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A Systematic Review of Personality Trait Change Through Intervention

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The current meta-analysis investigated the extent to which personality traits changed as a result of intervention, with the primary focus on clinical interventions. We identified 207 studies that had tracked changes in measures of personality traits during interventions, including true experiments and prepost change designs. Interventions were associated with marked changes in personality trait measures over an average time of 24 weeks (e.g., $d = .37$). Additional analyses showed that the increases replicated across experimental and nonexperimental designs, for nonclinical interventions, and persisted in longitudinal follow-ups of samples beyond the course of intervention. Emotional stability was the primary trait domain showing changes as a result of therapy, followed by extraversion. The type of therapy employed was not strongly associated with the amount of change in personality traits. Patients presenting with anxiety disorders changed the most, and patients being treated for substance use changed the least. The relevance of the results for theory and social policy are discussed.

Keywords: personality trait change, clinical psychology, personality change, intervention

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A significant body of evidence has accumulated to show that personality traits predict meaningful life outcomes, such as economic well-being, relationship success, health, and longevity (see, e.g., Borghans, Duckworth, Heckman, & Ter Weel, 2008; Ozer & Benet-Martinez, 2006; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007 for reviews). While important findings in their own right, the links between personality traits and important life outcomes invites the question of whether personality is fate. This question is really a question of whether personality traits change. If personality traits do change, then personality traits would not

necessarily be fate because people could develop in ways that might positively impact their future.

The answer to the question of whether personality traits change is yes, and not just early in the life span. Indeed, multiple studies have now provided evidence that personality traits change in young adulthood (e.g., Neyer & Asendorpf, 2001; Robins, Fraley, Roberts, & Trzesniewski, 2001), middle age (e.g., Hill, Turiano, Mroczek, & Roberts, 2012), and even old age (e.g., Möttus, Johnson, & Deary, 2012; Mroczek & Spiro, 2003; Small, Hertzog, Hultsch, & Dixon, 2003). Moreover, meta-analytic work has suggested that people become more confident, agreeable, conscientious, and emotionally stable with age (Roberts, Walton, & Viechtbauer, 2006). That said, the rate of personality trait change demonstrated in all of these studies is rather modest. Therefore, normative changes in personality traits may provide little comfort for those starting life low on key traits, such as conscientiousness. By the time these individuals might catch up with their peers, they may be too old to take advantage of the opportunities afforded by growth.

Nonetheless, the fact that personality traits do change throughout the life course invites a reasonable question: Can personality traits be changed through intervention? Moreover, if personality traits can be changed, how fast does the change occur and does the change remain once it has occurred? Within the field of personality psychology, there is a distinct lack of research investigating techniques to change personality traits, thus the question would appear

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to have not been systematically addressed. However, in clinical psychology there is a long-standing, if relatively unappreciated, current of research that incorporates personality trait measures to test the effectiveness of various forms of therapeutic interventions on clinical outcomes, such as anxiety and depression. In almost all cases, the focus of these studies was not to change personality traits (cf. Tang et al., 2009), but simply to track the effectiveness of therapy while using a wide variety of measures. The effort to be comprehensive in assessing potential outcomes has resulted in a surprisingly large corpus of studies that include change in personality trait measures.

The overarching question of whether measures of personality traits can and do change in therapy is not only an interesting question, but also one that is highly relevant to the theory and utility of personality traits. Theoretically and conceptually, the topic of personality trait development was not the focus of personality research for many years because the field was dominated by extreme positions that characterized personality either as essentially fixed (an essentialist position) or as so inconsistent that it was inconsequential (contextualist; see Roberts & Caspi, 2001). Accordingly, dominant theoretical models of personality provide little theoretical help to explain the fact that personality traits exhibit both continuity and change. Recently, however, theoretical frameworks have been developed that treat personality traits as developmental constructs (Durbin & Hicks, 2014; Roberts & Jackson, 2008). The central perspective within the more developmental take on personality traits is that they combine both continuity and change and typically change slowly and incrementally (Roberts, 2006) with meaningful change taking course over the span of years rather than weeks. The existence of personality trait change that occurs within the time-line of a typical therapeutic context would challenge these assumptions that have emerged largely through the examination of long-term, passive longitudinal studies.

The current article leverages the neglected, yet potentially important body of research in clinical psychology. In the following sections we first review clinical psychology perspectives on the changeability of personality traits in the context of therapeutic interventions. Second, we present a meta-analysis of interventions that assessed personality trait change to quantify whether and how much personality traits changed as a result of therapy. We also examine critical issues, such as whether the change endured once people left the therapeutic setting. Finally, we discuss how the findings are relevant to theoretical and applied issues surrounding the assessment and use of personality traits.

Can Personality Traits be Changed Through Clinical Intervention?

There is a long-standing literature within clinical psychology that has directly or indirectly addressed the question of whether typical clinical interventions, such as cognitive-behavioral therapy or pharmacological treatments, can and do change personality traits (e.g., Bagby, Joffe, Parker, Kalembo, & Harkness, 1995; Borkovec et al., 1987). More recently, several authors have considered more directly the topic of personality trait change in the context of therapeutic interventions (e.g., Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2014; Clark et al., 2003; Quilty et al., 2008; Soskin Carl, Alpert, & Fava, 2012). Two opposing positions have emerged concerning the changes seen in personality traits during

therapy. The assumption underlying both positions is that any given personality trait measure captures both state and trait variance to some unknown degree. The first position, the *state-artifact position*, argues that any changes seen in personality trait measures that appear as the result of therapy can be attributed to the state-level variance in personality trait measures (Du, Bakish, Ravindran, & Hrdina, 2002; Gracious, 1999; Marchevsky, 1999). For example, an episode of depression would cause an inflection in personality trait scores because it would push down the state variance of a relevant trait-like emotional stability. Once the depressive state is lifted, one would expect trait scores to rise too. The increase in personality traits, such as emotional stability, would not be attributable to change in the trait component of the construct but the state component instead. Therefore, what looks like personality trait change (e.g., the decrease in emotional stability associated with a major depressive episode and the subsequent increase in the trait as the depression lifted) would only be temporary state changes that result from the fact that our trait measures are imperfect and capture both state and trait variance.

Conversely, the *cause-correction hypothesis* (Soskin et al., 2012) proposes that the changes demonstrated in psychological outcomes, such as depression, are the result of changes in the trait component and not the state component of personality. For example, a double-blind placebo control trial (Tang et al., 2009) showed that taking paroxetine (an antidepressant) resulted in both improvement in depression and increases in emotional stability. Most importantly, the changes in emotional stability were differentiated from changes in depression and appeared to be the mechanism through which lasting changes in depression were made, as they mediated the effect of antidepressants on depression and long-term relapse.

Other types of clinical and nonclinical intervention studies also demonstrate that personality traits are amenable to change across the life span. Training programs, in which participants learn some type of life skill, appear to be especially effective in changing personality traits. For example, a mindfulness intervention was associated with personality trait changes in conscientiousness, agreeableness, empathy, and emotional stability among medical residents (Krasner et al., 2009). Similarly, a social-skill training program for recovering substance abusers led to increases in agreeableness, conscientiousness, and emotional stability (Piedmont, 2001; see also Oei & Jackson, 1980). Moreover, a cognitive training intervention for older adults was also associated with changes in a personality trait. Across 16 weeks, older adults learned inductive reasoning skills and completed 10 hr a week of crossword and Sudoku puzzles. Compared with a control condition, the intervention increased participants' levels of openness to experience (Jackson et al., 2012).

The clinical perspectives on personality trait change in therapeutic settings help to frame the research questions that need to be addressed to test whether the changes in personality traits are the result of changes in the state or trait structure of the construct. First, it is critical to both the state-artifact and cause-correction positions to show that personality trait measures change as a result of therapy. Thus, the first question is whether there is an association between attending therapy and personality trait change. But it is not enough to show that personality traits change during therapy as this type of effect could simply be attributable to changes in states. For example, a large number of studies described

below relate the experience of a specific form of therapy to personality trait change with no comparison group or control group. These types of results support the idea that personality traits can and do change quickly, but not the inference that the change is caused by therapy and not the result of the drift in states. A more stringent test of whether personality trait change is the result of therapy can be derived from experimental studies in clinical psychology. Experimental studies allow some advantage over observational studies in this case, as the control groups in clinical studies in this particular data set are typically wait-list-control studies. These control groups may show naturalistic recovery that is most consistent with the state-artifact position. If experimental studies show no advantage of therapy over a wait-list control group, this would support the state-artifact position. But the experimental effect on personality trait change, if it exists, could still be interpreted as state-level change, as it is possible that short-term therapy is truly more effective at changing state variance than "time," the presumed ingredient in a wait-list condition. Therefore, we also examine evidence that changes that occurred in therapy were long-lasting. Temporary, as opposed to long-term, changes in trait measures would be strong support for the state-artifact position. If trait change persists over long periods of time, however, then this would be more consistent with therapy shifting enduring trait variance, rather than state variance.

Given that personality trait change is complex with several alternative perspectives, we took a holistic approach to surveying the literature. No one effect size estimate or empirical trend is sufficient to determine the plausibility of therapeutically induced trait change. Instead, we integrate and interpret multiple sources of data drawing on complementary strengths of observational and experimental studies.

The Current Study

In early meta-analytic reviews, moderate changes in personality trait measures were reported as a result of psychotherapy (Jorm, 1989; Smith, Glass, & Miller, 1980). Since these reviews, hundreds more studies have tracked personality traits and how they changed during therapy. These studies can be organized into several categories. Some studies were designed as true experiments with random assignment to either a control or an experimental group. Many other studies lacked a control group and simply assessed change that putatively arose through therapy in relevant psychological outcomes like depression, as well as in personality traits. Finally, another category of studies conducted long-term follow-ups of interventions to determine whether the changes lasted.

The majority of studies used in the present review did not focus explicitly on changing personality traits. For example, it was common for researchers to default to including both the state and the trait versions of anxiety and anger measures (e.g., Spielberger, Jacobs, Russell, & Crane, 1983) because the inventories that measure these constructs often came in these two forms. In other studies, researchers chose scales that seemed therapeutically relevant, such as the Inventory of Interpersonal Problems (Horowitz et al., 1988), without considering that the scales of this inventory are essentially measures of pathological forms of low extraversion and low agreeableness. In some of the older studies, however, it appears that it was not considered unusual to track changes in

personality traits as they were more often considered the focal point of intervention in the middle of the 20th century (Worchel & Byrne, 1964).

Given the number of studies that have tested some form of intervention and shown that these interventions are associated with changes in personality traits, we have followed up on, and greatly expanded, the analyses originally reported in Smith et al. (1980). We conducted a meta-analysis of clinical and nonclinical interventions on personality trait change and added several dimensions to our analyses that were not addressed in earlier reviews. The first goal of this meta-analysis was to test the extent to which personality traits could be changed, even in relatively short periods of time common in clinical interventions, such as 12 to 15 weeks. It is common for researchers to assume that personality traits do not change quickly (e.g., Roberts, 2006), but this position is often derived from examining long-term passive longitudinal studies. Research based on passive longitudinal studies is problematic for making any inference about the time course of personality trait change, because they typically fail to track personality traits often enough to know whether traits change quickly or gradually over time. Researchers conducting passive longitudinal studies typically wait years between assessments of personality traits under the assumption that change does not happen quickly (e.g., Roberts et al., 2006). However, by not assessing personality over shorter intervals, these studies provide limited evidence for how fast or slow personality trait change can occur. Change may happen gradually over the whole span of years, quickly at some point during the long period in which no measurements were taken, or some combination of these options varying across individual (e.g., those in therapy compared to those not in therapy).

In spite of this shortcoming, there are some findings from passive longitudinal studies that presage the possibility of personality trait change can happen quickly. While most people fail to change much on the majority of traits over long periods of time, such as 8 years, most people change substantially on one trait out of five over this type of time span (Roberts et al., 2001). Though no longitudinal study has tracked personality trait change using short time intervals between assessments (i.e., weeks or months) over long periods of time, it is possible that traits could have changed quickly at any time in the long spans that typify longitudinal research, or gradually over the same time period. Research on clinical interventions that result in personality trait change would provide a much-needed perspective on whether change in personality traits is achievable in a shorter period of time.

The second goal of this meta-analysis was to compare the state-artifact and cause-correction hypotheses to the best of our ability given the data. To this end, we structured the outcome data in several ways in order to highlight conditions that should provide evidence to support either perspective. First, we tested whether personality trait change was associated with clinical interventions, broadly construed. Second, we examined personality trait change in true experimental studies that contrasted change in experimental groups to change in control groups. In most clinical intervention studies, even control groups may show modest improvements in measures of psychopathology (Smith et al., 1980). The key question relevant to the state-artifact hypothesis is whether intervention groups change more than control groups. Presumably any changes that occur in a control group, which receives no treatment, reflect the natural relief from the temporary inflection in personality

caused by syndromes such as depression. Assuming that control groups in clinical settings, which are typically waitlist control groups, will improve without treatment, they provide an excellent comparison group for the state-artifact perspective. If both groups show the same amount of personality trait change, then there would be little reason to infer that the many nonexperimental studies showing personality trait change after therapeutic interventions are showing anything more than state change.

Also relevant to the state-artifact and cause-correction hypotheses are the results from experimental designs in nonclinical studies. The literature search resulted in the identification of a set of studies that were not clinical interventions per se; they either used a nonclinical intervention, a nonclinical group, or a combination of these two factors. Like the clinical studies, demonstrating that the intervention groups increased more than control provides a good test for drawing the causal inference that the intervention can lead to personality trait change rather than state change. But possibly more important is the pattern of change in the control groups for the nonclinical studies. Unlike the clinical studies, there would be no reason to expect significant changes in these control groups, as they would not have a disproportionate number of individuals with preexisting forms of psychopathology. On the other hand, these control groups would address other artifactual reasons for slight changes over time, such as testing effects from repeated administration of the personality inventory. A final way to distinguish the state-artifact and cause-correction positions is to examine how long the changes persist. If therapy only affects change in states, then we would expect the putative effect of therapy to wear off with time and for the personality trait scores to return to their baselines. In order to test these effects, we compiled the subset of studies that performed follow-ups of the intervention samples after the termination of therapy.

In summary, this meta-analysis was designed to address a number of enduring questions about personality trait change. This study is also positioned to address many other central questions about interventions and personality change. First, we tracked changes across the Big Five trait categories (extraversion, agreeableness, conscientiousness, neuroticism, and openness to experience) to see in what domain(s) people show the most change. Given the overriding importance of neuroticism to most clinical issues, we expected to find the largest effects for neuroticism in studies of clinical orientation (Kotov et al., 2010). We also expected changes to occur on extraversion as it is strongly related to positive affect, and thus various forms of psychopathology, such as depression (e.g., [Clark & Watson, 1991](#); [Watson & Naragon-Gainey, 2014](#)). Second, we sought to determine whether the type of therapeutic intervention, such as cognitive-behavioral therapy or supportive therapy, made any difference to the outcome. It is common in meta-analytic studies to find little difference between various forms of psychotherapy on clinical outcomes, such as depression or anxiety ([Luborsky et al., 2002](#)), but the question remains whether the same is true for personality traits as outcomes. If the findings for typical clinical outcomes, such as depression, hold for personality traits, we should expect little differential treatment effects for various forms of therapy. Third, we examined whether the type of disorder being treated moderated the amount of change found over time. Specific psychological disorders, such as eating disorders, are known to be very difficult to treat ([Kaplan & Garfinkel, 1999](#)). We sought to determine whether several

additional factors, such as the length of the intervention, gender composition of the sample, and year of publication, moderated the amount of personality trait change.

Method

Literature Search Procedures

The data sources for the current meta-analysis were journal articles as well as unpublished dissertations and theses. Multiple steps were taken to identify usable studies. First, we went through the reference list of Smith et al.'s (1980) seminal meta-analytic work on the effect of psychotherapy and all relevant reviews to identify studies that examined the change of personality traits as a result of interventions. Next, we conducted an inclusive search in the American Psychological Association's PsycINFO database (1887–2012), Google Scholar, Web of Science, and the ProQuest Dissertations and Theses database for any combination of the key words *psychotherapy and personality change*, *selective serotonin reuptake inhibitors and personality*, *SSRIs and personality*, *personality* and a specific SSRI (*fluoxetine*, *fluvoxamine*, *citalopram*, *duloxetine*, *paroxetine*, *sertraline*, *escitalopram*, *venlafaxine*), *treatment outcomes and personality change*. We inspected the citations from each usable study and review in our database as well as studies that referred to each study for additional qualified studies and continued this iterative process until no new study could be found. We also contacted researchers who had authored multiple studies that tracked personality trait change in clinical intervention studies to acquire any studies we had missed and any unpublished studies. Finally, we circulated a request for relevant studies to several list serves including the Society for Personality and Social Psychology, Association for Research in Personality, Division 12 of APA, and the Society for the Research on Psychopathology asking for unpublished research on personality trait change in intervention research.

Studies were evaluated for inclusion in the current meta-analysis on the basis of the following criteria. First, the study needed to measure one or more personality trait variables. We chose a very specific operationalization of personality traits to make sure the focus was on measures typically thought of as traits. Specifically, we only included studies if the ratings of the items in the measures used were (a) global; (b) general in terms of overall functioning; and (c) focused on implicitly or explicitly enduring patterns of thoughts, feelings, and behaviors. A prototypical measure that fulfills these criteria asks people to rate general items about personality (e.g., I'm a talkative person) using rating scales intended to capture general patterns (e.g., How characteristic or uncharacteristic of you is this statement?). With these criteria applied, studies that examined state (e.g., affects, state anxiety) rather than trait variables, behavioral frequencies (e.g., frequency of aggressive behaviors), and social adjustment or maladjustment (e.g., marital satisfaction, relationship conflict) were excluded from the meta-analysis. Second, the study was considered for inclusion if it employed a prepost test design, that is, an experimental design such that personality trait change from pre- to posttest or the difference between control and treatment groups was reported. Third, effect sizes of personality trait change must have been reported in the study or able to be calculated from the reported results. Studies that reported incomplete results for calculating

effect sizes (e.g., trait scores for only one group at one time point, mean trait scores without any standard deviations) were excluded from our analysis.

After applying these inclusion criteria, our searches resulted in 207 studies (35 experimental studies) with a total of 357 samples (many studies included separate results for multiple samples), published or completed between 1959 and 2013. The overall sample of our meta-analytic database included a total of 20,024 participants with an overall average of 63.41% female. The mean age of the samples ranged from 19 to 73 and averaged 36.04 years. Nearly one quarter of the samples ($k = 77$) followed up the participants longitudinally to monitor the long-term effect of the interventions with the longest follow-up occurring 16 years after therapy ended. Although we focused on clinical interventions, we found a large number of “nonclinical” studies that either tested (a) a nonclinical intervention (e.g., efforts to improve cognitive functioning); or (b) a clinical intervention, such as administration of antidepressants, applied to a healthy sample of adults. We examined these studies separately. A full list of these studies is provided in supplementary Table 1. The data files used to estimate the results and supplementary tables can be found at <https://osf.io/4tys6/>.

Coding of Study Variables

At the onset of the meta-analysis, we developed a detailed codebook for recording a range of characteristics of the studies (see online appendixes), the samples, the interventions used in the studies, the personality variables measured, and the effect sizes for personality change. Each sample from every usable study was coded for key study variables, including study design (experimental design, prepost test design), type of treatment (cognitive-behavioral, supportive, psychoanalytic, pharmacological, hospitalization, and mixed), duration of intervention (in weeks), time interval for the follow-up in longitudinal studies (in weeks), sample mean age (in years), percentage of females in the sample, clinical and nonclinical sample type, type of psychological disorders/maladjustment presented (depression, anxiety, personality disorder, eating disorder, alcohol or drug abuse, and mixed), personality inventory used, and Big Five personality trait assessed. Several researchers coded the studies and agreement in coding was checked several times. Agreement was good for the coding of type of treatment ($\kappa = .79$), and for type of presenting psychological disorders/maladjustment ($\kappa = .96$). Agreement for computed effect sizes for each study was also quite high ($r = .98$). Any disagreement in the ratings was addressed by discussion between the authors.

Personality scales that did not directly assess the Big Five personality traits (e.g., the 18 scales in the California Personality Inventory) were sorted into corresponding Big Five categories, if any, based on multiple criteria: (a) information from studies that documented the development of the personality scales, (b) studies that examined the correlations among these personality scales and established Big Five measures, and (c) studies that used these personality scales in our database. When a personality scale was determined to be a combination of several Big Five trait dimensions, we coded the effect size as “blended” to reflect this determination. This classification most often occurred for clinically oriented traits (e.g., hysteria, assaultiveness), evaluative traits (e.g.,

mistrust, socialization), or higher-order traits (e.g., well-being, harm avoidance). In the prepost dataset, effect sizes marked as blended were most frequently associated with neuroticism ($n = 248$) and extraversion ($n = 208$), with substantially fewer observations associated with agreeableness ($n = 89$), conscientiousness ($n = 139$), and openness to experience ($n = 24$). A total of 606 observations were labeled blended. Three of the authors independently completed this process. Disagreement was resolved by discussion. A complete list of coding classifications for study variables is presented in supplemental Table 2.

Analytical Procedures

For the present analysis, we calculated effect sizes for personality trait change using Cohen’s d (Cohen, 1988). For studies that used prepost test design, effect sizes are computed as the difference between post- and pretest scores divided by the pretest standard deviation of these scores. For experimental studies, the effect sizes reflect postintervention difference between the treatment group and the control group divided by their pooled standard deviation. We reversed the sign of the effect sizes for change in negative personality traits to ensure that the effect sizes were always *positive* when patients improved after intervention (i.e., score change on a measure of emotional stability was in the positive direction) and were *negative* when patients exhibited undesirable change after intervention (i.e., score change on a measure of emotional stability was in the negative direction). Effect sizes reported in metrics other than Cohen’s d (e.g., correlation coefficient r , student t statistic, one-way F statistic) were converted to Cohen’s d before being analyzed.

A total of 2,144 effect sizes were obtained from the usable studies in our meta-analytic database. In most cases, multiple effect sizes were available for each sample because several personality scales (e.g., all five scales of the NEO Five Factor Inventory) were used to assess the participants. For the within-subject, prepost change scores, the variance estimates were based on Becker (1988, Equation 13). Because this equation requires knowledge of the test–retest correlation, which was almost never reported, we assumed an average test–retest of $r = .5$ and tested the robustness of the effect by systematically varying the test–retest correlations from .25 to .75.¹ We also calculated the 95% confidence intervals of our point estimates. A confidence interval not including zero means that the change of personality was significant at $\alpha = .05$. We also separated the experimental effect sizes from the prepost effect sizes, as these two types of studies are only directly comparable under restrictive assumptions (Morris & DeShon, 2002).

Our meta-analytic search produced a large body of literature with evidence concerning personality change in response to therapy. We extracted the maximum amount of information from each article, resulting in a number of dependencies within the dataset. For example, most articles reported personality change across a number of dimensions, and some articles reported estimates of change across different waves of longitudinal follow-ups to the sample. Each effect size is informative, particularly for testing

¹ We found no notable differences in our results using alternative estimated average test–retest stabilities so report only the estimates for $r = .50$.

moderator effects. To obtain robust effect sizes and standard errors, we applied two techniques. First, we used the COMPLEX survey option in *Mplus* 7.31 to correct standard errors for the nonindependence of observations derived from the same sample (Muthén & Muthén, 1985–2015). Second, we weighted the observations by the inverse of the number of observations derived from a given sample as well as the inverse of the sampling variance associated with the observation. These approaches allow the use of the full dataset to estimate accurate effect sizes and standard errors.

We used the general paradigm laid out by Cheung (2008) for random-effects and mixed-effects meta-analysis using structural equation modeling. Random-effects meta-analysis is generally considered the more robust and conservative meta-analytic approach compared with fixed-effects meta-analysis, which makes the assumption that all effect sizes are drawn from the same population. Given the tremendous diversity of studies included in the current meta-analysis, such an assumption is unlikely to hold. Using random-effects meta-analysis, between-study variance in effect sizes is estimated (denoted τ in the results). Mixed-effects meta-analysis attempts to explain such between-study variance by incorporating study-level moderators, such as Big Five dimension, intervention type, or presenting problem. Variance explained (i.e., R^2) by the moderators can be calculated as the ratio of between-study variance when moderators are included compared to when moderators are not included (e.g., .3 variance with moderators compared with .6 without implies the moderators explain 50% of the between-study differences). More broadly, our metaregression approach allows for the testing of moderators separately while simultaneously taking into account the nested structure of the data to generate robust effect size estimates, regression coefficients for the moderators, and standard errors.

Small study effects (i.e., statistical noise and publication bias) pose special problems for meta-analysis. Unfortunately, a good tool to adjust for various small study effects does not exist, and experts disagree about the circumstances under which adjustment for small study effects is appropriate. Techniques commonly used in psychological research, such as trim-and-fill (Duval & Tweedie, 2000) or the failsafe N (Rosenthal, 1979), have been thoroughly criticized (Moreno et al., 2009; Peters et al., 2007; Sutton, 2009). Others have argued for regression-based tests where precision (i.e., standard errors) is used to predict effect sizes (Egger, Davey Smith, Schneider, & Minder, 1997). More recently, some have argued that the intercept of such a model, where the implied standard error is zero, indicates the effect size for the theoretical perfect study (Stanley, 2008; Stanley & Doucouliagos, 2014). Again, such tests have drawn support and criticism (Peters et al., 2010; Sterne et al., 2011). Most critically, Ioannidis (2008) argued that when effect sizes are even moderately heterogeneous, regression-based tests are “inappropriate, meaningless, or both” (p. 954). Effect sizes in the current study are heterogeneous, drawn from different populations, disorders, and treatments. This result is demonstrated empirically below. Recently, the performance of various bias-correction strategies have been put to the test under more realistic circumstances with underwhelming results (e.g., Reed, Florax, & Poot, 2015; Inzlicht, Gervais, & Berkman, 2015). The primary recommendation of this line of work is that some correction is better than none, but no one technique is unam-

biguously better than others. Trustworthy effects should not be sensitive to the choice of correction.

We employ two techniques to provide potentially more conservative estimates of personality trait change, while acknowledging that such tests are incapable of providing exact metrics for personality trait change. First, we extend our random-effects meta-analysis to include the squared standard error as a moderator (PEESE model, following Stanley & Doucouliagos, 2014). This approach adjusts for the potential for small studies to report large effect sizes by interpreting the intercept of the model. We integrate the predictor of study precision into our larger metaregression framework in order to maintain consistency across analysis. Second, we complement the strictly statistical metaregression approach with a visual inspection of funnel plots. As can be seen in Figure S1, funnel plot asymmetry occurs for prepost effect sizes associated with standard errors greater than roughly .2. These relatively small studies tend to have substantially larger effect sizes. Therefore, we ran all models excluding effect sizes with standard errors greater than .2 (302 observations), leaving 83.71% of the original dataset. For comparison, the trim-and-fill approach indicated that there were eight effect sizes missing from the funnel plot, meaning our visual correction should be more conservative. Because the prepost dataset was so large, we were also interested in whether results held when only high precision studies were included. To test this question, we excluded effect sizes with standard errors greater than .1 (1,310 observations), leaving 29.34% of the original dataset. For comparison, Stanley, Jarrell, and Doucouliagos (2010) suggested analyzing only the studies in the top tenth percentile of precision. Our criterion is not quite as extreme, but tends toward that direction. The experimental dataset was much smaller, and as can be seen in Figure S2, visual evidence for small study effects was less definitive. As a comparable test, we excluded effect sizes with standard errors greater than .4 (53 observations), leaving 76.86% of the original sample. In this case, the trim-and-fill approach indicated that there were two missing effect sizes. We did not test more stringent cutoffs because of the smaller pool of observations. If the levels of personality trait change identified in the meta-analysis are meaningful, the effects should be robust across a variety of analytic choices.

Results

Descriptive Statistics

Supplementary Table 1 lists all 207 studies and the basic information about each study, including sample size, measures used, clinical versus nonclinical, type of therapy employed, outcomes, and how they were coded. The majority of studies were clinical interventions intended to treat some form of psychopathology. The most common disorder treated was depression, followed by anxiety and eating disorders. Nineteen studies examined change in personality traits in nonclinical samples. Table 1 provides descriptive statistics for the accumulated data. The average duration of therapy was 24 weeks (mode and median were 12 and 13 weeks, respectively), indicating that most studies tracked changes over a 3-to-6 month period,

Table 1
Descriptive Statistics of Meta-Analytic Database (K = 207)

Average publication year	2001 (<i>SD</i> = 10.57)
Percentage of females	63.41 (<i>SD</i> = 20.10)
Mean age of sample	36.04 (<i>SD</i> = 7.47)
Duration of treatment (in weeks)	23.75 (<i>SD</i> = 35.96)
<i>K</i>	207
Total <i>N</i>	20,024
Longest follow-up	Average (month): 6.80
	Number of studies that tracked changes beyond termination of therapy: 77
Big Five categories	Extraversion: 91 studies (18.35%) Agreeableness: 86 studies (14.14%) Conscientiousness: 74 studies (16.19%) Emotional stability: 199 studies (51.97%) Openness: 44 studies (4.86%)
Intervention type	Blended: 67 studies (33%) Pharmacological: 81 (22.69%) Cognitive-behavioral: 65 (18.21%) Supportive/humanistic: 13 (3.64%) Psychoanalytic: 39 (10.92%) Hospitalization not otherwise specified: 8 (2.24%)
Presenting problem	Mixed: 115 (32.21%) Depression: 74 (20.73%) Anxiety: 77 (21.57%) Personality disorder: 43 (12.04%) Eating disorder: 26 (7.28%) Substance use: 23 (6.44%)
	Mixed: 67 (18.77%) Other: 4 (1.12%) None: 43 (12.04%)
Number of studies with experiments	35 out of 207

Note. The parenthetical percentages for the Big Five categories, intervention type, and presentation problem represent the percentage of effect sizes when the entire data set was analyzed. Blended traits were those coded as reflective of more than a single trait domain.

though a handful of studies tracked changes for a much longer time interval.

Overall Effects of Interventions on Personality Trait Change

We present results in two complementary ways. First, Table 2 presents effect size expectations based on metaregression models applied to prepost effect sizes. These effect sizes are derived from the model-implied metaregression equation, and the reported confidence intervals are useful for determining whether an effect size is different from zero. Second, supplementary tables report the regression parameters from our models. Because we use effects coding for all (noncontinuous) analyses, the regression parameters represent deviations from the midpoint. Conceptually, these parameters indicate whether effect sizes within a specific moderator class (e.g., emotional stability) differ from the expected average of general effect sizes within that moderator. In this context, the reported confidence intervals are useful for determining whether a specific moderator class differs from the expected average, rather than zero. Figure 1 summarizes our results and integrates these two frames of reference. The solid lines represent the midpoint effect size for that class of moderators. The dots represent expected effect sizes for a specific class of effect sizes. The confidence intervals can be used to infer whether the specific class of effect size differs from the expected midpoint. Importantly, all analyses

make use of the entire relevant dataset, allowing for direct comparisons of effect sizes while correcting for the nested structure of the data. Here, we give primary attention to the effect size estimates.

Focusing first on the average prepost effect size, personality tended to change moderately in the studies, with a *d* of .37, 95% CI [.33, .40]. This effect was robust across different corrections for small study effects (minimum *d* = .21, 95% CI [.17, .24]). However, there was considerable evidence of effect size heterogeneity, with estimates of systematic between-study variance ranging from .22 to .27 across models (see Table S3). Thus, without partitioning the data in any way, and when taking into account publication bias, clinical and nonclinical interventions appear to change personality traits between one fifth to one third of a standard deviation, with robust evidence for heterogeneity and the possibility that moderators affected those estimates.

Findings Relevant to the State-Artifact and Cause-Correction Hypotheses

We next turned to the particular study designs that provided information that could inform whether the evidence in totality supports the state-artifact or the cause-correction hypothesis about the effects of clinical interventions on personality trait change. Specifically, we focused on (a) true experimental designs, which contrast groups receiving treatment against those who do not; and

Table 2
Pre-Post Personality Change Effect Size Estimates

Moderator categories	Full Model ES [95% CI]	PEESE Model ES [95% CI]	Visual Model ES [95% CI]	High Power ES [95% CI]
Panel 1: Average effect size				
Average effect size	.37 [.33, .40]	.21 [.17, .24]	.31 [.28, .34]	.28 [.24, .32]
Panel 2: Clinical vs. Nonclinical vs. Control				
Clinical treatment	.38 [.34, .42]	.23 [.19, .26]	.33 [.29, .36]	.29 [.24, .33]
Clinical control	.24 [.14, .34]	0 [−.11, .11]	.19 [.10, .28]	.17 [.09, .25]
Nonclinical treatment	.33 [.19, .46]	.06 [−.05, .17]	.25 [.14, .35]	
Nonclinical control	−.03 [−.08, .03]	−.24 [−.35, −.14]	−.03 [−.09, .04]	
Comparison group	.15 [.09, .21]	−.06 [−.15, .02]	.16 [.09, .22]	
Panel 3: Follow-up interval				
Immediate	.34 [.31, .37]	.18 [.14, .22]	.29 [.26, .32]	.28 [.24, .33]
6 month	.48 [.36, .60]	.30 [.20, .41]	.39 [.27, .51]	.35 [.17, .53]
12 month	.46 [.32, .60]	.27 [.16, .37]	.36 [.24, .49]	.16 [.04, .27]
1 year +	.37 [.26, .47]	.24 [.15, .32]	.30 [.21, .40]	.25 [.13, .36]
Panel 4: Big Five				
Extraversion	.23 [.17, .29]	.14 [.08, .21]	.22 [.16, .27]	.22 [.14, .29]
Agreeableness	.15 [.11, .20]	.06 [.02, .11]	.14 [.10, .18]	.12 [.07, .17]
Conscientiousness	.19 [.14, .23]	.10 [.05, .15]	.18 [.13, .23]	.17 [.11, .24]
Emotional stability	.57 [.52, .62]	.39 [.33, .44]	.49 [.44, .54]	.49 [.42, .55]
Openness	.13 [.07, .18]	.04 [−.02, .09]	.12 [.07, .17]	.10 [.03, .17]
Blended	.27 [.23, .31]	.17 [.12, .21]	.24 [.21, .28]	.24 [.19, .29]
Panel 5: Intervention type				
Pharmacological	.31 [.26, .35]	.14 [.09, .19]	.25 [.21, .29]	.25 [.19, .31]
Cognitive behavioral	.46 [.37, .54]	.26 [.18, .34]	.36 [.28, .43]	.34 [.23, .45]
Supportive	.49 [.36, .61]	.31 [.18, .44]	.45 [.33, .57]	.37 [.36, .38]
Psychodynamic	.38 [.28, .48]	.20 [.11, .29]	.35 [.25, .44]	.22 [.11, .34]
Hospital	.16 [.07, .26]	.09 [.00, .18]	.16 [.07, .25]	.15 [.06, .25]
Mixed	.41 [.34, .48]	.26 [.20, .32]	.36 [.29, .42]	.31 [.24, .38]
Panel 6: Presenting problem				
Depression	.36 [.31, .41]	.27 [.21, .32]	.35 [.30, .40]	.34 [.28, .40]
Anxiety	.54 [.42, .66]	.22 [.11, .32]	.36 [.26, .46]	.25 [.07, .43]
Personality disorder	.53 [.39, .67]	.25 [.15, .36]	.37 [.27, .48]	.25 [.06, .44]
Eating disorder	.24 [.15, .32]	.07 [−.02, .16]	.20 [.11, .29]	.10 [.04, .16]
Substance use	.22 [.15, .29]	.15 [.09, .22]	.22 [.15, .29]	.20 [.12, .28]
Mixed	.40 [.32, .48]	.25 [.18, .32]	.37 [.29, .44]	.31 [.21, .41]

Note. Expected effect sizes reported derived from meta-regression model. Full Model reports results based on weighted random effects model. PEESE Model reports results additionally controlling for the squared standard error to further correct for small study effects. Visual Model reports results based on a weighted random effects model where studies from asymmetrical portion of funnel plot are omitted ($SEs > .2$). High Power Model reports results based on a weighted random effects model only including high power studies ($SEs < .1$). ES = effect size; CI = 95% confidence interval. Due to limited data availability for nonclinical samples, the High Power Model for Panel 2 was estimated only for clinical effect sizes. The estimates of the prepost scores for the clinical treatment and control groups does not reflect the overall experimental effect because these estimates only include those studies that reported prepost scores which is a subset used to estimate the experimental effect.

(b) the duration of the effects of interventions after they had been terminated. Table 3 presents effect size expectations for experimental effect sizes across the full dataset. On average, the experience of psychotherapy led to an increase of .43, 95% CI [.30, .55] standard deviations compared to control groups.² The overall effect size was largely consistent across clinical and nonclinical settings (see Table S4, moderator $p = .53$).

To better understand the experimental effects, we also computed the prepost changes for a set of control groups that were available in the set of studies examined. Even with random assignment, difference scores between intervention and control groups may be affected by differences in means that existed at the initiation of the study, which may bias the differences at the end of therapy. Tracking control group prepost change scores allows for a check on the experimental results. Most of these control groups came from the experimental studies, but several comparison group prepost changes also were reported in quasi-experimental studies in which the participants were tracked for time periods equivalent to those receiving treatment. As can be seen

in Table 2, change patterns found in the various control groups were consistent with the inference that the interventions caused more change than not intervening. Specifically, the clinical control groups did show improvement, $d = .24$, 95% CI [.14, .34], the nonclinical and comparison groups showed markedly less change $d = -.03$, 95% CI [−.08, .03] and .15, 95% CI [.09, .21], respectively (see Table 2).

² The effect size estimate was reduced only slightly when omitting low power studies, but the PEESE correction indicated that the effect may be substantially smaller ($d = .13$, 95% CI [−.10, .36]). However, the imprecision of this estimate warrants some caution as the experimental dataset contained relatively few high precision studies (see Figure S2), a critical requirement for accurate PEESE estimates. As a symptom of this issue, one can inspect the increase in the confidence interval width when PEESE is applied. In the prepost dataset, this results in an increase of confidence interval width of 5.7%. For the experimental dataset, the increase is 89.4%. Taken as a whole, the experimental results are less robust than the prepost effects, but the more realistic visual correction still finds evidence of substantial treatment effects, $d = .40$, 95% CI [.28, .51].

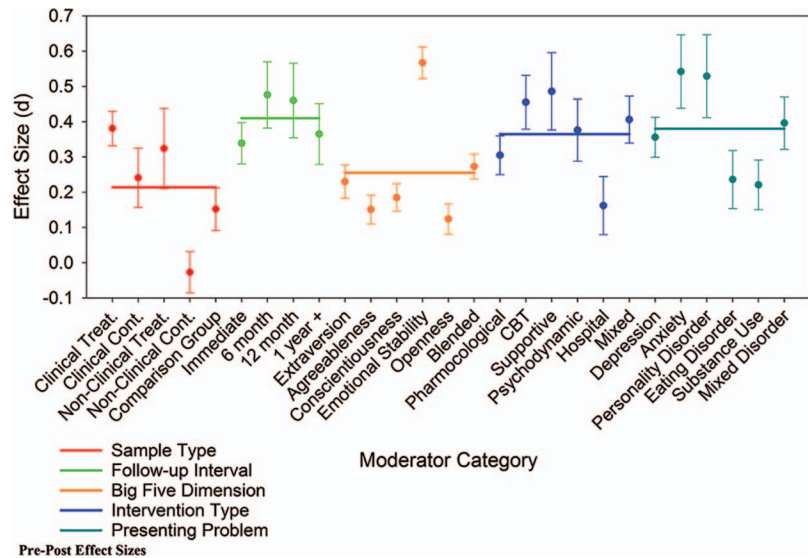


Figure 1. Deviation plot for major moderators.

These two differences between the clinical and other groups are statistically significant (see Table S3, $p < .001$ and $p < .05$, respectively). The findings for the nonclinical studies are highly relevant to the inference that interventions actually change personality traits as clinical studies are confounded by the nature of the participants seeking therapy. In contrast, people in nonclinical experiments would not necessarily be at a low point in their psychological functioning and would not be expected to be systematically affected by the simple passage of time. In sum, it appears that the prepost and experimental effects for interventions are associated with changes in personality traits that exceed what happens to individuals who are not receiving treatment.

A second way of addressing the state-artifact hypothesis is to test whether the changes due to therapy are short-lived. Thus, long-term follow-ups of the intervention groups are critical. If the effect of therapy fades when treatment ends, then one would assume that therapy provided a short-term boost to positive affect or alleviation of

negative affect, which should fade with time and, in turn, drag trait scores back down to prior levels of the true trait level. To evaluate this position, we broke the findings down by follow-up interval (see Table 2). As the results in Table 2 show, there appears to be no marked decrease in the effect sizes with time and follow-up interval. The average effect of treatment was $d = .34$, 95% CI [.31, .37] immediately following treatment, $d = .48$, 95% CI [.36, .60] for studies tracking changes zero to six months after treatment, $d = .46$, 95% CI [.32, .60] for studies tracking outcomes six months to one year. Finally, the studies following up samples for an even longer period of time showed no decline in the effect of therapy, with the changes from one or more years after treatment being $d = .37$, 95% CI [.26, .47]. It should be noted that all of the effects across various follow-up intervals were robust to bias. Moreover, the moderator analysis as shown on Table S3 indicates that the only estimate that differed was the effect immediately following treatment ($p = .02$, other p 's $> .17$).

Table 3
Experimental Personality Change Effect Size Estimates Comparing Treatment to Control

Moderator categories	Full Model ES [95% CI]	PEESE Model ES [95% CI]	Visual Model ES [95% CI]
Panel 1: Average effect size			
Average effect size	.43 [.30, .55]	.13 [−.10, .36]	.40 [.28, .51]
Panel 2: Clinical vs. Nonclinical			
Clinical	.45 [.29, .61]	.15 [−.08, .38]	.40 [.25, .55]
Nonclinical	.36 [.15, .57]	.04 [−.33, .41]	.39 [.20, .58]
Panel 3: Big Five			
Extraversion	.38 [.18, .58]	.20 [−.11, .51]	.39 [.19, .60]
Agreeableness	.23 [.08, .38]	.03 [−.29, .35]	.23 [.09, .38]
Conscientiousness	.06 [−.05, .16]	−.18 [−.52, .16]	.10 [.02, .18]
Emotional stability	.69 [.45, .93]	.39 [.07, .70]	.59 [.35, .82]
Openness	.36 [.23, .49]	.24 [−.04, .52]	.38 [.29, .46]
Blended	.27 [.10, .44]	.01 [−.26, .28]	.27 [.10, .44]

Note. Expected effect sizes reported derived from meta-regression models. Positive effect sizes indicate that personality changed more for treatment groups relative to control groups. Full Model reports results based on weighted random effects model. PEESE Model reports results additionally controlling for the squared standard error to further correct for small study effects. Visual Model reports results based on a weighted random effects model where studies from asymmetrical portion of funnel plot are omitted ($SEs > .4$). ES = effect size; CI = confidence interval.

Taken as a whole, the findings showed that clinical and nonclinical interventions resulted in increases in positive traits over very short time intervals in true experimental designs. Nonclinical control groups showed little evidence of placebo effects commonly seen in clinical control groups. Moreover, the changes tended to be retained following therapy for relatively long-time intervals. When combined, these results provide more support for the cause-correction hypothesis that therapeutic interventions may impart changes in personality traits. In contrast, the state-artifact hypothesis predicts that treatment and control groups would show similar personality increases due to artificially lowered means resulting from states, and because states are typically defined as lasting from moments to at most days, any personality change would not last a month, let alone over a year.

Which Domain of Personality Trait Changes the Most?

Next, we turned to the question of which trait domain changed the most in response to intervention (see Table 2). Clearly, we would expect the effect sizes to be larger for trait domains that are either implicitly or explicitly linked to the focus of the intervention. In the case of the clinical studies, most interventions concerned depression, anxiety, or eating disorders, which are comorbid for depression and anxiety. Therefore, we expected larger changes on the trait domains most strongly linked to affect: neuroticism (i.e., emotional stability reversed), which is associated with negative affect, and extraversion, which is closely aligned with positive affect. When broken out by the Big Five categories, the results for the clinical studies were consistent with these expectations. The largest effects were found for the domain of emotional stability, adjusted $d = .57$, 95% CI [.52, .62], followed by extraversion $d = .23$, 95% CI [.17, .29]. Blended traits, which represented a mixture of positive and negative emotionality, also tended to change more than other dimensions, $d = .27$, 95% CI [.23, .31]. Of the remaining Big Five domains, agreeableness $d = .15$, 95% CI [.11, .20] and conscientiousness $d = .19$, 95% CI [.14, .23] showed increases that were different from zero. The changes found for openness to experience ($d = .13$, 95% CI [.07, .18]) were not robust across corrections for small study bias. In particular the PEESE correction indicated no evidence of change for openness. However, the remaining small study estimates did show an indication that the changes in openness to experience were robust. Despite the clear pattern of expected effects for emotional stability and extraversion, only emotional stability had a significant moderator effect (Table S3, $p < .001$). Trait dimension explained a substantial portion of between-study variance (19%, Table S3), which was much larger than any other moderator ($M = 2\%$).

As these results for the Big Five reflect prepost change scores aggregated from mostly observational studies of the effectiveness of therapy, they do not provide evidence for differential causal effects of therapy for different domains of the Big Five. In order to identify potential causal effects on different Big Five domains, we aggregated the effects drawn from the experimental studies within each Big Five domain (see Table 3). Given the similarities of the effects across clinical and nonclinical studies, and because there a much smaller number of experimental studies, we aggregated all of the experimental studies together. Consistent with the results for prepost change scores, the largest effects were found for emotional

stability, $d = .69$, 95% CI [.45, .92]. Also consistent with expectations, the second largest effect was found for extraversion, $d = .38$, 95% CI [.18, .58], with notable effect sizes found for the remaining Big Five domains. Blended traits also displayed relatively large increases, $d = .27$, 95% CI [.10, .44]. However, the only effects that were robust to the small study bias analyses were those found for emotional stability, such that any changes found in the remaining Big Five should be interpreted with caution. Similar to the prepost results, emotional stability had a significant moderator effect ($p = .001$), such that it was significantly different from the other estimates of change (Table S4). Again, trait dimension explained a large amount of between-study variance (43%, Table S4), which was much larger than other moderators ($M = 4\%$).

Does the Type of Therapy Matter?

A number of other potential moderators and their combinations emerged in the compilation of the studies. Relevant to clinical psychology, many studies reported the effects of various psychosocial interventions, such as cognitive-behavioral therapy, or pharmacological interventions, largely focused on antidepressants. Consistent with the prevailing research on clinical outcomes, all forms of therapy, with the exception of hospitalization, showed similar levels of efficacy in changing personality traits (see Table 2). The effects for supportive therapy, cognitive behavior therapy, and psychodynamic approaches all exceeded .38 and were largely indistinguishable from one another. Supportive and cognitive-behavioral therapy were associated with a slightly larger change than the average treatment (Table S3), but the difference in magnitude was small (difference in d of approximately .1, with p 's = .03 and .02, respectively). Psychopharmacological therapies did exhibit a slightly smaller effect than the remaining approaches, $d = .31$, 95% CI [.26, .35], $p = .03$. In contrast, being hospitalized resulted in the smallest amount of change, $d = .16$, 95% CI [.07, .26] and was statistically significantly different from the average ($p < .001$). All of the estimates for the different types of therapy were robust to small study bias.

Does Type of Presenting Problem in Clinical Studies Matter?

Table 2 also shows the overall magnitude of personality trait change broken down by the presenting problem being treated in the clinical intervention studies. We were able to compile enough studies to compare six types of presenting problems: depression, anxiety, personality disorder, eating disorders, substance use disorders, and mixed diagnoses. As can be seen by the effect size estimates in Table 2, the type of presenting problem being treated does appear to moderate the amount of personality trait change that occurred. Specifically, the changes seen in patients presenting with anxiety, $d = .54$, 95% CI [.42, .66], and personality disorders, $d = .53$, 95% CI [.39, .67], were larger than those for patients presenting with other disorders (both moderator effects $p < .05$, Table S3). Conversely, changes associated with eating disorders and substance use disorders were smaller than the remaining categories (both moderator effects $p < .01$, Table S3). In fact, the PEESE estimator for eating disorders was not distinguishable from zero. Effect sizes associated with depression and mixed diagnoses were at roughly the midpoint of the other conditions (both moderator effects $p > .4$, Table S3).

Focusing on Changes in Emotional Stability

One potential biasing factor affecting the variability, or lack thereof, across the different moderators is the uneven distribution of types of measures used in individual studies. Some types of studies, for example, those focusing on supportive therapy, focused more strongly on personality traits that show more change, such as emotional stability. This may have affected the average magnitude of the effects found for each type of intervention shown in Table 2. To put the different forms of therapy and presenting problems on more equal footing for comparison, we limited the analyses to only the domain of emotional stability (see Table 4). Consistent with this interpretation, the effect sizes for all forms of therapy and all presenting problems were larger when examining changes in emotional stability. The most conspicuous change across these analyses is that all effects were now robust to small study bias. Substantively, when examining only emotional stability outcomes, the three most effective therapies were cognitive-behavioral, supportive, and mixed therapeutic approaches. Moreover, even hospitalization showed a robust association with improvement on measures of emotional stability, $d = .35$, 95% CI [.19, .52]. Mirroring the earlier results, cognitive-behavioral therapy elicited slightly more change and hospitalization slightly less (both $p < .01$, Table S5) compared with the other interventions.

The pattern of moderator effects for presenting problems became much more homogeneous when only examining emotional stability outcomes. While anxiety disorders did again show the largest effects, the effects of remaining categories were also quite large and robust to small study bias. Again, mirroring results from the full dataset, effect sizes associated with anxiety disorders displayed relatively large increases and substance use disorders displayed relatively smaller increases (both $p < .01$, Table S5) compared with the other disorders.

How Long Does an Intervention Have to be to Change Personality?

Extremely short interventions, lasting days rather than weeks, may be unlikely to change personality. To test this, we examined the relation between effect size and duration of intervention. Conceptually, a linear model is unlikely to hold as the effectiveness of therapy may reach an upper asymptote across time. Empirically, our data contained wide variability in intervention duration, and initial inspection of the scatterplot indicated that there may be an exponential relation between duration and effect size. Results are presented in Table S6 and graphically in Figure 2. The central time-related parameter was statistically significant in the full dataset as well as in the emotional stability subset and across all

Table 4
Prepost Personality Change Estimates for Emotional Stability Effect Sizes

Moderator categories	Full Model ES [95% CI]	PEESE Model ES [95% CI]	Visual Model ES [95% CI]	High Power ES [95% CI]
Panel 1: Average effect size				
Average effect size	.59 [.53, .64]	.38 [.33, .44]	.49 [.44, .54]	.48 [.41, .55]
Panel 2: Clinical vs. Nonclinical vs. Control				
Clinical treatment	.61 [.55, .67]	.41 [.35, .47]	.51 [.46, .56]	
Clinical control	.26 [.08, .44]	-.02 [-.20, .16]	.23 [.02, .44]	
Nonclinical treatment	.35 [.16, .55]	.10 [-.11, .31]	.31 [.12, .49]	
Nonclinical control	.09 [0, .17]	-.08 [-.20, .05]	.08 [0, .16]	
Comparison group	.22 [.14, .29]	-.0 [-.07, .07]	.22 [.13, .30]	
Panel 3: Follow-up interval				
Immediate	.50 [.46, .55]	.33 [.27, .38]	.44 [.39, .48]	.45 [.39, .50]
6 month	.81 [.64, .97]	.57 [.40, .75]	.68 [.48, .88]	.79 [.41, 1.17]
12 month	.82 [.70, .94]	.58 [.48, .67]	.69 [.58, .80]	.56 [.33, .79]
1 year +	.76 [.60, .92]	.57 [.44, .70]	.62 [.47, .77]	.52 [.35, .68]
Panel 4: Intervention type				
Pharmacological	.48 [.38, .58]	.31 [.23, .40]	.39 [.32, .46]	
Cognitive behavioral	.73 [.61, .86]	.45 [.34, .55]	.53 [.44, .62]	
Supportive	.68 [.47, .88]	.45 [.24, .66]	.61 [.39, .83]	
Psychodynamic	.49 [.39, .59]	.32 [.22, .41]	.45 [.36, .54]	
Hospital	.35 [.19, .52]	.28 [.11, .45]	.37 [.20, .53]	
Mixed	.66 [.56, .75]	.47 [.38, .56]	.57 [.47, .67]	
Panel 5: Presenting problem				
Depression	.59 [.51, .66]	.50 [.43, .57]	.58 [.50, .65]	
Anxiety	.75 [.61, .89]	.36 [.22, .51]	.51 [.37, .64]	
Personality disorder	.59 [.45, .72]	.35 [.23, .46]	.46 [.37, .55]	
Eating disorder	.61 [.40, .83]	.39 [.14, .64]	.57 [.31, .83]	
Substance use	.35 [.24, .46]	.28 [.18, .38]	.35 [.25, .45]	
Mixed	.55 [.44, .65]	.39 [.28, .50]	.50 [.39, .62]	

Note. Expected effect sizes reported derived from meta-regression models. Full Model reports results based on weighted random effects model. PEESE Model reports results additionally controlling for the squared standard error to further correct for small study effects. Visual Model reports results based on a weighted random effects model where studies from asymmetrical portion of funnel plot are omitted ($SEs > .2$). High Power Model reports results based on a weighted random effects model only including high power studies ($SEs < .1$). ES = effect size; CI = confidence interval. Due to limited coverage, we were unable to estimate the High Power Model for clinical vs. control, intervention type, or presenting problem. The estimates of the prepost scores for the clinical treatment and control groups does not reflect the overall experimental effect because these estimates only include those studies that reported prepost scores which is a subset used to estimate the experimental effect.

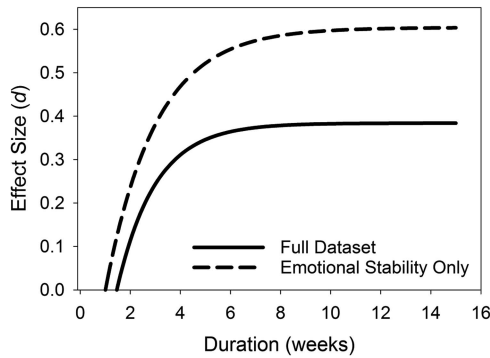


Figure 2. Exponential trends in treatment duration.

corrections for small study effects. All analyses converge on the finding that interventions lasting less than approximately 4 weeks tend to have small effects. Beyond roughly 8 weeks, longer interventions do not induce greater personality change. This result provides critical information about how quickly personality can change.

Ancillary Moderators

As noted, we also coded studies for several factors that, although not directly relevant to the issue of whether interventions change personality traits, may have acted as moderators of the findings. These moderators were year of publication, mean age of the sample, and percentage of the sample that was female. Publication year was weakly associated with effect size, with an estimated increase of .04, 95% CI [.02, .06] per decade. Age of the sample was not associated with effect size ($p = .63$). Percent of the sample that was female was not associated with effect size ($p = .56$). We also examined whether samples that were entirely composed of females compared with entirely males differed in effect size, but the effect sizes did not differ, $b = .03$, 95% CI [−.02, .09]. Next, we conducted exploratory analyses to investigate whether gender composition moderated the effectiveness of treatment or responsiveness of certain disorders. Gender (i.e., percentage of sample that was female) did not moderate the effectiveness of any treatment (p 's $> .23$). For presenting problem, studies focusing on eating disorders were almost entirely female (minimum percent female $>90\%$). Therefore, we omitted eating disorder effect sizes from this analysis. Once doing so, results indicated that personality traits changed less in studies of personality disorders when there was greater female representation in the sample ($p = .04$). However, upon inspection of the data, two studies were outliers in terms of very low female representation, and when omitted, the effect was no longer significant ($p = .50$), limiting confidence in the robustness of this result. Thus, gender composition of the sample does not have direct or interactive effects on personality trait change. Because these results are largely null, we did not investigate small study bias.

Discussion

The current meta-analysis sought to investigate the extent to which personality traits changed as a result of intervention, with the primary focus on clinical interventions. Focusing exclusively

on studies that included measures of personality traits, hundreds of studies were found and synthesized. One of the most salient features of the results was the pervasive positive and reasonably large patterns of change that were found on personality trait measures across various ways of examining the data. Interventions clearly are associated with changes in personality trait measures in the reported literature.

The most pressing question is whether the interventions actually changed personality traits or resulted in some other type of change, such as changes in episodic states rather than traits. Two clear positions have been outlined in prior theoretical and conceptual work on clinical interventions and personality trait change—the state-artifact and the cause-correction positions. Both positions argue that personality traits will change during a typical clinical intervention. Where the positions diverge is on the meaning of that change. According to the state-artifact position, the changes in personality traits during therapy can be attributed to state-content contamination of typical trait measures. People who change their moods will also change their ratings on trait measures, but these types of changes do not reflect real trait change. In contrast, the cause-correction position posits that the changes in personality trait measures are enduring and have real consequences for clinical outcomes such as recovery and relapse (Tang et al., 2009).

The findings of the primary studies contained in the meta-analysis offered various forms of evidence that, in aggregate, shine some light on whether the changes were the result of state-level contamination. First, and foremost, it was important to know whether interventions experienced by an experimental group—a true experimental condition—worked relative to a true control group. Fortunately, a group of studies tracked personality traits with both a clinical and a nonclinical focus. Across both types of interventions, experimental groups changed more than control groups and at magnitudes that were quite large relative to observational studies of personality change over similar time periods. For that matter, the change in emotional stability, the most highly relevant trait domain to clinical interventions, was approximately half of the change expected over the entire life span based on typical passive observational studies (Roberts et al., 2006). If aggregated, it appears that most people increase on emotional stability approximately 1 standard deviation from young adulthood through middle age. Therapy lasting 4 or more weeks achieves half that amount of change. Moreover, patterns of change in the control groups helped to rule out the argument that the changes found in personality traits were simply the result of rebounding from a low point in one's life as the changes in the experimental groups were much larger on average. It appears that interventions cause changes in personality trait ratings over the short-run.

Although the findings from the true experimental studies bolster the inference that interventions can cause short-term changes in personality traits, this does not address the primary argument behind the state-artifact position that the changes caused by therapy are short-lived because they reflect changes in states, not traits. To further address this idea, we compiled studies that followed up on samples after the participants had terminated their therapy. Regardless of the timeline, the effects consistently supported the cause-correction position. There was no evidence that the effects of therapy faded with time. Specifically, personality levels remained altered after more than a full year by between .24 and .37 standard deviations, and more impressively, emotional stability

levels remained altered after a similar time span by between .52 and .76 standard deviations, depending on the correction for small study effects. So, within the confines of the studies contained in the current meta-analysis, it appears that the preponderance of data supports the cause-correction hypothesis and the inference that seeing a therapist can lead to lasting personality trait change.

There is, however, one major caveat to this conclusion. It is possible that the changes that clinicians impart are not remaking someone's personality. Rather, what a clinician might be doing is bringing people back to the baseline that existed before their episode of psychopathology. It is a fact that clinical intervention studies all start with people who are suffering, so we do not know what the participants in controlled intervention studies were like well before they experienced depression, anxiety, or some other form of psychopathology. We do know from passive longitudinal studies that people who go into therapy typically score higher on neuroticism prior to engaging in a therapeutic relationship (e.g., Lüdtke et al., 2011). Unfortunately, this type of information is lacking for the entire corpus of intervention research since researchers do not have the ability to track entire populations out of which they select individuals for treatment. The key, unanswerable question is exactly where intervention study participants were on personality trait levels well before their clinical episodes. But this type of data is almost impossible to acquire.

However, the set of nonclinical studies provides further evidence that interventions do lead to meaningful personality trait change. These studies did not focus on samples that had a prior history of psychopathology, and therefore, any change exhibited by these types of interventions provides stronger evidence for real change that goes beyond bringing a person back to their prior baseline. Put differently, it is hard to imagine how the state-artifact hypothesis could apply to these effect sizes. As we found, non-clinical groups changed just as much as clinical samples on personality trait measures. Unfortunately, the set of studies in this category is much smaller, and none of these studies report long-term change patterns. It is still a possibility that these changes fade with time, though the evidence is suggestive that they do not.

It is also the case that the ideal data to differentiate state-level change from trait-level change was not provided in a form that could be tested systematically in this data set. In particular, an ideal test of the differentiation of the state-artifact and cause-correction positions would be to assess change in both states and traits and control for the change in states when examining change in traits. Unfortunately, too few studies provided the necessary data³ to test this more optimal approach to differentiating these two positions. Nonetheless, future research could help clarify this omission by examining state and trait change simultaneously.

In sum, the data found in this set of studies provides tentative support for the idea that interventions do lead to personality trait change over time. Nonetheless, the data are not complete and without evidence such as the long-term efficacy of change interventions in nonclinical samples, we believe it would be prudent to be cautious in making a strong case that clinical interventions change personality traits.

Personality Traits Can Change Quickly

Quite possibly the most significant finding of this research is the fact that personality traits can and do change more quickly than

commonly thought. The examination of the relation between duration of treatment and change showed that most of the gains were made within the first month of therapy. This empirical fact contradicts the widely held assumption that personality traits typically change slowly and gradually over many years (Roberts, 2006). Conceptually, the idea of gradual change was consistent with the bottom-up process of change informed by theories such as the sociogenomic model of personality traits (Roberts & Jackson, 2008). This model suggests that the slow accretion of small behavioral, attitudinal, and emotional changes that occur over time eventually become internalized and automatized. The sociogenomic assumption of slow and gradual change was derived from the large number of passive, observational, longitudinal studies on personality trait change, which show modest changes occurring over relatively long periods of time (Roberts et al., 2006). The findings from the current meta-analysis invite closer scrutiny of this conclusion.

Specifically, the appearance of progressive, incremental, and small amounts of change made over long periods of time may have been an artifact of the study designs typically employed in longitudinal personality research. Almost no longitudinal studies have tracked personality traits continuously during the length of the longitudinal study. If we imagine combining the typical clinical intervention design (continuous assessments every few weeks or months) with the typical personality longitudinal design (assessments spaced out across long time intervals), we may see a second explanation for the seemingly modest changes in personality traits over long periods of time. The modest changes that appear in passive longitudinal studies may have resulted from a minority of individuals changing dramatically over short periods of time, with most others remaining roughly the same on trait levels. The typical passive longitudinal design would be insensitive to this possibility, and the resulting modest pattern of change could be a misrepresentation of the process of change that occurs.

This possibility has been alluded to in several close examinations of change in passive longitudinal studies. Specifically, a set of longitudinal studies utilized a very conservative index of change, called the Reliable Change Index (RCI; Roberts et al., 2001; Robins et al., 2001), which was created to determine whether the changes that resulted from therapeutic interventions were larger than would be expected by chance (Jacobson & Truax, 1991). Interestingly, when the RCI index has been used, dramatic levels of change on personality traits have been found for most populations (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006; Pullman, Raudsepp, & Allik, 2006). More importantly, and relevant to the findings of the present study, only a minority of individuals shows dramatic changes on any given trait. In fact, the base rate of these dramatic changes has been estimated to happen on one in five traits over periods as long as 8 years (Roberts et al., 2001). Thus, the modest changes found at a population level may reflect very large changes that occur in a small subset of any given population on any given trait—a finding consistent with the evi-

³ Ideally, one would need state and trait measures such as state anxiety and trait anxiety and the correlation between each within the samples being followed. It is the latter information that is almost never reported in clinical intervention studies. We hope with the advent of online data repositories that this type of information can be recorded going forward.

dence from this study that personality traits can and do change in a short period of time.

Unfortunately, given the assumptions that underlie personality traits, this possibility has never been tested because very few researchers have tracked personality traits both continuously and over a long period of time. What this highlights is the fact that within personality science, we do not know or understand much about the process of personality trait change because we have yet to reliably track the change as it happens.

The solution to this oversight is relatively simple. Researchers need to conduct longitudinal studies of personality traits in which the traits or their concomitant behavioral, attitudinal, and affective states are assessed on a more regular basis, such as every week or month in order to capture when and how change occurs. It is possible that the process of personality trait change plays out in multiple ways, with some people changing slowly and incrementally, and others changing quickly and then solidifying the gains or losses they experienced and changing very little thereafter. These two process models of change—the incremental model and what we might describe as the “punctuated equilibrium” model of personality trait change—lie as untested explanations of the process of personality change.

Potential Moderators of Personality Trait Change

The clearest moderator of the effect of clinical interventions on personality trait change was the domain of the trait itself. Consistent with expectations, the two trait domains that changed the most were emotional stability (neuroticism) and extraversion for both clinical and nonclinical studies. These effects were seen in both the prepost change scores and the effects derived from true experiments. The magnitude of change in emotional stability was about one half of a standard deviation, which is notable, as it is roughly equivalent to half the gain typically made in these traits across an entire life course (Roberts et al., 2006). That clinical interventions, which are largely targeted at alleviating negative affect (e.g., anxiety) and addressing an absence of positive affect (e.g., depression), would impart change largely in these two trait domains is not surprising.

Of the remaining trait domains, we found modest changes that were not always robust to small sample bias. This mixed set of results would caution against strong arguments for the potential effect of therapy on trait domains other than extraversion and emotional stability. We view the lack of systematic change across all of the Big Five as a good sign. If we were to find substantial changes on all positive traits, it would invite the possibility that personality trait change that resulted from therapy reflected increases in self-presentation of being “better,” or the possibility of a global positivity effect of therapy (see [Mu, Luo, Nickel, & Roberts, 2016](#)). The relative specificity invites the inference that interventions might be tailored to address narrow traits within trait domains with high fidelity.

We also considered two major potential moderators of the types of changes that would occur on personality traits: the form of therapy being used and the presenting problem being treated. Consistent with the Dodo Bird Effect (Wampold et al., 1997), we found little systematic difference between the type of therapy used to treat patients, especially when examining emotional stability as the outcome. Clinical research has consistently found that type of

therapy is not strongly associated with differences in the efficacy of therapy ([Luborsky et al., 2002](#)). More to the point, therapy appears to lead to improvement regardless of what type of therapy is administered. Our findings for change in personality traits were surprisingly similar to prior research demonstrating little or no difference in the effectiveness of different forms of clinical interventions, with the exception of people who were hospitalized for their problems. This latter group may be less relevant to evaluating therapies, as it may be a better indicator of what happens when people are stabilized after experiencing the most severe episodes of psychopathology.

There were two apparent effects for moderation by presenting problems. Of the six categories of presenting problems, people presenting with anxiety-related issues and personality disorders showed the most change, and people presenting with eating disorders and substance use problems showed the least amount of change. These apparent differences were reduced markedly when we just examined emotional stability outcomes. This lack of difference between different presenting problems was interesting because one of the presenting categories was for personality disorder, which are often considered untreatable or difficult to treat at best. Therapists appear to be successfully changing individuals with personality disorders, even if therapists do not believe they are doing so. One possibility is that people with personality disorder are experiencing a much wider set of problems that are more severe than people with other types of disorders are experiencing. This might mean that the changes, though real, are still somewhat dwarfed by the multitude of problems (e.g., interpersonal difficulties, problems in work and family) that someone faces when diagnosed with, for example, borderline personality disorder ([Gunderson et al., 2011](#); [Skodol et al., 2005](#)). The answers to the questions like these raised by the review await more focused longitudinal and intervention research.

Of the remaining moderators, we found that duration of treatment had a nonlinear relation with personality trait change, such that interventions that lasted less than 1 month were less efficacious than those that lasted longer. We would caution making strong inferences based on these analyses as there were relatively few studies shorter than 1 month in duration. On the other hand, the asymptotic nature of long-term treatment is worthy of deeper study, as it implies that the effect of very long-term therapy may not be easily differentiated from more modest interventions. Year of publication, gender, and age did not have systematic relations to personality trait change. These null findings are interesting because of their implications. First, there appears to be no decline effect in this literature given the lack of association between year of publication and the reported effect sizes of personality trait change. Decline effects often occur when the first reports of an effect are larger than the subsequent reports, often because the nature of the publication incentives rewards provocative, if less well-designed, studies when first reporting results. The lack of a systematic effect of gender indicates that therapies are not currently differentially affecting men and women with respect to personality trait change. If one is hoping that current therapeutic approaches are equally effective across genders, then this is a positive finding. In contrast, if one hopes that certain therapies might be differentially effective with men or women due to targeting of unique etiological factors, these findings give no indication that the types of therapies studied here help in that regard. The

lack of an age effect is also interesting as it supports the plasticity of personality across the life course given the samples ranged from adolescence through old age. The fact that therapies appeared to be just as efficacious with young, middle-aged, and old populations would appear to support the plasticity principle that personality is an open system and amenable to change even if change decreases with age (Roberts, Wood, & Caspi, 2008).

Limitations and Future Directions

One salient limitation of the compiled data set is the reliance on self-report measures of personality traits. We did find a handful of studies that used more than one method, and these few studies reported that observer ratings of patients (often performed by the therapists themselves) also increased in a positive direction (e.g., Hoglend et al., 2008). That said, the inference that therapy results in a truly noticeable improvement on personality traits would benefit greatly from research designs where friends and interviewers who were not directly involved with the intervention were the source of observer ratings. Including observer ratings alongside self-reports would allow stronger inferences that personality traits are actually changing and not less compelling constructs, such as response sets. Moreover, developing some form of unobtrusive or objective index of personality traits that was not subject to self-presentational strategies or other biases would also benefit researchers' ability to infer that interventions resulted in true change.

Another limitation of the body of research concerns the construct validity of the changes that are found. That is, we do not know whether the changes incurred by therapy predict important outcomes. For example, in passive observational studies of personality development, it is now known that changes in personality traits are predictive of important outcomes above and beyond original standing on those same traits (Moffitt et al., 2011; Mroczek & Spiro, 2007; Takahashi et al., 2013). Likewise, it would alleviate concerns about the changes in self-reported personality traits if the changes themselves predicted consequential outcomes beyond the therapeutic intervention, such as later relapse (e.g., Tang et al., 2009; Vittengl, Clark, & Jarrett, 2010; Vittengl, Clark, Thase, & Jarrett, 2015).

The heterogeneity of our data was also an issue. Our meta-analytic results found substantial mean-level change in personality, but this change was not uniform over all of the observed studies. For example, we demonstrated that change was more pronounced for measures of emotional stability. However, substantial between-study variance remained even after including coded moderators. On the one hand, this result might be expected as we drew on an extremely diverse set of studies. We included studies of any personality dimension in any setting for any disorder (or no disorder), as long as an intervention was applied. On the other hand, between-study variance has two important statistical implications. First, between-study variance reduces statistical power (Pigott, 2012). Because we drew on a very large body of research, statistical power was not a large concern for our analyses, and indeed, we report narrow confidence intervals reflecting this fact. Second, large between-study variance implies that our aggregate effect sizes do not apply universally, but rather are the average expected effect size. Our results imply that there are many situations in which one would expect smaller effect sizes than the average, and many other

situations in which one would expect larger effect sizes than the average. Apart from personality dimension, our coded moderators accounted for relatively little of the between-study variance. Future work in this area will be necessary to tease apart the factors that lead to such variance.

Another limitation of the compiled data was the pervasive evidence for publication bias throughout many of the categories that were analyzed. According to the PEESE test and the funnel plot analyses, many of the estimates showed some signs of publication bias. The bias, though pervasive, was not enough to eliminate the effects of therapy on personality trait change when a variety of adjustments were made. Nonetheless, it indicates a need for more preregistered, controlled studies conducted by individuals who are not motivated to show the effectiveness of any given therapy or intervention. Also, most of the studies in the data set were powered only to detect medium or large effects. Larger sample sizes would potentially enhance the accuracy of future estimates of change. Moreover, most research examined interventions drawn from programs designed at a specific hospital or clinic. One opportunity afforded by focusing on personality traits, rather than psychopathology, is that interventions could be implemented outside of institutions that employ interventions as a rule and thus provide a more objective evaluation of the intervention efficacy.

The results of the study also challenge future researchers to create an empirical edifice that is more detailed and thus more easily tested and refuted. Specifically, we know very little about the magnitude of variability in personality and behavior across different time periods. The current study is novel in large part because personality developmental researchers have failed to consider that change could occur in very short periods of time when designing their longitudinal studies. Thus, we simply do not have data on how personality trait measures behave over weeks, months, and even a year because the assumptions have always been that years were needed to detect development. Without this information, it is difficult to gain perspective on the amount of change we found in this study and the absolute minimum amount of time sufficient for personality trait change to occur. While a half of one standard deviation appears large from the perspective of the average effect sizes in psychology, we still do not know whether that is meaningful. Mapping the magnitude of change across time and measures is a basic task that is in dire need of attention.

Another key necessity for future research is to identify the mechanisms responsible for personality change in the therapeutic setting. It is tempting to endorse specific theoretical and conceptual explanations typically invoked in clinical research. For example, one could propose that the cognitive reorganization intrinsic to a cognitive-behavioral approach to therapy is a viable mechanism that helps impart personality trait change. However, the fact that personality trait change happened across all types of therapeutic intervention militates against this argument. People clearly change across many different constructs as a result of going to see therapists, but the explanations for why remain elusive, especially in the case of personality trait change.

Conclusion

Modern personality trait theories have successfully moved beyond the false dichotomies posed by prior generations of research-

ers that painted a picture in which personality traits were either perfectly stable or permanently variable (Roberts, 2009). Personality traits not only show robust evidence for change across the life course, but also show meaningful changes in relation to life experiences (Roberts & Mroczek, 2008). Moreover, theoretical systems have developed that move modern trait theory to a stage in which these changes are not only possible, but it is possible to entertain the more provocative question of whether personality traits can be changed through specific interventions.

As this review has shown, the contemporary take on personality traits simply catches up with the wealth of evidence that has been accumulating in clinical science for decades. Clinicians and other interventionists have been changing personality traits for many years. Appropriately, clinical researchers have identified personality traits, primarily neuroticism, as key contributors to psychopathology (Lahay, 2009) and suggested that they should be the primary focus of interventions given their widespread relation to various forms of psychopathology (Barlow et al., 2014). It is time that these findings are fully appreciated in both personality psychology and other fields—such as economics, political science, and health psychology—that use personality traits as tools. Personality traits are not only robust predictors of important life outcomes, but also appear to be amenable to intervention. This fact opens the door to a new era of research that more strongly links personality and clinical psychology, and other groups, such as educational psychologists and economists, who are interested in changing people and their behaviors in order to help them with their lives.

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