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Psychiatric symptom versus neurocognitive correlates of diminished expressivity in schizophrenia and mood disorders

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ABSTRACT

Diminished expressivity is a poorly understood, but important construct for a range of mental diseases. In the present study, we employed computerized acoustic analysis of natural speech to understand diminished expressivity in patients with schizophrenia and mood disorders. We were interested in the degree to which speech characteristics tapping alogia (i.e., average pause duration) and blunted affect (i.e., prosody computed from fundamental frequency and intensity) reflected psychiatric symptoms (i.e., depression, anxiety, paranoia and bizarre behavior) versus neurocognitive deficits. Twenty-six subjects with schizophrenia and 22 subjects with mood disorders provided speech samples in response to a variety of laboratory stimuli and completed neuropsychological batteries assessing a range of abilities. For both the schizophrenia and mood disorder groups, attentional coding deficits were significantly correlated with increased pause time (at large effect size levels) and, for the schizophrenia group only, reduced prosody (also at a large effect size level). For the mood disorder but not the schizophrenia group, increased average pause time was also significantly associated with neurocognitive deficits on a range of other tests (medium to large effect size levels). Psychiatric symptoms were not significantly associated with speech characteristics for either group (generally, negligible effect sizes). These results suggest that there is a link between expressivity and neurocognitive dysfunctions for both patients with schizophrenia and mood disorders. Implications and future research directions are discussed.

1. Introduction

A reduction in expressive behaviors, which include symptoms such as blunted affect, motor retardation and alogia, manifests across a range of severe mental illnesses. Of note, clinically-rated expressive deficits have been found to be similar in severity across patients with schizophrenia and mood disorders (Kulhara and Chadda, 1987; Galynker et al., 2000; Tremeau et al., 2005; Mueser et al., 2010). Despite these deficits reflecting important Research Domain Criteria (RDoC; Insel et al., 2010), and hence, being useful for improving diagnosis and understanding of clinical syndromes (Insel et al., 2010; Cohen et al., 2012a), our understanding of their nature is poor. A critical obstacle in this endeavor is a reliance on interviewer-based rating scales for measuring diminished expression (e.g., Andreasen, 1984). Data from these scales often cover wide temporal swaths, are relatively insensitive to change given their relatively few response options and ambiguous operational definitions, produce ordinal data that are inappropriate for parametric statistics and are imprecise for isolating specific behaviors from other negative traits/symptoms (Mueser et al., 1994; Alpert et al., 2002; Stahl and Buckley, 2007; Cohen et al., 2008). Thus, these scales have limited use for providing all but a gross understanding of how expressive deficits modulate within individuals, how they differ across individuals, and how they are broadly related to neurocognitive, functional, pathophysiological, genetic and other variables. Emerging computer-based technologies have allowed for assessment of natural behavior in individuals with severe mental illness that offer near perfect inter-rater reliability and greater sensitivity and specificity than clinical rating scales (Alpert et al., 2002; Cohen et al., 2008). The present project applied computerized acoustic analysis of natural speech to understand the underpinnings of expressive deficits in stable outpatients with severe mental illness.

Within schizophrenia, it has been proposed that negative symptoms can reflect a number of potential causes, including "psychiatric" sources such as depression, anxiety, bizarre behavior (e.g., catanoia) and paranoia. Thus, a patient's lack of communicative behavior may stem from being too depressed, socially anxious, disorganized or suspicious of others. Alternatively, it has been proposed that expressive deficits can reflect basic neurocognitive liabilities (Barch and Berenbaum, 1996; Berenbaum and Oltmanns, 1992; see also Cohen et al., 2012c for a more recent discussion). Generally speaking, effective expression is theorized to draw upon a range of mental resources, and taxing these resources limits their availability for expressive behavior. In patients with severe mental illness, these expressive deficits could be magnified

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relative to the nonpsychiatric population due to weakened cognitive stores more generally. Support for this theory has been found in that experimental increases in cognitive demands during speaking tasks lead to decreases in expressive behavior in individuals with schizophrenia-spectrum disorders/traits (Barch and Berenbaum, 1996; Cohen et al., 2012c). It is worth noting that patients with schizophrenia and mood disorders have shown similar levels of neurocognitive impairment across a range of domains (e.g., Šoštarič & Zalar, 2011; Cohen et al., 2012a) — thus raising questions about whether neurocognitive dysfunctions are similarly related to expressive deficits for both groups. Within these patient groups, individuals with negative symptoms (Cohen et al., 2007) and melancholic depression (Withall et al., 2009) tend to show greater neurocognitive deficits relative to other patients within their diagnostic categories. With this in mind, the primary aim of this study was to evaluate the hypothesis that diminished expressivity would be differentially associated with neurocognitive versus "psychiatric" (e.g., anxiety, depressive, bizarre behavior and paranoia symptoms) factors, and that the relationship between these variables would not differ for patients with schizophrenia versus mood disorders. A secondary aim of the project was to compare the contribution of a) neurocognitive tests primarily tapping attentional/working memory processes (i.e., processing speed, concentration, working memory) to b) neurocognitive domains that are less dependent on attentional processes, in terms of diminished expressivity.

2. Methods

2.1. Subjects

The subjects were recruited from an outpatient community mental health clinic. These subjects included 26 patients with Diagnostic & Statistical Manual of Mental Disorders 4th edition (DSM-IV; American Psychiatric Association, 1994) diagnosed schizophrenia and 22 patients with a history of DSM-IV major depressive episode without a history of schizophrenia-spectrum disorder. Diagnoses were made based on information obtained from the patients' medical records and from a structured clinical interview (SCID; First et al., 1996). Exclusion criteria included the following: a) Global Assessment of Functioning (American Psychiatric Association, 1994) rating below 30, indicating symptom levels that could interfere with participation in the study, b) documented evidence of mental retardation from the medical records, c) current or historical DSM-IV diagnosis of alcohol or drug abuse suggestive of severe physiological symptoms (e.g., delirium tremens), and d) history of significant head trauma (requiring overnight hospitalization). All patients were clinically stable at the time of testing and were receiving pharmacotherapy under the supervision of a multi-disciplinary team. The patients received $40 for participation in this study. This study was approved by the appropriate Human Subject Review Boards and all the subjects offered informed consent prior to participating in the study. For further information on recruitment, see other published studies from this dataset (Cohen et al., 2012a, 2012b, 2012c).

2.2. Diagnostic and symptom ratings

The Brief Psychiatric Rating Scale (Lukoff et al., 1986) was used to characterize psychiatric symptoms. BPRS ratings were made using information obtained from medical records, the patients' treatment teams, self-report and behavioral observations made during the research interview. Individual subscales of anxiety, depression, suspiciousness and bizarre behavior, reflecting potential "psychiatric symptom" correlates of diminished expression (Kirkpatrick et al., 2001), were employed using scales from one to seven. Factor subscale scores reflecting positive (i.e., bizarre behavior, suspiciousness, unusual thought content, disorientation, and hallucinations items), negative (i.e., self-neglect, blunted affect, motor retardation, and emotional withdrawal items), and mania/excitement (i.e., motor hyperactivity, elevated mood, excitement, distractibility, hostility, and grandiosity items) symptoms (defined in Ventura et al., 2000) were also employed. Preliminary diagnoses and ratings were made by one of four doctoral level students who were trained to criterion (Intra-class Correlation Coefficient (ICC) values > .70). All research interviews were videotaped and diagnoses and ratings were reviewed during a monthly case conference meeting that was led by a licensed clinical psychologist with considerable diagnostic experience (Alex S. Cohen).

2.3. Speech samples

The subjects were asked to view and speak about affectively-valenced pictures from the International Affective Picture System (IAPS; Lang et al., 2005) in six separate, three-picture blocks grouped by emotional valence (good, bad and neutral) and arousal (high and low). Administrations, which employed different images, were performed twice — separated by a week epoch. The pictures were selected for their relative representation of their respective valence and arousal based on existing norms (Lang et al., 2005). Picture display was set at 20 s, and each speaking condition was exactly 60 s. Block order and picture order within each block were random. Blocks were separated by a 30-second interval during which the subjects were instructed to "relax and breathe deeply". While viewing the pictures, the subjects were asked to "discuss how the picture relates to them, what it means to them, what it reminds them of, and how it makes them feel". The subjects were encouraged to speak for the full recording time. Research assistants were not allowed to speak during the task, though hand gestures encouraging subjects to talk were permitted. In all, 720 s of speech was available for analysis for each subject.

2.4. Acoustic analysis

The Computerized assessment of Affect from Natural Speech protocol (Cohen et al., 2009, 2010), developed by our lab to assess vocal expression, was employed here. Speech was digitally recorded using headset microphones at a sampling rate of 44,100 kHz with 16-bit quantization. The digitized recordings were analyzed using PRAAT (Boersma and Weenink, 2006), a program that has been used extensively in acoustic analysis. The PRAAT system organizes sound files into "frames" for analysis which for the present study was set at a rate of 100 per second. During each of these frames, frequency and volume were quantified. Various MATLAB and Excel Macro functions were employed to compute our variables of interest from the PRAAT output. The variables examined in this study included the following: *average pause time* ($Pause_{\bar{x}}$) — computed as the average millisecond pause between utterances; *inflection* — computed as the standard deviation of the fundamental frequency, computed from the standard deviation scores within each utterance; *intensity* — computed as the mean volume across utterances, and *emphasis* — computed as the standard deviation of the volume, computed from the standard deviation scores within each utterance. The first symptom maps onto the construct of alogia whereas the others map onto blunted affect. The inflection, intensity and emphasis variables were converted to z-score format and summed to reduce the overall number of analyses. This variable is referred to as "prosody" in this paper. Note that all frequency values were log-transformed to control for nonlinear distributions. Increasing $Pause_{\bar{x}}$ and decreasing prosody values reflect increasing expressive deficits. For data reduction purposes, speech variables were aggregated across the various valence and arousal speaking conditions and across the two administrations. There were no significant changes in speech production as a function of group, time, valence or arousal using repeated measure × group ANOVAs after controlling for group differences in ethnicity. Data regarding temporal stability and group differences (as well as means and variability scores for these variables) are reported elsewhere (Cohen et al., 2012a).

2.5. Neurocognitive functioning

Neurocognitive abilities were assessed using the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph et al., 1998) a battery consisting of 5 domains: immediate verbal memory (list learning, story memory), visual–spatial (figure copy, line orientation), language (picture naming, semantic fluency), attention (digit span, coding) and delayed memory (list recall, story recall, picture recall). Due to low correlations between the digit span and coding tasks ($r[48] = .19$, $p = .21$), these tests were examined separately as opposed to combining them for the attention domain. Internal consistency, inferred from significant correlation values, was observed for the other domains. The RBANS is sensitive to neurocognitive impairments commonly observed in schizophrenia (Gold et al., 1999), and is relatively efficient to administer, sensitive, reliable, highly correlated with other neurocognitive batteries and related to functioning variables, such as employment status (Gold et al., 1999).

2.6. Analyses

Analyses were conducted in three steps. First, we computed descriptive and clinical variables for the schizophrenia and mood disorder groups to identify variables that might need to be controlled for in subsequent analyses. Second, we computed Spearman's correlations, a nonparametric statistic appropriate for analysis using relatively small sample sizes, among RBANS neurocognitive scores, psychiatric symptom scores and speech characteristics. We hypothesized that neurocognitive performance would be significantly associated with shorter average pause times and higher prosody for each group. We lacked a priori hypotheses regarding correlations involving psychiatric symptoms. Fisher r-to-z transformations were conducted to compare correlations when appropriate (Meng et al., 1992). Finally, we employed hierarchical regressions to evaluate the relative contributions to average pause time and prosody scores (dependent variables) made by psychiatric symptoms from the BPRS (step 2), global neurocognition (step 4; computed as a sum of all RBANS domains besides attention) and attention domain scores (step 6). Interaction terms were entered to determine whether the groups differed in their relationships between psychiatric symptoms × diagnostic group (step 3), global neurocognition × diagnostic group (step 5) and attention × group (step 7). Diagnostic group was entered in step 1. We hypothesized that steps four and six would be statistically significant for each speech characteristic, and that steps five and seven would not be significant. That is, we expected that both global neurocognitive and attentional performance would significantly contribute to the variance in speech characteristics, and that this contribution would not significantly differ between patients with schizophrenia versus mood disorder diagnoses. For reasons explicated below, attentional functioning was assessed using coding but not digit span scores in the regression analyses. All analyses in this study are two-tailed and all variables are normally distributed unless otherwise stated.

3. Results

3.1. Descriptive statistics

Test statistics for comparisons between the schizophrenia and mood disorder groups in descriptive, clinical and speech variables are presented in Table 1. The groups were largely similar in terms of sex, symptoms, neurocognitive performance, speech characteristics, and functioning. The schizophrenia group was significantly younger, more likely to be African-American and had more severe bizarre-behavior symptoms. Caucasians and African-Americans did not significantly differ for any of the speech measures, nor did males and females (ps > .10). Age was not significantly associated with any of the speech characteristics (ps > .10). All analyses in this study were recomputed including age, ethnicity and sex as covariates without substantive change (unless otherwise noted).

3.2. Correlational analyses

Table 2 contains the bivariate correlations between speech characteristics and neurocognitive and clinical variables. For the mood disorder group, increasing pause time was significantly associated with poorer immediate memory, language and coding performance. For the schizophrenia group, increasing pause time and decreasing prosody were both significantly associated with poorer coding performance. There are several other notable findings from the correlation analyses. First, none of the other 10 correlations between speech characteristics and neurocognition for the schizophrenia group exceeded a value of .16. In contrast, six of the 12 total correlations for mood disorder group exceeded a medium effect size level (e.g., r > .30), and all but two exceeded a small effect size level (e.g., rs > .17). For the schizophrenia group, the correlations between coding and prosody/pause time were significantly different than those for other neurocognitive scores and prosody/alogia (zs > 2.08, ps < .04). For the mood disorder group, the correlations between various neurocognitive scores and prosody/pause time were generally not different from each other. Second, the correlations between coding and prosody were significantly different in magnitude for the schizophrenia versus mood disorder groups, based on Fisher r-to-z transformations ($z = 2.03$, $p = .04$). The differences in magnitude between groups for the other correlations did not rise to the level of statistical significance (zs < 1.57, ps > .11).

With respect to the symptom correlates, none of the psychiatric symptoms were significantly associated with average pause length or prosody for either group. Only one of these correlations exceeded a small effect size level while 10 of the 16 correlations were in the negligible range. Depression severity was associated with *increasing*

Table 1
Descriptive statistics for demographic and clinical variables for the mood disorder and schizophrenia groups.

	Mood disorder Mean (SD)	Schizophrenia Mean (SD)	Test statistic
Age	46.17 (9.69)	39.87 (9.96)	2.31*,b
% Caucasian	70%	33%	6.84**,a
%African-American	30%	67%	
% Male	61%	67%	.19[a]
Number of hospitalizations	4.23 (4.69)	4.55 (4.80)	.24[b]
Psychiatric history			
Major depression	100%	47%	17.57**,a
Manic episodes	44%	27%	1.64[a]
Psychosis symptoms	30%	100%	29.89**,a
Psychiatric symptoms			
Anxiety	3.64 (1.76)	3.17 (1.85)	.91[b]
Depression	3.77 (2.14)	3.14 (1.79)	1.16[b]
Suspiciousness	2.27 (1.24)	2.79 (1.54)	1.29[b]
Bizarre behavior	1.32 (.64)	1.93 (1.22)	2.13*,b
Clinical syndromes			
Manic–Excitement	10.23 (5.47)	10.39 (4.63)	.26[b]
Negative	5.68 (2.98)	8.52 (3.70)	2.61*,b
Positive	7.78 (3.10)	12.26 (5.30)	3.53**,b
RBANS			
Immediate memory[d]	82.26 (18.62)	77.52 (13.24)	.99[b]
Verbal comprehension[d]	89.05 (18.95)	81.32 (17.93)	1.38[b]
Language[d]	91.53 (11.13)	89.24 (12.05)	.64[b]
Digit span (raw score)	10.71 (3.33)	10.15 (2.43)	.50[b]
Coding (raw score)	37.57 (9.63)	38.52 (10.95)	.76[b]
Delayed memory[d]	89.33 (13.76)	83.85 (16.30)	.22[b]
GAF	51.05 (8.97)	47.50 (9.44)	1.33[b]
Speech characteristics			
Pause $_{\bar{x}}$	4142.04 (2772.66)	4071.10 (2838.33)	.08[c]
Prosody	−.33 (1.97)	.27 (2.02)	1.05[c]

* = p < .05, ** = p < .01, a = chi-square value, b = t value, c = omnibus F condition value reflecting both T1 and T2, d = Standard Score. GAF = Global Assessment of Functioning Score, Pause $_{\bar{x}}$ = average pause time.

Table 2
Spearman's correlations between speech characteristics and neurocognitive, psychiatric symptom, and clinical variables.

	Mood disorder		Schizophrenia	
Neurocognitive measures	Pause $_{\bar{x}}$	Prosody	Pause $_{\bar{x}}$	Prosody
Immediate memory	−.45*	−.20	−.04	.04
Verbal comprehension	−.15	−.35	−.16	.01
Language	−.49*	−.03	−.05	−.06
Digit span (DS)	−.28	−.21	−.04	.03
Coding	−.58*	−.08	−.60**	.50**
Delayed memory	−.35	−.38	.00	.10
Neurocognitive sum (no coding or DS)	−.52*	−.17	−.09	.15
Psychiatric symptoms				
Anxiety	−.23	−.04	−.01	.19
Depression	.02	.04	.04	.34+
Suspiciousness	−.06	.28	.05	.07
Bizarre behavior	.23	.18	.09	.02
Sum of psychiatric symptoms	−.03	.19	.04	.31
Manic excitement factor	−.07	.50*	−.15	.21
Negative symptom factor	.31	−.15	.36+	−.34+
Positive symptom factor	.21	.30	.28	.05
GAF	−.42+	−.17	.09	−.12

+ = p<.10, * = p<.05, ** = p<.01. GAF = Global Assessment of Functioning Score, Pause $_{\bar{x}}$ = average pause time,

prosody at a trend level for the schizophrenia group. Increasing severity of manic–excitement symptoms was significantly associated with *increasing* prosody for the mood disorder group. Finally, increasing severity of negative symptoms was associated with greater pause time and less prosody at a trend level for the schizophrenia group.

3.3. Regression analyses

Although we originally hypothesized that attention, defined in terms of both digit span and coding performance, would be associated with speech characteristics, it was clear from the correlational analyses that this would not be the case. Accordingly, for the regression analyses (see Table 3), we evaluated attention exclusively in terms of coding. It is worth noting that the digit span task from the RBANS includes only digits forward and not backward, and is more a measure of brief concentration than of working memory. In total, the models explained between 26% (i.e., prosody) and 40% (i.e., average pause time) of the variance in the speech measures. There are four notable findings. First, with respect to average pause time, the contribution of global neurocognition (step 4) was statistically significant. Second, with respect to both average pause time and prosody, the contribution of coding (step 6) was significant. Third, none of the interactions were significant. Finally, the contribution of psychiatric symptoms for neither regression was significant, though it was not insubstantial for both average pause time and prosody. Collectively, these results suggest that neurocognitive variables, in particular, those related to attentional coding, are important for understanding speech characteristics — more so than psychiatric symptoms.

4. Discussion

The present study is the first to our knowledge to employ computerized acoustic analysis of natural speech to understand the relative contributions of psychiatric versus neurocognitive correlates of expressive deficits in stable outpatients with schizophrenia and mood disorders using acoustic analysis of natural speech. Overall, the present findings suggest that neurocognitive abilities are important for understanding at least some aspects of diminished expressivity in both patients with schizophrenia and mood disorders. At the same time, we found that psychiatric symptoms, at least those related to depression, anxiety, bizarre behavior and paranoia, were not significantly associated with expressive deficits in any meaningful regard. Thus, it

Table 3
Relative contributions of psychiatric symptoms, global neurocognition and coding performance to speech characteristics (dependent variables) in schizophrenia and mood disorder patient groups.

	Pause $_{\bar{x}}$		Prosody	
	ΔR^2	ΔF	ΔR^2	ΔF
Step 1: Group	.00	.04	.02	1.10
Step 2: Psychiatric symptoms	.04	1.97	.07	3.36
Step 3: Psychiatric symptoms × group	.07	3.59	.00	.14
Step 4: Global neurocognition	.14	8.14**	.00	.05
Step 5: Global neurocognition × group	.02	1.16	.03	1.54
Step 6: Coding	.11	7.82**	.09	4.94*
Step 7: Coding × group	.01	.58	.03	1.57

* = p<.05, ** = p<.01. Pause $_{\bar{x}}$ = average pause time.

seems reasonable to conclude that neurocognition is an important factor above and beyond psychiatric symptoms for understanding expressive deficits in severe mental illness. With some potential caveats detailed below, these findings appear to hold for both patients with schizophrenia and those with mood disorders. These findings provide potential insight into a treatment-resistant and deleterious facet of psychopathology.

Based on the present findings, there are some interesting implications for future research. First, it is worth noting that coding performance was associated with speech characteristics even after controlling for global neurocognitive performance. This would imply that some aspect of coding ability not captured as well by the other tests, possibly involving attentional vigilance, processing speed and psychomotor abilities, is particularly important for understanding diminished expressivity. Attributing this pattern of scores to a "differential deficit" in attentional abilities is problematic in that this study was correlational in design. Nonetheless, prior experimental studies support the notion that increases in attentional demands are associated with diminished expressivity in individuals with schizophrenia and schizotypy (Barch and Berenbaum, 1996; Cohen et al., 2012c). It is unclear why digit span scores were not significantly associated with diminished expressivity in this study, though it is noteworthy that the measure used in this study employs digits forward, not backward, and is thus, probably more a measure of brief concentration than of working memory. Collectively, these findings may be helpful for future studies attempting to isolate the neurocognitive substrates of diminished expressivity. Second, there is reason to suspect that there may be meaningful differences in how neurocognition is related to diminished expressivity in patients with schizophrenia versus mood disorders. Of note, coding was highly related to prosody for the schizophrenia but not for the mood disorder groups, and increased pause time was seemingly associated with a wider network of neurocognitive correlations for the mood disorder than the schizophrenia groups. Although the present study was not effectively powered or designed for evaluating these group differences, they do bear importance for future studies. Perhaps expressive deficits have different correlates in schizophrenia versus depression.

Some limitations warrant mention. First, while all of the subjects in this study were medicated and psychiatrically stable, it is impossible to meaningfully control for differences in medication type or dosage. There is little reason to think that individual differences in medication prescriptions affected the results of this study in any substantial way, particularly since patients with schizophrenia and mood disorders were similar in most respects. Second, there was no non-psychiatric control group for reference. The present findings are still quite informative since the most interesting findings regarded within-group differences. Third, the modest sample size was underpowered for detecting small effects. It is possible that some of the interactions in the regression analysis, notably involving psychiatric symptoms and average pause time, would have been statistically significant with a larger sample size. This does not, however, detract

from the important implications raised by the relative difference in expressive deficits accounted for by the primary neurocognitive versus psychiatric symptom factors. Fourth, the present study measured only blunted affect related to vocal expression and did not capture facial expressions and hand gestures, or even other negative symptoms such as anhedonia or amotivation. Fifth, the speaking task was somewhat artificial in that it involved speaking about evocative images, so the significant correlations between speech characteristics and neurocognition may, in part, be context specific. Sixth, most subjects in the depression group were not clinically depressed at the time of testing. The results may not generalize to patients with more severe depression. Seventh, extrapyramidal symptoms were not measured in the present study, and could reflect an important or potentially confounding variable of interest. Finally, correlational studies of this kind are not particularly well-equipped for matching variables in psychometric properties, so it is possible that differential "deficits" in neurocognitive versus psychiatric variables, or in coding versus other neurocognitive abilities, are in part, a reflection of differences in measure psychometrics. Given that it seems unlikely that the coding task is inherently more discriminating than the other tasks in the RBANS (particularly when computed as a sum score) and that aspects of pathology, such as depression, verbal comprehension deficits, delayed memory deficits and mania symptoms, were associated with *increased* prosody at a small to medium effect size level, it seems unlikely that the present results are an artifact of a generalized deficit. Nonetheless, more rigorous, preferably experimental designs should be used in future research.

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Contributors

Alex S. Cohen was the primary investigator for this project and designed the study and wrote the bulk of the manuscript. Yunjung Kim and Gina M. Najolia helped manage the literature searches and the analyses and provided conceptual material to the planning and presentation of this project. All authors contributed to and have approved the final manuscript.

Conflict of interest

There are no conflicts of interest to report.

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