

Mapping the biomarker landscape of Biofield Therapies: An exploration of measurable effects

Rick Sá

ricksa.info@omniness.com.br

Brazilian Academic Consortium for Integrative Health (CABSIN) <https://orcid.org/0009-0001-4700-029X>

Method Article

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Abstract

Background

Biofield Therapies (BTs), such as Reiki and Healing Touch, are increasingly utilized within integrative health. Despite their widespread use, the biological mechanisms underlying these non-invasive interventions remain poorly characterized, necessitating a systematic investigation of their physiological effects.

Objectives

This study aims to comprehensively map the biomarker landscape modulated by BTs to identify consistent physiological signatures and elucidate potential molecular and cellular mechanisms of action.

Methods

A focused analysis of a curated cohort of 15 studies was conducted, encompassing diverse BT modalities, experimental models (clinical, preclinical, and *in vitro*), and biomarker categories. Biomarkers were systematically classified by type, biological function, and clinical application. The analytical approach emphasized qualitative synthesis and exploratory pattern recognition due to significant methodological heterogeneity across studies.

Results

The analysis evaluated 73 biomarkers, distributed across human (37%), animal (34%), and *in vitro* (29%) models. Prognostic and prognostic-therapeutic biomarkers were predominant (58.9%). Inflammatory/immunological markers constituted the largest category (38.4%), followed by markers of cellular regulation (15.1%) and neuroendocrine activity (11.0%). The synthesis revealed consistent evidence of HPA axis regulation, enhanced immune function, cytoprotective effects, selective apoptosis, and telomere lengthening. Ultraweak photon emission was identified as a promising biophysical metric for quantifying biofield interactions.

Conclusion

BTs are associated with reproducible, multi-system physiological changes, supporting their role in integrated organismal regulation. This review provides a foundational biomolecular framework for BT mechanisms and highlights ultraweak photon emission as a key correlate for future research. Subsequent studies should prioritize diagnostic biomarker discovery and treatment optimization through dose-response studies utilizing such biophysical parameters.

1. INTRODUCTION

The term biomarker has undergone substantial evolution since its initial characterizations as biochemical markers or biological markers [1]. Formally introduced in 1973 by Rho and colleagues to indicate the presence or absence of specific biological material, the concept has since matured into a pillar of contemporary biomedical science [2]. According to the U.S. Food and Drug Administration (FDA), a biomarker is defined as a measurable indicator that offers utility across the entire disease continuum, from foundational research to clinical therapeutic development [3]. This broad applicability is reflected in resources such as *MarkerDB 2.0*, which archives over 34,000 distinct biomarkers encompassing genetic, proteomic, and metabolic entities. Rigorous validation and standardization of biomarkers now constitute a critical component of modern research, enabling precise quantification of physiological processes, disease mechanisms, and responses to treatment [4].

Parallel to these conceptual advances, the term human biofield was formally introduced in 1994 and has been accepted by the U.S. National Library of Medicine as a medical subject heading search (MeSH) term [5, 6]. It is described as a massless field, not necessarily electromagnetic, that both surrounds and permeates the human body and is hypothesized to play a fundamental role in homeostatic regulation [5, 7–9]. This biofield concept serves as the foundational framework for a distinct class of interventions known as Biofield Therapies (BTs). Recognized by the National Cancer Institute's Division of Cancer Treatment and Diagnosis (DCTD/NCI), this category includes practices such as Reiki, External Qigong, Therapeutic Touch, and Healing Touch [9]. These therapies are increasingly studied within the broader domains of complementary and alternative medicine, particularly under the subfield of energy medicine. Empirical observations of practitioners capable of transferring bioenergy to elicit therapeutic effects have led to proposals that this process may operate through principles analogous to energy transfer in electrical engineering. Such transfer is thought to facilitate a form of bioenergetic communication between living systems, suggesting a plausible mechanism for the observed physiological and clinical outcomes associated with BTs.

Global health organizations, including the World Health Organization (WHO), have increasingly acknowledged the necessity of scientifically evaluating Traditional, Complementary, and Integrative Medicine [10]. These practices are considered an important and often underestimated health resource, particularly in the prevention and management of lifestyle-related chronic diseases and in addressing the health needs of aging populations, thus potentially alleviating the burden on conventional health systems [10]. This effort includes investigating the mechanisms of BTs and establishing clinical parameters that ensure their safe and credible application. Within this context, a growing body of evidence, spanning controlled *in vitro* and *in vivo* studies to randomized controlled trials (RCTs), consistently reports significant physiological effects [11]. These outcomes are quantitatively demonstrated through modulations in a diverse array of objective biomarkers, providing a measurable basis for their therapeutic potential. Critically, if these biomarker modulations prove to be consistent and specific, they may represent measurable downstream effects of a fundamental yet elusive process: directed bioenergetic communication

between practitioner and recipient. Confirmation of such a phenomenon would constitute a profound paradigm shift, offering compelling objective evidence of a direct biological influence inherent to these practices. Such a contribution would transcend anecdotal discourse and firmly establish BTs as a legitimate field of biophysical inquiry.

Consequently, this review was designed to systematically explore this premise. It proposed to fill the current knowledge gap by: (I) curating a new, comprehensive bibliographic database detailing therapeutic protocols, measured biomarkers, and clinical outcomes; (II) organizing these biomarkers into a structured, mechanistic classification to elucidate potential bioenergetic signaling pathways and their therapeutic applications; and (III) performing a quantitative and narrative analysis of the biomarker data to discern statistically consistent modulation patterns.

2. METHODS

2.1. Review Design and Rationale

This study employs a focused, in-depth analysis of a cohort of 15 studies selected to explore biomarker patterns in BT research. Unlike systematic reviews that strive for comprehensive literature coverage, this analytical approach selected studies based on their methodological diversity and biomarker richness to enable a detailed cross-sectional comparison of molecular correlates across different biofield modalities. Studies were selected to represent: (1) the spectrum of cognitive therapies (Reiki, Healing Touch, Qigong, etc.); (2) diverse study designs (RCTs, preclinical, *in vitro*); and (3) a broad range of biomarker categories (inflammatory, hormonal, cellular).

2.2. Study Selection and Data Extraction

The 15 studies were selected through a sampling approach, aiming to maximize biomarker diversity rather than comprehensive coverage. Studies were included if they reported empirical data from randomized controlled trials (RCTs), preclinical studies (*in vivo* or *in vitro* models), or other controlled experimental designs that evaluated the effect of a defined BT on one or more measurable biomarkers in human, animal, or cellular models.

Exclusion criteria included studies that relied exclusively on physiological parameters, psychometric instruments, or imaging biomarkers without molecular/cellular biomarker data; non-peer-reviewed publications; and duplicate datasets.

2.3. Data Synthesis and Analysis

All analyses were explicitly exploratory given the purposive study selection and heterogeneous biomarker landscape.

2.4. Biomarker Classification

The biomarker classification in this analysis was based on a specialized three-tiered framework designed to ensure scientific rigor and contextual relevance, rather than automated platform-generated categories. This framework integrated established regulatory guidelines, physiological knowledge, and study-specific context to ensure consistent and evidence-based categorization.

First, formal definitions and consensus frameworks from regulatory bodies (e.g., U.S. FDA, NIH) and databases such as *MarkerDB* were applied to align with standardized nomenclature [4, 12]. Second, biomarkers were systematically classified by: Type (Molecular or Cellular Biomarkers); Biological Function (e.g., inflammatory, hormonal, metabolic; see Supplementary Material for complete list). According to these guidelines, biomarkers of Clinical Application were categorized into five primary types:

Diagnostic: Used to detect or confirm the presence of a disease or condition;

Prognostic: Identifies the likelihood of a clinical outcome (favorable or adverse), independent of treatment;

Therapeutic: Monitors responses to an intervention and is often the direct target of therapy;

Diagnostic & Prognostic: Serves both to diagnose conditions and predict disease trajectories;

Prognostic & Therapeutic: Predicts clinical outcomes and is modulated by therapeutic interventions, reflecting its dynamic role in treatment monitoring and mechanistic validation.

Third, study designs (human, animal, or *in vitro*) and experimental contexts were incorporated to refine classifications based on methodological relevance. This structured approach ensured a reproducible, biologically logical, and context-aware categorization system, facilitating robust cross-study comparisons and analyses.

3. RESULTS

The selected cohort comprised 15 studies, encompassing a diverse set of BTs: Reiki (n = 2), Therapeutic Touch (n = 2), Healing Touch (n = 2), Spirit "Passe" (n = 1), External Qigong (n = 2), and other modalities (n = 5). Of these, six were randomized controlled trials (RCTs) involving 313 human participants (experimental group: n = 134; control group: n = 179), representing 40% of the included studies. Four studies (26.66%) were conducted *in vitro*, three (20%) employed preclinical animal models, and the remaining two included one case-controlled *in vitro* double-blind study (6.67%) and one quasi-experimental mixed-methods design (6.67%). From each eligible study, data were extracted into a standardized coding spreadsheet. The extracted information included: (1) first author and year of publication; (2) Biofield Therapy; (3) populations (n); (4) study design (e.g., type, duration, number of sessions, distance from the

subject); (5) control group conditions; (6) dose; (7) type of intervention; (8) biomarkers; (9) biomarkers results; (10) clinical results and; (11) observations (See the Condensed Table 1).

Table 1
Condensed table of the 15 studies with detailed d

Author (Year)	Biofield Therapy	Population (n)	Study Design	Control	Dose	Type of Intervention	Bioma
Bektas et al. (2025) [17]	Reiki	Patients undergoing autologous BMT (n = 21 Reiki; n = 21 Control)	Prospective RCT	Usual care (no intervention)	30 min/day, days 0, 1 (in-person) and 2 (distance) post-BMT	With touch and at a distance	PCT, C Neutrc Platelets
Bai et al. (2021) [19]	1. Qigong External Qi 2. Qi from Herb (Astragali Radix) 3. RPV-Field (Torsion Field)	<i>In Vitro</i> : M-1 mouse kidney cells. <i>In Vivo</i> : Female C57BL/6J mice (8 months old). (n = 3 per group for animal study)	Pre-Clinical Study (<i>In Vitro</i> and <i>In Vivo</i>)	<i>In Vitro</i> : Untreated cells (for each Qi source). <i>In Vivo</i> : Mice administered water only	1. Qigong External Qi: Single 15-min session. 2. Astragali Radix: Cells: 50 µg/ml for 4h. Mice: 0.20 ml/10g daily for 7 days (intragastric). 3. RPV-Field: Cells: Exposure for 48h	No touch (for Qigong and RPV interventions)	Primar length Secon Telom activity TERT (expres
Carneiro et al. (2018) [14]	Spiritist "Passe"	Preterm newborns (NBs) in nursery (n = 13 SP; n = 12 Control)	Randomized, Double-Blind Controlled Trial	Sham "Passe" (Laying on of hands with healing intention but no spiritual component)	3 sessions (1x/day) of 10 min each, over 3 consecutive days	No touch (hands 10–15 cm from body)	Saliva
Cohen et al. (2024) [41]	Bengston Energy Healing Method	Human pancreatic cancer cells (PANC-1) <i>in vitro</i> (n = 40 sessions for live cell treatment, n = 20 for control cell treatment, n = 40 for sham control cells)	Experimental, double-blind, 2x2 (treatment vs. no-treatment; live cells vs. control) case study	1. Dead cells or medium-only (no cells) for human physiology comparison. 2. Sham-treated control cells (person mimicking movements/distance) for cellular outcome comparison	60 sessions total (6 sessions/day for 10 days). Each session: 15 min treatment (5 min still, 5 min movement allowed, 5 min still) preceded and followed by 2 min baseline	No touch (distance of ~ 12 inches from cells)	Intracel tubulir
Gronowicz et al. (2015) [21]	Therapeutic Touch (TT)	Female BALB/c mice with injected 66c14 mammary carcinoma cells. (Study 1: n = 16 TT, n = 16 Mock, n = 8 PBS; Study 2: n = 12 TT, n = 12 Mock, n = 8 PBS)	Pre-Clinical Animal Study	Mock Treatment (CA): Mice placed in apparatus for 10 min, 2x/wk with a non-TT person present. PBS Control (PBS): Mice injected with vehicle (PBS), no treatment	Study 1 (TT1): 10-min sessions, 2x/wk for 26 days (started 24h post-injection). Study 2 (TT2): 10-min sessions, 2x/wk for 2 weeks prior to injection and continued for 29 days post-injection	Non-Contact (hands 2–10 inches from apparatus)	Serum IL-1α, IL-1β, MIP-2, IL-4, IL-12(p40), CSF (3 panel) Immune (FACS): %CD44 ⁺ , %CD44 ⁺ , %CD44 ⁺ , %CD44 ⁺ , lympho, %CD11b ⁺ , macro Tumor PCNA (prolifer

Author (Year)	Biofield Therapy	Population (n)	Study Design	Control	Dose	Type of Intervention	Bioma
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Jain et al. (2012) [13]	Energy Chelation (Biofield Healing)	Fatigued breast cancer survivors (stages I-III A) (n = 27 Healing; n = 30 Mock; n = 19 Control)	Blinded RCT	Mock Healing (identical hand positions, no healing intent) and Waitlist	8 sessions (2x/week) of 1 hour each, over 4 weeks	Hands-on touch	Diurna slope (
Jhaveri et al. (2008) [38]	Therapeutic Touch (TT)	Human osteoblastic cells; n = 3 different lineages (passages 3–6)	<i>In vitro</i> experimental study	No TT (unmanipulated plate)	10 minutes per session, 1 session/day, for 10 days	No touch (approx. 5–10 cm above the cells)	DNA (quant PicoGr alkalin phosp (activi depos (Alizar
Kent et al. (2020) [49]	Reiki	Mouse intervertebral disc (IVD) cells <i>in vitro</i> (n = 2 cell plates per group, experiments in duplicate)	Laboratory <i>in vitro</i> study	Sham (practitioner with no knowledge of biofield therapy, instructed to have distracting thoughts)	10-minute sessions, once daily for 3 successive days	No touch (practitioner's hands placed under cell plate tray inside a light-tight box)	Collag gene e Collag gene e Aggre expres Biophc Emiss
Kokubo et al. (2007) [48]	Laying-on-of-hands (Qigong master), Prayer	Pairs of cucumber pieces (n = 15 Exp/Ctrl pairs Healing; n = 15 pairs Non-Tx; n = 16 pairs Thermal)	<i>In vitro</i> experimental study with paired samples	Non-Treatment (immediate measurement) & Active Control (Thermal stress at 40°C for 30 min)	Laying-on-of-hands: 15–30 min/session; Prayer: 5 min/session at 1.2m distance. (Single session per sample pair)	Non-contact (Hands near but not touching dish; prayer at a distance)	iophot intens (count pixels/
Lutgendorf et al. (2010) [15]	Healing Touch (HT)	Women with cervical cancer undergoing chemoradiation (n = 17 HT; n = 17 RT; n = 17 UC)	RCT	Relaxation (active) and Usual Care	4x/week, ~25min/session, for 6 weeks	With and without touch	NKCC, %NK, V

Author (Year)	Biofield Therapy	Population (n)	Study Design	Control	Dose	Type of Intervention	Bioma
Running et al. (2022) [16]	Healing Touch (HT)	University students (n = 21 Exp; n = 21 Ctrl)	Randomized Controlled Trial	Watched a demonstration video	Single 20-minute session	Mixed (hands-on and hands-off)	Saliva IL-6, S Diasto
Trivedi et al. (2023) [18]	Biofield Energy Healing (The Trivedi Effect®)	Adult subjects with psychological symptoms (n = 77; 35 Exp, 42 Ctrl)	Randomized Controlled Trial (RCT)	Naive attunement (sham)	2 in-person sessions (3 min each) at day 0 and 90	No touch (hands ~ 10–20 cm above head)	Immur CD8+C Neuro Norepi Dopan Acetyl Hormc Oxytoc estrad Anti-a Inflam Cytoki IL-1β, I Oxidat Isopro Oxidiz
Wilkinson et al. (2002) [22]	Healing Touch (HT)	Mixed-diagnosis adults (n = 22 total; n = 10 w/ more trained practitioners; n = 12 w/ less trained practitioners)	Mixed-method repeated measures (quasi-experimental & naturalistic)	No Treatment (NT) - rested on table for 30 min with practitioner present	2 sessions (1 HT, 1 HT+); 30–45 min each; over 2 weeks	Mixed (hands-on and hands-off)	Secret Immur (slgA)
Yan et al. (2004) [20]	External Qi of Yan Xin Life Sciences Technology (YXLST)	Primary retinal neurons from 0–2 day old Sprague-Dawley rats, cultured <i>in vitro</i> (n of replicates per group ranged from 3 to 9)	Laboratory <i>in vitro</i> study with dual-blind design	Sham-operated procedure in the tissue culture room (no real Qi emission)	1 session of 10 min of Qi emission, applied 30 min prior to toxic stimulus (H ₂ O ₂) in most experiments	No touch (distance; emitter in a separate locked room)	Cell Vi assay) (TUNE DNA lα PI3K e activity; expres (North
Yan et al. (2006) [39]	External Qi of Yan Xin Qigong (YXQ)	Human pancreatic cancer cells (BxPC3 line) and human fibroblasts in culture. (Exact n not specified; experiments repeated 3–6 times)	Pre-Clinical <i>in vitro</i> Study	Untreated cells (same conditions, no Biofield exposure)	Protocol 1 (Apoptosis): Single 5-min session. Protocol 2 (Lysis): Three 5-min sessions, with 25-min intervals between them (total protocol duration: 65 min)	No touch (cells were transferred to a treatment room for exposure)	Phosp Phosp PI3K a κB act (EMSA 3/8/9 PARP DNA fragm Sub-G populat releas

A total of 73 biomarkers were evaluated in the included studies, of which 48 (65.75%) were molecular and 25 (34.25%) were cellular in nature. This distribution highlights the balanced contribution of the three research models: human studies (n = 27, 37.0%), animal models (*in vivo*) (n = 25, 34.2%), and *in vitro* laboratory research (n = 21, 28.8%). This triad underscores a robust translational pipeline, where over half of the evidence base (pre-clinical + *in vitro* =

63.0%) is derived from non-clinical research, ensuring that clinical findings are underpinned by a solid foundation of mechanistic exploration. The hierarchical relationships and relative frequencies of these biomarkers are visually summarized in the sunburst plot (Fig. 1).

When classified by their research purpose, Prognostic biomarkers represented the largest group (n = 22; 30.14%), followed by combined Prognostic & Therapeutic biomarkers (n = 21; 28.77%), and Diagnostic & Prognostic biomarkers (n = 13; 17.87%). Purely Diagnostic biomarkers were less frequent (n = 9; 12.33%), and Therapeutic biomarkers alone represented only 6.86% (n = 5). Regarding the subclassification of biomarkers, the majority were related to Inflammatory & Immunological processes (n = 28; 38.36%), followed by markers of cell proliferation and apoptosis (n = 11; 15.07%), Hormonal & Stress (n = 8; 10.96%), and Cell Signaling & Metabolic Pathways (n = 7; 9.59%). Markers associated with Extracellular & Bone Matrix Remodeling (n = 4; 5.48%) and Aging & Longevity (n = 4; 5.48%) were less frequent, while Oxidation & Cellular Stress (n = 3; 4.11%), Acute Phase Proteins (n = 2; 2.74%), and Other unclassifiable biomarkers (n = 6; 8.22%) were sparsely represented (See the Table 2).

Data were pooled from seven clinical studies involving human subjects (n = 335), including six randomized controlled trials (RCTs) and one study with a repeated-measures design, it was possible to analyze the correlation between the molecular and cellular biomarkers evaluated and the demographic variables of gender, age, and distribution between experimental and control groups. The total sample was composed primarily of women (246 participants; 73.43%), while men represented 26.57% of the sample (89 participants). The average age varied considerably across the studies, encompassing young adults (~ 20 years) to middle-aged individuals (~ 50 years). Data on newborns were limited; in the included study, newborn admission to the nursery occurred between 2 and 15 days of life. The experimental group totaled 156 participants (46.57%), and the control group, 179 (53.43%).

Table 2. Characteristics of Included Studies, Participants, and Biomarkers. The table summarizes the distribution of study designs (15 studies in total), participant demographics (335 individuals in total), and classification of identified biomarkers (73 biomarkers in total).

Category	Total (n)	Proportion (%)	Proportion of Biomarkers per Study (%)
Study Designs			
Total Studies	15	100.00	-
RCTs	6	40.00	36.99
<i>In vitro</i> Studies	4	26.67	34.25
Preclinical Animal Studies	3	20.00	26.03
Case-Controlled Studies	1	6.67	1.37
Mixed Methods Studies	1	6.67	1.37
Participants (Human)			
Total Participants	335	100.00	-
Men	89	26.57	-
Women	246	73.43	-
Mean Age (Men)	~ 31.11	-	-
Mean Age (Women)	~ 43.87	-	-
Experimental Group	156	46.57	-
Control Group	179	53.43	-
Biomarkers			
Total Biomarkers	73	100.00	-
<i>By Type</i>			
Molecular Biomarkers	48	65.75	-
Cellular Biomarkers	25	34.25	-
<i>By Clinical Application</i>			
Diagnostic	9	12.33	-
Prognostic	22	30.14	-
Therapeutic	5	6.86	-
Diagnostic & Prognostic	13	17.81	-
Prognostic & Therapeutic	21	28.77	-
Not Specified	3	4.11	-
<i>By Biological Function</i>			
Inflammatory & Immunological	28	38.36	-
Hormonal & Stress	8	10.96	-
Oxidation & Cellular Stress	3	4.10	-
Cell Proliferation & Apoptosis	11	15.07	-
Cell Signaling & Metabolic	7	9.59	-
Extracellular & Bone Matrix	4	5.48	-
Aging & Longevity	4	5.48	-
Acute Phase Proteins	2	2.74	-
Others	6	8.22	-

The most frequently assessed biomarkers in humans included markers of stress and inflammation, such as salivary cortisol (or its diurnal variability) [13, 14, 16], neutrophil percentage [17], and NK cell percentage [15], which were evaluated in four studies. The proportion of female participants in these studies was 100%, 100%, 76,19% and 28.57%, respectively. The results of Trivedi et al. (2023) [18], which reported extremely large effect sizes, were considered outliers and analyzed separately due to intractable heterogeneity with the rest of the literature.

Exploratory examination of the four studies reporting stress and immune biomarkers revealed considerable variability in effects across different demographic contexts (Table 3). Studies with predominantly female samples (Jain et al. 2012: 100% female [13]; Running et al. 2022: 76% female [16]) showed positive effect sizes ranging from + 0.282 to + 0.735, while the study with balanced gender distribution (Bektas et al. 2025: 29% female [17]) showed

a more modest effect (+ 0.246). Similarly, effects appeared consistent across age groups, with both younger (Running et al. 2022: 20.2 years [16]) and older (Jain et al. 2012: 52 years [13]; Lutgendorf et al. 2010: 48.1 years [15]) samples showing positive directions. However, the small number of studies (n = 4) precluded meaningful statistical analysis of this demographic relationship.

Table 3. Results of moderator analysis for demographic variables: Mean Age and Percentage of Female Participants. The table presents statistics from the meta-analysis model, including residual heterogeneity and significance tests for the moderators.

Parameter	Moderator: Mean Age	Moderator: % Female
Model Statistics		
Number of studies (k)	4	4
Residual heterogeneity (τ^2)	0.1723	0.1966
I ² (%)	62.68	65.95
H ²	2.68	2.94
<i>Test for Residual Heterogeneity</i>		
QE (df)	5.30 (2)	5.77 (2)
p-value	0.071	0.056
Moderator Results		
Coefficient (β)	-0.0201	-0.0060
Standard Error (SE)	0.0209	0.0073
z-value	-0.964	-0.823
p-value	0.335	0.411
95% CI (β)	-0.0610, 0.0208	-0.0202, 0.0083
<i>Test of Moderators</i>		
QM (df)	0.93 (1)	0.68 (1)
p-value	0.335	0.411

4. DISCUSSION

4.1. Evidence Analysis: Biomarkers Modulated by BTs

The analysis of biomarkers in the included studies reveals a mature and multifaceted research strategy, focused on objectively validating the biological effects of BTs. The distribution reflects a perspective focused on understanding their potential to predict disease course and therapeutic outcomes, rather than focusing solely on diagnostic or treatment-specific outcomes. This is confirmed by the significant frequencies of Prognostic biomarkers (30.14%) and the combined Prognostic & Therapeutic category (28.77%), which go beyond the simple detection of a biological effect.

They highlight the field's ambition to demonstrate that biofield-induced modulation can influence the course and outcome of a disease. By monitoring biomarkers such as diurnal cortisol (for stress resilience) or NK activity (for immune competence in oncology), researchers are not simply looking for a biological corollary, but rather an indicator of better patient prognosis. The strong predominance of molecular biomarkers (65.75%) over cellular biomarkers (34.25%) indicates a focus on identifying precise and measurable biochemical signatures of treatment response.

This approach emphasizes practicality and analytical reliability, utilizing established laboratory methods such as ELISA and qPCR [19], to detect changes accurately and reproducibly. Functional categorization further clarifies this focus. The high frequency of Inflammatory & Immunological biomarkers (including IL-6, IL-1 β , IL-8, TNF- α , sIgA, CD8⁺CD28⁻, leucocytes) [18, 20, 22], red blood cells (RBC) and Natural Killer Cells (NKCC, NKAUC, and %NK) [15], neutrophils and platelets [17], and highlights that a key research interest lies in the ability of BTs to modulate immunoinflammatory responses, key processes in many chronic diseases. This strategic choice is clinically wise, given that such biomarkers (e.g., cytokines, natural killer cell activity, C-reactive protein) are widely recognized indicators of disease state and therapeutic response, and are readily measurable in serum or saliva samples.

The distribution of biomarkers across diverse functional categories in this review reflects a concerted effort to move beyond isolated markers and elucidate the complex, systems-level mechanisms underlying BTs. This approach aligns with the growing recognition that individual biomarkers, while valuable, cannot be considered the ultimate endpoint of biological discovery or therapeutic development. A true understanding of an intervention's effect is contingent upon mapping its complex ramifications across entire biological systems. That analysis, by cataloging biomarkers from Cell Proliferation & Apoptosis to Cell Signaling & Metabolic Pathways, represents a step in this direction. This multifaceted mapping is crucial, as the initial assessment of therapies has often relied on a relatively static set of assays, and extrapolations from animal models to human biology have frequently proven unreliable. The investigation into fundamental cellular processes, particularly evidenced *in vitro* studies, provides a necessary foundation for understanding bioenergy's specific mechanisms of action. Simultaneously, the emerging interest in categories like Extracellular Matrix & Bone and Aging & Longevity signals a novel frontier exploring the

regenerative and anti-aging potential of BTs. This shift is increasingly feasible due to drastic reductions in the cost of integrated genetic, genomic, and biological measurements [23, 24].

While the current lower representation of categories like Oxidation & Cellular Stress highlights an underdeveloped area, it also identifies a fertile ground for future research. Ultimately, a massive amount of validation for these increasingly complex models will be required. However, this work is essential to enable a more effective prediction of the impact of biofield interventions on integrative biology and clinical outcomes, moving from a focus on isolated biomarkers to a comprehensive network of biological effects.

4.2. Stress Response and Neuroimmunomodulation

BTs appear to operate through psychoneuroimmunological (PNI) mechanisms found in the literature [25–33], mainly modulating the hypothalamic-pituitary-adrenal (HPA) axis to promote allostatic balance where various clinical outcomes have been analyzed in different interventions [34–37]. This upstream neuroendocrine regulation directly influences downstream immune function, positioning HPA axis modulation as a central pathway for these interventions. Salivary cortisol findings are particularly revealing [13, 14, 16]. A double-blind study of preterm infants receiving Spiritist "passe" showed a strong trend toward reduced cortisol ($p = 0.05$), suggesting direct HPA modulation independent of cognitive placebo effects [14]. More notably, in breast cancer survivors, Energy Chelation significantly steepened the diurnal cortisol slope ($p < 0.04$; $d = 0.58$), indicating enhanced HPA resilience, an effect surpassing active control, implying practitioner-driven neurobiological influence [13].

Immunologically, HPA modulation translates into functional preservation rather than mere cell count changes. During cervical cancer chemoradiation, Healing Touch maintained NK cell cytotoxicity (NKCC and NKAUC; group-time interaction $p < 0.05$) despite systemic myelosuppression [15]. This aligns with PNI principles: neuroendocrine regulation protects immune function under stress [29, 30]. Similarly, increased secretory IgA after HT ($p = 0.021$) reflects a shift toward parasympathetic dominance, supporting mucosal immunity [22]. The Trivedi Effect study offers a systems-level view: simultaneous elevation of neurohormones (oxytocin $\uparrow 412.71\%$, dopamine $\uparrow 422.96\%$) alongside reduced inflammation (IL-6 $\downarrow 76.86\%$) and elevated anti-aging markers (klotho $\uparrow 685.33\%$) reflects coordinated allostatic regulation [18]. Briefly, the BTs likely initiate deep relaxation and HPA axis regulation, leading to: Preserved immune cell function under stress; Enhanced mucosal immunity; Reduced inflammatory burden; Improved neurobiological resilience. Thus, stress modulation is not ancillary but a core mechanism through which BTs influence diverse physiological outcomes. (As illustrated in Fig. 2).

4.3. Gene Expression and Molecular Signaling Pathways

In addition to systemic neuroendocrine effects, BTs exert direct, reproducible influences on gene expression and cellular signaling *in vitro*, pointing to a primary biophysical mechanism. Evidence shows that bioenergetic inputs can selectively reprogram cellular behavior.

It has been demonstrated in a controlled manner that intervention with Therapeutic Touch (TT) significantly modulates key biomarkers of osteoblastic function *in vitro*. In primary human osteoblasts (HOBs), TT treatment increased DNA synthesis, a marker of cell proliferation, and promoted extracellular matrix mineralization [38]. Simultaneously, the intervention elevated gene expression of key differentiation markers such as type I collagen, bone sialoprotein, and alkaline phosphatase. In contrast, in the SaOs-2 osteosarcoma cell line, the same intervention exerted an inhibitory effect, reducing mineralization and the expression of these same biomarkers. These results suggest that TT is capable of selectively modulating fundamental cellular processes, stimulating activity in healthy bone cells and inhibiting it in cancerous cells, highlighting its potential as a modulating therapeutic intervention with biologically distinct effects.

In primary rat retinal neurons, a brief 10-minute exposure to External Qi (Yan Xin) before oxidative insult (H_2O_2) activated the PI3K–Akt survival pathway, elevating PI3K activity by 3.5–6-fold ($p < 0.05$) and upregulating IGF-I mRNA. This pretreatment conferred significant resistance to

apoptosis ($p < 0.05$), demonstrating that bioenergetic stimulation can epigenetically prime cytoprotective responses. BTs exhibit striking cell-type specificity [20]. In human pancreatic cancer cells (BxPC3), Yan Xin Qigong suppressed survival pathways (Akt, ERK, NF- κ B) and triggered apoptosis, whereas in normal fibroblasts the same exposure transiently activated these pathways without inducing damage ($p < 0.05$) [39]. This bidirectional action, pro-death in malignant cells, pro-survival in healthy cells, suggests a targeted bioinformatic effect beyond nonspecific stimulation.

These *in vitro* findings have been corroborated *in vivo*. At the University of Texas MD Anderson Cancer Center, exposure to a putative biofield emitter (Sean Harribance) significantly reduced Lewis lung carcinoma (LLC) tumor growth in mice following 30-minute sessions, concomitant with PI3K/Akt/mTOR downregulation (decreased pAkt and pS6) in the tumor microenvironment [40]. Notably, this intervention also remodeled the immune landscape: it downregulated PD-L1, lowered circulating MCP-1, increased cytotoxic CD8⁺ T-cell infiltration, and reduced macrophages and regulatory T cells. A follow-up study using longer sessions (60 minutes) did not replicate the tumor volume reduction but revealed enhanced mechanistic effects [41]. BT increased apoptotic and necrotic cell death (necrotic area nearly fourfold higher), boosted CD8⁺ T cells 2.7-fold, reduced Tregs by 30% (CD8⁺/Treg ratio $\uparrow 3.1$ -fold), lowered macrophage density by 51% (CD68⁺), and shifted the M1/M2 phenotype toward an antitumor (M1) profile. Crucially, SOX2, a biomarker of cancer stemness, was reduced by 33%, suggesting a novel mechanism for recurrence and metastasis control. Together, these data delineate a dual mechanism: direct suppression of oncogenic signaling and stemness, combined with indirect immune modulation. The observation that exposure duration can pivot the primary outcome, from tumor burden reduction to immune-driven cell death, highlights the complexity of the biophysical interaction and underscores the need for dose–response optimization.

Further supporting precision, Bengston Energy Healing applied to PANC-1 pancreatic cancer cells selectively altered Ca^{2+} homeostasis (smaller increase vs. sham, $p = 0.03$) without affecting structural proteins. Functionally, this translated to dramatically reduced invasiveness ($p < 0.0001$) in post-intervention assays, indicating lasting modification of metastatic behavior [42]. Perhaps most remarkably, biofield stimuli directly affect genomic aging. Application of External Qi, energized Astragali Radix, or a torsion field to rat kidney cells increased telomere length by 28–43% within 4 hours. While Qigong Master Qi

increased TERT expression, other methods achieved telomere lengthening via TERT-independent pathways, suggesting multiple biophysical routes to genomic regulation [19].

These *in vitro* findings indicate that BTs can act as epigenetic modulators, influencing key processes such as: Selective activation of cell survival/death pathways; Tissue-specific gene expression promoting regeneration; Telomere maintenance and genomic stability; Long-term functional changes, including reduced cancer invasiveness. This body of evidence supports a model in which bioenergy signals are transduced into coherent cellular responses, offering a plausible mechanism for BTs' system-wide effects (As illustrated in Fig. 3)

4.4. Biophotons as biomarkers: A new paradigm in conventional medicine

The scientific investigation of BTs has long been constrained by the challenge of quantifying the intervention's "dose" and the physical nature of the putative healing agent, often attributed to intention or subtle energy [43]. A significant methodological advance that overcomes this limitation is the application of ultraweak photon emission (UPE), or biophoton emission, as an optical biomarker to assess biofield interactions and other biological materials [8, 44–62]. Importantly, this foundational research is now catalyzing a transformative shift within conventional medicine itself.

Preliminary studies have sought to define the potential of UPE as a sensitive biophysical biomarker capable of detecting various physiological and pathological states, particularly in cancers, based on their distinct oxidative metabolic profiles [50, 52, 57–59]. As an intrinsic property of biological systems, ultraweak photon emission represents a promising candidate for the development of optical biopsy techniques aimed at monitoring reactive oxygen species (ROS) levels during metabolic processes in both normal and abnormal cells, tissues, and organisms [45, 47, 57–60]. Within this context, UPE has already demonstrated diagnostic utility in cardiovascular diseases, serving as a non-invasive biomarker [50]. Furthermore, research in animal

models have shown that UPE intensity from the body surface can significantly distinguish breast cancer-bearing mice from healthy controls, with emission levels varying in relation to tumor size [57]. Subsequent work by the same research group characterized the spectral features of UPE throughout the progression of human breast cancer in mice, comparing them to healthy controls [58]. These findings have been extended to human studies, where biophoton count and intensity measurements on the body surface offer a promising avenue for non-invasive health monitoring and early disease detection.

Building directly on this diagnostic potential, emerging research is actively exploring the same UPE metric not just for assessment, but as a direct tool for intervention. Investigations into UPE measurements applied during BTs are now creating a crucial methodological hook, offering a tangible way to quantify the dose of the intervention and objectively measure its bioeffects on the recipient's energy field. This bridges the historical gap, allowing researchers to move beyond subjective reports and begin rigorously testing the mechanisms and efficacy of these therapies using a quantifiable biophysical correlate [48, 49].

Kokubo et al. (2007) [48], aiming to establish a standardized protocol, utilized sections of cucumber (*Cucumis sativus*) as a biosensor system. Following a 5–30 minutes' non-contact intervention (administered by a Qigong master or via prayer), UPE was measured over 18 hours. The results demonstrated a significantly higher biophoton intensity in treated samples compared to controls ($p = 0.002$), an effect not observed in samples subjected to thermal stress (40°C water). Crucially, the authors proposed the index $J = \ln(IE/IC)$, the logarithm of the ratio of experimental to control emission intensity, as a quantitative metric for comparing healing efficacy across practitioners or techniques.

Kent et al. (2020) [49] using a mammalian cell model. Employing a light-tight chamber with a photomultiplier tube (PMT), they measured photon emission from TNF- α -stimulated rat intervertebral disc cells before, during, and after Reiki application. Their findings aligned with Kokubo's: Reiki-treated cells exhibited a significant increase in photon emission post-treatment relative to both baseline and sham controls ($p < 0.05$). Corroborating this biophysical data, molecular analysis revealed that Reiki significantly upregulated anabolic genes (Collagen II and Aggrecan; $p < 0.05$), key markers of extracellular matrix repair, thereby confirming a biologically relevant therapeutic effect.

Collectively, these studies provide three crucial contributions. They establish that differential post-treatment UPE serves as an indirect, objective, and replicable measure of BT dosage, applicable to both plant and mammalian systems, and open a scientific field for investigating the biophysical characteristics of UPEs in BTs, such as spectral length, coherence, and electromagnetic flux density. This paradigm shifts the conceptualization of BT from an unspecified energetic phenomenon to a potential photonic modulator with measurable biological outcomes.

4.5. Limitations

This analysis is not intended to be a systematic review of the entire literature. The selection of studies was intentional, not exhaustive. A fundamental selection criterion was the methodological rigor of the studies, which led to the inclusion of several key randomized controlled trials (RCTs). These studies were prioritized for their robust design and comprehensive biomarker panels, which remain highly relevant for in-depth exploratory analysis. Thus, this approach sacrifices temporal breadth to allow for detailed cross-comparison between methodological domains, ensuring that conclusions are drawn from a high-quality evidence base.

5. CONCLUSION

This review synthesizes evidence demonstrating that BTs consistently induce significant modulations across a diverse spectrum of biomarkers, confirming their ability to elicit objective physiological changes that go beyond the subjective effects of placebo. Notably, exploratory examination suggested that key moderators, such as participant sex and age, did not show consistent patterns for Immunological & Inflammatory biomarkers, highlighting that the therapeutic efficacy of BTs may be independent of these demographic variables. To date, based on the data extracted from the fifteen trials for this review,

research has been dominated by studies investigating human biomarkers (e.g., cortisol, sIgA, cytokines) and *in vitro* parameters (e.g., cell viability, gene expression), which robustly demonstrate biological responsiveness but largely neglect diagnostic applications.

Consequently, the current evidence base positions BTs primarily in purely prognostic frameworks, aiming to increase patient resilience and outcomes, and in prognostic-therapeutic contexts, where they act as adjuncts to disease management. This predominant focus highlights a critical gap and a compelling direction for future research: exploring the diagnostic potential of BTs. Investigating cellular, molecular, and, particularly, biophotonic parameters, such as individualized UPE signatures, could open a new frontier for BTs, transforming them from exclusively interventional tools into modalities capable of assessing health status, predicting risk, and informing personalized treatment strategies.

Abbreviations

BP

Blood Pressure

BMT

Bone Marrow Transplant

BT

Biofield Therapy

BTs

Biofield Therapies

CRP

C-Reactive Protein

ELISA

Enzyme-Linked Immunosorbent Assay

ERK1/2

Extracellular Signal-Regulated Kinase 1/2

FACS

Flow Cytometry (Fluorescence-Activated Cell Sorting)

FACT-B

Functional Assessment of Cancer Therapy - Breast

Hb

Hemoglobin

Ht

Hematocrit

IGF-I

Insulin-like Growth Factor I

LDH

Lactate Dehydrogenase

MFSI-sf

Multidimensional Fatigue Symptom Inventory - Short Form

MTT

Cell Metabolism Assay (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide)

NF-κB

Nuclear Factor Kappa B

NIPS

Neonatal Infant Pain Scale

NKCC

Natural Killer Cell Cytotoxicity

NKAUC

Natural Killer Activity Area Under the Curve

PARP

Poly (ADP-ribose) Polymerase

PCT

Procalcitonin

PCNA

Proliferating Cell Nuclear Antigen

PI3K

Phosphatidylinositol 3-Kinase

RBC

Red Blood Cells

RPV-Field

Resonant Phase Virtual Field

slgA
Secretory Immunoglobulin A
TERT
Telomerase Reverse Transcriptase
TUNEL
Terminal deoxynucleotidyl transferase dUTP Nick-End Labeling (Apoptosis) Assays
VAS
Visual Analog Scale
WBC
White Blood Cells

Declarations

CONFLICT OF INTEREST

The author declares no conflict of interest.

SUPPLEMENTARY MATERIAL

Supplementary material is available on the publisher's website along with the published article.

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Figures

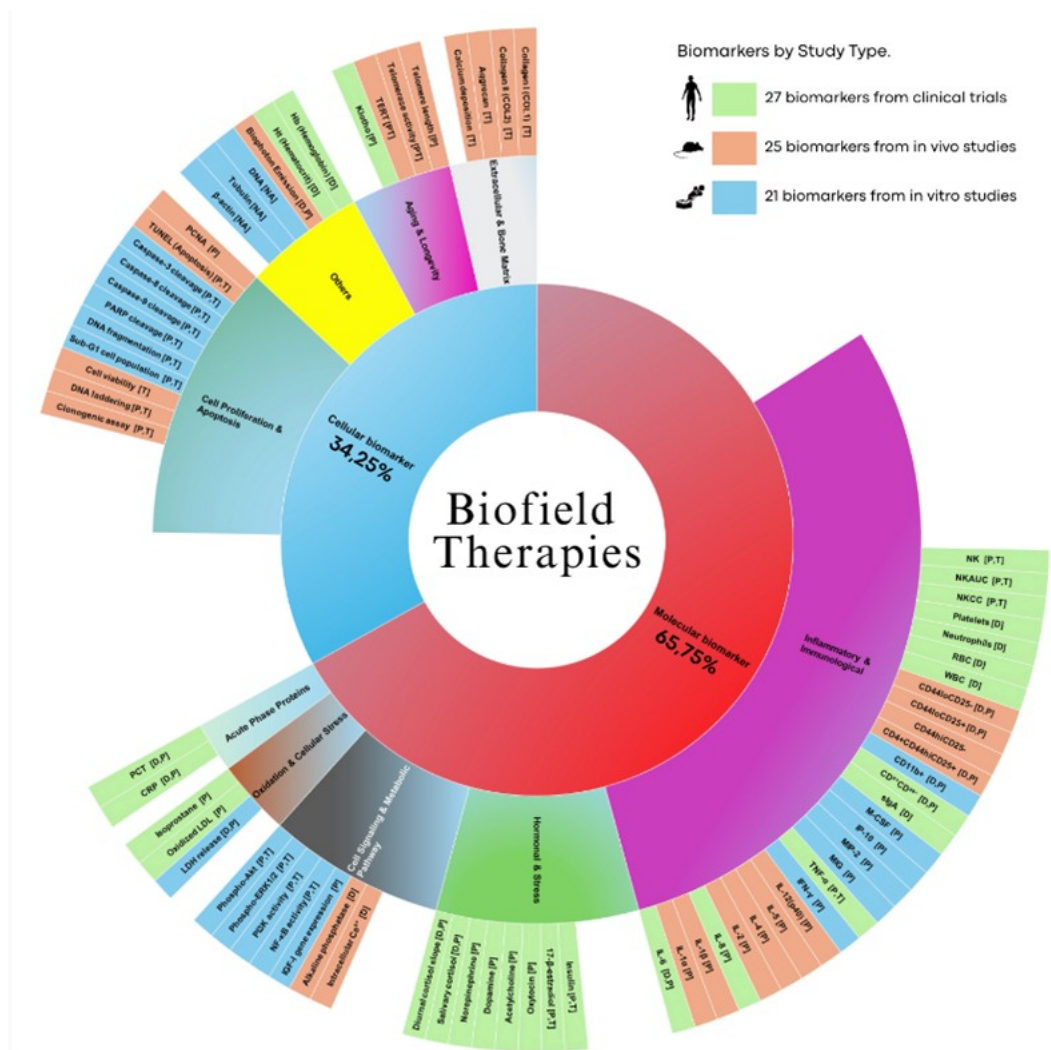


Figure 1

Sunburst representation of the hierarchy of biomarkers investigated in Biofield Therapy studies. Concentric levels represent main categories (internal), subcategories (intermediate), and individual biomarkers (external). Colors correspond to the classifications defined according to the methodology. Abbreviations: [P], Prognosis; [T], Therapeutic; [D], Diagnostic; [P, D], Prognosis & Therapeutic; [D, P], Diagnostic & Prognosis; [NA] = Not Applicable.

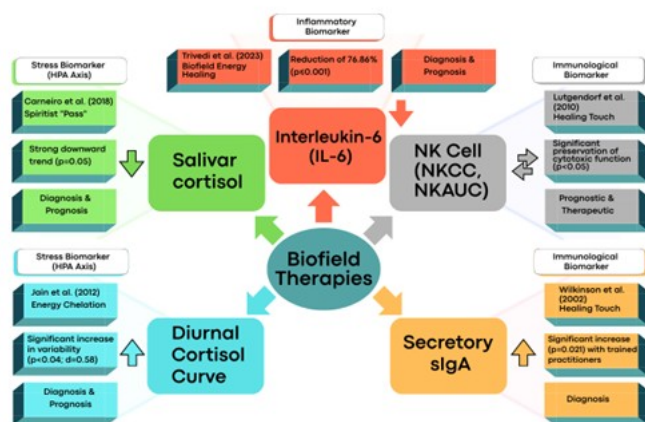


Figure 2

Modulation of stress and immunological biomarkers by Biofield Therapies in clinical studies. Data from selected studies demonstrate significant effects on the HPA axis (hypothalamic-pituitary-adrenal axis, e.g., cortisol) and immunological markers (e.g., IL-6 - Interleukin-6, cytotoxic function, slgA - Secretory Immunoglobulin A), suggesting applications in diagnosis, prognosis, and treatment.

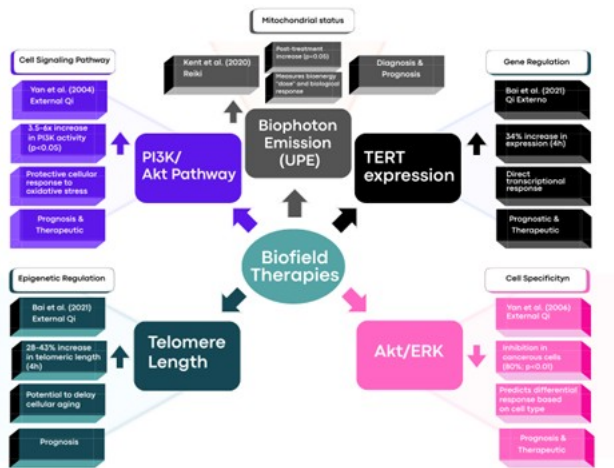


Figure 3

Molecular mechanisms modulated by Biofield Therapies in preclinical studies. Data demonstrate the modulation of cellular signaling pathways (e.g., PI3K - Phosphatidylinositol 3-kinase, Akt/ERK - Protein Kinase B / Extracellular Signal-Regulated Kinase), gene regulation (e.g., TERT - Telomerase Reverse Transcriptase), and mitochondrial parameters (e.g., UPE - Ultraweak Photon Emission), indicating potential applications in prognosis and therapy.