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ORIGINAL RESEARCH

The Mediating Roles of Self-Efficacy, Resilience, and Social Support in the Relationship Between Clinical Factors and Adaptation to Chronic Disease

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Purpose: Psychosocial adaptation to chronic disease plays a central role in determining quality of life. Yet the mechanisms linking clinical factors to adaptation remain insufficiently understood, especially in Eastern European contexts and across different chronic conditions. This study tested whether self-efficacy mediates the relationship between comorbidities and number of medications on adaptation to chronic disease (as indicators of clinical burden and treatment complexity) and examined perceived social support and resilience as alternative mediators.

Patients and Methods: Participants were 263 adults with chronic diseases recruited from public hospitals in Romania. Measures included the SEMCD (self-efficacy), CIAS (adaptation; total and the compliant behavior [CIAS-f2] and emotional support [CIAS-f4] subscales), BRS (resilience), and MOS-SSS (social support), with higher scores indicating higher levels. A cross-sectional design was used, and four simple mediation models were estimated, each with one mediator, using bootstrapping with 5000 resamples (95% bias-corrected CIs) in JASP 0.19.

Results: Using unstandardized coefficients, the indirect effect of comorbidities on adaptation through self-efficacy was significant ($a \times b = -0.17$, 95% CI −0.29 to −0.09), while the direct path was not, indicating complete mediation. For the number of medications, the indirect effect through self-efficacy was −0.02 (95% CI −0.04 to −0.01), with a nonsignificant direct effect. Mediation emerged for CIAS-f2 (compliant behavior) but not for CIAS-f4 (emotional support needs). Social support showed a direct positive association with adaptation ($b = 0.45$, $P < 0.001$) without mediation, whereas resilience showed no significant effects.

Conclusion: The findings indicate a significant indirect pathway through which comorbidities and complex medication regimens are associated with adaptation via self-efficacy. Social support showed a direct, rather than mediating, association with adaptation, whereas resilience appeared to have a limited role. These results suggest that clinical practice may benefit from focusing on simplifying treatment regimens, enhancing self-management efficacy through personalized education, and mobilizing social support.

Keywords: self-efficacy, chronic disease adaptation, psychosocial factors, mediation analysis, social support

Introduction

The rapid technological progress, including digitization and applications of artificial intelligence, has reshaped medicine and pharmacology in recent years. Over the past decade, international reports have documented improved survival or

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reduced mortality across major chronic disease categories, including end-stage renal disease, cardiovascular conditions (ischemic heart disease and stroke), and several types of cancer.[1–4] In parallel, analyses have shown a shift in focus from the initial "living longer" to the current perception of "living better", that is, from simply extending longevity to extending lifespan quality. [5–7] Long-term well-being has become an explicit goal of medicine, with positive implications for both life expectancy and mental health.[8] In this context, adaptation to chronic diseases is a central component of well-being and provides its psychological foundation.[9]

In chronic disease management, the primary goal is no longer just survival but sustained psychological adaptation. The TMA-LTC[9] transdiagnostic model posits that psychological processes (eg, self-efficacy) mediate the effects of biological factors on adaptation to chronic disease, hence the focus of this study on mechanisms rather than symptoms. In this study, adaptation is conceptualized as a dynamic process of integrating illness into one's identity and self-management practices, essential for living well with a chronic condition.[10] Insufficient adaptation is associated with more frequent use of health services and with less sustainable and poorer long-term health outcomes, thus underscoring its clinical importance.[11] In the present study, particular attention was given to the compliant behavior (factor f2) and emotional support (factor f4), as they capture behavioral and relational aspects of adaptation that are especially sensitive to clinical burden. Based on Bandura's theory,[12] self-efficacy in chronic disease management expresses the belief that specific self-management behaviors can be successfully implemented. In this study, self-efficacy is conceptualized broadly, in line with the SEMCD scale, capturing perceived competence across multiple self-management behaviors. In medical contexts, this reflects patients' confidence in tasks such as preventing fatigue/pain/distress from disrupting daily activities, communicating effectively with physicians, and administering medications correctly.[13,14] Other psychosocial processes relevant to adaptation, and considered in our conceptual framing, include resilience (the capacity to cope with and overcome disease-related adversity while maintaining daily functioning and psychosocial adjustment) and social support (relational resources from family, friends, and peers that facilitate disease management and are linked to better outcomes).[15]

Interventions focused on developing self-management have consistently improved self-efficacy and, in turn, patient-reported outcomes among people living with chronic disease[16] and among older adults.[17] Although recent studies have analyzed clinical factors as antecedents with self-efficacy[13,18,19] as an outcome, or self-efficacy as an antecedent with quality of life as an outcome[20–22] comparatively few have explicitly tested self-efficacy as a mediator between clinical factors (comorbidity, number of medications per day) and adaptation to chronic disease, rather than generic quality-of-life indices.[15] Moreover, another limitation in current research is the frequent reliance on diagnosis-specific samples (eg, HIV or cancer),[6,10] rather than transdiagnostic models that leverage dedicated instruments and specific subscales for a more comprehensive approach (eg, compliant behavior, emotional support). Finally, studies from Eastern European settings remain limited.[23] In Eastern European contexts, factors such as healthcare system constraints, access to resources, and sociocultural norms may shape how patients adapt to chronic disease. Recent evidence from Romania has highlighted the relevance of psychosocial mechanisms in health-related contexts,[24] and this study adds further data on adaptation to chronic disease within public hospitals in Romania.

Based on the TMA-LTC framework theorized by Carroll et al[9],the conceptual model of this research was outlined as the relation between the clinical factors represented by comorbidity and number of medications (antecedents), self-efficacy as the key psychological process (mediator), and adaptation to chronic disease (outcome), followed by specific, testable hypotheses.

The first two models tested examined the relationships between comorbidities (Model 1) and the number of medications per day (Model 2), as clinical antecedents, and adaptation to chronic disease, considering self-efficacy as a key mediator. The transdiagnostic TMA-LTC framework and Livneh's[15] synthesis explicitly place clinical factors as influences on psychological processes, including self-efficacy, which has a mediating role toward adaptation to chronic disease.[9,15]

Empirically, in people with multimorbidity, treatment burden is associated with lower self-efficacy, suggesting that the complexity of the health condition may erode the sense of self-efficacy and the capability to self-manage the diseases.[25,26] Following the same logic, a higher number of medications correlates with lower self-efficacy for disease and medication management.[19] In contrast, self-efficacy supports adherence, self-regulation, and coping, and is consistently related to adaptation and quality of life in chronic disease.[20,21,27]

From these premises, an indirect effect was expected to follow: greater comorbidity (and, analogously, more medications) determines lower self-efficacy, which determines poorer adaptation.

In addition to the main hypotheses, two secondary hypotheses (H3–H4) examined the potential mediating roles of perceived social support and resilience.

Two models in which psychological resilience and social support were proposed to mediate the link between comorbidities and adaptation to chronic disease were tested. In the TMA-LTC theoretical framework, such resources are embedded in processes through which clinical antecedents influence outcomes, justifying their examination as mediators.[9] In this conceptualization, resilience reflects an internal capacity to maintain functioning under adversity, whereas social support represents an external, relational resource; both are positioned as processes through which clinical burden may influence adaptation, rather than as moderators or background covariates.

Recent evidence indicates that resilience is associated with daily functioning and psychosocial adjustment,[28] and clinical complexity (number of illnesses, number of medications per day) correlates with lower resilience.[22] At the same time, social support is linked to resilience and better adaptation, acting as a relational resource that facilitates disease management.[29] Convergently, in children with chronic diseases, "family resilience" is associated with better psychosocial adjustment.[30]

Taken together, this rationale motivates the present study, in which these mediation pathways are tested, contrasting the basic (self-efficacy) model with the alternative (resilience/social support) model in a transdiagnostic sample.

The purpose of this study was to examine whether self-efficacy mediates the relationship between clinical factors (comorbidities and number of medications per day) and adaptation to chronic disease. In addition, the study aimed to test, in Models 3 and 4, the role of social support and resilience as possible mediators in this process. By examining these pathways in a transdiagnostic sample, the study shifts the focus from diagnosis-specific effects to shared psychosocial processes linking clinical burden and adaptation.

Research Question

Research purpose: The purpose of this study was to examine whether self-efficacy mediates the relationship between clinical factors (comorbidities and number of medications per day) and adaptation to chronic disease. In addition, the study aimed to test, in Models 3 and 4, the role of social support and resilience as possible mediators in this process.

RQ1. Does self-efficacy mediate the relationship between clinical factors (comorbidities, number of medications) and adaptation to chronic disease, and what role do social support and resilience play in this process?

Hypotheses

Model 1. Comorbidities – Self-Efficacy – Adaptation to Chronic Disease

H1a. Comorbidities will predict lower levels of self-efficacy.

H1b. Self-efficacy will positively predict adaptation.

H1c. Self-efficacy will mediate the relationship between comorbidities and adaptation – compliant behavior.

H1d. Self-efficacy will mediate the relationship between comorbidities and adaptation – need for emotional support.

Model 2. Number of Medications Per Day – Self-Efficacy – Adaptation to Chronic Disease

H2a. Higher number of medications per day will predict lower levels of self-efficacy.

H2b. Self-efficacy will positively predict adaptation.

H2c. Self-efficacy will mediate the relationship between number of medications per day and adaptation– compliant behavior.

H2d. Self-efficacy will mediate the relationship between number of medications per day and adaptation – need for emotional support.

Models 3 and 4 – for Social Support and Resilience as Mediators

H3. Social support will mediate the relationship between clinical factors and adaptation.

H4. Resilience will mediate the relationship between clinical factors and adaptation.

Materials and Methods

The sample used in this study is the same as the one described in Laza et al.[31] The two papers, however, address different aims and apply distinct analytical approaches. The earlier article examined the direct predictive role of psychosocial and clinical factors, specifically the type of diagnosis, comorbidities, time since diagnosis, and number of medications, on patients' adaptation to chronic disease. In contrast, the present study focuses on the psychological mechanisms that explain these associations. Specifically, in this cross-sectional design, it was examined whether self-efficacy mediates the relationship between clinical variables and adaptation within the TMA–LTC framework, which conceptualizes adaptation to chronic disease as the outcome of mediated processes involving psychological mechanisms relevant to self-management. In addition, two alternative mediators, social support and resilience, were explored to provide a complementary perspective on the pathways linking clinical complexity to adaptation. The clinical variables of interest were comorbidities and the number of medications taken daily, while the psychological mediators were self-efficacy, perceived social support, and resilience.

Participants

The study involved 263 adult patients with chronic conditions who were undergoing treatment in public healthcare institutions in Timișoara, Romania. The sample size was considered adequate for mediation analyses with bootstrapping, given the number of tested models and parameters. The same cohort of participants was previously described in detail in Laza et al[31] (2024). Patients were recruited from several hospital units for chronic conditions and all had advanced, non-remitting diseases. Participants were recruited using a convenience sampling approach, based on their availability during routine medical visits. Inclusion criteria were as follows: participants aged 18 years or older; a confirmed diagnosis of a non-remitting chronic disease; ongoing treatment in public healthcare institutions; ability to understand the study information and provide informed consent; and willingness to complete the questionnaires independently or with minimal assistance. Exclusion criteria included: severe cognitive impairment or acute psychiatric conditions that could compromise the validity of self-reported data; clinically unstable or critical health status at the time of recruitment; refusal or inability to provide informed consent; and incomplete data on the main study variables.

Of the participants, 63 (24%) were undergoing dialysis for chronic kidney disease, 49 (18.6%) had solid tumors, 61 (23.2%) had hematologic diseases, 64 (24.3%) were living with HIV infection, and 26 (9.9%) had tuberculosis. In total, 139 patients (52.9%) presented comorbidities requiring long-term treatment. The daily medication count ranged from 0 to 25 (M = 5.75, SD = 4.79). The ages of the patients ranged from 17 to 92 (M = 53.79, SD = 17.14). The sample included 153 men (58.2%), and 160 participants (60.8%) were from urban areas. Other characteristics of the cohort, such as time since diagnosis and type of treatment, were reported elsewhere (Laza et al, 2024). Descriptive statistics for the main demographic and clinical variables used in the present analyses are summarized in Table 1.

Instruments

Data were collected with an online questionnaire that included socio-demographic, clinical, and psychological measures. Sociodemographic and clinical data referred to age (open-ended question), sex, residential area, main diagnosis, number of medications per day, and comorbidities (dichotomized as presence/absence to reflect a clinically interpretable distinction and to ensure comparability across heterogeneous diagnostic groups).

Adaptation to chronic disease was measured with the Chronic Illness Adjustment Scale (CIAS).[32] The scale has 19 items grouped into five factors: f1 - illness denial (4 items), f2 - illness-compliant behavior (4 items), f3 - strategic positive engagement (4 items), f4 - emotional support (3 items), and f5 - emotional engagement (4 items). CIAS-f2 assesses the patient's consistency in following treatment and care routines, reflecting adherence behaviors. CIAS-f4

Table 1 Descriptive Summary of the Participants

Variable	n	%	M	SD
Clinical factors				
Chronic kidney disease (dialysis)	63	24.0		
Solid neoplasms	49	18.6		
Hematologic diseases	61	23.2		
HIV infection	64	24.3		
Tuberculosis	26	9.9		
Comorbidities present	139	52.9		
Comorbidities absent	124	47.1		
Number of medications/day			5.75	4.79
Sociodemographic factors				
Age (years)			53.79	17.14
Men	153	58.2		
Women	110	41.8		
Residence Urban	160	60.8		
Rural	103	39.2		

Abbreviations: N, number of participants; %, percentage; M, mean; SD, standard deviation.

captures the tendency to seek emotional support by sharing concerns and experiences with significant others, as an adaptive way to mobilize relational resources. Responses were rated from 1 (never) to 4 (always). Higher scores show better adaptation.

Self-efficacy was assessed with the Self-Efficacy to Manage Chronic Disease Scale (SEMCD).[33] It contains six items rated from 1 to 10 (totally confident). Higher values denote stronger perceived ability to manage the condition.

Resilience was measured with the Brief Resilience Scale (BRS), which has six items rated from 1 (strongly disagree) to 5 (strongly agree). Higher scores indicate higher resilience.[34]

Perceived social support was assessed with the Medical Outcomes Study Social Support Survey (MOS-SSS).[35] The instrument has 19 items rated from 1 (never) to 5 (all the time) and covers four types of support: emotional/informational, tangible, affectionate, and positive social interaction. Higher scores indicate higher perceived support. Although individual items were measured on ordinal Likert-type scales, composite scores were treated as approximately continuous variables.

Procedure

The study was approved by the Ethics Commission of Victor Babes Hospital Timisoara (no. 5925/05.07.2024). All ethical rules were respected. Participation was voluntary, patients signed informed consent, and confidentiality was assured. Data were collected in July–August 2024. Patients were invited to take part when they came for their regular medical visits. The study was explained, and those who agreed filled in the online questionnaire. Data were collected anonymously, with no identifying information recorded in the dataset. When assistance was required, the resident doctor provided only technical support (eg, clarifying instructions or reading items aloud), without offering interpretations, feedback, or guidance on responses. Participants were explicitly informed that their answers would not be accessible to clinical staff and would not affect their medical care.

Statistical Analysis

The data were analyzed using JASP (version 0.19).[36] For the mediation models, the bootstrap method with 5000 resamples and 95% confidence intervals was used. Unstandardized regression coefficients (B) with standard errors (SE) and 95% bias-corrected bootstrap confidence intervals are reported. Direct, indirect, and total effects were estimated, and mediation was considered significant when the confidence interval did not include zero. Analyses were first conducted for adaptation to chronic disease (CIAS total). Two subscales, illness-compliant behavior (CIAS-f2) and emotional support (CIAS-f4) were also tested because they showed the most consistent links with clinical factors. The other subscales did not reach significance and are not reported here due to limited reporting space. In Models 3 and 4, resilience (BRS) and social support (MOS-SSS) were also tested as potential mediators. Model fit was evaluated by reporting R^2 for the mediator and outcome equations, and the change in explained variance (ΔR^2) when including the mediator. Statistical significance was set at $P < 0.05$, two-tailed. There were no missing data on the main study variables.

Results

The results are presented for the four tested models. Model 1 examined the mediating role of self-efficacy in the relationship between the presence of comorbidities and adaptation to chronic disease, both at the total score level and for two subscales of adaptation: compliance and emotional support. Model 2 analyzed the number of medications per day as the clinical factor. Models 3 and 4 tested social support and resilience as possible mediators.

Model 1. Comorbidities – Self-Efficacy – Adaptation to Chronic Disease

To test Model 1 and hypotheses H1a–H1d, a mediation analysis was performed. The independent variable was the presence of comorbidities, coded as a dichotomous variable (presence/absence), the mediator was self-efficacy (SEMCD), and the dependent variable was adaptation to chronic disease (CIAS total and CIAS-f2, CIAS-f4). Table 2 presents the results for CIAS total, while results for CIAS-f2 and CIAS-f4 are presented in the text.

The mediation analysis for Model 1 is shown in Table 2. Comorbidities had a significant negative effect on self-efficacy (B = −0.53, SE = 0.12, P < 0.001, 95% CI −0.77 to −0.30). Self-efficacy positively predicted adaptation (CIAS total: B = 0.32, SE = 0.06, P < 0.001, 95% CI 0.20 to 0.45; CIAS-f2: B = 0.24, SE = 0.06, P < 0.001, 95% CI 0.11 to 0.36). For both CIAS total and CIAS-f2, the indirect effects were significant (CIAS total: B = −0.17, 95% CI −0.29 to −0.09; CIAS-f2: B = −0.13, 95% CI −0.23 to −0.06), confirming mediation. The total effect (c) of comorbidities on CIAS total was not significant (P > 0.05), which is consistent with the pattern of complete mediation. Because the direct effects (c′) on CIAS total and CIAS-f2 were not significant (P > 0.05), while the indirect effects were significant, this indicates

Table 2 Mediation Results for Model 1 (Comorbidities – Self-Efficacy – Adaptation to Chronic Disease)

Variable	B	SE	95% CI		t	P
			LLCI	ULCI		
Comorbidities → CIAS total (c′)	0.12	0.12	−0.12	0.35	0.94	0.345
Comorbidities → Self-efficacy (a)	−0.53	0.12	−0.77	−0.30	−4.45	<0.001
Self-efficacy → CIAS total (b)	0.32	0.07	0.20	0.45	5.31	<0.001
	Model fit indices: Mediator model (Self-efficacy ~ Comorbidities): R^2 =0.28, Adj. R^2 =0.27 Outcome model (CIAS total ~ Comorbidities): $R^2(0)$ =0.04 Outcome model (CIAS total ~ Comorbidities + Self-efficacy): $R^2(1)$ =0.32 ΔR^2 =0.28					

Notes: B = unstandardized regression coefficient; SE = standard error; LLCI = lower limit of the 95% confidence interval (CI); ULCI = upper limit of the 95% CI; *t* = t statistic; *P* = p value (two-tailed); R^2 = coefficient of determination; Adj. R^2 = adjusted coefficient of determination; ΔR^2 = change in R^2 between models.; CIAS total = Chronic Illness Adjustment Scale total score. Paths: a = Predictor → Mediator; b = Mediator → Outcome; c′ = Direct effect.The arrow symbol (→) indicates the direction of the tested regression paths in the mediation model.

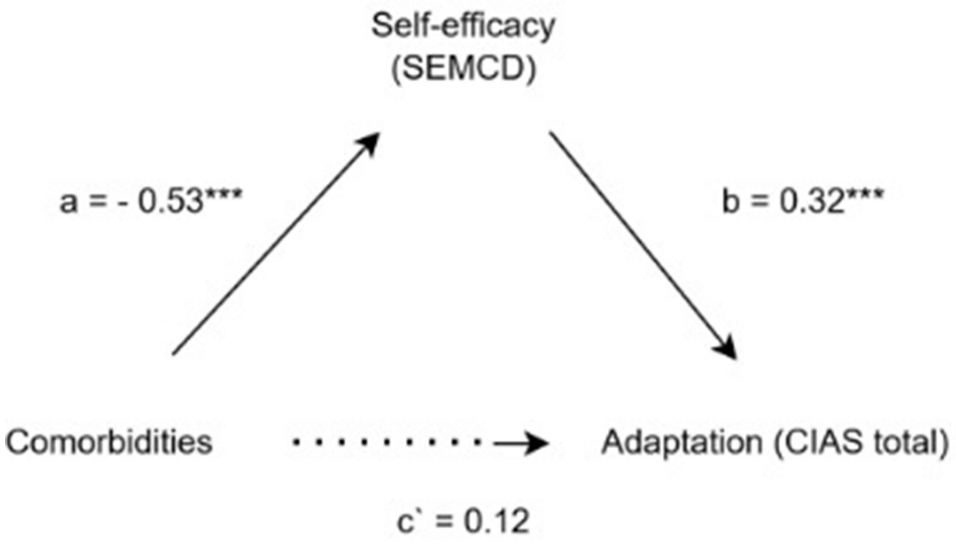

Figure 1 Mediation model of comorbidities and adaptation.
Notes: Values represent unstandardized coefficients (B). Solid lines indicate significant paths (p <0.05; *** p< 0.001).
Abbreviations: CIAS, Chronic Illness Anticipation Scale; SEMCD, Self-Efficacy for Managing Chronic Disease; dashed lines indicate non-significant paths.

complete mediation of the relationship between comorbidities and adaptation through self-efficacy. For CIAS-f4, no mediation was observed, but a direct positive effect of comorbidities on emotional support was found (B = 0.30, SE = 0.13, P = 0.02, 95% CI 0.04 to 0.54). Figure 1 presents the path diagram for Model 1.

The data revealed that comorbidities had a significant negative effect on self-efficacy (path a). Self-efficacy positively predicted adaptation (path b). The indirect effect was significant, which confirms the mediating role of self-efficacy in the relationship between comorbidities and adaptation (CIAS total and CIAS-f2, illness-compliant behavior). This mediation was complete for CIAS total and CIAS-f2, because the direct paths were nonsignificant once self-efficacy was included. For CIAS-f4 (emotional support), the mediation was not significant, but a direct effect of comorbidities on emotional support was observed. This pattern suggests that self-efficacy is relevant for behavioral aspects of adaptation, such as illness-compliant behaviors, whereas emotional support reflects a relation that may be more directly associated with clinical burden rather than mediated by individual self-management beliefs. These findings confirm H1a, H1b, and H1c. However, H1d was not supported, as no mediation emerged for CIAS-f4 (emotional support).

Model 2. Number of Medications – Self-Efficacy – Adaptation to Chronic Disease

To test Model 2 and hypotheses H2a–H2d, a mediation analysis was performed. The independent variable was the number of medications per day, considered an indicator of treatment complexity and clinical burden. The mediator was self-efficacy (SEMCD), and the dependent variable was adaptation to chronic disease (CIAS total, CIAS-f2, CIAS-f4). Table 3 presents the results for CIAS total, while results for CIAS-f2 and CIAS-f4 are presented in the text.

The mediation analysis for Model 2 is shown in Table 3. The number of medications per day had a significant negative effect on self-efficacy (B = −0.07, SE = 0.01, P < 0.001, 95% CI −0.10 to −0.04). Self-efficacy positively predicted adaptation (CIAS total: B = 0.31, SE = 0.06, P < 0.001, 95% CI 0.17 to 0.43; CIAS-f2: B = 0.26, SE = 0.06, P < 0.001, 95% CI 0.13 to 0.39). For both CIAS total and CIAS-f2, the indirect effects were significant (CIAS total: B = −0.02, 95% CI −0.04 to −0.01; CIAS-f2: B = −0.02, 95% CI −0.03 to −0.01), as indicated by bias-corrected bootstrap 95% CIs (5,000 resamples), confirming mediation. The total effect (c) of the number of medications on CIAS total was not significant (P > 0.05), which is consistent with a complete mediation pattern. Because the direct effects (c′) on CIAS total and CIAS-f2 were not significant (P > 0.05), while the indirect effects were significant, this indicates complete mediation of the relationship between the number of medications and adaptation through self-efficacy. For CIAS-f4, no mediation was observed, but a direct positive effect of the number of medications on emotional support was found (B = 0.03, SE = 0.01, P = 0.03, 95% CI 0.00 to 0.06). Although the indirect effects were statistically significant, their

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Table 3 Mediation Results for Model 2 (Number of Medications – Self-Efficacy – Adaptation to Chronic Disease)

Variable	B	SE	95% CI		t	P
			LLCI	ULCI		
Medications → CIAS total (c′)	0.00	0.01	−0.02	0.02	0.04	0.966
Medications → Self-efficacy (a)	−0.07	0.01	−0.10	−0.04	−5.69	<0.001
Self-efficacy → CIAS total (b)	0.31	0.06	0.17	0.43	4.96	<0.001
	Model fit indices: Mediator model (Self-efficacy ~ Medications): R^2 =0.07, Adj. R^2 =0.06 Outcome model (CIAS total ~ Medications): $R^2(0)$ =0.01 Outcome model (CIAS total ~ Medications + Self-efficacy): $R^2(1)$ =0.10 ΔR^2 =0.09					

Notes: B = unstandardized regression coefficient; SE = standard error; LLCI = lower limit of the 95% confidence interval (CI); ULCI = upper limit of the 95% CI; *t* = t statistic; *P* = p value (two-tailed); R^2 = coefficient of determination; Adj. R^2 = adjusted coefficient of determination; ΔR^2 = change in R^2 between models. CIAS total = Chronic Illness Adjustment Scale total score. Paths: *a* = Predictor → Mediator; *b* = Mediator → Outcome; *c′* = direct effect. The arrow symbol (→) indicates the direction of the tested regression paths in the mediation model.

relatively small magnitude suggests that medication burden may influence adaptation through self-efficacy in a modest but clinically meaningful way. Figure 2 presents the path diagram for Model 2.

The results showed that the number of medications per day had a significant negative effect on self-efficacy (path a). Self-efficacy positively predicted adaptation (path b). The indirect effect was significant, which confirms the mediating role of self-efficacy in the relationship between the number of medications and adaptation (CIAS total and CIAS-f2, illness-compliant behavior). This pattern shows that self-efficacy fully mediated the relationship for CIAS total and CIAS-f2, whereas in the case of CIAS-f4, self-efficacy did not act as a significant mediator. For CIAS-f4 (emotional support), the mediation was not significant, but a direct positive effect of the number of medications on emotional support was observed. These findings support H2a, H2b, and H2c. H2d was not confirmed, as self-efficacy did not mediate the relationship with CIAS-f4motional support.

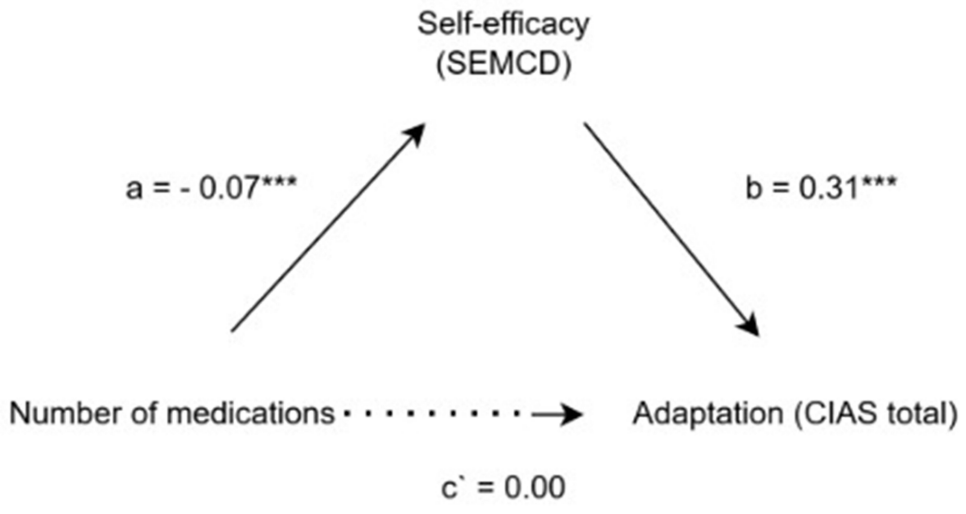

Figure 2 Mediation model of the number of medications and adaptation.
Notes: Values represent unstandardized coefficients (*B*). Solid lines indicate significant paths (p <0.05; *** p< 0.001).
Abbreviations: CIAS, Chronic Illness Anticipation Scale; SEMCD, Self-Efficacy for Managing Chronic Disease; dashed lines indicate non-significant paths.

Models 3 and 4. Social Support and Resilience as Mediators of Adaptation to Chronic Disease

Model 3 with social support (MOS-SSS) was tested using CIAS total as the outcome (Table 4).

For MOS, the effect of the number of medications on perceived social support was not significant (B = −0.01, SE = 0.01, P = 0.66, 95% CI −0.03 to 0.02). However, perceived social support had a strong positive effect on adaptation (B = 0.45, SE = 0.06, P < 0.001, 95% CI 0.35 to 0.54). The total effect (c) of medications on CIAS total was not significant (P > 0.05). The indirect effect was not significant (B = −0.00, 95% CI −0.01 to 0.01), as indicated by bias-corrected bootstrap 95% CIs (5,000 resamples). This indicates that, although social support was positively associated with adaptation, it did not mediate the relationship between treatment complexity and adaptation.

For MOS, the effect of comorbidities on perceived social support was also not significant (B = 0.01, SE = 0.12, P = 0.93, 95% CI −0.23 to 0.26). However, perceived social support had a strong positive effect on adaptation (B = 0.45, SE = 0.06, P < 0.001, 95% CI 0.35 to 0.55). The direct effect of comorbidities on adaptation was not significant (B = −0.06, SE = 0.11, P = 0.58, 95% CI −0.29 to 0.15), and the indirect effect through social support was also not significant (B = 0.01, SE = 0.06, P = 0.93, 95% CI −0.11 to 0.11). This indicates that, although social support was positively associated with adaptation, it did not mediate the relationship between comorbidities and adaptation. Figure 3 presents the path diagram for Model 3.

H3 was not supported, as no mediation was found. However, social support exerted a strong direct positive effect on adaptation.

Model 4 with resilience (BRS) was tested using CIAS total as the outcome (Table 5).

Table 4 Mediation Results for Model 3 (Number of Medications – Social Support – Adaptation to Chronic Disease) and (Comorbidities – Social Support – Adaptation to Chronic Disease)

Path	B	SE	95% CI		t	P
			LLCI	ULCI		
	Predictor: Number of medications per day					
Medications → MOS-SSS (a)	−0.01	0.01	−0.03	0.02	−0.44	0.66
MOS-SSS → Adaptation (CIAS total, b)	0.45	0.06	0.35	0.54	7.50	<0.001
Medications → Adaptation (CIAS total, c′)	0.02	0.06	−0.09	0.13	0.28	0.78
Indirect effect (a×b)	−0.00	-	−0.01	0.01	−0.44	0.66
	Model fit indices: Mediator model (MOS-SSS ~ Medications): R^2 =0.00, Adj. R^2 =0.00 Outcome model (CIAS total ~ Medications): $R^2(0)$ =0.01 Outcome model (CIAS total ~ Medications + MOS-SSS): $R^2(1)$ =0.20 ΔR^2 =0.19					
	Predictor: Comorbidities					
Comorbidities → MOS-SSS (a)	0.01	0.12	−0.23	0.26	0.08	0.93
MOS-SSS → Adaptation (CIAS total, b)	0.45	0.06	0.35	0.55	7.66	<0.001
Comorbidities → Adaptation (CIAS total, c′)	−0.06	0.11	−0.29	0.15	−0.55	0.58
Indirect effect (a×b)	0.01	0.06	−0.11	0.11	0.08	0.93
	Model fit indices (comorbidities): Mediator model R^2 ≈.00; Outcome model R^2 =0.20					

Notes: B = unstandardized regression coefficient; SE = standard error; LLCI = lower limit of the 95% confidence interval (CI); ULCI = upper limit of the 95% CI; *t* = t statistic; *P* = p value (two-tailed); R^2 = coefficient of determination; Adj. R^2 = adjusted coefficient of determination; ΔR^2 = change in R^2 between models. CIAS total = Chronic Illness Adjustment Scale total score. Paths: *a* = Predictor → Mediator; *b* = Mediator → Outcome; *c′* = direct effect. Indirect effect (a × b) represents the indirect effect of the predictor on the outcome through the mediator. MOS-SSS = Medical Outcomes Study Social Support Survey. The arrow symbol (→) indicates the direction of the tested regression paths in the mediation model.

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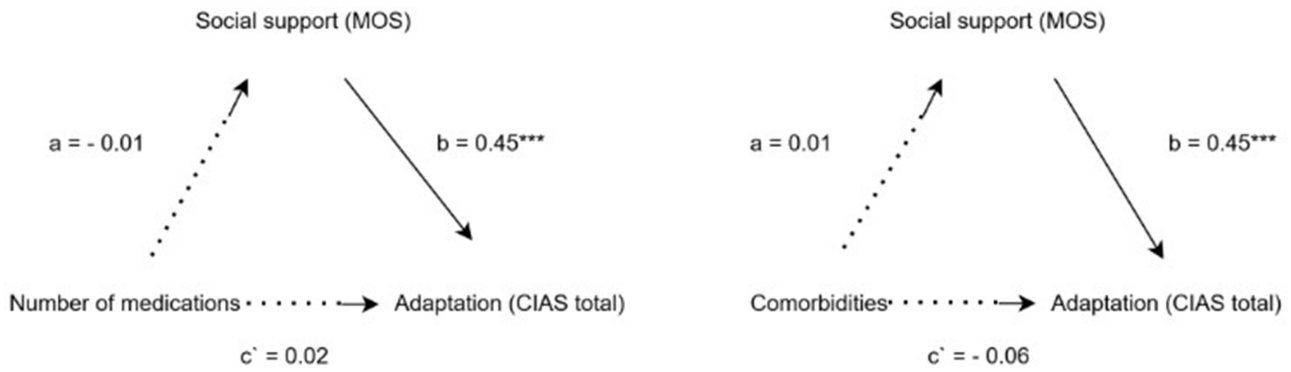

Figure 3 Mediation models of the relationship between the number of medications (left) and comorbidities (right) on adaptation, with social support as a potential mediator.
Notes: Values represent unstandardized coefficients (*B*). Solid lines indicate significant paths (p <0.05; *** p< 0.001), and dashed lines indicate non-significant paths.
Abbreviations: CIAS, Chronic Illness Anticipation Scale; MOS, Medical Outcomes Study-Social Support Survey.

For BRS, the effect of the number of medications on resilience was not significant (B = −0.01, SE = 0.01, P = 0.39, 95% CI −0.03 to 0.01), and the effect of resilience on adaptation was also nonsignificant (B = 0.11, SE = 0.06, P =0 .08, 95% CI −0.02 to 0.24). The total effect (c) of medications on CIAS total was not significant (P > 0.05). The indirect effect was not significant either (B = −0.00, 95% CI −0.01 to 0.00), as indicated by bias-corrected bootstrap 95% CIs

Table 5 Mediation Results for Model 4 (Number of Medications – Resilience – Adaptation to Chronic Disease) and (Comorbidities – Resilience – Adaptation to Chronic Disease)

Path	B	SE	95% CI		t	p
			LLCI	ULCI		
	Predictor: Number of medications per day					
Medications → Resilience (a)	−0.01	0.01	−0.03	0.01	−0.86	0.39
Resilience → Adaptation (CIAS total, b)	0.11	0.06	−0.02	0.24	1.75	0.08
Medications → Adaptation (CIAS total, c′)	0.02	0.06	−0.09	0.13	−1.54	0.13
Indirect effect (a×b)	−0.00	–	−0.01	0.00	−0.77	0.44
	Model fit indices: Mediator model (Resilience ~ Medications): R^2 =0.00, Adj. R^2 =0.00 Outcome model (CIAS total ~ Medications): $R^2(0)$ =0.01 Outcome model (CIAS total ~ Medications + Resilience): $R^2(1)$ =0.02 ΔR^2 =0.01					
	Predictor: Comorbidities					
Comorbidities → BRS (a)	−0.14	0.12	−0.40	0.11	−1.15	0.25
BRS → Adaptation (CIAS total, b)	0.11	0.06	−0.01	0.25	1.79	0.07
Comorbidities → Adaptation (CIAS total, c′)	−0.04	0.12	−0.29	0.19	−0.33	0.74
Indirect effect (a×b)	−0.02	0.02	−0.07	0.01	−0.97	0.33
	Model fit indices (comorbidities): Mediator model R^2 =0.01; Outcome model R^2 =0.013					

Notes: B = unstandardized regression coefficient; SE = standard error; LLCI = lower limit of the 95% confidence interval (CI); ULCI = upper limit of the 95% CI; t = t statistic; P = p value (two-tailed); R^2 = coefficient of determination; Adj. R^2 = adjusted coefficient of determination; ΔR^2 = change in R^2 between models. CIAS total = Chronic Illness Adjustment Scale total score. Paths: a = Predictor → Mediator; b = Mediator → Outcome; c' = direct effect. Indirect effect (a × b) represents the indirect effect of the predictor on the outcome through the mediator. BRS = Brief Resilience Scale. The arrow symbol (→) indicates the direction of the tested regression paths in the mediation model.

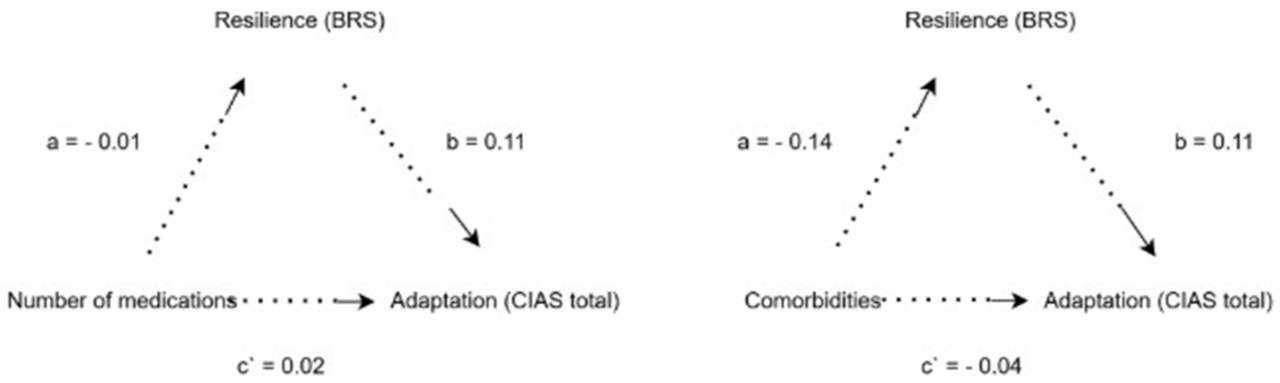

Figure 4 Mediation models of the relationship between the number of medications (left) and comorbidities (right) on adaptation, with resilience as a potential mediator.
Notes: Values represent unstandardized coefficients (*B*). All paths are non-significant ($p > 0.05$), as indicated by the dashed lines.
Abbreviations: BRS, Brief Resilience Scale; CIAS, Chronic Illness Anticipation Scale.

(5,000 resamples). This confirms the absence of a mediating role of resilience in the relationship between treatment complexity and adaptation.

For BRS, the effect of comorbidities on resilience was also not significant (B = −0.14, SE = 0.12, P = 0.25, 95% CI −0.40 to 0.11), and the effect of resilience on adaptation was also nonsignificant (B = 0.11, SE = 0.06, P = 0.07, 95% CI −0.01 to 0.25). The direct effect of comorbidities on adaptation was not significant (B = −0.04, SE = 0.12, P = 0.74, 95% CI −0.29 to 0.19), and the indirect effect through resilience was not significant either (B = −0.02, SE = 0.02, P = 0.33, 95% CI −0.07 to 0.01). These findings indicate that resilience did not mediate the relationship between comorbidities and adaptation. The lack of significant associations for resilience may reflect sample characteristics, such as advanced disease stages and high clinical burden, or the use of a brief, trait-oriented resilience measure that may be less sensitive to context-specific adaptation processes. Figure 4 presents the path diagram for this model.

H4 was not supported, as resilience showed neither a mediating role nor a significant direct effect on adaptation.

In summary, the mediation models were tested separately, and the inclusion of self-efficacy consistently led to an increase in the explained variance of adaptation, highlighting its central role within the proposed framework. Hypotheses H1a–H1c and H2a–H2c were supported, while H1d, H2d, H3, and H4 were not confirmed. The only significant direct effect beyond self-efficacy was found for social support, whereas resilience did not show significant associations with adaptation.

Models 3 and 4 with social support and resilience yielded no significant indirect effects. Adding self-efficacy changed the picture. The explained variance in adaptation increased clearly ($\Delta R^2 = 0.28$ in Model 1; $\Delta R^2 = 0.09$ in Model 2), pointing to a mediation pathway from clinical factors to adaptation. Social support raised the total R^2 to .20, but the path from clinical variables was absent. This suggests a direct contribution rather than a mediating role. Resilience showed no meaningful paths, reinforcing its marginal influence in this sample.

Discussions

This study aimed to examine whether self-efficacy mediates the relationship among clinical factors (comorbidities and number of medications per day) and adaptation to chronic disease. In addition, the study explored the functions of perceived social support and resilience as potential mediators in this process. This work complements a previous study[31] that analyzed which clinical factors predict patients' adaptation to chronic disease. A cross-sectional, correlational design with mediation models was used. Participants were 263 patients from public hospitals for chronic diseases in Romania. Guided by the literature, a transdiagnostic perspective was adopted, including patients with chronic kidney disease, solid tumors, hematologic diseases, HIV infection, and tuberculosis. Simple mediation analyses were conducted to test four models, discussed in the following section.

Model 1. Comorbidities – Self-Efficacy – Adaptation to Chronic Disease

In the first model, mediation analysis tested whether self-efficacy mediated the link between comorbidities and adaptation to chronic disease, assessed both at the overall level (CIAS total) and across specific adaptation dimensions. Adaptation was assessed both as the overall CIAS score and through two subscales: f2 (illness-compliant behavior) and f4 (need for emotional support).

The results supported H1a, showing that the presence of comorbidities was linked with lower self-efficacy, a result consistent with earlier findings.[18] In this study, comorbidity was coded simply as present or absent. Other researchers reached similar conclusions, whether they looked at the number of conditions[37] or at their severity.[38] Both approaches revealed the same pattern: the more conditions a patient has, the lower their self-efficacy. When multiple chronic conditions overlap, symptoms can also combine, treatments can interact, and in this context, treatment regimens become increasingly complicated. The consequence of this is a greater burden, which, over time, erodes patients' confidence.[39]

From a clinical perspective, to counteract this effect, patients should benefit from personalized and repeated counseling that focuses on practical self-management strategies. Useful measures may include simplifying treatment regimens, clarifying conflicting recommendations, and introducing motivational or brief problem-solving interventions.[40,41]

In both Model 1 and Model 2, the second hypothesis (H1b and H2b) targeted the final part of the mediation chain, namely the relationship between self-efficacy and adaptation to chronic illness (CIAS total). The analysis revealed a positive association between the level of self-efficacy and adaptive capacity, in line with previous research.[21,42]

From a theoretical perspective, this finding is consistent with Bandura's[12] self-efficacy theory. Patients with increased self-efficacy are prone to initiate and maintain self-care behaviors, persevere in daily management, and continue efforts even in the face of difficulties. In this sense, self-efficacy also shapes how illness-related demands are appraised and managed, influencing coping responses to ongoing stressors associated with chronic disease. They also tend to cope with symptoms more effectively and reinterpret the demands of the illness in a way that promotes better adaptation.[43–45] Other studies add that self-efficacy supports problem-focused coping,[46] improves adherence to treatment regimens,[47] and is associated with fewer emergency or outpatient visits and better health outcomes.[48]

Taken together, these results suggest that self-efficacy can be considered a key mechanism for improving adaptation to chronic diseases. Jiakpoona[29] emphasized that the management of chronic diseases should go beyond the mere medical treatment itself and that their management could include a broader holistic perspective. From this integrative perspective, psychosocial resources, such as self-efficacy, become essential. Therefore, strengthening patients' confidence in their own capabilities through interventions tailored to their needs may represent a promising strategy for improving adaptation.[16]

Regarding hypotheses H3a and H4a, the results obtained showed that self-efficacy mediates the relationship between comorbidities and adaptation at the dimensional level, focusing on two specific dimensions: compliance behavior (factor 2 of the CIAS) and the need for emotional support (factor 4 of the CIAS). Mediation was observed only for compliance behavior. Comorbidities were associated with lower self-efficacy, a pattern also documented in previous studies.[49]

By contrast, the relationship with the need for emotional support was not mediated by self-efficacy. Instead, a direct association was observed between comorbidities and higher emotional support needs. At an interpretive level, this distinction can be explained by the nature of the construct: self-efficacy is more closely related to behaviors than to emotional responses, which clarifies its role in adherence but not in emotional outcomes.[50] Patients with comorbidities often seek more emotional support regardless of their level of self-efficacy.[51] Instead, comorbidities and polypharmacy showed direct positive effects on patients' tendency to seek emotional support. Because f4 reflects the active mobilization of support as an adaptive strategy, these findings point to an affective–relational pathway triggered by clinical burden, which runs in parallel to the more instrumental pathway captured by self-efficacy. Clinically, patients with greater complexity should be systematically screened for emotional support needs and directed toward the psychological and social resources within the multidisciplinary team.

These findings suggest different directions for intervention. To strengthen compliant behavior, increasing self-efficacy through training programs, health education, or individualized coaching appears effective.[43,52,53] To reduce the emotional impact of comorbidities on adaptation, interventions need to focus on the affective side, not only on strengthening self-efficacy. Measures such as screening for depression or anxiety and participation in support groups are examples of useful strategies.[54]

To capture both overall adaptation and distinctions between its behavioral and emotional dimensions, the results were interpreted at the level of the global CIAS score as well as across the relevant subscales.

In summary, the results for Model 1 are supported by prior research showing that comorbidities can erode self-efficacy and reduce patients' capacity to manage their disease.[19,25] At the same time, the present study reinforces the importance of self-efficacy, confirming that patients with higher confidence in their own abilities are more likely to develop adaptive behaviors, as also reported in recent research.[20,21] Taken together, the results suggest partial and domain-specific mediation: the impact of comorbidities on adaptation is indirectly mediated by self-efficacy in a domain-specific manner, particularly for behavioral adaptation (CIAS-f2), while emotional support needs (CIAS-f4) appear to be shaped more directly by clinical burden.

Model 2. Number of Medications Per Day – Self-Efficacy – Adaptation to Chronic Disease

In the second model, mediation analysis tested whether self-efficacy in managing chronic disease mediates the relationship between number of medications per day and adaptation to chronic disease. Adaptation was considered both as the global CIAS score and through two subscales: f2 (illness-compliant behavior) and f4 (need for emotional support).

For H2a, results supported the expected association: a higher number of number of medications per day was linked with lower levels of self-efficacy. This aligns with findings from earlier research showing that polypharmacy is related to reduced confidence in self-management.[19] A large number of daily medications increases treatment burden and gradually weakens the feeling of control and competence in self-administration, which are central to self-efficacy.[55] Because taking many medications per day constitutes a risk factor for low self-efficacy, efforts should be made to simplify administration schedules together with patients[56] or to use individualized management strategies with monitoring and follow-up.[40]

The second hypothesis, H2b, was already addressed in the first model, and it was again confirmed here: self-efficacy in managing chronic disease was positively related to adaptation.

For H2c and H2d, the analysis tested whether self-efficacy would mediate the link between the number of daily medications and adaptation, with a focus on compliant behavior and the need for emotional support (CIAS factors 2 and 4). As in the previous model, results indicated that mediation occurred only for compliant behavior. A higher number of daily medications was associated with lower self-efficacy, and lower self-efficacy was in turn linked with poorer treatment compliance, a result also supported by other studies.[57,58]

Taking many medications each day creates an increased treatment burden. This burden is not limited to the physical effort of administering pills but also involves cognitive and emotional demands, which undermine the sense of control and competence and make adherence more difficult.[55] In addition, a direct relationship was found between the number of medications and the need for emotional support, with no mediation by self-efficacy. This means H2d was not supported. Self-efficacy refers to the perceived ability to carry out actions, in this case to manage a complex treatment regimen, while the need for emotional support represents a reaction to stress and emotional overload. Administering a large number of medications can reduce self-efficacy by adding complexity to treatment, which then lowers compliance.[58,59] At the same time, polypharmacy can directly heighten the need for emotional support, but this pathway is independent of self-efficacy.[60]

In summary, Model 2 shows that a larger number of daily medications weakens self-efficacy in managing disease. This is consistent with studies reporting that polypharmacy reduces confidence and increases treatment burden when regimens become more complex.[58] Simultaneously, the results confirm the central role of self-efficacy. Mediation was observed for compliant behavior: the number of medications lowered self-efficacy, and reduced self-efficacy in turn

diminished adherence, a mechanism already reported in prior studies on the link between self-efficacy and treatment compliance and the influence of regimen complexity.[61]

For the need for emotional support, the relationship with the number of medications remained direct, with no mediation by self-efficacy. This suggests that this outcome reflects the emotional strain created by complex regimens rather than perceived capability.[60] From a practical perspective, the findings suggest that reviewing and simplifying treatment regimens in collaboration with patients may reduce treatment burden and help maintain self-efficacy, in line with evidence from the literature on collaborative practices in chronic disease management.[62]

Models 3 and 4 – Social Support and Resilience as Mediators

In this study, the analysis of clinical factors, psychosocial processes and adaptation to chronic illness was framed by the TMA-LTC model.[9] According to this perspective, elements such as comorbidities or the number of prescribed medications act as antecedents that influence psychosocial mechanisms, which then shape adjustment outcomes. Building on this view, resilience and perceived social support, described by Livneh[15] as key processes in the adjustment to chronic disease, were examined as possible mediators.

Evidence from earlier research had suggested such effects,[28] and hypotheses H3 and H4 were formulated on that basis. However, the present findings did not confirm a mediating role for either resilience or social support. Even so, perceived social support showed a clear and positive direct effect on adaptation. This aligns with studies that underline the protective contribution of supportive networks, suggesting that psychological adjustment may occur regardless of clinical severity.[63]

In contrast, resilience was not found to be a significant factor, neither when analyzed as a mediator nor when analyzed as a direct predictor. This result differs from many of the results reported in the specialized literature, where resilience is frequently described as a key psychological resource for patients to cope with chronic illnesses.[64] A likely explanation for this incompatibility relates to the way in which the resilience construct was measured. One possible explanation would be the use of a unidimensional scale (BRS) which could have restricted the ability to capture the complexity of resilience. In addition, the clinical heterogeneity of the sample, including different diagnoses and disease trajectories, may represent another factor that limited the detection of consistent associations between resilience and adaptation. To overcome this problem, future work will need more nuanced instruments that are more sensitive to interindividual differences in resilience in different patient groups.

The results obtained thus highlight the specificity of self-efficacy compared to other mediators. Self-efficacy for managing chronic illnesses reflects beliefs and behaviors directly related to the daily management of the illness and is thus closely related to adaptation. Through this proximal positioning, clinical factors can influence adaptation through a clearly defined behavioral pathway. In contrast, another investigated mediator, perceived social support, is more general in nature and mainly reflects resources in the social environment. Although social support is strongly correlated with adaptation, it does not vary with clinical complexity but acts more as a direct facilitator than as a mediating mechanism. Resilience had even weaker links, probably due to the reduced ability of the instrument used to capture the nuances of this construct in a heterogeneous sample. In conclusion, self-efficacy functions as a proximal mechanism, while social support and resilience influence adaptation in an indirect way, functioning as background resources. This distinction points to the presence of two distinct mechanisms: a behavioral pathway mediated by self-efficacy and an emotional pathway that appears to be directly influenced by clinical burden.

The results indicate specific directions for multidisciplinary care. For example, self-efficacy can be strengthened through structured medical care protocols, patient education tailored to individual needs, together with psychological support focused on self-management of the disease. Step-by-step guidance to maintain daily health-related behaviors can be integrated into clinical routines. Personalized educational activities can also develop condition-specific skills and increase confidence in self-management. Earlier introduction of psychological support facilitates the transformation of clinical recommendations into stable self-management habits from the outset. For people with multimorbidity and complex medication regimens, these components can be incorporated into integrated care modalities to enhance adaptation. At the same time, social support can be mobilized through family involvement, peer initiatives, or community services. These provide emotional and relational resources that support behavioral change. Rather than acting as

a substitute, social support complements self-efficacy, and together they form a more resilient framework for long-term adaptation.[65]

The direct positive association between clinical complexity and the tendency to seek emotional support also has clinical implications that should also be considered. Patients with multiple comorbidities or a complex medication regimen may be more likely to need emotional support. This occurs when they are faced with increased psychological demands. Therefore, beyond the management of medical treatment, medical teams should also examine the emotional support needs of these patients. Early identification of emotional needs can allow for timely referral to psychosocial services, such as counseling, support groups, or social work interventions, ensuring that the affective-relational dimension of adaptation to chronic diseases is addressed.

Synthesizing the results, the study showed that self-efficacy mediated the relationship between clinical factors, comorbidity and the number of medications administered daily, and adaptation to chronic diseases. At the same time, direct links were highlighted between these clinical variables, self-efficacy, perceived social support, and the adaptation process. Thus, adaptation is not only determined by the severity of the disease but also by the interaction between internal resources and the support available in the immediate environment. To be effective, interventions could integrate medical management with programs that strengthen personal control and activate social support, resulting in better quality of life and reduced vulnerability.

Limitations

Some aspects of this research may limit the generalization of its findings and should therefore be acknowledged. First, the cross-sectional design does not allow for causal relationships between the investigated variables; it only captures a present reality. The identified associations suggest possible mechanisms, but these should be tested in longitudinal studies that follow the dynamics of adaptation to chronic diseases over time. In this context, longitudinal or repeated-measures studies would allow examination of how self-efficacy and adaptation evolve over time and provide a stronger basis for drawing inferences about directional relationships between variables.

Second, the sample includes patients with various diagnoses recruited from public hospitals in Romania. This transdiagnostic approach provides a broader picture of the psychosocial processes involved but, at the same time, may hide the specific differences of each disease. Consequently, the results cannot be generalized without reservations to other clinical or cultural contexts.

Another limitation concerns the way in which the psychological variables were measured. Resilience, for example, was assessed using a short unidimensional scale (BRS), which may not capture its complexity. At the same time, self-assessments can be influenced by subjective factors, such as mood or the tendency of patients to respond in a socially acceptable way.

The unbalanced distribution of patients according to the type of condition and the severity of the disease may also be a factor that influenced the results. In some subgroups, the small number of participants limits the statistical power of the analysis.

Conclusions

The results obtained show that self-efficacy functions as a central mechanism through which clinical factors, including comorbidities and the number of daily medications administered, influence adaptation to chronic disease, particularly in relation to compliance-related adaptive behaviors. This mediating effect was not observed for emotional-support needs. Patients' perception of their own ability to manage their disease modifies the way in which the complexity of treatment is reflected in coping behaviors. At the same time, the analysis indicated that perceived social support has a direct and consistent effect on adaptation, while resilience did not have a relevant role. This study complements the existing literature because it explicitly tests the mediating role of self-efficacy between clinical factors and adaptation.

Clinical practice and health policy should prioritize theoretically informed strategies that strengthen self-efficacy (eg, goal setting/action plans, feedback, problem-solving, peer support) and self-care education, including hybrid/digital formats.

From an intervention perspective, these data support the need for programs that combine medical management with psychosocial care. The increase in self-efficacy can be stimulated through health education, counseling and training in self-care skills, adapted to each patient. In parallel, strengthening support networks, either by involving the family or by facilitating contact with patient groups, can bring direct benefits in the adaptation process. Such an integrated approach, which takes into account the internal and external resources of the person, has the potential to more effectively support the quality of life of patients with chronic disease.

Data Sharing Statement

Data underlying the findings of this study are accessible from the corresponding author on reasonable request.

Ethics Statement

The research adhered to the ethical principles outlined in the Declaration of Helsinki and received approval from the Ethics Committee of the Victor Babeş Hospital for Infectious Diseases and Pneumophthisiology (approval no. 5925/05.07.2024). All participant data were treated with strict confidentiality, participation was voluntary, and written informed consent was obtained from each individual involved.

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Disclosure

The authors declare no conflicts of interest related to this article.

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