

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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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 ($M_{age} = [DA \text{ INSERIRE}]$, $SD = [DA$
192 $\text{INSERIRE}]$) from the University of Florence through course announcements and online
193 postings. The sample was restricted to female participants to control for sex-related
194 variation in vocal pitch characteristics (Gelfer & Mikos, 2005), which could obscure
195 personality-related effects and reduce statistical power. Fundamental frequency (F0) differs
196 substantially between males and females (approximately 100 Hz lower in males) due to
197 anatomical differences in vocal fold length and mass, making direct comparison
198 problematic without large samples or 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 ($\alpha = [INSERIRE]$), Detachment ($\alpha =$
228 [INSERIRE]), Antagonism ($\alpha = [INSERIRE]$), Disinhibition ($\alpha = [INSERIRE]$), and
229 Psychoticism ($\alpha = [INSERIRE]$). Items are rated on a 0-3 scale (0 = very false/often false,
230 3 = very true/often true). PID-5 domain scale scores were calculated as an average of the
231 three most representative facet scores included under each domain as per formal scoring
232 procedures (American Psychiatric 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 Although derived from speech signals, both F0 and NNE index peripheral
319 physiological processes involved in phonation and autonomic regulation, enabling their use
320 as objective, physiology-linked indicators that are largely independent of self-report.

321 **Theoretical rationale.** The selection of F0 and NNE was guided by converging
322 neurophysiological and psychophysiological models linking stress to alterations in muscular
323 activation and motor control during speech production (Scherer, 1986; Titze, 1994;
324 Giddens et al., 2013). Stress-related increases in general muscular tone, including at the
325 laryngeal level, are expected to elevate vocal fold tension and vibratory rate, resulting in
326 higher F0. In contrast, predictions for voice quality measures such as NNE are less
327 straightforward. Moderate increases in laryngeal tension may improve glottal adduction
328 and reduce aperiodic energy, whereas excessive or asymmetric tension may stiffen the vocal
329 fold mucosa, disrupt the regularity of the mucosal wave, and increase airflow turbulence at
330 the glottis, leading to higher noise levels (Titze, 1994). This mechanistic ambiguity is
331 consistent with the smaller and less consistent stress effects typically reported for voice
332 quality parameters relative to F0 (Giddens et al., 2013; Van Puyvelde et al., 2018).

333 **Data Quality and Missingness**

334 **EMA Compliance and Quality Control.** Participants with fewer than 5 or
335 more than 40 EMA assessments were excluded prior to analysis (n excluded =
336 [INSERIRE]). We implemented additional quality checks to identify careless responding:

337 1. **Within-subject variability:** Participants with SD < 0.30 on the Negative
338 Affectivity composite (which should exhibit temporal variation) were flagged for

339 review.

340 **2. Response patterns:** Excessive use of scale endpoints or preference for round
341 numbers (>80% responses divisible by 10 on 0-100 visual analog scales) indicated
342 potential inattention.

343 **3. A priori exclusions:** Based on suspicious response patterns identified in
344 preliminary screening, n = [INSERIRE] participants were excluded before analysis.

345 Final sample included N = 119 participants with complete voice data and valid EMA
346 responses.

347 **Convergent Validity of EMA Measures**

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

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

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

361 **Statistical Analysis**

362 Statistical analyses were conducted in a Bayesian framework using Stan. Voice
363 outcomes (mean F0 and NNE) were modeled using hierarchical linear regression models

³⁶⁴ with repeated observations nested within participants. Experimental effects were
³⁶⁵ parameterized using two orthogonal contrasts representing stress reactivity (stress
³⁶⁶ vs. baseline) and recovery (post-stress vs. stress).

³⁶⁷ Individual differences in personality were modeled as latent traits derived from
³⁶⁸ repeated EMA assessments of the five PID-5 domains. A multivariate measurement model
³⁶⁹ was used to estimate subject-level latent trait scores while accounting for measurement
³⁷⁰ error in the observed EMA indicators. These latent traits were then included in the
³⁷¹ outcome model both as main effects and as moderators of stress and recovery effects via
³⁷² cross-level interactions.

³⁷³ All models included subject-specific random intercepts and random slopes for the
³⁷⁴ stress and recovery contrasts. Weakly informative priors were used for all fixed effects,
³⁷⁵ variance components, and correlations. Posterior inference was based on full posterior
³⁷⁶ distributions, with model adequacy evaluated using posterior predictive checks and
³⁷⁷ approximate leave-one-out cross-validation.

³⁷⁸ **Estimation.** Models were estimated using Hamiltonian Monte Carlo with 4 chains
³⁷⁹ of 5,000 iterations each (2,500 warmup). Convergence was assessed via $Rhat < 1.01$ and
³⁸⁰ effective sample size > 400 . Adaptation parameters (`adapt_delta = 0.995`, `max_treedepth`
³⁸¹ $= 18$) prevented divergent transitions. For models with convergence difficulties, we
³⁸² increased iterations or simplified random effects structures.

³⁸³ **Inference.** Effects were considered credible if 95% credible intervals excluded zero.
³⁸⁴ We report posterior means and 95% CIs throughout. For key hypotheses, we computed
³⁸⁵ Bayes factors comparing moderation models to null models without interactions.

³⁸⁶ **Data and Code Availability.** All analysis code and de-identified data will be
³⁸⁷ made publicly available upon publication at [OSF LINK]. Models were fit using `brms 2.21`
³⁸⁸ with `cmdstanr 0.7`.

389

Results

390 All analyses used hierarchical Bayesian models implemented in Stan via the rstan
391 package, with four chains of 4,000 iterations each (2,000 warmup). Convergence was
392 verified through R-hat statistics (all < 1.01) and trace plot inspection. We report posterior
393 medians with 95% credible intervals (CrIs) and directional probabilities.

394 **Main Effects of Exam-Related Stress on Vocal Acoustics**

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

403 **Fundamental frequency.** Descriptive statistics revealed a progressive pattern
404 across timepoints. Mean F0 at baseline was 190.7 Hz (SD = 22.0), increasing to 194.0 Hz
405 (SD = 21.9) immediately before the exam and then declining slightly to 192.5 Hz (SD =
406 23.6) following the exam. The hierarchical model confirmed robust stress-related elevation.
407 The intercept parameter α , representing the estimated F0 at baseline, had a posterior
408 median of 192.48 Hz (MAD = 1.73, 95% CrI [189.07, 195.96]). The stress contrast β_1
409 showed a clear positive effect: F0 increased by 3.27 Hz (MAD = 1.25, 95% CrI [0.81, 5.71])
410 when comparing pre-exam to baseline recordings, with $P(\beta_1 > 0) = 0.995$. This finding
411 indicates that acute academic stress reliably elevates vocal pitch, consistent with increased
412 laryngeal tension and autonomic arousal. The recovery contrast β_2 was essentially null
413 (median = 0.14 Hz, MAD = 1.24, 95% CrI [-2.34, 2.59], $P(\beta_2 > 0) = 0.542$), indicating that
414 F0 plateaued after the exam with minimal further change during the brief recovery period.

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

421 **Normalized noise energy.** In contrast to F0, NNE exhibited a pattern consistent
422 with reduced glottal noise under stress. Descriptively, mean NNE at baseline was -26.55 dB
423 (SD = 2.64), decreasing to -27.09 dB (SD = 3.25) at the pre-exam assessment and
424 remaining relatively stable at -26.98 dB (SD = 2.91) post-exam. More negative NNE values
425 indicate a more periodic, harmonically stable signal. The hierarchical model confirmed
426 systematic stress-induced reduction in glottal noise. The intercept had a posterior median
427 of -26.87 dB (MAD = 0.20, 95% CrI [-27.28, -26.47]). The stress contrast showed a robust
428 negative effect: NNE decreased by 0.79 dB (MAD = 0.31, 95% CrI [-1.30, -0.30]), with
429 $P(\beta_1 < 0) = 0.995$. The recovery contrast was $\beta_2 = -0.19$ dB (MAD = 0.30, 95% CrI
430 [-0.69, 0.31]). The 95% credible interval includes zero and the directional probability is
431 weak ($P(\beta_2 < 0) = 0.219$), indicating minimal systematic change during the post-exam
432 period. Random effects estimates revealed considerable between-person heterogeneity in
433 baseline NNE ($\tau_1 = 2.14$, 95% CrI [1.85, 2.46]) and in stress-related change ($\tau_2 = 0.71$, 95%
434 CrI [0.06, 1.56]). Residual variability was $\sigma = 1.98$ dB (95% CrI [1.78, 2.17]).

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

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

445 **Personality Moderation of Vocal Stress Responses**

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

453 **Arousal-related pitch responses (F0).** For F0, we estimated ten moderation
454 parameters: five domains (Negative Affectivity, Detachment, Antagonism, Disinhibition,
455 Psychoticism) crossed with two contrasts (stress, recovery). Table 1 presents posterior
456 medians, 95% credible intervals, and directional probabilities for each interaction. Among
457 these ten tests, only one showed clear evidence of moderation: Negative Affectivity
458 amplified the stress-induced increase in F0 ($\gamma_1 = 3.14$ Hz per SD, 95% CrI [0.37, 5.89], PD
459 = 0.97). This effect indicates that individuals higher in emotional reactivity and stress
460 sensitivity (Negative Affect) exhibited stronger vocal arousal responses during anticipatory
461 stress. No other stress-phase moderation effects showed meaningful Bayesian evidence.
462 Detachment and Disinhibition exhibited no credible stress-phase moderation (PD = 0.59
463 and 0.64, respectively). During the recovery phase, Detachment showed a suggestive
464 tendency toward reduced F0 elevation ($\gamma_2 = -2.02$ Hz, PD = 0.88), whereas Disinhibition
465 showed no meaningful effect (PD = 0.67).

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

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

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

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

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

471 detecting interaction effects in the recovery phase.

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

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

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

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

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

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

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

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

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

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

482 modulation (Table 2).

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

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

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

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

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

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

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

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

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

492 noise, particularly during stress de-escalation.

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

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

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

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

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

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

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

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

527 Within-Person Temporal Covariation

528 Finally, we examined whether momentary fluctuations in personality states—assessed
529 via EMA immediately before each voice recording—covaried with concurrent F0 levels.
530 This tests whether trait-level moderation effects have within-person analogs: do individuals
531 show higher F0 when they report elevated negative affectivity in the moment?

532 We compared hierarchical specifications with fixed effects (population-average slopes
533 only) versus random slopes (allowing individual differences in within-person associations).
534 Random slopes models substantially outperformed fixed-effects specifications ($R^2 = 35\%$ vs
535 2.5% ; ELPD difference ≈ 40 points). However, with only three observations per person,
536 posterior distributions for individual slopes were extremely wide—99.7% of participant \times
537 domain combinations yielded credible intervals spanning zero.

538 This pattern reveals a critical distinction: the population shows clear evidence of
539 slope heterogeneity (nonzero σ_β for several domains), but we cannot reliably identify which
540 specific individuals have nonzero slopes. This reflects statistical power limitations rather

541 than modeling failure. Denser temporal sampling would be required to resolve
542 individual-level within-person dynamics.

543 Discussion

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

553 **Vocal acoustics as objective indicators of situational stress.** Exam-related
554 stress produced dissociable changes in vocal production. Consistent with prior
555 psychophysiological work, F0 increased during the pre-exam assessment, indicating
556 heightened laryngeal muscle tension and autonomic arousal (Giddens et al., 2013; Scherer,
557 2003). Importantly, this effect emerged in a real-world evaluative context rather than an
558 experimentally induced laboratory stressor, supporting the ecological validity of vocal pitch
559 as an objective marker of stress reactivity in everyday life.

560 In contrast to a simple “vocal degradation” account, NNE decreased by 0.79 dB
561 during stress, indicating a more periodic, less noisy signal. This pattern suggests that
562 evaluative stress can involve not only arousal-related activation (indexed by F0) but also
563 *compensatory phonatory control* (indexed by reduced glottal noise). Under
564 performance-oriented conditions, individuals may unconsciously adopt more effortful or
565 pressed phonation that increases periodicity, even while physiological arousal rises. This
566 arousal-control dissociation implies that stress responses in the voice are multidimensional

567 rather than unitary, and that different personality domains may preferentially shape
568 different components of the response.

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

579 **Domain- and phase-specific moderation of vocal stress responses.** Given
580 the absence of prior research on personality moderation of vocal stress markers, we adopted
581 an exploratory approach for most PID-5 domains, specifying a directional hypothesis only
582 for Negative Affectivity. The resulting pattern provides evidence that personality
583 pathology domains do not exert broad, uniform effects; instead, they appear to moderate
584 *specific acoustic parameters at specific phases* of the stress-response cycle.

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

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

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

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

606 Psychoticism showed minimal moderation of F0, aligning with mixed prior evidence on
607 physiological stress reactivity in schizotypy-related traits. However, Psychoticism uniquely
608 moderated *NNE during recovery*, with higher Psychoticism associated with increased
609 glottal noise (less negative NNE) post-exam. This specificity to a voice quality parameter,
610 and to the recovery phase, suggests that Psychoticism may be linked less to the magnitude
611 of acute arousal and more to processes involved in stabilizing phonatory control after stress
612 exposure. One interpretation is that cognitive–perceptual dysregulation may compromise
613 adaptive recalibration of motor control once stress cues recede, yielding less stable
614 phonation. Alternative explanations are also plausible, including persistent autonomic
615 dysregulation or context-dependent changes in speech effort. Discriminating among these
616 accounts will require concurrent measurement of autonomic markers (e.g., heart rate
617 variability) and, ideally, more continuous sampling across the recovery period.

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

619 Detachment showed weak evidence for *improved* rather than impaired F0 recovery (PD =

620 0.88, negative coefficient). This pattern is noteworthy because it runs counter to
621 predictions derived from the emotional inertia literature, which links restricted affectivity
622 and anhedonia to prolonged negative states and impaired affect repair (Koval et al., 2015;
623 Kuppens et al., 2010). One possible interpretation is that the emotional blunting
624 characteristic of Detachment may attenuate not only positive emotional experiences but
625 also sustained physiological activation following stress: if high-Detachment individuals are
626 less engaged with the evaluative implications of the stressor, they may show faster
627 normalization simply because there is less activation to resolve. Given the modest strength
628 of this effect, it should be treated cautiously and may represent sampling variability, but it
629 raises the possibility that restricted affectivity could function as a context-specific
630 protective factor in acute stress—even if it reflects broader affective impairment in other
631 domains.

632 Disinhibition showed no meaningful moderation of either acoustic parameter (all PD
633 < 0.75). This null finding is theoretically coherent rather than merely uninformative.
634 Unlike Negative Affectivity, which reflects arousal sensitivity and emotional reactivity,
635 Disinhibition is defined by impulsivity, irresponsibility, and poor behavioral
636 control—characteristics pertaining to volitional action regulation rather than automatic
637 physiological processes (Krueger et al., 2012). Meta-analytic evidence indicates that
638 conscientiousness-related traits show weak associations with physiological stress responses
639 despite moderating subjective stress perception and coping behaviors (Luo et al., 2023).
640 The present findings extend this dissociation to vocal acoustics: because F0 elevation
641 reflects automatic autonomic mechanisms (laryngeal tension from sympathetic activation)
642 rather than behavioral control processes, Disinhibition—which governs volitional rather
643 than automatic responses—would not be expected to moderate this pathway. More
644 broadly, these results reinforce the conclusion that moderation effects are not ubiquitous
645 across PID-5 domains but are *selective and mechanism-specific*, with different domains
646 operating through distinct psychophysiological pathways (Calà et al., 2025).

647 **Implications for transactional models of personality pathology.** The
648 dissociations observed across trait domains (Negative Affectivity, Antagonism,
649 Psychoticism), acoustic parameters (F0 vs. NNE), and temporal phases (stress reactivity
650 vs. recovery) are consistent with transactional models in which personality pathology
651 shapes *how* individuals respond to contextual demands rather than simply predicting static
652 levels of functioning (Bolger & Zuckerman, 1995). In this view, different domains may map
653 onto distinct stress-process mechanisms: Negative Affectivity primarily amplifies arousal
654 reactivity; Antagonism may be linked to persistence of activation following stress; and
655 Psychoticism may be associated with post-stressor instability in control-related vocal
656 processes. Importantly, these interpretations remain provisional and should be tested in
657 designs that incorporate richer characterization of situational features and more continuous
658 measurement during recovery.

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

666 **Methodological contribution: intensive ambulatory trait assessment.** A
667 central methodological contribution of this project is to show that brief trait measurements
668 assessed repeatedly via EMA can perform comparably to comprehensive baseline
669 questionnaires for predicting vocal stress trajectories. Across models, out-of-sample
670 predictive performance was similar for EMA-based and baseline-based PID-5 measures,
671 indicating a substantial overlap in the trait variance they capture. However, EMA-based
672 measurement yielded *more precise moderation estimates*, consistent with the idea that
673 repeated sampling can reduce measurement error and stabilize person-level trait estimates.

674 This distinction is important: even when overall predictive accuracy is equivalent,
675 improved inferential precision can strengthen theory tests of trait-by-context interactions.

676 These results support the feasibility of combining low-burden repeated trait
677 assessment with objective acoustic markers in longitudinal designs, particularly when
678 comprehensive questionnaires are impractical. At the same time, the present design relied
679 on brief standardized voice recordings rather than continuous passive sensing. Extending
680 this approach to more frequent recordings would allow stronger tests of within-person
681 coupling and more detailed modeling of recovery dynamics.

682 **Limitations and future directions.** Several limitations qualify the conclusions.
683 First, the sample consisted of female university students, and we excluded participants
684 with psychiatric disorders requiring treatment. Although PID-5 traits are dimensionally
685 meaningful in community samples, generalization to clinical populations and to males
686 requires replication. Second, although the examination period provides a naturalistic
687 stressor with real stakes, it is not equivalent to interpersonal stressors that are central to
688 many forms of personality pathology. Different stressor classes may yield different vocal
689 signatures and trait moderation patterns. Third, vocal data were collected at only three
690 time points, which constrains the temporal resolution with which stress-related dynamics
691 can be characterized. Although this design captures broad phases of baseline, anticipatory
692 stress, and early recovery, it cannot resolve the finer-grained time course through which
693 vocal parameters return to baseline following stressor offset. As a result, apparent
694 persistence of stress-related effects may reflect delayed physiological recovery, continued
695 contextual activation, or both. Future studies employing denser sampling, particularly in
696 the hours immediately following stress exposure, will be necessary to disentangle these
697 processes and to model recovery trajectories with greater precision. Fourth, vocal markers
698 are biologically plausible indices of stress, but they are not process-pure. Changes in F0
699 and NNE may reflect multiple mechanisms, including autonomic activation, cognitive load,
700 strategic self-presentation, and speech effort. Future studies should triangulate voice

701 measures with concurrent physiological indicators (e.g., heart rate variability, electrodermal
702 activity) and with contextual measures (perceived stress, task difficulty, preparedness) to
703 clarify underlying pathways.

704

References

- 705 • Bolger, N., & Zuckerman, A. (1995). A framework for studying personality in the
706 stress process. *Journal of Personality and Social Psychology*, 69(5), 890.
- 707 • Fleeson, W. (2001). Toward a structure-and process-integrated view of personality.
708 *Journal of Personality and Social Psychology*, 80(6), 1011.
- 709 • Giddens, C. L., Barron, K. W., Byrd-Craven, J., Clark, K. F., & Winter, A. S.
710 (2013). Vocal indices of stress: A review. *Journal of Voice*, 27(3), 390-e21.
- 711 • Hopwood, C. J., Bleidorn, W., & Wright, A. G. (2022). Connecting theory to
712 methods in longitudinal research. *Perspectives on Psychological Science*, 17(4),
713 884-894.
- 714 • Kent, R. D., & Kim, Y. (2003). Toward an acoustic typology of motor speech
715 disorders. *Clinical Linguistics & Phonetics*, 17(6), 427-445.
- 716 • Krueger, R. F., Derringer, J., Markon, K. E., Watson, D., & Skodol, A. E. (2012).
717 Initial construction of a maladaptive personality trait model and inventory for
718 DSM-5. *Psychological Medicine*, 42(9), 1879-1890.
- 719 • Mischel, W., & Shoda, Y. (1995). A cognitive-affective system theory of personality.
720 *Psychological Review*, 102(2), 246.
- 721 • Scherer, K. R. (2003). Vocal communication of emotion: A review of research
722 paradigms. *Speech Communication*, 40(1-2), 227-256.
- 723 • Scherer, K. R., Johnstone, T., & Klasmeyer, G. (2013). Vocal expression of emotion.
724 In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective
725 sciences* (pp. 433-456). Oxford University Press.
- 726 • Trull, T. J., & Ebner-Priemer, U. W. (2020). Ambulatory assessment in
727 psychopathology research: A review of recommended reporting guidelines and current
728 practices. *Journal of Abnormal Psychology*, 129(1), 56.
- 729 • Wright, A. G., & Simms, L. J. (2016). Stability and fluctuation of personality
730 disorder features in daily life. *Journal of Abnormal Psychology*, 125(5), 641.

- 731 • Wright, A. G., Gates, K. M., Arizmendi, C., Lane, S. T., Woods, W. C., & Edershile,
732 E. A. (2019). Focusing personality assessment on the person. *Assessment*, 26(3),
733 403-419.
- 734 American Psychiatric Association. (2014). *Online assessment measures: The personality
735 inventory for DSM-5 (adult)*.
736 <https://www.psychiatry.org/getmedia/594673a6-1b9b-4298-8b52-c4c652c4a4e2/APA-DSM5TR-ThePersonalityInventoryForDSM5FullVersionAdult.pdf>.
- 738 Bibbey, A., Carroll, D., Roseboom, T. J., Phillips, A. C., & Rooij, S. R. de. (2013).
739 Personality and physiological reactions to acute psychological stress. *International
740 Journal of Psychophysiology*, 90(1), 28–36.
- 741 Bottesi, G., Caudek, C., Colpizzi, I., Iannattone, S., Palmieri, G., & Sica, C. (2024).
742 Advancing understanding of the relation between criterion a of the alternative model for
743 personality disorders and hierarchical taxonomy of psychopathology: Insights from an
744 external validity analysis. *Personality Disorders: Theory, Research, and Treatment*.
- 745 Calà, F., Colpizzi, I., Sica, C., Caudek, C., Lanatà, A., & Frassineti, L. (2025). A
746 preliminary analysis on longitudinal effects of exam stress and personality traits over
747 acoustic properties. *Models and Analysis of Vocal Emissions for Biomedical
748 Applications: 15th International Workshop*, 145–148.
- 749 Colpizzi, I., Trull, T. J., Sica, C., Haney, A. M., & Caudek, C. (2025). State
750 self-compassion dynamics: Partial evidence for the bipolar continuum hypothesis.
751 *Mindfulness*, 1–16.
- 752 Dietrich, M., & Abbott, K. V. (2012). Vocal function in introverts and extraverts during a
753 psychological stress reactivity protocol. *Journal of Speech, Language, and Hearing
754 Research*, 55(3), 973–987. [https://doi.org/10.1044/1092-4388\(2011/10-0344\)](https://doi.org/10.1044/1092-4388(2011/10-0344))
- 755 Gelfer, M. P., & Mikos, V. A. (2005). The relative contributions of speaking fundamental
756 frequency and formant frequencies to gender identification based on isolated vowels.
757 *Journal of Voice*, 19(4), 544–554.

- 758 Giddens, C. L., Barron, K. R., Byrd-Craven, J., Clark, K. F., & Winter, A. S. (2013).
759 Progressive vocal stress modeling. *Behavioral Sciences*, 3(4), 571–587.
- 760 Kasuya, H., Ogawa, S., Mashima, K., & Ebihara, S. (1986). Normalized noise energy as an
761 acoustic measure to evaluate pathologic voice. *The Journal of the Acoustical Society of
762 America*, 80(5), 1329–1334.
- 763 Luo, J., Zhang, B., Cao, M., & Roberts, B. W. (2023). The stressful personality: A
764 meta-analytical review of the relation between personality and stress. *Personality and
765 Social Psychology Review*, 27(2), 128–194.
- 766 Manfredi, C., Lebacq, J., Cantarella, G., Schoentgen, J., Orlandi, S., Bandini, A., &
767 DeJonckere, P. H. (2017). Smartphones offer new opportunities in clinical voice
768 research. *Journal of Voice*, 31(1), 111–e1.
- 769 Mendoza, E., & Carballo, G. (1998). Acoustic analysis of induced vocal stress by means of
770 cognitive workload tasks. *Journal of Voice*, 12(3), 263–273.
- 771 Morelli, M. S., & Manfredi, S. O. C. (2019). BioVoice: A multipurpose tool for voice
772 analysis. *Proceedings of the 11th International Workshop Models and Analysis of Vocal
773 Emissions for Biomedical Applications, MAVEBA 2019*, 261–264. Firenze University
774 Press Firenze, Italy.
- 775 Scherer, K. R. (2003). Vocal expression of emotion. *Handbook of Affective Sciences*,
776 433–456.
- 777 Sica, C., Caudek, C., Colpizzi, I., Bottesi, G., Iannattone, S., & Patrick, C. J. (2024).
778 Comparing the DSM-5 dimensional trait and triarchic model conceptions of
779 psychopathy: An external validity analysis. *Journal of Personality Disorders*, 38(4),
780 368–400.
- 781 Titze, I. R. (1994). *Principles of voice production*. Prentice Hall.
- 782 Trull, T. J., & Ebner-Priemer, U. W. (2020). Ambulatory assessment in psychopathology
783 research: A review of recommended reporting guidelines and current practices. *Journal
784 of Abnormal Psychology*, 129(1), 56–63. <https://doi.org/10.1037/abn0000473>

- 785 Van Puyvelde, M., Neyt, X., McGlone, F., & Pattyn, N. (2018). Voice stress analysis: A
786 new framework for voice and effort in human performance. *Frontiers in Psychology*, 9,
787 1994.

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.