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People Typically Experience Extended Periods of Relative Happiness or Unhappiness due to Positive Feedback Loops Between LS and Variables Which are Both Causes and Consequences of LS

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Abstract

Long term panel data enable researchers to construct trajectories of LS for individuals over time. Bar charts of trajectories, and subsequent statistical analysis, show that respondents typically spend multiple consecutive years above and below their own long-term mean level of LS. We attempt to explain these multi-year waves of change by estimating structural equation models with two-way causal links between LS and variables usually treated as causes of LS, including health, frequency of physical exercise and frequency of social activities. Results are interpreted as showing positive feedback loops between these variables and LS, such that gains and losses of LS tend to be reinforced over time.

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1. Introduction

Until recently the most widely accepted theory of Life-Satisfaction (LS) was probably set-point theory (Lykken / Tellegen, 1996). The central claim of this theory is that adult LS is stable, except for temporary fluctuations due to life events. Analyses of long term panel data have gradually undermined the claim (Fujita / Diener, 2005; Headey / Muffels / Wagner, 2010). It is noteworthy that almost all contributors to a recent volume on "The Stability of Happiness" (Sheldon / Lucas, 2014) concluded that set-point theory is flawed, and that the main focus of research should now be on accounting for change. For a discussion of set points, and the role of personality traits and other fixed effects in econometric estimation of subjective well-being, see Easterlin (1974), Winkelmann / Winkelmann (1998), Ferrer-i-Carbonell / Frijters (2004), Frijters et al (2004), and Clark et al (2008).

In this paper we make some initial moves. We begin by analysing *individual trajectories of LS;* specifically, the trajectories of the 1873 prime age (25 – 64)

SOEP respondents who recorded their LS for twenty consecutive years in 1991 – 2010. Visual inspection of these trajectories, and subsequent statistical analysis, make it clear that many individuals go through extended periods in which their LS is either above or below their own long-term mean. In other words, people typically experience *multi-year periods of (relative) happiness or unhappiness.*

The rest of the paper involves trying to explain this phenomenon. We re-examine relationships between LS and variables usually treated as causes of LS: health, exercise and frequency of socialising with friends, relatives and neighbours. In reviews of LS research it is common to point out that these and many other variables could just as well be consequences of LS, or both causes and consequences (Diener / Suh / Lucas / Smith, 1999; Frey / Stutzer, 2002). However, in empirical work the possibility of two-way causation is usually ignored. In the case of the link between health and LS, this is plainly a mistake. It has been shown in prospective studies that happy people live longer, which could only happen if happiness promotes better health (e.g. Deeg / van Zonneveld, 1989). Although a priori reasoning is not so compelling in relation to other possible two-way links, it is surely plausible to hypothesise that happiness could cause as well as be caused by, frequency of exercise and social participation.

2. Waves of Change in LS

Below are six bar charts which show the trajectories of the first and last three SOEP respondents in the datafile who recorded their LS every year from 1991 to 2010. Clearly, six cases do not constitute a representative sample, but their LS trajectories are instructive, especially in view of the stable, flat-line trajectory predicted by set-point theory.

Visual inspection suggests that Cases A, B and D have roller-coaster lives. They do not appear to have any set-point around which their LS fluctuates. Case A was a 33 year old married woman in 1991, with eighteen years of education and a household income close to the national median. She rated about average on the personality traits of neuroticism and extroversion. Her LS rating was 8 on the 0 – 10 scale in 1991. It then fell every year until 1994, rose greatly in 1995, then kept falling again until 1998, was 4 or 5 for most of the time between 1998 and 2006, and then 6 or 7 in 2007 – 10.

By contrast, Cases C, E, and F had quite stable levels of LS in this period. Take Case F, a man age 30 in 1991 with 11.5 years of education and below median income, who rated above average on neuroticism and average on extroversion. His LS rating was 6 on the 0 – 10 scale in ten of these twenty years, and 7 in most other years. He dropped to 5 on just three occasions. Clearly, this man could reasonably be said to have an LS set-point of between 6 and 7.

Figure 1: Individual Trajectories of Life Satisfaction: Bar Charts

Inspection of many more trajectories suggests that about half the population just do not have a set-point. For the other half, the concept more or less applies.

A key feature of the trajectories is that, instead of recording short term fluctuations around a set-point, many respondents appear to spend several consecutive years above… or several years below their own long term (20-year) mean level of LS. This is suggested by pairs of within-person (fixed effects) regressions in which LS in year t is the independent variable and the dependent variables are, successively (i) LS in year t+1 and t−1 (ii) t+2 and t−2 (iii) t+3 and t−3 and (iv) t+4 and t−4.[1] The first pair of (metric) regression coefficients are 0.248 and 0.256, the second pair are 0.143 and 0.153, the third are 0.088 and 0.096, and the fourth are 0.045 and 0.050 ($p<0.001$). Recall that, in fixed effects analysis, the coefficients relate deviations from each individual's own grand mean on the *y* variable to deviations from his/her grand means on explanatory variable(s). So the coefficients indicate that, if an individual is above his/her own long term (1991–2010) mean of LS in a particular year, then he/she is more rather than less likely to be above it in each (but, given the size of the coefficients, not all) of the four years beforehand and the four years afterwards.[2] These apparently innocuous regression results may have non-trivial implications for LS theory. They show that LS tends to change in medium term periods rather than fluctuating short term around a stable set-point.

3. Two-way Causation Models: Positive Feedback Loops

We now attempt to account for medium term periods of relatively high and low LS with two-way causation models which include positive feedback loops between LS and variables usually treated as causes of LS. It must be conceded that disentangling two-way causation issues remains problematic, even with long term panel data. Previous researchers, using panel data, have claimed to find two-way causation between LS and domain satisfactions (mainly marriage and job satisfaction), and between LS and quality of social networks, health, housing and volunteering (Headey/Veenhoven/Wearing, 1991; Mathison et al, 2007; Nagazato/Schimmack/Oishi, 2011). These studies have been roundly criticised by other researchers who claim that structural equation models involving two-way causation can be unstable in the sense that small, reasonably plausible changes in model specification can lead to quite different conclusions about direction of causation (Scherpenzeel/Saris, 1996; see also Wooldridge, 2010).

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[1] Controlling for age and age-squared.

[2] Another way of making the same point: among individuals who were above their own 1991–2010 grand mean of LS in any particular year, just over 25% remained above it for all of the next four years. By chance only 6.25% would have done so.

In this paper we suggest that more stable and better fitting models of LS can be estimated by conceptualising relationships in terms of *Granger-causation* (Granger, 1969; Granger/Newbold, 1974). The intuitions behind Nobel Laureate, Clive Granger's concept of causation, are that (1) causes must precede effects and (2) if earlier values of an *x* variable (a presumed cause) can be shown to be statistically significantly related to later values of a *y* variable (a presumed effect) in equations which include multiple lagged versions of *y*, then it may be inferred that *x* is one cause of *y*. Granger's view is that, by including multiple lags of *y*, the researcher will usually take care of most of the variance due to omitted variables ('unobserved heterogeneity'), and so will avoid biased estimates and the autocorrelated error terms that indicate bias (Granger, 1969).

We suggest extending Granger's approach to two-way causation and panel data, and hypothesizing that *x* and *y* may cause each other. We estimate 5-wave panel models in which an *x* variable (e.g. health) at time t is hypothesized to influence LS at t+1, and LSt is hypothesized to influence *x* at t+1. All (Granger) lags of LS (t−1, t−2 etc) and of the *x* variable are initially included in equations. Also included are correlations linking the error terms of LS at *x* at each of the five waves. These correlated error terms should be routinely included in structural equation panel models as a further step towards netting out the effects of omitted variables (Finkel, 1996; Wooldridge, 2010).

Model identification

Specific strategies are always needed to achieve identification of two-way causation models. There are three main approaches, all of which are implemented in this paper:

(1) Exogenous variables may serve as instrumental variables. In our models fourteen exogenous variables – standard socio-economic variables, personality traits[3] and years in the panel – are linked to wave 1 versions of *x* and LS, but not to later waves. So they act as instruments to identify the equations for later waves.

(2) In previous research lagged versions of *x* and LS were commonly used as instruments to identify equations. However, in the models in this paper, the inclusion of multiple Granger-style lags increases rather than reduces the number of free parameters to be estimated. Our models are nevertheless still identified, due to inclusion of the exogenous variables. Further, in later computer runs we modified models by removing some Granger-style lags – specifically longer term lagged effects of *x* on LS, and LS on *x*. Consequently, in our final models, this second approach to identification came back into play.

[3] The so-called Big Five personality traits: neuroticism, extroversion, openness, agreeableness and conscientiousness (Costa/McCrae, 1991).

(3) Equality constraints may be imposed. That is, sets of coefficients may be fixed equal to each other, so reducing the number of free parameters to be estimated (Finkel, 1995). No equality constraints were imposed in initial models. However, both a priori reasoning and first computer runs indicated that, empirically, some causal links appeared to be almost exactly the same in consecutive waves of data. So equality constraints were added in later runs; they improved model parsimony without significantly worsening model fit.

The periods covered by our five-wave panel models are overlapping: 1991 – 95, 1992 – 1996 … up to 2006 – 10.[4] The reason for using all available five-year periods, instead of just four non-overlapping periods is to obtain more reliable results due to larger sample numbers. An assumption which has to be met for this decision to be sensible is that relationships among variables do not change much within the overall time period. Inspection of bivariate correlations within and across waves suggests that this assumption is plausible.

4. Estimates of Structural Equation Models: Positive Feedback Loops Between LS and *x* Variables

In analysing the five-wave panel data, we first estimated relationships between LS and each particular *x* variable, deploying a Granger-style model with multiple lags. We then modified this model, removing statistically insignificant links and imposing equality constraints where appropriate. We then compared our final Granger-style model – both substantive causal estimates and measures of model fit – with results from one-way causal models, and also from cross-lagged and simultaneous causation models of the kind deployed in previous studies of two-way causation.

Health and LS

Our first model assesses two-way links between LS and health. An internationally widely used, self-assessed health measure has been included in SOEP since the early 1990s. Respondents rate their own health on a 1 – 5 scale ('bad' to 'very good').

Our first step was to estimate a 'full' Granger-style model; that is, a model in which all lags of both self-assessed health and of LS were included in equations. It was immediately clear that all lags of the health measure were statistically significant in health equations, and that all lags of LS were significant in LS equations. This already suggested that the Granger approach was going to

[4] Strictly speaking, maximum likelihood (ML) estimation of structural models requires interval or ratio scales. Our scales are ordinal. It has become fairly routine in LS research to treat ordinal scales as interval-level; we have followed this lead. It was essential to do so to be able to use the measures of model fit described below.

greatly improve model fit, compared with previous approaches. It was also clear, however, that the model could be pruned. While health lagged by one year had a statistically significant effect on LS, and LS lagged by one year had a significant effect on health, little additional variance was accounted for by 'extra' (t−2, t−3 etc) cross-lags. No extra cross-lags of LS were significant, and although some two-year cross-lags of health were significant at the 0.05 level, their effects were substantively trivial. So we dropped extra cross-lags from our final model.

Table 1 gives both metric and standardized maximum likelihood estimates of the coefficients of main interest. The standardized results are particularly useful because they enable us to compare effect sizes. Table 1 also provides a fairly comprehensive set of measures of model fit. Readers are referred to the causal modelling literature for a description of these measures (e.g. Browne and Cudeck, 1993). Here it may be noted that a Root Mean Squared Error of Approximation (RMSEA) below 0.05 is conventionally regarded as satisfactory, as is a Standardized Root Mean Squared Residual (SRMR) below 0.08. Comparative Fit Indices (CFI) should be over 0.95, as should results for the Tucker-Lewis Index (TLI).

Table 1

Two-Way Causal Links
Between Self-Assessed Health and LS (N=35698)[a]

	Metric ML estimates	*Standardized ML estimates*	*Measures of model fit*	
$Health_t$ –>LS_{t+1}	0.190*** (0.005)	**0.093*** (0.003)**	LR Chi-square (df=136)	3773.20***
LS_t –>$Health_{t+1}$	0.027*** (0.001)	**0.056*** (0.002)**	RMSEA	0.027
Correlated error term	0.157*** (0.004)	**0.213*** (0.005)**	SRMR	0.027
			CFI	0.984
			TLI	0.977

a. N=person years.
*** significant at the 0.001 level.

Positive feedback loops are found between self-assessed health and LS. The size of the standardized path from Health to LS (0.093 $p<0.001$) is greater the path from LS to Health (0.056 $p<0.001$). The relative size of the two-way links is approximately the same for men and women, and for older and younger people. Correlated error terms, reflecting the effects of omitted variables, are also statistically significant (0.213 $p<0.001$).

This Granger-style health model has a satisfactory fit to the data. The RMEA and the SRMR are both 0.027, the CFI is 0.984 and the TLI is 0.977. No statistically significant autocorrelated errors are found.

It is crucial to compare the fit of this Granger-style model to reasonable alternatives. All the other models we consider are 'nested' versions of the Granger-style model, so model fit can be directly compared (Browne / Cudeck, 1993). A model with only one-way causation from health to LS, which includes 'extra' Granger lags, is a much worse fit to the data: RMSEA=0.104, SRMR=0.066, CFI=0.829 and TLI=0.757. A one-way model with causation running only from LS to health is even worse: RMSEA=0.105, SRMR=0.067, CFI=0.817 and TLI=0.740. Equally to the point, the Granger model is a closer fit to the data than either a cross-lagged model without 'extra' Granger lags, or a simultaneous causation model without extra lags. Both these alternative models give the following fit readings: RMSEA=0.055, SRMR=0.037, CFI=0.954, TLI=0.934.

Despite fitting the data less well, it is important to record that substantive estimates for the two-way links between LS and Health in both the cross-lagged and simultaneous causation models are broadly consistent with, although somewhat larger than estimates for our preferred model. The reason for these somewhat higher estimates is presumably that one consequence of omitting extra 'Granger' lags of outcome variables is to give an upward bias to estimates of the two-way links of main interest (Granger / Newbold, 1974).

In summary, finding significant two-way links between health and LS is compatible with the interpretation that feedback loops are operating, so that having better or worse health results in more or less LS, which in turn results in better or worse health, and so on.

Exercise and LS

SOEP respondents are asked annually about how frequently they engage in active sport or exercise. The response scale runs from 0 ('not at all') to 5 ('every day').

A full multiple-lag Granger model for links between frequency of exercise and LS indicated that both first and second lags of the two-way causal links relationships were statistically significant. However, the effect sizes of the second lags were small, suggesting just lingering effects. Again, as with the health model, inspection of the input correlation matrix made it clear that relationships within and between waves of data were quite similar over time. So we again opted for a model in which the cross-lagged causal links of main interest were constrained to be equal. As before, all Granger lags of outcome variables were included.

Two-way causal links are found between exercise and LS. Increased frequency of exercise increases LS. Then, in a positive feedback loop, enhanced LS leads to more frequent exercise. A comparison of the standardized coefficients indicates that the strength of the link from exercise to LS (0.036

p<0.001) is greater than the link from LS to exercise (0.014 p<0.001). Correlated error terms, reflecting the effects of omitted variables, are again statistically significant (0.027 p<0.001).

The Granger-style model has a satisfactory fit to the data: RMSEA=0.026, SRMR=0.022, CFI=0.987, TLI=0. The model is a closer fit to the input data than a one-way causation model (exercise->LS), or than either a cross-lagged or a simultaneous causation model. A one-way model has these fit readings: RMSEA=0.047, SRMR=0.025, CFI=0.960, TLI=0.943. Cross-lagged and simultaneous causation models both give the following fit readings: RMSEA=0.051, SRMR=0.030, CFI=0.956, TLI=0.937. The simultaneous model has negative correlated terms, which are almost certainly a sign of poor model specification (Finkel, 1995).

Frequency of socialising/social participation and LS

The social participation index used here combines two items about frequency of 'meeting with friends, relatives or neighbours' and 'helping out friends, relatives or neighbours'.

A modest degree of two-way causation is found between social participation and LS. The standardized link from social participation to LS is 0.030 (p<0.001), while the reverse link is 0.020 (p<001). The two-way links can again be interpreted as indicating positive feedback loops.

Alternative models – one-way causation, cross-lagged and simultaneous models – fit the data less well. The cross-lagged and simultaneous models both have CFIs of 0.950 and TLIs of 0.928, together with RMSEAs and SRMRs which are somewhat higher than the Granger-style model. Estimated correlated error terms in the simultaneous model are again negative, probably indicating model misspecification.

A multivariate two-way causal model: links between exercise, social participation, health and LS

So far we have only assessed two-way causation between LS and *x* variables, taking one *x* at a time. However, it is a plausible hypothesis that health, exercise and social participation exert combined and perhaps more or less simultaneous effects on LS. If so, they should be entered at the same step in a causal model. A multivariate model indicated that all three variables still have statistically significant reciprocal links with LS, although the standardized coefficients are slightly lower than those reported above for bivariate models. In summary, it appears that health, exercise and active social participation can combine to change happiness, and that changes in happiness then affect subsequent health, and subsequent frequency of exercise and social participation.

5. Discussion

The main current challenge for LS researchers is to develop a theory of change. Set-point theory is purely a theory of stability and does not account for evidence from panel surveys which shows that the LS trajectories of many respondents are subject to medium or long term change, and certainly do not fluctuate around a stable set-point.

The suggestion in this paper is that *positive feedback loops* between LS and associated variables may help to account for medium term periods of change. If our models are valid, they provide a preliminary explanation of why an individual might enjoy an extended period of time with a level of LS above his/her own long term average, and then perhaps spend several consecutive years below this long term average.

The feedback loops in the models should not be over-interpreted. They might appear to imply that the same people keep getting happier and happier, while others get steadily more miserable. However, since these feedback loop models are not within-person models, the importance of which have been well documented since Winkelmann/Winkelmann (1988), they do not imply that. Rather they indicate how changes (positive or negative changes) may be maintained for an extended period of time. Psychological adaptation mechanisms doubtless come into play and partly counteract the effects of both positive and negative changes. So, for example, one would expect that a change in exercise levels, or a change in health, would have a greater effect on LS in the short term than in the longer term. People partially adapt to almost all changes, but adaptation is often far from complete (Wilson/Gilbert, 2008).

From this perspective one key issue for future research is to explain why some individuals record more or less permanent changes, and 'lock in' at levels of LS substantially different from what they experienced earlier in life. This paper has focussed on medium term change. So far as we know, there is virtually no empirical research accounting for long term change.

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