Subjective well-being and cardiometabolic health: An 8–11 year study of midlife adults

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ABSTRACT

Objective: Individuals who are satisfied and experience frequent positive emotions tend to have reduced risk for coronary heart disease (CHD). However, conflicting evidence exists and little research has investigated whether well-being is associated with early-warning indicators of biological risk that precede CHD. We investigated whether life satisfaction and positive emotions longitudinally predicted reduced risk of incident cardiometabolic conditions and healthier cardiometabolic risk scores, which may provide insight into underlying mechanisms and novel prevention targets.
Methods: Initially healthy men and women ($N = 754$–854) reported their baseline life satisfaction and positive emotions. During follow-up, presence of manifest cardiometabolic conditions was assessed and a separate cardiometabolic risk score was constructed from eight biomarkers. Poisson and linear regression analyses tested whether life satisfaction and positive emotions were associated with reduced incident disease risk and lower cardiometabolic risk scores 8–11 years later.
Results: Life satisfaction and positive emotions were each prospectively associated with reduced risk of manifest conditions, controlling for demographics and family history of CHD. Associations were attenuated for positive emotions after adjusting for depressive symptoms and for life satisfaction after adjusting for health behaviors. Life satisfaction was associated with lower cardiometabolic risk scores until adding health behaviors, but positive emotions were not (regardless of the included covariates).
Conclusion: Well-being, particularly life satisfaction, is associated with reduced risk for incident cardiometabolic conditions in minimally-adjusted models. However, accounting for underlying behavioral pathways attenuates the association. Low levels of life satisfaction (but not positive emotions) may also provide early warning of cardiometabolic risk prior to disease development.

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1. Introduction

Individuals who are satisfied with their lives and who experience frequent positive emotions – that is, individuals with high levels of subjective well-being [1] – not only feel good, but may also have reduced risk for developing coronary heart disease (CHD) [2]. In one prospective study of British civil servants, individuals reporting the highest life satisfaction levels had a 13% reduced risk of CHD approximately five years later [3]. Similarly, Canadian men and women who displayed more versus fewer positive emotions had a 22% reduced risk of incident CHD ten years later [4]. Associations in both studies remained when controlling for demographics, health behaviors, clinical factors, and

negative emotions. However, null findings exist as well. Results from the above sample of British civil servants indicated that positive emotions were not related to incident CHD during twelve years of follow-up [5].

Thus, further research is needed to ascertain whether subjective well-being protects against manifest CHD. Moreover, little prior work has investigated whether life satisfaction and positive emotions are associated with conditions that *precede* risk for CHD (e.g., high blood pressure). Subjective well-being may buffer against the harmful health consequences of stress and exert direct influence on bodily systems, or may motivate healthy behavior [2]. If so, subjective well-being could be associated with cardiometabolic risk factors that appear earlier in life than manifest disease. Indeed, limited work suggests that life satisfaction and positive emotions are associated with healthier levels of blood pressure [6], high-density lipoprotein (HDL) cholesterol [7],

glycosylated hemoglobin [8], and C-reactive protein (CRP) [9]. However, null findings have also been reported [7,10]. Moreover, prior work used different study designs (e.g., cross-sectional vs. short-term longitudinal studies) and different methodologies for assessing biomarkers (e.g., ambulatory vs. laboratory-based assessment). Evidence of an association with biological risk factors that *precede* manifest disease would further support the importance of links between subjective well-being and reduced risk of adverse cardiovascular outcomes. In addition, such associations would allow for earlier identification of risk factors and protective factors—that is, before disease occurs as an early warning system for risk prediction.

The present, longitudinal investigation of middle-aged men and women investigated whether baseline assessments of life satisfaction and positive emotions were associated first with incident cardiometabolic conditions and second with a biologically-based cardiometabolic risk score approximately a decade later. Individuals with higher compared to lower levels of life satisfaction and positive emotions were hypothesized to be at reduced risk for incident cardiometabolic conditions and more likely to have lower cardiometabolic risk scores during follow-up. Cardiometabolic risk was assessed by eight biomarkers that could be potentially modified at younger ages to alter risk trajectories. Evidence suggests indicators of cardiometabolic dysregulation cluster together such that combining them yields a measure that improves prediction of disease risk, which may be particularly useful with relatively healthy or younger populations [11–13]. The eight biological markers in the risk score were also considered separately in sensitivity analyses to see if one marker in particular was driving effects with the overall composite. Individuals with higher baseline life satisfaction and positive emotions were expected to have healthier levels of each biomarker. Based on prior research [2], covariates were considered that might either confound (e.g., education, depressive symptoms) or indicate a potential pathway (e.g., smoking, physical activity) for the associations of interest.

2. Method

2.1. Participants

Participants included men and women from the Midlife in the United States (MIDUS) study. The MIDUS study was originally established in 1994-1995 to understand associations between psychological, social, and behavioral factors in aging individuals. The first phase comprised 7108 individuals ages 25–74 who were primarily recruited via random digit dialing. Other recruitment methods included oversampling certain metropolitan areas and recruiting twin pairs and siblings of some respondents [14]. The second phase occurred in 2004–2005 and involved expanded assessments, including biomarkers. The present research included the subset of participants who completed the biomarker assessment ($n = 1255$). Participants were eligible for this assessment if they were healthy enough to travel to a research clinic (at University of Wisconsin, Georgetown University, or University of California, Los Angeles) and had completed the psychosocial assessment [15]. Reflecting the requirement that participants travel to the research clinic and stay overnight, the response rate for the biomarker assessment was relatively low (43.1%) [15]. However, participants in the biomarker assessment did not differ from participants in the larger survey based on age, sex, race, marital status, income, chronic conditions, or body mass index, although they were more highly educated [15].

Analytic samples for the incident cardiometabolic conditions and cardiometabolic risk analyses were drawn from the 1255 participants with biomarker data. Those newly recruited at the second phase of MIDUS who did not have baseline measures of subjective well-being were excluded ($n = 201$). To limit potential concerns about reverse causality, participants who reported chronic cardiometabolic conditions (heart problems, hypertension, diabetes, stroke, high cholesterol, or medication use for these conditions), or who had missing values on

these conditions during the first phase ($n = 280$) were excluded from the incident cardiometabolic conditions analyses. In addition, 20 participants with missing data on subjective well-being, incident disease, or covariates were excluded, yielding 754 individuals in these analyses (187 of whom had a sibling or twin). For the cardiometabolic risk analyses, 140 participants were excluded due to baseline heart problems or stroke, using related medications, or missing data. We did not exclude participants with related cardiometabolic conditions or medication use at baseline (i.e., hypertension, diabetes, or high cholesterol) because this would essentially duplicate the incident analyses and would lead to a more restrictive sample that does not reflect the high prevalence of such conditions among U.S. adults [16]. In addition, participants with extreme levels of triglycerides (>2000 mg/dL), glycosylated hemoglobin (>15%), or CRP (>10 μg/mL) at follow-up were excluded from risk analyses ($n = 39$), as such levels may indicate outliers, incorrect values, or an active infection [17]. Finally, participants with missing data on subjective well-being, biomarkers of cardiometabolic risk, or covariates were excluded ($n = 21$), yielding 854 individuals in the risk analyses (217 of whom had a sibling or twin). Supplemental Fig. S1 describes how the analytic samples were obtained. Where possible, missing values on baseline demographics were imputed with values from the second phase.

Participants in the most inclusive analytic sample ($N = 854$) were more likely to be white, were more highly educated, and had higher household incomes relative to participants not included. All individuals provided informed consent to participate and Institutional Review Boards at participating institutions approved the research.

2.2. Measures

2.2.1. Subjective well-being

Two components of subjective well-being were assessed at baseline: life satisfaction and positive emotions. Life satisfaction and positive emotions are related ($r = .56$ in this sample), but are considered distinct with the former reflecting a cognitive evaluation and the latter reflecting an affective evaluation [1]. Recommendations to investigate them separately were followed [1].

Life satisfaction was assessed with a single item that asked participants to rate their overall life satisfaction on an 11-point scale ranging from 0 (*worst possible*) to 10 (*best possible*) [18] that has been used in previous MIDUS work [19]. In this study, mean life satisfaction was 7.94 ($SD = 1.40$; range = 0.00–10.00). Other research demonstrates that single-item measures of life satisfaction show validity equivalent to longer measures [20]. However, to ensure that life satisfaction findings were robust, sensitivity analyses also used a four item composite of life satisfaction that asked participants to rate their satisfaction relative to their work, relationship with their spouse/partner, relationship with their children, and life overall ($\alpha = .66$) [21]. Similar composites that average domain life satisfaction ratings are strongly correlated with global assessments of life satisfaction [22].

Positive emotions were assessed with six items that asked "During the past 30 days, how much of the time did you feel…" The six feelings included cheerful, in good spirits, extremely happy, calm and peaceful, satisfied, and full of life. Items were derived from validated measures of emotion and used a time frame of 30 days to be sensitive to both situational and personality-based factors [23]. Responses ranged from 1 (*all of the time*) to 5 (*none of the time*). Items were reverse-scored and then averaged for an overall score of positive emotions. Higher scores reflected more positive emotions. Mean positive emotion levels were 3.45 ($SD = 0.66$; range = 1.33–5.00) and internal consistency reliability was excellent ($\alpha = .89$).

Prior MIDUS research shows that subjective well-being is related to higher socioeconomic status, more future planning, better sleep quality, and fewer physical disorders [21,24–26]. Life satisfaction and positive emotions can be conceptualized as persistent characteristics that may have cumulative effects on health due to their relative stability over

time [27]. For example, the correlation between baseline and follow-up scores (up to 11 years later) for life satisfaction and positive emotion here were .39 and .48, respectively.

2.2.2. Incident cardiometabolic conditions

Status regarding cardiometabolic conditions, self-reported by participants at both phases, was indicated by the presence or absence of heart problems (including heart attack or heart failure; $N_{cases} = 72$, 9.55%), hypertension ($N_{cases} = 197$, 26.13%), diabetes ($N_{cases} = 48$, 6.37%), stroke ($N_{cases} = 19$, 2.52%), high cholesterol ($N_{cases} = 294$, 39.25%), or medication use for these conditions. Participants were classified as having a cardiometabolic condition if they reported at least one. Prior research has suggested that such self-reported health measures are valid and reliable [28].

2.2.3. Cardiometabolic risk score

Unlike other risk scores that include non-modifiable factors such as gender (e.g., the Framingham risk score), cardiometabolic risk was assessed with a set of biomarkers that are not susceptible to self-report bias; they are also potentially modifiable via improved health behaviors (e.g., exercising). Prior work has demonstrated that these scores predict general cardiovascular risk and have the potential to provide researchers and clinicians with tools for evaluating interim risk, which can facilitate primary prevention [12]. Following previous research [29–31], a cardiometabolic risk score was constructed from eight biomarkers assessed at the second phase: systolic blood pressure (SBP), diastolic blood pressure (DBP), triglycerides, HDL cholesterol, low-density lipoprotein (LDL) cholesterol, glycosylated hemoglobin, waist circumference, and CRP. SBP and DBP were each assessed three times, with the two most similar readings averaged together; other biomarkers were each assessed once. To maintain as much information as possible [32] and in accordance with past research [33], each biomarker was standardized ($M = 0$, $SD = 1$) and the eight standardized biomarkers were summed into an overall cardiometabolic risk score wherein higher scores indicated greater cardiometabolic dysregulation (HDL cholesterol was reverse scored prior to summing as higher levels indicate better health). The overall cardiometabolic risk score was standardized ($M = 0$, $SD = 1$; range $= -2.43–3.73$) for ease of interpretation.

2.2.4. Covariates

Covariates included confounders or potential mediators of the associations of interest. Self-reported covariates assessed at baseline included demographics, family history of CHD, health behaviors, and depressive symptoms. Demographics included age (years), gender (men, women), race (white, black and/or African American, other), marital status (married, separated/divorced, widowed, never married), education (less than high school, high school degree, some college, 4-year college degree or higher), and household income (U.S. dollars). Households with incomes greater than $300,000 were converted to a maximum value of $300,000 to minimize risk of deductive disclosure. Other confounders included family history of CHD (number of biological relatives who have ever had a heart attack) and, in cardiometabolic risk score analyses, medications used to manage high blood pressure, high cholesterol, or diabetes (yes/no). Medication use was self-reported during the follow-up assessment. Depressive symptoms were assessed with a screening question from the World Mental Health Organization's Composite International Diagnostic Interview Short Form: "During the past 12 months, was there ever a time when you felt sad, blue, or depressed for two weeks or more in a row?" [yes/no; 34]. This measure was developed for use in situations with limited assessment time, and is consistent with more lengthy diagnostic interviews [34]. Health behaviors, considered as potential mediators of the association between subjective well-being and cardiometabolic health [35], included smoking status (current smoker, past smoker, never smoker), alcohol consumption (no alcohol consumption, <3 drinks/week, ≥3 drinks/week), frequency of vigorous physical activity that is "long enough to work up a

sweat" (e.g., running; number of times/month), and frequency of moderate physical activity (e.g., bowling; number of times/month).

2.3. Statistical analyses

Statistical analyses were performed in SAS 9.3. Following preliminary descriptive analysis, subjective well-being's associations with incident cardiometabolic conditions and the cardiometabolic risk score were examined separately for life satisfaction and positive emotions. Poisson regression analyses were used with the dichotomous cardiometabolic conditions outcome because it was reasonably common [36], while linear regression analyses were used with the continuous cardiometabolic risk score outcome. In both cases, the first model adjusted for age. The second model controlled for demographics (age, gender, race, marital status, education, and household income) and family history of CHD. For the cardiometabolic risk score, the second model also adjusted for relevant medication use. The third model additionally adjusted for depressive symptoms. The fourth, fully-adjusted model further adjusted for potential mediating factors related to health behaviors (smoking status, alcohol consumption, vigorous physical activity, and moderate physical activity). For the linear regression models with the cardiometabolic risk score, effect size estimates of Cohen's f^2 were calculated [37]; f^2 statistics of .02, .15, and .35 are considered small, medium, and large, respectively [38]. Sensitivity analyses repeated the Poisson and linear regression models with the four-item composite of domain life satisfaction. Secondary analyses regressed each of the eight individual cardiometabolic biomarkers (standardized) on life satisfaction and positive emotions separately in linear regression models adjusting for demographics and relevant medication use. Due to the inclusion of siblings and twins in the sample, Poisson and linear regression analyses were repeated using generalized estimating equations (proc GENMOD with family in the repeated statement) to account for clustering of the data [39–41].

3. Results

3.1. Preliminary analyses

Participant characteristics for the 854 participants are shown in Table 1. Notably, approximately one quarter of participants reported having depressive symptoms. This is congruent with national estimates of lifetime depression [42], suggesting that the single item provided adequate assessment. Participant characteristics according to low, moderate, and high levels of life satisfaction (derived from sample-based tertiles) are shown in Supplemental Table S1. Similar patterns were evident for positive emotions (Supplemental Table S2) and when using the sample for incident analyses ($N = 754$; data not shown).

3.2. Subjective well-being and incident cardiometabolic conditions

Of the 754 participants without cardiometabolic conditions at baseline, 413 (54.77%) developed at least one incident condition over the follow-up period. Consistent with our hypotheses, higher baseline life satisfaction and positive emotions were associated with a 7% reduced risk of incident cardiometabolic conditions after adjusting for age (Tables 2–3). The pattern of associations was similar across the two indicators of subjective well-being, but the magnitude of effect was more robust with life satisfaction. For example, life satisfaction's protective association remained statistically significant when demographics, family history of CHD, and depressive symptoms were added to the model. Adding health behaviors as potential mediators attenuated life satisfaction's association with cardiometabolic conditions such that the association was no longer statistically significant. Positive emotion's association with cardiometabolic conditions was no longer statistically significant when depressive symptoms (or health behaviors) were added to the model.

Table 1
Distribution of participant characteristics ($N = 854$).

Mean life satisfaction (SD)	7.94 (1.40)
Mean positive affect (SD)	3.45 (0.66)
Mean age (SD), years	45.68 (11.56)
Sex	
Male	46.02%
Female	53.98%
Race	
White	94.03%
Black	2.69%
Other	3.28%
Marital status	
Married	72.60%
Separated or divorced	13.00%
Widowed	2.58%
Never married	11.83%
Education	
Less than high school	2.81%
High school degree	22.95%
Some college	27.75%
4-Year college degree or higher	46.49%
Mean household income (SD), US dollars	83,199 (61,275)
Mean number of biological family members who have had a heart attack, (SD)	0.47 (0.74)
Medication use for high blood pressure	
Yes	32.32%
No	67.68%
Medication use for high cholesterol	
Yes	27.75%
No	72.25%
Medication use for diabetes	
Yes	7.38%
No	92.62%
Depressive symptoms	
Yes	24.82%
No	75.18%
Mean vigorous physical activity (SD), occasions per month	6.68 (5.14)
Mean moderate physical activity (SD), occasions per month	9.94 (4.44)
Smoking status	
Current smoker	14.75%
Past smoker	29.39%
Never smoker	55.85%
Alcohol intake	
Never drinker	5.74%
Light or moderate drinker	55.62%
Heavy drinker	38.64%
Mean systolic blood pressure (SD), mmHg	130.44 (17.36)
Mean diastolic blood pressure (SD), mmHg	75.13 (10.24)
Mean triglycerides (SD), mg/dL	129.23 (80.25)
Mean high-density lipoprotein cholesterol (SD), mg/dL	55.09 (17.39)
Mean low-density lipoprotein cholesterol (SD), mg/dL	107.50 (34.42)
Mean glycosylated hemoglobin (SD), %	5.95 (0.81)
Mean waist circumference (SD), cm	96.30 (16.52)
Mean C-reactive protein (SD), µg/mL	2.13 (2.15)

3.3. Subjective well-being and the cardiometabolic risk score

As expected, higher baseline life satisfaction was also associated with lower cardiometabolic risk scores assessed 8–11 years later ($M = 9.07$ years; Table 4). This association was statistically significant until health behaviors were added to the model. Of note, adding depressive symptoms did not alter life satisfaction's association with lower cardiometabolic risk, suggesting that the absence of depression is not equivalent to the presence of subjective well-being. Positive emotions were not significantly associated with the cardiometabolic risk score (Table 5).

3.4. Additional analyses

Primary analyses (i.e., Poisson and linear regression models) were repeated for the four-item composite of domain life satisfaction. As shown in Supplemental Tables S3–S4, results for incident cardiometabolic conditions were identical and results for the cardiometabolic risk score were very similar when comparing findings for domain life satisfaction and the single item of overall life satisfaction.

The eight cardiometabolic biomarkers were separately examined in relationship to subjective well-being in regression models adjusting for demographics and relevant medication use (Supplemental Table S5). Greater life satisfaction and positive emotions were each significantly associated with lower levels of triglycerides. Greater life satisfaction was also associated with smaller waist circumference and positive emotions were associated with lower levels of LDL cholesterol. However, analyses do not suggest that any single biomarker is driving findings with the overall cardiometabolic risk score.

All analyses were repeated with generalized estimating equations to account for clustering within the data (i.e., twin and sibling pairs). These results were nearly identical to the findings described previously, suggesting that clustering did not bias estimates. Given the similarity of these findings with those of the Poisson and linear regression analyses, as well as the ease of interpretation with the latter, results from the generalized estimating equations are shown in Supplemental Tables S6–S7.

4. Discussion

This study examined whether subjective well-being is linked with reduced risk of incident cardiometabolic conditions and lower cardiometabolic risk scores 8–11 years later. Consistent with past research [3,4], findings indicated that higher levels of life satisfaction and positive emotions were associated with reduced risk of developing manifest cardiometabolic conditions, controlling for demographics and family history of CHD. Life satisfaction's association with incident conditions was maintained after controlling for depressive symptoms, suggesting that life satisfaction's effects are independent of emotional distress. However, positive affect's association with incident conditions was no longer statistically significant when accounting for depressive symptoms, which may suggest that positive emotions and depressive symptoms are more closely related. Moreover, adding health behaviors—which potentially lie on the pathway between subjective well-being and incident cardiometabolic conditions—attenuated life satisfaction's association.

Subjective well-being's association with early-warning markers of cardiometabolic risk—as defined by eight non-self-reported cardiometabolic biomarkers—was consistent with predictions for life satisfaction but not positive emotions. Specifically, life satisfaction was significantly associated with lower cardiometabolic risk scores controlling for demographics, family history of CHD, relevant medication use, and depressive symptoms, but not potential behavioral pathways. Positive emotions were not significantly associated with cardiometabolic risk in any model. When examining each individual biomarker of cardiometabolic risk, both life satisfaction and positive emotions showed statistically significant inverse associations with triglycerides; life satisfaction was also related to smaller waist circumference and positive emotions were related to lower triglycerides. Associations with other biomarkers did not reach statistical significance.

The presence of even one cardiometabolic risk factor in middle age dramatically increases risk of cardiovascular death [43]. However, one issue often considered with composite measures like the risk score is whether findings are driven primarily by a single biomarker, or if the whole is greater than the sum of its parts. Most research suggests findings can rarely be attributed to a single biomarker. For example, contextual factors within one's neighborhood were associated with a composite of cardiometabolic markers more strongly than individual biomarkers [30]. In the current study, we also saw the most consistent associations with the total cardiometabolic risk score. Summary measures are informative because they predict long-term disease risk via early-warning indicators across multiple systems. Our findings suggest that life satisfaction in particular is associated with lower cardiometabolic risk and the subsequent likelihood of developing CHD.

Table 2
Prevalence ratios (95% confidence intervals) for the association between one standard deviation increase in life satisfaction and risk of incident cardiometabolic conditions across 9 years of follow-up (N = 754)[a]

	Model 1[b]	Model 2[c]	Model 3[d]	Model 4[e]
Life satisfaction (standardized)	0.93* (0.88, 0.99)	0.93* (0.88, 0.99)	0.93* (0.88, 0.99)	0.95⁻ (0.89, 1.01)
Age at baseline	1.02*** (1.02, 1.03)	1.02*** (1.01, 1.03)	1.02*** (1.01, 1.03)	1.02*** (1.01, 1.02)
Female (vs. male)		1.05 (0.92, 1.19)	1.05 (0.92, 1.19)	1.04 (0.90, 1.19)
Black (vs. white)		1.33⁻ (0.96, 1.83)	1.32⁻ (0.96, 1.83)	1.29 (0.93, 1.80)
Other race (vs. white)		1.07 (0.78, 1.48)	1.07 (0.77, 1.47)	1.07 (0.77, 1.48)
Separated or divorced (vs. married)		0.97 (0.80, 1.17)	0.97 (0.79, 1.17)	0.96 (0.79, 1.17)
Widowed (vs. married)		0.88 (0.62, 1.24)	0.88 (0.62, 1.24)	0.90 (0.62, 1.31)
Never married (vs. married)		0.91 (0.71, 1.18)	0.92 (0.71, 1.19)	0.92 (0.71, 1.18)
High school degree (vs. <high school)		0.92 (0.65, 1.28)	0.92 (0.66, 1.29)	0.94 (0.67, 1.32)
Some college (vs. <high school)		0.91 (0.65, 1.27)	0.91 (0.65, 1.27)	0.93 (0.67, 1.31)
4-Year college degree or higher (vs. < high school)		0.89 (0.64, 1.23)	0.89 (0.64, 1.24)	0.93 (0.67, 1.30)
Household income		1.00 (1.00, 1.00)	1.00 (1.00, 1.00)	1.00 (1.00, 1.00)
Biological family members with heart attack history		1.13*** (1.06, 1.21)	1.13*** (1.06, 1.21)	1.12*** (1.05, 1.19)
Depressive symptoms			1.03 (0.87, 1.20)	1.02 (0.86, 1.19)
Vigorous physical activity				0.98** (0.97, 0.99)
Moderate physical activity				1.00 (0.98, 1.01)
Past smoker (vs. never smoker)				1.15* (1.00, 1.33)
Current smoker (vs. never smoker)				1.14 (0.94, 1.37)
Light or moderate drinker (vs. never drinker)				1.06 (0.80, 1.41)
Heavy drinker (vs. never drinker)				1.10 (0.81, 1.49)

[a] 413 cases of incident cardiometabolic conditions.
[b] Adjusted for age.
[c] Adjusted for demographics (age, gender, race, marital status, education, and household income) and family history of heart disease.
[d] Adjusted for demographics, family history of heart disease, and depressive symptoms.
[e] Adjusted for demographics, family history of heart disease, depressive symptoms, and health behaviors (smoking status, alcohol consumption, vigorous physical activity, and moderate physical activity).
⁻ p ≤ .10.
* p ≤ .05.
** p ≤ .01.
*** p ≤ .001.

Our results add to the growing literature indicating that positive psychological states are associated with better cardiometabolic health, often independently of poor psychological functioning [2]. These findings are consistent with a cross-sectional investigation from the MIDUS study that examined baseline associations between mental health and cardiovascular disease (assessed similarly to cardiometabolic conditions in the

Table 3
Prevalence ratios (95% confidence intervals) for the association between one standard deviation increase in positive emotions and risk of incident cardiometabolic conditions across 9 years of follow-up (N = 754)[a]

	Model 1[b]	Model 2[c]	Model 3[d]	Model 4[e]
Positive emotions (standardized)	0.93* (0.87, 0.99)	0.94* (0.88, 1.00)	0.94⁻ (0.88, 1.01)	0.96 (0.89, 1.03)
Age at baseline	1.02*** (1.02, 1.03)	1.02*** (1.01, 1.03)	1.02*** (1.01, 1.03)	1.02*** (1.01, 1.02)
Female (vs. male)		1.05 (0.92, 1.19)	1.04 (0.92, 1.19)	1.03 (0.90, 1.19)
Black (vs. white)		1.33⁻ (0.97, 1.82)	1.33⁻ (0.97, 1.82)	1.30 (0.94, 1.79)
Other race (vs. white)		1.08 (0.78, 1.50)	1.08 (0.77, 1.49)	1.08 (0.78, 1.49)
Separated or divorced (vs. married)		1.00 (0.83, 1.21)	1.00 (0.82, 1.21)	0.98 (0.81, 1.20)
Widowed (vs. married)		0.89 (0.63, 1.28)	0.89 (0.62, 1.28)	0.93 (0.63, 1.34)
Never married (vs. married)		0.93 (0.72, 1.21)	0.93 (0.72, 1.21)	0.91 (0.72, 1.20)
High school degree (vs. <high school)		0.95 (0.69, 1.31)	0.95 (0.69, 1.32)	0.97 (0.70, 1.34)
Some college (vs. <high school)		0.96 (0.70, 1.32)	0.96 (0.70, 1.32)	0.98 (0.71, 1.35)
4-Year college degree or higher (vs. <high school)		0.93 (0.68, 1.27)	0.93 (0.68, 1.27)	0.96 (0.70, 1.32)
Household income		1.00 (1.00, 1.00)	1.00 (1.00, 1.00)	1.00 (1.00, 1.00)
Biological family members with heart attack history		1.13*** (1.06, 1.21)	1.13*** (1.06, 1.20)	1.12*** (1.05, 1.19)
Depressive symptoms			1.03 (0.87, 1.21)	1.02 (0.87, 1.20)
Vigorous physical activity				0.98** (0.97, 0.99)
Moderate physical activity				0.10 (0.98, 1.01)
Past smoker (vs. never smoker)				1.15⁻ (0.99, 1.33)
Current smoker (vs. never smoker)				1.13 (0.94, 1.37)
Light or moderate drinker (vs. never drinker)				1.07 (0.80, 1.43)
Heavy drinker (vs. never drinker)				1.11 (0.82, 1.50)

[a] 413 cases of incident cardiometabolic conditions.
[b] Adjusted for age.
[c] Adjusted for demographics (age, gender, race, marital status, education, and household income) and family history of heart disease.
[d] Adjusted for demographics, family history of heart disease, and depressive symptoms.
[e] Adjusted for demographics, family history of heart disease, depressive symptoms, and health behaviors (smoking status, alcohol consumption, vigorous physical activity, and moderate physical activity).
⁻ p ≤ .10.
* p ≤ .05.
** p ≤ .01.
*** p ≤ .001.

Table 4
Parameter estimates (standard error) for the association between one standard deviation increase in life satisfaction and the standardized cardiometabolic risk score ($N = 854$)

	Model 1[a]	Model 2[b]	Model 3[c]	Model 4[d]
Life satisfaction (standardized)	−0.07* (0.03)	−0.07* (0.03)	−0.07* (0.03)	−0.06~ (0.03)
Age at baseline	0.008** (0.003)	0.002 (0.003)	0.002 (0.003)	0.002 (0.003)
Female (vs. male)		−0.68*** (0.06)	−0.69*** (0.07)	−0.71*** (0.07)
Black (vs. white)		−0.02 (0.19)	−0.02 (0.19)	−0.02 (0.20)
Other race (vs. white)		0.16 (0.17)	0.15 (0.18)	0.15 (0.18)
Separated or divorced (vs. married)		0.07 (0.10)	0.07 (0.10)	0.05 (0.10)
Widowed (vs. married)		0.05 (0.21)	0.05 (0.21)	0.09 (0.21)
Never married (vs. married)		0.08 (0.11)	0.08 (0.11)	0.07 (0.11)
High school degree (vs. <high school)		−0.19 (0.20)	−0.18 (0.20)	−0.17 (0.20)
Some college (vs. <high school)		−0.34~ (0.20)	−0.33~ (0.20)	−0.31 (0.20)
4-Year college degree or higher (vs. <high school)		−0.51** (0.20)	−0.50** (0.20)	−0.47* (0.20)
Household income		−8.57E−7 (5.56E−7)	−8.38E−7 (5.57E−7)	−7.82E−7 (5.59E−7)
Family history of heart disease		0.10* (0.04)	0.09* (0.04)	0.09* (0.04)
Medication use for high blood pressure		0.28*** (0.08)	0.28*** (0.08)	0.27*** (0.08)
Medication use for high cholesterol		−0.18* (0.08)	−0.18* (0.08)	−0.18* (0.08)
Medication use for diabetes		0.69*** (0.12)	0.69*** (0.12)	0.64*** (0.12)
Depressive symptoms			0.04 (0.08)	0.04 (0.08)
Vigorous physical activity				−0.01* (0.01)
Moderate physical activity				−0.001 (0.008)
Past smoker (vs. never smoker)				−0.05 (0.07)
Current smoker (vs. never smoker)				0.10 (0.10)
Light or moderate drinker (vs. never drinker)				0.14 (0.14)
Heavy drinker (vs. never drinker)				0.11 (0.15)
Effect size (f^2)	0.02	0.25	0.25	0.26

[a] Adjusted for age.
[b] Adjusted for demographics (age, gender, race, marital status, education, and household income), family history of heart disease, and medication use for high blood pressure, high cholesterol, or diabetes.
[c] Adjusted for demographics, family history of heart disease, medication use, and depressive symptoms.
[d] Adjusted for demographics, family history of heart disease, medication use, depressive symptoms, and health behaviors (smoking status, alcohol consumption, vigorous physical activity, and moderate physical activity).
~ $p \le .10$.
* $p \le .05$.
** $p \le .01$.
*** $p \le .001$.

Table 5
Parameter estimates (standard error) for the association between one standard deviation increase in positive emotions and the standardized cardiometabolic risk score ($N = 854$)

	Model 1[a]	Model 2[b]	Model 3[c]	Model 4[d]
Positive emotions (standardized)	−0.05 (0.03)	−0.05~ (0.03)	−0.05 (0.03)	−0.04 (0.03)
Age at baseline	0.01* (0.003)	0.002 (0.003)	0.002 (0.003)	0.002 (0.003)
Female (vs. male)		−0.68*** (0.06)	−0.69*** (0.07)	−0.71*** (0.07)
Black (vs. white)		−0.01 (0.20)	−0.02 (0.20)	−0.02 (0.20)
Other race (vs. white)		0.17 (0.18)	0.15 (0.18)	0.15 (0.18)
Separated or divorced (vs. married)		0.11 (0.10)	0.10 (0.10)	0.08 (0.10)
Widowed (vs. married)		0.08 (0.21)	0.07 (0.21)	0.10 (0.21)
Never married (vs. married)		0.09 (0.11)	0.10 (0.11)	0.09 (0.11)
High school degree (vs. <high school)		−0.16 (0.20)	−0.15 (0.20)	−0.14 (0.20)
Some college (vs. <high school)		−0.29 (0.20)	−0.28 (0.20)	−0.27 (0.20)
4-Year college degree or higher (vs. <high school)		−0.47* (0.20)	−0.46* (0.20)	−0.43* (0.20)
Household income		−8.98E−7 (5.56E−7)	−8.73E−7 (5.58E−7)	−8.17E−7 (5.59E−7)
Family history of heart disease		0.09* (0.04)	0.09* (0.04)	0.09* (0.04)
Medication use for high blood pressure		0.27*** (0.08)	0.28*** (0.08)	0.27*** (0.08)
Medication use for high cholesterol		−0.18* (0.08)	−0.18* (0.08)	−0.18* (0.08)
Medication use for diabetes		0.70*** (0.12)	0.70*** (0.12)	0.65*** (0.12)
Depressive symptoms			0.05 (0.08)	0.05 (0.08)
Vigorous physical activity				−0.01* (0.01)
Moderate physical activity				−0.002 (0.01)
Past smoker (vs. never smoker)				−0.05 (0.07)
Current smoker (vs. never smoker)				0.10 (0.10)
Light or moderate drinker (vs. never drinker)				0.15 (0.14)
Heavy drinker (vs. never drinker)				0.12 (0.15)
Effect size (f^2)	0.01	0.24	0.24	0.25

[a] Adjusted for age.
[b] Adjusted for demographics (age, gender, race, marital status, education, and household income), family history of heart disease, and medication use for high blood pressure, high cholesterol, or diabetes.
[c] Adjusted for demographics, family history of heart disease, medication use, and depressive symptoms.
[d] Adjusted for demographics, family history of heart disease, medication use, depressive symptoms, and health behaviors (smoking status, alcohol consumption, vigorous physical activity, and moderate physical activity).
~ $p \le .10$
* $p \le .05$.
*** $p \le .001$.

current study) [44]. Why might well-being and cardiometabolic health be linked? A mediating behavioral pathway is an oft-cited explanation [2,45]. That is, greater well-being is thought to lead to healthier behaviors, which in turn promote health. Although direct tests of this hypothesis were not possible given available data, findings suggest that health behaviors may play a role, at least for life satisfaction. However, depressive symptoms attenuated positive emotion's association with cardiometabolic conditions before health behaviors were even considered. Thus, while behaviors such as exercise may be relevant in well-being's associations with health, other mediating factors—such as social support [45], sleep, or direct biological effects [2]—should also be evaluated.

Several limitations apply to the current research. Findings are generalizable only to mostly white U.S. middle-aged, primarily middle class, men and women. Findings are correlational and it is possible that the association between subjective well-being and cardiometabolic health is an artifact of unmeasured confounders such as genetic factors. However, few studies to date have identified genes or polymorphic gene scores that underlie phenotypic measures of well-being. It is also possible that the single item measure of life satisfaction may be unreliable and bias findings toward the null, although findings were nearly identical with a four-item composite of domain life satisfaction and other work demonstrates that single items are as valid as longer measures [20]. Moreover, positive emotions were assessed with regard to the previous 30 days, which could make them susceptible to contextual effects; however, the high correlation of positive emotions measured up to 11 years apart suggests a relatively persistent experience. In addition, self-reported health status could be subject to bias, although prior work suggests that self-report of medical conditions is reliable insofar as the conditions have become manifest [46]. Self-reported health status may not capture the presence of subclinical disease at baseline, however. Thus, future work should include baseline and repeated assessments of cardiometabolic biomarkers. Although similar versions of the cardiometabolic risk score have been validated [12], the exact cardiometabolic risk score used here has not been validated against hard health outcomes. Moreover, having time-to-event data for cardiometabolic conditions would have allowed us to conduct survival analyses rather than less powerful Poisson regression analyses. Finally, the associations of interest are relatively small (e.g., greater subjective well-being was associated with 4–7% reduced risk of incident cardiometabolic conditions). However, even small associations can have an impact at the population level [47], especially if effects accumulate across time [48,49], which subjective well-being and cardiometabolic risk could be expected to do.

These limitations are balanced by a longitudinal design that included only participants known to be initially free from heart problems or stroke, which reduces concerns about reverse causality. Moreover, incident analyses further excluded individuals with other cardiometabolic conditions (e.g., diabetes) and risk score analyses controlled for relevant medications. The approximate decade-long follow-up during early and middle adulthood not only provided a lengthy period in which to detect changes in cardiometabolic health, but also focused on an important point in life when risk factors begin to accumulate [50,51] and when early warning may be particularly valuable. Other strengths include consideration of possible confounders and underlying pathways. Subjective well-being was assessed with measures reflecting both its cognitive and affective components. Past work has more frequently investigated positive emotions in relation to health [52], so the current study further contributes to findings regarding life satisfaction. Additionally, by combining across multiple biomarkers, the cardiometabolic risk score may serve as an "early warning system" for likelihood of future disease [53]. The findings with the cardiometabolic risk score, which is comprised of modifiable factors, raise an important question for future work: namely, whether improving life satisfaction would reduce cardiometabolic risk downstream.

In sum, this longitudinal study investigated subjective well-being in relation to incident cardiometabolic conditions and early cardiometabolic risk. Life satisfaction was consistently associated with incident cardiometabolic conditions and biological deterioration constituting risk for development of manifest disease; this association may be partly accounted for by health-related behaviors. Positive emotions were also associated with incident conditions in minimally-adjusted models, but not with biological deterioration. This weaker finding for positive emotions is consistent with null findings that have been previously reported [5], and suggests that different types of well-being may have differential associations with cardiometabolic health, perhaps depending on how closely linked they are to distress [2]. In particular, life satisfaction could be considered a health asset. More broadly, identifying health assets may be an important future direction, thereby providing novel targets for prevention and intervention. Taken together, findings suggest one possible protective factor or health asset—namely, life satisfaction—that may foreshadow who will eventually experience cardiometabolic risk and cardiometabolic conditions.

Conflict of interest

The authors have no competing interests to report.

Acknowledgements

Appendix A. Supplementary material

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.jpsychores.2016.03.018.

References

[1] E. Diener, E.M. Suh, R.E. Lucas, H.L. Smith, Subjective well-being: three decades of progress, Psychol. Bull. 125 (1999) 276–302.

[2] J.K. Boehm, L.D. Kubzansky, The heart's content: the association between positive psychological well-being and cardiovascular health, Psychol. Bull. 138 (2012) 655–691.

[3] J.K. Boehm, C. Peterson, M. Kivimaki, L.D. Kubzansky, Heart health when life is satisfying: evidence from the Whitehall II cohort study, Eur. Heart J. (2011).

[4] K.W. Davidson, E. Mostofsky, W. Whang, Don't worry, be happy: positive affect and reduced 10-year incident coronary heart disease: the Canadian Nova Scotia Health Survey, Eur. Heart J. 31 (2010) 1065–1070.

[5] H. Nabi, M. Kivimaki, R. De Vogli, M.G. Marmot, A. Singh-Manoux, Positive and negative affect and risk of coronary heart disease: Whitehall II prospective cohort study, BMJ 337 (2008) a118.

[6] A. Steptoe, J. Wardle, Positive affect and biological function in everyday life, Neurobiol. Aging 26 (2005) 108–112.

[7] C.D. Ryff, G.D. Love, H.L. Urry, D. Muller, M.A. Rosenkranz, E.M. Friedman, et al., Psychological well-being and ill-being: do they have distinct or mirrored biological correlates? Psychother. Psychosom. 75 (2006) 85–95.

[8] V.K. Tsenkova, G.D. Love, B.H. Singer, C.D. Ryff, Socioeconomic status and psychological well-being predict cross-time change in glycosylated hemoglobin in older women without diabetes, Psychosom. Med. 69 (2007) 777–784.

[9] A. Steptoe, K. O'Donnell, E. Badrick, M. Kumari, M. Marmot, Neuroendocrine and inflammatory factors associated with positive affect in healthy men and women: The Whitehall II study, Am. J. Epidemiol. 167 (2008) 96–102.

[10] A. Steptoe, J. Wardle, M. Marmot, Positive affect and health-related neuroendocrine, cardiovascular, and inflammatory processes, Proc. Natl. Acad. Sci. 102 (2005) 6508–6512.

[11] P.E. Szmitko, S. Verma, C-reactive protein and the metabolic syndrome: Useful addition to the cardiovascular risk profile? J. Cardiometab. Syndr. 1 (2006) 66–69 quiz 70-1.

[12] M. Marino, Y. Li, M.J. Pencina, R.B. D'Agostino Sr., L.F. Berkman, O.M. Buxton, Quantifying cardiometabolic risk using modifiable non-self-reported risk factors, Am. J. Prev. Med. 47 (2014) 131–140.
[13] P. Palatini, Heart rate and the cardiometabolic risk, Curr. Hypertens. Rep. 15 (2013) 253–259.
[14] B.T. Radler, C.D. Ryff, Who participates? Accounting for longitudinal retention in the MIDUS national study of health and well-being, J. Aging Health 22 (2010) 307–331.
[15] G. Dienberg Love, T.E. Seeman, M. Weinstein, C.D. Ryff, Bioindicators in the MIDUS National Study: protocol, measures, sample, and comparative context, J. Aging Health 22 (2010) 1059–1080.
[16] V.L. Roger, A.S. Go, D.M. Lloyd-Jones, E.J. Benjamin, J.D. Berry, W.B. Borden, et al., Heart disease and stroke statistics—2012 update: a report from the American Heart Association, Circulation 125 (2012) e2–e220.
[17] T.A. Pearson, G.A. Mensah, R.W. Alexander, J.L. Anderson, R.O. Cannon 3rd, M. Criqui, et al., Markers of inflammation and cardiovascular disease: Application to clinical and public health practice: a statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association, Circulation 107 (2003) 499–511.
[18] C. Hadley, The pattern of human concerns, Rutgers University Press, New Brunswick, NJ, 1965.
[19] C.W. Karlson, M.W. Gallagher, C.A. Olson, N.A. Hamilton, Insomnia symptoms and well-being: longitudinal follow-up, Health Psychol. 32 (2013) 311–319.
[20] F. Cheung, R.E. Lucas, Assessing the validity of single-item life satisfaction measures: results from three large samples, Qual. Life Res. Int. J. Qual. Life Asp. Treat. Care Rehab. 23 (2014) 2809–2818.
[21] K.M. Prenda, M.E. Lachman, Planning for the future: a life management strategy for increasing control and life satisfaction in adulthood, Psychol. Aging 16 (2001) 206–216.
[22] U. Schimmack, P. Krause, G.G. Wagner, J. Schupp, Stability and change of well being: an experimentally enhanced latent state-trait-error analysis, Soc. Indic. Res. 95 (2010) 19–31.
[23] D.K. Mroczek, C.M. Kolarz, The effect of age on positive and negative affect: a developmental perspective on happiness, J. Pers. Soc. Psychol. 75 (1998) 1333–1349.
[24] A.D. Ong, D. Exner-Cortens, C. Riffin, A. Steptoe, A. Zautra, D.M. Almeida, Linking stable and dynamic features of positive affect to sleep, Ann. Behav. Med. 46 (2013) 52–61.
[25] E.B. Weiser, Associations between positive and negative affect and 12-month physical disorders in a national sample, J. Clin. Psychol. Med. Settings 19 (2012) 197–210.
[26] J.K. Boehm, Y. Chen, D.R. Williams, C. Ryff, L.D. Kubzansky, Unequally distributed psychological assets: are there social disparities in optimism, life satisfaction, and positive affect? PLoS ONE 10 (2015) e0118066.
[27] S. Lyubomirsky, K.M. Sheldon, D. Schkade, Pursuing happiness: the architecture of sustainable change, Rev. Gen. Psychol. 9 (2005) 111–131.
[28] R.B. Wallace, A.R. Herzog, Overview of the health measures in the Health and Retirement Study, J. Hum. Resour. 30 (1995) S84–S107.
[29] R.P. Juster, B.S. McEwen, S.J. Lupien, Allostatic load biomarkers of chronic stress and impact on health and cognition, Neurosci. Biobehav. Rev. 35 (2010) 2–16.
[30] K.E. King, J.D. Morenoff, J.S. House, Neighborhood context and social disparities in cumulative biological risk factors, Psychosom. Med. 73 (2011) 572–579.
[31] A.L. Non, M. Rewak, I. Kawachi, S.E. Gilman, E.B. Loucks, A.A. Appleton, et al., Childhood social disadvantage, cardiometabolic risk, and chronic disease in adulthood, Am. J. Epidemiol. (2014).
[32] O. Naggara, J. Raymond, F. Guilbert, D. Roy, A. Weill, D.G. Altman, Analysis by categorizing or dichotomizing continuous variables is inadvisable: an example from the natural history of unruptured aneurysms, AJNR Am. J. Neuroradiol. 32 (2011) 437–440.
[33] K. Wickrama, C.W. O'Neal, T.K. Lee, T. Wickrama, Early socioeconomic adversity, youth positive development, and young adults' cardio-metabolic disease risk, Health Psychol. 34 (2015) 905–914.
[34] R.C. Kessler, G. Andrews, D. Mroczek, B. Ustun, H. Wittchen, The World Health Organization Composite International Diagnostic Interview Short-Form (CIDI-SF), Int. J. Methods Psychiatr. Res. 7 (1998) 171–185.
[35] N. Grant, J. Wardle, A. Steptoe, The relationship between life satisfaction and health behavior: a cross-cultural analysis of young adults, Int. J. Behav. Med. 16 (2009) 259–268.
[36] D. Spiegelman, E. Hertzmark, Easy SAS calculations for risk or prevalence ratios and differences, Am. J. Epidemiol. 162 (2005) 199–200.
[37] A.S. Selya, J.S. Rose, L.C. Dierker, D. Hedeker, R.J. Mermelstein, A practical guide to calculating Cohen's f(2), a measure of local effect size, from PROC MIXED, Front. Psychol. 3 (2012) 111.
[38] J. Cohen, A power primer, Psychol. Bull. 112 (1992) 155–159.
[39] P. Burton, L. Gurrin, P. Sly, Extending the simple linear regression model to account for correlated responses: an introduction to generalized estimating equations and multi-level mixed modelling, Stat. Med. 17 (1998) 1261–1291.
[40] J.A. Hanley, A. Negassa, M.D. Edwardes, J.E. Forrester, Statistical analysis of correlated data using generalized estimating equations: an orientation, Am. J. Epidemiol. 157 (2003) 364–375.
[41] N.J. Horton, S.R. Lipsitz, Review of software to fit generalized estimating equation regression models, Am. Stat. 53 (1999) 160–169.
[42] R.C. Kessler, P. Berglund, O. Demler, R. Jin, D. Koretz, K.R. Merikangas, et al., The epidemiology of major depressive disorder: results from the National Comorbidity Survey Replication (NCS-R), J. Am. Med. Assoc. 289 (2003) 3095–3105.
[43] D.M. Lloyd-Jones, A.R. Dyer, R. Wang, M.L. Daviglus, P. Greenland, Risk factor burden in middle age and lifetime risks for cardiovascular and non-cardiovascular death (Chicago Heart Association Detection Project in Industry), Am. J. Cardiol. 99 (2007) 535–540.
[44] C.L. Keyes, The nexus of cardiovascular disease and depression revisited: the complete mental health perspective and the moderating role of age and gender, Aging Ment. Health 8 (2004) 266–274.
[45] A. Steptoe, S. Dockray, J. Wardle, Positive affect and psychobiological processes relevant to health, J. Pers. 77 (2009) 1747–1776.
[46] G.A. Colditz, P. Martin, M.J. Stampfer, W.C. Willett, L. Sampson, B. Rosner, et al., Validation of questionnaire information on risk factors and disease outcomes in a prospective cohort study of women, Am. J. Epidemiol. 123 (1986) 894–900.
[47] H.S. Friedman, S. Booth-Kewley, The 'disease-prone personality': a meta-analytic view of the construct, Am. Psychol. 42 (1987) 539–555.
[48] B.W. Roberts, N.R. Kuncel, R. Shiner, A. Caspi, L.R. Goldberg, The power of personality: the comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes, Perspect. Psychol. Sci. 2 (2007) 313–345.
[49] R.P. Abelson, A variance explanation paradox: when a little is a lot, Psychol. Bull. 97 (1985) 129–133.
[50] T. Nwankwo, S.S. Yoon, V. Burt, Q. Gu, in: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics (Eds.),Hypertension Among Adults in the United States: National Health and Nutrition Examination Survey, 2011–2012, 2013 (Hyattsville, MD).
[51] C.L. Ogden, M.D. Carroll, B.K. Kit, K.M. Flegal, in: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics (Eds.), Prevalence of obesity among adults: United States, 2011–2012, National Center for Health Statistics, Hyattsville, MD, 2013.
[52] S.D. Pressman, S. Cohen, Does positive affect influence health? Psychol. Bull. 131 (2005) 925–971.
[53] T.L. Gruenewald, T.E. Seeman, C.D. Ryff, A.S. Karlamangla, B.H. Singer, Combinations of biomarkers predictive of later life mortality, Proc. Natl. Acad. Sci. U. S. A. 103 (2006) 14158–14163.