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Is dispositional happiness contagious? The impact of the well-being of family members on individual well-being

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Psychologists have historically been much more interested in negative states than positive states (Diener, Suh, Lucas, & Smith, 1999); however, this focus has changed as people have recognized that positive emotions are predictive of a variety of health-related outcomes, like who will contract illness (Smart Richman et al., 2005) and who will live longest (Danner, Snowdon, & Friesen, 2002). This begs the question, how do people become happy? What factors predict happiness? To answer these questions, we must first define what we mean by happiness.

Consensus has not yet been reached on how to best assess happiness. Diener (2009) pointed out that the specific measurements used in happiness research can lead to different conclusions; therefore, it is important to consider the degree to which different studies assess the same construct. Much research in this area focuses on subjective well-being, a construct composed of positive affect, negative affect, and life satisfaction (e.g., Bartels & Boomsma, 2009; DeNeve & Cooper, 1998; Diener, 2009). Although the measure used in the current study—the well-being scale of the Multidimensional Personality Questionnaire (MPQ) (Tellegen & Waller, 2008)—is typically considered a personality measure, it similarly assesses affect and a cognitive evaluation of life. We suspect that an optimistic mood state that persists over time (i.e., happiness) and the tendency to view events in a positive manner (i.e., affect) are likely to be highly related. Additionally, considerable fluctuation in well-being has been demonstrated by retest reliabilities around only .50 (e.g., Lykken & Tellegen, 1996), which is similar to the stabilities reported by Diener (2009) for SWB measures. In fact, the 3-year stability of well-being in the current study was only .41, the lowest of all MPQ scales in this sample (other scales' correlations ranged from .54–.73). Furthermore, in a recent multivariate study of SWB and personality, Weiss, Bates, and Luciano (2008) found that although nonshared environmental influences differed, the genetic influences on SWB were completely accounted for by genetic influences on personality as measured by the five-factor model. Thus, it's plausible that SWB and MPQ well-being describe the same construct.

Regardless of how happiness is defined, twin studies have consistently shown that genetic factors account for 40–50% of happiness variance, measured by both MPQ well-being and SWB (Bartels & Boomsma, 2009; Bouchard & McGue, 2003; Lykken & Tellegen, 1996). In addition, nonshared environmental influence (e^2 , that which makes siblings different) has

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been shown to comprise most or all of the environmental influence on happiness while shared environmental influence (c^2 , that which makes siblings similar) is usually estimated as zero (e.g., Lykken & Tellegen, 1996). However, the twin design has important limitations that can lead to doubt about these research findings. For instance, it is assumed that monozygotic (MZ) twins do not share more similar environments than do dizygotic (DZ) twins. If MZ twins do in fact share more similar environments and if similarity is related to the phenotype of interest, heritability may be overestimated and shared environmental effects underestimated. Indeed, research utilizing an entirely different (non-behavior genetic) method calls into question the findings of these twin studies.

A recent study of the impact of the happiness level of people in one's social network has garnered much attention (Fowler & Christakis, 2008). The authors performed a social network analysis of happiness on participants of the Framingham Heart Study. Using regression models of target (focal individual) happiness on the happiness of people connected to the target, including relatives, friends, spouses, coworkers, and neighbors, and with adjustment for various covariates, they found that individuals were 15.3% more likely to be happy if a close associate was happy (averaged across associate type). This effect continued up to three degrees of separation (e.g., friend's friend's friend). These findings have been interpreted as evidence of happiness as a "contagion" that is spread from person to person in social networks.

These authors have used the same data and methodology to examine the social network effects of many health-related phenomena, including obesity (Christakis & Fowler, 2007) and smoking (Christakis & Fowler, 2008), for example. They have found that individuals cluster together according to their behavioral status on every outcome. It is easy to imagine how others' behavior may influence an individual to gain weight or start smoking; however, similar results have been found for outcomes for which contagion makes little sense (i.e., skin problems, headaches, and height; Cohen-Cole & Fletcher, 2008a). It is thus important to consider alternative explanations for similarity among individuals within a network.

One possibility is that unmeasured variables contribute to network similarity; if they are positively correlated with the outcome of interest, then the estimate of a social network effect will be biased upward. Cohen-Cole and Fletcher (2008b) pointed out that the social network design cannot directly control for all relevant contextual influences and so cannot rule them out as a possibility. Another possible explanation for similarity is selection (homophily). Fowler and Christakis attempted to account for this by including previous alter and ego happiness in their regressions, but unless selection is conditioned only on these variables, homophily is still a possibility (Aral, Muchnik, & Sundararajan, 2009; Cohen-Cole & Fletcher, 2008a, 2008b). Since individuals create their environments through selection of jobs, locations, activities, etc. that expose them to individuals with similar attributes, it is possible that the people with whom they spend the most time, live near, and date already share important behavioral characteristics.

A final limitation of Fowler and Christakis (2008) is that the inclusion of relatives in the network confounds environmental and genetic influences. Family members share both genes and environment, so similarity may be due to either. The inclusion of relatives raises the

possibility that the resemblance between egos and alters reflects shared genetic effects rather than social contagion. In fact, of the 4,739 egos included in the study, only 45% had a friend in the network (average = 0.7 friend ties per ego), only 39% had a coworker in the network, and only 10% had a neighbor in the network. So, out of the average 10.4 ties per ego, the majority of them must have been family members.

We tested the contagion hypothesis of happiness by comparing correlations among both biological and adoptive family members; if happiness were contagious, we would expect that family members would experience similar levels of happiness regardless of biological relation. Additionally, we employed biometrical modeling of well-being, which decomposes variance into components of additive genetic (a^2), c^2 , and e^2 . To test the significance of parameters, fit was compared between the full model (containing all parameters, A, C, and E) and a model with fewer parameters. Fit is often evaluated by change in chi square value, which is itself distributed as a chi square. If we could drop the shared environment parameter from the model without significant loss of fit, we could conclude that it is not significantly different from zero and thus not an important influence on well-being. Figure 1 shows the model of the adoption design; note that the observed correlation between adoptive siblings directly estimates the shared environmental component. This is because adoptees share 0% of their genes but 100% of their environment, so any similarity can be attributed to the shared environment.

The inclusion of adoptees allows us to directly control for genetic relatedness and homophily because in adoptive homes, unrelated family members are presumably no more genetically or phenotypically similar than are strangers. Indeed, research has shown that selective placement of adoptees into homes with parents of similar personality does not occur; correlations in personality between adoptive and biological parents were negligible in one of the few studies that could assess them (Plomin, Corley, Caspi, Fulker, & DeFries, 1998). Thus, we are able to observe the effect of an unrelated, unselected individual's happiness on that of an individual who resides in the same home. Because we estimate the impact of the total shared environment, what we are really examining is the combined impact of the contagion effect and other contextual variables that may contribute to similarity. On one hand, this means that we cannot isolate the contagion effect to estimate its size; on the other hand, if shared environment is not an important influence on happiness, we can rule out the possibility of a contagion effect.

Furthermore, the importance of physical proximity was highly emphasized in the results of Fowler and Christakis' study (e.g., while friends who lived within a mile of the ego significantly affected the ego's happiness, friends who lived more than a mile away had no effect). This suggests that the contagion effect arises through the frequency of interaction (presumably people who live near each other interact more often than those who do not). All of the offspring included in our sample were still living at home with their siblings and parents at first assessment (and most at time 2), so this degree of physical proximity should result in the largest contagion effects and therefore constitute the best test of the contagion hypothesis.

Additionally, the adoption design presents an opportunity to confirm previous etiological findings with an alternative research method. As previously discussed, past research has found no significant variance accounted for by shared environmental effects; however, these findings were based on twin studies which have some important limitations. Thus, an alternative test of shared environmental effects is needed. If shared environmental effects on happiness are found in analysis of adoptive family data, then behavioral genetic research may not be completely out of line with Fowler and Christakis' findings.

We addressed the following questions:

Question #1 – To what extent is the happiness of immediate family members (mother, father, siblings) related to target happiness when family members are not genetically related?

Because the contagion effect from only one individual to another may be much smaller than the compounded effect of many individuals, we asked the second question,

Question #2 – Is overall level of family happiness related to target happiness when family members are not genetically related?

Because it is conceivable for family members' current level of happiness to be dissimilar and yet affect change in happiness over time, we asked a third question,

Question #3 – Is family members' level of happiness associated with change in target happiness when family members are not genetically related?

Finally, because social contagion can be considered a shared environmental influence and because an adoption design is a direct measure of c^2 , we asked a fourth question,

Question #4--Is the shared environmental estimate of happiness variance different from zero?

Method

Participants

Participants were from the Sibling Interaction and Behavior Study (SIBS; McGue et al., 2007). At least one parent and two adolescent offspring (M age = 14.92 years, SD = 1.56) comprised each family, and study eligibility was limited to those families living within driving distance of the research lab and having adolescent offspring within five years of age of each other. In addition, adolescents in adoptive families were required to have been placed for adoption before reaching age 2 years (M = 4.7 months, SD = 3.4 months). Records of large adoption agencies and state birth records were used to identify a representative sample of adoptive and nonadoptive (biological) families, respectively. Once identified, a parent was interviewed to establish family eligibility, and most eligible families agreed to participate in the study (63% of the adoptive families and 57% of the biological families). Moreover, non-participating but eligible families differed minimally from participating families in socioeconomic status and parent ratings of their children's behavioral problems (McGue et al., 2007). The final sample consisted of 615 families, including 284 adoptive (both children adopted), 208 nonadoptive (both children biologically

related to parents), and 123 mixed (one adoptive and one biological child) families. Details concerning recruitment of the SIBS sample can be found in McGue et al. (2007).

Measures

Happiness was measured as the well-being scale from a 198-item version of the MPQ (Tellegen & Waller, 2008) in participants 16 and older and with the 133-item Personality Booklet—Youth, Abbreviated (PBYA) (both developed specifically for this lab) in participants younger than 16. The well-being scale is comprised of 18 items scored from 1 = *Definitely true* to 4 = *Definitely False*, and most items are reverse-scored so that higher scores represent higher levels of happiness; items do not differ between the two MPQ versions. In the current sample, the coefficient alpha for the well-being scale was 0.89. Well-being was assessed twice across a 3-year interval ($M = 40.27$ months, $SD = 5.35$ months) allowing analysis of change over time; however, parent personality was assessed only once. Of the adolescents participating at intake, 83% returned and completed the well-being measure at follow-up. The intake (assessment 1) well-being scores of those who did not complete the well-being measure did not differ significantly from the well-being scores of those who did return.

Analysis

Raw scores were adjusted for age and sex effects. Mx statistical software was used for correlational and biometrical modeling analyses. Well-being correlations at intake among targets, their parents, and the family average (average of all members excluding the target) were calculated in adoptive, biological, and mixed families. We used Mx to calculate correlations because it allows us to set offspring-parent correlations to be equal for both siblings, thus using all relevant data without double-entry. Correlations were calculated between parent intake data and offspring data from follow-up and change over time (residuals from a regression of time 2 scores on time 1 scores). Biometrical analysis was conducted on intake data; we compared the fit of the ACE model to one where the C parameter has been constrained to zero.

Results

Table 1 presents descriptive statistics of target (offspring) well-being scores by sex and family type, and Table 2 presents descriptive statistics of parent well-being scores by sex and family type. There was no evidence of sex differences in well-being of targets or parents. Also, there was no evidence of important differences in means or variances of well-being among the family types, suggesting that restriction of range in adoptive families is not a problem for this phenotype.

Correlations of well-being scores at intake, follow-up, and change over time are presented in Table 3. All correlations in the adoptive families were close to zero and not significant while all correlations in the biological families were small but significant at intake. At follow-up, adoptive correlations remained near zero, and biological correlations remained small but significant, with the exception of the target-dad relationship. Finally, correlations with well-

being change were close to zero and not significant for both family types, with exception of target-sib and target-mom correlations in biological families.

Variance component estimates in the full ACE model were .39, .02, and .60 for a^2 , c^2 and e^2 ($\chi^2(1204) = 8484.28$, $AIC = 6076.28$). Model fit was not significantly reduced in the more parsimonious AE model ($\chi^2(1) = .12$, $p = .73$), and there is evidence for a moderate genetic influence on well-being ($a^2 = .42$, $e^2 = .58$).

Discussion

The present study tested the contagion hypothesis of happiness using a novel adoptive family study design. If happiness were contagious, then family members would have similar levels of happiness even when they are not genetically related. However, we found no support for the contagion hypothesis over a 3-year period. The happiness of family members, either singly or in aggregate, was unrelated to target happiness. Our finding of moderate familial correlations among genetically related family members, which is consistent with a larger behavioral genetic literature based on studies of twins (e.g., Finkel and McGue, 1997), suggests that our failure to observe resemblance in adoptive families does not owe to some inherent limitation of our measure. Both our consistent observation that target happiness is unrelated to the happiness of individuals with whom he or she has lived for an extended period of time but who are not genetically related or actively selected (i.e., homophily) and the fact that biometrical modeling produced a c^2 estimate that was essentially zero presents challenges to the contagion hypothesis.

One limitation of this study is that sibling pairs may be comprised of either same-sex or opposite-sex siblings. While we observed no sex difference in well-being scores, it is still possible that same-sex siblings are more similar than opposite-sex siblings. It may be the case that contagion is (or other shared environmental factors are) important in same-sex individuals but not in opposite-sex individuals. To address this issue, we split the sample into same-sex pairs and opposite-sex pairs and compared correlations (not reported). There was no indication of a contagion effect for either type of pair.

Another possible limitation is that the spousal correlations we report look different for adoptive and biological families, which suggests fundamental differences between these family types; however, these correlations were not significantly different. Even so, the correlations are lower than those usually observed in research on SWB (Bookwala & Schulz, 1996; Dyrenforth, Kashy, Donnellan, & Lucas, 2010). They are, however, well within the range reported in studies using the MPQ well-being scale (e.g., Humbad et al., 2010). This may be due to the fact that, although the MPQ well-being scale includes items reflecting both affect and cognitive appraisal, it clearly weights affective items more heavily. The previous studies included measures that weight these components differently; for example, Dyrenforth et al. (2010) used a 1-item measure of life satisfaction and as such included no aspect of affect. Bookwala and Schulz (1996) used a number of measures but the highest spousal correlation (.34) corresponded to a cognitive appraisal while the lowest correlation (.26) corresponded to a measure of affect. Thus, the smaller spousal correlations we report may partially be an effect of the composition of happiness measures.

Discrepancy between our findings and those of Fowler and Christakis (2008) may be accounted for by differences in study design and sample. Whereas we used a straightforward behavioral genetic design, they performed social network research, critiques of which are now emerging in the literature (Lyons, 2011; Shalizi & Thomas, 2011). Our sample included adolescents and adults whereas the previous study included all adults. However, if the contagion hypothesis is correct, it may actually be *more* likely that we would see its impact within parent-offspring and sibling relationships because they live in the same home and can exhibit more influence on each other.

Also, our sample included adoptees, which may not be ideal test cases for the contagion hypothesis. An assumption we must make is that families with adopted children are representative of all families. While there is evidence of range restriction in such homes for socioeconomic status (SES) and parent disinhibitory psychopathology (McGue et al., 2007), it was found that selection on these variables did not attenuate the regression coefficients of delinquency, drug use, or IQ on SES and parent disinhibitory psychopathology. This provides some evidence that shared environmental effects are not underestimated in the adoption design; however, it is not clear whether range restriction of these environmental factors influences their impact on well-being. Other researchers found that family interactions differ between adoptive and non-adoptive families, and they suggested that such interactions may influence child adjustment (Rueter, Keyes, Iacono, & McGue, 2009). In this study, both parents and adolescents in adoptive families reported greater parent-child conflict than did members of non-adoptive families. However, in the present study, adoptees and non-adoptees did not significantly differ in well-being at any time. If greater conflict occurs within these families, it doesn't seem to be affecting reports of happiness, early or late in adolescence.

Finally, whereas Fowler and Christakis (2008) used items from the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977), we used the MPQ well-being scale as our measure of happiness. Although the CES-D was developed to examine depression, these measures are similar in content. In the previous study only a subset of items were chosen to represent happiness, and they included: "I felt hopeful about the future," "I was happy," "I enjoyed life," and "I felt that I was just as good as other people." There are four items on the well-being scale that are nearly identical to the CES-D items: "My future looks bright to me," "Basically I am a happy person," "Most days I have moments of real fun or joy," and "Without being conceited, I feel pretty good about myself."

The main difference between the CES-D and MPQ is that the former asks about experiences and feelings *in the last week* and the MPQ asks about those same experiences and feelings *in general*. This is an important difference, but it may not necessitate different interpretations of each study's results. For one thing, pairs in the previous study could have been assessed up to a year apart in time. It is thus unclear whether pair similarity actually reflects influence on state-level happiness rather than trait happiness. Additionally, in a different sample (described in McGue, Hirsch, & Lykken, 1993), researchers observed a correlation of $-.51$ between CES-D and MPQ well-being scores. This is comparable to the MPQ 3-year retest correlation of $.41$ reported in the current study and both the CES-D 30-day ($.67$) and the 1-

year (.32–.49) retest correlations reported previously (Radloff, 1977); scores across assessments are just as similar as those on the same assessment over a short period of time.

Other social network research has raised doubts about the validity of the contagion hypothesis. In their analysis, Cohen-Cole and Fletcher (2008a) identified what appear to be contagion effects on improbable traits including acne, headaches and height. When they incorporated school-level fixed effects into their regressions, the apparent contagion effects were reduced by over 50%. Aral et al. (2009) applied a dynamic matched sample framework to adoption of a mobile service application in a very large network of instant messaging users. They showed that standard approaches to social network analysis substantially overestimated social contagion effects (by 300–700%) and that homophily explained over 50% of the similarity in mobile service adoption. The complexities of differentiating network effects from homophily underscore the need for alternative approaches to testing for social contagion effects. While the finding that shared environment does not significantly influence happiness may be expected given previous behavioral genetic research, the novel use of an adoption study addressed concerns surrounding the impact of genetic confounding and homophily on social network research as well as confirmed previous null findings of a shared environmental effect. Our findings provide support for recent critiques of social network modeling.

In our behavior genetic analyses, we found significant similarity of well-being scores among biological but not among adopted relatives and thus no evidence for shared environmental influences on happiness. Although shared environmental effects refer to those that make siblings more similar while a contagion effect refers to influence of one person on another regardless of relationship, families living together provide the strongest test of contagion as they are in very close physical proximity. Since adopted relatives are neither genetically related nor selected, our results suggest that the effects of genetic confounding and homophily may have been underestimated in previous social network research on happiness.

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Figure 1.

Path diagram of the ACE model in the adoption design. Note. S_1 = sibling 1, S_2 = sibling 2, A = additive genetic effects, C = shared environmental effects, E = non-shared environmental effects, bio = biologically related siblings, adopt = unrelated siblings.

Table 1

MPQ well-being (WB) scores in offspring by sex and family type.

	Adoptive Families			Biological Families			Mixed Families		
	Mean (SD)	N		Mean (SD)	N		Mean (SD)	N	
Males									
Intake	56.64 (7.55)	234		56.21 (7.43)	200		55.52 (7.53)	113	
Follow-Up	55.24 (8.00)	197		56.61 (7.86)	177		55.38 (7.69)	85	
Change	-0.08 (1.02)	190		0.09 (0.96)	175		0.02 (0.93)	85	
Females									
Intake	56.82 (8.61)	326		56.84 (8.92)	211		56.73 (8.42)	130	
Follow-Up	55.04 (8.51)	277		56.77 (7.76)	180		56.92 (7.40)	100	
Change	-0.12 (1.08)	277		0.09 (0.98)	177		0.15 (0.83)	97	

Note: Change = WB scores at follow-up controlling for WB scores at intake.

Table 2

MPQ well-being (WB) scores in parents by sex and family type.

	Adoptive Families		Biological Families		Mixed Families	
	Mean (SD)	N	Mean (SD)	N	Mean (SD)	N
Moms	56.82 (7.01)	284	57.30 (8.22)	205	56.61 (7.49)	121
Dads	54.57 (8.31)	258	56.45 (7.52)	186	55.74 (7.47)	111

Note: Means are from intake data.

Table 3
Correlation coefficients (confidence intervals) of target and family member well-being scores.

	Adoptive Families		Biological Families		
	<i>r</i> (CI)	N	<i>r</i> (CI)	N	
Target – Sib	Intake	0.05 (–0.08 – 0.17)	399	0.22 (0.08 – 0.34)	203
	Follow-Up	–0.01 (–0.14 – 0.12)	309	0.25 (0.10 – 0.38)	170
	Change	0.06 (–0.08 – 0.13)	304	0.18 (0.04 – 0.28)	166
Target – Mom	Intake	0.05 (–0.02 – 0.12)	680	0.16 (0.08 – 0.24)	528
	Follow-Up	0.02 (–0.06 – 0.10)	568	0.16 (0.07 – 0.26)	449
	Change	0.02 (–0.09 – 0.09)	560	0.11 (0.01 – 0.19)	444
Target – Dad	Intake	0.05 (–0.04 – 0.13)	629	0.13 (0.02 – 0.24)	469
	Follow-Up	0.03 (–0.05 – 0.11)	568	0.11 (0.00 – 0.21)	441
	Change	0.00 (–0.12 – 0.08)	560	0.07 (–0.04 – 0.16)	437
Target – Family Average					
	Intake	0.08 (–0.01 – 0.17)	681	0.24 (0.15 – 0.33)	532
	Follow-Up	0.03 (–0.05 – 0.11)	568	0.22 (0.13 – 0.31)	446
	Change	0.02 (–0.08 – 0.09)	560	0.08 (–0.02 – 0.15)	442
Mom – Dad					
	Intake	0.06 (–0.06 – 0.17)	369	0.19 (0.05 – 0.32)	284

Notes: Spousal correlations were not significantly different across family type. Intake = correlations of target and family member well-being scores at intake. Follow-up = correlations of target well-being at follow-up with family member well-being at intake. Change over time = correlations of target follow-up scores controlling for intake scores with family member well-being at intake. Bold text indicates significance.