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Global positive expectancies in adolescence and health-related behaviours: Longitudinal models of latent growth and cross-lagged effects

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Abstract

Constructs representative of global positive expectancies (GPE) such as dispositional optimism and hope have been theoretically and empirically linked to many positive mental and physical health outcomes. However such expectancies' health implications for adolescents, as well as their trajectory over time, are less well understood than for adult populations. This study tested whether GPE predict the key indicators of adolescents' future physical health status, their health-related behaviours. A prospective longitudinal study design was employed whereby a diverse population-based cohort ($N = 744$; mean age at baseline = 12) completed three surveys over approximately 18 months. Rigorous tests of causal predominance and reciprocal effects were conducted through latent growth and cross-panel structural equation models. Results showed GPE systematically decreased during the course of the study, yet higher initial levels of GPE predicted less alcohol drinking, healthier food choice and greater physical activity over time. GPE's protective relationships towards health protective behaviours (vs. health risk behaviours that also included tobacco smoking) appear more independent from depressive symptomatology, and the primary findings were robust across socio-demographic groups.

Keywords

expectancies; optimism; hope; health behaviour; substance use; physical activity; adolescents

Introduction

Generalised expectancies have been theorised as health protective dispositions for over five decades (e.g. Rotter, 1954; Scheier & Carver, 1985; Schwarzer & Fuchs, 1996; Snyder et al., 1991; Wallston, 1992). Two exemplary constructs and central within the emerging field of positive psychology (Gallagher & Lopez, 2009; Rand, 2009; Roesch & Vaughn, 2006), are *dispositional optimism* (Scheier & Carver, 1985) and *hope* (Snyder et al., 1991). Despite extensive research on their health implications, gaps remain that require prospective designs and dynamic models to address (Aspinwall & Tedeschi, 2010; Rand, 2009; Smith, 2006).

Dispositional optimism reflects generalised outcome expectancies (Scheier & Carver, 1985) and has been linked to many health benefits over the lifespan (Benyamini & Roziner, 2008; Scheier, Carver, & Bridges, 1994). Primary mechanisms that these expectancies promote health are positive immune function, (usually) adaptive coping strategies in response to specific threats and positive lifestyle habits. This investigation focuses on the latter, as Jessor, Turbin, and Costa (2010) affirm, adolescents' future health status is strongly tied to their health-related behaviours. The preponderance of research suggests optimism predicts more health protective behaviours and fewer risk behaviours (e.g. Baker, 2007; Carvajal, Evans, Nash, & Getz, 2002; Giltay, Geleijnse, Zitman, Buijsse, & Kromhout, 2007; Kelloniemi, Ek, & Laitinen, 2005; Milam, Richardson, Marks, Komper, & McClutchman, 2004; Steptoe, Wright, Kunz-Ebrecht, & Iliffe, 2006).

Snyder et al. (1991) define hope as general *control* expectancies, specifically goal-related determination ('agency') and perceived efficacy in planning to meet goals ('pathways'). Hope and other related constructs (e.g. general self-efficacy, perceived self-regulation competence, habitual self-control and self-mastery), are associated with increased protective behaviours and decreased risk behaviours (e.g. Carvajal et al., 2002; Irving, Snyder, & Crowson, 1998; Scheier & Carver, 1985; Schröder & Schwarzer, 2005; Schwarzer, 1994).

The distinctiveness of optimism, hope and related constructs has been debated (Aspinwall & Tedeschi, 2010; Lagacé-Séguin & d'Entremont, 2010; Roesch & Vaughn, 2006; Smith, 2006; Tennen, Affleck, & Tennen, 2003). Studies with younger populations have found strong associations between optimism and hope scales (e.g. r 's 0.65–0.80; Bryant & Cvengros, 2004; Carvajal et al., 2002; Ey et al., 2005; Lagacé-Séguin & d'Entremont, 2010). Such measures have also been included as part of broad health protective intrapersonal resources (de Ridder, Fournier, & Bensing, 2004; Lauriola, Laicardi, Artistic, & Baldassarri, 2000; Marshall, Wortman, Vickers, Kusulas, & Hervig, 1994; Matthews, Räikkönen, Gallo, & Kuller, 2008; Schröder & Schwarzer, 2001; Schwarzer, 1994; Yi, Vitaliano, Smith, Yi, & Weinger, 2008), though there is no consensus on terminology (e.g. 'functional optimism', 'optimistic beliefs', 'reserve capacity', 'resiliency', 'optimistic-control', 'personal resourcefulness'). Further, multiple studies have identified a higher order structure of optimism and hope, and defined this generalised or global 'positive expectancies' dimension (Carvajal et al., 2002; Gallagher & Lopez, 2009; also note Rand, 2009 and Bryant & Cvengros, 2004, each estimated a comparable factor termed 'goal attitude' and 'future orientation', respectively).

This study

This *epidemiological personology* (Krueger, Caspi, & Moffit, 2000) investigation tests the trajectory and predictive efficacy of global positive expectancies (GPE) within a three-wave prospective longitudinal population-based design. Less is known about the health protective role of GPE (as well as optimism and hope) in general adolescent populations than in adult and/or clinical samples (Benyamini & Roziner, 2008; Heinonen et al., 2006). Jessor et al. (2010) also note distal influences may account for unique variation in health behaviours, and be of particular importance for multiple such behaviours' developmental trajectories (in contrast to more narrow predictive contexts of proximal factors). The primary outcomes are

the four leading causes of premature mortality in the USA: tobacco smoking, alcohol use, healthy food choice and physical activity (Centers for Disease Control and Prevention, 2004).

This research aims to compliment health psychology research on central personality domains (Smith, 2006) and context specific research on more narrowly banded constructs (Aspinwall & Tedeschi, 2010). To date, little is known about developmental trajectories of global expectancy constructs. Adolescence is a known critical time of personality change (Caspi, Roberts, & Shiner, 2005); persons tend to follow the maturity principle: exhibiting greater openness, agreeableness, conscientiousness and lower neuroticism as they age (Johnson, Hicks, McGue, & Iacono, 2007; Klimstra, Akse, Hale, Raaijmakers, & Meeus, 2010; Lüdtke, Trautwein, & Husemann, 2009). A study involving Australian parochial high school students showed hope systemically decreased (Heaven & Ciarrochi, 2008); there are no similar studies on optimism.

Personality-health scholars also note the preponderance of studies relies on adult samples or two data points, limiting tests of more nuanced questions on personality development in adolescence (Caspi et al., 2005). This study employs rigorous non-experimental methods for testing causal predominance and reciprocal influences (Bollen & Curran, 2004; Curran, Stice, & Chassin, 1997); thus if engagement in health protective or risk behaviours alters individuals' positive dispositions and mood is considered (White, Kendrick, & Yardley, 2009). The four study aims are to test if: (1) GPE change within a relatively brief early adolescent period; (2) initial and subsequent levels of GPE predict initial and subsequent levels of health-related behaviours; (3) GPE's prediction of behaviours is distinct from a competing explanatory construct and (4) socio-demographics moderate the relations between GPE and behaviours.

Systematic change of GPE will be tested using *latent growth* (LG) models (Muthén & Asparouhov, 2011). LG models have not been used to test for systematic change in GPE (nor dispositional optimism), thus knowledge about its relative mutability and trajectory in adolescents is limited. The LG approach uses a structural equation modelling (SEM) framework to test a multi-level or hierarchical linear model estimating individual change. These models extend the potential inferences capable of generalised linear models (GLMs) or standard SEM because they afford tests of systemic within-individual changes in study variables and their relations to other endogenous factors (Benyamini, Ein-Dor, Ginzburg, & Solomon, 2009; Heaven & Ciarrochi, 2008). Integrated LG models of GPE and the health-related behaviours will be used to test if initial levels or escalation of GPE predict initial or escalation of those behaviours. As self-regulation is critical to engaging in healthy lifestyle-related behaviours (Schröder & Schwarzer, 2005; Schwarzer & Fuchs, 1996), it is expected that GPE will negatively predict the risk behaviours and positively predict the protective behaviours. Each outcome will be predicted independently so as to not assume GPE is equivalently protective across health domains (Tennen et al., 2003).

Of note, the health protective effects of optimism or hope may diminish when considered with depressive symptomatology (DS) or a sub-component, negative affect (Benyamini & Roziner, 2008; Chang, 1998; de Ridder et al., 2004; Lai et al., 2005; Räikkönen &

Matthews, 2008). Thus, the degree GPE predict health behaviours beyond DS which will be a further test of its robustness as an explanatory factor. For these tests, multi-wave cross-panel SEM (Curran et al., 1997) will be estimated, a technique particularly useful for testing mutual influences of personality factors and health behaviours (Klimstra et al., 2010). Finally, there is debate of the generality of optimism and hope in diverse populations (Edwards, Ong, & Lopez, 2007; Rääkkönen, & Matthews, 2008; Roesch & Vaughn, 2006). Socio-demographic factors found to predict changes in adolescents' dispositions or health behaviours (Carvajal & Granillo, 2006; Ek, Remes, & Sovio, 2004; Heinonen et al., 2006) will be examined as moderators.

Method

Participants and procedures

This study utilised a diverse population-based cohort ($N = 744$) in the West Coast of the USA. The baseline mean age of respondents was 12 (97% were 11 or 12). Respondents' self-identified as follows: 57% female; 49% having a parent with some college education; 41% Latino; 29% European American or White; 18% Asian American; 7% African American; and 6% multi-ethnic/other. Procedures were developed following the American Psychological Association's ethical guidelines; the study also complied with applicable federally approved internal review boards. Active consent – one signed consent form from a parent allowing the child's participation, and one approved by the child, were required for study enrollment.

Participants completed three self-report surveys. The median and mean duration of follow-up were 18 and 19 months, with comparable intra-wave intervals. The cohort was established by randomly selecting among all 6th grade classrooms in a large urban school district. The baseline participation rate was 67%. Non-responses were primarily due to the inability to acquire a signed parental consent form, with refusal on this form less than 7% and student refusal less than 1%. It should be noted that this baseline response rate is greater than that for the most comparable US-sponsored epidemiological youth health survey in the encompassing region and nearby locales (Centers for Disease Control and Prevention, 2004). Research staff collected data through group administered paper and pencil questionnaires during class periods. If participants could not be reached in school for follow up, multiple efforts to obtain questionnaires via mail were attempted.

Cohort attrition—Given that this study was theory-driven and etiological, the study completion rate was closely monitored. Due to intensive follow-up procedures, 86% of respondents completed a Wave II follow-up and 80% completed a Wave III follow-up. Primary reasons for lack of follow up were those who moved out of district (and whose forwarding addresses and secondary contacts failed) and chronic absentees who also failed to respond to multiple school and mail attempts. Implications of attrition were also evaluated. GPE (baseline) was unrelated to completion of Wave II data, those with higher scores were marginally more likely to complete Wave III ($\eta = 0.08, p < 0.05$). Also smoking less was associated with completion of Wave II ($\eta = 0.07, p < 0.05$), but unrelated to completion of Wave III ($p < 0.05$). Less alcohol use was unrelated to completion of Wave II,

but was positively associated with completion of Wave III ($\eta = 0.08$, $p < 0.05$). Baseline depressive symptoms, healthy food choice and physical activity were unrelated to completion of a Wave II or Wave III survey. As no variable accounted for 1% of the variance in follow-up completion status, the likelihood for attrition substantively impacting the findings is modest.

Questionnaire and measures

Participants completed a self-report questionnaire. Socio-demographic variables that were assessed included age, gender, ethnicity and parental education (a reliable socioeconomic status indicator for youth; Heinonen et al., 2006; Wills, Ainette, Stoolmiller, Gibbons, & Shinar, 2008).

Global positive expectancies—GPE was measured by eight items. Challenges to measuring dispositional optimism by the most established scales, the Life Orientation Test (LOT; Scheier & Carver, 1985) and Revised Life Orientations Test (LOT-R; Scheier et al., 1994), are well documented for late adolescent or younger populations (Carvajal et al., 2002; Ey et al., 2005; Lemola et al., 2010; Rääkkönen & Matthews, 2008). Guided by focus groups with youth – which indicated comprehension concerns for the negatively worded items, four positively worded items representing optimism were used. They were selected from a prior youth instrument adapted from the LOT and LOT-R, with further guidance from factor loadings from prior adolescent studies that favoured the positive items (Carvajal et al., 2002; Ey et al., 2005).

Four of the six items from the Children's Hope Scale (Snyder et al., 1997) completed the GPE measure. These items were selected because they had superior loadings from prior research, and balanced agency/pathway components. The two agency items included in this study are also the same ones Edwards et al. (2007) identified as the sole emergent indicators of agency in their analysis of the Children's Hope Scale in adolescents.

In totality, the eight GPE items were as follows (in the order of presentation): 'I always look on the bright side of things' (dispositional optimism); 'I think I am doing pretty well.' (hope, agency); 'I can think of many ways to get things in life that are important to me.' (hope, pathways); 'I believe some good can come out of every situation.' (dispositional optimism); 'I am doing just as well as other kids my age.' (hope, agency); 'When I have a problem, I can come up with lots of ways to solve it.' (hope, pathways); 'I always feel good about my future'. (dispositional optimism); 'Overall, I expect more good things to happen to me than bad'. (dispositional optimism). Respondents used the categories from the Children's Hope Scale as it was preferred in pilot research, they are: 'All of the time' (4), 'Most of the time' (3), 'Some of the time' (2) and 'None of the time' (1). (Note: Ey et al., 2005, changed the response formats of the LOT-R when developing the Youth Life Orientations Test due to similar issues).

The GPE measure had high internal consistency at all three data collections: $\alpha = 0.87$ (Wave I), $\alpha = 0.89$ (Wave II), $\alpha = 0.90$ (Wave III). Additionally, a unidimensional confirmatory factor analytic model fit the data adequately at each data collection (at Wave I: CFI, comparative fit index = 0.98; GFI, goodness of fit = 0.98, RMSEA, root mean square error

of approximation = 0.05; at Wave II CFI = 0.96, GFI = 0.96; RMSEA = 0.08; at Wave III CFI = 0.96; GFI = 0.95; RMSEA = 0.09). Guided by these findings, GPE was represented from these eight indicators.

Depressive symptomatology—DS was measured with an eight-item version of the CES-D (Radloff, 1997) guided by prior research (Kohout, Berkman, Evans, & Cornoni-Huntley, 1993; Santor & Coyne, 1997). Four symptom domains of depression are reported in the past 7 days: negative affect (e.g. ‘I felt down and unhappy’, (absence of) positive affect (e.g. ‘I was happy’), somatic complaints (e.g. ‘I didn’t sleep as well as I usually sleep’) and interpersonal problems (e.g. ‘I felt people didn’t like me’). Response categories include: ‘5–7 days’, ‘3–4 days’, ‘1–2 days’, ‘Less than 1 day’ and ‘Never’. Given the purpose of this study to use DS as a competing explanatory construct, it was kept continuous (e.g. Chang, 1998; Heaven & Ciarrochi, 2008), with higher values corresponding to more DS (α 's = 0.87, 0.89, 0.90).

Health-related behaviours were based on measures employed in US national surveys of adolescent health status (e.g. Centers for Disease Control and Prevention, 2004). Logarithmic transformation of smoking and alcohol items were applied as they were moderately to severely non-normally distributed. *Current cigarette smoking* was determined from responses to ‘During the past 30 days’... ‘how many days did you smoke a cigarette’, ‘how many times did you have a puff on a cigarette?’ and ‘on the days you smoked, how many cigarettes did you smoke per day?’. Each question had 5–6 response categories and a composite score was derived (α 's = 0.88, 0.89, 0.88). *Current alcohol use* was assessed with the item, ‘During the past 30 days’... ‘how many days did you have at least one drink of alcohol?’. Response categories were ‘0 days’, ‘1–2 days’, ‘3–9 days’, ‘10–19 days’ or ‘20+ days’. Higher scores represent more smoking and drinking, respectively.

Healthy food choice—Healthy food choice was based on six items on the number of servings of fruits or vegetables consumed the week prior. The questions were phrased ‘During the past 7 days, how many times did you’... ‘eat green salad?’, ‘eat carrots?’, etc. Response formats were ‘Less than 1 time’, ‘1 to 3 times’, ‘4 to 6 times’, ‘1 time per day’, ‘2 times per day’ and ‘3+ times per day’. Higher aggregate scores reflect more consumption of fruits and vegetables (α 's = 0.86, 0.86, 0.85).

Physical activity—Physical activity was assessed by three items indicating various activities in the past week. Specifically, they assessed the number of days respondents: exercised or participated in (vigorous) physical activity for a minimum of 20 minutes (e.g. ‘that made you sweat and breathe hard. For example, basketball, running, swimming..’), participated in (light) physical activity for a minimum of 30 minutes (e.g. ‘fast walking’, ‘slow bicycling’, ‘shooting baskets..’), and exercised to strengthen or tone muscles (e.g. ‘push-ups’, ‘sit-ups’, ‘weight lifting’). Response formats ranged from ‘0 days’ to ‘7 days’ (α 's = 0.66, 0.69, 0.69).

Data analysis

Prior to conducting the analysis one consideration was if to proceed in two (observations nested with individuals) or three-level analysis (add nesting within school at baseline) for the LG models. Intraclass correlations (ICC) were estimated to show the degree respondents within schools had more interrelated responses relative to those in other schools. ICC was defined as follows: $\rho = \sigma^2_s / \sigma^2_t$, expressing the variance between schools over the total variance for the outcomes, with adjustment for planned covariates and using robust estimation. In total, 12 ρ s were estimated, the endogenous indicators of GPE, DS, smoking, drinking, healthy food choice and physical activity. ρ associated with school differences for GPE = 0.013 (T2) and = 0.004 (T3). The other 10 estimates of ρ ranged from 0.0001 to 0.047 (T2 activity). From these coefficients (no school level covariates were employed), it was determined that the combined influence of the nesting within schools at baseline was negligible on parameter estimates and inferential tests for the primary models [estimates of the largest variance inflation factor (VIF) = 0.0006]; thus within and/or between individual-level models were conducted.

Given the sample size and the study variables' distributions, non-normality was accounted for using the Satorra-Bentler scaled χ^2 (Bentler, 2006). With sample sizes greater than 250, maximum likelihood estimation with adjustments for the $S-B\chi^2$ performs well (Bentler, 2006; Herzog & Boomsma, 2009; Lai & Kelley, 2011), thus there was high confidence in using this approach to generate accurate parameter estimates and standard errors. Power to detect significant parameter estimates (Lai & Kelley, 2011) was also high. Given study conditions, there was 80% power to detect lower bound small effects consistent with the personality-health literature (i.e. $\beta = |0.10|$) and 99% power to detect upper bound small effects (i.e. $\beta = |0.20|$).

To limit further model complexity (and improve convergence) while adjusting for measurement error, attenuation was adjusted from α coefficients (alcohol used the smoking estimate). Goodness of model fit was evaluated by multiple indices from different theoretical classes. Acceptable fit was based on criteria of Bentler (2006) and Bollen and Curran (2004), and evidenced if: (1) the CFI > 0.9 (superior fit 0.95); (2) the GFI > 0.9; (3) the RMSEA < 0.10 (superior fit 0.06); and 4) the standardised residuals are well distributed. EQS (Bentler, 2006) was used to conduct all SEM analysis, including LG models (means analysis). Potential moderation of GPE (by demographics) was tested in GLMs with hierarchically added cross-product interaction terms.

Results

Study variables' distributions and inter-correlations are presented in Table 1. GPE were most strongly related (negatively) with DS measured at the same time point (-0.42 r 's -0.47). GPE were significantly ($p < 0.05$) associated with all variables observed at the same time point or later. GPE were negatively associated with the risk behaviours and positively associated with the protective behaviours.

LG models

The incipient model tested LG parameters of GPE. Paths from each GPE measure to the intercept variable were fixed to one. Given equivalent wave intervals (Mehta & West, 2000), paths from GPE at Wave I to the slope parameter was fixed to 0, GPE Wave II to 1, and GPE Wave III to 2. This model thus expresses initial level of GPE and an estimate of linear change over time. Paths from a constant to each latent variable were also modelled to provide overall mean estimates for the intercept and slope.

The LG model (Figure 1) of GPE fit the data well (CFI = 1.00; RMSEA = 0.02; GFI = 1.00). Unless otherwise noted (by the dashed lines and indicated by 'ns'), the standardised coefficients presented in the LG figures are statistically significant ($p < 0.05$). The paths from the constant to the intercept represent the initial overall mean of GPE ($\mu = 3.14$, $p < 0.05$). The paths from the constant to the slope represent average rate of change ($\mu = -0.08$, $p < 0.05$); thus GPE is systematically decreasing in the study period. There were significant variance components ($\psi = 0.19, 0.06$, p 's < 0.05) for the intercept and slope, evidencing individual variation in initial levels of GPE and rates of change. The negative correlation ($r = -0.28$, $p < 0.05$) for the intercept and slope shows that individuals with higher initial GPE had a larger rate of decrease in GPE.

Models including the relationships among the LG estimates of GPE to LG estimates of the risk behaviours were then tested. The procedures to develop the LG models of smoking and alcohol were the same as that for GPE and had parameters consistent with prior work (Benyamini et al., 2009; Curran et al., 1997; Wills et al., 2008). The first of these models (Figure 2) tested GPE and cigarette smoking and fit the data well (CFI = 1.00; RMSEA = 0.01; GFI = 1.00). This model evidenced participants' smoking rose over time ($\mu = 0.02$, $p < 0.05$) and there was significant individual variation in initial levels of smoking and rate of change (estimates provided in the text only for clarity). The intercepts from these models were negatively correlated ($r = -0.17$, $p < 0.05$; Figure 2) showing initial GPE was significantly negatively correlated with initial reported smoking. Also, the slopes were negatively correlated ($r = -0.19$, $p < 0.05$), suggesting participants' change in GPE were inversely related to change in smoking (i.e. those whose levels of GPE dropped more were likely to increase their smoking). Neither initial GPE predicted change in smoking nor initial smoking predicted change in GPE.

Next, LG models of GPE and alcohol use were tested and fit the data well (CFI = 0.99; RMSEA = 0.03; GFI = 1.00). Alcohol use systematically increased over time ($\mu = 0.03$, $p < 0.05$), and there was significant individual variation in initial use and rates of change. The intercepts and slopes of GPE and alcohol (Figure 3) were both negatively correlated (r 's = $-0.18, -0.23$, p 's < 0.05); initial level and change in these factors were inversely associated. Also, initial level of GPE negatively predicted escalation (growth) of alcohol use over time ($\beta = -0.18$, $p < 0.05$) beyond initial use. Thus, those with higher initial levels of GPE started at lower levels of drinking and showed a less rapid escalation of drinking over the study period than other participants. Initial levels of alcohol use did not significantly predict changes in GPE.

A model of the relations among LG models of GPE and healthy food choice was tested. This model fit the data well (e.g. CFI = 0.97; RMSEA = 0.07; GFI = 0.99) and showed healthy food choice systematically degraded over time ($\mu = -0.43, p < 0.05$) and there was significant individual variation in food choice and rate of change. Initial levels of healthy food choice were unrelated to rate of change in food choice ($p > 0.05$). Figure 4 shows initial GPE and healthy food choice ($r = 0.39, p < 0.05$) were significantly related as were their slopes ($r = 0.31, p < 0.05$). Initial GPE was negatively related to the degradation of food choice ($\beta = -0.22, p < 0.05$) above initial levels of food choice. Thus, participants ate less healthy foods over time; those who initially reported higher levels of GPE initially ate more fruits and vegetables as well as decreased such consumptions at slower rate than the overall trend. Initial healthy food choice did not predict changes in GPE.

The last of the LG analyses included GPE and physical activity, however activity responses over time fit poorly to a LG model. Thus, a model was estimated with independent physical activity responses at each time point with an auto-regressive correlated error structure (Bollen & Curran, 2004; Curran et al., 1997). To most parallel prior models, the intercept of GPE was correlated with the physical activity at Wave I, the slope of GPE correlated with both successive reports of activity, and the intercept of GPE predicted successive reports of activity. This model fit the data well (e.g. CFI = 0.99; RMSEA = 0.03; GFI = 1.00) and is presented in Figure 5. Initial GPE were predictive of greater reported physical activity at Wave II ($\beta = 0.27, p < 0.05$) and Wave III ($\beta = 0.12, p < 0.05$) statistically controlling for all previous reports of activity. Wave I physical activity was correlated with initial GPE ($r = 0.16, p < 0.05$) but did not predict change in GPE. Those who were more active at baseline showed neither faster nor slower degradation in GPE.

Cross-panel models incorporating DS

Relations of GPE and health-related behaviours while considering DS were then tested. Models initially included paths where all previous indicators predict future ones, and those measured at the same time point (or their residuals for endogenous variables) are correlated (Bollen & Curran, 2004). Then each path was deleted (with the exception of the 2nd order auto-regressive correlated error structure maintained regardless of statistical criteria) until only significant paths remained. Unless otherwise noted (by the dashed lines and indicated by 'ns'), the standardised coefficients and correlations presented graphically in the cross-panel model figures are significant ($p < 0.05$).

The incipient series of models predict cigarette smoking by GPE and DS, and fit the data well (CFI = 1.00; RMSEA = 0.02; GFI = 1.00). GPE negatively predicted future levels of DS across the time points (Figure 6), but only DS significantly predicts future smoking when all prior variables were considered ($\beta = 0.15, p < 0.05$; between the 2nd and 3rd time points). Those reporting more DS at Wave II reported more smoking at Wave III. Wave I smoking also predicts Wave III DS ($\beta = -0.11, p < 0.05$). Next, these models were estimated with alcohol use. The final cross-lagged alcohol use model (Figure 7) fit the data well (CFI = 1.00; RMSEA = 0.03; GFI = 0.99). GPE predicts future DS, but in this model GPE significantly predicts future alcohol use in the last interval when all prior variables were

considered ($\beta = -0.17, p < 0.05$). Higher GPE at Wave II predicts lower drinking at Wave III.

Cross-lagged models incorporating the health protective behaviours were then tested. The final model with healthy food choice fit the data well (CFI = 1.00; RMSEA = 0.02; GFI = 0.99). GPE and healthy food choice (Figure 8) positively and significantly predict future levels of one another in two of the four paths estimated (Wave I GPE to Wave III healthy foods, $\beta = 0.08, p < 0.05$; Wave II healthy foods to Wave III GPE, $\beta = 0.09, p < 0.05$). Last, these models with physical activity fit the data well (CFI = 1.00; RMSEA = 0.02; GFI = 1.00). Both previous measures of GPE predict the subsequent measure of activity (Figure 9) beyond that accounted for by prior activity and DS ($0.11 \leq \beta s < 0.14, p's < 0.05$). Activity at Wave II also positively predicts GPE at Wave III ($\beta = 0.11, p < 0.05$). All associations between GPE and activity showed higher levels of one were related to higher levels of the other. DS and food choice were unrelated ($p's > 0.05$) in both final health protective models (Figures 8 and 9).

Testing the robustness of GPE in a diverse sample

Associations among demographics and the primary variables were examined. GPE was unrelated to gender, age and ethnicity. However, GPE was positively associated with education at Wave III ($\eta = 0.14, p < 0.01$). Those with a parent who had a college degree reported higher GPE. Also, girls reported significantly ($p < 0.05$) higher DS (all Waves), less smoking (Wave II only) and more physical activity (Wave II only). Those older reported more alcohol use and smoking (at Wave I only). Those of lower education reported higher DS, more smoking, more alcohol use, and less activity at one or more time points. Latinos (at Wave III only) reported more alcohol use than Whites and those of another ethnicity. Whites ate fewer healthy foods and were more active at multiple time points.

Potential moderators of the relationships between GPE to health-related behaviours or DS due to gender, age, ethnicity and education were then tested. GLMs that correspond to the cross-panel models were developed, then all paired interaction terms with each demographic variable were hierarchically added (entered one at a time with significance evaluated by R^2). There were 24 such tests for gender, and only one with GPE was significant. Wave I GPE $\times$ gender was significant in predicting smoking at Wave II ($R^2 = 0.007, p < 0.05$), stratified analysis showed that within this model GPE only significantly predicted smoking in boys. Other tests of the interactions with gender showed differential prediction of DS ($p < 0.05$). Subsequent analysis showed DS at both time points were more strongly predicted by GPE for girls than for boys (Though DS at Wave II for boys was still significantly predicted by GPE at Wave I).

None of the 24 GPE $\times$ age interactions terms were significant ($p > 0.05$). There were 48 tests of interactions between GPE and education or ethnicity. In no case did GPE differentially predict behaviour. However, GPE $\times$ ethnicity was significant ($p < 0.05$) in predicting DS at both follow-ups. Stratified analyses showed GPE independently predicted lower DS in Whites and those of other ethnicity at future time points, but not in the Latinos. Though one would expect to two or more significant ($p < 0.05$) interactions in latter tests, the fact the

interaction terms with ethnicity and GPE were significant in predicting DS at both follow ups suggest this effect may be robust.

Discussion

This study examined systematic change in GPE and its role in influencing health-related behaviours. Underutilised techniques for informing upon causal relationships and for testing interrelated developmental trajectories were employed. GPE systematically decreased in this early adolescence period, consistent with trajectories of hope (Heaven & Ciarrochi, 2008) and self-esteem development (Robins & Trzesniewski, 2005). The findings also showed higher initial GPE predicted slower escalation of drinking alcohol, slower degradation of healthy food choices and increased future physical activity. Further, and unexamined in previous studies, initial health-related behaviours did not predict rate of change of GPE. Thus, the findings suggest GPE (and dispositional optimism and hope) have a greater influence on health behaviour engagement than those behaviours influence them.

The protective relations of GPE and health behaviours observed were largely independent from DS, with one exception. DS could account for the relations between initial GPE and future cigarette smoking; for smoking DS may act as a mediator (Figure 6; Baker, 2007; Lagacé-Séguin & d'Entremont, 2010). However DS could not explain relations among GPE with drinking alcohol, healthy food choice or physical activity over time, in fact DS was not associated with the latter two outcomes. Also, the cross-panel models suggest GPE more influence DS than DS influence GPE, and GPE' protective relations to health behaviours were generally robust across socio-demographic groups. The lone exception was Wave II reported smoking, where the significant protective influence of GPE was restricted to boys.

The findings contribute to the understanding of relations of dispositional optimism and healthy eating. Some prior studies have not found dispositional optimism to predict physical activity or weight-related outcomes (Davidson & Prkachin, 1997; Fontaine & Cheskin, 1999; Milam et al., 2004), while other investigations have found protective relationships like the current findings (Baker, 2007; Giltay et al., 2007; Ironson et al., 2005; Mulkana & Hailey, 2001; Schröder & Schwarzer, 2005; Steptoe et al., 2006). Taken collectively, when samples are more population-based (versus clinical) and when the interval of the physical activity measured is longer, optimism appears more predictive of physical activity. Benyamini and Raz (2007) as well as Schröder and Schwarzer (who examined optimism as well as other general expectancy constructs) note individual differences in self-regulation may be more critical when health behavioural engagement or maintenance encounter sustained challenges over time.

Though all observed significant relationships with GPE were health protective, predictions of risk behaviours were less robust in this study. It should be noted the smoking and drinking reports referenced frequency of use in the past month and was infrequent in these adolescents. Thus models that discriminate different classes of drinkers or smokers could not be estimated with the data. It is possible GPE may have a stronger protective influence on problem drinking or regular smoking (versus chippers, experimenters or light drinkers), or with cessation from alcohol and tobacco problems (Ohannessian, Hesselbrock, Tennen, &

Affleck, 1994). For such outcomes, self-regulation processes may have enhanced importance and the influences of generalised expectancies from affect may be further distinguishable (Benyamini & Roziner, 2008). Finally, the findings showed DS could not explain GPE's health protective behavioural relations.

Within personality-health scholarship, GPE would be a middle-unit in Mischel and Shoda's (1995) taxonomy of social-cognitive traits and personality domains. Associations between dispositional optimism (and self-mastery) and domains most linked to health – neuroticism and conscientiousness, have been found to be of moderate magnitude in other studies (Marshall et al., 1994) and there is some evidence of optimism predicts health outcomes beyond neuroticism (e.g. Scheier et al., 1994). Further, when one contrasts developmental trajectories of domains in adolescence, there is further evidence that GPE is not merely a reflection of them. Research suggests conscientiousness increases and neuroticism decreases in this period (Bogg & Roberts, 2004; Klimstra et al., 2010; Lüdtke et al., 2009). Such patterns are inverse to what would be expected if GPE solely measured (low) neuroticism and (high) conscientiousness.

Embedded in moderately stable and perhaps mutable cognitive–social–motivational systems of behaviour, GPE may have application for health promotion research. This study show systemic instability and changes (decreases) in GPE, and some evidence of behaviours influencing later GPE levels. Potential environmental (e.g. poverty, positive educational settings and neighbourhoods) or social developmental influences (e.g. family, parenting, peers, teachers) that may affect persons' initial levels and changes in these expectancies should be further investigated (Aspinwall & Tedeschi, 2010; Ek et al., 2004; Heinonen et al., 2006; Heaven & Ciarrochi, 2008; Lagacé-Séguin & d'Entremont, 2010; Smith, 2006). There may also be downstream health promotion approaches where GPE are relevant. For instance Berg, Snyder, and Hamilton (2008) evidenced an effective 16 minute intervention that raised levels of trait hope and (objective) pain tolerance. Likewise Weis and Ash (2009) affirms identifying meaningful goals based on hope theory as a means to improve youth clients' therapeutic satisfaction as well as their health.

While not the focus of this investigation, it should be noted the fit of the unidimensional model of GPE was stronger at earlier data collections. In fact across studies dispositional optimism and hope appear increasingly differentiated as samples are older; and, uni-polar optimism, dispositional pessimism, agency and pathway components may also be differentiable in some populations (Bryant & Cvengros, 2004; Rand, 2009; Roesch & Vaughn, 2006; Schweizer & Rauch, 2008). Given the challenges of measuring dispositional optimism in youth (Carvajal et al., 2002; Ey et al., 2005; Lemola et al., 2010; Ráikkönen & Matthews, 2008), future studies may aim to try to improve the precision in measuring GPE distinct from positive self-views and consider potential unique contributions of specific subfactors.

There were important limitations and strengths of the study. Though the response rate was higher than the most comparable US health survey and the attrition rate met study objectives, the inability to collect data on all respondents is a limitation. However, as participants at all stages of this study were in grades lower than 9th, the study is not

susceptible to the exclusion of dropouts – whose omission is particularly problematic for making population inferences on adolescents' risk behaviours. Also, given the field setting and broad goals of the parent study, a comprehensive personality assessment and other related explanatory variables could not be employed. It should be noted however that the GPE measure had items (particularly two from the Children's Hope Scale) that are quite similar to those also represented within other predictive dispositional self-regulation measures; such as the general self-efficacy (e.g. Schwarzer & Fuchs, 1996; <http://userpage.fu-berlin.de/~health/engscal.htm>), the self-regulation competence questionnaire (Schröder & Schwarzer, 2001) and self-control (Wills et al., 2008). Further, the GPE measure derived from extensive prior work with adolescents, had high internal consistency, and measurement error was accounted for.

Though typical to population-based adolescent cohort studies, another limitation is that all data were from self-reports. The use of recent indicators of health behavioural measures validated in other studies was done to moderate these concerns. One particular weakness was the assessment of physical activity, which had modest reliability and did not discriminate activity contexts. Some contexts (e.g. physical activity unrelated to the standard school curricula) may lead to stronger inter-relations among activity and GPE. Future work should consider alternative measures (e.g. Arvidsson, Slinde, & Hulthén, 2005) more sensitive to individuals' motivation-tied changes in physical activity, such as those exhibited in leisure time.

A major strength was that the longitudinal data and analyses employed afford strong causal inferences from non-experimental designs. As all previous levels of GPE and behaviours are considered in the panel models, only the degree to which social desirability or other unmeasured constructs systematically change during the same period would provide alternative explanations for the findings. Also, the LG models provide a flexible and powerful approach to examine trajectories among groups and individuals on the study variables. Such models can be used to better understand dynamic properties of health outcomes (e.g. Benyamini et al., 2009; Curran et al., 1997; this study), 'distal' (Jessor et al., 2010) dispositional predictors (Heavin & Ciarrochi, 2008; this study), and proximal predictors (e.g. those grounded in social cognitive behavioural models like behavioural intentions or self-efficacy; Schroder & Schwarzer, 2005). With the availability of five or more observations (under some conditions with fewer), models integrating the strengths of LG and autoregressive models can also be modelled (Bollen & Curran, 2004). Despite their epistemological strengths, these methods however cannot replace the causal interpretations and intervention relevance of experimental research aiming to increase positive expectancies (or their determinants), and where subsequent effects on behaviours and cognitions are monitored (e.g. White et al., 2009).

In summary, this study extends the literature on GPE, and related constructs such as dispositional optimism and hope, by identifying that these expectancies are dynamic and predictive of future health-related behaviours. Relations with health protective behaviours were also distinct from DS and robust across diverse groups. Future research might consider investigating specific mechanisms in which GPE most influence health behaviours and include biological markers of adolescents' health. Such research could further illuminate

pathways leading adolescents to be more or less healthy adults and potential intervening variables to be targeted in health promotion programmes.

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Figure 1.

LG model of GPE. Overall model: $\chi^2(1) = 1.21$, $p = 0.27$, CFI = 1.00, RMSEA = 0.02 and GFI = 1.00.

Figure 2.

GPE and smoking LG models. Overall model: $\chi^2(7) = 7.21$, $p = 0.41$, CFI = 1.00, RMSEA = 0.01 and GFI = 1.00.

Figure 3.

GPE and alcohol LG models. Overall model: $\chi^2(7) = 11.23, p = 0.13, CFI = 0.99, RMSEA = 0.03$ and $GFI = 1.00$.

Figure 4.

GPE and healthy food choice LG models. Overall model $\chi^2(8) = 34.36$, $p = 0.001$, CFI = 0.97, RMSEA = 0.07 and GFI = 0.99.

Figure 5.

GPE (LG) and physical activity. Overall model $\chi^2(7) = 12.10$, $p = 0.10$, CFI = 0.99, RMSEA = 0.03 and GFI = 1.00.

Figure 6.

Cross-lagged model predicting cigarette smoking. Overall model S-B $\chi^2(14) = 13.97$, $p = 0.45$, CFI = 1.00, RMSEA = 0.02 and GFI = 1.00.

Figure 7.

Cross-lagged model predicting alcohol drinking. Overall model S-B $\chi^2(15) = 18.63$, $p = 0.23$, CFI = 1.00, RMSEA = 0.03 and GFI = 0.99.

Figure 8.

Cross-lagged model predicting health food choice. Overall model $\chi^2(14) = 16.78, p = 0.27$, CFI = 1.00, RMSEA = 0.02 and GFI = 0.99.

Figure 9.

Cross-lagged model predicting physical activity. Overall model: $\chi^2(13) = 13.35, p = 0.42$, CFI = 1.00, RMSEA = 0.02 and GFI = 1.00.

Table 1

Correlations and descriptive statistics for target study variables ($N=744$).

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1 GPE T1	0.93	0.50	0.40	-0.51	-0.38	-0.27	-0.15	-0.11	-0.12	-0.14	-0.11	-0.18	0.33	0.16	0.23	0.15	0.18	0.19
2 GPE T2	0.44	0.94	0.60	-0.31	-0.53	-0.42	-0.04	-0.15	-0.13	-0.05	-0.15	-0.23	0.22	0.31	0.24	0.06	0.30	0.27
3 GPE T3	0.35	0.54	0.95	-0.26	-0.39	-0.55	-0.06	-0.15	-0.16	-0.06	-0.08	-0.20	0.17	0.25	0.29	0.11	0.29	0.35
4 DS T1	-0.42	-0.26	-0.21	0.88	0.57	0.49	0.15	0.03	0.11	0.13	0.10	0.11	-0.08	-0.05	-0.10	-0.02	-0.08	-0.13
5 DS T2	-0.32	-0.44	-0.33	0.45	0.89	0.64	0.06	0.13	0.19	0.09	0.22	0.19	-0.12	-0.4	-0.09	-0.02	-0.13	-0.13
6 DS T3	-0.23	-0.36	-0.47	0.39	0.52	0.90	-0.03	0.08	0.27	0.07	0.16	0.32	-0.07	-0.06	-0.11	0.08	-0.11	-0.16
7 Cig T1	-0.13	-0.03	-0.04	0.12	0.05	-0.3	0.93	0.33	0.17	0.55	0.17	0.10	-0.03	-0.05	-0.06	0.01	0.04	-0.05
8 Cig T2	-0.10	-0.14	-0.13	0.03	0.11	0.07	0.29	0.94	0.31	0.26	0.38	0.23	-0.02	-0.03	-0.07	0.08	-0.01	-0.06
9 Cig T3	-0.11	-0.11	-0.15	0.10	0.16	0.24	0.15	0.28	0.95	0.15	0.27	0.50	-0.04	-0.06	-0.03	0.03	-0.07	-0.08
10 Alc T1	-0.12	-0.03	-0.09	0.10	0.08	0.04	0.48	0.23	0.13	0.93	0.31	0.28	-0.03	-0.05	-0.09	0.01	0.02	0.01
11 Alc T2	-0.09	-0.14	-0.13	0.09	0.20	0.15	0.15	0.34	0.24	0.27	0.94	0.43	-0.04	-0.08	-0.08	0.00	-0.03	-0.5
12 Alc T3	-0.15	-0.20	-0.21	0.09	0.18	0.29	0.07	0.20	0.45	0.23	0.39	0.95	-0.03	-0.06	-0.05	0.03	-0.05	-0.05
13 HF T1	0.28	0.19	0.20	-0.06	-0.10	-0.05	-0.03	-0.02	-0.03	-0.03	-0.04	-0.03	0.90	0.43	0.47	0.54	0.30	0.32
14 HF T2	0.14	0.28	0.23	-0.04	-0.04	-0.05	-0.04	-0.02	-0.05	-0.4	-0.08	-0.05	0.37	0.93	0.58	0.27	0.52	0.38
15 HF T3	0.20	0.22	0.27	-0.09	-0.08	-0.09	-0.05	-0.06	-0.03	-0.08	-0.07	-0.04	0.41	0.49	0.93	0.26	0.37	0.52
16 PA T1	0.12	0.04	0.09	-0.01	-0.01	-0.07	0.01	0.07	0.03	0.00	0.00	0.03	0.40	0.20	0.20	0.81	0.40	0.42
17 PA T2	0.15	0.23	0.22	-0.06	-0.09	-0.08	0.03	-0.01	-0.06	0.02	-0.03	-0.04	0.23	0.40	0.29	0.26	0.83	0.58
18 PA T3	0.17	0.20	0.28	-0.11	-0.10	-0.13	-0.04	-0.06	-0.07	0.01	-0.04	-0.05	0.24	0.27	0.40	0.28	0.40	0.83
M	3.14	3.07	2.97	1.97	2.00	2.12	0.04	0.06	0.09	0.05	0.09	0.11	4.27	4.22	3.48	4.82	4.86	4.83
SD	0.58	0.62	0.64	0.77	0.78	0.82	0.19	0.22	0.27	0.16	0.21	0.24	3.48	3.52	3.23	1.71	1.77	1.81
SKEW	-0.77	-0.48	-0.03	1.12	0.99	0.77	5.27	3.94	3.24	3.21	2.13	1.69	1.35	1.32	1.74	-0.10	-0.01	-0.03
Range	1-4	1-4	1-4	1-5	1-5	1-5	0-2	0-2	0-2	0-8	0-8	0-8	0-15	0-15	0-15	1-8	1-8	1-8

Notes: The diagonal represents the fixed estimates used in EQS to adjust for measurement error. Estimates below the diagonal represent raw zero-order correlations among study variables. Estimates above the diagonal represent zero-order correlations adjusted for attenuation (estimates for alcohol use were approximated based on internal consistency estimates for cigarette smoking).

GPE, global positive expectancies; DS, depressive symptomatology; cigarette smoking (Log) (Cig); alcohol drinking (Log)(Alc); HF, healthy food choice; PA, physical activity, Wave I (T1); Wave II (T2); and Wave III (T3).

For $r > |0.07|$, $p < 0.05$; $r > |0.09|$, $p < 0.01$; $r > |0.12|$, $p < 0.001$.