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Psychological Inflexibility and Quality of Life in Breast Cancer

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Abstract

Background: Psychological inflexibility (PI) is a transdiagnostic risk factor for emotional distress and diminished quality of life (QoL), yet the indirect pathways linking PI to QoL through distress remain largely untested in breast cancer. Methods: This exploratory secondary analysis used a publicly available longitudinal dataset of 40 women with non-metastatic breast cancer assessed at baseline and 2-month follow-up. Bootstrapped mediation (5000 resamples, BCa CIs) tested whether depression, anxiety, and stress (DASS-21) statistically mediated the association between PI (AAQ-II) and QoL (WHOQOL-BREF) across 27 models. Hierarchical regressions, Kruskal–Wallis tests, and extended correlations supplemented the analysis. Results: At follow-up, all nine cross-sectional indirect effects were significant (all 95% BCa CIs excluding zero), with distress accounting for 33–51% of the total association between PI and QoL. The largest indirect effects were observed for depression on psychological QoL (indirect = −0.845, $p < 0.001$) and anxiety on physical QoL (indirect = −0.739, $p < 0.001$). No indirect effects were significant at baseline or for change scores (all $p > 0.05$). Concurrent PI independently predicted psychological QoL ($\Delta R^2 = 0.094$, $p = 0.002$), general QoL ($\Delta R^2 = 0.084$, $p = 0.015$), and anxiety ($\Delta R^2 = 0.122$, $p = 0.004$) in hierarchical regressions. Anxiety severity was associated with impairment across all five QoL domains (all p_adj < 0.05). Conclusions: These preliminary findings suggest that emotional distress may partially account for the association between PI and QoL in breast cancer, consistent with predictions from the ACT model. However, the small sample size and cross-sectional nature of the indirect effects observed at follow-up preclude causal inference. Replication in adequately powered prospective designs is essential.

Keywords: psychological inflexibility; experiential avoidance; quality of life; breast cancer; mediation analysis; ACT

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1. Introduction

Breast cancer is the most frequently diagnosed malignancy among women worldwide, accounting for approximately 2.3 million new cases and 670,000 deaths in 2022 [1]. Beyond the direct physical consequences of the disease and its treatment, breast cancer imposes a substantial psychological burden characterised by depression, anxiety, stress, sleep disturbances, altered body image, and diminished quality of life (QoL) [2]. Systematic reviews have demonstrated that breast cancer survivors face significantly elevated risks of depression, anxiety, and suicidal ideation compared to women without a cancer history [3], and meta-analytic evidence indicates that these comorbidities adversely affect both cancer

prognosis and all-cause mortality [4]. Indeed, a meta-analysis of 94 interview-based studies estimated that mood disorders affect 30–40% of patients in oncological settings, with prevalence rising further when self-report measures are used [5]. These findings underscore the need for transdiagnostic theoretical frameworks that elucidate the psychological mechanisms underlying emotional distress and QoL impairment in this population.

Acceptance and Commitment Therapy (ACT) provides such a framework by identifying psychological inflexibility (PI) as a core transdiagnostic process driving psychopathology [6]. PI is operationally defined as the rigid dominance of internal reactions over chosen values and goals, encompassing experiential avoidance—the tendency to suppress or escape unwanted thoughts, emotions, and bodily sensations—and is reliably measured by the Acceptance and Action Questionnaire-II (AAQ-II) [7]. According to the ACT model, PI maintains psychological distress not through the content of negative cognitions but through patterns of experiential avoidance that narrow behavioural repertoires and disconnect individuals from valued life domains [6,8].

Converging evidence supports the clinical relevance of this framework in oncology. A meta-analysis of randomised controlled trials demonstrated moderate-to-large effects of ACT on reducing depression, anxiety, and stress in breast cancer [9], and clinical trials have shown benefits for fear of recurrence [10] and pain acceptance [11]. Reductions in experiential avoidance have been identified as a key process through which psychological interventions are associated with improvements in anxiety, depression, and well-being [12,13], and PI has emerged as a consistent correlate of emotional distress across diverse clinical populations [14,15]. Despite these advances, whether emotional distress statistically accounts for the association between PI and QoL in breast cancer remains largely untested.

Trindade and colleagues provided preliminary longitudinal evidence for the role of experiential avoidance in breast cancer outcomes [16]. However, their analysis was restricted to three outcome variables, did not test mediation pathways, did not model anxiety as a dependent variable, and omitted multiple QoL domains from regression analyses. The present study addresses these gaps through a theory-driven secondary analysis of the dataset, employing mediation across all DASS-21 distress subscales and WHOQOL-BREF QoL domains. The aim is to examine whether emotional distress statistically accounts for the association between PI and health-related QoL in breast cancer patients and to characterise PI's predictive associations across psychological and functional outcomes.

2. Materials and Methods

2.1. Data Source and Ethical Considerations

This study is a secondary analysis of a publicly available longitudinal dataset originally collected and reported by Trindade et al. [16]. The dataset is openly accessible via Mendeley Data [17]. As the dataset is publicly available and fully anonymised, no additional ethical approval was required for this secondary analysis.

2.2. Study Design and Participants

A prospective longitudinal design with two assessment points was employed: baseline (M0) and 2-month follow-up (M2). The interval between assessments was approximately two months. The sample comprised 40 women diagnosed with non-metastatic breast cancer (stages I–III), aged 36 to 85 years (M = 60.00, SD = 10.13), recruited from the Radiotherapy Service of the University Coimbra Hospital (Centro Hospitalar Universitário de Coimbra), Coimbra, Portugal. The average time since diagnosis was 6.70 months (SD = 4.26). No participant was receiving psychosocial treatment during the study period. No demographic or clinical covariates beyond age and cancer stage were available in the public dataset. The original data collection was conducted by researchers affiliated with CINEICC (Center

for Research in Neuropsychology and Cognitive and Behavioral Intervention), Faculty of Psychology and Educational Sciences, University of Coimbra.

2.3. Measures

The Acceptance and Action Questionnaire–II (AAQ-II) is a 7-item self-report measure of psychological inflexibility and experiential avoidance [7]. Items are rated on a 7-point Likert scale (1 = never true; 7 = always true), with higher total scores indicating greater psychological inflexibility. Total scores in the range of 24 to 28 and above have been used to denote clinically elevated psychological inflexibility [7]. The AAQ-II has demonstrated a single-factor structure, good internal consistency (mean $\alpha = 0.84$ across six validation samples), and adequate 12-month test–retest reliability ($\alpha = 0.79$) [7].

The Depression Anxiety Stress Scales–21 (DASS-21) [18] is a 21-item self-report instrument assessing three dimensions of negative emotional states: depression (7 items), anxiety (7 items), and stress (7 items). Items are rated on a 4-point severity/frequency scale (0 = did not apply to me at all; 3 = applied to me very much or most of the time). Subscale scores are computed by summing item responses and multiplying by two to correspond with the full DASS-42 norms. Higher scores indicate greater symptom severity. Clinical severity classifications (normal, mild, moderate, severe, extremely severe) follow the previously validated cut-offs [18]. The corresponding DASS-42 cut-offs for depression are normal 0–9, mild 10–13, moderate 14–20, severe 21–27, and extremely severe $\geq$ 28; those for anxiety are normal 0–7, mild 8–9, moderate 10–14, severe 15–19, and extremely severe $\geq$ 20; and those for stress are normal 0–14, mild 15–18, moderate 19–25, severe 26–33, and extremely severe $\geq$ 34 [18].

The World Health Organization Quality of Life–BREF (WHOQOL-BREF) [19] is a 26-item generic quality of life instrument assessing four domains, physical health (7 items), psychological (6 items), social relationships (3 items), and environment (8 items), plus two items assessing overall quality of life and general health satisfaction. Domain scores are transformed to a 0–100 scale, with higher scores indicating better quality of life; the WHOQOL-BREF has no established clinical cut-off. Internal consistency in the present sample ranged from $\alpha = 0.64$ to 0.90 across domains and timepoints [16].

2.4. Mediation Analysis Strategy

Guided by the ACT model of psychological inflexibility [6] and meta-analytic evidence linking experiential avoidance to depression ($r = 0.56$) and anxiety ($r = 0.51$) [20], we tested whether DASS-21 subscales mediated the relationship between AAQ-II and WHOQOL-BREF domains. Three mediators (depression, anxiety, stress) were crossed with three theoretically grounded QoL outcomes: physical (continuity with the original study [16]), psychological (strongest ACT-theorised link) [21], and general (global QoL indicator). Social and environmental domains were excluded from mediation analyses due to weaker theoretical grounding in the inflexibility–distress pathway [21]. Although social and environmental QoL are clinically important outcomes, this exclusion reflects the fact that the hypothesized ACT pathway primarily focuses on intrapersonal processes linking psychological inflexibility, emotional distress, and individual functioning. This yielded 27 planned models (3 mediators $\times$ 3 outcomes $\times$ 3 timepoints: baseline, follow-up, and change scores); all combinations were retained to enable evaluation of the full pattern across timepoints rather than selectively reporting significant results. Mediation was tested using the mediation package in R [22] with 5000 bootstrap resamples and bias-corrected accelerated (BCa) confidence intervals. Significance was determined by whether the 95% BCa CI excluded zero. As the mediator and outcome were measured

concurrently at each timepoint, these models estimate cross-sectional indirect effects and do not support causal inference [23].

2.5. Statistical Analysis

Five outcomes not examined by Trindade et al. [16] were tested in hierarchical multiple regression: WHOQOL-BREF psychological, social, environmental, and general domains and DASS-21 anxiety. For each outcome, a three-step model was fitted: Step 1 entered the baseline score of the dependent variable as an autoregressive control; Step 2 added concurrent psychological inflexibility (AAQ-II at M2); and Step 3 added baseline psychological inflexibility (AAQ-II at M0) to evaluate the incremental predictive value of earlier inflexibility levels. DASS-21 subscale scores were classified into clinical severity bands (normal, mild, moderate, severe, extremely severe) following the manual cut-offs [18]. Between-group differences in WHOQOL-BREF domain scores across severity categories were tested using Kruskal–Wallis tests, with Benjamini–Hochberg correction for multiple comparisons applied across the 15 tests (3 DASS subscales $\times$ 5 QoL domains). Pearson correlation matrices were computed at baseline, follow-up, and for change scores (Δ = M2 $-$ M0), including variables omitted from Trindade et al.'s [16] original correlation table. Pearson coefficients were chosen for comparability with the original study; given the non-normal distributions of several variables, these should be interpreted with caution. Baseline-to-follow-up changes were evaluated using paired *t*-tests for variables with normally distributed difference scores and Wilcoxon signed-rank tests where Shapiro–Wilk tests indicated significant departures from normality. Effect sizes were computed as Cohen's *d* for paired samples. All analyses were conducted in R (version 4.3). Statistical significance was set at $\alpha = 0.05$ throughout, with corrections for multiple comparisons applied where indicated.

3. Results

3.1. Descriptive Statistics

Table A1 presents descriptive statistics for all study variables at baseline and 2-month follow-up. On average, participants reported relatively low psychological inflexibility at baseline (AAQ-II: $M = 17.35$, $SD = 9.32$). Although the mean fell in the lower portion of the AAQ-II range, scores spanned almost the full scale (range 7–40), and a clinically relevant subgroup scored within the range commonly used to denote elevated psychological inflexibility (27.5% scored ≥ 24 and 20.0% ≥ 28). At follow-up the mean remained low ($M = 15.95$, $SD = 7.71$), with 15.0% scoring ≥ 24. Emotional distress was low-to-mild across DASS-21 subscales (depression: $M = 4.08$, $SD = 5.32$; anxiety: $M = 4.03$, $SD = 4.75$; stress: $M = 5.74$, $SD = 5.14$). Quality of life scores were moderate across all WHOQOL-BREF domains, with psychological QoL being highest ($M = 69.48$, $SD = 19.11$) and general QoL being lowest ($M = 61.88$, $SD = 15.75$). Shapiro–Wilk tests indicated significant departures from normality for the AAQ-II and all three DASS-21 subscales at both timepoints (all $p < 0.01$), necessitating non-parametric alternatives where appropriate.

3.2. DASS-21 Clinical Severity Distribution

At baseline, the majority of participants fell within the normal range for depression (70.0%), anxiety (56.4%), and stress (69.2%). Nevertheless, a clinically meaningful minority presented with elevated symptomatology: 12.5% reported moderate depression and 7.5% endorsed extremely severe depressive symptoms, while 12.8% reported extremely severe anxiety. By follow-up, the proportion classified as normal increased notably for stress (from 69.2% to 85.0%), whereas distributions for depression and anxiety remained relatively stable (Table A2).

3.3. Temporal Stability: Paired Comparisons

No statistically significant changes were observed between M0 and M2 on any variable (Table A3). Effect sizes were uniformly small ($|d| \leq 0.28$), indicating broad stability across the assessment interval. Physical QoL showed the largest positive trend ($\Delta = 3.57$, $d = 0.26$, $p = 0.103$).

3.4. Internal Consistency

Individual item-level data were not available in the public dataset; therefore, internal consistency coefficients were not recomputed. Trindade et al. [16] reported acceptable to good reliability across all instruments in this sample (AAQ-II $\alpha = 0.93$–0.94; DASS-21 depression $\alpha = 0.91$–0.95; anxiety $\alpha = 0.73$–0.81; stress $\alpha = 0.83$–0.92; WHOQOL-BREF domains $\alpha = 0.64$–0.90).

3.5. Mediation Analysis

The hypothesised mediation model is depicted in Figure 1. Twenty-seven mediation models were tested, crossing three DASS-21 mediators with three WHOQOL-BREF outcomes at three timepoints. Results are summarised in Table A4 and Figure 2.

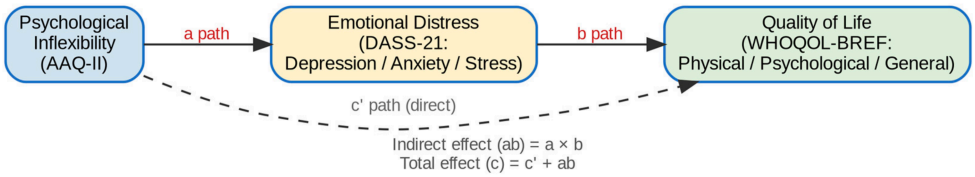


Figure 1. Conceptual mediation model. Psychological inflexibility (AAQ-II) is hypothesised to affect quality of life (WHOQOL-BREF) both directly (c′ path) and indirectly through emotional distress (DASS-21: depression, anxiety, stress). The indirect effect (ab) represents the product of the a and b paths.

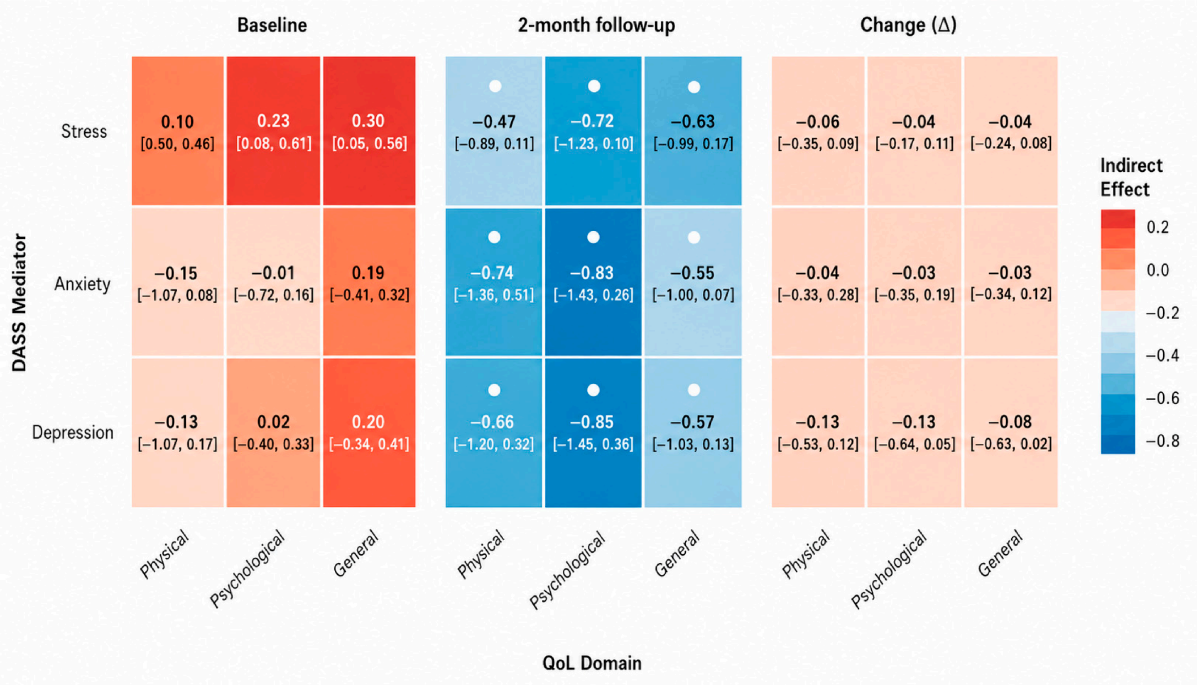


Figure 2. Heatmap of indirect effects across all 27 mediation models (3 DASS-21 mediators × 3 WHOQOL-BREF outcomes × 3 timepoints). Cell values represent point estimates of the indirect effect with 95% BCa confidence intervals in brackets. White dots denote statistically significant indirect effects ($p < 0.05$). Colour intensity reflects effect magnitude: blue = positive (suppression), red = negative (mediation in the

expected direction). Values represent indirect effects with 95% bias-corrected and accelerated confidence intervals (95% BCa CIs). White dots indicate statistically significant indirect effects ($p < 0.05$). Positive values indicate positive indirect effects, whereas negative values indicate negative indirect effects. Baseline, 2-month follow-up, and change (△) represent the indirect effects at baseline, at the 2-month follow-up, and the change in indirect effects from baseline to follow-up, respectively.

At baseline, none of the nine indirect effects reached statistical significance. The strongest trend was observed for the stress-mediated AAQ-II–general QoL indirect effect (indirect = 0.301, 95% BCa CI [−0.080, 0.546], $p = 0.092$), suggesting a potential but underpowered mediation effect. In contrast to baseline, all nine indirect effects at follow-up were statistically significant (Figure 2), suggesting a strengthening of the cross-sectional associations at this timepoint. Depression yielded a significant indirect effect on physical QoL (indirect = −0.661, 95% BCa CI [−1.195, −0.317], $p = 0.002$; 45.7% mediated), psychological QoL (indirect = −0.845, 95% BCa CI [−1.448, −0.365], $p < 0.001$; 47.7% mediated), and general QoL (indirect = −0.573, 95% BCa CI [−1.030, −0.131], $p = 0.009$; 44.5% mediated). Anxiety yielded the largest proportion mediated for physical QoL (51.1%), whilst stress showed the strongest indirect effect on the AAQ-II–general QoL association (48.8%). Proportions mediated at M2 ranged from 32.8% to 51.1%, indicating that emotional distress accounted for a substantial share of the total association between psychological inflexibility and quality of life. Figure 3 illustrates the depression indirect effect path at M2 as an exemplar.

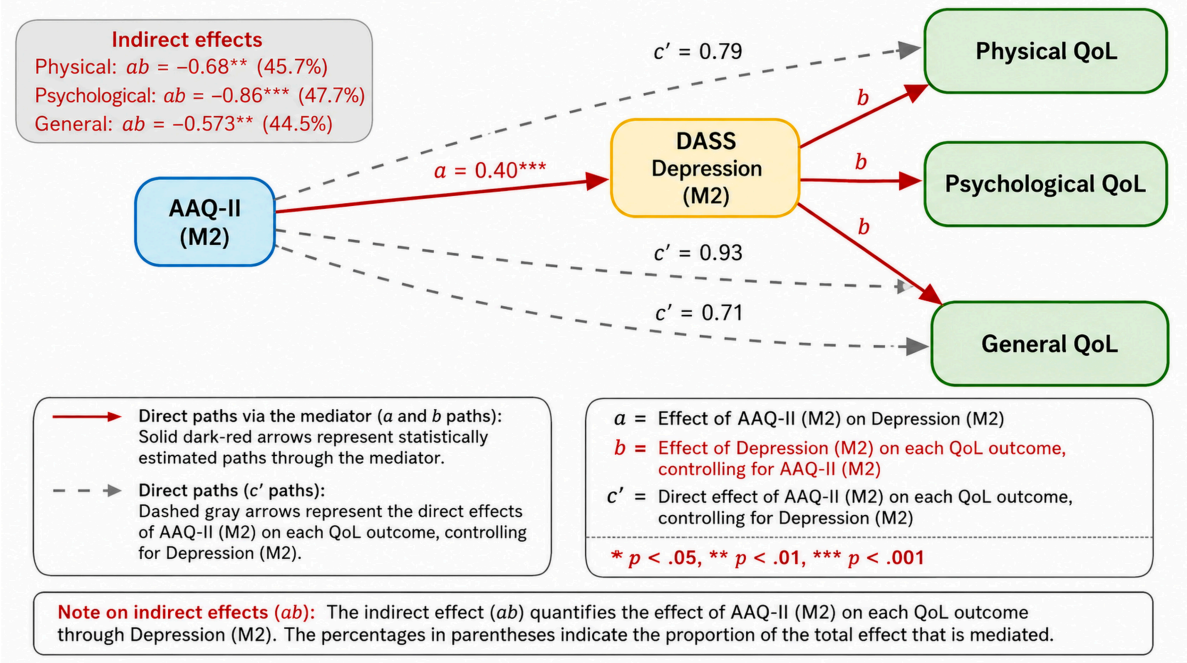


Figure 3. Path diagram showing indirect effects of psychological inflexibility (AAQ-II) on quality of life through depression (DASS-21) at 2-month follow-up (M2). Solid arrows indicate significant paths; dashed arrows indicate non-significant direct effects (c′ paths). Indirect effects (ab), proportions mediated, and significance levels are presented for physical, psychological, and general QoL outcomes.

No change-score mediation models yielded significant indirect effects (all $p > 0.05$), likely reflecting insufficient statistical power for detecting mediated effects in change scores with the present sample size [24]. Figure 4 illustrates the contrast across the three timepoint specifications.

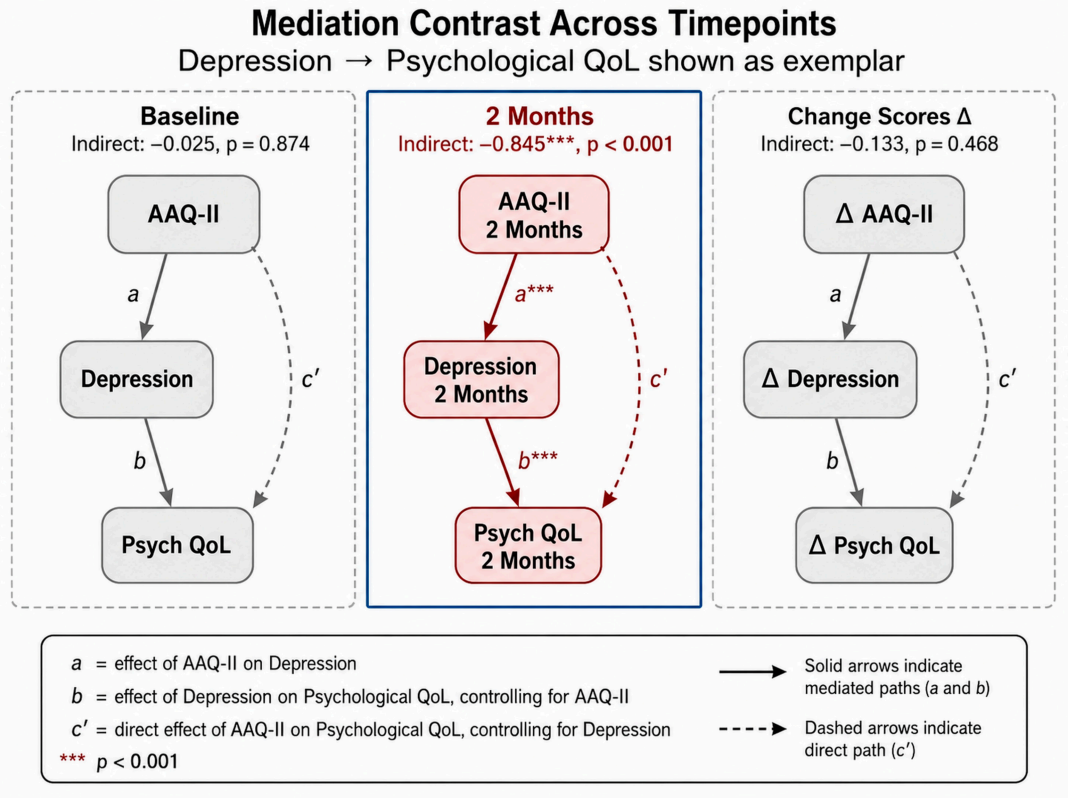


Figure 4. Mediation contrast across timepoints for an exemplar pathway (AAQ-II → Depression → Psychological QoL). Significant indirect effects emerged exclusively at follow-up (M2), transitioning from non-significant at baseline (indirect = −0.025, $p = 0.874$) to significant at follow-up (indirect = −0.845, $p < 0.001$). Change-score models remained non-significant (indirect = −0.133, $p = 0.468$).

3.6. Hierarchical Regression

Hierarchical regression analyses were conducted for five outcomes not examined in the original study (Table A5). Across all models, the baseline score of the dependent variable (Step 1) accounted for substantial variance ($R^2 = 0.416$–0.698). The addition of concurrent psychological inflexibility (AAQ-II at M2) at Step 2 significantly improved model fit for psychological QoL ($\Delta R^2 = 0.094$; $B = -0.97$, $p = 0.002$), general QoL ($\Delta R^2 = 0.084$; $B = -0.75$, $p = 0.015$), and anxiety ($\Delta R^2 = 0.122$; $B = 0.26$, $p = 0.004$). Environmental QoL showed a trend-level association ($B = -0.51$, $p = 0.053$) that reached significance at Step 3 ($B = -0.64$, $p = 0.034$). In contrast, concurrent inflexibility did not predict social QoL ($B = -0.23$, $p = 0.472$).

The inclusion of baseline inflexibility (AAQ-II at M0) at Step 3 contributed unique predictive variance only for anxiety ($B = 0.20$, $p = 0.037$), indicating that earlier psychological inflexibility was independently associated with subsequent anxiety symptoms, over and above concurrent inflexibility and baseline anxiety levels. For all QoL outcomes, baseline inflexibility did not add incrementally to prediction after accounting for concurrent inflexibility and the autoregressive effect (all $p > 0.35$).

3.7. DASS-21 Clinical Severity and Quality of Life

Kruskal–Wallis tests examined whether baseline QoL differed across DASS-21 clinical severity categories (Table A6; Figure 5). Anxiety severity was associated with statistically significant differences in all five QoL domains after Benjamini–Hochberg correction (all $p$adj < 0.05), with social QoL showing the largest effect ($H(4) = 20.41$, $p$adj = 0.006). Depression severity differentiated social QoL ($H(4) = 16.67$, $p$adj = 0.016) and physical QoL

($H(4) = 11.84$, $p$adj = 0.040), but not psychological, environmental, or general QoL after correction. Stress severity was associated only with social QoL ($H(4) = 14.66$, $p$adj = 0.016). These results identify social QoL as the domain most consistently differentiated by emotional distress severity across all three DASS-21 subscales.

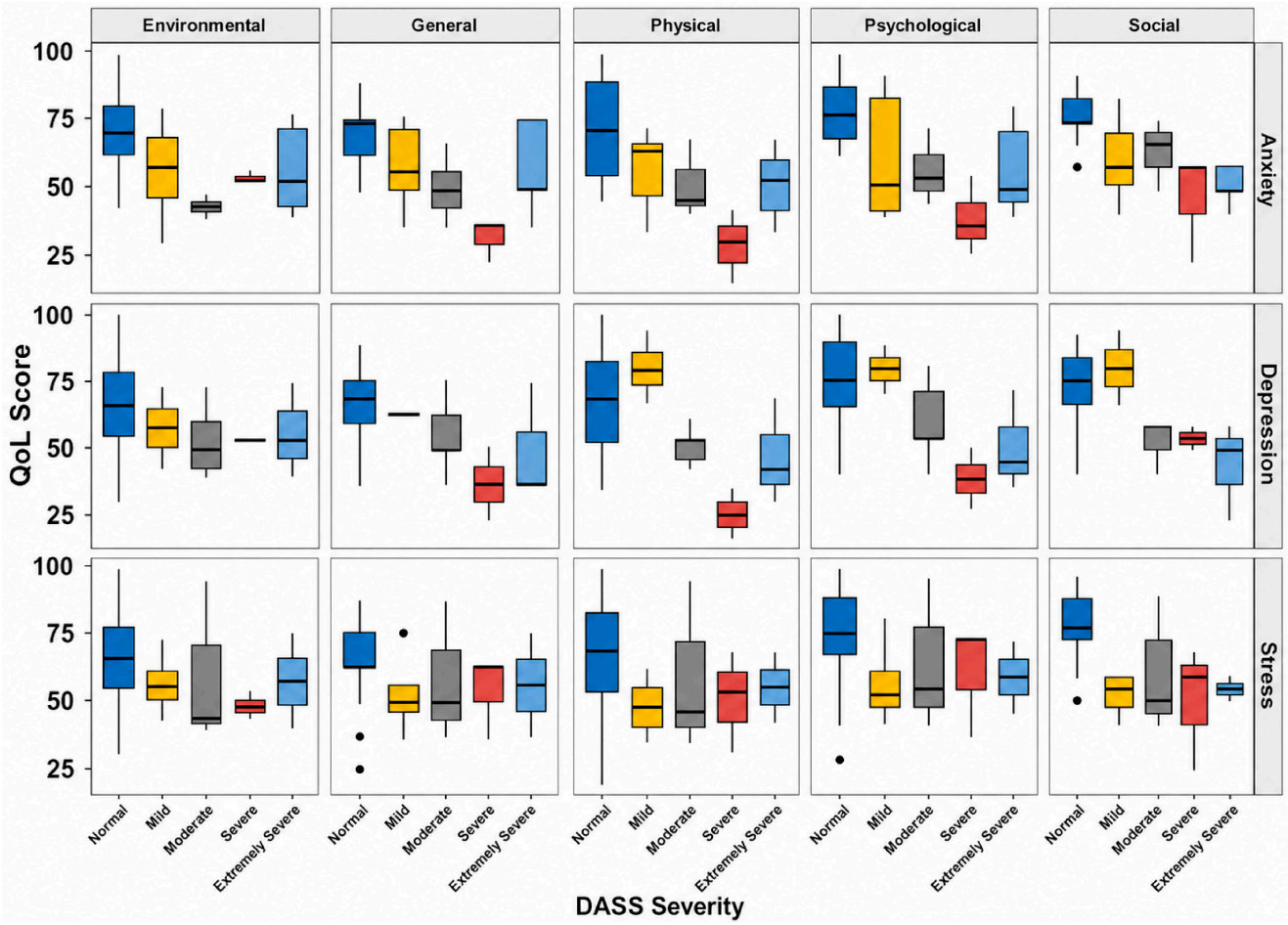


Figure 5. Quality of life scores (WHOQOL-BREF domains) by DASS-21 clinical severity classification at baseline. Each panel represents a QoL domain (columns) by DASS-21 subscale (rows) combination. Boxes represent interquartile ranges with median lines; whiskers extend to 1.5 × IQR.

Pearson correlations (Supplementary Tables S1–S3 revealed that AAQ-II scores were strongly associated with all QoL domains at baseline ($r = -0.645$ to -0.805, all $p < 0.001$) and with DASS-21 subscales ($r = 0.653$–0.714). At follow-up, associations between DASS-21 subscales and QoL domains strengthened considerably (e.g., depression–psychological QoL: $r = -0.563$ at baseline vs. $r = -0.816$ at 2-month follow-up), consistent with the emergence of significant mediation at 2-month follow-up. Change-score correlations were substantially attenuated; AAQ-II changes correlated significantly only with changes in psychological QoL ($r = -0.330$, $p = 0.038$) and general QoL ($r = -0.327$, $p = 0.039$). After controlling for total DASS-21 distress, the AAQ-II retained significant partial associations with all three QoL domains (partial r = −0.33 to −0.78, all $p < 0.05$) and accounted for incremental variance beyond distress in hierarchical regression ($\Delta R^2 = 0.05$ to 0.47), indicating that psychological inflexibility captured variance distinct from concurrent emotional distress.

4. Discussion

We conducted an exploratory secondary analysis of a publicly available longitudinal dataset [16,17] to examine whether emotional distress statistically accounts for the asso-

ciation between PI and QoL in women with non-metastatic breast cancer. Extending the original three-outcome analysis by Trindade et al. [16], we tested bootstrapped mediation across all DASS-21 distress dimensions and WHOQOL-BREF QoL domains, supplemented by hierarchical regression and clinical severity classification. To our knowledge, this is among the first studies to formally test indirect effects linking PI to multiple QoL domains through emotional distress in breast cancer, providing preliminary findings consistent with the ACT model [6]. All nine cross-sectional indirect effects at the two-month follow-up were significant, with emotional distress accounting for 33% to 51% of the total association between PI and QoL. This pattern is consistent with the ACT theoretical proposition that PI is associated with diminished functioning partly through elevated emotional distress [6,8], and the proportions mediated are broadly comparable to those reported in non-cancer populations [25,26]. In advanced cancer, Panjwani et al. [27] found that experiential avoidance statistically mediated the association between intolerance of uncertainty and anxiety, while Kashdan et al. [28] reported similar indirect effects in trauma survivors. Our findings extend this work by showing that indirect effects through distress are observed consistently across depression, anxiety, and stress as mediators and across physical, psychological, and general QoL as outcomes in the breast cancer context.

No indirect effects were significant at baseline despite strong bivariate associations. The most parsimonious explanation is that indirect effects are estimated imprecisely with small sample sizes. Moreover, the stronger cross-sectional associations at follow-up yielded larger and more stable product-of-coefficients estimates. Importantly, the follow-up models used concurrent data, meaning that all variables were measured at the same timepoint. These findings therefore represent cross-sectional indirect effects and should be interpreted as statistical associations rather than evidence of causal mediation [23]. Stein et al. [29] similarly cautioned that concurrent indirect effects may not replicate in cross-lag designs and recommended prospective tests with larger samples and additional assessment points.

Hierarchical regression results revealed that concurrent PI significantly predicted follow-up psychological QoL, general QoL, and anxiety after controlling for autoregressive effects, aligning with the longitudinal study by Al-Abbadey et al. [21] in which psychological flexibility predicted anxiety, depression, stress, and QoL in cancer survivors over three months. The observation that baseline PI contributed unique predictive variance only for anxiety is notable given that anxiety has been identified as a particularly persistent distress dimension in breast cancer [30,31], though this isolated finding requires replication. In contrast, PI did not predict social QoL, suggesting that social relationships in this population may be more closely associated with external interpersonal factors such as social support availability and relationship quality than with internal psychological processes [32,33]. This is consistent with our severity classification analysis, in which social QoL was the domain most consistently differentiated by emotional distress severity across all three DASS-21 subscales, pointing to social QoL as a sensitive indicator of distress burden potentially driven by relational rather than intrapersonal mechanisms. This finding highlights the importance of social functioning in breast cancer populations and suggests that future studies should investigate whether interpersonal and contextual factors interact with psychological inflexibility to influence social quality of life.

From a clinical perspective, anxiety severity was associated with impairment across all five QoL domains, whereas depression and stress each differentiated only selected domains. This is consistent with evidence that anxiety co-occurs with prognostic uncertainty and fear of recurrence in breast cancer [10,34] and may be more strongly associated with global QoL deterioration than depression in long-term survivors [35]. Current NCCN guidelines recommend routine distress screening in oncology settings [36]; if replicated in adequately powered studies, our findings would suggest that incorporating a brief measure of PI

alongside conventional distress measures may warrant investigation. Given the transdiagnostic nature of PI [20,21], ACT-based interventions that simultaneously address multiple distress dimensions [9,37] have shown feasibility in breast cancer populations [10,11,38] and represent a promising avenue. Future adequately powered prospective studies with three or more assessment points and structural equation modelling [39,40] are needed to establish the temporal sequence of these associations and provide more robust estimates of indirect effects.

This study has several limitations.

The sample comprised only 40 participants, which substantially limits statistical power, particularly for mediation analyses and change-score models. Although bootstrapping can improve estimation of indirect effects in small samples, mediation analyses with $N = 40$ are adequately powered primarily to detect relatively large indirect effects, whereas small-to-moderate effects may remain undetected. Consequently, the non-significant findings observed in the baseline and change-score models may reflect limited power rather than the absence of meaningful associations. In addition, indirect effects estimated from small samples may be unstable and sensitive to sampling variability. A total of 27 mediation models and 5 regression models were examined. Because the study was exploratory and hypothesis-generating in nature, corrections for multiple testing were not applied across these analyses. Therefore, some statistically significant findings may represent false-positive results, and the observed pattern of associations should be interpreted cautiously pending replication in larger, adequately powered prospective studies.

The follow-up mediation models used concurrent data, precluding causal inference. Furthermore, recent psychometric analyses have questioned whether the AAQ-II is fully separable from general emotional distress and negative affect [41,42]; because it served as the sole index of psychological inflexibility, it may share method and content variance with the DASS-21. Reassuringly, the AAQ-II retained associations with QoL after total distress was partialled out (partial r = -0.33 to -0.78) and contributed incremental predictive variance ($\Delta R^2 = 0.05$ to 0.47), indicating the two constructs were not redundant in this sample, although this does not fully resolve the construct-overlap concern. Item-level data were unavailable, preventing recomputation of internal consistency, and no demographic or clinical covariates beyond age and cancer stage were available. As a secondary analysis, this study was constrained by the original study design and available variables [43,44].

Future research should replicate these findings in larger, prospectively designed samples with multiple assessment points and use structural equation modeling to test longitudinal mediation. Investigating ACT-based interventions targeting psychological inflexibility and multiple distress dimensions, as well as incorporating PI alongside routine distress screening, may improve identification and support of breast cancer patients at risk for reduced QoL.

5. Conclusions

In conclusion, these exploratory findings suggest that emotional distress may partially account for the cross-sectional association between PI and multiple QoL domains in women with non-metastatic breast cancer, consistent with predictions from the ACT model. Given the small sample and concurrent measurement, these results should be regarded as hypothesis-generating. Replication in adequately powered prospective samples is essential to establish temporal precedence and inform the design of targeted interventions [45].

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/jmms13030014/s1, Table S1: Pearson Correlation Matrix–Baseline (M0); Table S2: Pearson Correlation Matrix–Follow up (M2); Table S3: Pearson Correlation Matrix–Change Scores (Δ).

Author Contributions: Conceptualization, I.M. and Y.S.; methodology, I.M.; formal analysis, I.M.; resources, I.M.; data curation, I.M.; writing—original draft preparation, I.M.; writing—review and editing, I.M.; visualization, I.M.; supervision, Y.S. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: Ethical review and approval were waived for this study due to this study being a secondary analysis of a publicly available fully anonymized dataset.

Informed Consent Statement: Patient consent was waived due to this study being a secondary analysis of publicly available data.

Data Availability Statement: The dataset analysed in this study is publicly available via Mendeley Data (DOI: 10.17632/8yz89ct4br.1) [17].

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

ACT	Acceptance and Commitment Therapy
BC	Breast Cancer
PI	Psychological Inflexibility
QoL	Quality of Life

Appendix A

Table A1. Descriptive Statistics for All Measures at Baseline and 2-Month Follow-Up.

Measure	Time	*n*	M	SD	Mdn	Skew	Kurt	SW *p*
AAQ-II	Baseline	40	17.35	9.32	17.00	0.61	−0.76	0.003
AAQ-II	2-month	40	15.95	7.71	14.00	0.60	−0.77	0.004
DASS Depression	Baseline	40	4.08	5.32	2.00	1.30	0.69	<0.001
DASS Depression	2-month	40	3.35	5.13	1.00	1.89	2.99	<0.001
DASS Anxiety	Baseline	39	4.03	4.75	3.00	1.34	1.47	<0.001
DASS Anxiety	2-month	40	3.75	4.93	1.50	1.64	2.36	<0.001
DASS Stress	Baseline	39	5.74	5.14	5.00	0.90	−0.26	0.001
DASS Stress	2-month	40	4.70	4.25	4.50	1.50	3.48	<0.001
QoL Physical	Baseline	40	62.77	20.04	64.29	0.06	−0.80	0.395
QoL Physical	2-month	40	66.34	17.86	69.64	−0.18	−0.91	0.331
QoL Psychological	Baseline	40	69.48	19.11	70.83	−0.23	−1.00	0.213
QoL Psychological	2-month	40	70.52	18.40	75.00	−0.76	−0.19	0.016
QoL Social	Baseline	40	68.54	15.84	75.00	−0.47	−0.27	0.042
QoL Social	2-month	40	67.50	17.38	66.67	−0.29	−0.73	0.147
QoL Environmental	Baseline	39	63.46	16.97	62.50	0.33	−0.65	0.359
QoL Environmental	2-month	40	63.28	17.97	65.62	0.11	−0.63	0.450
QoL General	Baseline	40	61.88	15.75	62.50	−0.36	−0.78	0.008
QoL General	2-month	40	63.75	15.45	62.50	−0.18	−0.28	0.008

Note. M = mean; SD = standard deviation; Mdn = median; Skew = skewness; Kurt = excess kurtosis; SW *p* = Shapiro–Wilk test *p*-value; AAQ-II = Acceptance and Action Questionnaire-II; DASS = Depression Anxiety Stress Scales-21; QoL = quality of life (WHOQOL-BREF domains). All decimals are reported with a leading zero.

Table A2. DASS-21 Severity Classification: n (%) at Baseline and 2-Month Follow-Up.

Subscale	Time	Normal	Mild	Moderate	Severe	Ext. Severe
Depression	Baseline	28 (70.0%)	2 (5.0%)	5 (12.5%)	2 (5.0%)	3 (7.5%)
Depression	2-month	29 (72.5%)	3 (7.5%)	4 (10.0%)	1 (2.5%)	3 (7.5%)
Anxiety	Baseline	22 (56.4%)	6 (15.4%)	3 (7.7%)	3 (7.7%)	5 (12.8%)
Anxiety	2-month	26 (65.0%)	4 (10.0%)	3 (7.5%)	2 (5.0%)	5 (12.5%)
Stress	Baseline	27 (69.2%)	4 (10.3%)	3 (7.7%)	3 (7.7%)	2 (5.1%)
Stress	2-month	34 (85.0%)	2 (5.0%)	2 (5.0%)	1 (2.5%)	1 (2.5%)

Note. Severity bands based on DASS-21 manual cut-offs [18]. Ext. Severe = Extremely Severe.

Table A3. Paired Comparisons: Baseline vs. 2-Month Follow-Up (N = 40).

Measure	M (Baseline)	M (2-Month)	Δ	Test	*p*	d	Sig
AAQ-II	17.35	15.95	−1.40	Wilcoxon	0.493	−0.20	ns
DASS Depression	4.08	3.35	−0.72	Wilcoxon	0.423	−0.16	ns
DASS Anxiety	4.03	3.72	−0.31	Wilcoxon	0.957	−0.07	ns
DASS Stress	5.74	4.59	−1.15	Wilcoxon	0.126	−0.28	ns
QoL Physical	62.77	66.34	3.57	Paired t	0.103	0.26	ns
QoL Psychological	69.48	70.52	1.04	Wilcoxon	0.843	0.08	ns
QoL Social	68.54	67.50	−1.04	Wilcoxon	0.500	−0.09	ns
QoL Environmental	63.46	63.70	0.24	Paired t	0.882	0.02	ns
QoL General	61.88	63.75	1.88	Wilcoxon	0.409	0.15	ns

Note. Wilcoxon signed-rank test used when Shapiro–Wilk test on difference scores was significant ($p < 0.05$); otherwise, paired *t*-test. d = Cohen's d for paired samples. M = mean.

Table A4. Theory-Driven Mediation Results: AAQ-II → DASS-21 → WHOQOL-BREF (Bootstrap 5000, BCa CIs).

Timepoint	Mediator	Outcome	Indirect [95% CI]	*p*	Direct	Total	% Med
Baseline (M0)	Depression	Physical	−0.133 [−1.07, 0.17]	0.513	−1.25	−1.39	9.6%
	Depression	Psychological	0.025 [−0.40, 0.33]	0.874	−1.67	−1.64	−1.5%
	Depression	General	0.200 [−0.34, 0.41]	0.297	−1.56	−1.36	−14.7%
	Anxiety	Physical	−0.151 [−1.07, 0.08]	0.370	−1.25	−1.40	10.8%
	Anxiety	Psychological	−0.010 [−0.72, 0.16]	0.903	−1.68	−1.69	0.6%
	Anxiety	General	0.186 [−0.41, 0.33]	0.316	−1.59	−1.40	−13.3%
	Stress	Physical	0.096 [−0.50, 0.46]	0.673	−1.49	−1.39	−6.9%
	Stress	Psychological	0.227 [−0.08, 0.61]	0.156	−1.87	−1.64	−13.8%
	Stress	General	0.301 [−0.08, 0.55]	0.092	−1.66	−1.36	−22.1%
Follow-up (M2)	Depression	Physical	−0.661 [−1.20, −0.32] *	0.002	−0.79	−1.45	45.7%
	Depression	Psychological	−0.845 [−1.45, −0.37] *	<0.001	−0.93	−1.77	47.7%
	Depression	General	−0.573 [−1.03, −0.13] *	0.009	−0.71	−1.29	44.5%
	Anxiety	Physical	−0.739 [−1.36, −0.50] *	<0.001	−0.71	−1.45	51.1%
	Anxiety	Psychological	−0.830 [−1.43, −0.26] *	0.007	−0.94	−1.77	46.9%
	Anxiety	General	−0.551 [−1.00, −0.07] *	0.023	−0.74	−1.29	42.8%
	Stress	Physical	−0.474 [−0.89, −0.11] *	0.022	−0.97	−1.45	32.8%
	Stress	Psychological	−0.723 [−1.20, −0.18] *	0.005	−1.05	−1.77	40.8%
	Stress	General	−0.628 [−0.99, −0.17] *	0.008	−0.66	−1.29	48.8%

Table A4. *Cont.*

Timepoint	Mediator	Outcome	Indirect [95% CI]	*p*	Direct	Total	% Med
Change (Δ)	Depression	Physical	−0.126 [−0.53, 0.12]	0.480	−0.08	−0.20	61.6%
	Depression	Psychological	−0.133 [−0.64, 0.06]	0.468	−0.46	−0.59	22.4%
	Depression	General	−0.084 [−0.63, 0.02]	0.405	−0.52	−0.60	13.9%
	Anxiety	Physical	−0.035 [−0.33, 0.28]	0.812	−0.15	−0.18	19.5%
	Anxiety	Psychological	−0.035 [−0.35, 0.19]	0.817	−0.58	−0.61	5.6%
	Anxiety	General	−0.028 [−0.34, 0.12]	0.768	−0.60	−0.63	4.4%
	Stress	Physical	−0.059 [−0.35, 0.09]	0.545	−0.16	−0.22	26.9%
	Stress	Psychological	−0.037 [−0.17, 0.12]	0.776	−0.57	−0.61	6.1%
	Stress	General	−0.037 [−0.24, 0.08]	0.696	−0.56	−0.60	6.2%

Note. * $p < 0.05$ (95% BCa CI excludes zero% Med = proportion of total effect mediated; values for non-significant indirect effects are unstable and should not be interpreted substantively [24]. Direct = direct effect (c′ path). Total = total effect (c path). Indirect effects tested via bootstrapped product of coefficients (5000 resamples, BCa confidence intervals).

Table A5. Summary of Hierarchical Regression Analyses for Untested Outcomes at 2-Month Follow-Up.

Outcome	R^2 S1	R^2 S2	ΔR^2	AAQ 2-Month (S2)	R^2 S3	AAQ 2-Month (S3)	AAQ Baseline (S3)
QoL Psychological	0.606	0.700	0.094	−0.97 **	0.703	−0.91 **	−0.18
QoL Social	0.603	0.609	0.006	−0.23	0.609	−0.21	−0.07
QoL Environmental	0.698	0.729	0.031	−0.51 †	0.735	−0.64 *	0.24
QoL General	0.438	0.522	0.084	−0.75 *	0.523	−0.71 *	−0.10
DASS Anxiety	0.416	0.538	0.122	0.26 **	0.593	0.15	0.20 *

Note. S1/S2/S3 = regression step. B values shown are unstandardised coefficients. ΔR^2 = change from Step 1 to Step 2. * $p < 0.05$, ** $p < 0.01$, † $p < 0.10$.

Table A6. Kruskal–Wallis Tests: QoL Domains by DASS-21 Severity Bands at Baseline.

DASS	QoL Domain	H	df	*p*	*p* (adj)	Sig
Anxiety	Physical	14.99	4	0.005	0.016	*
Anxiety	Psychological	15.49	4	0.004	0.016	*
Anxiety	Social	20.41	4	<0.001	0.006	**
Anxiety	Environmental	11.03	4	0.026	0.049	*
Anxiety	General	14.11	4	0.007	0.017	*
Depression	Physical	11.84	4	0.019	0.040	*
Depression	Psychological	9.70	4	0.046	0.076	ns
Depression	Social	16.67	4	0.002	0.016	*
Depression	Environmental	4.33	4	0.363	0.411	ns
Depression	General	7.67	4	0.104	0.156	ns
Stress	Physical	6.21	4	0.184	0.251	ns
Stress	Psychological	5.31	4	0.257	0.321	ns
Stress	Social	14.66	4	0.005	0.016	*
Stress	Environmental	4.17	4	0.384	0.411	ns
Stress	General	3.75	4	0.441	0.441	ns

Note. H = Kruskal–Wallis test statistic. *p* (adj) = Benjamini–Hochberg adjusted *p*-value. * $p_adj < 0.05$, ** $p_adj < 0.01$. ns = not statistically significant.

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