Journal of Current Medical Research and Opinion
CMRO 09 (09) 4861-4873 (2026)
https://cmro.in/index.php/jcmro/article/view/1204/1133
www.cmro.in
CMRO
ISSN (O) 2589-8779
(P) 2589-8760

Original Research

Clinical Evaluation of a Broad-Spectrum Therapeutic Method across Multiple Disease Conditions

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Abstract

Background

Broad-spectrum therapeutics are increasingly explored as interventions that can work on multiple diseases, multiple classes of pathogens, or multiple organ systems through a shared biological mechanism, as opposed to intervening on a specific abnormality associated with only one disease. Examples are antimicrobial agents that are active against multiple pathogens, host-directed drug-repurposing candidates like niclosamide, chemical chaperones like 4-phenylbutyrate, and mechanistic target of rapamycin inhibitors targeting pathways involved in multiple age-related disorders[1-5]. Another hypothesis suggests that gravitational or electromagnetic environments will be manipulated with therapeutic results in a variety of disease conditions. However, the breadth of a biological mechanism does not establish clinical efficacy across diseases, and such claims require separate assessment of physical plausibility, dosimetry, safety, and disease-specific clinical outcomes[1,2].

Methods

A rapid evidence synthesis was undertaken using five supplied peer-reviewed articles as seed literature, backward citation searching of their references, and targeted searches of PubMed, regulatory sources, and physical measurement standards. Priority was given to randomised controlled trials, prospective human studies, clinical pharmacology investigations, and authoritative regulatory or metrological sources. Evidence concerning artificial gravity, electromagnetic interventions, niclosamide, 4-phenylbutyrate, mTOR inhibition, drug repurposing, and broad-spectrum antimicrobial treatment was critically compared. No pooled meta-analysis was undertaken because the interventions, exposures, diseases, and outcomes were substantially heterogeneous.

Results

The available evidence supports the existence of genuinely broad or multi-indication therapeutic mechanisms but does not support a universal treatment. The proposed gravitational exposure requires physical redefinition because N m^2 kg^{-2} describes the dimensions of the gravitational constant rather than gravitational acceleration[6,7]. Human artificial-gravity studies demonstrate partial protection against selected consequences of prolonged unloading, including some musculoskeletal, orthostatic, metabolic, and skeletal effects, but results are endpoint-specific and do not demonstrate treatment of unrelated diseases [8,14].

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Received: 11.06.2026 | **Revised:** 04.07.2026 | **Accepted:** 28.08.2026 | **Published:** 05-09.2026

Electromagnetic interventions provide stronger proof that physical fields can have therapeutic effects, as demonstrated by transcranial magnetic stimulation and tumour-treating fields, while pulsed electromagnetic-field studies in musculoskeletal disease have yielded heterogeneous results[15-19]. Pharmacological examples similarly show that broad mechanistic activity does not ensure broad clinical efficacy. Niclosamide has extensive in-vitro activity but inconsistent human efficacy and formulation-dependent pharmacokinetics[3,20-25]. Sodium phenylbutyrate has established metabolic indications and experimental activity across several disease mechanisms, although positive early ALS findings were not confirmed in a subsequent phase 3 programme[5,26-36]. mTOR inhibition has clinically validated effects in selected diseases but exhibits dose-, disease-, and patient-dependent efficacy and toxicity[4,37-40].

Conclusion

The concept of a broad-spectrum therapeutic method is scientifically credible when it denotes intervention acting on a validated mechanism shared by several diseases. Current evidence does not establish altered gravitational or electromagnetic exposure as a universal disease treatment. A field-based intervention should therefore progress through quantitative exposure definition, engineering validation, mechanistic testing, phase I safety evaluation, and separate sham-controlled trials in biologically justified disease populations before any broad-spectrum clinical claim is made.

Keywords: Broad-Spectrum Therapeutics; Electromagnetic Fields; Artificial Gravity; Drug Repurposing; Niclosamide; Phenylbutyrate; Mtor; Clinical Translation; Physical Medicine; Multi-Disease Therapy

1. Introduction

Although clinical medicine is largely practiced around the concept of disease-specific diagnosis and treatment, there are many common biological processes that underpin many diseases such as inflammation, deregulated protein homeostasis, mitochondrial dysfunction, cellular senescence, deregulated immune signalling, oxidative stress, and deregulated metabolic regulation[4,5,34-38]. The convergence creates a legitimate biological argument to consider interventions that might have more than one effect for more than one diagnosis. Walters and Cox, for example, reviewed mTOR inhibition across cancer, neurodegeneration, immunosenescence, osteoporosis, rheumatoid arthritis, diabetic nephropathy and cardiovascular disease, while emphasising that efficacy and adverse effects differ between indications[4]. Bobat et al. similarly examined 4-phenylbutyrate across 13 diseases involving six organ systems because endoplasmic-reticulum stress, protein misfolding and inflammatory responses recur across those disorders [5].

In infectious disease, Firth and Prathapan proposed the term broad-spectrum therapeutic for antimicrobials active against multiple pathogen types and argued that repurposing could identify agents with clinically meaningful activity beyond their original indication[1]. Their framework is important because it distinguishes *biological breadth* from *proven clinical breadth*. Their drug-repositioning evidence-level system progresses from no experimental evidence through in-vitro and animal findings to incomplete human evidence and, finally, well-documented clinical endpoints at safe doses[1]. This distinction is especially relevant to interventions described as potential "panaceas," because mechanistic plausibility alone cannot demonstrate therapeutic benefit.

The field-based hypothesis considered in this paper proposes that modification of gravitational or electromagnetic exposure may correct disturbances believed to contribute to congenital or acquired disease. The initial formulation proposes treatment within a cylindrical environment and specifies values of 2.3–4.27 N m^2 kg^{-2} while attributing recovery to correction of atomic or molecular abnormalities involving hydrogen[6-7]. The hypothesis is potentially testable,

but its original formulation does not yet contain clinical observations, control subjects, validated disease-specific outcomes, quantitative electromagnetic parameters, including magnetic flux density (T), or an experimentally demonstrated molecular mechanism. Furthermore, the assertion that treatment cannot produce adverse effects is not scientifically sustainable before systematic safety evaluation.

The purpose of this study was therefore not to presume therapeutic efficacy but to determine whether the broader concept is compatible with existing clinical science. Four questions were examined: whether the proposed physical exposure can be quantitatively defined, whether gravitational or electromagnetic manipulation has demonstrated human therapeutic effects, what established broad-spectrum therapeutics reveal about translation from mechanism to clinical benefit, and what experimental pathway would be required to test a multi-disease field-based intervention responsibly.

2. Methods

A rapid evidence-synthesis approach was used because the research question crosses clinical pharmacology, physical medicine, space physiology, infectious disease, geroscience and device-based therapy. Five supplied publications were used as seed articles: Firth and Prathapan on broad-spectrum therapeutics[1], Siva et al. on the transition from broad-spectrum to precision antimicrobials[2], Xu et al. on niclosamide[3], Walters and Cox on mTOR inhibition[4], and Bobat et al. on 4-phenylbutyrate[5]. The references contained in these publications were examined to identify relevant mechanistic and clinical studies.

Targeted literature searching prioritised human randomised controlled trials, prospective clinical studies and regulatory evidence. Searches covered artificial gravity and prolonged bed rest, transcranial magnetic stimulation, tumour-treating fields, pulsed electromagnetic fields, niclosamide, phenylbutyrate, mTOR inhibition, antibiotic breadth and drug repurposing. Physical quantities were checked against National Institute of Standards and Technology reference values[6,7]. Evidence was interpreted hierarchically, with replicated randomised clinical evidence weighted more heavily than uncontrolled human observations, animal experiments or in-vitro findings. This approach parallels the DREL framework proposed for broad-spectrum therapeutic classification[1].

This study is therefore an evidence appraisal rather than a systematic review or meta-analysis. Formal pooled effect estimates were considered inappropriate because “broad-spectrum therapy” encompasses fundamentally different interventions and outcomes. The term clinical evaluation in the title refers to evaluation of available clinical evidence and translational requirements and should not be interpreted as reporting a newly conducted clinical trial.

3. Physical Definition and Biological Plausibility of a Field-Based Intervention

A reproducible clinical intervention must first be expressed using physically valid exposure variables. The Newtonian gravitational constant (G) has the numerical value 6.67430×10^{-11} m^3 kg^{-1} s^{-2}, which is dimensionally equivalent to N m^2 kg^{-2} 6[6]. Standard gravitational acceleration at the Earth's surface is defined as 9.80665 m/s^2 [7]. Consequently, the proposed therapeutic range of 2.3–4.27 N m^2 kg^{-2} cannot presently be interpreted as a local gravitational acceleration. If 2.3–4.27 m/s^2 were intended instead, these values would represent approximately 0.23–0.44 standard (g), but that is a possible reinterpretation rather than a property established by the original hypothesis. In addition to gravitational parameters, any electromagnetic component of the capsule environment should be quantitatively defined using magnetic flux density measured in tesla (T), together with the associated field characteristics required for engineering validation.

This distinction is clinically material. Artificial gravity in human research is ordinarily produced through centrifugation, where acceleration depends on angular velocity and distance from the axis of rotation. Kramer et al. used individually

adjusted short-arm centrifugation producing approximately 1 (g) at the centre of mass and approximately 2 (g) at the feet during a 60-day head-down bed-rest experiment[9]. The intervention did not preserve aerobic capacity but attenuated selected losses in plantar-flexor strength and jumping power. Linnarsson et al. found partial preservation of orthostatic tolerance following short-term bed rest[10], whereas Attias et al. found no measurable artificial-gravity effect on the motor-unit outcomes studied during 60 days of bed rest[11]. More recent studies suggest partial protection of bone, metabolic rate or muscle volume under particular protocols without universal preservation of physiological function [12-14].

Spaceflight research provides strong evidence that marked alteration of gravitational loading can influence multiple human systems, including cardiovascular, musculoskeletal, immune, metabolic and molecular processes[8]. The NASA Twins Study documented multi-system biological adaptations during long-duration spaceflight, but adaptation to microgravity does not establish that ordinary terrestrial diseases originate from small disturbances in gravitational fields or that lowering gravitational acceleration will reverse unrelated disease mechanisms[8]. Existing artificial-gravity research is therefore better interpreted as evidence for a countermeasure against unloading-associated deconditioning, not evidence for a universal disease treatment.

Electromagnetic therapy requires equally precise definition. Frequency, magnetic flux density expressed in tesla (T), electric-field strength, waveform, pulse duration, duty cycle, coil or electrode geometry, treatment location, exposure duration and resulting tissue dose may all influence biological effect. Clinical transcranial magnetic stimulation provides a good example of this specificity. FDA-cleared TMS systems create short bursts of magnetic fields that produce localised currents in the cortex, and the NeuroStar system was cleared for specific patients with MDD, not for general systemic types of illness[16]. O'Reardon et al. demonstrated efficacy in a multicentre sham-controlled depression trial[15], supporting a therapeutic effect of carefully targeted electromagnetic stimulation without implying efficacy against unrelated disorders. For the proposed capsule concept, the inclusion of Tesla as a physical quantity indicates that the internal electromagnetic environment would require deliberate field generation, measurement, and control rather than being considered an unspecified background condition.

Tumour-treating fields provide another example. Alternating electric fields applied using disease-specific parameters improved survival when added to maintenance temozolomide in patients with glioblastoma in randomised clinical investigation[17]. By contrast, pulsed electromagnetic-field studies in knee osteoarthritis have produced variable outcomes. Ay and Evcik observed improvements within both active and placebo groups but did not demonstrate a significant between-group benefit for overall pain or total functional score[18], whereas other protocols have reported beneficial outcomes[19]. These studies demonstrate a central principle: the biological activity of an electromagnetic field is parameter- and indication-dependent.

There is currently no established clinical mechanism by which a therapeutic gravitational or electromagnetic environment would restore disease by repositioning hydrogen atoms or molecules. Atomic and molecular organisation in biological tissues depends on electromagnetic interactions, chemical bonding, molecular conformation, thermal motion and biochemical energy landscapes, while ordinary gravitational interactions at molecular scale are extremely weak relative to electromagnetic interactions. Accordingly, a molecular correction hypothesis would require direct measurements showing a reproducible exposure-dependent change in a defined molecular target followed by a causal link to clinically meaningful disease improvement. The existing field-therapy literature does not supply that evidence.

4. Clinical Evidence across Field-Based and Broad-Spectrum Therapeutic Approaches

Table 1 Representative evidence relevant to broad-spectrum therapeutic claims

Intervention	Clinical context	Highest relevant evidence	Main finding	Implication
Artificial gravity	Bed-rest/microgravity deconditioning	Randomised controlled bed-rest studies [9–14]	Partial protection of selected musculoskeletal, orthostatic, skeletal or metabolic outcomes; other endpoints unchanged	Physiological effects are real but endpoint-specific
TMS	Major depressive disorder	Sham-controlled randomised trials and regulatory clearance [15,16]	Therapeutic benefit in defined neuropsychiatric populations	Electromagnetic therapy requires anatomical and dosing specificity
Tumour-treating fields	Glioblastoma	Randomised clinical trial [17]	Improved survival when combined with standard therapy	Field therapy can be clinically effective without being broadly curative
PEMF	Knee osteoarthritis	Randomised placebo-controlled studies [18,19]	Heterogeneous results depending on outcome and protocol	Field modality alone does not predict efficacy
Niclosamide	Viral, inflammatory and other repurposed indications	Phase 1–2 trials and later RCTs [20–25]	Strong preclinical breadth but variable clinical efficacy and major formulation dependence	In-vitro breadth must not be equated with clinical breadth
4-Phenylbutyrate	Urea-cycle disease, CF, ALS and experimental systemic applications	Established metabolic use, small trials and ALS RCTs [26–36]	Valid activity in specific indications; inconsistent replication across diseases	Shared mechanisms require disease-specific validation
mTOR inhibition	Cancer, immunosenescence and age-related pathology	Multiple clinical trials [37–40]	Efficacy in selected settings with dose-dependent toxicities	A common pathway is not a universal therapeutic target
Broad-spectrum antibacterials	Serious bacterial infection	Phase 3 trials and observational studies [2,43,45,48]	Essential in appropriate settings but unnecessary breadth can increase toxicity and ecological harm	Therapeutic breadth carries trade-offs

The evidence summarised in Table 1 **Representative evidence relevant to broad-spectrum therapeutic claims**argues against a binary choice between "disease-specific therapy" and "panacea." Therapeutic breadth is better conceptualised as a continuum requiring evidence for each extension of indication.

5. Lessons from Validated Broad-Spectrum Therapeutics

5.1 Niclosamide: strong mechanistic breadth, difficult clinical translation

Niclosamide provides a particularly useful translational case. It is an established anthelmintic that has demonstrated activity against SARS-CoV, MERS-CoV, Zika virus, hepatitis C virus, adenovirus and other viruses in cellular experiments[3,24,25]. Xu et al. additionally describe effects on Wnt/β-catenin, mTORC1, STAT3, NF-κB and other signalling processes, illustrating genuine pharmacological pleiotropy[3]. However, the same review identifies poor aqueous solubility, incomplete intestinal absorption, variable systemic exposure and approximately 10% oral bioavailability as major barriers to clinical translation.

These limitations are clinically important. In a blinded phase 2 COVID-19 trial involving 73 randomised participants, conventional oral niclosamide did not significantly improve day-3 respiratory viral clearance compared with placebo and did not significantly shorten symptom duration[20]. Conversely, a small open-label ulcerative-colitis study reported clinical remission in 10 of 17 patients receiving rectal niclosamide but appropriately concluded that placebo-controlled studies were needed[21]. A 2025 randomised study of a nanohybrid formulation designed to improve niclosamide exposure reported greater viral-load reduction and faster symptom improvement in selected analyses, illustrating that formulation can materially alter clinical performance[22]. Yet another large prophylaxis study involving 1,651 high-risk renal patients did not establish intranasal niclosamide as a broadly effective preventive intervention[23]. The lesson is that extensive molecular activity cannot substitute for indication-, route- and formulation-specific trials.

5.2 4-Phenylbutyrate: shared cellular pathways but disease-dependent outcomes

4-Phenylbutyrate provides a second instructive example. It has established pharmacological use as an ammonia-scavenging treatment in urea-cycle disorders, while its chemical-chaperone and histone-deacetylase-related effects have motivated studies involving proteostasis, endoplasmic-reticulum stress, neurological disease, renal disease, cardiovascular injury, metabolic dysfunction, ocular disorders and infectious disease[5,29-36]. Bobat et al. reviewed 13 diseases across cardiovascular, hepatic, renal, neurological, ocular and integumentary systems, but their evidence base included in-vitro, animal, preclinical and clinical models rather than equivalent clinical proof across all 13 diseases[5].

This distinction is important as shown by human data. Phenylbutyrate has been compared clinically with other nitrogen-scavenging therapies in urea-cycle disorders[29,32,33], and early cystic-fibrosis studies demonstrated measurable effects on CFTR-related physiology without establishing it as a definitive modern CF therapy[30,31]. In ALS, the CENTAUR randomised trial of sodium phenylbutyrate plus taurursodiol reported a slower mean monthly decline in ALSFRS-R score than placebo over 24 weeks, although secondary endpoints were not significantly different[26]. Longer-term analyses subsequently suggested a survival difference[27]. However, the larger 664-participant PHOENIX phase 3 programme did not reproduce the primary functional benefit, with the sponsor reporting no significant difference in 48-week ALSFRS-R change and no significant secondary-endpoint benefit[28]. Thus, even an intervention targeting biologically fundamental processes such as endoplasmic-reticulum stress cannot be presumed effective across disorders.

5.3 mTOR: a shared pathway with clinically selective value

mTOR is a central regulator of growth, nutrient sensing, autophagy and cellular metabolism and is

implicated in ageing, senescence, immune function and oncogenesis[4]. Walters and Cox therefore described mTOR inhibitors as candidate broad-spectrum therapeutics for age-related pathology while simultaneously highlighting dose-related mucosal, haematological and metabolic adverse effects[4]. Human studies support beneficial but selective effects. Low-dose mTOR inhibition enhanced influenza-vaccine responses in older adults[37], and subsequent TORC1-directed work reported improved immune measures and reduced infection rates[38].

In oncology, everolimus combined with exemestane substantially prolonged progression-free survival in hormone-receptor-positive advanced breast cancer but increased grade 3–4 adverse events[39]. Temsirolimus improved survival compared with interferon alfa in selected patients with poor-prognosis metastatic renal-cell carcinoma[40]. However, there are significant tumour type differences in clinical responses to mTOR inhibition and the review provided specifically states that there is a need for biomarker selection, not the assumption that all tumours with pathway involvement will respond[4] [4]. Thus a common molecular pathway provides a potential for widespread application, but does not eliminate biological heterogeneity.

5.4 Broad-spectrum antimicrobial therapy: breadth can produce harm as well as benefit

Another example of the panacea concept is the use of broad-spectrum antibiotics. They are essential in situations of severe infection where empirical coverage is needed prior to microbiological identification, especially in sepsis and critical illness[2]. However, any additional width will contribute to selection pressure for antimicrobial resistance and create a potential imbalance in the beneficial microbial communities[2,46]. The provided 2026 review, therefore, calls for a shift to pathogen-specific and mechanism-targeted therapy, where available.

This caution is underlined by clinical evidence. Webb et al. studied 1,995 adults hospitalised with community-onset pneumonia and found that 39.7% received broad-spectrum antibiotics although resistant pathogens were recovered in only 3%; broad-spectrum treatment was associated with increased mortality and other adverse outcomes after adjustment, although observational confounding cannot be completely eliminated[45]. Likewise, azithromycin had a sufficient antimicrobial and immunomodulatory breadth to attract repurposing interest during COVID-19 but both RECOVERY and PRINCIPLE hospital and community trials failed to show clinically meaningful routine benefit[41,42] . By contrast, purpose-built broad antibacterial agents have shown promise when tested against a specific indication: gepotidacin was shown to be non-inferior to nitrofurantoin in phase 3 trials of uncomplicated UTI[43] and BWC0977 is a broad spectrum agent that progressed as a treatment for multidrug resistant bacterial pathogens[48].

All of the above are examples of what constitutes the proper scientific definition of a broad-spectrum therapy. It is NOT a treatment that is supposed to cure all diseases. It is an intervention that has a mechanism of action that may be effective in more than one biologically defined condition, and efficacy and safety have to be demonstrated for each relevant clinical setting.

6. Translational Framework for Evaluating the Proposed Method

Current field-based hypothesis should be viewed as evidence in the exploratory stage, not as a clinical treatment. An intervention with primarily theoretical evidence but no direct experimental evidence would most closely fit level 0; level 4 would be an intervention with well-documented clinical endpoints at doses or exposures that are known to be safe[1]. This classification does not refute the hypothesis. It identifies the experiments that need to be undertaken to progress it.

The first one is engineering specification. A gravitational intervention should include acceleration (in m/s2 or multiples of (g)), spatial gradients, how it was generated, body orientation, duration, intermittency, and for centrifugation, radius and angular velocity. Magnetic/electric

field strength, frequency spectrum, pulse characteristics, waveform, field uniformity, tissue distribution and exposure duration should be an independent specification of an electromagnetic intervention. Gravity and electromagnetic exposure should not be lumped together as a single undefined exposure variable, since they are different types of physical exposures, and they should be dosimetred differently.

The second is mechanistic validation. Experiments should determine a reproducible change in a measurable biological response that is correlated with exposure before exposing patients who have more than one disease. For each hypothesis, the outcomes of interest are defined signalling pathways, membrane potentials, inflammatory mediators, oxidative-stress markers, mitochondrial parameters, proteostasis markers, and/or validated physiological responses. An assertion concerning hydrogen positioning could only be made on the basis of direct biophysical measurement, not on the basis of symptomatic improvement.

The third requirement is safety evaluation. An initial human study should assess tolerability under controlled exposure conditions with prospective recording of cardiovascular, neurological, vestibular and other modality-specific adverse events. A sham condition is particularly important for device interventions because expectation and treatment context can influence patient-reported outcomes. Claims of absence of side effects should be made only after adequate sample size, exposure duration and surveillance.

The fourth requirement is disease-specific proof of concept. The first patient trial should involve one condition for which a plausible mechanism, measurable biomarker and validated clinical endpoint can be justified. Enrolling heterogeneous diseases and classifying all participants simply as “improved” or “recovered” would make causal inference extremely weak. Clinical endpoints should instead follow accepted standards for each disease, and protocol-defined primary and secondary outcomes should be specified before recruitment.

The fifth requirement is independent cross-disease replication. A broad-spectrum designation would become defensible only after reproducible benefit is demonstrated in more than one biologically distinct disease population using adequately controlled trials. Evidence that an intervention improves one outcome should not automatically be generalised to cancer, infection, cardiovascular disease, neurological disease or congenital disorders. This principle is consistent with the development framework proposed for broad-spectrum drugs, in which pharmacology, toxicology, clinical trials and post-marketing surveillance constitute separate evidence gates [1]. The supplied framework explicitly progresses from candidate selection through clinical testing and surveillance rather than directly from mechanistic plausibility to therapeutic deployment.

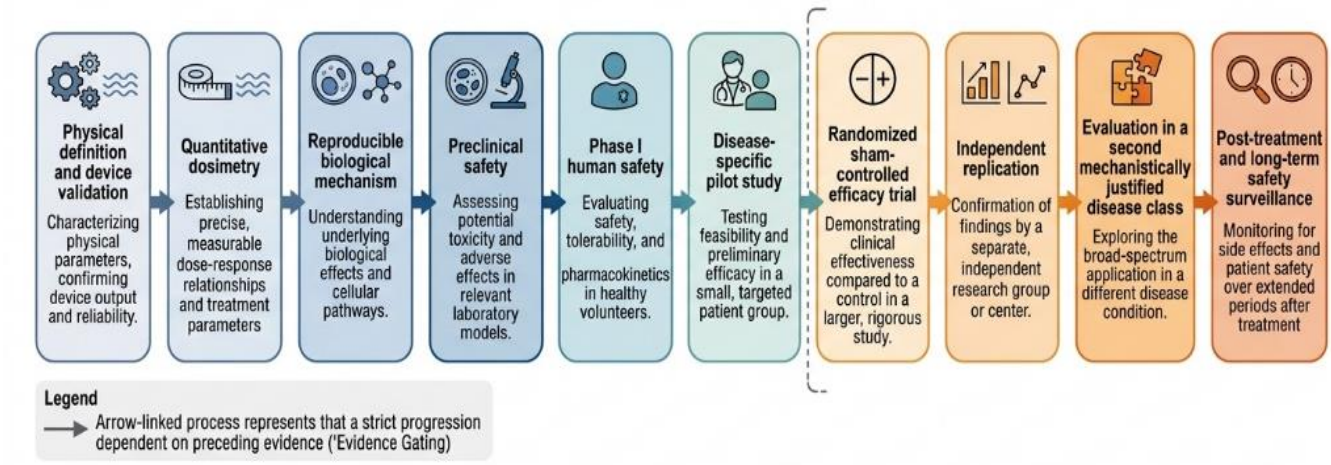

Figure 1. Proposed evidence-gated development pathway for a field-based broad-spectrum therapeutic method

This is an original synthesis developed for the present manuscript. Conceptually, it draws on the staged broad-spectrum therapeutic-development model of Firth and Prathapan[1] and the precision-treatment pipeline presented by Siva et al.[2]. The former advances from biological sampling to molecular detection to genomic characterisation to resistance prediction and targeted treatment selection; the general rule being that the more biological information, the more targeted treatment selection should be.

7. Limitations

There are several limitations of this rapid evidence synthesis. However, the subject is wide-spreading and was not created as a PRISMA-conforming systematic review, and thus, publication-selection bias and incomplete retrieval are possible. Considerable heterogeneity exists between pharmacological agents, gravitational interventions and electromagnetic devices, preventing quantitative pooling. Evidence from spaceflight and bed-rest studies primarily concerns physiological adaptation and prevention of deconditioning rather than treatment of established disease. Likewise, preclinical evidence supporting niclosamide, phenylbutyrate or mTOR-related activity should not be interpreted as human clinical efficacy where corresponding controlled trials are absent. Finally, the proposed field-based method currently lacks engineering validation, direct mechanistic experiments and patient-level clinical data, preventing any efficacy estimate.

8. Conclusion

Evidence across pharmacology, infectious disease, physical medicine and geroscience supports the principle that one intervention can influence more than one disease when those diseases share a therapeutically actionable biological process. Niclosamide, 4-phenylbutyrate, mTOR inhibitors and broad-spectrum antimicrobials illustrate this possibility, but they also demonstrate that mechanistic breadth frequently fails to translate into uniform clinical efficacy[1-5,20-48].

Physical-field therapies provide complementary evidence. Artificial gravity can modify selected physiological consequences of unloading, while TMS and tumour-treating fields demonstrate that appropriately dosed electromagnetic interventions can achieve clinical efficacy in specific indications[9-19]. None of these findings provides proof that changes in the gravitational and electromagnetic environment can cure all or most diseases.

As such, a gravitational/EM therapeutic cylinder should at the present time be presented as a therapeutic hypothesis to be tested, rather than a proven panacea. It will be dependent on the correction of the physical exposure definition, the demonstration of a reproducible mechanism, the establishment of safety and the generation of randomised disease specific clinical evidence. Those stages then, if they prove to have beneficial effects in other classes of diseases studied independently, could give empirical justification for referring to them as a broad-spectrum therapeutic method.

Declarations

Ethics approval: No new human participants, biological specimens or identifiable patient data were included in this evidence synthesis. Ethics approval was therefore not applicable to the present review. Any future prospective evaluation of the proposed intervention would require independent ethics approval and informed consent before participant enrolment.

Clinical trial registration: Not applicable to the present evidence synthesis. Any prospective interventional human study should be prospectively registered before enrolment.

Funding: To be completed by the authors.

Conflicts of interest: To be completed by the authors.

Data availability: All evidence evaluated in this manuscript was derived from published literature and publicly accessible scientific or regulatory sources.

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