The Determinants of Cancer-Related Fatigue: A Systematic Review

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Abstract: *Cancer-related fatigue (CRF) remains one of the most prevalent and functionally disruptive symptoms in oncology, yet its determinants are still frequently examined in isolation. This systematic review synthesizes recent evidence using an integrated model in which physiological, psychological, and treatment-related determinants shape fatigue severity, with sleep disturbance positioned as a probable mediating pathway and social support as a contextual moderator. A qualitative systematic synthesis aligned with PRISMA 2020 was conducted across PubMed, Scopus, Web of Science, and CINAHL for peer-reviewed studies published between 2021 and 2025. Of 274 records identified, 46 studies met the inclusion criteria and were retained for synthesis. The evidence consistently links inflammatory and metabolic dysregulation, pain burden, physical deconditioning, low activity, anxiety, depressive symptoms, treatment intensity, and advanced disease burden to higher fatigue severity. Recent evidence suggests that these determinants do not operate independently; rather, they accumulate across domains and cluster with poorer sleep. Sleep disturbance emerges as the most consistently implicated proximal pathway through which multidomain burden intensifies daytime fatigue, whereas stronger social support appears to attenuate fatigue escalation by buffering distress and supporting coping. The review contributes a study-by-factor matrix, a theory-informed synthesis, and an integrated conceptual framework for understanding variation in CRF severity across patients and care phases.*

Keywords: cancer-related fatigue, fatigue severity, sleep disturbance, social support, physiological factors, psychological factors, treatment-related factors.

1. Introduction

Cancer-related fatigue is a persistent and distressing form of physical, emotional, and cognitive exhaustion that is disproportionate to recent activity and not fully relieved by rest (Bower et al., 2024). Meta-analytic evidence continues to show that fatigue is among the most prevalent symptom burdens experienced by people with cancer (Al Maqbali et al., 2021). Severity-based estimates further indicate that clinically meaningful fatigue remains common across both active treatment and survivorship settings (Kang et al., 2023). A recent meta-analysis of working cancer survivors further indicates that CRF remains common in populations attempting to sustain everyday functioning and role participation after diagnosis (Matsunaga et al., 2025). Despite the volume of research, the evidence base remains weakly integrated and conceptually heterogeneous (Zdun-Ryżewska et al., 2025). Physiological correlates, psychological distress, symptom clusters, sleep disruption, and treatment burden are still often examined in parallel rather than within a single explanatory structure, which

limits understanding of why patients with comparable disease status can follow markedly different fatigue trajectories (Brownstein et al., 2022).

This review addresses that fragmentation by organizing the recent literature around an integrated conceptual architecture in which physiological, psychological, and treatment-related determinants constitute the primary domains, sleep disturbance functions as a mediating pathway, and social support operates as a moderating contextual condition. This framing is consistent with the Theory of Unpleasant Symptoms, which allows physiological, psychological, and situational influences to interact within a common explanatory structure (Wang et al., 2022). Accordingly, the review aims to synthesize recent evidence, map the determinant structure of cancer-related fatigue, and develop a conceptually coherent framework for explaining variation in fatigue severity.

2. Methodology

This review adopted a qualitative systematic review design and was reported in accordance with PRISMA 2020 to strengthen transparency in study identification, screening, eligibility assessment, and inclusion decisions (Page et al., 2021). A qualitative synthesis was selected because the purpose was explanatory rather than statistical pooling: the review sought to clarify how multiple determinant domains relate to cancer-related fatigue (CRF) severity and how those domains may interact. The review question, screening logic, and data-extraction structure were aligned with the conceptual framework in Figure 2, in which physiological, psychological, and treatment-related factors were treated as the principal determinant domains, sleep disturbance as a mediating pathway, and social support as a moderating contextual condition.

Searches were conducted in PubMed, Scopus, Web of Science, and CINAHL for peer-reviewed English-language studies published between 2021 and 2025, using combinations of terms related to cancer-related fatigue, fatigue severity, determinants, biomarkers, anxiety, depression, sleep disturbance, social support, chemotherapy, radiotherapy, symptom clusters, trajectories, and risk prediction. Supplementary backward and forward citation chaining was used to capture high-value reviews and cohort studies with direct relevance to CRF mechanisms or fatigue trajectories. As summarized in Figure 1, 274 records were identified, 145 records were excluded at title and abstract screening, 129 full-text articles were assessed for eligibility, 83 articles were excluded after full-text review, and 46 studies were retained for synthesis. Studies were included when they examined at least one construct represented in the conceptual framework or otherwise informed CRF pathways in adult oncology populations; intervention studies were retained only when they clarified the mechanistic relevance of sleep, physical activity, distress, or support-related processes for fatigue. Data extraction captured study design, cancer population, determinant coverage, and the reported direction of association with fatigue severity, after which the included studies were synthesized through study-by-factor mapping and thematic comparison.

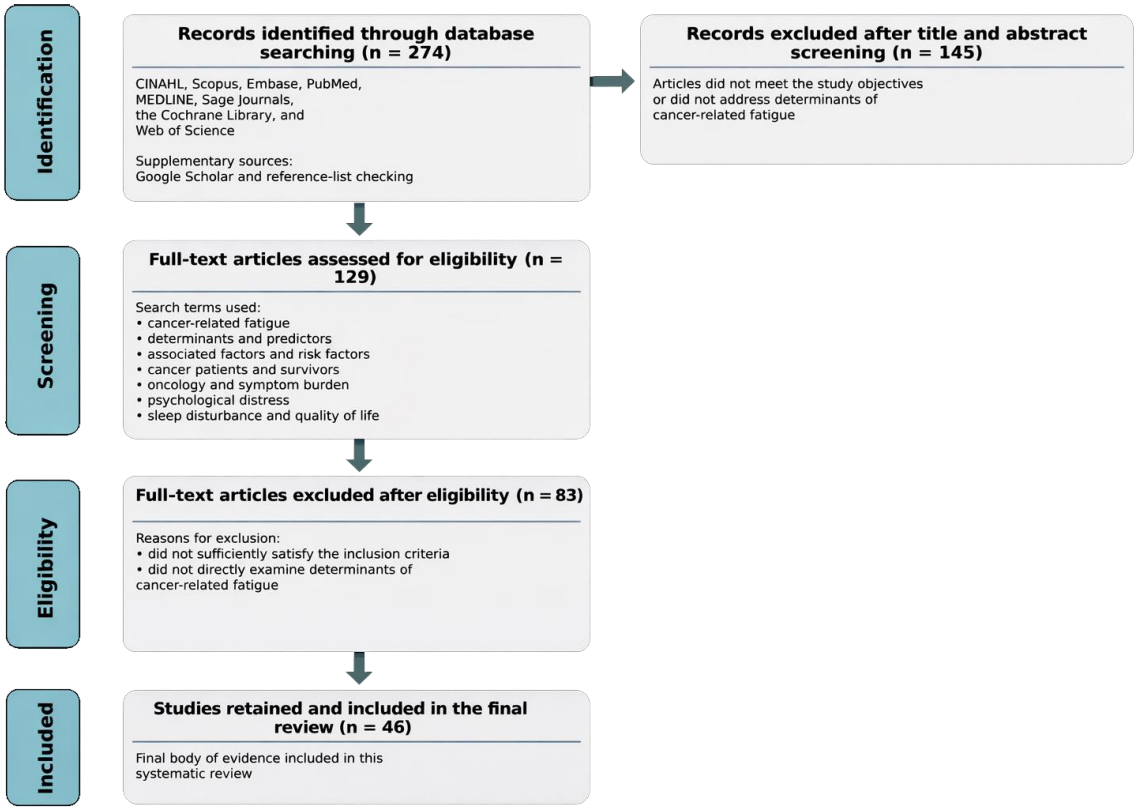

Figure 1: The Systematic Review Process

Table 1: Literature Review Matrix

No	Study	Inflamm.	Metab./hema.	Pain urden	Low activity	Func. decline	Anxiety	Depression	Coping/effic.	Treat. load	Disease stage	Sleep dist.	Support/ isolation
1.	Al Maqbali et al. (2021)									✓	✓		
2.	Bécourt et al. (2025)									✓	✓		✓
3.	Beenhakker et al. (2025)						✓	✓		✓	✓	✓	
4.	Bettariga et al. (2025)				✓	✓							
5.	Blickle et al. (2025)				✓		✓	✓				✓	✓
6.	Bower et al. (2021)						✓	✓		✓		✓	
7.	Bower et al. (2024)				✓							✓	✓
8.	Brownstein et al. (2022)	✓			✓	✓	✓	✓					
9.	Cihan et al. (2025)				✓	✓							
10.	Deng et al. (2024)						✓	✓				✓	
11.	Di Meglio et al. (2022)				✓		✓	✓		✓		✓	
12.	Dun et al. (2022)											✓	
13.	Ebbestad et al. (2023)			✓		✓		✓				✓	
14.	Ernst et al. (2024)				✓	✓							
15.	García-González et al. (2025)	✓	✓			✓							

No	Study	Inflamm.	Metab./hema.	Pain urden	Low activity	Func. decline	Anxiety	Depression	Coping/effic.	Treat. load	Disease stage	Sleep dist.	Support/ isolation
16.	Hao et al. (2024)						✓	✓	✓	✓	✓		
17.	Hou et al. (2024)							✓				✓	
18.	Hsiao et al. (2024)						✓	✓				✓	
19.	Huang et al. (2022)			✓	✓		✓	✓		✓	✓	✓	
20.	Kang et al. (2023)									✓	✓		
21.	Kim et al. (2025)		✓				✓	✓					
22.	Kittel et al. (2025)					✓		✓					✓
23.	Lee et al. (2024)			✓			✓	✓				✓	
24.	Maass et al. (2021)						✓	✓					
25.	Mallard et al. (2025)				✓	✓				✓			
26.	Mao et al. (2025)			✓						✓	✓	✓	
27.	McCloy et al. (2022)							✓				✓	
28.	Momayyezi et al. (2021)											✓	
29.	Pagliuca et al. (2025)				✓			✓		✓		✓	
30.	Richard et al. (2025)			✓			✓	✓				✓	✓
31.	Romero-Elías et al. (2025)			✓	✓	✓					✓		
32.	Rosas et al. (2023)			✓			✓	✓		✓		✓	
33.	Ruiz-Casado et al. (2021)	✓	✓		✓					✓		✓	
34.	Shen et al. (2024)					✓				✓			
35.	Tao et al. (2024)						✓	✓		✓		✓	
36.	Thong et al. (2025)				✓	✓		✓			✓		✓
37.	Vanrusselt et al. (2024)	✓	✓										
38.	Vaz-Luis et al. (2022)			✓				✓		✓		✓	
39.	Wang et al. (2022)			✓			✓	✓	✓	✓		✓	✓
40.	Wang et al. (2024)						✓	✓	✓	✓			
41.	Wu et al. (2025)						✓			✓		✓	✓
42.	Xian et al. (2021)		✓	✓						✓		✓	
43.	Ye et al. (2025)			✓				✓		✓	✓	✓	
44.	Yuan et al. (2024)									✓	✓	✓	
45.	Zdun-Ryżewska et al. (2025)									✓		✓	
46.	Zhang, Zhou, et al. (2025)			✓			✓	✓		✓	✓	✓	

Table 1 shows a stable contemporary pattern in the CRF literature: treatment burden, psychological distress, and sleep disturbance are modeled most often, whereas biomarker-based mechanisms and social-context variables are increasingly acknowledged but remain less consistently integrated within unified analytical frameworks (Doppenberg-Smit et al., 2025).

2.1 Determinants of Cancer-Related Fatigue Severity

2.2 Physiological factors

The physiological literature does not support reducing CRF to a purely subjective complaint attributable only to treatment aversion or generalized distress. Recent biomarker and mechanism reviews instead converge on inflammation, immune dysregulation, autonomic disturbance, endocrine disruption, and metabolic burden as plausible biological substrates of fatigue severity (Vanrusselt et al., 2024). This mechanistic signal is reinforced by survivor-level studies linking higher fatigue to deconditioning, reduced cardiorespiratory reserve, poorer muscle function, and lower physical capacity, while recent exercise-mechanism work also points to sleep quality, strength, and psychological distress as dynamically related to fatigue improvement (Mast et al., 2025). Broader umbrella evidence further indicates that physical activity is linked to more favorable fatigue and functional outcomes across cancer survivorship, which strengthens the interpretation of deconditioning as a clinically relevant contributor rather than a peripheral correlate (Filis et al., 2025). Brownstein et al. (2022) showed that physiological and psychosocial correlates coexist, but fatigue severity also tracks measurable physical compromise. Evidence on muscle strength and fitness further suggests that diminished physical reserve is clinically relevant in oncology populations (Bettariga et al., 2025).

Evidence from breast cancer survivorship has also become more coherent. García-González et al. (2025) identified inflammatory signaling, autonomic dysregulation, and metabolic disturbance as recurrent post-treatment mechanisms, and Ruiz-Casado et al. (2021) reached a similar conclusion in a focused review of breast cancer survivors. Although these studies do not yet establish a stable diagnostic biomarker panel, they provide a credible physiological rationale for persistent fatigue. Recent review work on objective fatigue assessment similarly shows that the field still lacks clinically standardized multimethod indicators that are robust enough for routine stratification or monitoring (Beeler et al., 2025). Observational data further show that fatigue is associated with pain, poorer perceived health, sedentary behavior, and broader symptom burden at or near diagnosis, indicating that biological vulnerability may already be present before treatment burden intensifies (Romero-Elías et al., 2025).

2.3 Psychological factors

Psychological determinants are among the most reproducible predictors of CRF severity, and recent symptom-network work continues to position fatigue near the interface between somatic and psychological burden (Doppenberg-Smit et al., 2025). Anxiety, depressive symptoms, negative illness appraisal, low perceived control, and reduced self-efficacy recur across cancer sites, study designs, and phases of care (Wang et al., 2024). In trajectory and risk-model research, psychological variables frequently distinguish patients with severe or persistent fatigue from those whose fatigue remains transient (Bower et al., 2021). Evidence from colorectal cancer is particularly clear. Wang et al. (2022) showed that psychosocial resources and symptom interpretation matter alongside clinical variables, and Wang et al. (2024) further demonstrated that anxiety, depressive symptoms, coping style, and body image contribute meaningfully to fatigue severity. Meta-analytic evidence likewise indicates that insomnia, anxiety, depression, pain, and lower physical activity tend to operate together rather than as isolated correlates (Huang et al., 2022).

The broader symptom-cluster literature reaches the same conclusion. Hsiao et al. (2024) showed that fatigue in breast cancer survivors is embedded in a wider psychoneurophysiological symptom network, and Richard et al. (2025) demonstrated through network-analysis studies that fatigue rarely operates as a standalone symptom. Models that treat CRF as a single-variable outcome while disregarding distress, sleep, and appraisal are therefore analytically incomplete (Richard et al., 2025).

Recent evidence also extends beyond adult solid tumors. Deng et al. (2024) identified distinct fatigue-psychological symptom profiles among long-term survivors diagnosed in childhood, adolescence, or young adulthood, and Maass et al. (2021) linked persistent fatigue in breast cancer survivors to depressive and anxiety symptoms years after treatment.

2.4 Treatment-related factors

Treatment-related determinants remain central, but the recent literature supports a more nuanced interpretation than a simple exposure-effect model. The strongest contemporary evidence suggests that treatment intensity does not produce uniform fatigue; instead, it interacts with baseline vulnerability, disease burden, and ongoing symptom accumulation (Mallard et al., 2025). In colorectal cancer, meta-analytic evidence shows that chemotherapy exposure, advanced stage, and low physical activity increase fatigue risk, especially when insomnia, pain, anxiety, or depression are already present (Huang et al., 2022). Longitudinal studies are particularly informative because they show how fatigue changes during treatment rather than merely coexisting with treatment exposure. Xian et al. (2021) reported rising fatigue across chemotherapy cycles in colorectal cancer, and Yuan et al. (2024) identified distinct postoperative lung-cancer fatigue trajectories during chemotherapy. Mallard et al. (2025) similarly showed that patients with early breast cancer do not follow a single fatigue course during chemotherapy, reinforcing the need for risk stratification rather than uniform symptom management.

Disease stage also matters because advanced disease increases both physiological strain and treatment intensity. Hao et al. (2024) found that tumor stage, chemotherapy cycle, anxiety, depression, and self-efficacy all contributed to fatigue severity in diffuse large B-cell lymphoma. Ye et al. (2025) and Mao et al. (2025) likewise showed that advanced disease burden and treatment exposure are embedded within fatigue-pain-sleep symptom clusters rather than acting independently. Treatment-related fatigue is therefore better understood as the product of cumulative treatment load acting on patients who differ in biological reserve, symptom burden, emotional distress, and available support (Di Meglio et al., 2022). This interpretation is made explicit in recent risk-model work, particularly in breast cancer survivorship research (Beenhakker et al., 2025).

2.5 Sleep disturbance as a mediating pathway

Sleep disturbance appears to be the most consistently implicated proximal pathway linking multiple determinant domains to fatigue severity (Tao et al., 2024). Poor sleep quality is consistently associated with worse fatigue across cancer populations, but the more important finding is that sleep often sits between upstream burden and downstream functional exhaustion rather than merely co-occurring with both (Momayyezi et al., 2021). Emerging evidence further suggests that maladaptive sleep beliefs and poor sleep hygiene can help explain how this pathway intensifies during chemotherapy, particularly in lung cancer populations (Yan et al., 2025). Longitudinal evidence supports this mediating interpretation. Tao et al. (2024) showed that sleep disturbance, fatigue, and psychological distress co-develop over time in breast cancer, and Rosas et al. (2023) identified insomnia as a key feature of high and persistent fatigue trajectories after radiotherapy. Sleep therefore appears to transmit the combined effects of pain, treatment burden, and emotional distress into daytime fatigue severity.

Intervention evidence indirectly reinforces this interpretation. Sleep-targeted interventions improve fatigue and quality of life, which would be difficult to explain if sleep were merely a passive consequence of fatigue rather than an active mechanism within the pathway (Dun et al., 2022). A recent comprehensive meta-analysis of insomnia interventions in cancer patients and survivors likewise found that CBT-I produced the most consistent improvements in insomnia severity, further

strengthening the clinical relevance of the sleep pathway (Nissen et al., 2024). Exercise-based insomnia interventions also appear promising, although the evidence base remains smaller and more heterogeneous than the CBT-I literature (Drozd et al., 2024). Related work on mindfulness and yoga likewise shows that fatigue tends to improve when sleep and distress improve (Hou et al., 2024). This mediating position is also consistent with symptom-cluster research, in which sleep disturbance is commonly linked to pain, depression, and impaired functioning, making it a credible conduit through which broad symptom burden is translated into exhaustion and poorer daily performance (Ebbestad et al., 2023).

2.6 Social support as a moderating resource

Social support is better conceptualized as a moderating resource than as a background characteristic, because current evidence suggests that social-context variables can alter how distress and symptom burden translate into sustained fatigue (Wang et al., 2022). In studies that model psychosocial pathways directly, support appears to weaken the progression from distress and symptom burden to severe fatigue by improving coping, adherence, and day-to-day recovery capacity (Wang et al., 2022). Wang et al. (2022) found that internal family support was independently associated with lower fatigue among colorectal cancer patients undergoing postoperative adjuvant therapy. Support is therefore not peripheral to CRF severity; it alters the strength of the pathway through which burden escalates.

Recent observational evidence further links social isolation to worse fatigue, poorer physical functioning, and poorer mental health. Kittel et al. (2025) showed that isolation co-occurs with fatigue and functional impairment, underscoring the clinical importance of social context. Thong et al. (2025) similarly linked long-term fatigue in survivors to broader psychosocial and functional disadvantage. Although intervention-based evidence remains comparatively limited and formal moderation models are still underdeveloped, the overall empirical direction is consistent: stronger support tends to attenuate fatigue escalation, whereas isolation removes a potentially important buffer. This gap suggests that support-related variables should be modeled more explicitly rather than treated as background demographics or residual covariates.

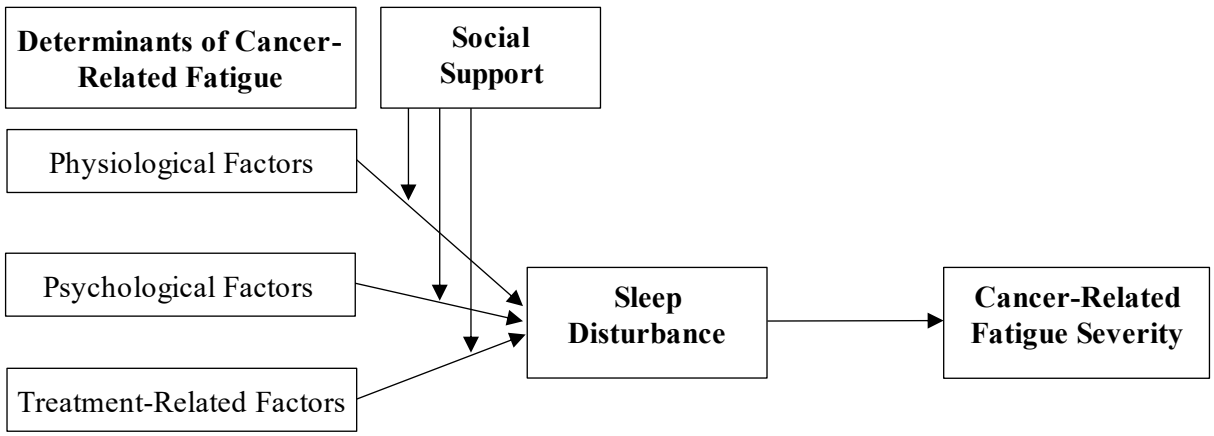

Figure 2: Proposed Conceptual Framework of the Determinants of Cancer-Related Fatigue Severity

3. Discussion

The synthesis supports the framework in Figure 2 as a defensible integrative interpretation of CRF, although the evidence remains stronger for patterned co-occurrence than for definitive causal ordering. Across the included studies, physiological burden, psychological distress, and treatment

load repeatedly clustered with poorer sleep and higher fatigue severity, while social support appeared to weaken fatigue escalation when psychosocial context was modeled directly. Recent network-based syntheses likewise indicate that fatigue often occupies a central bridging position within broader symptom systems rather than behaving as an isolated endpoint (Richard et al., 2025). The framework is therefore best read as a theory-informed explanatory model in which upstream burdens accumulate across domains, sleep disturbance functions as the most plausible proximal pathway linking those burdens to daytime exhaustion, and social-context conditions shape the strength of fatigue escalation. This interpretation is consistent with the Theory of Unpleasant Symptoms and with longitudinal evidence showing that sleep disturbance, psychological distress, and fatigue develop in mutually reinforcing patterns over time (Tao et al., 2024).

The clinical implication is that CRF management should move beyond generic symptom acknowledgment toward structured, multidomain risk stratification (Beenhakker et al., 2025). A patient with moderate fatigue plus insomnia, pain, depressive symptoms, deconditioning, and weak support should not be treated as clinically equivalent to a patient with the same fatigue score but fewer interacting burdens, because the likelihood of persistence and functional deterioration is plausibly different. Assessment should therefore combine fatigue severity with determinant profiling, and supportive care should be matched to the dominant burden pattern: physical rehabilitation when functional decline is prominent, sleep-focused management when insomnia is central, distress-focused care when anxiety or depression dominates, and proactive supportive-care planning when isolation or limited family support is evident. This applied interpretation is consistent with current survivorship guidance, which prioritizes structured assessment of contributing factors and nonpharmacological management for CRF (Bower et al., 2024). Recent narrative synthesis likewise supports aerobic and strength exercise, along with selected integrative oncology approaches, for fatigue reduction in survivorship settings (Ee et al., 2024). Recent comparative evidence further suggests that exercise modality matters, which strengthens the case for tailored rather than uniform fatigue management (Zhou et al., 2025). Resistance training also appears effective for CRF reduction, although dose and population tailoring remain important (Gray et al., 2024). Guided digital psychological interventions may also strengthen distress-focused care, with better fatigue and distress outcomes than unguided formats in the current evidence base (Akdemir et al., 2024). Emerging digital approaches may also help extend fatigue support beyond in-person care, although the evidence remains preliminary and population-specific (Jiang et al., 2025). At the same time, the framework should be interpreted with methodological discipline: much of the literature remains cross-sectional, so the proposed mediating and moderating paths are better understood as a clinically useful synthesis and a research agenda than as conclusive proof of causation.

Table 2: Summary of Key Determinant Domains Influencing Cancer-Related Fatigue Severity

Domain	Representative factors	Interpretative role
Physiological factors	Metabolic burden, pain, low activity, reduced functional reserve	Increase biological vulnerability and reduce recovery capacity
Psychological factors	Depressive symptoms, maladaptive appraisal, low self-efficacy	Intensify symptom appraisal and fatigue persistence
Treatment-related factors	Radiotherapy, cumulative cycles, advanced stage, multimodal burden	Add direct symptom load and interact with patient vulnerability
Sleep disturbance	Poor sleep quality, circadian disruption	Mediates the translation of burden into daytime fatigue severity
Social support	Family support, perceived support, reduced isolation, support-based care	Moderates fatigue escalation by buffering stress and enabling coping

3.1 Research Gaps in the Literature

A major limitation of the literature is temporal design. Many determinant studies remain cross-sectional, yet they discuss mediation, escalation, or pathway effects without establishing temporal order. Claims that sleep mediates fatigue progression, for example, require repeated measurement and stronger longitudinal designs than much of the current literature provides (Tao et al., 2024). A second limitation is measurement heterogeneity. Recent methodological work shows that CRF is still conceptualized differently across diagnostic, mechanistic, and intervention studies, which weakens direct comparison and helps explain why findings do not always accumulate cleanly across cancer sites or treatment phases (Zdun-Ryżewska et al., 2025). This challenge is compounded by the limited integration of objective indicators alongside patient-reported fatigue measures, which constrains multimethod assessment and clinical translation (Beeler et al., 2025).

A third limitation is the early developmental stage of prediction research. Risk-model studies are increasing, but many remain restricted to narrow samples, short follow-up windows, or cancer-specific settings, and only a limited subset is sufficiently mature for routine clinical deployment (Zhang, Li, et al., 2025). A fourth limitation is the underuse of social-context variables within explanatory models, despite evidence that isolation and support meaningfully shape fatigue-related functioning and mental health (Kittel et al., 2025). Social support is often treated as a static background characteristic rather than as a mechanism that can alter how distress, treatment burden, and sleep disruption translate into fatigue severity. A fifth gap concerns intervention delivery: digital mental-health and symptom-management approaches are expanding, but the evidence remains heterogeneous across modalities, outcomes, and cancer populations (Zhong et al., 2025). Finally, biomarker research is promising but not yet decisive. The field has identified plausible mechanisms, yet it still lacks standardized panels that are stable, specific, and practical enough for CRF prediction or monitoring in routine oncology care (Vanrusselt et al., 2024).

4. Conclusion

Cancer-related fatigue severity is best understood as the product of interacting physiological, psychological, and treatment-related determinants rather than as a single downstream side effect of cancer therapy. The recent literature is most informative when it models accumulation and interaction rather than isolating variables (Richard et al., 2025). The integrated framework developed in this review highlights two clinically important points. First, sleep disturbance is best treated as a proximal mediating pathway through which multiple burdens converge into worsening fatigue. Second, social support is not peripheral; it is a moderating resource that can weaken the translation of burden into disabling fatigue. Future research should move beyond fragmented correlational models toward longitudinal, theory-driven designs that explicitly test pathways, trajectories, and interaction effects across cancer types and treatment phases, while also improving multimethod measurement and intervention personalization (Beeler et al., 2025).

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