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## FEELING GOOD: HAPPINESS AND HEALTH IN DUTCH OLDER ADULTS

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FEELING GOOD: HAPPINESS AND HEALTH IN DUTCH OLDER ADULTS

BY

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## **Abstract**

Statement of the problem: Positive psychology research has sought to identify cost efficient and intuitive ways to increase subjective happiness, with broad implications to personal health and overall quality of life (Lee Duckworth, Steen, & Seligman, 2005; Rashid, 2009; Seligman, 2008; Seligman, Ernst, Gillham, Reivich, & Linkins, 2009; Seligman, Rashid, & Parks, 2006). A complication in this research has been conflicting findings in the longitudinal stability of happiness (Easterlin, 2006). This analysis examines age related changes in happiness and physiological immune responsiveness to clarify the implications of positive health interventions.

Methods: Pretest-posttest structural equation modeling is employed to examine longitudinal association. Data from the Longitudinal Aging Study Amsterdam are analyzed (Hoogendijk et al, 2020). Operationalizations of two theories of happiness as well as interleukin-6, a measure of physiological inflammation, are considered.

Associations are examined between baseline rates and changes at follow-up. In addition to estimating associations among the primary outcome variables, age is also included as a predictor, in addition to possible confounds.

Summary of results: Consistent with hypotheses, significant positive associations were identified among changes in happiness and inflammation response. Conversely, relationships with age were nonsignificant or directionally opposite from the positive association identified in previous literature. It is suggested that while lifelong deterministic trends in subjective experience and inflammation may plateau during older adulthood, the variables may still demonstrate meaningful association. This analysis provides a methodically rigorous comparison of two leading theories of subjective well-

being. It is concluded that feelings of social support and connection may mitigate some of the challenges faced by older adults, such as the mortality of friends and family.

Further, this could help older adults facilitate positive aging and longevity.

## **Acknowledgements**

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## **Introduction**

In discussing modern theories of happiness, the work of positive psychology founder Martin Seligman provides a conceptual foundation (Diener, 2009; Kaczor, 2015; Seligman & Csikszentmihalyi, 2000; Seligman, Steen, Park, & Peterson, 2005). Though revisions have been made over the years, the focus of the theory centers on five essential sources of happiness: positive affect, engagement, relationships, meaning, and achievement (Scorsolini-Comin, Fontaine, Koller, & Santos, 2013; Seligman, 2002, 2012). Positive affect refers to the subjective experience of enjoyment (Seligman, 2002). Engagement is largely based on Mihaly Csikszentmihalyi's (1990) theory of flow, which describes a state of focus where personal skills are maximized to accomplish a challenging task. Relationships address the importance of friends and family members to personal well-being (Demir & Weitekamp, 2007). Meaning emphasizes the importance of a sense of purpose and connection to something bigger than the self (Steger, 2009). Lastly, achievement accounts for the need for personal accomplishment beyond a general positive attitude (Walker, Winn, & Lutjens, 2012). Seligman (2012) suggests that improvements in any of these sources of happiness should improve overall well-being. Within a public health methodology, interventions to increase happiness have been proposed, implemented, and evaluated (Lee Duckworth, Steen, & Seligman, 2005; Rashid, 2009; Seligman, Ernst, Gillham, Reivich, & Linkins, 2009; Seligman, Rashid, & Parks, 2006).

Responding to Seligman's (2002, 2012) work, the stability of happiness has been raised as a subject of interest (Easterlin, 2006). Seligman (2012) had originally focused on happiness as a personality (Peterson, Park, & Seligman, 2005; Peterson, Ruch,

Beermann, Park, & Seligman, 2007; Scorsolini-Comin, Fontaine, Koller, & Santos, 2013). Defining happiness as a personality, a stability to happiness is assumed, a perspective supported by other theories. Lyubomirsky (2007, 2013) is an advocate for the theory of the hedonic treadmill, which argues happiness is more or less fixed to a set range within individuals over the lifespan. One implication of this theory is that what may appear to be a large personal setback will eventually be mitigated over time; this has been empirically supported (Luhmann, Hofmann, Eid, & Lucas, 2012; Lyubomirsky, 2013; Uglanova & Staudinger, 2013). The goal of this research has been to identify the behaviors that increase subjective happiness within the set range, in order to help people live at the high end of their range (Jacobs Bao & Lyubomirsky, 2015; Layous, & Lyubomirsky, 2014; Lyubomirsky, 2010; Lyubomirsky, Dickerhoof, Boehm, & Sheldon, 2011; Lyubomirsky, Sheldon, & Schkade, 2005; Sheldon, Boehm, & Lyubomirsky, 2013).

In an effort to examine the more personal aspects of happiness, Fredrickson (2001, 2004a, 2013) created the broaden-and-build theory of positive emotions. The foundation of this theory is that positive experiences beget greater number and greater qualitative diversity of future positive experiences in an upward spiral of personal growth (Fredrickson, 2001, 2004a, 2013). This process is viewed as a psychological change toward increasing awareness of positive experiences (Fredrickson, 2002, 2004b; Fredrickson & Branigan, 2005; Fredrickson & Joiner, 2002). Additionally, physiological implications have been identified, especially through what Fredrickson (2013) calls the vagal tone. The vagus nerve is physiologically involved with coordinating basic processes of functioning, such as breathing and heart rate (Rosenzweig, Leiman, &

Breedlove, 1996). The responsiveness of the vagus nerve, or vagal tone, has been described as a neurological indicator of stress reactivity (Porges, 1995). Previous literature has focused on how greater reported happiness often indicates calmer vagal tones and less physiological stress response (Fredrickson, 2013; Fredrickson, Mancuso, Branigan, & Tugade, 2000; Kok et al, 2013; Kok, B. E., & Fredrickson, 2010).

These theories share similar epistemological foundations, including a scientific methodology, a focus on the positive aspects of personal well-being, and the goal of increasing happiness via interventions (Gable & Haidt, 2005). Despite this, theoretical implications related to change over time are inconsistent. Seligman (2012) does not directly address the issue of personal changes in happiness over time, but rather focuses on sources that predict personal happiness. Lyubomirsky (2007, 2013) is more deterministic, suggesting that happiness is generally resistant to change. Fredrickson (2013) implies that increases in happiness are expected and self-perpetuating. A consideration is that different authors have differing conceptualizations and measurement of happiness as a phenomenon, a criticism that has been raised of positive psychology research (Froh, 2004; Gable & Haidt, 2005; Held, 2004; Pawelski, 2016; Waterman, 2007, 2013).

To resolve inconsistencies, Winston (2016) created the existential-humanistic-positive theory of human motivation, which identified similarities between theories in the history of psychology on similar subjects. Specifically, she suggests that there is an underlying psychological development across the personality-based theory of happiness from Seligman (2002), humanistic need fulfillment from Maslow (1987), existential despair by Kierkegaard (1989), and sense of morality described by Kohlberg (1984). An

entirely theoretical writing, Winston (2016) suggests that there is a broader personal transition in subjective worldview that transcends these theories. She argues that development starts with a selfish need for immediate pleasure and ends with deep satisfaction derived from a selfless sense of holistic interconnection.

A strength of Winston's (2016) theory is that it provides an explanation for these differing results. The focus is not on the absolute volume of experienced happiness, but rather the phenomenon of happiness experienced. This theory allows for both Lyubomirsky's (2007, 2013) modest improvements in happiness person to person, and Fredrickson's (2013) broad cycles of individual growth. According to Winston (2016), a person will experience meaningful growth over the life course, similar to how Fredrickson (2013) describes. That said, as Lyubomirsky (2007, 2013) has found, this may not result in a very large change in overall happiness, at most a slight increase proportional to previously reported happiness. Instead, the change will be in relation to sources of happiness, as described by Seligman (2012). In Winston's (2016) theory, the first priority will be on positive affect, shifting toward engagement and achievements, and finally ending with a focus on meaning and relationships.

The largest limitation of Winston's (2016) theory is that it is entirely theoretical, with no data analysis provided as support. There have since been some empirical studies examining Winston's (2016) theory, examining possible developmental and physiological correlates. Consistent with both theories of happiness: data from a large sample of Americans revealed that older age predicted a more developed conceptualization of happiness per Winston (2016), but had minimal relationship with general happiness as defined by Lyubomirsky (2007, 2013; Tanzer, 2019a). A more

direct investigation by Tanzer (2019b) provided a preliminary examination of the empirical plausibility of Winston's (2016) theory in a small sample of university students. Canonical correlation analysis was used to examine relationships between questionnaires of happiness with biomarkers of stress response. The results suggested that there may be some plausibility to Winston's (2016) grouped definitions of happiness. Further, it provided some evidence of a relationship between happiness and stress response such that a more mature experience of self-reported happiness correlated with a reduced physiological stress response in that sample.

This point was further examined in a meta-analysis of neuroimaging research on Winston's (2016) forms of happiness. Discrete cerebral activation likelihoods were identified based on how happiness was defined (Tanzer & Weyandt, 2019). A less developed conceptualization of happiness tended to co-occur with activation of the insula and amygdala, structures closely related to stress response signaling (Rosenzweig, Leiman, & Breedlove, 1996). A more developed conceptualization of happiness tended to co-occur with activity in the prefrontal cortex and insula, but not the amygdala. The insula has been described as physiologically processing disgust, and the amygdala as processing fear (Rosenzweig, Leiman, & Breedlove, 1996). A less mature conceptualization of happiness was more reactive to external stimuli, demonstrated by activation in the amygdala. A more mature conceptualization of happiness started with similar neurological pathways in the insula, but the signal tends to get cut off before the amygdala initiates a bodily stress response (Tanzer & Weyandt, 2019). It was concluded that there was some evidence that Winston's (2016) developmental progression can be identified.

These results together provide some broad support for Winston's (2016) theory, however they are imprecise. One study was able to account for longer-term development, however it did not account for physiological origins (Tanzer, 2019a). One study included direct measures of happiness and physiological response, however the sample was small and all under the age of 30 (Tanzer, 2019b). The meta-analysis was able to examine physiological underpinnings, however there were few age-based or longitudinal designs identified in the literature (Tanzer & Weyandt, 2019). The present analysis will seek to examine all of these variables in context: older age, two timepoints, measures of happiness, and a measure of physiological stress response. This will allow for a more detailed and generalizable investigation of Winston's (2016) theory within personal context.

### **Methods**

**Participants.** The sample analyzed is of older adults from the Longitudinal Aging Study Amsterdam (LASA; Hoogendijk et al, 2020). The LASA began in 1992 and has been continuously collecting data ever since (Hoogendijk et al, 2020). The original sample included 3,107 Dutch older adults who had previously participated in the Living Arrangements and Social Networks of older adults (LSN) study (Knipscheer, Jong-Gierveld, van Tilburg, & Dykstra, 1995). The sample was intended to be nationally representative. Specifically, the cities of Zwolle, Oss, and Amsterdam were sampled to account for known population densities relating to religious history (Protestantism, Catholicism, and secularization). Table 1 presents the sample characteristics.

**Materials.** Two subjective measures related to happiness were included in the analysis, both extracted from face-to-face interviews conducted in Dutch using validated scale

translations (Hoogendijk et al, 2020). First, a subset of the Centers for Epidemiological Studies Depression scale (CES-D) was used (Radloff, 1977). Although the whole scale is intended to measure depression, there are four items that are reverse scored and directly measure happiness (“I felt just as good as other people,” “I felt hopeful about the future,” “I was happy,” and “I enjoyed life.”). Items are scored ordinally representing the weekly frequency of feeling happy. Specifically, 1 represents “rarely or none of the time (less than one day),” 2 represents “some or little of the time (1-2 days),” 3 represents “occasionally or a moderate amount of time (3-4days),” and 4 represents “most or all of the time (5-7 days).” Internal consistency for the happiness-only subscale has been calculated to be appropriate within the LASA sample ( $\omega=0.755$ ; Zhang et al, 2011). Additionally, factor analyses generally identify the positive affect subscale as loading onto a separate factor from the negative affect items (Shafer, 2006). This scale was taken as representing a general happiness such is often measured in the theory of the hedonic treadmill (Lyubomirsky, 2007; Waterman, 2007).

The second subjective measure was a rating of social support. During structured interviews, participants were prompted to list as many contacts as they could remember specifically related to the following domains of social interactions: household, children/-in-law, other kin, neighborhood, work/school, organizations, and other non-kin. For the twelve most influential people, participants were then asked to rate them across four modalities of social support: giving instrumental support, receiving instrumental support, giving emotional support, and receiving emotional support. Responses were rated from 0 (representing never giving nor receiving support) to 3 (representing often giving or receiving support).



Ratings of social support have been identified as related to older adult happiness and well-being (Ferreira & Sherman, 2007; Kafetsios & Sideridis, 2006; Krause, 1986; McAuley et al, 2000; Thomas, 2009). More importantly, this measure was selected because it can account for developments in Winston's (2016) conceptualization of happiness. A more mature view of happiness is described as prioritizing relationships and an acceptance of unpleasantness. Participants with a more mature view of happiness would likely have more active social support networks, especially so far as it relates to giving social support. It is not always enjoyable to provide social support to another, however, according to Winston (2016), a mature view of happiness would suggest that it is worth the investment. This helps identify developments in subjective happiness contrasted with the general happiness discussed by Lyubomirsky (2007) and measured by the CES-D four item happiness subscale.

In addition to these two subjective constructs, two objective variables were included, specifically interleukin-6 and age. Interleukin-6 is an inflammation cytokine that modulates stress response (Malven, 1993). Previous work by Tanzer (2019b) indicated this biomarker as a correlate of happiness as defined by Winston (2016). Interleukin-6 was extracted from saliva samples, which were initially centrifuged after collection and stored at -80° C until being determined in 2004. Age was selected because Winston's (2016) theory is a developmental theory. Additionally, over the course of the lifespan, physiological inflammation and stress response undergo many changes (Graham, Christian, & Kiecolt-Glaser, 2006). Parallel, age-related developmental trajectories of interleukin-6 and happiness will be the focus of this analysis, to clarify the dynamic interrelationships between these aspects of well-being.

Lastly, while these four variables account for the primary research questions, some possible confounders were included in the model to identify how robust the findings may be to statistical isolation. Subjective ratings of personal health and personal health relative to peers were included, each measured as a scale from 1 to 5. As an objective measure of personal physique, the Activities of Daily Living index was included (Katz, Ford, Moskowitz, Jackson, & Jaffe, 1963; Shelkey & Wallace, 2012). This measure entails rating a series of activities from 0 (representing “task can be completed without difficulty”) to 4 (representing “task cannot be completed”). Example activities include climbing stairs, walking, and getting dressed. Lastly, to account for more general social context, the number of years of education and gender binary identification (measured as male or female) were also included. Accounting for these additional covariates will provide a check on how robust the results for the primary research question are when accounting for possible confounds.

**Analytic strategy.** Structural equation modeling will be used as described by Raykov (1992). Structural equation modeling is a regression-based method that examines the patterns of variances and covariances within a hypothesized model specified by the researcher (Harlow, 2014). Regression predictions can be specified, as well as item loadings onto latent variables, called factors. The analytic specification for a pretest-posttest design described by Raykov (1992) includes two factors estimated for each construct. The factors represent baseline value and change at follow-up. Baseline observations of the same construct only load onto the baseline factor. The follow-up observations load onto both the baseline factor and the change factor. This specification was designed in this way because the baseline factor represents a latent mean value,

which informs both exogenous measures at baseline and exogenous measures at follow-up. The change factor, on the other hand, represents difference at follow-up, which does not predict already measured baseline values. Lastly, errors between repeated observations of the same measure are correlated to properly estimate the model. This allows for an examination of how participants changed from baseline to follow up, as well as the possible inclusion of exogenous predictors of both baseline and change components. Figure 1 provides Raykov's (1992) original diagrams for representing these pretest-posttest models.

Within longitudinal structural equation modeling, multiple trends can be fit at once to examine how multiple variables change together over time, called a parallel process model (Cheong, MacKinnon, & Khoo, 2003). Parallel process models are an extension of other longitudinal models, where each variable is fit as its own change process, however the factor error terms, called disturbances, are covaried. A significant covariance indicates a meaningful relationship between the two latent terms. For example, if there is a significant positive covariance between two baseline factors, that would indicate that participants with high scores on the first variable at baseline tended to also have high scores on the second variable at baseline. As another example, a significant negative relationship between the baseline factor and a change factor would indicate that participants who had high values on the first variable at baseline tended to decrease at follow-up for the second variable. Longitudinal interrelationships can be examined in detail using the parallel process model design.

Figure 2 represents the model that will be fit in this research analysis. There are three longitudinal models as described by Raykov (1992): 1) a multiple item model for

self-reported social support ratings; 2) a single item model for general happiness as measured by the CES-D happiness subscale; and, 3) a single item model for interleukin-6. This results in six latent factors, for baseline and change for each of these constructs. Additionally, age is specified in the model to predict each of these factors. This will indicate whether or not older participants were more likely to start at higher values than younger participants, as well as whether or not older participants will increase in these variables more than younger participants. Factor disturbances between all of them will be covaried. This will provide statistical tests as to which variables were associated over time. Lastly, after initial model estimation, the covariates will be added predicting all of the factors to see how consistent results were with and without accounting for possible confounds.

**Hypotheses.** The goal of this analysis is to compare theories of happiness over the course of the lifespan. Some pragmatic changes are expected, such as age-based changes to health and social structure. The oldest adults face a unique difficulty relating to social support, such that mortality of friends and family may pose a significant threat to support and well-being (Baird, Lucas, & Donnellan, 2010). It is hypothesized that age will predict a negative change in social support. It is also hypothesized that age will predict higher levels of interleukin-6 at baseline, as has been observed and described as a normal physiological development with age (Graham, Christian, & Kiecolt-Glaser, 2006).

It is hypothesized that there will be a nonsignificant or small relationship between age and happiness as measured by the CES-D at baseline and follow-up, theoretically in line with the theory of the hedonic treadmill (Lyubomirsky, 2007). The focus of this analysis, however will be in theoretically testing Winston's (2016) theory through

longitudinal mediation. It is hypothesized that age will predict baseline levels of subjective social support, and the disturbance on the baseline levels of social support factor will have a positive covariance with the change in happiness factor disturbance, and a negative covariance with the change in interleukin-6 factor disturbance.

Relationships of this pattern would suggest that older participants are more likely to have a more mature conception of happiness, resulting in improvements in happiness and reduction in stress response as suggested by Winston (2016) and Tanzer (Tanzer, 2019a, 2019b; Tanzer & Weyandt, 2019;). This will be specifically tested by constraining the parameters to zero and assessing how this impacts model fit. If the chi-squared value increases significantly, the RMSEA increases, or CFI decreases when leaving out relevant paths, then it will be concluded that these paths are important to include and that Winston's (2016) theory would have some support in the data (Harlow, 2014).

To some extent, this is an exploratory analysis for other relationships among the variables. Beyond testing the hypothesized longitudinal mediation, additional longitudinal associations can be identified within the model. For example, if there is a positive covariance between changes in social support and changes in interleukin-6, this may indicate a cross-sectional closeness between Winston's (2016) theory of happiness and physiological stress response, as suggested by Tanzer (2019b). Relationships of this nature will be explored outside of the specified hypotheses.

**Analysis diagnostics.** With a longitudinal design, loss to follow-up is always a risk to validity. Within this sample, the baseline included 3,107 participants, the second wave included 2,545, 81.91%, of the original sample. To address this, the LASA intentionally oversampled older adults, to preempt expected heterogeneous loss to follow-up given

age. Additionally, missing data imputation was used (Little & Rubin, 2019). Figure 3 is a flow chart describing the specific steps of the analysis, to statistically account for these limitations in the data, and the measures taken to examine threats to validity they pose. The bootstrapped estimates of the model were used, wherein the parameter estimates are based on reiterative resamples of the data. For 100 iterations, 3,107 random participants from the dataset were sampled with replacement to generate 100 unique datasets containing characteristics proportional to those of the original dataset. Previous research has suggested that this should be a sufficient number of bootstrap samples with a sample as large as this (Efron, & Tibshirani, 1994; Hall, 1988). For each resampled dataset, all missing values were filled in with conditional draws. Conditional draws are based on a regression equation fit to predict each variable in the dataset. Based on the parameter estimates, each missing value is imputed as the expected value for each individual, plus random error is added based on the variance of the variable being imputed. This fits each missing value with the most likely value while adding in random variation, as would be expected for any measurement. Lastly, the structural equation model was estimated using the complete imputed dataset, and the parameter estimates were recorded. The final model estimates which are reported are based on the mean of all 100 bootstrapped model estimates, and standard error was based on the variance of the bootstrapped model estimates.

There also was concern that some of the values for interleukin-6 may be unreliable. Due to the sensitivity of the saliva assay, the lowest values in the dataset were below the threshold of reliable detection. This problem affected 51.24% of the baseline samples and 36.69% of the follow-up samples. To address this, the unreliable estimates

were treated like missing data. After all missing data imputations had been completed, the unreliable values were also replaced with conditional draws. This allowed for a correction to the possible unreliable values that had been originally observed.

Lastly, it was important to verify the model design was appropriate for the data. Before adding covariates, there are 13 exogenous variables in the model, which means there are 91 variance and covariances estimated. There are 57 parameters in the model, which means that there are 34 degrees of freedom in the model, which should be sufficient. Additionally, ignoring participants lost to follow-up to be conservative, there remain 2,545 participants in the dataset. This is well above the general recommendation that structural equation modeling should have at least 300-400 participants (Harlow, 2014). Power contrasting nested structural equation models can be calculated using G\*Power (Faul, Erdfelder, Lang, & Buchner, 2007). The noncentrality parameter lambda with a chi-squared distribution can be calculated as a function of sample size, degrees of freedom between the two models, and RMSEA values for each model (Hermida, Luchman, Nicolaidis, & Wilcox, 2015). Initial model fit was estimated as RMSEA=0.10, 0.07, and 0.05. Additionally, change in model fit was estimated as  $\Delta$ RMSEA=0.01, 0.02, and 0.03. Across all nine conditions, power was estimated to be exceptional (power>0.999). With sufficient degrees of freedom and strong power, the hypothesized model was concluded to be appropriate for the analysis.

## **Results**

**Data diagnostics.** First all variables were examined for skewness and kurtosis (see Table 1). Because errors are modeled as normally distributed, skewness greater than |1.0| or kurtosis greater than 2.0 may be at risk for improper estimation, although sample

estimates | for skewness or kurtosis may be acceptable if even somewhat larger (Harlow, 2014). All variables were appropriately distributed except for interleukin-6, which showed high skewness and kurtosis at both timepoints. The model was fit with the log transformed values, but this did not meaningfully impact the results, so the untransformed values were used in the final reported model.

**Social support diagnostics.** Next, the sample was randomly split in half, so that a portion of the sample could be examined and then validated in the second portion of the sample. First, the social support scale was examined. It was expected that all four items load onto a single social support factor. This model fit the data reasonably well (CFI = 0.93, 95% CI [0.90, 0.96]; RMSEA = 0.07, 90% CI [0.06, 0.08], SMRM = 0.04, 95% CI [0.04, 0.05]). All loadings were highly significant ( $p < 0.0001$ ), however it was clear that the scale measured emotional support (baseline: giving,  $\lambda = 0.66$ ; receiving,  $\lambda = 0.60$ ; follow-up: giving,  $\lambda = 0.58$ ; receiving,  $\lambda = 0.59$ ) more saliently than instrumental support (baseline: giving,  $\lambda = 0.19$ ; receiving,  $\lambda = 0.17$ ; follow-up: giving,  $\lambda = 0.16$ ; receiving,  $\lambda = 0.21$ ). Importantly, giving and receiving support were similarly measured, indicating that this may be a valid operationalization on Winston's (2016) theory. Reliability for the measure was not strong, but sufficient (coefficient omega = 0.72, 95% CI [0.69, 0.74]).

**Interleukin-6 diagnostics.** Because there was concern that some of the estimates for interleukin-6 were unreliable, a brief analysis of validity was performed. As previously discussed, it was expected that inflammation would be increased among older participants. As a diagnostic check, the correlation between age and interleukin-6 was estimated. Additionally, available to the researchers was C reactive protein, another



inflammation biomarker, that would also be expected to have a positive correlation with interleukin-6 if the variable were modeled properly. Five missing data techniques were used to estimate the correlation: pairwise complete cases, pairwise complete cases with unreliable values of interleukin-6 removed, bootstrapped multiple imputation with no bias to imputations, bootstrapped multiple imputation with high bias to imputations, and multiple imputation with low bias to imputations. The average correlation values and mean squared error of estimate are reported in Table 2.

Contrary to expectations, age had minimal association with interleukin-6 or C reactive protein. This is most surprising for C reactive protein, which had some missing data but no concern of unreliable quantification from assays. Despite this, while correlations were small, the measurements of biomaterial supported convergent validity for interleukin-6. The strongest correlation was between the repeated observations of interleukin-6 (baseline-follow-up,  $r = 0.19$ ,  $p < 0.0001$ ), followed by the correlations between interleukin-6 and C reactive protein at the same timepoint (baseline,  $r = 0.13$ ,  $p < 0.0001$ ; and follow-up,  $r = 0.14$ ,  $p < 0.0001$ ). Discriminant validity was also demonstrated, as the correlations between interleukin-6 and C reactive protein at different timepoints were near zero and not significant (Interleukin-6<sub>1</sub>-C reactive protein<sub>2</sub>,  $r = 0.04$ ,  $p = 0.1519$ ; Interleukin-6<sub>2</sub>-C reactive protein<sub>1</sub>,  $r = 0.01$ ,  $p = 0.8095$ ). This suggests that while the age trajectories were counterintuitive, the inflammation response that was measured appeared to be as expected despite some concern of unreliability.

At this point, the structural equation model was fit. Before interpreting the results, a sensitivity analysis was performed on the imputation. The imputations were intentionally biased high and low, by 0.8 standard deviations, reflective of a large effect

per Cohen (1992). No changes to estimate significance were identified and model fit was essentially the same (i.e., difference in  $X^2 < 2$ ). This suggests that parameter estimate bias was not likely dependent on missing data and the interleukin-6 correction, so the final model results reported are those using the unbiased model imputations. At this point, the model was analyzed.

**Statistical Model.** Model fit estimates and hypothesis tests are reported in Table 3, Table 4 presents the developmental effects, and Table 5 provides the covariate effect estimates. Most models demonstrated strong model fit (i.e.,  $X^2/df < 5.00$ , CFI  $> 0.95$ , RMSEA  $< 0.05$ , SRMR  $< 0.05$ ). The hypothesized model was especially well fitting (model 0,  $X^2/df = 2.70$ ; CFI  $> 0.99$ , 95% CI [0.97, 1.00]; RMSEA = 0.03, 90% CI [0.01, 0.05]; SRMR = 0.02, 95% CI [0.01, 0.02]). The addition of covariates was clearly not parsimonious, with large increases to the chi-squared value (model 1,  $X^2/df = 9.55$ ; difference,  $X^2(10) = 143.43$ ,  $p < 0.0001$ ) and decreased model fit by the CFI (0.89, 95% CI [0.85, 0.93]) and RMSEA (0.07, 90% CI [0.06, 0.09]).

Next, specific hypotheses were tested by constraining model parameters to be zero. First, longitudinal mediation was tested, testing the path from age to baseline social support, and the associations between baseline social support and change in happiness and interleukin-6. If the model fit well, this would be taken as evidence for Winston's (2016) theory. This resulted in significant increases in the chi-squared statistic (model 2,  $X^2/df = 9.55$ ; difference,  $X^2(3) = 50.60$ ,  $p < 0.0001$ ) and RMSEA (0.06, 90% CI [0.04, 0.08]) and decreases in fit by the CFI (0.93, 95% CI [0.88, 0.97]). Looking at the specific parameter estimates, counter to expectations, the path from age to baseline happiness was significant and negative ( $\gamma = -0.28$ ,  $p < 0.0001$ ). The covariances were very small

and not significant (social support baseline, happiness change  $\psi < 0.01$ ,  $p = 0.9840$ ; social support baseline, interleukin-6 change  $\psi = 0.06$ ,  $p = 0.8572$ ). As a follow up test of longitudinal mediation, the effect of age was left free to be estimated and the two nonsignificant factor covariances were constrained to zero. Despite nonsignificant parameter estimates, assuming orthogonal relationships was significantly detrimental to model fit (model 3,  $X^2/df = 3.11$ ; difference,  $X^2(2) = 9.03$ ,  $p = 0.0109$ ), however CFI and SRMR were similar between models but with wider confidence intervals (CFI = 0.98, 95% CI [0.93, 1.02]; SRMR = 0.02, 95% CI [0.00, 0.04]). This upholds support for longitudinal mediation, even in the absence of the significant age effect.

Next, the hypothesis was tested to see whether or not there was common variation among baseline factors. All three covariances were constrained to zero, however this significantly reduced model fit (model 4,  $X^2/df = 3.27$ ; difference,  $X^2(3) = 13.74$ ,  $p = 0.0033$ ; CFI = 0.97, 95% CI [0.95, 0.99]; RMSEA = 0.04, 90% CI [0.02, 0.05]; SRMR = 0.02, 95% CI [0.02, 0.03]). Examining individual parameter estimates, the strongest relationship was between social support and interleukin-6 ( $\psi = -0.29$ ,  $p = 0.0004$ ), with extremely small and nonsignificant relationships between social support and happiness ( $\psi = -0.02$ ,  $p = 0.6312$ ) and between happiness and interleukin-6 ( $\psi = 0.03$ ,  $p = 0.5619$ ). All age effects were significant and negative (social support,  $\gamma = -0.28$ ,  $p < 0.0001$ ; happiness,  $\gamma = -0.11$ ,  $p < 0.0001$ ; interleukin-6,  $\gamma = -0.17$ ,  $p = 0.0034$ ).

To see whether or not there was common variation among changes, these covariance parameters were next constrained to zero. This change improved model fit slightly (model 5,  $X^2/df = 1.82$ ; difference,  $X^2(3) = 0.73$ ,  $p = 0.8661$ ; CFI = 0.99, 95% CI

[0.97, 1.01]; RMSEA = 0.02, 90% CI [0.00, 0.04]; SRMR = 0.02, 95% CI [0.01, 0.02]). None of the covariance estimates were significant. Together, this suggests changes within individual were not meaningfully associated over time. The only significant association with age was in relation to change in happiness ( $\gamma = -0.07, p = 0.0060$ ), however this effect was reduced to near zero when covariates were added ( $\gamma = -0.03, p = 0.8181$ ).

At this point, covariates were added. The effect of age on change in happiness was the only effect that was significantly changed by this addition. Although the model including all covariates was clearly not parsimonious, it was worth examining the individual effects of the covariates that stood out. The covariates with the strongest relationships were the subjective health ratings variables, with health rating significantly associated with baseline happiness ( $\gamma = -0.34, p < 0.0001$ ), and health ratings relative to peers significantly associated with baseline social support ( $\gamma = 0.09, p = 0.0349$ ), change in happiness ( $\gamma = 0.06, p = 0.0466$ ), and baseline interleukin-6 ( $\gamma = -0.13, p = 0.0178$ ). Lastly, female gender identity was associated with lower ratings of baseline happiness ( $\gamma = -0.11, p < 0.0001$ ) and years of education was positively associated with baseline happiness ( $\gamma = 0.05, p = 0.0188$ ).

## **Discussion**

The goal of this analysis was to compare the developmental trajectories of two theories of happiness. There was specific interest in evaluating whether or not there might be longitudinal mediation such that older adults have a more mature conception of happiness as discussed by Winston (2016), later demonstrating increased happiness as discussed by Lyubomirsky (2007, 2013) and reduced inflammation response. There was

evidence of longitudinal mediation, however the association with age was opposite from what was expected. Older respondents tended to have lower ratings of social support, contrary to expectations. Despite this, participants with higher ratings of social support did indicate higher ratings of happiness.

Unexpected relationships with age were also demonstrated with stress responsiveness. Whereas it is often demonstrated that inflammation increases with age, the model identified older participants as having reduced inflammation response, and when assessing the validity of the measures associations with age were minimal. This may suggest that lifelong trajectories related to inflammation responsiveness and subjective happiness are not unilateral. This sample specifically included older adults, so the age-based relationships may no longer be clearly positive in association. One possible explanation is due to mortality. The oldest living adults among a group of older adults may have disproportionately low inflammation expression which would otherwise be mitigated in a more general sample of all ages. An analysis of people across the entire lifespan may show more expected developmental trajectories.

While age-based associations were different than what was expected, there were clear distinctions between modeling elements related to Winston's (2016) and Lyubomirsky's (2007, 2013) theories of happiness. Specifically, social support, an operationalization of Winston's (2016) theory, showed a closer relationship to inflammation activity than did happiness, an operationalization of Lyubomirsky's (2007, 2013) theory. Whether it was giving or receiving social support, the more respondents endorsed social support, the less inflammation response they tended to have, in line with the hypothesis.

Conversely, happiness that was operationalized in Lyubomirsky's (2007, 2013) theory showed much closer relationships to the covariates. What was an association with age disappeared when covariates were added. Further, differences were identified by gender identity (women tending toward less happiness), education (more educated respondents tending toward more happiness), and health ratings (healthier identifying respondents starting lower on happiness but increasing slightly over time). This is important because it demonstrates the relevance of circumstance to the Lyubomirsky's (2007, 2013) construct of happiness. This construct has been characterized by temporal stability, although the theory has attributed a significant portion of variation in happiness to personal circumstance (Lyubomirsky, 2007, 2013). This result adds support to the relevance of circumstance, even if individual happiness is fairly consistent over time.

These results highlight the ways these different phenomena of happiness warrant alternative considerations within older adult populations. Previous literature has raised concern about how older adults are higher risk for circumstantial negative life changes (e.g., loss of friends and family members; Baird, Lucas, & Donnellan, 2010). These results support this concern for subjective well-being, however the results also indicate that these circumstantial changes may be less of a risk for public health of older adults. Feelings of social support and rich satisfaction may be more relevant to the physical health of this group, as has been suggested in previous research (Chen & Feeley, 2014; Fiori & Jager, 2012; Montpetit, Nelson, & Tiberio, 2017). Identifying and evaluating interventions targeting this may be of interest for future research.

The largest limitation of this analysis is the measurement of inflammation expression. Interleukin-6 was of particular interest due to previous literature related to

interleukin-6, however it is not ideal that there were both missing data and concerns about reliability of measurement. The analysis supported the validity of the measure, however it would be preferable to have more accurate measures to begin with. Future research may want to provide additional consideration of the method used to address the unreliable values by treating them like missing data. A simulation using this procedure to address this form of measurement uncertainty would be insightful. Another limitation was that this variable was not normally distributed. Transformations of the variable did not indicate any changes to the results, however it would be preferable for this to be a more normally distributed. Lastly, the analysis would be improved by incorporating more inflammation response biomarkers (e.g., C-reactive protein, cortisol). They could be modeled, for example, as a multiple item factor, like social support was.

The operationalization of subjective happiness could also have been improved. Here social support was taken as a measure of Winston's (2016) theory of happiness, however this may not be a valid indicator. Furthermore, its reliability was acceptable (i.e., coefficient omega = 0.72), but could be improved. Lyubomirsky's (2007, 2013) happiness was operationalized by the CES-D happiness subscale, which intends to measure happiness for the purpose of identifying a lack of happiness. While the individual items clearly represented happiness, a measure specifically intended to quantify happiness as conceptualized by Lyubomirsky (2007, 2013) would be preferable. Replicating these results with more direct measures of the constructs would be informative.

Lastly, the largest unexpected result was the relationships to age. Future research should investigate this further and consider developmental trajectories while stratifying

for age groups. Comparing the lifelong relationships between age, and measures of happiness and inflammation, to the same relationships when the data are subdivided by age could provide insights into the nature of these results.

Ultimately, the goal of this analysis was to compare the longitudinal changes between two constructs of subjective happiness. Results indicated that there were some relationships between constructs over time, however their differences were more outstanding. Lyubomirsky's (2007, 2013) conceptualization of happiness showed stronger relationships to personal circumstance, whereas Winston's (2016) was more associated with inflammation expression.

Identifying ways to exploit this relationship to benefit public health may be worth further consideration. Consider the case of the Lighten UP! intervention (Friedman et al, 2017). Based on school age interventions, this program enrolls older adults in 60-minute group sessions over the course of eight weeks. During sessions, participants share and discuss personal experiences and histories with the intention of increasing subjective well-being. Evaluations of this program have shown significant increases in measures of subjective well-being, however longer-term assessments are required to further address efficacy (Friedman et al, 2017, 2019). The results of this analysis bolster the possible implications of this approach. While there was no intervention in these data, results indicated longitudinal association of physical health measures and feelings of community integration. This provides support for the possible benefits of this kind of intervention.

This analysis has employed rigorous analytic methods to compare two theoretically distinct conceptualizations of happiness. Understanding the important differences between the two could provide insight into possible targets for intervention.



The unique circumstances older adults face complicate subjective appreciation of life. That said, there may be opportunity to preserve broader health and well-being in light of these results. Social support networks may be an avenue to mitigate some of the age specific realities older adults must endure. Because this was an observations study, future research is necessary. However, the longitudinal nature of this analysis over the course of multiple years and the inclusion of physiological variables support the possible efficacy of this approach.

**Table 1****Sample Characteristics Statistics**

<b>Variable</b>	<b>Mean</b>	<b>SD</b>	<b>Skew</b>	<b>Kurtosis</b>	<b>% Missing</b>
Age (years, in 1991)	70.11	8.77	-0.07	-1.24	0.00
<u>Happiness (Scale from 0 to 16)</u>					
Baseline	7.97	7.80	1.67	3.53	1.64
Follow-up	7.98	7.83	1.62	3.28	28.81
<u>Social support (Scale from 0 to 3)</u>					
Receiving instrumental support, baseline	0.80	0.73	0.75	-0.12	7.50
Giving instrumental support, baseline	0.67	0.72	0.92	0.00	7.47
Receiving emotional support, baseline	1.71	0.77	-0.48	-0.38	7.60
Giving emotional support, baseline	1.58	0.83	-0.30	-0.72	7.56
Receiving instrumental support, follow-up	0.86	0.72	0.63	-0.36	29.26
Giving instrumental support, follow-up	0.69	0.73	0.92	0.05	29.35
Receiving emotional support, follow-up	1.60	0.80	-0.33	-0.57	29.29
Giving emotional support, follow-up	1.73	0.74	-0.53	-0.18	29.32
<u>Interleukin-6</u>					
Baseline	9.11	5.21	2.39	6.25	43.84
Follow-up	11.30	10.54	3.25	12.98	58.58
<u>Confounders</u>					
Years of education	8.76	3.32	0.94	0.29	0.26
Subjective health rating (scale from 1 to 5)	2.40	0.93	0.70	0.30	0.80
Subjective health rating relative to peers (scale from 1 to 5)	2.47	0.92	0.00	-0.21	1.29
Functional limitations (scale from 0 to 8)	1.40	1.22	0.91	0.84	0.64
<b>Gender identification</b>					
	<b>n</b>	<b>%</b>			
Male	1506	48.47			
Female	1601	51.53			
Missing	0	0.00			

**Table 2****Measurement Diagnostics**

Average correlation		<u>Baseline</u>		<u>Follow-up</u>	
	Age	Interleukin-6	C reactive protein	Interleukin-6	C reactive protein
Age	1.00				
<u>Baseline</u>					
Interleukin-6	0.07 (0.00)*	1.00			
C reactive protein	-0.04 (0.01)	0.13 (0.01)*	1.00		
<u>Follow-up</u>					
Interleukin-6	0.01 (0.00)	0.19 (0.01)*	0.01 (0.00)	1.00	
C reactive protein	0.04 (0.00)	0.04 (0.01)	0.11 (0.04)*	0.14 (0.02)*	1.00

*Note:* Correlations were estimated using pairwise complete cases, pairwise complete cases with unreliable values of interleukin-6 removed, bootstrapped multiple imputation with no bias to imputations, bootstrapped multiple imputation with high bias to imputations, and multiple imputation with low bias to imputations. The average correlation value across all five estimates is reported and parentheses is the mean squared error for the estimate.

\* $p < 0.05$

**Table 3****Model Fit and Hypothesis Tests**

#	$X^2$	df	$p$	$X^2/df$	CFI [95% CI]	RMSEA [90% CI]	SRMR [95% CI]
0	18.92	7	0.0084	2.70	0.99 [0.97, 1.00]	0.03 [0.01, 0.05]	0.02 [0.01, 0.02]
1*	162.35	17	< 0.0001	9.55	0.89 [0.85, 0.93]	0.07 [0.06, 0.09]	0.03 [0.02, 0.04]
2*	69.52	10	< 0.0001	6.95	0.93 [0.88, 0.97]	0.06 [0.04, 0.08]	0.03 [0.02, 0.05]
3*	27.95	9	0.0010	3.11	0.98 [0.93, 1.02]	0.03 [0.01, 0.05]	0.02 [0.00, 0.04]
4*	32.66	10	0.0003	3.27	0.97 [0.95, 0.99]	0.04 [0.02, 0.05]	0.02 [0.02, 0.03]
5	18.19	10	0.0518	1.82	0.99 [0.97, 1.01]	0.02 [0.00, 0.04]	0.02 [0.01, 0.02]

\*Significantly worse fit from hypothesized model, model 0, at  $p < 0.05$

*Note:* Model zero is the hypothesized model, and all model comparisons are made to this model. Model 1 is the hypothesized with the inclusion of covariates. Model 2 provides a test of longitudinal mediation, wherein the path from age to baseline social support and the covariances between baseline social support and each of change in happiness and interleukin-6 were constrained to zero. Model 3 is a follow up test to model 2, in which only the covariances between baseline social support and each of change in happiness and interleukin-6 were constrained to zero. Model 4 tests the assumption of no orthogonal baseline traits by constraining covariances between baseline social support, happiness, and interleukin-6 to zero. Lastly, model 5 tests the assumption of orthogonal change traits by constraining covariance between change in social support, happiness, and interleukin-6 to zero.

**Table 4****Developmental Effects**

<b>Age effects</b> <b>Outcome</b>	<b><u>Without covariates<sup>0</sup></u></b>			<b><u>With covariates<sup>1</sup></u></b>		
	<b>PE</b>	<b>Z</b>	<b>p</b>	<b>PE</b>	<b>Z</b>	<b>p</b>
Social support baseline <sup>2</sup>	-0.28	-6.82	< 0.0001	-0.27	-6.43	< 0.0001
Social support change	0.00	0.04	0.9681	-0.02	-0.19	0.8493
Happiness baseline	-0.11	-4.16	< 0.0001	-0.08	-3.15	0.0016
Happiness change	-0.07	-2.75	0.0060	-0.03	-0.23	0.8181
Interleukin-6 baseline	-0.17	-2.93	0.0034	-0.20	-3.15	0.0016
Interleukin-6 change	0.10	0.43	0.6672	-0.01	-0.02	0.9840

<b>Factor 1</b>	<b>Factor 2</b>	<b><u>Without covariates<sup>0</sup></u></b>			<b><u>With covariates<sup>1</sup></u></b>		
		<b>PE</b>	<b>Z</b>	<b>p</b>	<b>PE</b>	<b>Z</b>	<b>p</b>
<u>Baseline covariances</u>							
Social support baseline	Happiness baseline <sup>4</sup>	-0.02	-0.48	0.6312	-0.03	-0.90	0.3681
Social support baseline	Interleukin-6 baseline <sup>4</sup>	-0.29	-3.57	0.0004	-0.29	-3.63	0.0003
Happiness baseline	Interleukin-6 baseline <sup>4</sup>	0.03	0.58	0.5619	0.01	0.11	0.9124
<u>Change covariances</u>							
Social support change	Happiness change <sup>5</sup>	0.05	0.95	0.3421	0.03	0.42	0.6745
Social support change	Interleukin-6 change <sup>5</sup>	0.02	0.08	0.9362	0.27	0.22	0.8259
Happiness change	Interleukin-6 change <sup>5</sup>	0.02	0.23	0.8181	-0.14	-0.07	0.9442
<u>Within factor covariances</u>							
Social support baseline	Social support change	-0.31	-0.90	0.3681	-0.13	-0.39	0.6965
Happiness baseline	Happiness change	-0.07	-0.90	0.3681	-0.47	-0.16	0.8729
Interleukin-6 baseline	Interleukin-6 change	0.10	0.25	0.8026	0.50	0.27	0.7872
<u>Cross factor covariances</u>							
Social support baseline	Happiness change <sup>2,3</sup>	0.00	0.02	0.9840	-0.01	-0.10	0.9203
Social support baseline	Interleukin-6 change <sup>2,3</sup>	0.06	0.18	0.8572	-0.15	-0.12	0.9045
Social support change	Happiness baseline	0.05	1.29	0.1971	0.03	0.80	0.4237
Social support change	Interleukin-6 baseline	-0.01	-0.09	0.9283	-0.04	-0.26	0.7949
Happiness baseline	Interleukin-6 change	-0.01	-0.10	0.9203	0.00	-0.03	0.9761
Happiness change	Interleukin-6 baseline	-0.03	-0.59	0.5552	-0.04	-0.21	0.8337

*Note:* PE represents parameter estimate, gamma for the age effects and psi for the factor covariances

0: Model 0, the hypothesized model

1: Model 1, the hypothesized model with covariates

2: Model 2, parameter constrained to zero to test longitudinal mediation

3: Model 3, parameter constrained to zero as a follow up test of longitudinal mediation

4: Model 4, parameter constrained to zero to test the orthogonal baseline traits

5: Model 5, parameter constrained to zero to test orthogonal change traits

**Table 5****Covariate Effects**

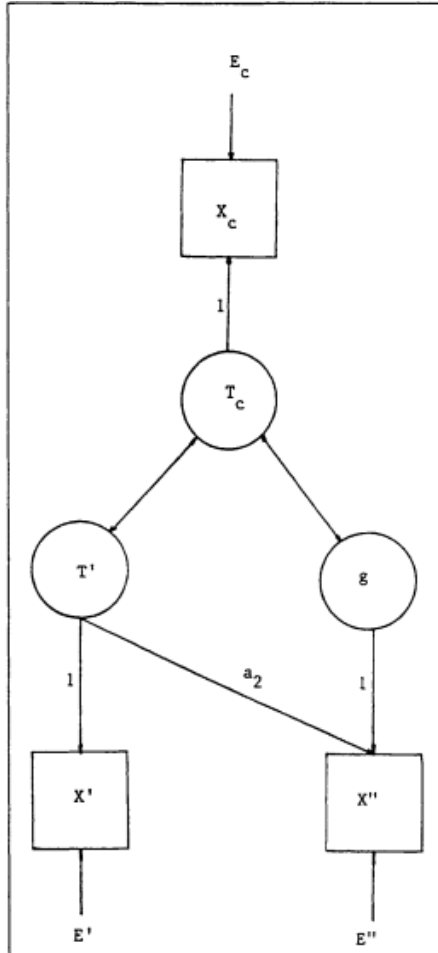
<b>Covariate</b>	<b>PE</b>	<b>Z</b>	<b>p</b>
<u>Outcome: social support baseline</u>			
Female gender identity	-0.12	-1.72	0.0854
Years education	0.04	1.03	0.3030
Subjective health	-0.04	-0.79	0.4295
Subjective health relative to peers	0.09	2.11	0.0349
Functional limitations	0.02	0.48	0.6312
<u>Outcome: social support change</u>			
Female gender identity	-0.05	-0.49	0.6241
Years education	-0.04	-0.94	0.3472
Subjective health	-0.03	-0.51	0.6101
Subjective health relative to peers	-0.02	-0.51	0.6101
Functional limitations	0.02	0.36	0.7188
<u>Outcome: happiness baseline</u>			
Female gender identity	-0.11	-4.74	< 0.0001
Years education	0.05	2.35	0.0188
Subjective health	-0.34	-10.87	< 0.0001
Subjective health relative to peers	-0.01	-0.43	0.6672
Functional limitations	-0.03	-1.33	0.1835
<u>Outcome: happiness change</u>			
Female gender identity	-0.08	-0.24	0.8103
Years education	-0.01	-0.05	0.9601
Subjective health	-0.03	-0.04	0.9681
Subjective health relative to peers	0.06	1.99	0.0466
Functional limitations	-0.05	-1.08	0.2801
<u>Outcome: interleukin-6 baseline</u>			
Female gender identity	0.01	0.11	0.9124
Years education	0.03	0.49	0.6241
Subjective health	-0.02	-0.26	0.7949
Subjective health relative to peers	-0.13	-2.37	0.0178
Functional limitations	0.04	0.58	0.5619
<u>Outcome: interleukin-6 change</u>			
Female gender identity	0.02	0.12	0.9045
Years education	0.04	0.15	0.8808
Subjective health	-0.04	-0.07	0.9442
Subjective health relative to peers	-0.07	-0.16	0.8729
Functional limitations	0.01	0.02	0.9840

*Note:* PE represents parameter estimate, gamma. Results only reflect model 1, which is the hypothesized model plus these covariate effects.

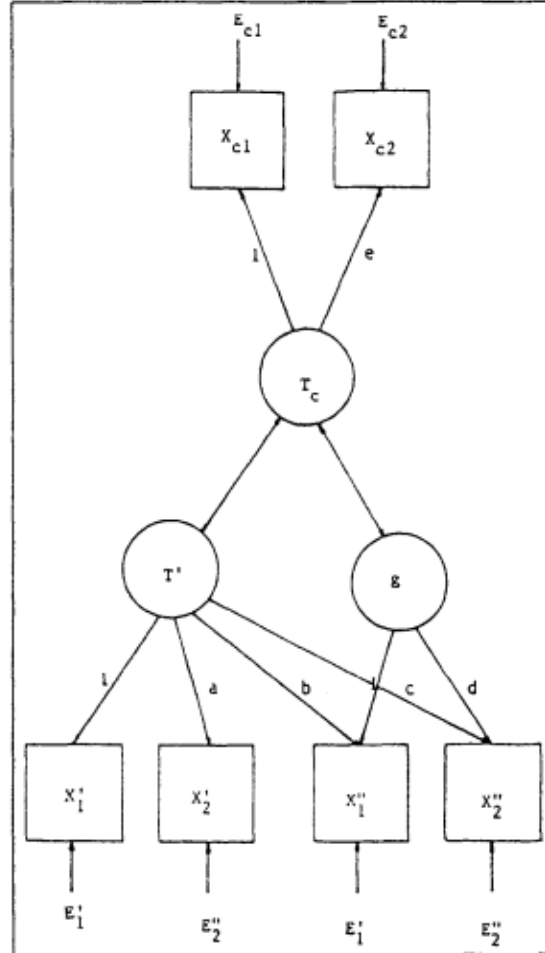
**Figure 1**

**Model design**

Single item design:



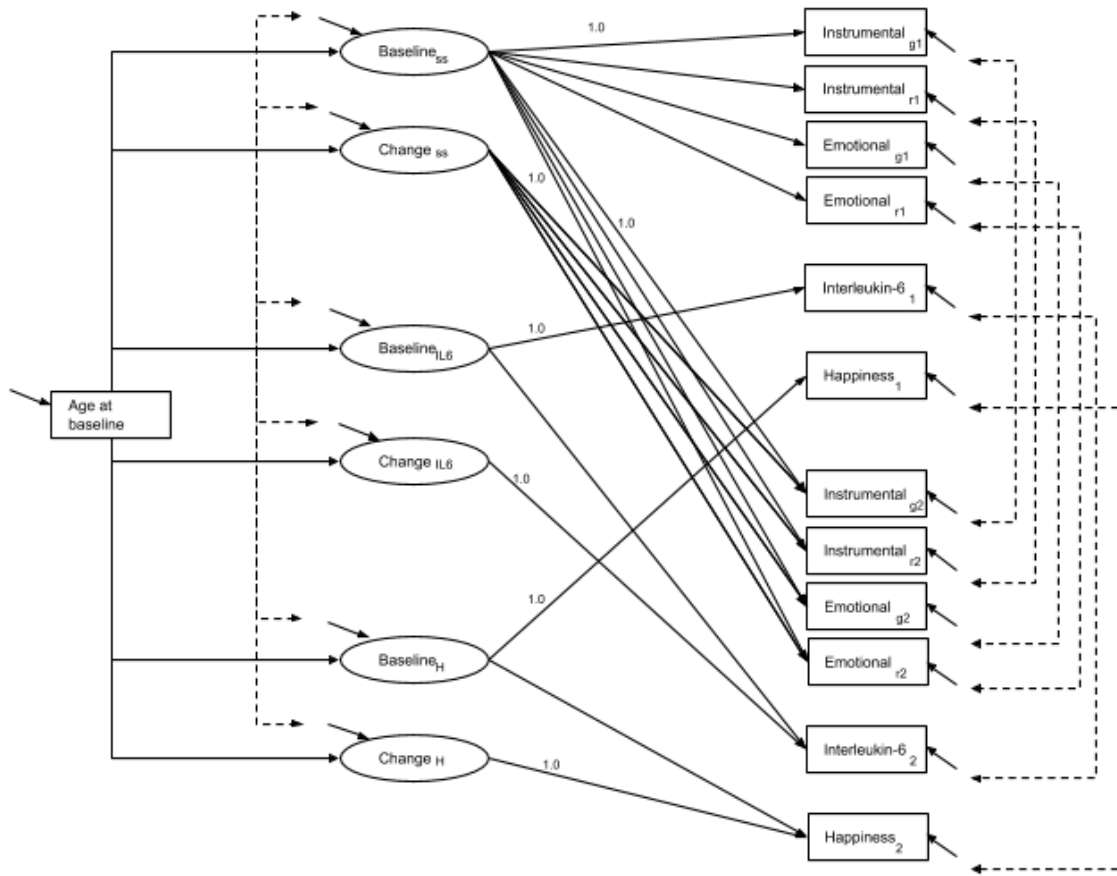
Multiple item design:



*Note: From Raykov (1992)*

**Figure 2**

**Hypothesized model**

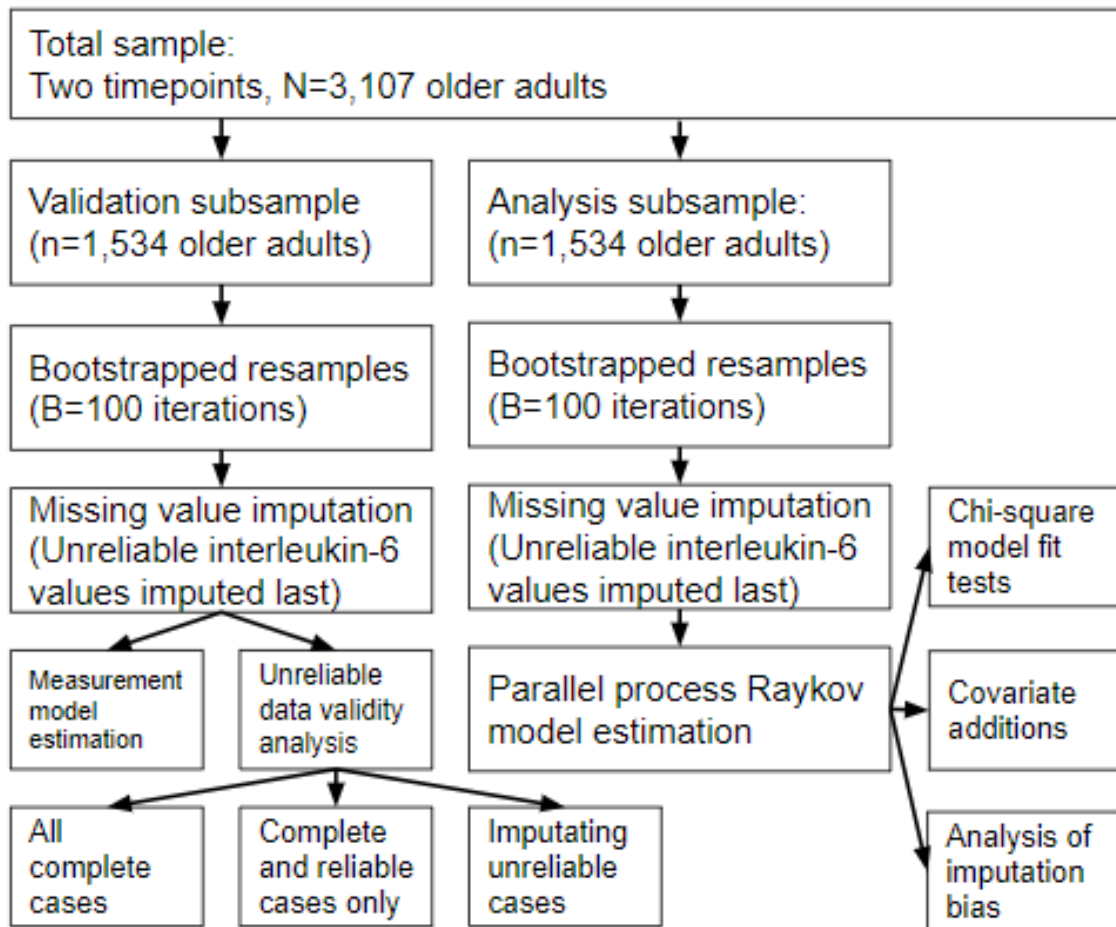


*Note: For clarity, double-headed covariances between error terms have been marked with dashed lines, while all other single-headed regression paths, factor loadings, and error variances are solid. One loading from the baseline factor to the baseline measurement, and one loading from the change factor to follow-up measurement is constrained to 1.0. Error variances are covaried between repeated measures of the same variable. Loadings from baseline to follow-up measurement are freely estimated. This is specified in the analytic design for this model, described by Raykov (1992). All disturbances are covaried and freely estimated between all six baseline and change factors to allow for an analysis of relationships over time.*



**Figure 3**

**Flow chart of analysis design**



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