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WORKING MEMORY IN PATIENTS WITH MILD AND MODERATE DEPRESSIVE EPISODE

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ABSTRACT

Aim. To establish what is known about working memory specifically in adults with a mild or moderate depressive episode: whether the deficit exists in its own right or reduces to generalised slowing of processing and to a lowered willingness to expend effort; whether its magnitude depends on episode severity, number of past episodes and medication status; whether it persists in remission; and what follows from this for outpatient practice.

Methods. Literature review. PubMed, Scopus, eLibrary and CyberLeninka were searched with priority given to work published in 2021–2026, using combinations of the terms "working memory", "n-back", "digit span", "executive function", "major depressive disorder", "depressive episode", "severity", "remission", "rumination" and their Russian equivalents. Earlier work was included only where it introduces a concept or remains the sole source of a value not recomputed since. Meta-analyses, systematic reviews, controlled studies and clinical guidelines concerning working memory or executive function in adults with unipolar depression were eligible. Bibliographic data and content of every source were verified against its PubMed record or DOI; work whose numerical results could not be confirmed was either excluded or is cited without numerical values, with this stated explicitly. The review includes 47 sources, of which 35 were published in 2021–2026 and 27 are meta-analyses or systematic reviews. Effect sizes were neither recalculated nor pooled; disagreements between sources are stated plainly rather than smoothed over.

Results. The working memory deficit in depression is real and does not reduce to generalised slowing: in a meta-analysis of 31 n-back studies, accuracy did not differ from controls on the 0-back condition and fell significantly at 1-, 2- and 3-back, peaking at 2-back, whereas response latencies were prolonged at every load including zero. The overall cognitive deficit in MDD is SMD −0.39 (95% CI −0.47 to −0.31), but only −0.19 (−0.37 to −0.00) in unmedicated patients. In remission, between-group differences in executive function become nonsignificant once processing speed is controlled for, except in auditory and spatial working memory, where a residual effect of g = 0.19–0.31 persists. Mechanistic accounts have weakened over the past five years: the association between repetitive negative thinking and cognitive control is selective but small (all $|r| < 0.15$), and a path analysis of 223 studies places

rumination downstream of symptoms rather than as a mediator. The procognitive effect of antidepressants is confined to one molecule and small (g = −0.23 to −0.29), and aerobic exercise does not improve working memory (g = 0.08; −0.01 to 0.17).

Conclusions. In the mild and moderate episode, the working memory deficit is selective: manipulation of held material is impaired rather than storage capacity as such, and its magnitude is smaller than pooled meta-analytic estimates obtained on more severely ill samples. Three points matter for practice. The memory complaint and the memory test measure different things — their pooled correlation is 0.14, under 2% shared variance. The mechanism remains unestablished: neither rumination nor elevated effort cost survives scrutiny by recent syntheses in its earlier formulation. Full cognitive recovery in the wake of mood improvement should not be expected, yet no intervention with proven action on working memory specifically is currently available.

Keywords: Working memory; depressive episode; mild and moderate severity; n-back; rumination; executive function; residual cognitive impairment; processing speed.

Introduction

Depression remains the most prevalent mental disorder: GBD 2019 estimates 279.6 million cases of major depressive disorder worldwide (95% UI 251.6–310.3), with an age-standardised prevalence of 3440 per 100 000 population [1]. The same project updated to 2023 gives 1.17 billion cases (1.06–1.31) across all mental disorders and a rise in case numbers of 95.5% since 1990 against a rise in age-standardised prevalence of only 24.2% — that is, most of the increase is explained by population growth and ageing rather than by the illness itself becoming more frequent [2]. The claim that the 2020 pandemic sharply added to morbidity rests on a modelled estimate and is not borne out by direct measurement: in the national NEMESIS-3 sample, the prevalence of disorders before and during the pandemic did not differ (26.7% vs 25.7%), although over the decade from 2007–2009 to 2019–2022 it rose from 17.4% to 26.1% [3].

What matters clinically, however, is not the total figure but its composition. In the National Comorbidity Survey Replication all 12-month cases of MDD were independently reclassified by the QIDS-SR: 10.4% proved mild, 38.6% moderate, 38.0% severe and 12.9% very severe [4]. These data are more than twenty years old, yet no later estimate exists: not one population survey published in 2021–2026 reports the distribution of depressive episodes by severity, so this distribution remains the only available anchor. Roughly every second patient with depression has an episode of mild or moderate severity. Such patients are all but absent from the inpatient samples on which the bulk of the neuropsychological literature is built: they are treated as outpatients, more often by a general practitioner or a neurologist than by a psychiatrist, and they stay in work.

It is in these patients that the complaint of poor memory and concentration comes to the fore, and the complaint is not a secondary matter: diminished ability to think is part of the definition of the illness. DSM-5-TR formulates criterion A8 as “diminished ability to think or

concentrate, or indecisiveness, nearly every day, either by subjective account or as observed by others" [5]. ICD-11 includes impaired concentration among the symptoms of a depressive episode and separates the mild from the moderate degree functionally: in the mild degree the patient has "some difficulty" in continuing to function in at least one domain, in the moderate degree "significant difficulty" in several domains at once [6]. ICD-10 draws the boundary by symptom count: F32.0 requires two typical symptoms plus two additional ones, F32.1 two typical plus three, preferably four, additional ones [7]. All three classifications define the cognitive component through the complaint and through its functional consequences. None of them requires measurement: nowhere is a test provided for.

How persistent this complaint is was shown by Conradi, Ormel and de Jonge, who followed 267 primary care patients weekly for three years. Cognitive problems were present for 94% of the time spent in a depressive episode and 44% of the time spent in remission, full or partial; only lack of energy (90% and 35%) and sleep disturbance (85% and 39%) were equally persistent [8]. The complaint is thus one of the three most enduring residual symptoms. There is little ground for expecting treatment to remove it: in the meta-analysis by Ahern et al., which covered 69 studies and 4639 patients across 44 cognitive measures, change over the course of the illness was nonsignificant, negligible or small for more than 95% of the measures, and the contribution of a practice effect could not be ruled out [9]. To understand what lies behind the complaint, one must move from the complaint itself to the construct that describes it.

In the model of Alan Baddeley and Graham Hitch, working memory is not a store but a workbench: a system that holds a small amount of information for exactly as long as an operation upon that information requires [10]. The phonological loop holds acoustic material and refreshes it by subvocal rehearsal, the visuospatial sketchpad does the same for images and their locations, and the central executive allocates resource between them, switches attention and suppresses interference. Half a century later the same authors revised the architecture of the model: the episodic buffer, once introduced as a passive binding system, has been renamed the buffer of awareness and moved to the centre of the diagram, since it is there that storage, executive manipulation and attentional control converge [11]. The difference from short-term memory is not terminological: short-term memory is measured by how many items a person can hold, working memory by what the person can do with them while holding them.

The internal architecture of the executive is conventionally described after Miyake et al.: shifting, updating of contents and inhibition of a prepotent response correlate moderately with one another yet remain statistically separable, and each predicts performance on complex frontal lobe tasks in its own way [12]. This model is not beyond dispute. On re-examination neither the three-factor nor the nested model achieved acceptable fit to the data, and exploratory analysis yielded a different structure: an expanded working memory factor, a combined shifting-and-inhibition factor and a separate factor for the Stroop task [13]. It is telling that working memory is precisely what survived as a factor in its own right — the authors call it the most robustly operationalised construct among the executive functions. For depression it is updating that matters: the ability to clear from the buffer material that has lost

its relevance. It will be shown below that of the three functions it is chiefly updating that is impaired.

Working memory is measured by several families of task, and these are not interchangeable — in the literal, psychometric sense. Backward digit span and Corsi block-tapping yield capacity: how many elements a person can hold and reproduce in reverse order, auditory elements in the first case, visuospatial ones in the second. Operation span and reading span load the executive with a dual task: counting or reading while simultaneously holding a list. The n-back measures updating directly: the participant decides whether the current stimulus matches the one presented n steps earlier, and as n increases the load on the buffer rises stepwise. Owen et al. pooled 668 sets of activation coordinates from 24 neuroimaging studies and showed that the n-back reproducibly engages one and the same frontoparietal network: dorsolateral and ventrolateral prefrontal cortex, dorsal cingulate, and medial and lateral posterior parietal cortex [14]. The n-back and the span tasks cannot, however, be placed in a single row. Burgoyne et al., in 1272 participants, obtained a correlation of r = 0.25 between them, and at the level of latent factors only 20% of the variance proved to be shared [15]. Span tasks load storage, whereas the n-back demands rapid disengagement from outdated contents, and the authors warn explicitly against treating one as a substitute for the other. This distinction will be needed twice: first when effect sizes are examined, then when mechanism is examined.

The magnitude of the deficit is given by several large syntheses, and they agree better than is commonly supposed. Rock et al., working with CANTAB data, found significant moderate deficits of executive function, memory and attention in the symptomatic phase, with Cohen's d ranging from −0.34 to −0.65; for spatial working memory specifically, across 15 studies and 1088 participants, d was −0.54 (95% CI −0.75 to −0.33), and −0.46 (−0.84 to −0.09) in the subgroup of unmedicated patients [16]. Snyder pooled 113 studies and obtained a spread of effect sizes for executive function from 0.32 to 0.97, the working memory measures falling within that range in order of increasing manipulation load: forward digit span d = 0.39 (0.25–0.53), backward digit span d = 0.55 (0.39–0.71), n-back d = 0.63 (0.11–1.15) [17]. Rhee et al., across 33 studies and 2596 participants (1337 patients vs 1259 healthy controls), obtained an overall SMD of −0.39 (95% CI −0.47 to −0.31), with a deficit present in every domain except reaction time [18]. The same work contains a qualification of direct relevance to the present topic: in unmedicated patients, that is in those on average less severely ill, the effect was only −0.19 (−0.37 to −0.00), whereas in treatment-resistant depression it was −0.56 (−0.78 to −0.34).

Snyder's series of values lines up in order of increasing manipulation load, and this used to be read as direct proof that storage is less impaired than storage with manipulation. Since Burgoyne's work that reading has to be softened. Forward digit span and the n-back measure different constructs rather than two levels of a single scale, and the gap between d = 0.39 and d = 0.63 partly reflects that very difference and not the rise in load alone. The argument stands, but it must rest on a gradient within a single task rather than on a comparison of tasks with one another.

Such a gradient is supplied by the meta-analysis of Nikolin et al., confined to the n-back task alone: 31 studies, 1666 participants [19]. Accuracy in patients did not differ from controls at 0-back, where the task amounts to simple attention with no load on the buffer, and fell significantly at 1-, 2- and 3-back, the largest effect falling on 2-back. Response latency, meanwhile, was prolonged at all four levels, including 0-back. One conclusion follows: slowing in depression is generalised and extends even to tasks with no storage requirement, whereas the fall in accuracy is load-dependent and appears exactly where updating of contents comes into play. If the whole working memory deficit reduced to generalised slowing, accuracy at 0-back would suffer as much as at 2-back — it does not suffer at all. This is the most direct argument for a working memory deficit in depression in its own right. Effect sizes for the individual load levels are not reported in the abstract of that work and are deliberately omitted here: the argument rests on the shape of the relationship, not on particular numbers.

Whether the deficit depends on episode severity is a question to which the evidence base gives only a partial answer, and this incompleteness has a specific cause. Snyder ran a meta-regression on severity separately for each measure, and the result was selective: severity significantly predicted the deficit in verbal working memory overall ($\beta = 0.32$; $p = 0.049$) and in the manipulation composite ($\beta = 0.43$; $p = 0.031$), was borderline for backward digit span ($\beta = 0.43$; $p = 0.051$), and did not predict the deficit in forward digit span ($\beta = -0.01$; $p = 0.9$) [17]. For the n-back no meta-regression was run at all, for want of power. In other words, what depends on the severity of the episode is not the ability to hold material but the ability to manipulate what is held. Indirect support comes from the subgroup contrast in Rhee et al.: −0.19 in unmedicated patients against −0.56 in treatment-resistant depression [18].

More recent data call for a qualification, and it complicates the picture. Ross-Adelman et al. followed 475 participants of the RADAR-MDD cohort for a mean of 14 months, accumulating 2036 assessments, and separated two questions that are usually conflated. Between individuals, severity is associated with objective test performance, but this association is weaker than that between severity and self-reported cognitive difficulty. Within an individual, working memory does not track fluctuations in severity at all: only self-report, processing speed and attention were significant, and working memory was not among them [20]. Patients with more severe symptoms perform worse on the tests on average, yet in one and the same patient a worsening of the episode is not accompanied by a worsening of working memory. For the clinician who hopes to monitor the course of an episode with a working memory task, this is a direct warning.

At this point the principal weakness of the literature comes to light, and it should be named plainly. Not one of the meta-analyses listed above reports an effect separately for the mild and for the moderate episode. The syntheses are built on other axes: episode versus remission, first episode versus recurrent course, medicated versus unmedicated, treatment-resistant versus treatment-responsive. The ICD or DSM axis of “mild — moderate — severe” scarcely enters them, because the primary studies do not report it: instead of a category, a mean Hamilton or MADRS score is given, on the strength of which the group is retrospectively characterised as moderately to severely ill. Over the past five years the position has not changed — stratification by degree of severity has still not appeared, whereas stratification by treatment

resistance and by number of episodes has, and it yields larger values than the overall estimate. As a result, the physician treating a patient with a mild episode is obliged to extrapolate to that patient figures obtained predominantly on more severely ill and often hospitalised samples. If the dependence on severity is indeed continuous, such extrapolation systematically overstates the expected deficit.

The severity problem can be partly circumvented by another axis, that of the first episode. Lee et al. pooled 15 independent samples, 644 patients with a first episode of MDD of mean age 39.4 years, and found deficits of small to medium magnitude in psychomotor speed, attention, visual learning and all aspects of executive function [21]. The systematic review by Varghese et al. across 52 studies confirms this: in 22 of 32 comparisons (68.8%) first-episode patients performed below controls, and the domains most often affected were processing speed and executive function together with working memory [22]. Impairment is therefore already detectable in the first episode — before chronicity, before an accumulated medication history and before years of illness — and cannot be wholly a consequence of treatment or a residue laid down by repeated exacerbations. Kennedy and Ahern add a qualification absent from earlier reviews: on comparing 1288 first-episode patients with 1199 patients with a recurrent course, working memory diverged more than any other domain ($g = -0.47$), which means that an appreciable part of the deficit accrues with the number of episodes rather than being present from the outset [23]. More than half the comparisons in that same work were nonsignificant, so no across-the-board deterioration with recurrence can be claimed.

A second way of separating state from trait is to consider what persists once mood has normalised, and here a recent synthesis has shifted the emphasis. Semkovska et al. pooled 244 studies, 12 814 remitted patients and 10 578 healthy controls. On timed tasks a deficit persisted almost throughout, with magnitudes from small ($g = 0.24$) to large ($g = 0.86$). Once processing speed was controlled for, however, between-group differences ceased to be significant — with one exception. The exception was auditory-verbal and spatial working memory span, where a residual effect (g from 0.19 to 0.31) was independent of speed [24]. The authors' conclusion is unambiguous: executive dysfunction after remission is largely explained by persistent slowing of processing. For the present review this is at once a loss and a gain. The loss is that one can no longer speak of a broad enduring cognitive deficit after an episode, and that the magnitude of what remains is negligible or small. The gain is that working memory is one of the two domains that survived statistical adjustment for speed — that is, precisely the residue with which the present review is concerned.

A third line of argument comes from people who have no illness at all. MacKenzie, Uher and Pavlova pooled 54 independent samples and 8468 participants — 3246 first-degree relatives of patients with MDD and 5222 controls — and found overall cognitive performance reduced by SMD −0.19 (−0.27 to −0.11), and by −0.20 (−0.35 to −0.05) in the memory domain [25]. People who have never been ill themselves fall about a fifth of a standard deviation below controls. A caveat is obligatory: that work contains no separate working memory domain, only a broad memory domain, and its finding cannot be carried over to the n-back directly. The direction of the effect is nevertheless plain: part of the cognitive impairment seen in depression precedes the illness and travels with familial risk.

The residue has to be not merely recorded but explained by a mechanism. The most fully developed explanation ties it to rumination, and that explanation must now be viewed differently from five years ago. The experimental link holds. In an updating paradigm in which the participant memorises two lists of emotional words and must then drop one of the lists from storage, depressed patients showed a greater intrusion effect for negative words — in a neutral and in a sad mood alike — and the size of the intrusion correlated with self-reported rumination [26]. The chain runs as follows: negative self-referential material enters the buffer and is given priority; it cannot be removed on a forget instruction, because it is not perceived as irrelevant; the executive spends resource on holding it instead of on the task material; at low loads the remaining resource suffices, at 2- and 3-back it no longer does. No meta-analytic test of this paradigm itself appeared in 2021–2026, and the study cited remains the sole load-bearing support for the experimental link.

Testing on adjacent material yields a more modest result than was previously supposed. Schulze et al. pooled 109 studies and 10 637 participants on rumination and found the association between cognitive control and rumination to be selective but weak: significant associations emerged for inhibition and for updating but not for shifting, while all magnitudes remained small ($|r| < 0.15$) amid substantial heterogeneity [27]. The selectivity works in the explanation's favour: the component impaired is exactly the one at issue. The magnitude works against it: the association proved weaker than is commonly assumed. The objection concerning direction is graver still. Lopez et al. pooled 223 studies and 239 samples, 28 of them longitudinal, with 50 987 participants in total, and a path analysis of competing models yielded the reverse of the accepted ordering: a deficit of self-control predicts symptoms of depression and anxiety, and these in turn predict repetitive negative thinking [28]. In such a model rumination is a consequence of symptoms rather than a mediator between impaired control and depression. Finally, causal testing by intervention does not help the explanation either: in a meta-analysis of 44 studies, working memory training improved voluntary emotion regulation and reduced anxiety symptoms but had no effect on depressive symptoms [29]. Had rumination linked the working memory deficit to depression, training should have moved depressive symptoms as well. The position on this question is better put thus: rumination in depression is pronounced — in a transdiagnostic synthesis of 619 studies it proved the most pronounced maladaptive emotion regulation strategy (SMD = 1.51 against controls) [30] — but its role as the mechanism of the working memory deficit is at present unproven.

This explanation has a competitor to which the literature formerly paid less attention: the deficit may reflect not an inability but a reduced willingness to expend effort. Vinckier et al., in 35 patients and 35 matched healthy controls, used computational modelling of choice in a task with parametric variation of effort and reward to show that elevated effort cost is a distinctive characteristic of depressed patients [31]. A later study on a larger and unmedicated sample disputes this conclusion: Valton et al. examined 41 patients in an episode, 46 in remission and healthy controls, and found that the difference is determined not by altered sensitivity to effort or to reward but by a lower willingness to accept effort as such [32]. A transdiagnostic meta-analysis of 115 effect sizes from 68 studies (N = 3697) places this dispute in comparative context: the deficit in effort-based decision making in MDD is modest

(g = −0.28) and smaller than in schizophrenia (−0.40) and bipolar disorder (−0.39), so it is not a distinguishing feature of depression in particular [33]. For the present topic the implication is unchanged: 2-back and 3-back demand deliberate exertion sustained over minutes, and poor accuracy on them is compatible both with a malfunctioning buffer and with a decision not to invest effort. These explanations can be told apart only by tasks with external reinforcement for accuracy; meta-analyses of working memory in depression contain no such separation.

Neuroimaging might have settled this dispute, but over the past five years it has rather demonstrated the limits of its own resolving power. Two coordinate-based meta-analyses from the same group, with an identical search window, produced opposite results for working memory: activation likelihood estimation across 69 studies (2073 patients and 2009 healthy controls) revealed hyperactivation of the right middle and superior frontal gyri [34], whereas anisotropic effect-size signed differential mapping across 46 studies (1558 and 1468) left not a single cluster surviving the primary correction threshold [35]. When the sign of an effect depends on the choice of synthesis method, neuroimaging confirmation of the working memory deficit cannot be cited as established fact. The reason is evident from the methodological literature: reproducible associations between features of the brain and behaviour require thousands of observations, whereas the median sample of a neuroimaging study is about 25 people, so that effect sizes in early work are predictably inflated and reproducibility is low [36]. The entire early n-back literature in depression is built on groups of 10–15 people and is therefore serviceable as first description but not as proof.

Structural data have produced an even more definite result. The claim that hippocampal volume is reduced in depression, derived earlier from a small number of small studies, was not confirmed at all when tested in three clinical cohorts totalling 4021 individuals (1764 patients and 2257 healthy controls): no differences were found within the hippocampus, and the largest effect across the whole brain fell on the anterior insular cortex and amounted to partial $R^2 = 0.01$ [37]. The clinical implication is direct: in a patient with a mild or moderate episode there is no ground for seeking a structural substrate of the cognitive complaint. Of the influences that act on function rather than on structure, inflammation has undergone quantitative testing, and its result likewise tempers expectations: Morrens et al., across 75 studies and 29 104 participants, obtained an association between a pro-inflammatory index and global cognition of $r = −0.076$ ($p = 0.003$), which they termed very weak; individual cognitive domains, working memory included, did not differ from that level [38].

The clinical significance of all the above turns on an observation that the primary care physician should keep in mind at all times: the memory complaint and the memory test measure different things. An umbrella review with meta-analysis, pooling 50 reviews across various conditions, gives a pooled correlation between subjective and objective cognition of 0.14 (95% CI 0.08–0.21), that is under 2% of shared variance, and concludes that a discrepancy between complaint and test is in itself so common as to carry no diagnostic value [39]. It does not follow from a near-zero correlation, however, that the complaint may be disregarded. Vicent-Gil et al., in 325 patients, showed the opposite: in patients in remission, subjective appraisal of one's own executive function was the principal predictor of psychosocial functioning ($\beta = 1.09$; $p < 0.001$; $R^2 = 0.427$), while outside remission both

appraisals were significant, the subjective one weighing appreciably more than the objective (β = 1.34 against β = −0.36) [40]. The logic is this: the objective test says what a person is able to do, the subjective appraisal what the person is prepared to do, and it is the second that bears on capacity for work.

The association between the objective deficit and functioning, for its part, has turned out to be both narrower and weaker than was believed. Ahern et al. pooled 49 independent studies and 383 effect sizes (37 studies, 6991 participants) and found processing speed, attention, verbal learning and memory, shifting and global cognition to be significantly associated with psychosocial outcomes, whereas working memory was not among them, and more than half of the associations proved nonsignificant altogether [41]. Basing a prognosis of return to work on working memory is therefore premature, which makes the subjective complaint all the more valuable as a clinical indicator in its own right.

The effect of antidepressants on cognition is an area in which marketing generalisation is particularly easy to confuse with research data, and a recent synthesis makes the difference plain. In a network meta-analysis of 40 randomised trials, 32 of which evaluated antidepressants, only one drug separated significantly from placebo on cognitive outcomes — vortioxetine, and with a small effect size at that (g from −0.23 to −0.29); escitalopram, citalopram, paroxetine and fluoxetine did not differ from placebo [42]. Even this result cannot be generalised to working memory, for two reasons. First, the registration trials of vortioxetine were built around the DSST, a test of processing speed and attention rather than of storage with manipulation, and working memory was not singled out in them as a separate endpoint. Second, the result is not reproduced where the sample comes closest to the present topic. Baune et al., in 152 working patients with MDD, compared vortioxetine 10 mg, paroxetine 20 mg and placebo and by the eighth week obtained improvement on the DSST and the UPSA-B in all three groups with no significant differences between them [43]. In working outpatients precisely — that is, predominantly in mild and moderate episodes — the effect disappeared.

Non-pharmacological approaches yield more direct numbers for working memory, but these too have diverged. Legemaat et al., across 21 controlled trials of cognitive remediation, obtained an overall effect on cognition of g = 0.60, on functioning of 0.22 and on depressive symptoms of 0.28 [44]. For physical exercise the picture is more definite and less favourable: a three-level meta-analysis of 10 randomised trials and 780 patients showed that aerobic exercise significantly improves inhibitory control (g = 0.19; 95% CI 0.07–0.32; p = 0.004) and cognitive flexibility (g = 0.17; 0.01–0.33; p = 0.043), whereas working memory it does not (g = 0.08; −0.01 to 0.17; p = 0.070) [45]. For a review that deliberately keeps working memory apart from cognition at large, this is a central observation: the familiar generalisation that “exercise improves cognition in depression” does not hold for working memory in particular.

The weak points of the evidence base deserve listing without mitigation. The first has already been named: the ICD and DSM gradation of severity scarcely enters the meta-analyses, and there are virtually no data on the mild and moderate episode as a category in its own right. The second concerns medication status: the effect of the illness and the effect of its treatment cannot be separated in observational samples, and the contrast between −0.19 in unmedicated

patients and −0.56 in treatment-resistant depression shows how material this is [18]. The third concerns mechanism: neither the rumination account nor the account through effort cost has passed recent testing in its earlier formulation, and as matters stand the review is obliged to record a deficit in the absence of an established mechanism. The fourth concerns specificity: cognitive dysfunction is found in practically every mental disorder, so the working memory deficit in depression is diagnostically nonspecific and cannot be interpreted clinically out of context.

A separate question is what of all this is testable in our own setting. Thibaut et al. pooled 29 studies and 19 100 participants from 11 low- and middle-income countries, found a deficit in every depressed group and for memory obtained d = −1.60 (−2.02 to −1.18) — a value so large against the 0.2–0.6 usual in this literature that it points in itself to heterogeneity of methods and of study quality rather than to a real gap; the authors' principal conclusion, however, is that culturally validated cognitive tests are lacking [46]. Russian-language literature on the subject exists, but almost all of it describes cognition as a whole rather than working memory as a separate function: Preobrazhenskaya, for instance, treats the decline of short-term and working memory as the core of the cognitive complaint in depression and notes that the impairment persists after the episode has resolved [47]. No work on working memory in the depressive episode specifically, carried out in Uzbekistan or Kazakhstan, could be found, nor could any current population estimate of the prevalence of depression for the region. The gap is evident and can be filled: a prospective study using the n-back and backward digit span in outpatients with F32.0 and F32.1, stratified by the PHQ-9 and assessed before and after eight weeks of treatment, requires neither costly equipment nor large samples.

The upshot consists of four statements of differing strength. A working memory deficit in the depressive episode exists and does not reduce to generalised slowing: accuracy falls in a load-dependent manner from 1-back onwards while remaining normal at 0-back [19], and working memory span turns out to be one of the two measures that survive statistical adjustment for processing speed in remission [24]. The magnitude of the deficit in the mild and moderate episode is in all likelihood smaller than pooled meta-analytic estimates imply: in unmedicated patients it is half the overall value [18], its dependence on severity concerns manipulation of held material rather than simple storage [17], and within a single patient the test does not track fluctuations of the episode at all [20]. Direct data on the ICD gradations do not exist, and this is a defect of the literature, not a feature of the illness. The mechanism is not established: the rumination account retains its selectivity but loses magnitude and possibly direction [27, 28], while the account through elevated effort cost has been disputed on a larger sample and proved not to be specific to depression [32, 33]. Two conclusions follow for practice. The memory complaint and the test result measure different things, their shared variance falling short of two per cent [39], so a normal test does not annul the complaint, and it is the subjective appraisal that better predicts functioning [40]. And full cognitive recovery in the wake of mood should not be expected: the cognitive complaint persists for 44% of the time spent in remission [8], change over the course of the illness is nonsignificant, negligible or small for 95% of measures [9], the procognitive effect of antidepressants is small and tied to a single molecule [42], and aerobic exercise, while improving inhibition and flexibility, does not affect

working memory [45].

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