—	0.991	—	0.988	0.035	[0.019 , 0.051]
	2	Equal slope factor means	25	59.289	14.947 ***	0.985	0.006	0.982	0.043	[0.029 , 0.057]
	3	Equal slope factor means and covariance	26	62.000	2.711	0.984	0.001	0.982	0.043	[0.029 , 0.057]
	4	Equal slope factor means and factor covariances	27	62.037	0.037	0.985	0.001	0.983	0.042	[0.028 , 0.055]
<i>Unhealthy diet & Sun exposure</i>	1	Equal forms	24	243.700	—	0.980	—	0.975	0.063	[0.056 , 0.070]
	2	Equal slope factor means	26	329.649	85.949 ***	0.972	0.008	0.968	0.071	[0.064 , 0.078]
	3	Equal slope factor means and covariance	27	330.620	0.971	0.972	0.000	0.969	0.069	[0.063 , 0.076]
	4	Equal slope factor means and factor covariances	28	343.751	13.131 ***	0.971	0.001	0.969	0.069	[0.063 , 0.076]
<i>Sun exposure & Smoking</i>	1	Equal forms	21	62.934	—	0.979	—	0.970	0.052	[0.037 , 0.067]
	2	Equal slope factor means	23	72.758	9.824 **	0.975	0.004	0.968	0.054	[0.040 , 0.068]
	3	Equal slope factor means and covariance	24	74.319	1.561	0.975	0.000	0.969	0.053	[0.040 , 0.067]
	4	Equal slope factor means and factor covariances	25	74.396	0.077	0.975	0.000	0.970	0.051	[0.038 , 0.065]

Note: DF = degrees of freedom; CFI = Comparative Fit Index; NNFI = Non-Normed Fit Index; RMSEA = Root mean square error of approximation; Intercept and slope factors are uncorrelated within diet behavior; $\Delta\chi^2$ and Δ CFI computed between nested models for each invariance constraint; for $\Delta\chi^2$, * $p < .05$; ** $p < .01$; *** $p < .001$.

Table 16. Model estimated stage membership probabilities by cancer risk behavior.

Behavior	Time point	PC	C	PR	A/M
<i>Smoking</i>	Year 0 (Baseline)	0.357	0.440	0.203	—
	Year 1	0.328	0.384	0.160	0.127
	Year 2	0.304	0.344	0.146	0.206
<i>Unhealthy diet</i>	Year 0	0.523	0.145	0.331	—
	Year 1	0.404	0.164	0.198	0.234
	Year 2	0.404	0.148	0.189	0.259
<i>Sun exposure</i>	Year 0	0.324	0.235	0.441	—
	Year 1	0.354	0.320	0.158	0.169
	Year 2	0.356	0.141	0.192	0.311

Note: PC = Precontemplation; C = Contemplation; PR = Preparation; A/M = Action/ Maintenance.

Table 17. Model fit statistics for 2-year stage transition patterns by cancer risk behavior.

Behavior	Model 2A	Stage Transition Pattern	DF	LL	G ²	ΔG ²	AIC	BIC
<i>Smoking</i>	1	Baseline model, all transition paths free	2024	-5915.477	1095.539	—	11876.954	12007.588
	2	Two-forward, two-backward	2027	-6145.857	1473.318	460.929 ***	12331.715	12445.309
	3	Two-forward, one-backward	2030	-6267.977	1719.116	704.577 ***	12569.954	12666.510
	4	One-forward, one-backward	2034	-7014.808	2567.816	2194.755 ***	14055.617	14129.453
<i>Unhealthy diet</i>	1	Baseline model, all transition paths free	2024	-20570.905	3192.298	—	41187.810	41344.536
	2	Two-forward, two-backward	2027	-24288.259	3565.696	7540.782 ***	48616.517	48752.801
	3	Two-forward, one-backward	2030	-26550.011	4096.880	12087.956 ***	53134.021	53249.862
	4	One-forward, one-backward	2034	-29490.592	5621.663	17810.165 ***	59007.184	59095.768
<i>Sun exposure</i>	1	Baseline model, all transition paths free	2024	-21094.532	3236.122	—	42235.064	42392.911
	2	Two-forward, two-backward	2027	-21635.132	4118.344	1075.357 ***	43310.264	43447.523
	3	Two-forward, one-backward	2030	-23805.414	4707.131	5392.107 ***	47644.828	47761.497
	4	One-forward, one-backward	2034	-25519.856	5845.748	8809.245 ***	51065.713	51154.931

Note: DF = degrees of freedom; LL = log likelihood; G² = likelihood ratio; AIC = Akaike information criterion; BIC = Bayesian information criterion; For scaled difference ΔG², all models were compared to Model 1 of respective behavior, *** denotes $p < .001$ for ΔG².

Table 18. Model fit statistics for 2-year stationary and nonstationary stage transition patterns by cancer risk behavior.

Behavior	Model 2A	Stage Transition Pattern	DF	LL	G ²	ΔG ²	AIC	BIC
<i>Smoking</i>	1	Baseline model, all transition paths free	2024	-5915.477	1095.539	—	11876.954	12007.588
	1S	Stationary model, equal tau	2033	-5920.517	1105.618	10.367	11869.033	11948.549
<i>Unhealthy diet</i>	1	Baseline model, all transition paths free	2024	-20570.905	3192.298	—	41187.810	41344.536
	1S	Stationary model, equal tau	2033	-20601.577	3176.270	63.102 *	41231.155	41326.553
<i>Sun exposure</i>	1	Baseline model, all transition paths free	2024	-21094.532	3236.122	—	42235.064	42392.911
	1S	Stationary model, equal tau	2033	-21121.139	3206.683	54.962 *	42270.278	42366.359

Note: DF = degrees of freedom; LL = log likelihood; G² = likelihood ratio; AIC = Akaike information criterion; BIC = Bayesian information criterion; Nested model comparison of stationary models to nonstationary model of respective behavior; * denotes $p < .001$ for ΔG².

Table 19. Stage transition parameter estimates by cancer risk behavior in full analytical sample.

Risk Behavior	Model	Baseline Stage	<u>Year 1 Stage</u>				Year 1 Stage	<u>Year 2 Stage</u>			
			PC	C	PR	A/M		PC	C	PR	A/M
<i>Smoking</i>	1S	PC	0.651	0.220	0.052	0.076	PC	0.651	0.220	0.052	0.076
		C	0.183	0.528	0.168	0.121	C	0.183	0.528	0.168	0.121
		PR	0.075	0.360	0.333	0.231	PR	0.075	0.360	0.333	0.231
							A/M	0.063	0.083	0.090	0.765
<i>Unhealthy diet</i>	1	PC	0.543	0.126	0.126	0.204	PC	0.628	0.120	0.105	0.147
		C	0.299	0.237	0.213	0.251	C	0.336	0.250	0.235	0.179
		PR	0.231	0.192	0.304	0.273	PR	0.236	0.191	0.358	0.214
							A/M	0.206	0.089	0.160	0.545
<i>Sun exposure</i>	1S	PC	0.666	0.136	0.151	0.047	PC	0.666	0.136	0.151	0.047
		C	0.321	0.311	0.292	0.076	C	0.321	0.311	0.292	0.076
		PR	0.156	0.114	0.480	0.250	PR	0.156	0.114	0.480	0.250
							A/M	0.060	0.029	0.282	0.629

Note: PC = Precontemplation; C = Contemplation; PR = Preparation; A/M = Action/Maintenance.

Table 20. Model fit statistics by cancer risk behavior for 2-year stage transition pattern with intervention condition included as time-invariant covariate.

Behavior	Model 2C	Stage Transition Pattern	DF	LL	G²	AIC	BIC
<i>Smoking</i>	1	Baseline model, all transition paths free	4048	−7396.222	1119.882	14886.444	15153.391
<i>Unhealthy diet</i>	1	Baseline model, all transition paths free	4048	−25183.758	3424.337	50461.516	50781.782
<i>Sun exposure</i>	1	Baseline model, all transition paths free	4048	−25891.936	3861.821	51877.871	52200.428

Note: DF = degrees of freedom; LL = log likelihood; G² = likelihood ratio; AIC = Akaike information criterion; BIC = Bayesian information criterion.

Table 21. Model estimated baseline stage membership probabilities across intervention condition by cancer risk behaviors.

Behavior	Condition	N	PC	C	PR
<i>Smoking</i>	Comparison	1117	0.353	0.427	0.220
	Intervention	1047	0.361	0.453	0.186
<i>Unhealthy diet</i>	Comparison	3494	0.526	0.140	0.334
	Intervention	3235	0.520	0.151	0.328
<i>Sun exposure</i>	Comparison	3684	0.328	0.242	0.429
	Intervention	3381	0.320	0.226	0.453

Note: PC = Precontemplation; C = Contemplation; PR = Preparation.

Table 22. Stage transition parameter estimates across intervention condition for smoking risk.

		<u>Year 1 Stage</u>			
		PC	C	PR	A/M
<u>Baseline Stage</u>	PC	0.598 (-0.051)	0.247 (0.041)	0.064 (-0.007)	0.091 (0.017)
	C	0.183 (-0.017)	0.478 (-0.068)	0.181 (0.017)	0.158 (0.068)
	PR	0.073 (-0.010)	0.350 (-0.022)	0.275 (-0.070)	0.302 (0.102)
		<u>Year 2 Stage</u>			
		PC	C	PR	A/M
<u>Year 1 Stage</u>	PC	0.737 (0.084)	0.209 (-0.009)	0.020 (-0.019)	0.034 (-0.056)
	C	0.145 (-0.041)	0.593 (0.074)	0.139 (-0.040)	0.123 (0.007)
	PR	0.071 (0.003)	0.298 (-0.102)	0.384 (0.045)	0.247 (0.054)
	A/M	0.062 (-0.003)	0.053 (-0.069)	0.065 (-0.058)	0.820 (0.130)

Note: PC = Precontemplation; C = Contemplation; PR = Preparation; A/M = Action/Maintenance;
Values in parentheses represent the difference in the intervention condition parameter estimate compared to the comparison condition.

Table 23. Stage transition parameter estimates across intervention condition for unhealthy diet risk.

		<u>Year 1 Stage</u>			
		PC	C	PR	A/M
<u>Baseline Stage</u>	PC	0.481 (-0.111)	0.146 (0.035)	0.128 (0.003)	0.245 (0.073)
	C	0.288 (-0.022)	0.234 (-0.005)	0.205 (-0.015)	0.273 (0.042)
	PR	0.226 (-0.009)	0.179 (-0.023)	0.283 (-0.039)	0.312 (0.071)
		<u>Year 2 Stage</u>			
		PC	C	PR	A/M
<u>Year 1 Stage</u>	PC	0.592 (-0.060)	0.124 (0.008)	0.123 (0.029)	0.161 (0.023)
	C	0.323 (-0.024)	0.257 (0.011)	0.203 (-0.058)	0.217 (0.071)
	PR	0.222 (-0.026)	0.179 (-0.022)	0.351 (-0.013)	0.248 (0.061)
	A/M	0.168 (-0.079)	0.105 (0.032)	0.144 (-0.034)	0.583 (0.081)

Note: PC = Precontemplation; C = Contemplation; PR = Preparation; A/M = Action/Maintenance;
Values in parentheses represent the difference in the intervention condition parameter estimate from the comparison condition.

Table 24. Stage transition parameter estimates across intervention condition for sun exposure risk.

		<u>Year 1 Stage</u>			
		PC	C	PR	A/M
<u>Baseline Stage</u>	PC	0.589 (-0.104)	0.174 (0.034)	0.165 (0.035)	0.072 (0.035)
	C	0.264 (-0.082)	0.273 (-0.042)	0.338 (0.076)	0.125 (0.048)
	PR	0.114 (-0.087)	0.099 (-0.022)	0.463 (0.002)	0.324 (0.107)
		<u>Year 2 Stage</u>			
		PC	C	PR	A/M
<u>Year 1 Stage</u>	PC	0.650 (-0.057)	0.109 (-0.012)	0.193 (0.058)	0.048 (0.011)
	C	0.311 (-0.046)	0.307 (-0.052)	0.334 (0.083)	0.048 (0.015)
	PR	0.141 (-0.014)	0.096 (-0.045)	0.507 (-0.004)	0.256 (0.063)
	A/M	0.037 (-0.053)	0.014 (-0.035)	0.229 (-0.122)	0.720 (0.210)

Note: PC = Precontemplation; C = Contemplation; PR = Preparation; A/M = Action/Maintenance;
Values in parentheses represent the difference in the intervention condition parameter estimate from the comparison condition.

FIGURES

Figure 1. Unconditional model of 2-year growth trajectory for single cancer risk behavior.

Figure 2. Conditional model of 2-year growth trajectory for single cancer risk behavior with intervention condition as a time-invariant covariate.

Figure 3. Unconditional parallel-process model of 2-year growth trajectories in co-occurring risk behavior dyad (behaviors A and B).
 Note: For simplicity, model shown assumes independence (no association) between intercept and slope factors across behaviors.

Interval (a)

Interval (b)

Figure 4. Stage transition model.

Note: PC = Precontemplation; C = Contemplation; PR = Preparation; A/M = Action/Maintenance stages of behavior change.

Transition probabilities ($\tau_{S_{i2}|S_{i1}}$) to be estimated for intervals:

(a) Baseline to 1-year, and (b) 1- to 2-years.

Model 1: Baseline model, all paths free.

Model 2: Two-forward, two-backward.

Model 3: Two-forward, one-backward.

Model 4: One-forward, one-backward.

Figure 5. Stage transition pattern models.

Note: PC = Precontemplation; C = Contemplation; PR = Preparation;

A/M = Action/Maintenance stages of behavior change.

Dashed arrows denote transition paths conditioned on membership in Action/Maintenance stages at previous time point and are only estimated for interval between Year 1 and 2.

Figure 6. Conditional 2-year LTA model for single cancer risk behavior with intervention condition as a time-invariant covariate.

$\chi^2(2, N=2117) = 24.218$; CFI = 0.973; NNFI = 0.960; RMSEA = .072, 90% CI [.048, .100].

Figure 7. Unconditional model of 2-year growth trajectory for smoking behavior with standardized parameter estimates.

Note: α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$;

Indicators for smoking behavior are number of cigarettes/day (square-rooted transformed for normalization) at baseline, 1- and 2-years;

$\chi^2(4, N=6651) = 169.992$; CFI = 0.980; NNFI = 0.985; RMSEA = .079, 90% CI [.069, .089].

Figure 8. Unconditional model of 2-year growth trajectory for healthy eating behavior with standardized parameter estimates.

Note: α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$;

Indicators for diet behavior are DBQ mean scores at baseline, 1- and 2-years.

$\chi^2(3, N=7021) = 268.23$; CFI = 0.963; NNFI = 0.963; RMSEA = .112, 90% CI [.101, .124].

Figure 9. Unconditional model of 2-year growth trajectory for sun protection behavior with standardized parameter estimates.

Note: α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$;

Indicators for sun protection behavior are SPBI mean scores at baseline, 1- and 2-years.

$\chi^2(3, N=2117) = 24.078$; CFI = .975; NNFI = .950; RMSEA = .058, 90% CI [.038, .080].

Figure 10. Conditional model of 2-year growth trajectory for smoking behavior with standardized parameter estimates.

Note: α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$;

Indicators for smoking behavior are number of cigarettes/day (square-rooted transformed for normalization) at baseline, 1- and 2-years;

$\chi^2(5, N=6651) = 177.131$; CFI = .980; NNFI = .976; RMSEA = .072, 90% CI [.063, .081].

Figure 11. Conditional model of 2-year growth trajectory for healthy eating behavior with standardized parameter estimates.

Note: α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$;

Indicators for diet behavior are DBQ mean scores at baseline, 1- and 2-years.

$\chi^2(4, N=7021) = 270.621$; CFI = .963; NNFI = .945; RMSEA = .097, 90% CI [.088, .170].

Figure 12. Conditional model of 2-year growth trajectory for sun protection behavior with standardized parameter estimates.

Note: α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$;

Indicators for sun protection behavior are SPBI mean scores at baseline, 1- and 2-years.

$\chi^2(6, N=2117) = 24.355$; CFI = .987; NNFI = .966; RMSEA = .038, 90% CI [.023, .054].

Figure 13. Conditional model of 2-year growth trajectory for smoking behavior with multiple time-invariant covariates and standardized parameter estimates.

Note: TRT = intervention condition; Tempt = situational temptations;

α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$;

Indicators for smoking behavior are number of cigarettes/day (square-rooted transformed for normalization) at baseline, 1- and 2-years;

$\chi^2(8, N=6651) = 179.158$; CFI = .981; NNFI = .964; RMSEA = .057, 90% CI [.050, .064].

Figure 14. Conditional model of 2-year growth trajectory for healthy eating behavior with multiple time-invariant covariates and standardized parameter estimates.

Note: TRT = intervention condition; Tempt = situational temptations;

α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$;

Indicators for diet behavior are DBQ mean scores at baseline, 1- and 2-years.

$\chi^2(7, N=7021) = 335.447$; CFI = .973; NNFI = .943; RMSEA = .082, 90% CI [.074, .089].

Figure 15. Conditional model of 2-year growth trajectory for sun protection behavior with multiple time-invariant covariates and standardized parameter estimates.

Note: TRT = intervention condition; Conf = self-efficacy;

α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$;

Indicators for sun protection behavior are SPBI mean scores at baseline, 1- and 2-years.

$\chi^2(13, N=1496) = 42.710$; CFI = .987; NNFI = .985; RMSEA = .039, 90% CI [.026, .052].

Figure 16. Unconditional parallel-process model of 2-year growth trajectories for smoking and healthy diet behaviors with standardized parameter estimates.

Note: α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$;

Indicators at baseline, 1- and 2-years for smoking behavior are number of cigarettes/day (square-rooted transformed for normalization), and mean DBQ scores for diet behavior.

$\chi^2(14, N=4681) = 303.24$; CFI = .974; NNFI = .972; RMSEA = .066, 90% CI [.060, .073].

Figure 17. Unconditional parallel-process model of 2-year growth trajectories for healthy diet and sun protection behaviors with standardized parameter estimates.

Note: α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$.

Indicators at baseline, 1- and 2-years for healthy eating behavior are mean DBQ mean scores, and mean SPBI scores for sun protection behavior.

$\chi^2(12, N=1499) = 77.913$; CFI = .967; NNFI = .959; RMSEA = .061, 90% CI [.048, .074].

Figure 18. Unconditional parallel-process model of 2-year growth trajectories for smoking and sun protection behaviors with standardized parameter estimates.

Note: α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$.

Indicators at baseline, 1- and 2-years for smoking behavior are number of cigarettes/day (square-rooted transformed for normalization), and mean SPBI score for sun protection behavior.

$\chi^2(23, N=1496) = 44.342$; CFI = .991; NNFI = .988; RMSEA = .035, 90% CI [.019, .051].

Figure 19. Unconditional parallel-process model of 2-year growth trajectories for smoking and healthy diet behaviors with standardized parameter estimates across intervention condition.

Note: Parameter estimates for control condition are presented in parentheses;

α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$.

Indicators at baseline, 1- and 2-years for smoking behavior are number of cigarettes/day (square-rooted transformed for normalization), and mean DBQ scores for diet behavior.

$\chi^2(24, N=4681) = 243.70$; CFI = .980; NNFI = .975; RMSEA = .063, 90% CI [.056, .070].

Figure 20. Unconditional parallel-process model of 2-year growth trajectories for healthy diet and sun protection behaviors with standardized parameter estimates across intervention condition.

Note: Parameter estimates for control condition are presented in parentheses;

α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$.

Indicators at baseline, 1- and 2-years for healthy eating behavior are mean DBQ mean scores, and mean SPBI scores for sun protection behavior.

$\chi^2(21, N=1499) = 62.934$; CFI = .979; NNFI = .970; RMSEA = .052, 90% CI [.037, .067].

Figure 21. Unconditional parallel-process model of 2-year growth trajectories for smoking and sun protection behaviors with standardized parameter estimates across intervention condition.

Note: Parameter estimates for control condition are presented in parentheses;

α = estimated factor mean; ψ = estimated factor variance;

* $p < .05$; ** $p < .01$; *** $p < .001$.

Indicators at baseline, 1- and 2-years for smoking behavior are number of cigarettes/day (square-rooted transformed for normalization), and mean SPBI score for sun protection behavior.

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