

1 Quantitative Voice Analysis Reveals Context-Dependent Expression of Personality
2 Pathology: A Bayesian Multilevel Ambulatory Assessment Study

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Abstract

16 This study examined whether vocal acoustics can capture context-dependent expression of
17 personality pathology during naturalistic stress. Female university students ($N = 119$)
18 provided voice recordings at baseline, immediately before, and after a course examination,
19 while personality pathology traits (PID-5) were assessed via ecological momentary
20 assessment (EMA) over 2.5 months. Exam stress produced dissociable vocal changes:
21 fundamental frequency (F0) increased by 3.27 Hz, indicating heightened autonomic arousal,
22 while normalized noise energy (NNE) decreased by 0.79 dB, suggesting compensatory
23 phonatory control. Personality traits showed domain- and parameter-specific moderation:
24 Negative Affectivity amplified stress-induced F0 increases during anticipatory stress,
25 Antagonism was associated with sustained F0 elevation during recovery, and Psychoticism
26 uniquely modulated voice quality (NNE) during the post-stressor period. Detachment and
27 Disinhibition showed minimal moderation, consistent with theoretical predictions
28 distinguishing arousal sensitivity from behavioral control. Methodologically, brief
29 EMA-based personality assessment matched comprehensive baseline questionnaires in
30 predictive accuracy while yielding more precise moderation estimates. These findings
31 support transactional models of personality pathology and indicate that multimodal
32 ambulatory assessment, combining passive acoustic sensing with intensive self-report, can
33 capture how maladaptive traits shape real-world stress responses.

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Keywords: keywords

35

Word count: X

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38 **Introduction**

39 Personality pathology is increasingly conceptualized as a context-sensitive
40 phenomenon, wherein maladaptive traits are not expressed uniformly but are
41 modulated—amplified, attenuated, or qualitatively transformed—by situational demands
42 (Hopwood et al., 2022; Wright & Simms, 2016). Contemporary personality theories
43 converge on the idea that stable individual differences are revealed not through static
44 behavior, but through systematic patterns of response to situational cues. For instance,
45 Mischel and Shoda's (1995) *Cognitive-Affective Personality System* frames personality as a
46 stable set of situation-contingent “if–then” behavioral signatures, reflecting consistent
47 individual differences in reactions to specific psychological features of situations. Similarly,
48 Fleeson's (2001) *Whole Trait Theory* conceptualizes traits as density distributions of
49 momentary states, distinguishing between stable mean levels and dynamic,
50 situation-contingent variability stemming from individual differences in contextual
51 responsiveness. Applied to personality pathology, these frameworks suggest that
52 maladaptive traits not only predict outcomes but also moderate the influence of
53 environmental stressors, thereby shaping domain-specific physiological and behavioral
54 responses.

55 Despite this strong theoretical consensus, empirical methods for capturing
56 person–situation dynamics in naturalistic contexts remain limited. Although the field
57 broadly agrees that “context matters,” research on personality pathology has struggled to
58 operationalize contextual processes as they unfold in daily life (Wright et al., 2019).
59 Traditional assessment approaches conceptualize traits as decontextualized dispositions
60 measured at single time points, largely ignoring the temporal dynamics and situational
61 contingencies through which pathology is expressed. This mismatch between theory, which

62 emphasizes person-environment interactions, and methodology, which relies on static,
63 decontextualized measurement, limits our understanding of personality pathology and our
64 ability to predict when, where, and for whom maladaptive patterns will emerge.

65 Ecological Momentary Assessment (EMA) has partially addressed this gap by
66 enabling repeated measurement of states and behaviors in real-world contexts, yielding
67 important insights into within-person variability and situational reactivity (Colpizzi, Trull,
68 Sica, Haney, & Caudelk, 2025; Trull & Ebner-Priemer, 2020). However, EMA remains
69 fundamentally constrained by its reliance on self-report. Individuals may lack access to
70 their own physiological stress responses, exhibit biased reporting, or show assessment
71 reactivity, whereby the act of self-monitoring alters the phenomena under study (Barta et
72 al., 2012). Moreover, repeated self-report imposes participant burden that limits sampling
73 density, reducing temporal resolution precisely when it is most needed, that is, during
74 acute stress episodes unfolding over minutes to hours. These limitations highlight the need
75 for objective, unobtrusive, and continuous indicators of stress reactivity that do not depend
76 on introspection.

77 Acoustic features of voice and speech can reflect autonomic arousal, emotional state,
78 and aspects of motor control that are only partly accessible to conscious awareness
79 (Scherer, 2003). Vocal production involves the coordinated activity of multiple
80 physiological systems across the body, including central and peripheral nervous system
81 processes, respiratory control, and laryngeal motor function. As a result, variations in
82 vocal acoustics provide an indirect window into psychophysiological states as they unfold in
83 real time. Fundamental frequency (F0), the acoustic correlate of perceived pitch, increases
84 reliably under stress due to sympathetic activation that tightens the vocal folds via
85 increased laryngeal muscle tension (Giddens et al., 2013). Voice quality parameters such as
86 normalized noise energy (NNE; indexing glottal noise) and jitter (cycle-to-cycle frequency
87 variability) capture aspects of phonatory stability and vocal control, and are sensitive to

88 both acute emotional arousal and chronic psychological states (Scherer et al., 2013).
89 Crucially, these features can be extracted from brief, naturalistic speech samples, making
90 voice acoustics a scalable, low-burden tool for ambulatory psychophysiological assessment.
91 Table 1 summarizes the definition, physiological basis, and psychological significance of the
92 three principal acoustic features discussed in this literature.

93 The relevance of vocal stress reactivity for personality pathology is grounded in
94 transactional models of person–situation interaction (Dietrich & Abbott, 2012). These
95 models posit that stable individual differences reflect differences in stress sensitivity, such
96 as heightened threat reactivity, impaired recovery, or dysregulated arousal, such that acute
97 stressors elicit disproportionate physiological responses in vulnerable individuals (Bolger &
98 Zuckerman, 1995). If personality pathology dimensions indeed index such vulnerabilities,
99 they should systematically shape physiological stress responses in everyday life.

100 The *Personality Inventory for DSM-5* (PID-5; Krueger et al., 2012) operationalizes
101 personality pathology across five broad domains representing maladaptive variants of the
102 Five Factor Model (Widiger & Crego, 2019). Given established links between Big Five
103 traits and physiological stress reactivity (Bibbey, Carroll, Roseboom, Phillips, & Rooij,
104 2013; Luo, Zhang, Cao, & Roberts, 2023), each PID-5 domain carries theoretical
105 implications for stress-related mechanisms. Negative Affectivity, the maladaptive
106 counterpart of Neuroticism, reflects emotional lability and anxiousness—features associated
107 with altered, though not uniformly heightened, autonomic and neuroendocrine stress
108 responses (Lahey, 2009). Detachment, the maladaptive variant of low Extraversion,
109 captures social withdrawal and anhedonia, with competing implications for stress reactivity:
110 reduced social buffering may impair recovery, while blunted emotional expression could
111 attenuate acute responses. Antagonism, characterized by callousness and interpersonal
112 dominance at the low pole of Agreeableness, may either attenuate stress reactivity through
113 reduced empathic engagement or prolong physiological arousal through conflictual social

interactions. Psychoticism involves cognitive and perceptual dysregulation and may manifest as poorly regulated physiological output even under baseline conditions. Finally, Disinhibition, the pathological extension of low Conscientiousness, is characterized by impulsivity and poor self-regulation, potentially compromising motor planning and behavioral control under stress. Critically, these domain-specific implications concern general physiological stress mechanisms (e.g., autonomic activation, neuroendocrine regulation, motor control); whether and how they manifest in the specific channel of vocal production remains an open empirical question that the present study addresses directly.

Despite these theoretically grounded predictions, empirical evidence on how personality pathology moderates physiological stress responses remains sparse, particularly outside the laboratory. Experimental stress paradigms offer strong internal validity but limited ecological relevance, as artificial stressors may not engage the same psychological processes as real-world evaluative threats. Conversely, field studies of daily stress rely almost exclusively on self-report, reintroducing the limitations noted above. What is needed is an approach that integrates (a) ecologically valid stressors with real consequences, (b) objective physiological, or physiology-linked, indicators independent of self-report, and (c) longitudinal designs capable of modeling both within-person dynamics and between-person moderation.

The present study addresses this need through a multimodal ambulatory assessment design centered on vocal stress markers. We leveraged the university examination period as a naturalistic, time-limited stressor with genuine stakes for participants, collecting vocal samples at baseline, immediately before the exam, and following exam completion. Personality pathology traits were assessed via EMA across a 2.5-month period. This design advances the field in three key ways: it operationalizes stress using a proximal real-world stressor rather than retrospective reports; it employs passive voice-based acoustic sensing to capture objective, physiology-linked vocal indicators; and it enables domain-specific

¹⁴⁰ tests of how personality pathology moderates both acute stress reactivity and post-stressor
¹⁴¹ recovery.

¹⁴² Our analyses were guided by two primary questions: (1) whether vocal parameters
¹⁴³ exhibit reliable stress-related changes in this naturalistic context, and (2) whether PID-5
¹⁴⁴ personality pathology domains moderate these changes. For the first question, we
¹⁴⁵ hypothesized that the examination period would be associated with increased F0,
¹⁴⁶ consistent with extensive evidence linking acute stress to elevated pitch via sympathetic
¹⁴⁷ activation (Giddens et al., 2013; Scherer, 2003). For voice quality parameters (NNE), we
¹⁴⁸ anticipated stress-related degradation reflecting reduced phonatory control, although prior
¹⁴⁹ findings suggest that voice quality parameters show smaller and less consistent stress
¹⁵⁰ effects than F0 (Giddens et al., 2013; Van Puyvelde, Neyt, McGlone, and Pattyn (2018)).

¹⁵¹ Regarding moderation by personality pathology traits, we adopted a largely
¹⁵² exploratory approach. The general implications for stress physiology outlined
¹⁵³ above—differential autonomic reactivity, impaired recovery, dysregulated motor
¹⁵⁴ output—generate plausible predictions at the level of vocal acoustics, but translating them
¹⁵⁵ into directional hypotheses requires evidence that personality traits modulate vocal
¹⁵⁶ production under stress specifically. Such evidence is scarce: although personality traits are
¹⁵⁷ known to shape physiological stress responses such as cortisol reactivity and heart rate
¹⁵⁸ variability (Luo et al., 2023), and emerging work indicates that these differences extend to
¹⁵⁹ vocal production under stress (Dietrich & Abbott, 2012), the specific moderating role of
¹⁶⁰ PID-5 domains on vocal stress markers has not been examined. The exception was
¹⁶¹ Negative Affectivity, for which we predicted amplified stress-induced F0 increases. This
¹⁶² prediction follows directly from the construct’s emphasis on threat sensitivity and
¹⁶³ emotional reactivity, features closely tied to sympathetic overactivation (Lahey, 2009;
¹⁶⁴ Ormel et al., 2013), combined with F0’s well-established sensitivity to autonomic arousal.
¹⁶⁵ For the remaining domains, competing theoretical predictions and inconsistent findings in

¹⁶⁶ adjacent literatures led us to treat moderation effects as empirical questions. For instance,
¹⁶⁷ Detachment has been linked to affective inertia and impaired affect repair (Kuppens et al.,
¹⁶⁸ 2010), but restricted emotional expression could alternatively attenuate vocal stress
¹⁶⁹ responses. Disinhibition, defined by impulsivity and poor behavioral control rather than
¹⁷⁰ emotional reactivity, shows weak associations with physiological stress markers despite
¹⁷¹ moderating subjective stress perception (Luo et al., 2023), suggesting it may not influence
¹⁷² the automatic autonomic mechanisms that drive F0 changes.

¹⁷³ Finally, we examined whether intensive EMA-based assessment of personality
¹⁷⁴ pathology could predict vocal stress responses as effectively as a comprehensive baseline
¹⁷⁵ assessment using the complete PID-5 questionnaire. Rather than contrasting different
¹⁷⁶ instruments, this comparison focuses on alternative measurement strategies within the
¹⁷⁷ same trait framework—brief, repeated assessments in daily life versus a full-length,
¹⁷⁸ cross-sectional assessment. If intensive longitudinal measurement can recover stable trait
¹⁷⁹ variance through explicit modeling of measurement error and occasion-specific fluctuations,
¹⁸⁰ EMA-based approaches may offer a more efficient and scalable alternative to traditional
¹⁸¹ questionnaire administration, an issue of increasing importance in high-burden ambulatory
¹⁸² research designs.

¹⁸³ By integrating passive voice-based acoustic sensing with intensive longitudinal
¹⁸⁴ assessment in a real-world stress context, this study advances both theory and
¹⁸⁵ methodology in personality pathology research. It provides an objective, low-burden index
¹⁸⁶ of stress reactivity, tests core predictions of transactional models in daily life, and moves
¹⁸⁷ the field beyond static, decontextualized trait assessment toward a dynamic, situated
¹⁸⁸ understanding of maladaptive personality processes.

189

Methods

190 **Participants**

191 We recruited 141 female university students ($M_{age} = 22.06$, $SD = 3.74$) from the
192 University of Florence through course announcements and online postings. The sample was
193 restricted to female participants to control for sex-related variation in vocal pitch
194 characteristics (Gelfer & Mikos, 2005), which could obscure personality-related effects and
195 reduce statistical power. Fundamental frequency (F0) differs substantially between males
196 and females (approximately 100 Hz lower in males) due to anatomical differences in vocal
197 fold length and mass, making direct comparison problematic without large samples or
198 complex statistical controls.

199 All participants were native Italian speakers with no reported history of voice
200 disorders or current respiratory illness. Exclusion criteria included: (1) current or past
201 psychiatric disorders requiring treatment, (2) substance use disorders, (3) self-reported
202 hearing impairments that could affect voice monitoring, and (4) professional voice training
203 (e.g., singing lessons), which could alter baseline vocal characteristics. Participants
204 provided written informed consent and received course credit for participation. The study
205 was approved by the University of Trieste Ethics Committee (protocol #05/23052025).

206 **Design and Procedure**

207 We employed a naturalistic stress manipulation design, capitalizing on the university
208 examination period as an ecologically valid acute stressor. The study comprised three
209 assessment waves across 2.5 months:

210 1. **Baseline assessment (T1):** Administered 3-4 weeks before scheduled exams,
211 participants completed the full PID-5 questionnaire and provided vocal recordings in
212 a laboratory setting.

- 213 2. **Pre-exam assessment** (T2): The day before a major course examination,
214 participants recorded vocal samples in the same laboratory.
- 215 3. **Post-exam assessment** (T3): The day after the examination, participants provided
216 final vocal recordings.

217 The course examination was administered digitally via the Moodle platform, with
218 automated scoring that provided participants with their grade immediately upon
219 completion. This procedural feature ensured that, by the time of the T3 recording,
220 uncertainty about the exam outcome had been resolved.

221 Between T1 and T3, participants completed twice-weekly ecological momentary
222 assessments (EMA) via smartphone application, yielding an average of 27.0 EMA
223 observations per participant (range: 12-31).

224 Measures

225 **Personality Pathology. Full PID-5 (Baseline).** At T1, participants completed
226 the 220-item Personality Inventory for DSM-5 (Krueger et al., 2012), which assesses five
227 maladaptive trait domains: Negative Affectivity ($\omega = 0.85$), Detachment ($\omega = 0.84$),
228 Antagonism ($\omega = 0.82$), Disinhibition ($\omega = 0.84$), and Psychoticism ($\omega = 0.90$). Items are
229 rated on a 0-3 scale (0 = very false/often false, 3 = very true/often true). PID-5 domain
230 scale scores were calculated as an average of the three most representative facet scores
231 included under each domain as per formal scoring procedures (American Psychiatric
232 Association, 2014).

233 **Brief PID-5 for EMA.** To reduce participant burden while maintaining construct
234 coverage, we administered a brief version of the PID-5 in the EMA assessments, comprising
235 a small subset of items per domain. Items were selected based on their factor loadings and
236 domain representativeness in a large independent sample drawn from the same population,
237 as reported in prior validation studies (Bottesi et al., 2024; Sica et al., 2024). This selection

238 strategy aimed to maximize construct validity while minimizing redundancy and
239 assessment burden in intensive longitudinal designs.

240 EMA data were collected via the m-Path smartphone application (RoQua, Tilburg,
241 Netherlands), a validated platform for ambulatory assessment research. Participants
242 received push notifications twice weekly on non-consecutive days between 18:00 and 20:00
243 over the 2.5-month study period. Each prompt requested ratings of current affect states
244 and brief personality-relevant items. Items were rated on the same 0–3 scale used in the
245 full PID-5, but were rephrased to assess current states (e.g., ‘At this moment, I worry a lot
246 about being alone’), whereas the original PID-5 items assess general tendencies (e.g., ‘I
247 worry a lot about being alone’).

248 In addition to routine EMA assessments, two exam-related prompts were
249 administered: one immediately before a scheduled exam (same day, 1–4 hours prior) and
250 one the day after exam completion. These exam-linked assessments were paired with voice
251 recordings (see Voice Recordings section) to capture stress-related vocal changes.

252 **Data quality and compliance.** Participants with fewer than 50% response rate to
253 EMA prompts were excluded from analysis prior to data processing to ensure adequate
254 sampling of trait-relevant behaviors.

255 **Voice Recordings.** At each of the three assessment sessions (T1, T2, and T3),
256 participants produced a set of standardized voice recordings in a sound-attenuated room.
257 The recording protocol included sustained vowel phonations, a coarticulation task, and a
258 standardized sentence. In addition, using the m-Path application, participants recorded a
259 short spontaneous audio message spoken with conversational pitch, loudness, and rhythm,
260 based on predefined prompts described below (Manfredi et al., 2017).

261 All recordings were obtained in quiet indoor environments. Participants were
262 instructed to position the recording device approximately 15 cm from the mouth at a 45°

263 angle to minimize plosive artifacts and lateral acoustic distortions. Instructions emphasized
264 maintaining consistent microphone placement across sessions.

265 **Stimuli.** Participants were asked to produce the following vocalizations:

- 266 1. Three repetitions of sustained Italian cardinal vowels (/a/, /i/, /u/), each held for a
267 minimum of 3 s.
- 268 2. A coarticulation task consisting of counting aloud from 1 to 10 in Italian.
- 269 3. A standardized, continuously voiced Italian sentence: “*Io amo le aiuole della*
270 *mamma*” (English translation: “*I love my mother’s flowerbeds*”).

271 All speech tasks were performed using conversational pitch and loudness to preserve
272 ecological validity while ensuring sufficient acoustic quality for analysis.

273 For sustained-vowel features, acoustic parameters were extracted separately for each
274 vowel token (/a/, /i/, /u/; three repetitions each) and then averaged across vowels and
275 repetitions within each session to yield a single session-level estimate per participant.

276 **Acoustic Feature Extraction.** Acoustic features were extracted using two
277 complementary approaches: (1) traditional vocal parameters derived from sustained vowel
278 phonations, and (2) mel-frequency cepstral coefficients (MFCCs) extracted from continuous
279 speech.

280 **Sustained vowel analysis.** The open-source BioVoice software (Morelli & Manfredi,
281 2019) was used to extract frequency- and time-domain acoustic parameters from sustained
282 vowel productions. Analyses focused on the steady-state portion of each vowel, excluding
283 onset and offset segments to ensure stable phonatory conditions. For each recording
284 session, measures were computed separately for each vowel token (/a/, /i/, /u/) and then
285 averaged across vowels and repetitions.

286 **Fundamental frequency (F0).** Mean and median F0 were calculated as summary

287 indices of vocal fold vibratory rate during sustained phonation, while the standard
288 deviation of F0 indexed short-term pitch variability. In addition, the temporal location of
289 the maximum F0 value (T0) was extracted as a marker of phonatory dynamics.
290 Fundamental frequency is the most extensively validated acoustic correlate of psychological
291 stress and arousal, reflecting changes in laryngeal muscle tension and autonomic activation.
292 Meta-analytic and experimental evidence consistently demonstrates reliable increases in F0
293 under a wide range of stressors, including public speaking, cognitive load, and evaluative
294 threat (Giddens, Barron, Byrd-Craven, Clark, & Winter, 2013; Scherer, 2003).
295 Physiologically, these increases are attributed to sympathetic nervous system activation,
296 which enhances cricothyroid and vocalis muscle tension, increases vocal fold stiffness, and
297 consequently elevates vibratory frequency (Titze, 1994). As such, F0 provides a direct
298 acoustic index of the arousal component of the stress response.

299 **Normalized Noise Energy (NNE).** Normalized noise energy, expressed in
300 decibels, quantifies the relative contribution of aperiodic (noise) energy to the overall voice
301 signal and serves as an index of phonatory quality and glottal closure completeness
302 (Kasuya, Ogawa, Mashima, & Ebihara, 1986). Higher NNE values indicate a greater
303 proportion of noise relative to harmonic energy and are typically associated with reduced
304 or incomplete glottal closure and breathier voice quality. Unlike perturbation measures
305 such as jitter and shimmer, which primarily capture cycle-to-cycle instability, NNE is
306 sensitive to broader stress-related changes in phonatory control and vocal effort. Empirical
307 findings suggest that acute stress can have bidirectional effects on vocal noise: it may
308 increase NNE through incomplete glottal closure and increased airflow turbulence under
309 heightened arousal, or decrease NNE through compensatory hyperadduction and pressed
310 phonation aimed at maintaining vocal control (Mendoza & Carballo, 1998; Scherer, 2003).
311 The direction of these effects appears to depend on individual coping strategies and task
312 demands. Accordingly, NNE may capture individual differences in stress-related vocal
313 regulation, reflecting whether arousal is accompanied by vocal degradation or

314 compensatory control. Examining NNE alongside F0 therefore allows dissociation between
315 arousal-driven changes in pitch and stress-related adjustments in phonatory control,
316 clarifying whether stress manifests primarily as vocal degradation or as compensatory
317 tightening of the phonatory system.

318 **Data Quality**

319 **EMA Compliance and Quality Control.** EMA data quality was ensured by
320 applying compliance and quality control criteria prior to analysis. Participants with fewer
321 than 5 assessments were excluded. Additional checks targeted careless responding,
322 including insufficient within-subject variability and atypical response patterns (e.g.,
323 excessive use of scale endpoints). Applying these criteria resulted in the exclusion of 12
324 participants, reducing the sample from $n = 141$ to $n = 119$. The final sample included
325 participants with complete voice data and valid EMA responses.

326 **Convergent Validity of EMA Measures**

327 To establish construct validity of the brief EMA assessment, we computed
328 correlations between person-level EMA domain scores (aggregated across all assessments)
329 and full baseline PID-5 domains.

330 **Selection of Acoustic Parameters.** We focused on two acoustic parameters (F0
331 and NNE) selected for their theoretical relevance to stress-related vocal changes and their
332 complementary information about distinct physiological mechanisms [REF].

333 This dual-parameter approach aligns with multidimensional models of vocal stress
334 responses (Giddens et al., 2013) and allows us to test competing hypotheses about how
335 personality traits might selectively moderate arousal versus control components of the
336 stress response. We did not examine other acoustic features (e.g., formant frequencies,
337 spectral tilt, speech rate) as these are more strongly influenced by linguistic content and
338 articulation than by the phonatory physiology most directly affected by autonomic stress

339 responses.

340 Statistical Analysis

341 Statistical analyses were conducted in a Bayesian framework using Stan. Voice
342 outcomes (mean F0 and NNE) were modeled using hierarchical linear regression models
343 with repeated observations nested within participants. Experimental effects were
344 parameterized using two orthogonal contrasts representing stress reactivity (stress
345 vs. baseline) and recovery (post-stress vs. stress).

346 Individual differences in personality were modeled as latent traits derived from
347 repeated EMA assessments of the five PID-5 domains. A multivariate measurement model
348 was used to estimate subject-level latent trait scores while accounting for measurement
349 error in the observed EMA indicators. These latent traits were then included in the
350 outcome model both as main effects and as moderators of stress and recovery effects via
351 cross-level interactions.

352 All models included subject-specific random intercepts and random slopes for the
353 stress and recovery contrasts. Weakly informative priors were used for all fixed effects,
354 variance components, and correlations. Posterior inference was based on full posterior
355 distributions, with model adequacy evaluated using posterior predictive checks and
356 approximate leave-one-out cross-validation.

357 **Estimation.** Models were estimated using Hamiltonian Monte Carlo with 4 chains
358 of 5,000 iterations each (2,500 warmup). Convergence was assessed via $Rhat < 1.01$ and
359 effective sample size > 400 . Adaptation parameters (`adapt_delta = 0.995`, `max_treedepth`
360 $= 18$) prevented divergent transitions. For models with convergence difficulties, we
361 increased iterations or simplified random effects structures.

362 **Inference.** Effects were considered credible if 95% credible intervals excluded zero.
363 We report posterior means and 95% CIs throughout. For key hypotheses, we computed

364 Bayes factors comparing moderation models to null models without interactions.

365 Data and Code Availability. All analysis code and de-identified data will be
366 made publicly available upon publication at [OSF LINK]. Models were fit using brms 2.21
367 with cmdstanr 0.7.

368 Results

369 Main Effects of Exam-Related Stress on Vocal Acoustics

We first examined whether acute exam-related stress altered fundamental frequency and glottal noise independently of personality traits. Hierarchical models incorporated random intercepts and random slopes for two orthogonal contrasts: a stress contrast (c_1) comparing pre-exam to baseline recordings, and a recovery contrast (c_2) comparing post-exam to pre-exam recordings. This parameterization allowed us to distinguish the immediate impact of anticipatory stress from subsequent recovery dynamics. For sustained-vowel outcomes (F0 and NNE), values represent session-level averages computed across the three sustained vowels (/a/, /i/, /u/) and their repetitions.

Fundamental frequency. Descriptive statistics revealed a progressive pattern across timepoints. Mean F0 at baseline was 190.7 Hz (SD = 22.0), increasing to 194.0 Hz (SD = 21.9) immediately before the exam and then declining slightly to 192.5 Hz (SD = 23.6) following the exam. The hierarchical model confirmed robust stress-related elevation. The intercept parameter α , representing the estimated F0 at baseline, had a posterior median of 192.48 Hz (MAD = 1.73, 95% CrI [189.07, 195.96]). The stress contrast β_1 showed a clear positive effect: F0 increased by 3.27 Hz (MAD = 1.25, 95% CrI [0.81, 5.71]) when comparing pre-exam to baseline recordings, with $P(\beta_1 > 0) = 0.995$. This finding indicates that acute academic stress reliably elevates vocal pitch, consistent with increased laryngeal tension and autonomic arousal. The recovery contrast β_2 was essentially null (median = 0.14 Hz, MAD = 1.24, 95% CrI [-2.34, 2.59], $P(\beta_2 > 0) = 0.542$), indicating that F0 plateaued after the exam with minimal further change during the brief recovery period.

390 Between-person variability was substantial. The standard deviation of random
391 intercepts was $\tau_1 = 19.86$ (95% CrI [17.68, 22.44]), reflecting considerable individual
392 differences in baseline vocal pitch. The standard deviation of random slopes for the stress
393 contrast was $\tau_2 = 1.08$ (95% CrI [0.04, 4.45]), indicating modest heterogeneity in stress
394 reactivity after accounting for personality moderation (see below). Residual variability
395 within individuals was $\sigma = 9.10$ Hz (95% CrI [8.34, 9.96]).

396 **Normalized noise energy.** In contrast to F0, NNE exhibited a pattern consistent
397 with reduced glottal noise under stress. Descriptively, mean NNE at baseline was -26.55 dB
398 (SD = 2.64), decreasing to -27.09 dB (SD = 3.25) at the pre-exam assessment and
399 remaining relatively stable at -26.98 dB (SD = 2.91) post-exam. More negative NNE values
400 indicate a more periodic, harmonically stable signal. The hierarchical model confirmed
401 systematic stress-induced reduction in glottal noise. The intercept had a posterior median
402 of -26.87 dB (MAD = 0.20, 95% CrI [-27.28, -26.47]). The stress contrast showed a robust
403 negative effect: NNE decreased by 0.79 dB (MAD = 0.31, 95% CrI [-1.30, -0.30]), with
404 $P(\beta_1 < 0) = 0.995$. The recovery contrast was $\beta_2 = -0.19$ dB (MAD = 0.30, 95% CrI
405 [-0.69, 0.31]). The 95% credible interval includes zero and the directional probability is
406 weak ($P(\beta_2 < 0) = 0.219$), indicating minimal systematic change during the post-exam
407 period. Random effects estimates revealed considerable between-person heterogeneity in
408 baseline NNE ($\tau_1 = 2.14$, 95% CrI [1.85, 2.46]) and in stress-related change ($\tau_2 = 0.71$, 95%
409 CrI [0.06, 1.56]). Residual variability was $\sigma = 1.98$ dB (95% CrI [1.78, 2.17]).

410 **Summary.** Exam-related stress produced dissociable changes in vocal production.
411 Fundamental frequency increased robustly under stress, reflecting heightened autonomic
412 arousal and laryngeal tension. In contrast, NNE decreased, indicating reduced glottal noise
413 and a more controlled, periodic phonatory signal. These patterns suggest that acute stress
414 does not simply destabilize the voice but instead induces simultaneous increases in
415 physiological arousal (indexed by F0) and compensatory phonatory control (indexed by

416 reduced noise). The consistent directionality and strong posterior probabilities ($P > 0.99$)
417 that the effects differed from zero for both F0 and NNE) underscore the robustness of these
418 vocal signatures of stress. In contrast, recovery effects showed weaker evidence, with
419 credible intervals including zero for both parameters.

420 **Personality Moderation of Vocal Stress Responses**

421 To examine whether PID-5 personality domains moderated vocal stress responses, we
422 added to the previously-described models a trait \times contrast interactions. Personality traits
423 were modeled as latent variables derived from EMA assessments, incorporating explicit
424 measurement error correction. We report moderation effects as the change in the stress or
425 recovery contrast effect associated with a one-standard-deviation increase in the trait. We
426 first present F0 moderation results, then examine whether personality domains
427 differentially modulate voice quality (NNE).

428 **Arousal-related pitch responses (F0).** For F0, we estimated ten moderation
429 parameters: five domains (Negative Affectivity, Detachment, Antagonism, Disinhibition,
430 Psychoticism) crossed with two contrasts (stress, recovery). Table 1 presents posterior
431 medians, 95% credible intervals, and directional probabilities for each interaction. Among
432 these ten tests, only one showed clear evidence of moderation: Negative Affectivity
433 amplified the stress-induced increase in F0 ($\gamma_1 = 3.14$ Hz per SD, 95% CrI [0.37, 5.89], PD
434 = 0.97). This effect indicates that individuals higher in emotional reactivity and stress
435 sensitivity (Negative Affect) exhibited stronger vocal arousal responses during anticipatory
436 stress. No other stress-phase moderation effects showed meaningful Bayesian evidence.
437 Detachment and Disinhibition exhibited no credible stress-phase moderation (PD = 0.59
438 and 0.64, respectively). During the recovery phase, Detachment showed a suggestive
439 tendency toward reduced F0 elevation ($\gamma_2 = -2.02$ Hz, PD = 0.88), whereas Disinhibition
440 showed no meaningful effect (PD = 0.67).

441 For the recovery contrast, Antagonism showed the strongest moderation ($\gamma_2 = 3.16$

442 Hz per SD, 95% CrI [0.51, 5.78], PD = 0.97), suggesting that individuals higher in

443 callousness and interpersonal hostility (Antagonism) exhibited continued F0 elevation

444 during the post-exam period. However, this effect should be interpreted cautiously given

445 the weak main effect of the recovery contrast itself and the relatively small sample for

446 detecting interaction effects in the recovery phase.

447 **Voice Quality Moderation (NNE).** In contrast to F0, NNE showed minimal

448 moderation during the stress phase, with all domains exhibiting weak directional certainty

449 (all PD < 0.83 for γ_1). However, the recovery phase revealed a distinct pattern:

450 Psychoticism demonstrated strong directional certainty for recovery moderation ($\gamma_2 = 0.88$

451 dB, 95% CrI [0.05, 1.72], PD = 0.96, SNR = 1.74). This effect indicates that individuals

452 higher in odd or eccentric thinking (Psychoticism) exhibited less negative NNE values (i.e.,

453 increased glottal noise) during the post-exam period, reflecting reduced phonatory control

454 following stress exposure. Antagonism showed suggestive evidence for recovery moderation

455 in the opposite direction ($\gamma_2 = -0.43$ dB, 95% CrI [-1.37, 0.49], PD = 0.82), though this fell

456 below conventional evidence thresholds. No other domains showed meaningful NNE

457 modulation (Table 2).

458 **Summary.** The selective moderation patterns reveal striking domain-specificity in

459 personality influences on vocal stress responses. Whereas Negative Affectivity reliably

460 shaped arousal-related pitch responses during stress anticipation, voice quality (NNE)

461 showed a completely distinct pattern: only Psychoticism modulated NNE, and exclusively

462 during the recovery phase ($\gamma_2 = 0.88$ dB, PD = 0.96). This dissociation suggests that

463 different personality domains influence distinct temporal phases and acoustic dimensions of

464 stress responses. Internalizing traits (Negative Affectivity) appear to primarily modulate

465 autonomic arousal indexed by F0 during stress exposure, whereas thought disorder

466 characteristics (Psychoticism) influence phonatory control mechanisms reflected in glottal

467 noise, particularly during stress de-escalation.

468 [TABLE 1 HERE: PID-5 Domain × Stress/Recovery Interactions for F0]

469 [TABLE 2 HERE: PID-5 Domain × Stress/Recovery Interactions for NNE]

470 **Comparing EMA-Based and Baseline PID-5 Assessments**

471 A methodological question central to our design was whether repeated measurement
472 via EMA provided advantages over comprehensive single-occasion assessment. To address
473 this, we compared three modeling approaches using leave-one-out cross-validation
474 (LOO-CV): (1) EMA-only, incorporating three EMA assessments within a latent variable
475 measurement model; (2) Baseline-only, using the full 220-item PID-5 from a single
476 administration; and (3) Combined, simultaneously estimating both EMA latent traits
477 (estimates from repeated EMA assessments) and baseline domain scores.

478 Out-of-sample predictive performance, quantified via expected log pointwise
479 predictive density (ELPD), was comparable across the three approaches (Table 3). The
480 EMA-based model showed numerically the highest ELPD, but differences relative to the
481 Combined model (Δ ELPD = -3.0, SE = 3.6) and Baseline-only model (Δ ELPD = -4.6,
482 SE = 4.0) did not exceed the conventional threshold for meaningful differences ($|\Delta| < 2$
483 SE). This equivalence indicates that ambulatory assessment and comprehensive
484 single-occasion assessment provide similar predictive accuracy for vocal F0 trajectories
485 during acute stress.

486 However, examination of moderation effect estimates revealed an important
487 distinction. Table 3 presents a focused comparison for Negative Affectivity, the domain
488 showing the strongest stress moderation. The EMA-based model yielded a more precise
489 estimate ($\gamma_1 = 3.07$ Hz, 95% CrI [-0.44, 6.55], PD = 0.96) compared to the baseline model
490 ($\gamma_1 = 2.65$ Hz, 95% CrI [-2.20, 7.52], PD = 0.86). The EMA estimate showed a 28%

491 narrower credible interval and stronger directional evidence. This pattern was consistent
492 across other PID-5 domains: EMA-derived estimates systematically showed tighter
493 uncertainty bounds despite comparable point estimates (Supplementary Table S2).

494 The Combined model, which simultaneously estimated both measurement approaches,
495 produced moderation estimates intermediate between the two single-source models.
496 Neither measurement approach dominated when both were included, suggesting that EMA
497 and baseline assessment capture largely overlapping rather than complementary variance in
498 predicting vocal stress reactivity. Together, these results indicate that while EMA does not
499 improve aggregate predictive performance, it does enhance inferential precision for
500 moderation effects—a distinction relevant for theory testing even when forecasting
501 accuracy is equivalent.

502 Discussion

503 Across an ecologically valid academic stressor, we observed reliable stress-related
504 changes in two complementary vocal parameters, fundamental frequency (F0) and
505 normalized noise energy (NNE), and found *selective, domain- and phase-specific moderation*
506 by PID-5 trait domains. In addition, repeated brief EMA-based trait assessment achieved
507 *comparable predictive accuracy* to a complete baseline PID-5 questionnaire while yielding
508 *greater precision* in key moderation estimates. Together, these findings support
509 transactional accounts of personality pathology by showing that maladaptive trait domains
510 are expressed not only in average tendencies, but also in *how individuals'*
511 *psychophysiological responses change across situational demands and recovery periods*.

512 **Vocal acoustics as objective indicators of situational stress.** Exam-related
513 stress produced dissociable changes in vocal production. Consistent with prior
514 psychophysiological work, F0 increased during the pre-exam assessment, indicating
515 heightened laryngeal muscle tension and autonomic arousal (Giddens et al., 2013; Scherer,
516 2003). Importantly, this effect emerged in a real-world evaluative context rather than an

517 experimentally induced laboratory stressor, supporting the ecological validity of vocal pitch
518 as an objective marker of stress reactivity in everyday life.

519 In contrast to a simple “vocal degradation” account, NNE decreased by 0.79 dB
520 during stress, indicating a more periodic, less noisy signal. This pattern suggests that
521 evaluative stress can involve not only arousal-related activation (indexed by F0) but also
522 *compensatory phonatory control* (indexed by reduced glottal noise). Under
523 performance-oriented conditions, individuals may unconsciously adopt more effortful or
524 pressed phonation that increases periodicity, even while physiological arousal rises. This
525 arousal-control dissociation implies that stress responses in the voice are multidimensional
526 rather than unitary, and that different personality domains may preferentially shape
527 different components of the response.

528 Notably, average *recovery effects* were weak for both parameters over the short
529 post-exam window. Because exam grades were communicated to participants immediately
530 upon completion (via automated Moodle scoring), the persistence of stress-related vocal
531 changes at T3 cannot be attributed to lingering uncertainty about the outcome. Rather,
532 the absence of clear mean-level recovery likely reflects the slow decay of physiological
533 activation following stress, individual differences in recovery speed, or both. These
534 considerations highlight the value of modeling heterogeneity in recovery trajectories rather
535 than relying solely on mean-level recovery effects. In this sense, recovery may represent a
536 particularly sensitive phase for revealing individual differences that are obscured at the
537 level of average responses.

538 **Domain- and phase-specific moderation of vocal stress responses.** Given
539 the absence of prior research on personality moderation of vocal stress markers, we adopted
540 an exploratory approach for most PID-5 domains, specifying a directional hypothesis only
541 for Negative Affectivity. The resulting pattern provides evidence that personality
542 pathology domains do not exert broad, uniform effects; instead, they appear to moderate

543 specific acoustic parameters at specific phases of the stress-response cycle.

544 **Negative Affectivity: amplified acute arousal reactivity.** As predicted,
545 Negative Affectivity reliably amplified stress-induced increases in F0. Individuals higher in
546 Negative Affectivity showed a stronger pitch elevation during anticipatory stress, consistent
547 with models linking this domain to heightened threat vigilance, emotional lability, and
548 sympathetic activation (Lahey, 2009; Ormel et al., 2013). The specificity of the effect to *F0*
549 *during the stress phase*, rather than to NNE or to recovery, supports the interpretation that
550 Negative Affectivity primarily modulates arousal-driven pathways (i.e., the autonomic
551 component of the response), rather than broadly destabilizing vocal output.

552 **Antagonism: evidence for heterogeneous post-stressor trajectories.**

553 Antagonism showed its strongest association with the *recovery contrast* for F0, suggesting
554 that individuals higher on antagonistic traits may exhibit different post-stressor trajectories
555 of arousal. Because the average recovery effect in the sample was near zero, this pattern is
556 best interpreted not as a robust population-level “impaired recovery,” but as *preliminary*
557 *evidence of heterogeneity* in the degree to which arousal persists once the acute stressor has
558 ended. One plausible mechanism may involve prolonged engagement with the evaluative
559 episode (e.g., continued anger focus rumination, irritability, or interpersonal conflict in the
560 aftermath), which could maintain physiological activation beyond the stressor offset.
561 However, resolving whether this reflects affective inertia, continued contextual activation,
562 or other processes will require denser post-stressor sampling and measures of post-exam
563 cognitions and contexts.

564 **Psychoticism: selective modulation of voice quality during recovery.**

565 Psychoticism showed minimal moderation of F0, aligning with mixed prior evidence on
566 physiological stress reactivity in schizotypy-related traits. However, Psychoticism uniquely
567 moderated *NNE during recovery*, with higher Psychoticism associated with increased
568 glottal noise (less negative NNE) post-exam. This specificity to a voice quality parameter,
569 and to the recovery phase, suggests that Psychoticism may be linked less to the magnitude

570 of acute arousal and more to processes involved in stabilizing phonatory control after stress
571 exposure. One interpretation is that cognitive–perceptual dysregulation may compromise
572 adaptive recalibration of motor control once stress cues recede, yielding less stable
573 phonation. Alternative explanations are also plausible, including persistent autonomic
574 dysregulation or context-dependent changes in speech effort. Discriminating among these
575 accounts will require concurrent measurement of autonomic markers (e.g., heart rate
576 variability) and, ideally, more continuous sampling across the recovery period.

577 ***Detachment and Disinhibition: limited evidence for moderation.***

578 Detachment showed weak evidence for *improved* rather than impaired F0 recovery (PD =
579 0.88, negative coefficient). This pattern is noteworthy because it runs counter to
580 predictions derived from the emotional inertia literature, which links restricted affectivity
581 and anhedonia to prolonged negative states and impaired affect repair (Koval et al., 2015;
582 Kuppens et al., 2010). One possible interpretation is that the emotional blunting
583 characteristic of Detachment may attenuate not only positive emotional experiences but
584 also sustained physiological activation following stress: if high-Detachment individuals are
585 less engaged with the evaluative implications of the stressor, they may show faster
586 normalization simply because there is less activation to resolve. Given the modest strength
587 of this effect, it should be treated cautiously and may represent sampling variability, but it
588 raises the possibility that restricted affectivity could function as a context-specific
589 protective factor in acute stress—even if it reflects broader affective impairment in other
590 domains.

591 Disinhibition showed no meaningful moderation of either acoustic parameter (all PD
592 < 0.75). This null finding is theoretically coherent rather than merely uninformative.
593 Unlike Negative Affectivity, which reflects arousal sensitivity and emotional reactivity,
594 Disinhibition is defined by impulsivity, irresponsibility, and poor behavioral
595 control—characteristics pertaining to volitional action regulation rather than automatic
596 physiological processes (Krueger et al., 2012). Meta-analytic evidence indicates that

597 conscientiousness-related traits show weak associations with physiological stress responses
598 despite moderating subjective stress perception and coping behaviors (Luo et al., 2023).
599 The present findings extend this dissociation to vocal acoustics: because F0 elevation
600 reflects automatic autonomic mechanisms (laryngeal tension from sympathetic activation)
601 rather than behavioral control processes, Disinhibition—which governs volitional rather
602 than automatic responses—would not be expected to moderate this pathway. More
603 broadly, these results reinforce the conclusion that moderation effects are not ubiquitous
604 across PID-5 domains but are *selective and mechanism-specific*, with different domains
605 operating through distinct psychophysiological pathways (Calà et al., 2025).

606 **Implications for transactional models of personality pathology.** The
607 dissociations observed across trait domains (Negative Affectivity, Antagonism,
608 Psychoticism), acoustic parameters (F0 vs. NNE), and temporal phases (stress reactivity
609 vs. recovery) are consistent with transactional models in which personality pathology
610 shapes *how* individuals respond to contextual demands rather than simply predicting static
611 levels of functioning (Bolger & Zuckerman, 1995). In this view, different domains may map
612 onto distinct stress-process mechanisms: Negative Affectivity primarily amplifies arousal
613 reactivity; Antagonism may be linked to persistence of activation following stress; and
614 Psychoticism may be associated with post-stressor instability in control-related vocal
615 processes. Taken together, these findings underscore the importance of moving beyond
616 global notions of “stress reactivity” in personality pathology research. The PID-5 domains
617 do not appear to represent interchangeable indicators of generalized vulnerability; instead,
618 they map onto partially dissociable psychophysiological pathways that are differentially
619 engaged across phases of the stress-response cycle. This process-oriented interpretation is
620 consistent with transactional models of personality pathology, which emphasize dynamic
621 person–situation interactions rather than static trait effects (Bolger & Zuckerman, 1995;
622 Wright & Simms, 2016). More broadly, the results suggest that the theoretical value of the
623 PID-5 lies not only in its descriptive coverage of maladaptive traits, but also in its potential

624 to guide mechanistically informed hypotheses about when, how, and through which
625 biological channels personality pathology is expressed in everyday life. Importantly, these
626 interpretations remain provisional and should be tested in designs that incorporate richer
627 characterization of situational features and more continuous measurement during recovery.

628 At the same time, the present findings suggest that vocal acoustics can contribute to
629 a more differentiated phenotyping of stress responses. Relying solely on pitch-based
630 markers may primarily detect internalizing-related vulnerability (Negative Affectivity),
631 whereas incorporating voice-quality indices may reveal additional processes relevant to
632 cognitive-perceptual dysregulation (Psychoticism) or other domains. Future ambulatory
633 work that models multiple vocal dimensions across multiple stressor types may help build
634 more precise, trait-informed accounts of vulnerability and adaptation.

635 **Methodological contribution: intensive ambulatory trait assessment.** A
636 central methodological contribution of this project is to show that brief trait measurements
637 assessed repeatedly via EMA can perform comparably to comprehensive baseline
638 questionnaires for predicting vocal stress trajectories. Across models, out-of-sample
639 predictive performance was similar for EMA-based and baseline-based PID-5 measures,
640 indicating a substantial overlap in the trait variance they capture. However, EMA-based
641 measurement yielded *more precise moderation estimates*, consistent with the idea that
642 repeated sampling can reduce measurement error and stabilize person-level trait estimates.
643 This distinction is important: even when overall predictive accuracy is equivalent,
644 improved inferential precision can strengthen theory tests of trait-by-context interactions.

645 These results support the feasibility of combining low-burden repeated trait
646 assessment with objective acoustic markers in longitudinal designs, particularly when
647 comprehensive questionnaires are impractical. At the same time, the present design relied
648 on brief standardized voice recordings rather than continuous passive sensing. Extending
649 this approach to more frequent recordings would allow stronger tests of within-person

650 coupling and more detailed modeling of recovery dynamics.

651 **Limitations and future directions.** Several limitations qualify the conclusions.

652 First, the sample consisted of female university students, and we excluded participants

653 with psychiatric disorders requiring treatment. Although PID-5 traits are dimensionally

654 meaningful in community samples, generalization to clinical populations and to males

655 requires replication. Second, although the examination period provides a naturalistic

656 stressor with real stakes, it is not equivalent to interpersonal stressors that are central to

657 many forms of personality pathology. Different stressor classes may yield different vocal

658 signatures and trait moderation patterns. Third, vocal data were collected at only three

659 time points, which constrains the temporal resolution with which stress-related dynamics

660 can be characterized. Although this design captures broad phases of baseline, anticipatory

661 stress, and early recovery, it cannot resolve the finer-grained time course through which

662 vocal parameters return to baseline following stressor offset. As a result, apparent

663 persistence of stress-related effects may reflect delayed physiological recovery, continued

664 contextual activation, or both. Future studies employing denser sampling, particularly in

665 the hours immediately following stress exposure, will be necessary to disentangle these

666 processes and to model recovery trajectories with greater precision. Fourth, vocal markers

667 are biologically plausible indices of stress, but they are not process-pure. Changes in F0

668 and NNE may reflect multiple mechanisms, including autonomic activation, cognitive load,

669 strategic self-presentation, and speech effort. Future studies should triangulate voice

670 measures with concurrent physiological indicators (e.g., heart rate variability, electrodermal

671 activity) and with contextual measures (perceived stress, task difficulty, preparedness) to

672 clarify underlying pathways.

673

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Table 1

Principal Acoustic Features of Speech: Definitions and Psychological Significance

Feature	Definition	Physiological basis	Psychological significance
Fundamental frequency (F0)	Rate of vocal fold vibration (Hz); acoustic correlate of perceived pitch	Cricothyroid and vocalis muscle tension regulates vocal fold stiffness and vibratory rate	Most robust acoustic marker of stress: F0 increases reliably under acute stress, cognitive load, and evaluative threat via sympathetic activation
Normalized Noise Energy (NNE)	Ratio of inharmonic to harmonic spectral energy (dB); index of glottal noise	Degree of glottal closure completeness during phonation	Reflects phonatory control and vocal effort; stress may increase noise (incomplete closure under arousal) or decrease it (compensatory hyper-adduction and pressed phonation)
Jitter ^a	Cycle-to-cycle variation in F0 period (%); measure of frequency perturbation	Irregularity of successive vocal fold vibratory cycles	Proposed as an index of vocal instability and reduced neuromuscular control; elevated in some emotional and clinical states

Note. F0 and NNE were retained as primary outcome variables in the present study.

^aJitter was extracted but not included in the main analyses for the following reasons: (a) the literature on jitter and psychological stress is inconsistent, with effect sizes that are small, heterogeneous in direction, and often non-significant across studies (Giddens et al., 2013); (b) jitter is highly sensitive to recording conditions, signal-to-noise ratio, and extraction algorithm parameters, resulting in substantial measurement noise in field settings (Titze, 1994); and (c) unlike F0 and NNE, which index theoretically distinct mechanisms (autonomic arousal vs. phonatory control), jitter does not map onto a clearly separable psychophysiological pathway, limiting its interpretive value in the context of personality moderation hypotheses.

Table 2

Personality Moderation of Fundamental Frequency (F0)

Domain	Stress (γ_1)		Recovery (γ_2)	
	Median [95% CrI]	PD	Median [95% CrI]	PD
Negative Affectivity	3.14 [0.37, 5.89]	0.97	-0.31 [-3.13, 2.51]	0.60
Detachment	-0.38 [-3.10, 2.31]	0.59	-2.02 [-4.84, 0.76]	0.88
Antagonism	0.09 [-2.51, 2.69]	0.52	3.16 [0.51, 5.78]	0.97
Disinhibition	0.61 [-2.60, 3.82]	0.64	0.68 [-2.51, 3.87]	0.67
Psychoticism	-0.13 [-2.74, 2.46]	0.54	-1.22 [-3.81, 1.32]	0.80

Note. Moderation effects in Hz per SD of trait. PD = Probability of

Direction. Bold = strong certainty (PD > 0.95). CrI = Credible Interval.

Table 3

Personality Moderation of Normalized Noise Energy (NNE)

Domain	Stress Moderation (γ_1)		Recovery Moderation (γ_2)	
	Median [95% CrI]	PD	Median [95% CrI]	PD
Negative Affectivity	-0.46 [-1.45, 0.52]	0.83	-0.39 [-1.38, 0.62]	0.79
Detachment	0.29 [-0.69, 1.30]	0.72	0.21 [-0.77, 1.21]	0.66
Antagonism	-0.01 [-0.94, 0.90]	0.51	-0.43 [-1.37, 0.49]	0.82
Disinhibition	0.33 [-0.86, 1.51]	0.71	-0.39 [-1.56, 0.79]	0.74
Psychoticism	-0.02 [-1.04, 0.97]	0.52	0.88 [0.05, 1.72]	0.96

Note. Moderation effects represent the change in NNE (dB) associated with

a one-standard-deviation increase in the trait. Positive values indicate less

negative NNE (increased glottal noise). PD = Probability of Direction

(maximum of $P(\gamma > 0)$ and $P(\gamma < 0)$). Bold indicates strong directional

certainty (PD > 0.95). CrI = Credible Interval.

Table 4

Model Comparison: Out-of-Sample Predictive Performance

Model	ELPD		LOOIC	
	Estimate (SE)	Δ (SE)	Estimate (SE)	p_loo (SE)
EMA	-1247.3 (16.8)	—	2494.6 (33.7)	110.3 (9.2)
Combined	-1250.3 (16.7)	-3.0 (3.6)	2500.6 (33.3)	118.0 (9.4)
Baseline	-1251.9 (16.9)	-4.6 (4.0)	2503.8 (33.8)	109.0 (8.9)

Note. ELPD = Expected Log Predictive Density, LOOIC = Leave-One-Out Information Criterion, SE = Standard Error. The Δ ELPD column shows the difference relative to the best-performing model (EMA).

Table 5

Comparison of Negative Affectivity \times Stress Moderation Estimates Across Measurement Approaches

Model	Negative Affectivity \times Stress (γ_1)			
	Mean (Hz)	90% CrI	PD	Improvement
EMA	3.07	[-0.44, 6.55]	0.96	+28%
Baseline	2.65	[-2.20, 7.52]	0.86	—

Note. γ_1 = moderation effect of Negative Affectivity on stress-induced F0 change; PD = probability of direction (proportion of posterior above/below zero); CrI Width = credible interval width; Improvement = precision gain of EMA relative to Baseline [(Baseline width - EMA width) / Baseline width]. The EMA-based estimate shows **28% narrower uncertainty bounds** while maintaining comparable point estimates, reflecting enhanced precision through explicit measurement error modeling across repeated assessments.