Differential Adherence: Orthogonal Decomposition of Medication Nonadherence Reveals Clinically Significant Heterogeneity Destroyed by Sum-Score Instruments

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Abstract

Background: Medication nonadherence costs health care systems billions annually and is associated with increased morbidity and mortality. Although the distinction between intentional nonadherence (deliberate modification of prescribed regimens) and unintentional nonadherence (forgetting or carelessness) has been recognized for decades, the most widely used adherence instruments, including the Morisky Medication Adherence Scale (MMAS), collapse these mechanistically distinct behaviors into a single sum-score. The information lost in this collapsing operation has never been formally quantified.

Methods: Here our objective is to demonstrate that an orthogonal decomposition of adherence into sum (total nonadherence magnitude) and difference (differential adherence direction) components captures statistically significant clinical heterogeneity that sum-scoring destroys by collapsing a two-dimensional measurement to one**.** Secondary analysis of the NeuroGerAd study (N=907 adults with neurological disorders). The Stendal Adherence to Medication Score (SAMS) sub-factor means for intentional modification (9 items) and unintentional forgetting (5 items) were decomposed into orthogonal components: a sum-score proxy (S = intentional +

unintentional, analogous to MMAS) and a Differential Adherence Index (Δ = intentional − unintentional). Orthogonality was tested via Pearson correlation. Between-group discrimination was compared via one-way ANOVA across 5 neurological diagnosis groups for both S and Δ.

Results: The sum-score and Δ were near-perfectly orthogonal ($r = -0.077$, sharing only 0.6% of variance). Diagnosis groups differed significantly on Δ ($F(4,902) = 2.63$, $P = .03$, $\eta^2 = 0.012$) but not on the sum-score ($F(4,902) = 1.48$, $P = .21$, $\eta^2 = 0.007$). The strongest pairwise contrast was movement disorder versus neuromuscular (Δ difference: $t(468) = -2.85$, $P = .005$, Cohen $d = 0.28$), consistent with cognitive decline driving forgetting-dominant profiles in movement disorders versus preserved cognition allowing deliberate modification in neuromuscular conditions.

Conclusions: Sum-score adherence instruments destroy a statistically significant, clinically interpretable dimension of medication-taking behavior. The Differential Adherence Index recovers this information at zero additional measurement cost when sub-factor scores are available, enabling targeted intervention matching. Analysis code and reproducibility materials are openly available.

Keywords: medication adherence; intentional nonadherence; unintentional nonadherence; Morisky Medication Adherence Scale; orthogonal decomposition; neurological disorders; SAMS; differential adherence

Background

Medication nonadherence is among the most consequential yet inadequately measured problems in clinical medicine. The World Health Organization has estimated that adherence to long-term therapies in developed countries averages only 50%, with rates substantially lower in developing nations [1]. The downstream consequences are severe: nonadherence accounts for an estimated 125,000 deaths and $100–289 billion in avoidable health care costs annually in the United States alone [2]. Neurological conditions carry a particularly high nonadherence burden, as patients often manage complex polypharmacy regimens in the context of cognitive, motor, and psychiatric comorbidities [3,4].

A growing body of work has recognized that nonadherence is not a unitary construct. Lehane and McCarthy [5] proposed a comprehensive framework distinguishing intentional nonadherence—deliberate decisions to modify, skip, or discontinue prescribed medication—from unintentional nonadherence—passive failures arising from forgetting, carelessness, or misunderstanding. Clifford et al [6] demonstrated that intentional and unintentional nonadherers hold fundamentally different medication beliefs: intentional nonadherers perceive lower necessity and higher concerns, while unintentional nonadherers resemble adherers in their belief profiles. Wroe [7] framed the distinction in terms of decision-making processes, and Gadkari and McHorney [8] provided large-scale epidemiological evidence that unintentional nonadherence is far more prevalent yet often co-occurs with intentional nonadherence in ways that sum-scoring cannot disentangle.

Despite this conceptual consensus, the most widely used self-report adherence instruments continue to collapse intentional and unintentional behaviors into a single number. The Morisky Medication Adherence Scale (MMAS-4 and MMAS-8) [9,10], which has been cited over 4000 times and translated into more than 80 languages, produces a sum-score that treats forgetting a

dose and deliberately stopping a medication as interchangeable contributions to the same index. This practice has been questioned on psychometric grounds: Martinez-Perez et al [11] found low internal consistency and a 3-factor structure in Spanish diabetes patients, and multiple validation studies have reported that the assumed unidimensionality of the MMAS does not hold across clinical populations [12,13].

The Stendal Adherence to Medication Score (SAMS) [14,15] offers a more granular alternative. Developed and validated as an 18-item self-report instrument, the SAMS has been shown via confirmatory factor analysis and network analysis to contain 3 distinct sub-factors: forgetting (unintentional nonadherence), intentional modification of medication, and missing knowledge about the regimen [15,16]. Franke et al [16] identified these sub-factors across pooled samples totaling 1746 patients, confirming their structural robustness.

The critical question, which to our knowledge has not been formally addressed, is: how much clinically relevant information is destroyed when these validated sub-factors are collapsed into a sum-score? This is not merely a psychometric curiosity. If patients with identical sum-scores differ systematically in the direction of their nonadherence—some predominantly forgetting, others predominantly choosing to modify—then the sum-score is masking heterogeneity that requires different clinical responses. Forgetting-dominant patients benefit from reminders, simplified regimens, and pill organizers; intention-dominant patients require shared decision-making, side-effect counseling, and motivational interventions [5,6,17].

In this study, we apply an orthogonal decomposition to SAMS sub-factor scores from the NeuroGerAd study [3,4], a large cohort of adults with neurological disorders. We decompose the 2-dimensional (intentional, unintentional) adherence space into 2 orthogonal components—a

sum-score (analogous to MMAS) and a Differential Adherence Index (Δ = intentional − unintentional)—and test whether diagnosis groups differ on Δ when the sum-score shows no between-group discrimination. A positive finding would constitute the first formal demonstration that sum-scoring destroys clinically significant signal recoverable at zero additional measurement cost.

Methods

Data Source

This study is a secondary analysis of the NeuroGerAd study, a longitudinal observational study of medication adherence in adults with neurological disorders conducted at the Department of Neurology, Jena University Hospital, Germany, between February 2019 and March 2020 [3]. The study enrolled 910 consecutive inpatients aged 55 years and older, with diagnoses classified into 5 groups: movement disorders (n=303), cerebrovascular disorders (n=231), neuromuscular disorders (n=167), epilepsy (n=48), and others (n=158). The dataset is freely available for noncommercial scientific purposes from OSF [18]. The original study was approved by the ethics committee of Jena University Hospital (approval number 5290-10/17) and all participants provided written informed consent [3].

Instrument

Medication adherence was assessed using the SAMS [14], an 18-item self-report instrument with items scored 0 (never) to 4 (most of the time), where higher scores indicate greater nonadherence. The SAMS yields a total score (range 0–72) as well as 3 validated sub-factor scores identified via confirmatory factor analysis and network analysis [15,16]:

forgetting/unintentional nonadherence (items 6, 14, 15, 16, 18; 5 items), intentional medication modification (items 4, 7, 8, 9, 10, 11, 12, 13, 17; 9 items), and missing knowledge (items 1, 2, 3, 5; 4 items). Because the sub-factors contain unequal numbers of items, we computed sub-factor means (range 0–4) rather than sums to ensure comparability. Sub-factor means were computed using person-mean scoring (ie, averaging available items within each subscale), which retains participants with partial item-level data provided at least 1 item per subscale was answered. Three participants with entirely missing subscale data were excluded, yielding the analytic sample of N=907.

Orthogonal Decomposition

Let I denote the intentional modification sub-factor mean and U denote the unintentional (forgetting) sub-factor mean for each patient. We define 2 derived variables:

Sum-score proxy: $S = I + U$ (analogous to MMAS-type scoring, capturing total nonadherence magnitude)

Differential Adherence Index: $\Delta = I - U$ (capturing nonadherence directionality)

This decomposition represents a 45-degree rotation of the (U, I) coordinate system. Because $\mathrm{Cov}(S, \Delta) = \mathrm{Var}(I) - \mathrm{Var}(U)$, the sum and difference are exactly uncorrelated whenever the two sub-factor variances are equal, and approximately so when they are close. Any between-group variance captured by Δ but not by S therefore represents clinically relevant signal that is not identifiable from S alone: the map $(I, U) \rightarrow S$ is a many-to-one projection that provably destroys individual-level directionality.

Statistical Analysis

Orthogonality between S and Δ was assessed via Pearson correlation. Between-group discrimination was tested using one-way ANOVA across 5 diagnosis groups for both S and Δ, with effect sizes reported as eta-squared (η^2). Pairwise comparisons used independent-samples t tests with Cohen d as the effect size. Significance was set at $\alpha = .05$ (2-tailed). No corrections for multiple comparisons were applied to the pairwise tests, as these were exploratory. All analyses were conducted in Python 3.12 (tested with NumPy 1.26, pandas 2.2, SciPy 1.14, and matplotlib 3.9). Analysis code is openly available [19]. Reporting follows STROBE guidelines (Additional File 1).

Sensitivity Analysis

To assess robustness to missing item responses, analyses were repeated under progressively stricter inclusion criteria: person-mean scoring with no minimum item threshold (primary analysis, N=907), ≥50% item completion per subscale (N=881), ≥80% item completion per subscale (N=832), and complete-case analysis requiring all items answered (N=755). As expected, stricter criteria reduced sample size and statistical power.

Results

Descriptive Statistics

The analytic sample comprised 907 adults with neurological disorders across 5 diagnosis groups (Table 1). On the 0–4 scale, mean unintentional nonadherence (M = 0.425, SD = 0.542) was higher than mean intentional nonadherence (M = 0.297, SD = 0.507), indicating that forgetting was the predominant nonadherence mechanism in this population. The mean Differential Adherence Index was negative ($\Delta = -0.129$, SD = 0.528), reflecting this population-level

forgetting dominance. However, 53.5% of patients (n=485) had $\Delta \geq 0$ (intentional-dominant or balanced), indicating substantial individual-level heterogeneity not captured by the population mean.

Table 1. Population Statistics by Diagnosis Group

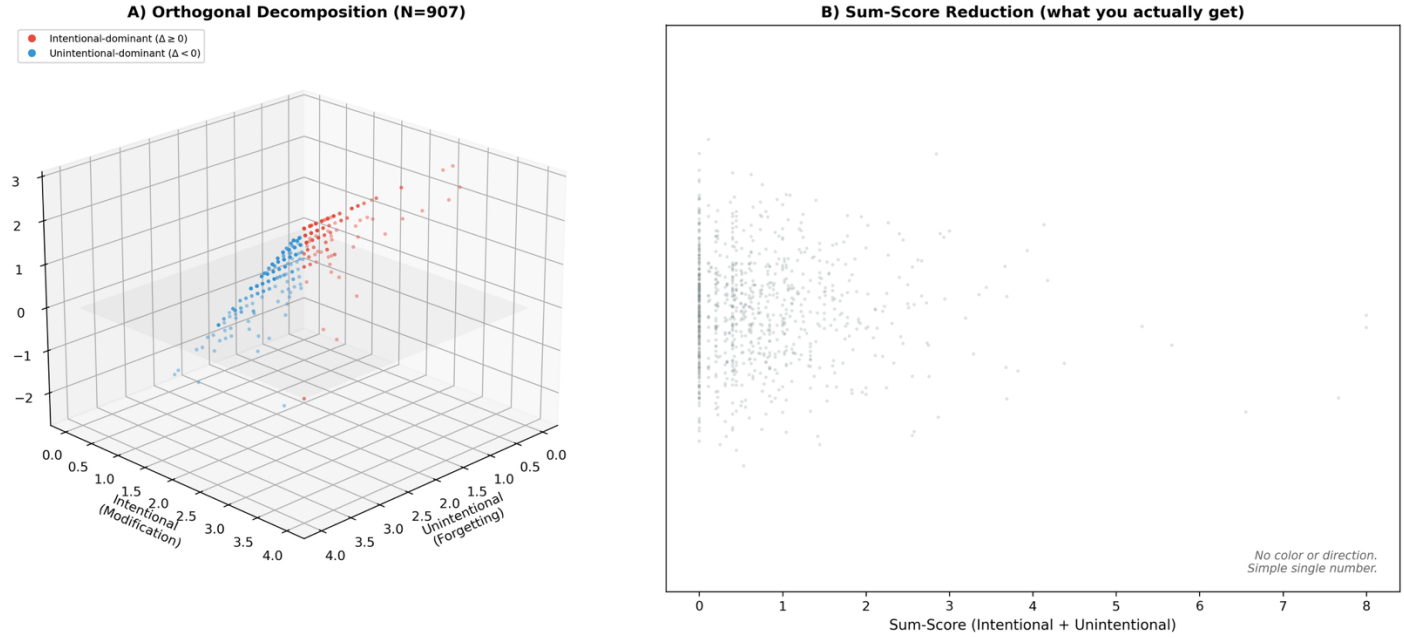
Diagnosis	N	Δ Mean	Δ SD	% Int	S Mean	S SD	P (vs NM)
Movement disorder	303	−0.180	0.589	46.9	0.825	0.957	.005
Cerebrovascular	231	−0.108	0.443	57.1	0.672	0.972	.088
Others	158	−0.146	0.545	53.8	0.682	0.775	.041
Epilepsy	48	−0.197	0.497	50.0	0.674	0.679	.038
Neuromuscular	167	−0.027	0.496	61.1	0.657	0.883	Ref

Δ = intentional − unintentional (sub-factor means, 0–4 scale). % Int = percentage of patients with Δ ≥ 0 (intentional-dominant). S = sum-score proxy (intentional + unintentional). P (vs NM) = P value for pairwise t test on Δ versus neuromuscular group (reference). All P values 2-tailed.

Orthogonality

The Pearson correlation between S and Δ was $r = -0.077$ ($P = .02$), corresponding to only 0.6% shared variance. This confirms near-perfect orthogonality: Δ captures a dimension of nonadherence that is effectively independent of S. The small but significant negative correlation reflects slightly higher variance in unintentional (SD = 0.542) than intentional (SD = 0.507) scores; perfect orthogonality obtains when Var(I) = Var(U) exactly (see Methods). Figure 1A displays the orthogonal decomposition, and Figure 1B illustrates the information lost by collapsing to a sum-score.

Figure 1.



Orthogonal Decomposition vs Sum-Score Reduction

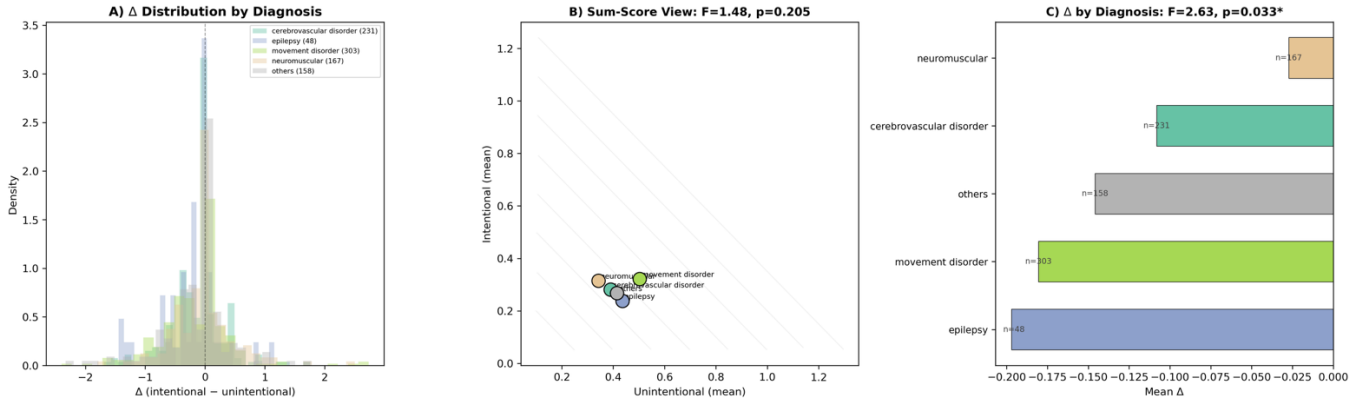
(A) Three-dimensional visualization of the orthogonal decomposition. The horizontal axes represent unintentional (forgetting) and intentional (modification) sub-factor means. The vertical axis represents the Differential Adherence Index (Δ = intentional − unintentional). Red: intentional-dominant patients (Δ ≥ 0); blue: unintentional-dominant patients (Δ < 0). The gray plane marks Δ = 0. (B) Sum-score reduction: the same 907 patients collapsed to a single number along the horizontal axis. Vertical jitter is added solely to visualize point density and carries no information; the data are one-dimensional. All directional structure visible in Panel A—the red/blue distinction, the vertical spread, the diagnostic-group separation—is unrecoverable from the sum score alone (many-to-one projection).

Between-Group Discrimination

One-way ANOVA revealed a statistically significant effect of diagnosis group on Δ ($F(4,902) = 2.63$, $P = .03$, $\eta^2 = 0.012$) but not on the sum-score ($F(4,902) = 1.48$, $P = .21$, $\eta^2 = 0.007$). That is, diagnosis groups differed in the direction of their nonadherence while appearing indistinguishable in its magnitude. This is the core finding: the information captured by Δ is statistically significant, clinically interpretable, and invisible to sum-score instruments.

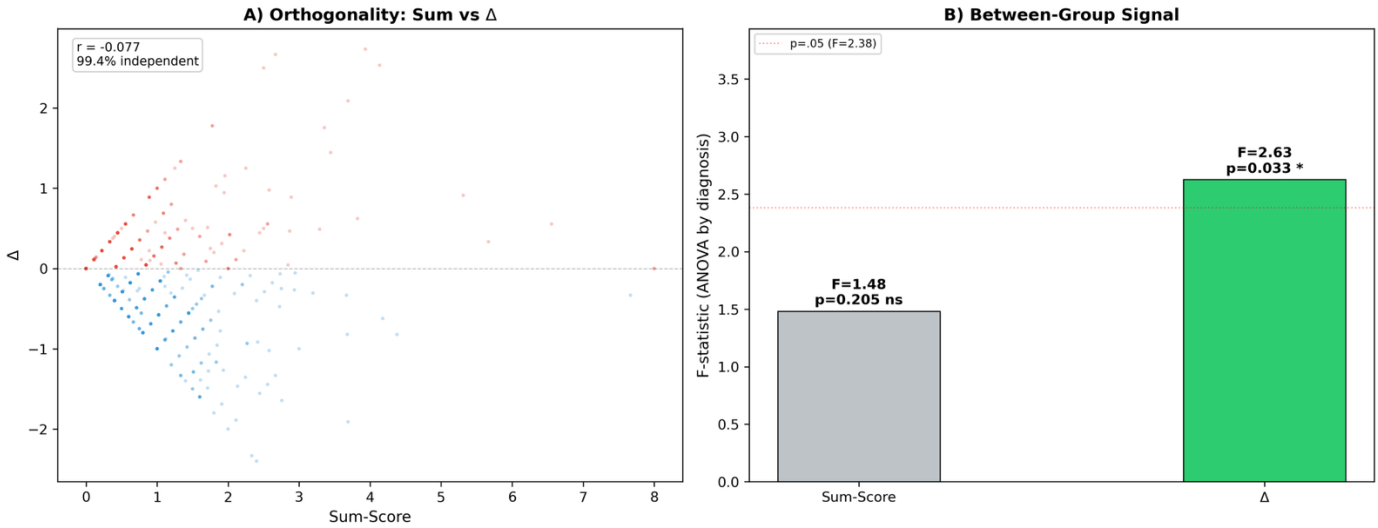
Pairwise comparisons on Δ (Table 1) revealed the strongest contrast between movement disorder and neuromuscular groups ($t(468) = -2.85$, $P = .005$, $d = 0.28$). Movement disorder patients showed the most forgetting-dominant profile ($\Delta = -0.180$), while neuromuscular patients were nearly balanced ($\Delta = -0.027$). Epilepsy versus neuromuscular ($t(213) = -2.09$, $P = .04$, $d = 0.34$) and neuromuscular versus others ($t(323) = 2.05$, $P = .04$, $d = 0.23$) also reached significance. These contrasts were entirely absent from the sum-score analysis (Figure 2).

Figure 2. Group-Level Heterogeneity in Differential Adherence



(A) Density plots of Δ by diagnosis group. (B) Group means in the sum-score view (intentional vs unintentional axes); gray iso-sum lines connect points with identical sum-scores. Groups cluster indistinguishably ($F = 1.48$, $P = .21$). (C) Mean Δ by diagnosis group reveals significant between-group heterogeneity ($F = 2.63$, $P = .03$) invisible to sum-scoring.

Figure 3. Information Loss Quantification



(A) Scatter plot of sum-score (S) versus Δ, colored by nonadherence directionality. Near-zero correlation (r = −0.077) confirms orthogonality. (B) F-statistic comparison: Δ exceeds the P = .05 critical threshold for between-group discrimination; the sum-score does not. The red dashed line indicates the critical F value (F = 2.38 at α = .05).

Sensitivity Analysis

The Differential Adherence Index (Δ) consistently differentiated movement disorder and neuromuscular diagnosis groups across all inclusion criteria (Table 2). The primary pairwise contrast was statistically significant and directionally stable under all specifications (P range: .005–.016, d range: 0.26–0.28). The omnibus ANOVA was sensitive to sample size reduction, reaching P = .05 at the 80% item threshold and P = .14 under complete-case analysis. The sum-score ANOVA remained nonsignificant under all specifications.

Table 2. Sensitivity Analysis Across Missing-Data Handling Approaches

Approach	N	Δ F	Δ P	Sum P	t (pw)	P (pw)	d
Person-mean	907	2.63	.033	.205	−2.85	.005	0.28
≥50% items	881	2.51	.040	.088	−2.78	.006	0.27
≥80% items	832	2.37	.051	.078	−2.72	.007	0.27
Complete-case	755	1.75	.137	.112	−2.41	.016	0.26

Δ F and Δ P = omnibus ANOVA F-statistic and P value for the Differential Adherence Index across 5 diagnosis groups. Sum P = omnibus ANOVA P value for the sum-score. t (pw), P (pw), d = pairwise t test, P value, and Cohen d for the movement disorder versus neuromuscular contrast. All P values 2-tailed.

Discussion

Principal Findings

This study provides the first formal demonstration that sum-score adherence instruments destroy clinically significant between-group signal. By decomposing the SAMS intentional and unintentional sub-factor scores into orthogonal components, we showed that the Differential Adherence Index (Δ) shares only 0.6% of variance with the sum-score and captures statistically significant diagnostic-group heterogeneity (P = .03) where the sum-score captures none (P = .21). The mathematical operation is trivial—a 45-degree rotation of the existing 2-dimensional measurement space—yet its clinical implications are substantial.

Mechanistic Interpretation

The observed pattern of group differences on Δ is consistent with known disease mechanisms. Movement disorder patients, who showed the most forgetting-dominant profile (Δ = −0.180), include those with Parkinson disease and related conditions characterized by progressive

cognitive decline, executive dysfunction, and memory impairment [20]. These deficits directly impair the capacity to maintain medication routines, producing unintentional nonadherence. Conversely, neuromuscular patients, who showed the most balanced profile ($\Delta = -0.027$), typically have preserved cognition, allowing more deliberate engagement with their medication regimens, including intentional modifications based on symptom fluctuation or side-effect management. Epilepsy patients showed the strongest forgetting dominance ($\Delta = -0.197$), consistent with seizure-related cognitive disruption and the known effects of antiepileptic medications on memory and attention [21]. These mechanistically coherent patterns are exemplary of the precise class of signal that should inform clinical decision-making but which is invisible to instruments that report only a sum-score.

Relation to Prior Work

The conceptual distinction between intentional and unintentional nonadherence is well-established. Lehane and McCarthy [5] argued for its clinical centrality nearly 2 decades ago, and Clifford et al [6] demonstrated that the 2 types are associated with distinct belief structures. Gadkari and McHorney [8] showed that they co-occur in complex patterns across 24,017 patients with chronic conditions. Network analysis of SAMS data by Franke et al [16] identified 3 structural clusters corresponding to forgetting, intentional modification, and missing knowledge. What has been lacking is a formal demonstration of what is lost when these validated sub-factors are collapsed. Our orthogonality analysis provides this: the sum and difference are algebraically (and near-perfectly empirically) independent, meaning that no statistical manipulation of the sum-score can recover the information encoded in Δ.

Limitations

Several limitations should be considered. First, the effect size for the between-group Δ difference was small ($\eta^2 = 0.012$). The significance of this finding lies not in the absolute magnitude of variance explained, but in the ratio: Δ captures between-group signal where the sum-score captures none. Second, sub-factor means were computed using person-mean scoring, which retains participants with partial item-level data (152 of 907 participants had at least 1 missing item within a subscale). The attenuation of omnibus significance under complete-case analysis (Table 2) reflects loss of power rather than loss of signal, as evidenced by stable effect direction and preserved pairwise contrasts across all inclusion thresholds. This behavior is consistent with detection of small but clinically meaningful effects in heterogeneous populations. Third, difference scores have well-known psychometric properties that may reduce reliability relative to their component sub-factors [22]. If the sub-factor reliabilities are not both high, Δ inherits amplified measurement error. The SAMS sub-factor structure has been validated via confirmatory factor analysis [15,16], which provides partial reassurance, but future work should report sub-factor reliabilities explicitly. Fourth, the NeuroGerAd sample consists exclusively of German neurological inpatients aged 55 years and older; generalization to other populations, clinical settings, and age groups requires replication. Fifth, the SAMS is a self-report instrument subject to social desirability bias and recall error, limitations shared with all self-report adherence measures, including the MMAS [9,10]. Sixth, the epilepsy subgroup (n=48) is small, limiting the reliability of pairwise comparisons involving this group. Pairwise contrasts were uncorrected exploratory comparisons; the movement disorder versus neuromuscular contrast survives Bonferroni correction for 10 comparisons ($\alpha = .005$), but other pairwise results should be interpreted cautiously.

Clinical Implications

The practical implication is immediate and requires no new data collection. Any adherence instrument with validated sub-factors—including the SAMS, and potentially reclassified MMAS items—can be decomposed into sum and difference components. The Δ score is computable from existing data and provides actionable information about the direction of nonadherence: patients with $\Delta > 0$ are intentional-modification-dominant and may benefit from shared decision-making, side-effect counseling, and motivational interviewing; patients with $\Delta < 0$ are forgetting-dominant and may benefit from reminders, simplified regimens, and pill organizers [5,6,17]. This targeting is impossible when only a sum-score is reported. Clinical decision support systems could readily incorporate Δ alongside existing adherence scores to support intervention matching.

Future Directions

The orthogonal decomposition presented here is the simplest possible rotation of the adherence measurement space. Future work should explore whether the information-geometric structure of the intentional–unintentional space supports richer decompositions, potentially incorporating higher-order interactions between adherence dimensions and time-varying covariates. The missing knowledge sub-factor of the SAMS, not analyzed here, may contribute additional orthogonal information. Longitudinal analysis of Δ trajectories could reveal whether patients drift between intentional and unintentional nonadherence profiles over time, which would have implications for adaptive intervention design.

Conclusions

Sum-score adherence instruments collapse mechanistically distinct forms of nonadherence into a single number, destroying clinically significant between-group signal in the process. The Differential Adherence Index recovers this information through a mathematically trivial orthogonal decomposition of existing sub-factor scores. In the NeuroGerAd cohort, diagnosis groups that appeared identical on a sum-score metric differed significantly on Δ in mechanistically coherent patterns. Reporting Δ alongside sum-scores requires no additional measurement burden and enables targeted intervention matching. We recommend that adherence research report sub-factor scores and their derived difference components rather than relying solely on sum-scores that provably discard clinically relevant information.

Declarations

Ethics approval and consent to participate

This study is a secondary analysis of deidentified, publicly available data from the NeuroGerAd study. The original study was approved by the ethics committee of Jena University Hospital (approval number 5290-10/17), and all participants provided written informed consent [3]. Because the present analysis used only the publicly released, deidentified dataset [18], no additional ethics review was sought, consistent with German and international guidelines exempting secondary analysis of anonymized data from further institutional review. No personally identifiable information was accessed or analyzed. Participants in the original study were not compensated specifically for the adherence assessment component; compensation details for the parent study are reported in Prell et al [3].

Consent for publication

Not applicable.

Availability of data and materials

The NeuroGerAd dataset is freely available for noncommercial scientific purposes from OSF [18]. Analysis code (Python) and reproducibility materials are available at https://github.com/amr28693/orthogonal_differential_adherence [19].

Competing interests

The author declares no competing interests.

Funding

The present analysis received no external funding.

Authors' contributions

AMR: conceptualization, methodology, software, formal analysis, visualization, writing–original draft, writing–review and editing.

Acknowledgements

The NeuroGerAd data were collected by Prell, Schönenberg, Mendorf, Mühlhammer, Grosskreutz, and Teschner at Jena University Hospital, funded by BMBF grant 01GY1804 to T. Prell. The author gratefully acknowledges their contribution in making the dataset openly available.

Abbreviations

ANOVA: analysis of variance

MMAS: Morisky Medication Adherence Scale

OSF: Open Science Framework

SAMS: Stendal Adherence to Medication Score

Additional file 1: STROBE checklist for cross-sectional studies.

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