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Proximal Processes, Temperament, and Pathological Narcissism: An Empirical Exploration from the Longitudinal Study of Personality Disorders

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Abstract
Introduction: It is argued that all personality pathology represents the final emergent product of a complex interaction of underlying neurobehavioral systems, which are reflected in personality factors, in conjunction with environmental inputs. Neurobehavioral systems manifest themselves in dispositional temperament and personality processes. Environmental inputs include, obviously, interpersonal relationships (e.g., parenting, social, and mentoring relations) as well as other factors such as abuse, neglect, and/or environmental insults (e.g., economic hardship, deprivation). Narcissistic personality disorder (NPD) is hypothesized to reflect both dispositional and environmental inputs to its pathogenesis. Temperament and personality-based theorizing regarding NPD proposes high dispositional levels of anger and related temperament features that could shape early development and subsequent NPD. Many classic theorists (e.g., Freud, Kernberg, Kohut, Miller) have also proposed that profound parenting failures are implicated in the emergence of NPD, each suggesting some failure in proper engagement and responsivity with the developing child. Such a failure in parenting can be thought of as reflecting diminished proximal process engagement with the developing child. ***Method:*** Using data from the *Longitudinal Study of Personality Disorders*, the present study examines both proximal process and temperament factors in relation to clinically significant NPD features from a prospective perspective. ***Results:*** Results suggest that both proximal process and temperament (notably anger) factors independently predict the level of NPD features over time. ***Conclusion:*** Both interpersonal relationships and temperament should be considered in models of etiology of NPD, it is not just one or the other.

Introduction

We have long argued that all personality pathology represents the final emergent product of a complex interaction of underlying neurobehavioral systems, which are reflected in temperament and personality factors, as well as environmental inputs [1–4]. Neurobehavioral systems, which manifest themselves in temperament and personality processes, define a theoretical reaction potential, and such neurobehavioral systems exert their influence in a

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"bottom-up" manner. Environmental inputs include, obviously, early interpersonal relationships (e.g., parenting, mentoring relations) as well as other factors such as abuse, neglect, and/or environmental insults (e.g., economic hardship, deprivation). Although all personality disorders likely reflect an admixture of temperament, personality, and environmental inputs, narcissism and narcissistic pathology are of considerable interest from a developmental psychopathology vantage point given proposed models of pathogenesis explicitly focused on temperament and early psychosocial experience.

Narcissism and narcissistic personality pathology have long been of interest in psychiatry, psychoanalysis, clinical psychological science, and other behavioral science vectors (e.g., personnel selection). Much of the work in this broad area of inquiry stands on the shoulders of early clinical observers such as Freud [5] and, later, Kernberg [6] and Kohut [7, 8]. Normal personality science scholars have probed narcissism as a normative personality dimension or trait, whereas psychopathologists and clinicians have probed highly dysfunctional pathological narcissism and narcissistic disturbances as mental disorders [9, 10]. This active area of inquiry has continued to grow and has been summarized in recent reviews and position pieces [11–15]. With increased empirical research and substantive model evaluation, critical theoretical and descriptive insights have been gleaned such as: (a) parsing self-esteem from normal range narcissism [16]; (b) the delineation of grandiose and vulnerable dimensions of pathological narcissism [10]; (c) the importance of well-known personality constructs, such as agentic extraversion, agreeableness, and neuroticism, in describing narcissism [14]; and (d) the severe psychological impact of what is known as malignant narcissism [17].

The etiology and pathogenesis of narcissistic personality pathology remain of considerable interest to clinicians and researchers alike. Efforts to understand the processes related to narcissistic experience and emergence of the disorder have ranged from the classic functional psychological proposition of Stolorow [18] to neurobehavioral theorizing [1–4]. However, the specific etiology [19] of narcissistic personality pathology, not unlike many forms of psychopathology, remains opaque. Considerable insight has been gleaned from the intensive assessment and treatment of individuals affected by pathological narcissism, or narcissistic personality disorder (NPD), over the past 100 years. Clinically informed theorizing has presented clear-cut hypotheses regarding temperamental (dispositional) factors as well as psychosocial inputs from parenting (and other caretaking individuals) to the development of such pathology. For example, temperament and personality-based theorizing regarding NPD proposes high dispositional levels of anger and related features that could shape early development and subsequent NPD. At the same time, expert clinical observation argues for the essential role of psychosocial failures in early relations impacting the development of children and how such failures might set the stage (even cause) narcissistic pathology [5–8, 20–23]. However, the jury remains out as to whether dispositional factors or environmental inputs separately, or *both* together, cause narcissistic disorders. Consider Kernberg's [6] observation: "These patients present a pathologically augmented development of oral aggression, and it is hard to evaluate to what extent this development represents a constitutionally determined strong aggressive drive, a constitutionally determined lack of anxiety tolerance regarding aggressive impulses, or severe frustration in their first years of life. Chronically cold parental figures with covert but intense aggression are a very frequent feature of the background of these patients" ([6], p. 58). In short, the question is: What causes narcissistic pathology, namely dispositional factors (anger, distress), environmental/interpersonal factors, or both?

A connection between NPD and temperament and personality is plausible. In brief, temperament refers to more genetically influenced and biologically based behavioral propensities observable early in life, whereas personality traits are thought to be built upon temperament and they emerge over time in interaction with the environment (allowing for genetic influences as well) [24]. It is clearly established that NPD features are correlated with personality factors in normal personality frameworks (e.g., five-factor approach: high angry hostility is present along with low trust, straightforwardness, altruism, compliance, modesty ([25], see also [14]). Similarly, in the newer *DSM-5* Alternative Model of Personality Disorders [26], one sees impairments in identity, self-direction, intimacy, and empathy along with elevated grandiosity and attention seeking coalesce around NPD features. Available data suggest that NPD is subject to considerable genetic influences whether using a traditional NPD phenotype or the newer AMPD dimensional approach [27–30]. Thus, there is considerable evidence linking the narcissistic pathology construct to personality factors as well as genetic influences, which is an empirical pattern consistent with existing hypotheses regarding the importance of *dispositional* factors in the etiology of narcissistic pathology.

In narcissistic pathology, theory posits a developmental picture of the interpersonal/social relations of the child and important caregivers (e.g., parents, other significant persons), such as it has emerged thus far, that suggests a failure of parents or other caregiver to meet basic emotional needs of the child. This could be, perhaps, inattention to emotional needs of the child, failure to provide a healthy emotional involvement with the child, and/or failure to engage with the child in an emotionally invested manner. Both Kernberg [6] and Kohut [7, 8] have hypothesized that early rearing social relations play a critical role in the development of narcissistic disorders. Horton [23] reports on a variety of parenting features (e.g., warmth, overindulgence, control, monitoring, and so on) are related to increased narcissistic features on normative narcissism personality trait inventories, some tapping grandiose and vulnerable pathological narcissism separately. However, most empirical studies relevant to this parenting/psychosocial input question do not address NPD, the *clinical* entity associated with significant impairment in social, occupational, and family functioning. Moreover, extant studies measure parental traits or demographic features, but not necessarily psychological *processes* thought to be central to the development of psychopathology.

How might one conceive of a process-oriented psychosocial/environmental approach to child development, particularly one that is centered on social relations? One possible approach to understand the manner in which environmental inputs interact with developing psychological systems is to use the *proximal process* construct advocated by the late Cornell developmental psychologist Urie Bronfenbrenner [31]. According to Bronfenbrenner's *person-process-context-time* model [31]:

> "…human development takes place through processes of progressively more complex reciprocal interaction between an active, evolving biopsychological human organism and the persons, objects, and symbols in its immediate external environment. To be effective, interaction must occur on a fairly regular basis over extended periods of time. Such enduring forms of interaction in the immediate environment are referred to as proximal processes. Examples of enduring patterns of proximal process are found in …making plans, performing complex tasks, and acquiring new knowledge and know-how" ([31], p. 996).

This conceptualization of the proximal process owes a considerable intellectual debt to Vygotsky's [32] theoretical concept of the *zone of proximal development*, which refers to that difference between what a person seeking to learn or master a task or skill can do without assistance versus and what he or she can do with assistance. In other words, in attempting to master a skill, one can reach a certain level of accomplishment on one's own, yet there is additional accomplishment possible if one gets help or guidance – this is the zone of proximal development. Examples of what Bronfenbrenner and Morris [31] would consider proximal processes are: making plans with a child to pursue an activity or project, problem-solving with a child, doing reading, artwork, or other creative activities with a child, learning to play a musical instrument, or developing other goals and pursuing them. An essential aspect of proximal processes so conceived is that they require reciprocal interaction between the child and people in their environment as well as other aspects of their environment. According to Bronfenbrenner and Ceci's general model [31, 33], a proximal process describes a mechanism of organism-environment interaction such that "the effect of increased levels of proximal processes" "foster the development of effective psychological functioning ([33], p. 571)" and "proximal processes *raise* [italics added] levels of effective developmental/psychological functioning ([33], p. 572)." They further theorize that proximal processes are the mechanism "through which genetic potentials for effective psychological functioning are actualized" ([33], p. 568) and they may help "to buffer genetic potentials for developmental dysfunction," showing their greatest effect in disadvantaged/disorganized environments (less so, in stable/advantaged environments). Finally, the term *effective*, as used in this formulation, means: "(a) differentiated perception and response; (b) directing and controlling one's own behavior; (c) coping successfully under stress; (d) acquiring knowledge and skill; (e) establishing and maintaining mutually rewarding relationships; and (f) modifying and constructing one's own physical, social, and symbolic environment" ([33], p. 569). For this to be optimal, the engagement needs to help bring the child along to a higher level or more complex level of psychological functioning. In short, proximal processes are thought to (a) have health producing effects, (b) help to actualize the genetic potentials that underpin psychological development, and (c) serve as a mechanism by which the developing child can advance in psychological adaptation, complexity, and health over time. A clear assumption in this model is that social experience and processes can influence the development of psychological and psychosocial systems that are mediated by neurobiological systems, an assumption that is viewed as entirely plausible [34].

In this context, it is important to clarify precisely what proximal process is and is not. Proximal process refers to the specific quality of an *active, engaged interpersonally-*

based psychological interaction that occurs between an adult and a child as proposed by Bronfenbrenner ([31], see also [33]). Insofar as other common developmental psychology constructs are concerned, proximal process is *not* perceived parenting quality on the part of the child or observer, it does not refer to inner working models hypothesized by the attachment perspective, nor does it refer to social support in childhood as commonly described in sociological-developmental approaches. The primary person involved in the proximal process relationship, in addition to the child, is often a parent or other caregiver, but not necessarily so. For example, a child could have a significant adult in his or her life that serves to engage the child in the manner hypothesized by the proximal process notion, even when his/her parents are dysfunctional themselves. In fact, in this latter scenario, the presence of an adult/child relationship characterized by rich proximal process may be even more important in facilitating development in the face of adversity[1].

Therefore, for this special issue that focuses on the importance of relationships in mental health, I use narcissistic personality pathology as a focus to discuss interpersonal relations, proximal processes, temperament, and psychopathology. The data for this exercise are from the *Longitudinal Study of Personality Disorders* (LSPD) [35]. In overview, a proximal process measure will be explored in relation to NPD features while simultaneously taking childhood temperament into account across a multiyear period of prospective longitudinal study.

An important assumption concerning the putative role played by proximal processes in development is that proximal processes help to actualize genetically determined potentials for various psychological and behavioral outcomes; that is, proximal processes help to moderate the expression of latent genetic potentials for expressed psychological or behavioral phenotypes. Thus, it is important to document that the intermediate and end-product constructs at play in the model at hand concerning NPD are, indeed, known to be subject to genetic influences. To wit, the temperament constructs under consideration in this study – emotionality, activity, sociability – are well established as being under considerable genetic influences, in childhood and adulthood [36] and, as noted previously, NPD is also known to be subject to genetic influences [27–30].

[1] It is conceivable that proximal processes may be active in effective, health producing parenting practices. However, the scope of the present paper precludes an extensive review of the parenting literature and how it may or may not address the issue of proximal process as defined by Bronfenbrenner [31].

Thus, to state explicitly the intention of the study at hand, the present empirical study examines the relations between temperament (dispositional factors) and proximal processes (environmental factor) of possible etiological significance for NPD features in adulthood across time. Are these factors predictive of NPD features separately? Are they interactive in predicting narcissistic PD features? Does their predictive relationship with the outcome measure maintain over time? The data used for illustration of this heuristic proposal are drawn from the LSPD [35], which is a prospective, multiwave study of personality disorders, personality, and temperament.

Methods

Dataset

The Study and Procedures: LSPD

The data for this study come from the LSPD, which has been described in great detail in numerous prior publications [35, 37, 38]. The LSPD, begun in 1991, as the first NIMH sponsored longitudinal study of PD's, has a prospective multiwave longitudinal design with subjects evaluated at three points in time, corresponding to their 1st, 2nd, and 4th years in college. The LSPD was begun in a nonclinical setting (a university) in Ithaca, NY (USA). Interview assessments for PD (i.e., Axis II) and Axis I disorders were conducted by PhD or advanced MSW clinicians with extensive Axis II diagnostic experience, at each of the three assessment waves [39]. All subjects in the LSPD also completed a self-report inventory for personality pathology at all three assessment waves. Finally, as the LSPD is a naturalistic prospective study, subjects were free to seek psychological treatment of their own accord. As noted, extensive detail regarding the LSPD, including reliability assessments and other technical matters, is given in prior publications and the interested reader is referred to those papers [35, 37, 38].

Subjects

The 258 subjects in the LSPD were drawn initially from an initial population consisting of 2,000 first-year undergraduate students [35, 37, 39]. All subjects gave voluntary written informed consent and received an honorarium of USD 50.00 at each wave of data collection. Extensive detail concerning the initial subject selection procedure and sampling is given elsewhere [35, 37, 39] and is not repeated here. Of the initial 258 participants, 250 completed all three assessment waves and are included in the current analysis (53% female; 47% male). Approximately half (n = 129) of the subjects were deemed to be at increased risk for some form of personality disorder, while the remainder (n = 121) were not. A summary of a selection of subjects' demographic characteristics appears in Table 1.

As a prospective, multiwave study, it is important to delineate the precise schedule of assessments in the LSPD. At each wave of data collection, the study subjects (a) were interviewed by experienced, trained clinicians for both Axis I and *all* Axis II conditions as noted above and (b) assessed on a wide variety of self-report measures of personality disorder, personality, temperament, sex role conformity, and mental state (anxiety and depression). It is

important to note that *all* study staff were blind as to the group membership status (i.e., Possible Personality Disorder [PPD] vs. No Personality Disorder [NoPD]) of the study subjects throughout the study period. Moreover, for the interview-based assessments, the same interviewer *never* saw the same subject more than once during the three waves of data collection. The subjects were seen for their wave I, II, and III assessments in their 1st, 2nd, and 4th years in college. Details regarding the assessment schedule and technical features are given elsewhere [35].

Initial Personality Disorder Screening Measure (Young Adulthood, Age 17/18)

IPDE DSM-III-R Screen (IPDE-S) [39]. The *IPDE-S* is a 250-item self-administered True-False PD screening inventory developed by A. W. Loranger. The diagnostic efficiency and psychometric properties of the IPDE-S, in a two-stage screen application, were generally excellent and have been described previously [39]. The grouping variable (PPD vs. NoPD) for the study subjects is *not* a focus of the current set of analyses, but it is noted as it is a study design feature.

Clinical and Individual Difference Measures (Emerging Adulthood, Ages 18–21)

Axis II Measurement: NPD Features. International Personality Disorder Examination (IPDE). The IPDE is the well-known semi-structured interview procedure that assesses both *DSM-IV* and *ICD-10* PD features [40–42] and was used in the WHO/ADAMHA-sponsored *International Pilot Study of Personality Disorders* [42]. The IPDE has excellent psychometric properties and has been shown to be robust as a diagnostic assessment tool for PD's even in the face of mental state (anxiety, depression) changes. The *DSM-III-R* criteria were assessed in this study as these were the criteria in effect at the time the LSPD was undertaken. I note that the *DSM-III-R* and the later *DSM-IV/DSM-5* criteria bear considerable resemblance to one another, and the fundamental PD constructs are the same in both nomenclatures. Clinically experienced interviewers received intensive training in IPDE administration and scoring by Dr. Armand W. Loranger and were supervised throughout the project by the author M.F.L., who was blind to the participants' identity, putative PD status, and all prior assessment information. The inter-rater reliability for IPDE assessments (based on intraclass correlation coefficients) was excellent at all three waves, ranging between 0.84 and 0.92 for all PD dimensions. The interviewers were: (a) blind to the putative PD group status of the participants, (b) blind to all prior LSPD PD assessment data, and (c) the same interviewer never assessed the same subject more than once. I defined the SZDPD outcome variable as the dimensional score total for the disorder derived from the IPDE.

Axis I Disorders: Diagnostic Measurement

Structured Clinical Interview for DSM-III-R: Nonpatient Version (SCID-NP) [43]. This is a semistructured *DSM-III-R* [44] Axis I clinical interview for use with nonpatients. These data are not reported here as they are not germane to the present project.

Temperament Measure: Early Adulthood Assessment of Childhood Temperaments. The EAS Adult Temperament Scale [45], is a well-known 20-item self-report measure, for use with adults, of the three major temperament constructs, emotionality (E) (which breaks down further to "fear," "distress," and "anger" sub-dimensions), activity (A), and sociability (S), thought to underlie personality processes in children and adults. The EAS requires a subject to read 20 statements and decide how characteristic or typical each statement is of himself or herself on a 5-point scale. Factor analysis of the childhood and adult versions of the EAS has revealed a consistent structure that supports the basic three temperaments model of Buss and Plomin [45] (see also [46, 47]) and test-retest reliability of the Adult EAS is acceptable. Finally, as noted above, the major components of the EAS have been shown to be highly heritable [36]. The EAS was administered at all three study waves, however, only the wave I temperament scores are used in this analysis so as to be methodologically consistent with the proximal process measure that was only assessed at wave I.

Social/Interpersonal Process Measure: Proximal Process Assessment (Early to Middle-Childhood, Ages 5–12. At the time LSPD data collection commenced, fall 1991, there was no existing measure of a *proximal process* construct such as that hypothesized by Bronfenbrenner [31]. In fact, Bronfenbrenner's *person-process-context-time* model [31, 33] that included the proximal process construct was not fully formed or articulated. Therefore, in consultation with Urie Bronfenbrenner, I developed a measure of proximal process – provisionally designated the *proximal process index* – that consisted of four (4) focal questions (see online suppl. Material 1; see www.karger.com/doi/10.1159/000524796 for all online suppl. material). These four questions were designed to be asked within a semistructured interview format. The use of an interviewer-based assessment in which questions were asked, examples were provided, clarifications were sought was seen as prefer-

Table 1. Descriptive statistics for selected demographic characteristics of participants in the *LSPD* sample (*N* = 250)

	Father, %	Mother, %
Parental education		
1–8 years	1.6	0.8
9–11 years	2.4	2.8
12 years	8.4	15.2
13–15 years	16.0	20.4
16+ years	68.8	58.8
Not available	2.8	2
Parental occupation		
Laborer/service	2.0	2.4
Operatives (machine)	2.8	1.2
Craftsman/foreman	3.2	1.6
Clerical/sales	4.0	16.8
Management/official	26.8	12.8
Professional/technical	52.4	42.0
Not available or homemaker	8.8	23.2
Race	%	
African-American	3.6	
Latin-Hispanic	4.8	
Caucasian-Anglo	72.0	
Asian-Pacific Islander	17.2	
Native American	0.8	
Other	1.6	

Table 2. Descriptive statistics for the principal study variables (n = 250)

Variable	Mean	SD
Proximal process	3.77	0.56
EAS distress – W-I	2.15	0.83
EAS fearfulness – W-I	2.17	0.75
EAS anger – W-I	2.35	0.79
EAS activity – W-I	3.18	0.76
EAS sociability – W-I	3.64	0.79
IPDE Nar PD – W-I	1.39	2.51
IPDE Nar PD – W-II	0.67	1.76
IPDE Nar PD – W-III	0.43	1.10

Proximal process refers to proximal process score. EAS sociability, EAS fear, EAS distress, EAS anger, and EAS activity refer, respectively, to temperament dimensions as assessed by the Buss and Plomin [45] EAS Adult Temperament Scale assessed at wave I. Nar PD – W-I, Nar PD – W-II, and Nar PD – W-III refer to the dimensional score for *DSM-III-R* NPD as assessed using the *IPDE* [40] across three prospective study waves.

Table 3. Correlations between predictor variables and narcissism personality disorder symptoms at three longitudinal assessment waves of the *LSPD* (N = 250)

	Nar PD-W-I	Nar PD-W-II	Nar PD-W-III
EAS sociability	−0.16**	−0.15*	−0.18**
EAS distress	0.28**	0.13*	0.14*
EAS anger	0.32**	0.24**	0.19**
EAS activity	0.15*	0.03	−0.02
EAS fear	0.19**	0.05	0.14*
Proximal process	−0.19**	-0.17**	−0.16*

Values are Pearson product-moment correlation coefficients based on 250 cases (all subjects have complete data). Proximal process refers to proximal process score. EAS sociability, EAS fear, EAS distress, EAS anger, and EAS activity refer to temperament dimensions as assessed by the Buss and Plomin [45] EAS Adult Temperament Scale. Nar PD-W-I, Nar PD-W-II, and Nar PD-W-III refer to the dimensional score for *DSM-III-R* NPD as assessed using the *IPDE* [40] across three prospective study waves. * Significance: $p < 0.05$. ** $p < 0.01$.

able to simple self-report approach. In short, the interviewer could use his or her skills to elicit more information from the subjects. Inter-rater reliability of the proximal process measure was excellent (intraclass correlation coefficient = 0.95). Content validity, which is typically assessed via expert judgment, of the proximal process measure was deemed acceptable by Professor Bronfenbrenner. Criterion relations involving the positive impact of proximal process on personality disorder symptom level and improvement as well as the development of the affiliation system have been reported in prior publications [48, 49]. The proximal process measure was administered only at wave I.

Assessment of these proximal process items did rely upon retrospective recall on the part of the subjects, with a focus on the childhood/pre-teen years (covering the 5- to 12-year age span). The benefits of interviewer-based assessments for retrospective reports have been described [50–52]. Clearly, the potential shortcomings of retrospective recall are known [50–52] and give some pause in the service of caution in interpreting observed associations between childhood processes and other factors (e.g., adversity) and later psychopathology. However, at the same time, it is just as important not to "throw out the baby with the bathwater" in considering the potential value of associations discovered between retrospectively assessed input variables and later output variables [50–52]. This is so because the common methodological complaints about retrospective reports – namely, lower reliability (and by implication lower validity) – are probably overblown or exaggerated [50]. Importantly, the benefits of *interviewer-based* assessments for retrospective reports have been described [50–52].

Statistical Analyses

The primary statistical analytic approaches used in this study were correlational analysis and multiple regression analysis [53]. The level of NPD features was correlated with the EAS dimensions as well as the proximal process measure. This analysis was done for the NPD scores at waves I, II, and III of the LSPD dataset. Multiple regression analysis was done such that all EAS temperament dimensions and the proximal process index were entered simultaneously (forced entry) as a single block in the prediction of NPD features for each of the three study waves. The semipartial (part) correlation coefficient was used to assess the relatively unique contribution of each variable in the prediction of NPD features in the regressions as per Darlington [53].

Results

The analyses for this report are based on the 250 subjects that have complete data across all three waves of LSPD. Of the original 258 LSPD subjects, 5 PPD and 3 NoPD subjects did not complete all three waves of data collection, 2 of these noncompleting subjects died in accidents during the study, and one in each subject group. The study period covered in this phase of the LSPD corresponds to what is often termed "emerging adulthood," the years from 18 through 21. Descriptive statistics for the principal variables under consideration are found in Table 2. The study subjects presented a wide range of NPD symptoms ranging from none to 14 as assessed by the IPDE dimensional scoring system.

The primary objective of the present study was to determine if level proximal process as well as level of temperament factors were associated with level NPD features in longitudinal perspective. It is important to note that

Table 4. Regression analysis results for regressors in the prediction of NPD symptoms at three longitudinal assessment waves of the *LSPD* (*N* = 250)

Regressors	β	*t*	*p*	*pr*	*R*	R^2
Assessment wave I – Narcissistic PD						
Process	−0.21	−3.52	0.001	−0.20		
EAS-distress	0.18	2.87	0.004	0.17		
EAS-anger	0.23	3.58	0.001	0.21		
EAS-activity	0.11	1.94	0.054	0.11	0.43	0.18
Assessment wave II – Narcissistic PD						
Process	−0.17	−2.76	0.006	−0.17		
EAS-distress	0.05	0.70	0.49	0.04		
EAS-anger	0.21	3.17	0.002	0.19		
EAS-activity	0.01	0.11	0.91	0.01	0.29	0.09
Assessment wave III – Narcissistic PD						
Process	−0.16	−2.49	0.013	−0.15		
EAS distress	0.08	1.15	0.25	0.07		
EAS anger	0.16	2.30	0.022	0.14		
EAS activity	−0.04	−0.58	0.57	−0.04	0.25	0.06

Process refers to proximal process score. EAS distress, EAS anger, and EAS activity refer to temperament dimensions as assessed by the Buss and Plomin [45] EAS Adult Temperament Scale. *DSM-III-R* NPD features at wave-I, wave-II, and wave-III as assessed using the *IPDE* [40]. β, standardized coefficients; *t*, *t* statistic, *p*, *p* value, *pr*, part correlation, *R*, multiple correlation; R^2, squared multiple correlation.

this hypothesis concerns *levels* of NPD features; it does not address the issue of change over time (which would tap an entirely different set of questions). Moreover, this study also sought to establish if *both* proximal process and temperament factors independently predicted the level of NPD features and if these predictive relationships maintained over time. The correlational analyses (Table 3) provided clear evidence that lower levels of proximal process in earlier life were associated with higher levels of NPD features in early adulthood and that these associations maintained over time. Similarly, the correlational analyses provided clear support for the supposition that temperament factors are associated with NPD features in early adulthood and that these associations maintained over time. This pattern of results is consistent with the theoretical conjectures of Kernberg [6], who posited the either dispositional (i.e., temperament factors, especially aggression) or parenting failures were causes of NPD, and they are also congenial with the proposal by Kohut [7, 8] regarding parenting failures in the emergence of NPD in adulthood.

The regression analyses were conducted in three stages. First, to reduce the number of regressors, an analysis was conducted to see which temperament dimensions were most closely associated with wave I NPD features when all temperament factors were considered in a multivariate manner. This analysis pointed to the importance of the EAS dimensions of anger, distress, and activity (all betas $p < 0.05$). Second, these three EAS dimensions along with proximal process were then explored in a set of regressions in which they were modeled as predictors of NPD features at the three different time points in prospective longitudinal perspective (i.e., wave I, wave II, and wave III taken separately) (Table 4). What emerged clearly from these three sets of regressions, one for each study wave, was a compelling pattern that both proximal process and EAS anger were predictive of NPD features at three different assessment waves (i.e., over time). In short, when considered in the same block of regressors in which all variables were forced to enter the prediction of NPD features, lower levels of proximal process and higher levels of EAS anger were significant predictors of increased levels of NPD features. In short, both proximal process (a social relations/parenting related construct) as well as EAS anger (a dispositional construct) were predictive of NPD features, even when considered statistically at the same time. Third, a set of regressions were conducted in which interaction terms for proximal process and each of the EAS dimensions were entered into the analyses after the main effects were entered. No interaction terms were significant for the prediction of NPD features and thus these results are not reported here to conserve space (available upon request).

Two additional sets of regressions were conducted to illuminate the research question fully. One supplementary set of regressions used all EAS variables (instead of the reduced set reported here) and was conducted for each study wave parallel to those reported above. The results of this set of analyses were essentially identical to main regression analyses reported above, namely proximal process *and* EAS anger emerged as the significant predictors of the level of NPD features at each of the three study waves. A second supplementary set of regressions used the reduced EAS variable set along with the proximal process variable and alternated entry of the variables as blocks. First, one subset of regressions entered the temperament variables as a block, followed by the proximal process variable in its own block in the prediction of NPD features. Next, another subset of regressions entered the proximal process variable as a block first, followed by the block of temperament variables. In short, in both subsets of regressions, whether entered first or second as blocks, *both* temperament and proximal process blocks were statistically significant for all NPD variables (W-I, W-II, W-III).

In order to conduct a sensitivity analysis for a construct plausibly related to NPD, the role of biological sex was considered (note this is sex, not gender) and used in a supplementary set of correlations and regressions. What emerged was that maleness was indeed associated with higher levels of NPD features in both the correlational analyses as well as two of the three regression models. However, inclusion of the sex variable in the regression analyses did not alter the results in any meaningful manner, such that proximal process and anger remained strong predictors of NPD features even with sex included in the block of regressors.

As noted above, the current research focus was on the relationships between *levels* of the variables in question, namely proximal process, temperament dimensions at wave I, and NPD features at waves I, II, and III. This study did not seek to address change in NPD features over time. Another statistical approach to the levels hypotheses under consideration here would be: (a) to compute individual growth curves (IGCs) [54] for each subject with NPD features defined as a function of time (Level I) and (b) determine if proximal process or temperament variables at wave I would predict the initial level (intercept) component of the IGCs (Level II) using a hierarchical linear modeling approach [38]. Such an analysis was conducted, and the results paralleled the regression findings reported above; namely, (lower) proximal process ($p < 0.001$) and (higher) EAS anger ($p < 0.001$) were both significantly associated with the initial level (intercept) of NPD IGCs.

Discussion

The results of this study, drawn from the prospective multiwave longitudinal study known as the *LSPD*, provide clear evidence that *both* proximal process and temperament factors, especially anger, are predictive of NPD features. The observed correlations of the index of proximal process and temperament (notably anger, distress, and activity) with (later and continuing) NPD features suggest that the presence of an engaged parent/caregiver in childhood and/or diminished temperamental anger (dispositional) were predictive of lower levels of NPD over time. The unique power of prospective longitudinal data is that they allow one to assess whether cross-sectional associations (wave I relations) hold up over time (wave II and III relations), a methodological perspective that allows one more leverage in the discussion of developmental phenomena. The results from this analysis clearly indicate that both proximal process and temperament maintain their relatively unique contributions to NPD features over the early adult years. However, there is some evidence in the data that the amount of variance predicted by the proximal process and EAS anger variables decreased over time. Although still significant as predictors of NPD features, the R^2 on the regression models decreases from 0.18 (wave I) to 0.06 (wave III).

As we have argued for over 20 years, namely that personality pathology is the emergent product of interacting neurobehavioral systems (manifesting as temperament and/or personality factors) in concert with environmental inputs [1–4], these results point to the joint importance of temperament and proximal process (i.e., environmental) inputs in the determination of NPD. Such a view is also consistent with what is known about the joint contributions of both genetic influences and environmental influences that are implicated in personality disorders, including narcissism, based on twin studies focused on either the traditional DSM (e.g., [28]) or the more recent Alternative Model of Personality Disorder [29] taxonomies. Finally, psychodynamic thinkers have long speculated that NPD can develop from either poor parenting influences [6–8, 55] or temperamental predispositions tilting toward aggression [6, 55]; these results are consistent with such theory.

An empirical literature has begun to emerge from follow-up studies supporting the association between the absence of nurturant parenting styles and/or the presence of emotional neglect/emotional abuse in the early childhood histories of those adults that were seen as having high levels of narcissism, with some likely presenting

NPD later in the life course [23]. This association was observed in studies that used retrospective as well as some prospective data collection methods [23]. There has been considerable speculation regarding the mechanism or process involved, typically drawn from intensive psychotherapeutic treatment, but a large-scale empirical study in this area remains sparse. However, beyond the establishment of the basic association between the parenting factors and later narcissistic pathology, little is known empirically about the *mechanism* or *process* by which a child might proceed from the quality of parenting experienced and later narcissistic PD. The present study has added some empirical support to the importance of both psychosocial and dispositional factors in the development of NPD. The proximal process construct, it is argued, goes beyond something akin to nurturance or warmth, rather it points to an active process of ever-increasing psychological sophistication and engagement between a child and a parent (or caregiver) that ostensibly yields healthier psychological development [31]. The current findings differ from those of Brummelman et al. [56] who found support of parental overvaluation as a determinant of childhood narcissism (as opposed to lack of parental warmth). However, that study focused on relatively young children (7–11 years old at baseline) that were followed for 2 years and assessed normative narcissism (not NPD). A focus, therefore, on pathological narcissism such as we studied (i.e., NPD) may point to different parental/environmental inputs. The present findings are also consistent with a literature that broadly links childhood adversities with later personality pathology [57, 58].

A striking feature of the results was the consistency with which anger emerged as a predictor of NPD features. Indeed, anger was a significant predictor of NPD features across all three LSPD study waves. It has long been known that individuals displaying NPD can be prone to angry, even rageful, outbursts in response to a variety of interpersonal stimuli [6, 7]. Moreover, there is a growing empirical literature that clearly relates anger to pathological narcissism [59–61] and the current results, which views anger as a component of temperament, are consistent with that literature.

A number of features of this study should be kept in mind when considering these results. For example, one might wonder if the effects of proximal processes reflected in this study sample were maximally impactful [33], recalling that Bronfenbrenner [31, 33] argued that the effects of proximal processes would be greatest in conditions characterized by severe disadvantage. The LSPD study sample consisted of individuals that were not typically subject to such severe disadvantage. The sample is largely Caucasian hailing from educated parents working in higher level jobs/professions. In other words, the majority of the study subjects in the LSPD were *not* drawn from the outer edges, so to speak, of the socioeconomic range. Rather their average expectable childhood rearing environments consisted of influences associated with relatively mainstream environments. More than 80% have some college or beyond (mothers and fathers), of those working most have managerial or professional occupations. Thus, it could very well be that the associations between the proximal process index and narcissistic pathology in the LSPD sample actually *underestimate* the power of these relationships, owing to the restriction of range on rearing environment variability among the subjects. Another feature of this sample is that the assessments were conducted during the early adult years, covering a 4-year period. One might consider whether the effects of proximal processes on narcissistic pathology will persist further into adulthood as suggested by this model. One could, of course, ponder whether temperamental factors will continue to exert their effects into adulthood. These empirical questions can be answered and will be a focus of data analyses associated with the planned wave IV assessments of the LSPD subjects, who are now in their late 40s.

Some other issues must also be kept in mind regarding potential limitations. The assessments of proximal process levels were done using a retrospective methodological approach. Although substantial methodological reviews [50] have supported the general utility of the retrospective recall approach, a degree of caution in the evaluation of data gathered using this method is reasonable. For example, could it be that narcissistically impaired young adults recall erroneously a childhood characterized by the absence of interpersonal connections with parents/caregivers? Another concern is that the EAS was used to collect information on temperament in these subjects. Although it is conjectured that the EAS, when administered in adulthood, can actually tap temperamental dispositions that were likely in place in childhood [45], one can reasonably ask if these subjects would have been characterized similarly in terms of temperament if they had been studied as children.

These results are offered in the context of discovery and their heuristic potential is emphasized. The results of the present study suggest an important role for *both* proximal process and anger (i.e., temperament disposition) in relation to NPD features assessed over time, beginning in early adulthood. It is important to emphasize, however,

that the present model and set of findings are *not* suggesting that the etiology of NPD is purely environmental in origin, rather that the level of proximal process present in the early childhood years may impact the developing personality system that eventually manifests itself in NPD. The same can be said of temperament. In sum, the take-home message, consistent with the theoretical speculation of Kernberg [6, 55] and congenial to the views of Kohut [7, 8], is that both social relationships as indexed through proximal processes and temperamental (dispositional) factors (notably anger) are likely important in the development, emergence, and continuation of NPD over time.

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Statement of Ethics

The *Longitudinal Study of Personality Disorder* (LSPD) was reviewed and received full approval from the Cornell University Institutional Review Board for Human Participant Research (IRB) prior to its inception in 1991. All subjects provided written informed consent to participate as adults in the LSPD at the time of study entry. The LSPD adhered to all ethical principles as specified by the American Psychological Association (USA), which accord with the World Medical Association Declaration of Helsinki.

Conflict of Interest Statement

No conflicts to report.

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Author Contributions

Dr. Lenzenweger is the sole author and responsible for all aspects of this paper.

Data Availability Statement

The data from the LSPD are not available for distribution due to confidentiality agreements in place since the inception of the study.

References

1 Lenzenweger MF, Depue RA. Personality disturbances as emergent phenomena reflective of underlying neurobehavioral systems: beyond dimensional measurement, phenotypic trait descriptors, and factor analysis. Psychopathology. 2020;53(3):213–20.
2 Lenzenweger MF, Depue RA. Toward a developmental psychopathology of personality disturbance: a neurobehavioral dimensional model incorporating genetic, environmental, and epigenetic factors. In: Cicchetti D, editor. Maladaptation and psychopathology. 3rd ed. Developmental psychopathology. New York: Wiley; 2016. Vol. 3. p. 1–31.
3 Depue RA, Lenzenweger MF. A neurobehavioral dimensional model of personality disorders. In: Livesley WJ, editor. The handbook of personality disorders. New York: Guilford; 2001. p. 136–76.
4 Depue RA, Lenzenweger MF. A neurobehavioral model of personality disturbance. In: Lenzenweger MF, Clarkin JF, editors. Major theories of personality disorder. 2nd ed. New York: Guilford; 2005. p. 391–453.
5 Freud S. On narcissism: an introduction. In: Strachey J, translator, editors. The standard edition of the complete psychological works of Sigmund Freud. London: Hogarth; 1914. Vol. 14. p. 111–40.
6 Kernberg OF. Factors in the psychoanalytic treatment of narcissistic personalities. J Am Psychoanal Assoc. 1970 Jan;18(1):51–85.
7 Kohut H. The analysis of the self. New York: International Universities Press; 1971.
8 Kohut H. The restoration of the self. New York: International Universities Press; 1977.
9 Cain NM, Pincus AL, Ansell EB. Narcissism at the crossroads: phenotypic description of pathological narcissism across clinical theory, social/personality psychology, and psychiatric diagnosis. Clin Psychol Rev. 2008 Apr 1;28(4):638–56.
10 Pincus AL, Lukowitsky MR. Pathological narcissism and narcissistic personality disorder. Annu Rev Clin Psychol. 2010 Apr 27;6:421–46.
11 Ackerman RA, Donnellan MB, Wright AGC. Current conceptualizations of narcissism. Curr Opin Psychiatry. 2019 Jan 1;32(1):32–7.
12 Caligor E, Levy KN, Yeomans FE. Narcissistic personality disorder: diagnostic and clinical challenges. Am J Psychiatry. 2015 May 1;172(5):415–22.
13 Krizan Z, Herlache AD. The narcissism spectrum model: a synthetic view of narcissistic personality. Pers Soc Psychol Rev. 2018 Feb;22(1):3–31.
14 Miller JD, Back MD, Lynam DR, Wright AGC. Narcissism today: what we know and what we need to learn. Curr Dir Psychol Sci. 2021 Dec 1;30(6):519–25.
15 Miller JD, Lynam DR, Hyatt CS, Campbell WK. Controversies in narcissism. Annu Rev Clin Psychol. 2017 May 8;13:291–315.
16 Rosenthal SA, Hooley JM. Narcissism assessment in social – personality research: does the association between narcissism and psychological health result from a confound with self-esteem? J Res Pers. 2010 Aug 1;44(4):453–65.

17 Lenzenweger MF, Clarkin JF, Caligor E, Cain NM, Kernberg OF. Malignant narcissism in relation to clinical change in borderline personality disorder: an exploratory study. Psychopathology. 2018;51(5):318–25.
18 Stolorow RD. Toward a functional definition of narcissism. Int J Psychoanal. 1975;56:179–85.
19 Meehl PE. Specific etiology and other forms of strong influence: some quantitative meanings. J Med Philos. 1977 Jan 1;2(1):33–53.
20 Miller A. Depression and grandiosity as related forms of narcissistic disturbances. Int Rev Psychoanal. 1979;6:61–76.
21 Miller A. Prisoners of childhood: the drama of the gifted child and the search for the true self. New York: Basic Books; 1981.
22 Horton RS, Bleau G, Drwecki B. Parenting narcissus: what are the links between parenting and narcissism? J Pers. 2006 Apr;74(2):345–76.
23 Horton RS. Parenting as a cause of narcissism: empirical support for psychodynamic and social learning theories. In: Campbell WK, Miller JD, editors. The handbook of narcissism and narcissistic personality disorder: theoretical approaches, empirical findings, and treatments. New York: Wiley; 2011. p. 181–90.
24 Shiner RL, DeYoung C. The structure of temperament and personality traits: a developmental perspective. In: Zelazo PD, editor. The oxford handbook of developmental psychology. Self and other. New York: Oxford University Press; 2013. Vol. 2. p. 113–41.
25 Campbell WK, Miller JD. Narcissistic personality disorder and the five-factor model: delineating narcissistic personality disorder, grandiose narcissism, and vulnerable narcissism. In: Widiger TA, Costa PT, editors. Personality disorders and the five-factor model of personality. Washington, DC: American Psychological Association; 2013. p. 133–45.
26 American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-5). 5th ed. Arlington, VA: American Psychiatric Publishing; 2013.
27 Jackson DN, Vernon PA. Genetic and environmental contributions to dimensions of personality disorder. Am J Psychiatry. 1993 Dec;150(12):1826–31.
28 Kendler KS, Aggen SH, Czajkowski N, Røysamb E, Tambs K, Torgersen S, et al. The structure of genetic and environmental risk factors for DSM-IV personality disorders: a multivariate twin study. Arch Gen Psychiatry. 2008 Dec 1;65(12):1438–46.
29 Wright ZE, Pahlen S, Krueger RF. Genetic and environmental influences on diagnostic and statistical manual of mental disorders-(DSM-5) maladaptive personality traits and their connections with normative personality traits. J Abnorm Psychol. 2017 May;126(4):416–28.
30 Markon KE, Krueger RF, Bouchard TJ Jr, Gottesman II. Normal and abnormal personality traits: evidence for genetic and environmental relationships in the Minnesota study of twins reared apart. J Pers. 2002 Oct;70(5):661–93.
31 Bronfenbrenner U, Morris P. The ecology of developmental processes. 5th ed. In: Lerner RM, editor. Handbook of child psychology: theoretical models of human development. New York: Wiley; 1998. p. 993–1028.
32 Vygotsky LS. Mind in society: development of higher psychological processes. Cambridge, MA: Harvard University Press; 1978.
33 Bronfenbrenner U, Ceci SJ. Nature-nurture reconceptualized in developmental perspective: a bioecological model. Psychol Rev. 1994 Oct;101(4):568–86.
34 Cicchetti D. The impact of social experience on neurobiological systems: illustration from a constructivist view of child maltreatment. Cognitive Dev. 2002 Sep 1;17(3–4):1407–28.
35 Lenzenweger MF. The longitudinal study of personality disorders: history, design considerations, and initial findings. J Pers Disord. 2006 Dec;20(6):645–70.
36 Plomin R. Development, genetics, and psychology. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc; 1986.
37 Lenzenweger MF. Stability and change in personality disorder features: the longitudinal study of personality disorders. Arch Gen Psychiatry. 1999 Nov 1;56(11):1009–15.
38 Lenzenweger MF, Johnson MD, Willett JB. Individual growth curve analysis illuminates stability and change in personality disorder features: the longitudinal study of personality disorders. Arch Gen Psychiatry. 2004 Oct 1;61(10):1015–24.
39 Lenzenweger MF, Loranger AW, Korfine L, Neff C. Detecting personality disorders in a nonclinical population: application of a 2-stage procedure for case identification. Arch Gen Psychiatry. 1997 Apr 1;54(4):345–51.
40 Loranger AW. International personality disorder examination: DSM-IV and ICD-10 interviews. Odessa, FL: Psychological Assessment Resources; 1999.
41 Loranger AW, Lenzenweger MF, Gartner AF, Susman VL, Herzig J, Zammit GK, et al. Trait-state artifacts and the diagnosis of personality disorders. Arch Gen Psychiatry. 1991 Aug 1;48(8):720–8.
42 Loranger AW, Sartorius N, Andreoli A, Berger P, Buchheim P, Channabasavanna SM, et al. The international personality disorder examination: the World Health Organization/alcohol, drug abuse, and mental health administration international pilot study of personality disorders. Arch Gen Psychiatry. 1994 Mar 1;51(3):215–24.
43 Spitzer RL, Williams JB, Gibbon M, First MB. User's guide for the structured clinical interview for DSM-III-R: SCID. Arlington, VA: American Psychiatric Association; 1990.
44 American psychiatric association diagnostic and statistical manual of mental disorders (DSM-III-R). 3rd ed. Revised. Arlington, VA: American Psychiatric Association; 1987.
45 Buss AH, Plomin R. Temperament: early developing personality traits. Hillsdale, NJ: Erlbaum; 1984.
46 Mathiesen KS, Tambs K. The EAS temperament questionnaire – factor structure, age trends, reliability, and stability in a Norwegian sample. J Child Psychol Psychiatry. 1999 Mar;40(3):431–9.
47 Naerde A, Røysamb E, Tambs K. Temperament in adults – reliability, stability, and factor structure of the EAS temperament survey. J Pers Assess. 2004 Feb 1;82(1):71–9.
48 Lenzenweger MF. A source, a cascade, a schizoid: a heuristic proposal from the longitudinal study of personality disorders. Dev Psychopathol. 2010 Nov;22(4):867–81.
49 Hallquist MN, Lenzenweger MF. Identifying latent trajectories of personality disorder symptom change: growth mixture modeling in the longitudinal study of personality disorders. J Abnorm Psychol. 2013 Feb;122(1):138–55.
50 Brewin CR, Andrews B, Gotlib IH. Psychopathology and early experience: a reappraisal of retrospective reports. Psychol Bull. 1993 Jan;113(1):82–98.
51 Rutter M, Maughan B. Psychosocial adversities in childhood and adult psychopathology. J Pers Disord. 1997 Mar;11(1):4–18.
52 Maughan B, Rutter M. Retrospective reporting of childhood adversity: issues in assessing long-term recall. J Pers Disord. 1997 Mar;11(1):19–33.
53 Darlington RB. Regression and linear models. New York, NY: McGraw-Hill; 1990.
54 Rogosa DR, Willett JB. Understanding correlates of change by modeling individual differences in growth. Psychometrika. 1985 Jun 1;50(2):203–28.
55 Kernberg OF. The almost untreatable narcissistic patient. J Am Psychoanal Assoc. 2007 Jun;55(2):503–39.
56 Brummelman E, Thomaes S, Nelemans SA, De Castro BO, Overbeek G, Bushman BJ. Origins of narcissism in children. Proc Natl Acad Sci U S A. 2015 Mar 24;112(12):3659–62.
57 Roberts A, Yang M, Zhang T, Coid J. Personality disorder, temperament, and childhood adversity: findings from a cohort of prisoners in England and Wales. J Forensic Psychiatry Psychol. 2008 Dec 1;19(4):460–83.
58 Shiner RL, Masten AS, Roberts JM. Childhood personality foreshadows adult personality and life outcomes two decades later. J Pers. 2003 Dec;71(6):1145–70.
59 Russ E, Shedler J, Bradley R, Westen D. Refining the construct of narcissistic personality disorder: diagnostic criteria and subtypes. Am J Psychiatry. 2008 Nov;165(11):1473–81.
60 Glover N, Miller JD, Lynam DR, Crego C, Widiger TA. The five-factor narcissism inventory: a five-factor measure of narcissistic personality traits. J Pers Assess. 2012 Sep 1;94(5):500–12.
61 Williams R. Anger as a basic emotion and its role in personality building and pathological growth: the neuroscientific, developmental and clinical perspectives. Front Psychol. 2017 Nov 7;8:1950.