Table S1. Pharmacological and nutraceutical interventions with reported clinical or mechanistic signals in ME/CFS.

Target: Immune dysregulation and pathogen-related signalling.

Intervention	Therapeutic category	Evidence type	Reported signals	Mechanistic relevance to review model	Limitations / caution
Valacyclovir (1, 2)	Antiviral (anti-herpesvirus; EBV/HSV)	Small prospective and retrospective cohort studies in EBV-positive ME/CFS subsets	Reported improvement in fatigue and functional capacity in selected patients with elevated EBV titres	Supports a model of abortive viral reactivation or persistent antigenic stimulation, rather than active lytic infection	Effects are subset-specific; inconsistent replication; long treatment durations required
Valganciclovir (3)	Antiviral (CMV/HHV-6 active)	Small randomised and open-label studies in HHV-6 / EBV-positive cohorts	Reported improvements in cognitive function and fatigue in selected patients	Relevant to herpesvirus reactivation and immune activation models	Potential toxicity; requires careful monitoring; benefit limited to virologically defined subgroups
Rintatolimod (4, 5)	Immunomodulatory antiviral-response amplifier	Phase 3 randomised controlled trials	Improvement in exercise tolerance	Demonstrates that modulation of antiviral pathways, rather than direct viral suppression, may be more relevant	Included for mechanistic contrast
Intravenous immunoglobulin (IVIG) **(6-13)**	Immunomodulatory therapy (pooled IgG; humoral immune modulation)	Early randomised controlled trials and subsequent heterogeneous clinical studies in ME/CFS	Mixed results: some studies report improvements in fatigue and functional outcomes in subsets; others show no significant benefit	Directly relevant to circulating immune effectors, autoantibody -mediated signalling, and immune-complex biology**;** may modulate Fc receptor activity, complement activation, and B-cell function**;** aligns with emerging models of pathogenic IgG and endothelial/immune interactions	Inconsistent efficacy across studies; likely subgroup-dependent**;** high cost and limited availability; potential adverse effects; not standard of care
Low-dose naltrexone (LDN) **(14-17)**	Opioid receptor antagonist (low-dose; immunomodulatory / neuroimmune regulator)	Mechanistic studies (including ion-channel work), observational reports, ongoing clinical trials	Restoration of TRPM3-like calcium signalling in NK cells; reported improvements in fatigue and pain in some patients	Strong alignment with **T**RPM3 dysfunction, Ca signalling, microglial activation, and immune modulation; links directly to ion-channel and neuroimmune components of the model	Lack of large-scale RCTs in ME/CFS; dosing variability; clinical efficacy not yet fully established
Echinacea (spp. *Purpurea)* **(18)**	Botanical immunomodulator	Mechanistic studies; clinical evidence largely from upper respiratory tract infection (URTI) studies	No robust ME/CFS-specific therapeutic signal	May modulate innate immune activity**,** cytokine production, and macrophage function	Effects are context-dependent**;** may stimulate rather than regulate immune responses; no ME/CFS trials
Rituximab (19)	Anti-CD20 monoclonal antibody (B-cell depletion)	Phase II and phase III randomised controlled trials in ME/CFS	Early studies suggested delayed clinical improvement in subsets, with some patients having transient immune-response but rituximab is not broadly effective for ME/CFS. Larger phase III trial failed to demonstrate significant efficacy overall.	Mechanistically relevant to B-cell dysregulation, autoantibody generation, and maladaptive immune-memory states**;** delayed responses were interpreted as potentially consistent with immune remodelling.	Negative phase III outcome; heterogeneous responses; does not target plasma cells; infection risk and immunosuppression; findings support subgroup heterogeneity rather than universal efficacy

Daratumumab (20)	Anti-CD38 monoclonal antibody (plasma-cell depletion)	Mechanistic and theoretical relevance; An observational clinical trial in ME/CFS (n=10)	Pilot study in 10 ME/CFS patients reported tolerability and clinical improvement in six patients, with transient IgG reduction; Results from the randomised trial required before efficacy can be inferred.	Potential relevance to persistent autoantibody production, plasma-cell survival, and chronic humoral immune activation; conceptually extends humoral immune-targeting beyond rituximab-sensitive B-cell populations.	Highly speculative in ME/CFS; substantial immunosuppressive risk; should be regarded as a hypothesis-generating future-direction intervention only. No published data yet.
Cyclo-phosphamide (21, 22)	Broad immunosuppressive / cytotoxic immunomodulatory agent	Open-label phase II study in ME/CFS	Clinical improvement reported in a subset of patients, (especially those with certain HLA Haplotypes) including fatigue and functional measures.	Supports the possibility that persistent maladaptive immune activation or immune-memory states contribute to disease maintenance; mechanistically relevant to B-cell and T-cell dysregulation within post-infectious immune-remodelling models	No placebo-controlled phase III confirmation; significant toxicity profile (infection risk, infertility, malignancy risk); effects heterogeneous and subgroup-dependent; not suitable as routine therapy
Immuno-adsorption (23-26)	Extracorporeal antibody-removal therapy; humoral immune modulation	Prospective cohort and pilot evidence in post-COVID ME/CFS and autoantibody-enriched ME/CFS/PCC populations; related evidence from plasmapheresis/IVIG studies	Reported reductions in GPCR autoantibodies and symptomatic improvement in subsets, particularly in patients with elevated β2-adrenergic receptor autoantibodies	Directly targets circulating autoantibodies, immune complexes, Fc-mediated signalling, endothelial dysfunction, autonomic receptor dysregulation and vascular control; supports a model in which humoral immune effectors contribute to ME/CFS pathophysiology in selected subgroups	Invasive and resource-intensive; evidence remains preliminary and subgroup-dependent; clinical response does not always correlate cleanly with antibody titre reduction; not established for unselected ME/CFS; requires controlled biomarker-stratified trials
Target: Neuroimmune, ion channel and central signalling.					
Selective serotonin reuptake inhibitors (SSRIs) (e.g. sertraline, fluoxetine, citalopram) **(27-29)**	Antidepressant / serotonergic modulation	Observational use; limited controlled trials in ME/CFS	May improve mood, anxiety, and coping; inconsistent effects on fatigue or core symptoms	Indirect relevance via central neurotransmission, autonomic tone, and neuroimmune signalling	Do not address underlying disease mechanisms; mixed efficacy for fatigue; potential side effects
Serotonin - noradrenaline reuptake inhibitors (e.g. duloxetine) **(30)**	Antidepressant / pain modulation	Extrapolated from fibromyalgia and chronic pain studies	May improve pain and affective symptoms in some patients	Relevant to central sensitisation and pain processing, not core ME/CFS biology	Limited ME/CFS-specific evidence
(low dose) Tricyclic antidepressants (e.g. amitriptyline) **(31, 32)**	Sedative antidepressant / analgesic	Clinical use and small studies	Improvement in sleep and pain reported	Targets sleep disturbance and nociceptive amplification	Anticholinergic side effects; not disease-modifying
Gabapentinoids (e.g. pregabalin, gabapentin) **(33, 34)**	Neuropathic pain modulators	Extrapolated from fibromyalgia and neuropathic pain	May improve pain and sleep in some patients	Relevant to central sensitisation and neuronal excitability	Limited ME/CFS-specific evidence; side effects common

Aripiprazole (35)	Partial dopamine D2 receptor agonist / atypical antipsychotic (low-dose use)	Retrospective cohort studies in ME/CFS patients	Reported improvements in fatigue, cognitive function, and sleep in subsets	Potential relevance to central dopaminergic signalling, neuroinflammation, and microglial modulation	Evidence limited to observational data; risk of side effects; mechanism not fully defined
Target: Autonomic, vascular and haemodynamic dysfunction.					
Pyridostigmine (36-39)	Acetylcholinesterase inhibitor (cholinergic/ autonomic modulation)	Randomised, double-blind, placebo-controlled acute physiological study (invasive CPET paradigm)	Improved peak VO_2 and cardiac output during exercise in ME/CFS patients; effect mediated via enhanced preload and autonomic regulation	Directly supports neurovascular and autonomic contributions to exertional intolerance**;** aligns with **s**tate-dependent endothelial and circulatory dysfunction described in the manuscript	Acute study design; not evidence of sustained clinical benefit; subgroup responsiveness likely
β-blockers - Propranolol, Bisoprolol, Atenolol (39-41)	β-adrenergic receptor antagonists (autonomic modulation)	Observational studies; extrapolated evidence from POTS and dysautonomia cohorts	Reduction in tachycardia, palpitations, and orthostatic symptoms in selected patients	Supports autonomic dysregulation and β-adrenergic signalling abnormalities**,** consistent with impaired cardiovascular control and neurovascular instability	May exacerbate fatigue, exercise intolerance, and hypotension; effects are symptom-modulating rather than disease-modifying; response is highly subgroup-dependent
Midodrine (42, 43)	α1-adrenergic agonist (peripheral vasoconstrictor)	Observational studies; evidence from orthostatic intolerance and POTS cohorts	Improvement in orthostatic tolerance, dizziness, and fatigue in patients with low blood pressure or impaired vasoconstriction	Directly supports vascular dysregulation, impaired peripheral resistance, and reduced venous return**,** contributing to state-dependent circulatory insufficiency	Risk of supine hypertension; benefit limited to patients with demonstrable orthostatic dysfunction; does not address upstream pathology
Fludrocortisone (44-47)	Mineralocorticoid (plasma volume expansion)	Observational studies; extrapolated from POTS and orthostatic hypotension management	Improvement in orthostatic symptoms, fatigue, and exercise tolerance in selected patients	Supports a model of reduced circulating blood volume and impaired preload**,** contributing to exercise intolerance and autonomic dysfunction	Risk of fluid retention, hypokalaemia, hypertension; variable efficacy; requires monitoring; not disease-modifying
Target: Mast-cell, histamine and inflammatory mediator pathways.					
H1 & H2 and dual blockade antihistamines (e.g. cetirizine, loratadine, famotidine) (48-52)	Histamine H1 receptor antagonists (OTC) Histamine H2 receptor antagonists	Observational studies, case series cohorts with ME/CFS. A single randomized double-blind trial in Long-COVID is informative	Improvements in fatigue, cognitive symptoms, and orthostatic intolerance reported in subsets Reported symptom improvement when combined with H1 blockade	Supports a mechanistic role for mast-cell activation, histamine signalling, and neurovascular dysregulation being present in the disease.	Effects not yet validated in controlled ME/CFS trials
Ketotifen (53)	Mast-cell stabiliser + H1 antagonist	Small studies, off-label use	Reported improvements in fatigue, sleep and allergic-type symptoms	Directly targets mast-cell degranulation and mediator release	Sedation; limited high-quality ME/CFS-specific data

Montelukast (54)	Leukotriene receptor antagonist; Mast-cell / inflammatory mediator modulator	Specialist clinical use in ME/CFS; provides indirect mechanistic evidence from macrophage and inflammatory disease models	Reported symptomatic benefit in selected patients with allergy /inflammation or MCAS-like features; no definitive ME/CFS RCT evidence.	Blocks CysLT1R signalling and may reduce leukotriene-mediated mast-cell, endothelial and macrophage activation; experimental evidence indicates suppression of NLRP3/caspase-1-dependent IL-1β and IL-18 release, supporting possible flare-modulating effects.	Evidence in ME/CFS remains indirect and subgroup-dependent; neuropsychiatric adverse effects require caution; should be considered adjunctive and symptom-/flare-modulating, not disease-modifying
Quercetin (55)	Antioxidant / mast-cell stabiliser / senolytic candidate	Mechanistic evidence only	No robust ME/CFS clinical trials	Relevant to mast-cell activation, oxidative stress, and inflammageing	Should be classified as speculative
Curcumin (56, 57)	Anti-inflammatory / NF-κB modulation / mast-cell stabilisation	Mechanistic and preclinical evidence	Anti-inflammatory and anti-oxidant effects; no definitive ME/CFS clinical trials	Maps to chronic immune activation, mast-cell signalling, and cytokine regulation	Poor bioavailability; clinical relevance uncertain
Target: Mitochondrial, metabolic and redox dysfunction.					
Coenzyme Q10 (± NADH) (58-64)	Mitochondrial electron transport / redox	Randomised, double-blind, placebo-controlled trials	Improvements in fatigue scores, sleep quality and QoL	Direct support for mitochondrial dysfunction and redox imbalance	Symptomatic benefit; not disease-modifying
NAD^+ precursors (e.g. nicotinamide riboside, NMN) **(62)**	Mitochondrial metabolism / sirtuin signalling	Mechanistic + indirect clinical evidence (no RCTs)	Improvements in fatigue in other conditions; limited ME/CFS-specific data	Aligns with impaired oxidative phosphorylation, redox state, and metabolic flexibility	Evidence extrapolated; dose and bioavailability uncertain
Liposomal glutathione (65-69)	Glutathione replacement with enhanced bioavailability.	Mechanistic evidence; limited clinical data in ME/CFS	Anecdotal and small-cohort reports of improved fatigue and cognitive clarity	Directly relevant to oxidative stress, mitochondrial redox balance, and immune-cell function	Lack of ME/CFS RCTs; variability in formulation and absorption; clinical efficacy not established
Hydroxo-Cobalamin (Vitamin B12) **(70-73)**	Methylation / nitric oxide scavenging / neuroimmune support	Observational studies and small uncontrolled cohorts	Reported improvement in fatigue and neurological symptoms in subsets	Relevant to NO signalling, autonomic dysfunction, and methylation pathways	Heterogeneous response; often combined with folate; limited controlled data

Methylfolate (5-MTHF) (74, 75)	One-carbon metabolism / epigenetic support	Mechanistic + adjunct clinical use	Used alongside B12; reported symptomatic benefit in some cohorts	Relevant to epigenetic regulation and immune-cell transcriptional programming	No robust standalone ME/CFS trials
N-acetylcysteine (NAC) (57, 76)	Glutathione precursor / redox modulation	Mechanistic + limited clinical evidence	Supports antioxidant capacity; limited direct ME/CFS data	Relevant to oxidative stress and mitochondrial redox imbalance	Clinical efficacy not established
Magnesium (77, 78)	Cellular energy metabolism / neuromuscular stability	Small clinical studies	Some reports of improved energy and muscle function	May support ATP utilisation and neuromuscular signalling	Non-specific; low-quality evidence
Acetyl-L-carnitine / L-carnitine (79)	Fatty acid transport into mitochondria	Small trials and observational studies	Mixed results; some reports of improved fatigue	Relevant to lipid metabolism abnormalities & mitochondrial substrate utilisation	Inconsistent findings across studies
Inspiritol (multi-component formulation) (80)	Nutraceutical combination targeting mitochondrial, redox, and metabolic pathways	Mechanistic rationale based on constituent components; expert observational reports suggesting immunological effects (e.g. T-cell function)	Anecdotal reports of improved fatigue and cognitive symptoms; reported modulation of T-cell responses in informal or observational settings	Integrates mitochondrial bioenergetics, redox balance, and substrate metabolism; potential indirect effects on T-cell activation, differentiation, and immune signalling via metabolic reprogramming	Evidence of safety and efficacy is preliminary. No RCTs yet in ME/CFS.
Alpha-lipoic acid (ALA) (64)	Mitochondrial cofactor / antioxidant	Limited clinical and mechanistic evidence	Reported fatigue reduction in other metabolic disorders	Supports redox balance and mitochondrial enzyme function	No high-quality ME/CFS-specific trials
D-ribose (81)	ATP substrate support	Small uncontrolled studies	Reported improvements in energy in small cohorts	Directly targets impaired ATP regeneration	Weak evidence base
Target: Lipid signalling and membrane biology.					
Evening primrose oil (source of γ-linolenic acid) **(82)**	Lipid-modulating nutraceutical	Limited clinical studies (mainly outside ME/CFS); mechanistic evidence	No consistent ME/CFS-specific benefit demonstrated	Relevant to lipid signalling**,** membrane composition, and inflammatory mediator balance (eicosanoid pathways)	Weak evidence base; indirect relevance; effects modest and inconsistent

Omega-3 fatty acids (83, 84)	Lipid signalling / membrane composition	Mechanistic + indirect clinical evidence	Anti-inflammatory effects; limited ME/CFS-specific data	Aligns with lipid raft biology and inflammatory signalling pathways	Evidence extrapolated from other conditions
Target: Systemic metabolic control and Endocrine - Immune coupling.					
Metformin (85-88)	AMPK-activating metabolic and immunometabolic modulator	Strong mechanistic literature; indirect relevance from Long COVID and inflammatory disease studies; limited direct ME/CFS evidence	Potential improvements in inflammatory tone, metabolic regulation and post-infectious symptom burden suggested in related conditions	Modulates AMPK/mTOR signalling, mitochondrial stress responses, insulin sensitivity, immune-cell metabolism, and NLRP3 inflammasome activation; aligns closely with models of immune–metabolic dysregulation and impaired physiological adaptability.	No direct ME/CFS trial evidence exists. Gastrointestinal side effects common; may worsen symptoms in patients with severe energy limitation or low BMI; not established as disease-modifying therapy
GLP-1 receptor agonists (semaglutide, liraglutide, tirzepatide) (89, 90)	Incretin-based immune-metabolic and endocrine modulators	Extensive mechanistic and cardiovascular/ metabolic literature; No direct ME/CFS evidence has been published	Potential improvements in inflammatory tone, endothelial function, metabolic flexibility and neuroimmune regulation suggested in related post-inflammatory conditions	Modulate immune –metabolic signalling, mitochondrial function, endothelial biology, autonomic regulation, oxidative stress, and NLRP3 inflammasome activation; Strongly aligned with models of impaired physiological adaptability and systemic metabolic dysregulation	Direct ME/CFS efficacy evidence is minimal; Potential confounding by weight loss and metabolic improvement; Gastrointestinal adverse effects common; should be regarded as exploratory rather than an established therapy.
Selenium + CoQ10 (91, 92)	Antioxidant / mitochondrial–endocrine redox support	Clinical supplementation study	Modest improvement in fatigue and quality of life reported	Relevant to mitochondrial redox balance, thyroid-related selenium biology, antioxidant defence and immune regulation	Combination therapy; effect cannot be attributed to selenium alone
Oxaloacetate (93, 94)	Tricarboxylic-acid-cycle intermediate / metabolic modulator	Non-randomised controlled trial; subsequent randomised controlled trial and follow-on analyses	Reported reduction in fatigue, with additional signals for cognitive function and upright activity in subgroups	Directly relevant to impaired metabolic flexibility, anaplerotic support, redox balance, and energy-substrate handling	Commercial supplement; dose-dependent effects; replication and subgroup definition still needed
Creatine (95)	Cellular energy-buffering substrate / phosphocreatine support	Small feasibility study using magnetic resonance spectroscopy	Increased brain creatine levels and reported improvement in fatigue and some cognitive outcomes	Supports impaired energy buffering in brain and muscle; relevant to ATP regeneration and neuroenergetic resilience	Small, non-definitive study; requires placebo-controlled replication
Melatonin + zinc (96)	Circadian–endocrine and antioxidant/immune support	16-week randomised, double-blind, placebo-controlled trial	Reduced physical fatigue and improved physical functioning scores compared with placebo	Links circadian signalling, antioxidant buffering, immune regulation and endocrine–metabolic control	Effect of zinc alone is uncertain; melatonin evidence is mixed; may worsen nocturnal hypotension in some patients

Vitamin D (replacement, where deficient) **(97, 98)**	Secosteroid hormone / immune-endocrine modulator	Mixed evidence; negative high-dose intermittent ME/CFS trial; possible benefit when correcting deficiency	High-dose intermittent vitamin D did not improve fatigue or vascular function in CFS/ME, but replacement may be rational in documented deficiency	Relevant to immune regulation, endothelial biology and endocrine–immune signalling	Should be framed as deficiency correction, not ME/CFS treatment
Probiotics / prebiotics / synbiotics (99-108)	Gut–immune–metabolic axis modulation	Pilot studies; mixed ME/CFS evidence; stronger adjacent post-COVID evidence	Reported improvements in mood, fatigue, sleep, cognition and activity tolerance in some studies	Directly relevant to gut barrier function, microbial metabolites, immune tone, mucosal immunity and systemic inflammatory signalling	Heterogeneous formulations; mixed results; not yet standardisable
Red ginseng (109, 110)	Botanical adaptogen / immunometabolic modulator	Open-label pilot and preclinical fatigue models	Reported improvements in energy, stamina, well-being and cognitive clarity in a mixed ME/CFS/post-viral/fibromyalgia cohort	Potential relevance to HPA-axis modulation, mitochondrial function, immune tone and metabolic resilience	Open-label data; formulation-dependent; insufficient controlled ME/CFS evidence
Vitamin C (ascorbic acid) **(31, 111)**	Antioxidant / redox modulator / cofactor in enzymatic reactions	Observational and mechanistic studies; limited direct ME/CFS clinical trial evidence	Reported modest improvements in fatigue and oxidative stress markers in some cohorts; widely used as adjunctive support	Relevant to oxidative stress buffering, mitochondrial protection, endothelial function, and immune-cell activity; supports redox–immune interface and stress-response pathways	Lack of robust ME/CFS-specific RCTs; effects modest and non-specific; high-dose intravenous use not established in ME/CFS; primarily supportive rather than disease-modifying
Target: Downstream symptom-modulating therapies.					
Ibuprofen	Non-steroidal anti-inflammatory drug (NSAID; COX inhibitor)	General clinical use; no ME/CFS-specific trials	Partial relief of musculoskeletal pain and headache in some patients	Targets prostaglandin-mediated pain and inflammation, downstream of immune activation	Does not address core immune–metabolic–vascular pathology; gastrointestinal and renal risks with prolonged use
Paracetamol (acetaminophen)	Central analgesic / antipyretic	General clinical use	Relief of mild-to-moderate pain and headache	Acts centrally on pain perception pathways; minimal anti-inflammatory effect	Symptomatic only; no disease-modifying effect
Aspirin	NSAID / antiplatelet Prostaglandin pathway modulation	Mechanistic rationale + limited observational use	Possible benefit in vascular or microthrombotic symptoms in subsets	Links to platelet activation, endothelial function, and inflammatory signalling	Bleeding risk; not mast Cell - specific. No ME/CFS evidence
Opioid analgesics (e.g. codeine, tramadol)	Opioid receptor agonists	Clinical use in chronic pain	Pain reduction in selected patients	Modulates central pain pathways; indirect relevance to neuroimmune signalling	Risk of dependence, tolerance, and symptom masking; generally discouraged for chronic use

Zopiclone (112)	Non-benzodiazepine hypnotic (Z-drug)	Clinical use; no disease-specific trials	Improves sleep initiation and maintenance	Addresses sleep dysregulation, which is a major symptom domain	Risk of tolerance, dependence, cognitive side effects; does not restore physiological sleep architecture
Fluconazole / systemic antifungals (113)	Antifungal	Anecdotal and observational use	No consistent evidence of benefit	Hypothesised relevance via gut dysbiosis or fungal overgrowth, not core ME/CFS biology	Evidence remains speculative and indirect

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