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OBM Integrative and Complementary Medicine

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Review

Biomarkers in Biofield Therapies: Analysis of Measurable Effects

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Academic Editor: Peta Stapleton

OBM Integrative and Complementary Medicine
2026, volume 11, issue 2
doi:10.21926/obm.icm.2602022

Received: April 02, 2026
Accepted: June 08, 2026
Published: June 24, 2026

Abstract

Biofield Therapies (BTs), such as Reiki and Healing Touch, are used in integrative and complementary medicine, but their biological mechanisms remain poorly understood. This review aimed to explore the physiological biomarkers modulated by BTs in a selected sample of studies to identify common patterns and elucidate potential mechanisms of action. A focused narrative synthesis of 14 intentionally selected studies was performed, encompassing various BT modalities, study designs (including randomized clinical trials, preclinical studies, and *in vitro* models), and biomarker types. A total of 64 biomarkers were extracted and classified based on type, clinical application, and biological function. Data synthesis included descriptive statistics to summarize the distribution of biomarker categories within this sample. In the RCTs, the analysis of the 17 biomarkers pointed to diagnostic and diagnostic-prognostic contexts, with a relevant subset of prognostic-therapeutic biomarkers (23.5%) suggesting an adjuvant role in predicting clinical outcomes and in therapeutic monitoring. The most frequently reported biomarkers in the selected studies were inflammatory and immunological (40.6%), followed by cellular regulatory markers. The main findings included evidence of modulation of the hypothalamic-pituitary-adrenal (HPA) axis, enhanced immune function (Natural Killer (NK) cell activity), cytoprotective effects in *in vitro* models, and exploratory observations of increased telomere length. This synthesis suggests that BTs are associated with measurable physiological changes in multiple systems, reinforcing the need for further mechanistic investigations. Future research should prioritize investigations into the diagnostic

and prognostic potential of BT in oncological contexts, as well as the development of individualized biophotonic signatures to promote personalized, rapid, and non-invasive applications.

Keywords
Biofield therapies; biomarkers; neuroimmune modulation; Ultra-weak Photon Emission (UPE)

1. Introduction

The term *biomarker* has undergone substantial conceptual evolution since its initial characterization as a biochemical or biological marker [1]. Formally introduced in 1973 by Rho and colleagues to denote the presence or absence of specific biological material, the concept has since matured into a cornerstone of contemporary biomedical science [2]. According to the U.S. Food and Drug Administration (FDA), a biomarker is defined as a measurable indicator with 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 are now critical components of modern research, enabling precise quantification of physiological processes, disease mechanisms, and treatment responses [4].

In parallel, the term *Biofield* has been proposed in biomedical contexts based on traditional medicines, as a conceptual construct to describe integrated patterns of biophysical activity that may include bioelectrical, biochemical, and electromagnetic components [5-9]. This construct serves as the foundational framework for a distinct class of interventions known as Biofield Therapies (BTs). Recognized by the Division of Cancer Treatment and Diagnosis (DCTD/NCI) of the National Cancer Institute as energy or healing therapies, this category includes practices such as Reiki, Therapeutic Touch, Healing Touch, Spirit “Passe”, and External Qigong [9]. These therapies are increasingly studied within complementary and integrative medicine, particularly within the subfield of energy medicine. Empirical observations of practitioners reportedly transferring internal bioenergy to produce therapeutic effects have motivated hypotheses about biophysical mechanisms; however, the nature of these mechanisms remains speculative and requires rigorous investigation [8].

Global health organizations, including the World Health Organization (WHO), have increasingly acknowledged the importance of scientifically evaluating Traditional, Complementary, and Integrative Medicine [10]. The clinical evidence base for BTs is still developing and contains mixed findings [11]; nonetheless, a growing number of studies employ objective biomarkers to explore potential biological correlates of these interventions. These studies, while heterogeneous in design and scope, provide a measurable basis for generating testable hypotheses about BT mechanisms.

This review pursues three main objectives: (1) to synthesize biomarker data from a purposively selected, methodologically diverse set of studies on BTs, categorizing findings by study design (RCTs, animal models, and *in vitro* experiments); (2) to classify biomarkers according to a clinical taxonomy (diagnostic, prognostic, therapeutic, and combinations) applied exclusively to RCTs in humans, recognizing the conceptual limitations of extending such classification to preclinical studies; and (3) to identify patterns and promising candidate biomarkers, including inflammatory markers, HPA axis

indicators, and cell-signaling markers, that warrant further investigation in larger, rigorously designed clinical trials. Based on these findings, we offer a perspective on emerging biophysical measures, including ultraweak photon emission (UPE), as potential tools for mechanistic and dose-response research. It is emphasized that all conclusions should be interpreted within the limitations of a purposive, non-systematic narrative synthesis.

2. Methods

2.1 Review Design and Rationale

This study employs an exploratory narrative synthesis focused on a purposively selected set of studies (n = 14) investigating biomarker patterns in BT research. This design was intentionally chosen to capture methodological diversity across biofield modalities (Reiki, Therapeutic Touch, Qigong) and study types (RCTs, preclinical, *in vitro*). Critically, this is not a systematic review, and the purposive sampling approach precludes claims of exhaustive or definitive literature mapping. Selection bias is an inherent limitation of this approach and is explicitly acknowledged. Accordingly, quantitative summaries (e.g., biomarker frequencies) reflect only this specific sample and cannot be generalized to the broader BT research literature. The primary objective is to provide a cross-sectional comparison of biomarker patterns to guide hypothesis generation and future research design.

2.2 Study Selection, Data Extraction, and Analysis

Studies were included if they: (1) evaluated a defined BT modality; (2) reported empirical data on at least one quantifiable molecular or cellular biomarker; and (3) utilized a controlled experimental design. Studies relying exclusively on physiological or psychometric outcomes, without molecular or cellular biomarkers, were excluded.

All analyses were explicitly exploratory, given the purposive study selection and heterogeneous biomarker landscape. Data organization, descriptive statistics, and categorical synthesis were performed using *Microsoft Excel*. To explore potential moderating effects of key demographic variables (mean age and percentage of female participants) on reported biomarker outcomes, an exploratory moderator analysis (meta-regression) was conducted using the *R software environment* (version 4.5.2 for *Windows*, 2025). A Sunburst diagram was constructed to visually represent the hierarchical classification of biomarkers across the included BT studies.

It is acknowledged that the study selection was not based on a pre-specified, systematic search protocol. Potential sources of selection bias include the non-exhaustive search, the intentional focus on methodological diversity, and the limited number of RCTs available in this field. These limitations are discussed further in Section 5.

2.3 Biomarker Classification Framework

Biomarker classification was based on a specialized three-tiered framework integrating established regulatory guidelines, physiological knowledge, and study-specific context. Instead of adopting fully automated categories generated by the platform, formal definitions and consensus frameworks from regulatory bodies (FDA, NIH) and the MarkerDB 2.0 database were applied [4, 12], aligning with standardized nomenclature.

Biomarkers were systematically classified by: (a) Type (Molecular or Cellular Biomarkers), following the classification adopted by Bodaghi et al. [1], which provides a comprehensive and widely cited framework for biomarker typology in the context of disease diagnosis, prognosis, and treatment monitoring; and (b) Biological Function (e.g., inflammatory, hormonal, metabolic; see Table S1 for complete list). It is acknowledged that this framework was custom-developed for this synthesis rather than derived from a formally validated taxonomy such as the FDA-NIH BEST Resource. Accordingly, inter-rater reliability was not formally established, which constitutes a limitation. Future work should consider formal validation of this classification system against established standards.

According to clinical guidelines, biomarkers from RCTs were categorized into five primary types of clinical application [1-3]:

- 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.

This clinical classification was applied exclusively to biomarkers from RCTs, in which clinical outcomes and disease trajectories are conceptually relevant. For animal and *in vitro* studies, this clinical taxonomy is not applicable, as these models lack the clinical context (diagnosis, prognosis in the human sense) required for such classification. Biomarkers from animal and *in vitro* studies were therefore classified only by Type (Molecular/Cellular) and Biological Function (e.g., Inflammatory, Cell Proliferation), and are reported as "Not Applicable (NA)" for clinical subcategories.

Study design (human, animal, or *in vitro*) and experimental context were incorporated to refine classifications based on methodological relevance. This structured approach was designed to ensure a reproducible, biologically logical, and context-aware categorization system, facilitating cross-study comparisons.

3. Results

3.1 Study and Participant Characteristics

The selected cohort comprised 14 studies encompassing a diverse range of BTs: Reiki (n = 2), Therapeutic Touch (n = 2), Healing Touch (n = 3), Spirit "Passe" (n = 1), External Qigong (n = 2), and other modalities (n = 4). Of these, five were RCTs, constituting 35.71% of the included studies, pooling a total of 189 human participants (experimental group: n = 99; control group: n = 90). Six studies (42.86%) were conducted exclusively *in vitro*, one (7.14%) employed a preclinical animal model exclusively, one (7.14%) combined *in vitro* and *in vivo* designs within the same study, and one (7.14%) was a quasi-experimental mixed-methods design involving human participants.

From each eligible study, data were extracted into a standardized coding spreadsheet capturing: (1) first author and year of publication; (2) BT modality; (3) sample size (n); (4) study design (type, duration, number of sessions, distance from subject); (5) control group conditions; (6) dose; (7) type of intervention; (8) biomarkers assessed; (9) biomarker results; (10) clinical outcomes; and (11) observations (see Table S1).

3.2 Biomarker Characteristics and Clinical Classification

A total of 64 biomarkers were evaluated across the 14 studies: 40 molecular (62.50%) and 24 cellular (37.50%). Of these, 17 biomarkers (26.56%) were identified in the 5 RCTs and were eligible for clinical subclassification. The remaining 47 biomarkers (73.44%) were derived from animal (*in vivo*) and *in vitro* studies and are reported as "Not Applicable (NA)" for clinical subcategories (see Supplementary Materials S2).

Among the 17 RCT-derived biomarkers, the distribution by clinical purpose was as follows: Diagnostic only (n = 7; 41.2%), Diagnostic & Prognostic (n = 5; 29.4%), Prognostic & Therapeutic (n = 4; 23.5%), Prognostic only (n = 1; 5.9%), and Therapeutic only (n = 0; 0%). These percentages reflect the specific subset of biomarkers measured in RCTs and should not be generalized to the broader set of 64 biomarkers or to preclinical studies. The hierarchical relationships and relative frequencies of these biomarkers are visually summarized in the Sunburst plot (Figure 1).

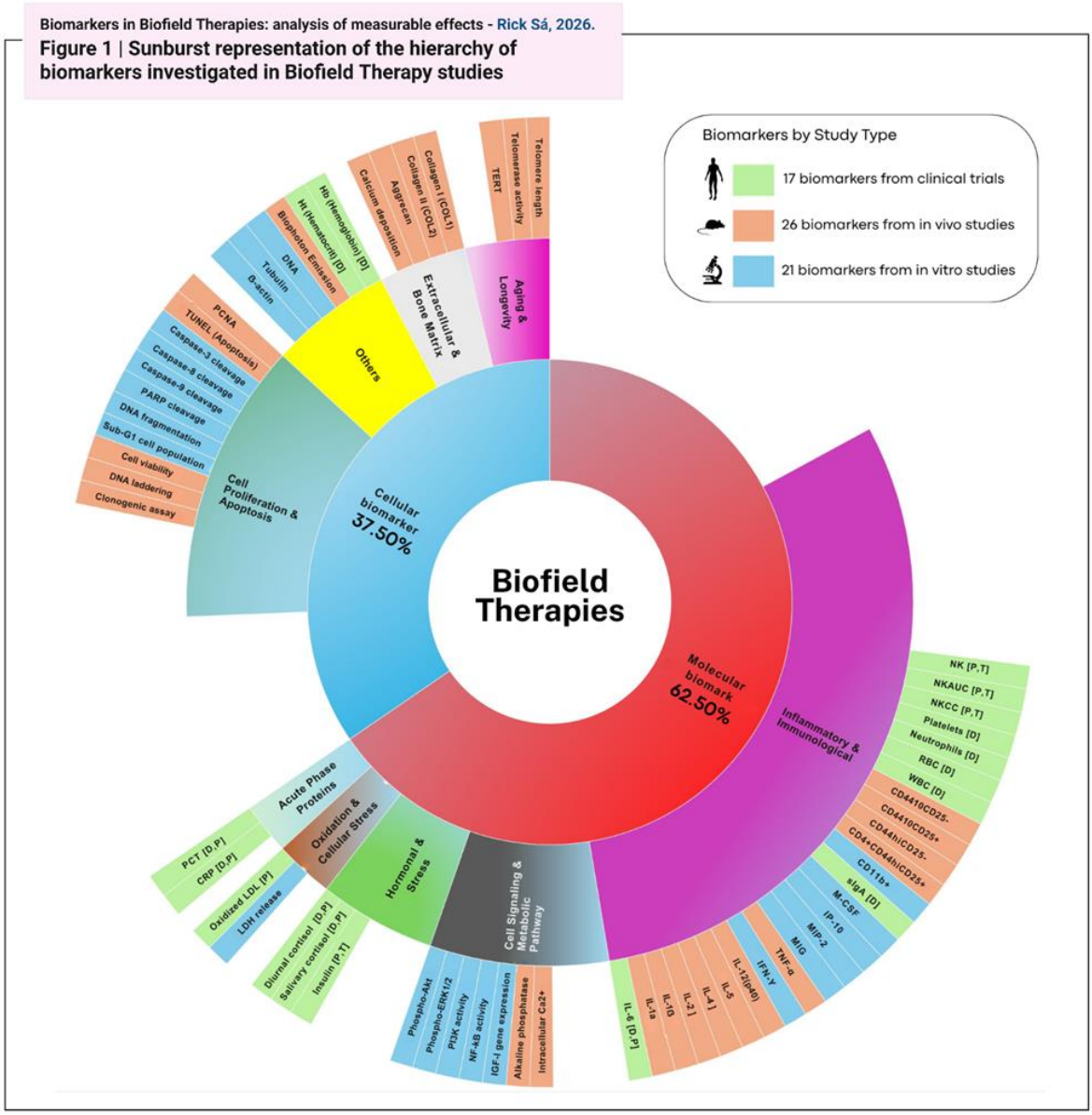

Figure 1 Sunburst representation of the hierarchy of biomarkers investigated in Biofield Therapy studies. Concentric levels represent the main (internal) categories,

subcategories (intermediate), and individual (external) biomarkers. The colors correspond to the classifications defined according to the methodology. Abbreviations: [P], Prognostic; [D], Diagnostic; [D, P], Diagnostic & Prognostic; [P, T], Prognostic & Therapeutic. Note: The clinical classification shown in this figure was applied only to the 17 biomarkers from RCTs (Randomized Clinical Trials). For animal and *in vitro* biomarkers (n = 47), the categories reflect their biological function (Inflammatory, Cell Proliferation, Hormonal) and are interpreted as not applicable (NA) for clinical subcategories. *Note*: For practical visualization purposes, biomarkers from studies with an *in vitro* component are grouped in this figure, combining the *in vitro* (exclusive) and mixed *in vitro*/*in vivo* categories. Among the three biomarkers identified in this hybrid study—telomere length, TERT expression, and telomerase activity—only the *in vivo* measurement was considered. The corresponding *in vitro* (cellular) measurements are not detailed separately in this figure. See the study design column of Table S1 for the complete distinction between exclusively *in vitro* studies, the exclusively animal preclinical study, and the mixed *in vitro*/*in vivo* design of this study.

When classified by biological function across all 64 biomarkers, the majority were related to Inflammatory & Immunological processes (n = 26; 40.6%), followed by Cell Proliferation & Apoptosis (n = 11; 17.2%), Cell Signaling & Metabolic Pathways (n = 7; 10.9%), and Extracellular & Bone Matrix Remodeling (n = 4; 6.3%). Smaller categories included: Hormonal & Stress (n = 3; 4.7%), Aging & Longevity (n = 3; 4.7%), Oxidation & Cellular Stress (n = 2; 3.1%), and Acute Phase Proteins (n = 2; 3.1%), with six additional biomarkers (9.4%) classified as Other (see Table 1).

Table 1 Characteristics of Included Studies, Participants, and Biomarkers.

Object	Total (n)	Proportion (%)	Proportion of biomarkers per study (%)
Studies	14	100.00	-
RCTs	5	35.71	26.56 (17 human)
In vitro (exclusive)	6	42.86	28.12 (18 *in vitro*)
Preclinical Animal (exclusive)	1	7.14	40.62 (26 animal)
Mixed *In Vitro*/*In Vivo*	1	7.14	4.69 (3 mixed)
Mixed Methods (quasi-experimental, human)	1	7.14	N/A (Included in the Humans/RCTs category)
Humans in the studies	189	100.00	-
Men	49	25.93	-
Men Age (Mean)	≈31.11	-	-
Women	140	74.07	-
Women Age (Mean)	≈43.87	-	-
Experimental group	99	52.38	-
Control group	90	47.62	-
Biomarkers	64	100.00	-
Molecular	40	62.50	-
Cellular	24	37.50	-

Diagnostic (D) only	7	41.10	-
Prognostic (P) only	1	5.98	-
Therapeutic (T) only	0	0	-
Diagnostic and Prognostic (D, P)	5	29.5	-
Prognostic and Therapeutic (P, T)	4	23.42	-
|NA|	47	73.44	-
Subcategory			
Inflammatory and Immunological	26	40.6	-
Hormonal and Stress	3	4.7	-
Oxidation and Cellular Stress	2	3.1	-
Cell Proliferation and Apoptosis	11	17.2	-
Cell Signaling and Metabolic Pathway	7	10.9	-
Extracellular and Bone Matrix	4	6.3	-
Aging and Longevity	3	4.7	-
Acute Phase Proteins	2	3.1	-
Others	6	9.4	-

Characteristics of Included Studies, Participants, and Biomarkers. The table summarizes the distribution of study designs (14 studies in total), participant demographics (189 individuals in total), and classification of identified biomarkers (64 biomarkers in total).

3.3 Participant Demographics

From the five RCTs, data were pooled from 189 human participants. The sample was predominantly female (n = 140; 74.1%), with men comprising 25.9% (n = 49). Mean age varied considerably across studies, ranging from young adults (~20 years) to middle-aged individuals (~50 years). One RCT included preterm newborns, admitted to the nursery between 2 and 15 days of life.

3.4 Exploratory Moderator Analysis

Among the 17 biomarkers identified in the RCTs, the most frequently assessed were markers of stress and inflammation, including salivary cortisol (or its diurnal variability) [13-15], neutrophil percentage [16], and natural killer (NK) cell percentage [17]. These biomarkers were reported in four of the five RCTs, allowing for an exploratory moderator meta-regression to examine whether demographic variables (mean age and sex distribution) influenced the direction or magnitude of reported effects.

The exploratory moderator meta-regression revealed no statistically significant moderation of effect size by either mean age ($\beta = -0.020$, SE = 0.021, $z = -0.964$, $p = 0.335$; 95% CI: -0.061 to 0.021) or percentage of female participants ($\beta = -0.006$, SE = 0.007, $z = -0.823$, $p = 0.411$; 95% CI: -0.020 to 0.008). Full model statistics are reported in Table 2.

Table 2 Exploratory Moderator Analysis of Stress and Immune Biomarkers in Four RCTs of Biofield Therapies.

Parameter	Moderator: Mean Age	Moderator: % Female
Model Statistics		
Number of studies (n)	4	4
Residual heterogeneity (τ^2)	0.1723	0.1966
I^2 (%)	62.68	65.95
H^2	2.68	2.94
Test for Residual Heterogeneity		
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
Test of Moderators		
QM (df)	0.93 (1)	0.68 (1)
p-value	0.335	0.411

Results of the moderator analysis (meta-regression) for demographic variables: Average Age and Percentage of Female Participants. The table presents model statistics, including residual heterogeneity and significance tests for the moderators.

Importantly, these results must be interpreted with extreme caution. With only four studies, the meta-regression is severely underpowered: the statistical power to detect a medium-sized moderator effect ($f^2 = 0.15$) at $\alpha = 0.05$ with $k = 4$ studies is below 0.30, far below the conventionally recommended threshold of 0.80. Accordingly, the non-significant moderator coefficients should not be interpreted as confirmatory evidence that age and sex do not influence BT treatment response; a Type II error cannot be excluded. The high residual heterogeneity observed in both models (I^2 = 62.68% for age; I^2 = 65.95% for sex) indicates that a substantial proportion of between-study variance remains unexplained by the demographic variables examined, which is unsurprising given the heterogeneity of biomarkers, BT modalities, and clinical populations across the four studies. The non-significant tests of moderators (QM_{age} (1) = 0.93, p = 0.335; QM_{sex} (1) = 0.68, p = 0.411) are consistent with this interpretation.

To support methodological transparency, the raw group-level data and individual effect sizes underlying the meta-regression are presented later in this work. Effect sizes were computed as Cohen's d using the pooled standard deviation weighted by degrees of freedom:

$$D_{pooled} = \sqrt{\frac{(n_1 - 1)SD_1^2 + (n_2 - 1)SD_2^2}{n_1 + n_2 - 2}} \quad (1)$$

$$d = \frac{M_{exp} - M_{ctrl}}{SD_{pooled}} \quad (2)$$

This approach, consistent with the implementation available in the *metafor* package for *R*, provides a more stable pooled variance estimate than the unweighted alternative when group sizes differ, as was the case for Jain et al. [13] (n_{exp} = 27; n_{ctrl} = 19). All four studies yielded positive effect sizes, indicating higher biomarker values in the experimental group relative to the control group. The largest effect was observed in Jain et al. [13] (d = 0.736), assessing diurnal cortisol slope in breast cancer survivors receiving Energy Chelation, followed by Lutgendorf et al. [17] (d = 0.400), which measured the NK cell percentage during chemoradiation with Healing Touch. Smaller effects were observed for Running et al. [15] (d = 0.283), assessing salivary cortisol in university students, and Akpinar et al. [16] (d = 0.247), measuring neutrophil percentage in bone marrow transplant patients receiving Reiki. By conventional benchmarks, three effect sizes fall within the small-to-medium range (0.20 ≤ d < 0.50), while Jain et al. [13] approach a medium effect (d = 0.736). The data are reported in Table 3.

Table 3 Raw Data and Effect Sizes for Stress and Immune Biomarkers in Four RCTs of Biofield Therapies.

Study	Biomarker	Control Group			Experimental Group			SD Pooled (Cohen's d)	Effect Size (d)	Mean Age (years)	Female (%)
		Mean	SD	n	Mean	SD	n				
Akpinar et al. (2025) [16]	Neutrophils	2.70	0.83	21	3.20	2.74	21	2.024	0.247	34.4	28.6%
Jain et al. (2012) [13]	Diurnal Cortisol Slope	1.10	0.86	19	1.80	1.01	27	0.951	0.736	52.0	100%
Lutgendorf et al. (2010) [17]	NK Cell %	7.68	6.33	17	9.90	4.63	17	5.546	0.400	48.1	100%
Running et al. (2022) [15]	Salivary Cortisol	0.18	0.15	21	0.23	0.20	21	0.177	0.283	20.3	76.2%

The table presents, for each study, the group-level descriptive statistics (mean and standard deviation) for both control and experimental groups, the pooled standard deviation (SD Pooled), and the individual effect size (Cohen's d), alongside the demographic moderators examined in the meta-regression (mean age and percentage of female participants).

4. Discussion

4.1 Overview of Biomarker Evidence

The analysis of biomarkers across the 14 included studies reveals a multifaceted research landscape aimed at objectively characterizing the potential biological effects of BTs. When examined as a whole, the 64 biomarkers span molecular and cellular levels, with a notable concentration in inflammatory, immunological, and cell-proliferation pathways. A more refined picture emerges when the clinical classification framework (Diagnostic, Prognostic, and Therapeutic) is applied exclusively to the 17 biomarkers derived from RCTs, for which these categories are conceptually valid. All interpretations below are qualified by the exploratory and preliminary nature of the evidence.

Among the 17 RCT-derived biomarkers, the most frequent categories were Diagnostic and Diagnostic & Prognostic. The prominence of purely diagnostic biomarkers, including hemoglobin, hematocrit, leukocytes, erythrocytes, neutrophils, platelets, and secretory IgA, likely reflects the routine hematological and immunological monitoring commonly employed in clinical trials rather than deliberate design to validate BTs as diagnostic tools per se. However, the presence of five biomarkers classified as Diagnostic & Prognostic (procalcitonin, C-reactive protein, interleukin-6, diurnal cortisol slope, and salivary cortisol) suggests that some researchers explored how BT-induced biological alterations may contribute both to disease detection and to predicting clinical trajectories.

The identification of Prognostic & Therapeutic biomarkers (insulin, NKCC, NKAUC, and %NK; 23.5% of RCT biomarkers) represents a conceptually meaningful advance. Rather than merely asking whether BTs produce any biological effect, these studies examine markers that both predict clinical outcomes and serve as potential targets for therapeutic monitoring. This shift is particularly notable in oncology, where the preservation of NK cell cytotoxicity during chemoradiation has been interpreted as a favorable prognostic indicator and a candidate therapeutic target [17]. However, these interpretations remain tentative: the small number of RCT biomarkers (n = 17) and the considerable heterogeneity of clinical populations, ranging from breast and cervical cancer patients to bone marrow transplant recipients, preterm newborns, and healthy university students, substantially limit the generalizability of these findings.

Functional categorization further clarifies the strategic focus of the field. The high frequency of inflammatory and immunological markers (IL-6, IL-1β, TNF-α, sIgA, leukocytes, red blood cells, NK cells, neutrophils and platelets) [16-20], highlights the central research interest in modulating immunoinflammatory responses. This emphasis is clinically sensible given the widespread recognition of these biomarkers as indicators of disease status and therapeutic response, and their practical accessibility in serum and saliva samples.

4.2 Stress Response and Neuroimmunomodulation

BTs appear to engage psychoneuroimmunological (PNI) mechanisms [21-30], with evidence suggesting potential modulation of the hypothalamic-pituitary-adrenal (HPA) axis. While the HPA axis appears to be a plausible pathway in these studies, it is important to note that it is not the only neuroendocrine mediator of the stress response [31, 32]. The sympathetic nervous system (SNS), particularly through catecholamine release (epinephrine, norepinephrine), is a major and rapid

mediator of neuroendocrine-immune communication [32]. The SNS directly innervates lymphoid organs and modulates immune cell activity, contributing to the physiological changes observed in stress contexts. Future research should aim to disentangle the relative contributions of the HPA axis and the SNS to the effects observed following BT interventions, for example, by simultaneously measuring salivary alpha-amylase (a marker of SNS activity) alongside cortisol.

Salivary cortisol findings from the available RCTs are suggestive. A double-blind study of preterm infants receiving Spiritist "passe" showed a trend toward reduced cortisol ($p = 0.05$), consistent with possible HPA modulation. However, the marginal statistical significance and small sample size preclude firm conclusions [14]. In breast cancer survivors, Energy Chelation was associated with a steeper diurnal cortisol slope ($p < 0.04$; $d = 0.58$), suggesting enhanced HPA axis regulation; however, the modest sample size and the absence of practitioner blinding represent important caveats [13]. Collectively, these observations are consistent with BTs potentially modulating neuroendocrine stress pathways, but independent replication in adequately powered trials is essential (as illustrated in Figure 2).

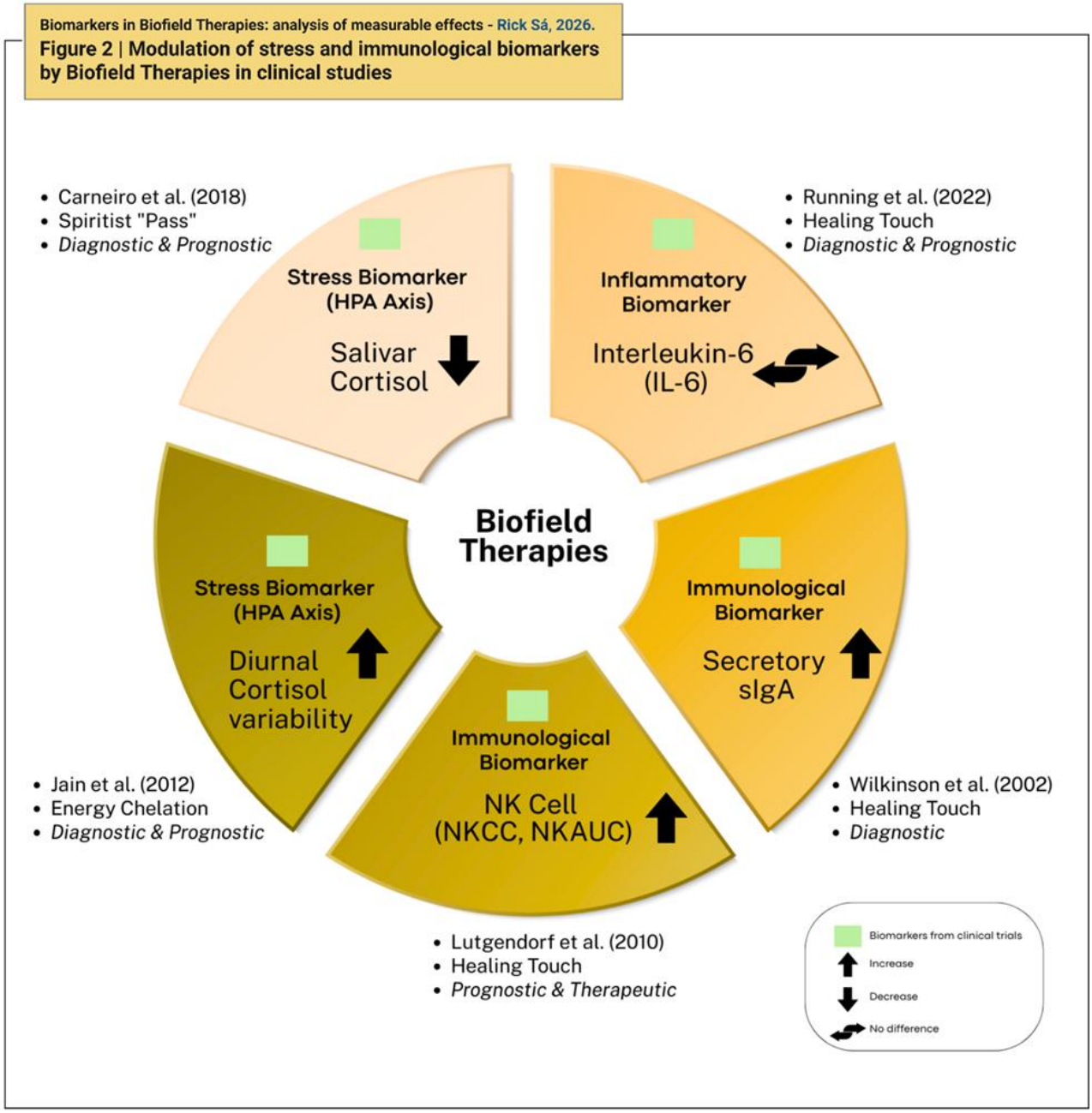

Figure 2 Modulation of stress and immunological biomarkers by Biofield Therapies in clinical studies. Data from selected studies demonstrate significant effects on the HPA axis (hypothalamic-pituitary-adrenal axis, e.g., cortisol) and immunological markers (e.g., IL-6 - Interleukin-6, cytotoxic function, sIgA - Secretory Immunoglobulin A), suggesting applications in diagnosis, prognosis, and treatment.

At the immunological level, HPA-axis modulation may translate into functional preservation of immune responses. During cervical cancer chemoradiation, Healing Touch was associated with maintenance of NK cell cytotoxicity (group-time interaction $p < 0.05$), consistent with PNI principles linking neuroendocrine regulation to immune function under stress [17, 25, 26]. Similarly, increased secretory IgA after Healing Touch ($p = 0.021$) may reflect a shift toward parasympathetic dominance, supporting mucosal immunity [19]. These findings are consistent with a model in which BTs may initiate relaxation and HPA axis regulation, potentially preserving immune cell function under stress,

reducing the inflammatory burden, and improving mucosal immunity. However, caution is warranted given the small and heterogeneous evidence base.

The absence of statistically significant moderation by age or sex (Table 2) in the exploratory analysis is noteworthy. However, this null result must be interpreted in light of the severe limitations of a four-study meta-regression. Larger, adequately powered clinical trials with pre-specified demographic stratification are required to determine whether treatment response varies across age and sex groups.

4.3 Gene Expression, Molecular Signaling, and Cellular Effects

In addition to systemic neuroendocrine effects, a body of *in vitro* and preclinical evidence suggests that BTs may influence gene expression and cellular signaling. These findings should be interpreted as exploratory, given the significant methodological challenges in this area, including the difficulty of blinding, the absence of independent replication in most studies, and the uncertain extrapolability of *in vitro* findings to clinical populations.

In primary human osteoblasts (HOBs), Therapeutic Touch (TT) treatment was associated with increased DNA synthesis and promotion of extracellular matrix mineralization, alongside elevated gene expression of type I collagen, bone sialoprotein, and alkaline phosphatase [33]. In contrast, in SaOs-2 osteosarcoma cells, the same intervention was associated with reduced mineralization and expression of these markers. These results suggest cell-type-specific effects; however, independent replication is required before mechanistic conclusions can be drawn.

In primary rat retinal neurons, brief exposure to External Qi (Yan Xin) before oxidative insult activated the PI3K-Akt survival pathway and upregulated IGF-I mRNA, conferring resistance to apoptosis [18]. In human pancreatic cancer cells, Yan Xin Qigong suppressed survival pathways (Akt, ERK, NF-κB) and promoted apoptosis. In contrast, normal fibroblasts showed transient pathway activation without cell damage [34] (as illustrated in Figure 3). While these bidirectional, cell-type-specific effects are mechanistically intriguing, they require independent replication and should not be interpreted as evidence of an established mechanism.

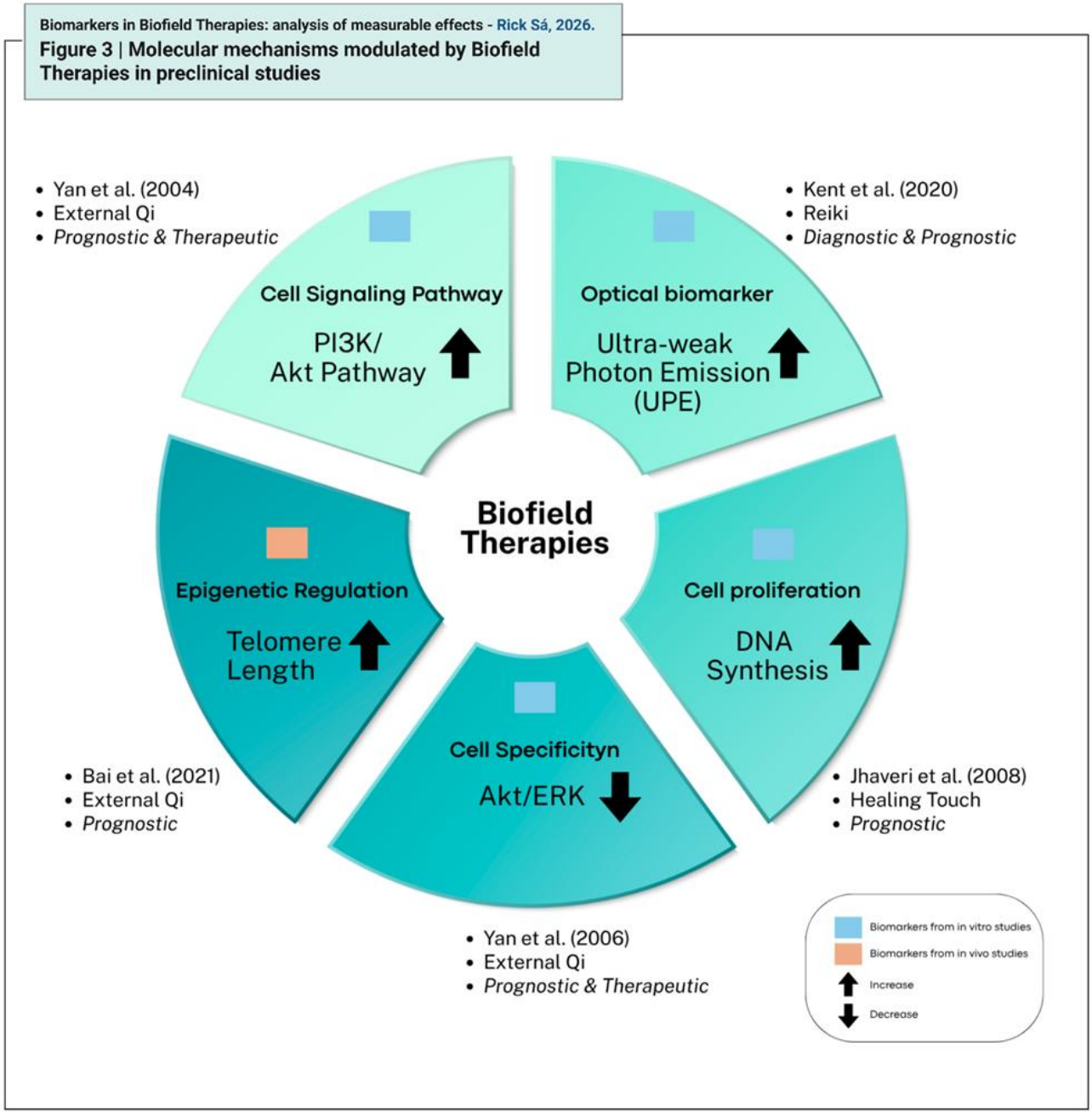

Figure 3 Molecular mechanisms modulated by Biofield Therapies in preclinical studies. Data demonstrate the modulation of cellular signaling pathways (e.g., PI3K - Phosphatidylinositol 3-kinase, Akt/ERK - Protein Kinase B/Extracellular Signal-Regulated Kinase), gene regulation (e.g., TERT - Telomerase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

Preclinical *in vivo* studies at MD Anderson Cancer Center reported that exposure to a putative biofield emitter was associated with reduced Lewis lung carcinoma tumor growth in mice, alongside PI3K/Akt/mTOR downregulation, immune landscape remodeling (increased CD8+ T-cell infiltration, reduced PD-L1 expression), and in a follow-up study using longer sessions a fourfold increase in necrotic area and reduction of cancer stemness marker SOX2 by 33% [35, 36]. These findings are mechanistically provocative; however, the use of a single putative biofield emitter, the absence of

independent replication, and the inherent challenges of biofield research methodology substantially limit their interpretability.

The application of external Qi and other methods to rat kidney cells was associated with increases in telomere length of 28 to 43% within 4 hours, via TERT-dependent and TERT-independent pathways [37]. These findings require cautious interpretation for several reasons, based on established telomeric biology and the data reported in the study itself. According to canonical mechanisms, telomere elongation is a replication-dependent process, driven by telomerase activity, that occurs gradually over multiple cell cycles; a timescale of hours is not consistent with sustained elongation through known enzymatic pathways [38, 39]. Furthermore, the authors themselves report that the study demonstrates a correlation but does not prove causality due to limitations in experimental design and transient effects, and explicitly call for further research to elucidate the underlying mechanisms [37].

Similarly, Bengston Energy Healing applied to PANC-1 pancreatic cancer cells was associated with selective alterations in Ca^{2+} homeostasis and a dramatic reduction in invasiveness [40]. In contrast to the Bai et al. [37] findings on telomere length, which, as noted above, do not establish causal directionality, the MD Anderson research program from which this finding derives has progressively developed more rigorous causal frameworks for BT effects on cancer cell biology. A simultaneous electrophysiological and cellular assessment study conducted under double-blind conditions demonstrated bidirectional Granger-causal associations between the BT practitioner's EEG activity and Ca^{2+} dynamics in PANC-1 cells, suggesting that the observed cellular changes were not merely coincident with, but directionally linked to, measurable practitioner physiological states [40]. Importantly, this causal analysis relies on statistical directionality (Granger causality) rather than experimental manipulation, and therefore does not establish mechanistic causation in the biological sense; nonetheless, it represents a methodological advance over purely correlational *in vitro* designs.

Building on this foundation, a more recent and comprehensive preclinical study conducted with three independent BT practitioners using a pre-registered, sham-controlled protocol provided the first systematic characterization of candidate molecular and bioelectrical mechanisms underlying BT effects on pancreatic ductal adenocarcinoma [41]. Across multiple human and mouse PDAC cell lines and patient-derived organoids, BT consistently suppressed proliferation and invasiveness, induced G0/G1 cell-cycle arrest, and induced marked mitochondrial ultrastructural changes. At the molecular level, BT significantly downregulated FOXM1, a master regulator of cell cycle progression, metastasis, and stemness in pancreatic cancer, along with its signaling-loop partners ALKBH5, cyclin B1, and CDK1, while upregulating the tumor suppressor p21. Critically, FOXM1 knockout abrogated BT-induced cell cycle arrest and anti-invasive effects, whereas FOXM1 overexpression augmented them, providing mechanistic evidence that FOXM1 downregulation is at least partially responsible for the observed phenotypic changes. At the bioelectrical level, BT reduced resting membrane voltage potential in PANC-1, MiaPaCa-2, and KPCY cells, consistent with cancer cell hyperpolarization, a bioelectrical state associated with suppression of the metastatic phenotype. In orthotopic mouse models, BT significantly reduced liver metastasis of both PANC-1 and KPCY cells across two independent studies, with effects on liver tumor burden comparable to gemcitabine in one study. While these findings collectively represent the most methodologically rigorous and mechanistically detailed preclinical evidence on the effects of BT on cancer biology to date, several important limitations remain: the two studies mentioned above used a single BT modality (Bengston Method), independent replication by external research groups is not yet available, the physical

mechanism by which BT exerts these effects remains unknown, and the extrapolation of these *in vitro* and murine findings to human clinical outcomes is uncertain. The authors themselves acknowledge that potential mechanisms, including biophoton emissions and electromagnetic fields, were not directly assessed and warrant systematic investigation in future studies.

4.4 Ultraweak Photon Emission as a Candidate Biomarker

The scientific investigation of BTs has long been constrained by the challenge of quantifying both the intervention's dose and the physical nature of the putative therapeutic agent [42]. Ultraweak Photon Emission (UPE), or biophoton emission, has been associated as a possible biophysical biomarker in living systems [10, 43-63]. However, the application of UPE as a BT dose metric remains conceptually ambitious and exploratory.

Biophotons are defined as ultraweak photons emitted spontaneously or elicited by living systems, resulting from the radiative decay of electronically excited states, generated primarily by reactive oxygen species (ROS) and possibly by quantum processes consistent with biomolecular arrangements [43]. In the context of biocommunication, the hypothesis is that biophotons can be considered not as random noise, but as potential carriers of information whose coherence, spectral distribution, and temporal patterns can encode physiological states and mediate intercellular or interorganismal signaling [63]. It must be emphasized, however, that this information-carrier hypothesis, while conceptually compelling, remains unproven and constitutes a frontier research question rather than an established biophysical mechanism.

From a physical standpoint, UPE is a well-documented biological phenomenon reflecting the spontaneous emission of photons from metabolic processes, particularly redox reactions [43, 44, 46, 50-54]. Its potential as a diagnostic biomarker has been demonstrated in non-BT contexts: tumor tissues have been shown to emit significantly higher photon counts than normal tissues in murine models [57], establishing a proof-of-concept for UPE as an optical biomarker of metabolic and oxidative state. Subsequent research expanded this scope to include whole-body surface UPE mapping, revealing consistent anatomical emission patterns across individuals, with highest intensities originating from the palms and the head [60], a finding of particular relevance to BT research given that practitioners typically direct treatment through the hands. Further evidence indicates that mental concentration can significantly alter biophoton emission from fingertips [45]. That negative emotional states produce measurable increases in trunk UPE intensity [54], suggesting that practitioner physiological and intentional states may modulate their own UPE output. Most recently, photoencephalography, defined as the passive measurement of UPEs from the human scalp correlating with functional brain states, has opened a new avenue for non-invasive neuroimaging [46], with potential implications for characterizing the practitioner's physiological state during BT sessions.

Key experimental constraints on UPE measurement include the extremely low emission intensity, requiring photomultiplier tubes or cooled CCD detectors in light-tight chambers, susceptibility to thermal and optical confounds, and the absence of standardized protocols for clinical measurement. The spectral characteristics, coherence properties, and electromagnetic flux density of any BT-associated UPE changes have not been systematically characterized, and the physical basis for any practitioner-induced modification of recipient UPE remains hypothetical.

Preliminary studies have explored UPE as an objective metric for BT effects. Using cucumber sections as a biosensor, a significant difference in biophoton intensity between treated and control samples was reported following non-contact Qigong or prayer interventions ($p = 0.002$) [48]. In a mammalian cell model, Reiki application was associated with a significant increase in photon emission post-treatment ($p < 0.05$), alongside upregulation of anabolic extracellular matrix genes [49].

These observations converge on a specific and testable mechanistic hypothesis for BT research: that natural photobiomodulation may occur when a patient's tissues absorb UPEs emitted by a biofield practitioner, with downstream biomolecular effects mediated by the spectral and coherence properties of the received photonic signal. An urgent investigation is needed to validate this causal pathway, characterize the biomolecular effects of practitioner-derived UPE on target tissues, and develop standardized dosimetric protocols to quantify the photonic dose delivered during BT sessions. Until such validation is achieved, UPE-based metrics for BT effects must be regarded as a promising but unproven research frontier.

4.5 Limitations

This synthesis has several important limitations that must be considered in interpreting the findings. First, this is a narrative synthesis based on a purposive, non-systematic sample of 14 studies. The selection was designed to maximize biomarker diversity, not to provide exhaustive or representative coverage of the literature. Consequently, all quantitative summaries reflect only this specific sample and cannot be generalized to the field as a whole. Publication bias and selection bias cannot be excluded. Second, the included studies are highly heterogeneous in terms of BT modalities, study designs (RCTs, preclinical animal models, *in vitro*), outcome measures, and methodological quality. This heterogeneity precludes formal pooling of effect sizes and limits cross-study comparisons. Third, the evidence base remains preliminary. Most *in vitro* experiments have not been independently replicated. The small number of RCTs ($n = 5$) substantially limits the generalizability of clinical findings, and all five RCTs have methodological limitations including small sample sizes, heterogeneous populations, and, in most cases, the inherent difficulty of double-blinding BT interventions. Fourth, the biomarker classification framework, while grounded in FDA/NIH guidelines, was custom-developed for this synthesis and has not been independently validated. Fifth, the moderator analysis (Table 2) was based on only four studies, which severely limits statistical power and substantially increases the risk of Type II error. These results should be treated as exploratory and hypothesis-generating. Sixth, the field currently lacks standardized protocols for BT administration (dose, duration, practitioner experience), complicating cross-study comparisons and the interpretation of apparently inconsistent findings. Finally, several extraordinary findings in this synthesis, including rapid increases in telomere length, selective anticancer signaling effects, and UPE-based dose metrics, require substantially stronger qualification and independent replication before they can be incorporated into any mechanistic framework for BTs.

5. Conclusion

This narrative synthesis synthesizes biomarker data from a purposively selected, methodologically diverse set of studies on BTs, revealing a complex and multifaceted research

landscape. The available evidence is consistent with the possibility that BTs may modulate a range of biological processes from inflammatory markers and HPA-axis activity to gene expression and cellular signaling *in vitro*. Emerging research on UPE offers a potentially quantifiable biophysical correlate for both the dose and effect of these interventions. However, the physical basis and experimental limitations of UPE measurement require more systematic investigation.

The evidence base, while generating important hypotheses, remains preliminary, heterogeneous, and largely unreplicated. The primary contribution of this synthesis is to identify specific biomarkers and pathways, including NK cell function, cortisol regulation, and PI3K/Akt signaling, as priority targets for future research. Future studies should prioritize rigorous RCT designs, pre-specified primary endpoints, adequate sample sizes, standardized BT protocols, and validation of biophotonic measurement techniques as objective biomarkers. Ultimately, this field requires a substantial body of high-quality, independently replicated validation, and the work reviewed here represents a foundational step, not a definitive conclusion, toward understanding the potential biological impact of biofield interventions.

Acknowledgments

The author would like to gratefully acknowledge the Brazilian Academic Consortium for Integrative Health (CABSIN) for its invaluable support and for fostering the development of the Biofield Therapies research landscape.

Author Contributions

R. S. analyzed the literature, drafted the manuscript.

Funding

The author declares that no funds, grants, or other support was received during the preparation of this manuscript.

Competing Interests

The author declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Data Availability Statement

The data included in this manuscript are available from the corresponding author.

AI-Assisted Technologies Statement

Artificial intelligence tools were used solely for English-language revision in the preparation of this manuscript. All scientific content, data analysis, interpretation, and conclusions are the sole responsibility of the author.

Additional Materials

The following additional materials are uploaded at the page of this paper.

1. Table S1: Condensed table of the 14 studies with detailed data.
2. Supplementary Materials S2: Classification, proportion, and distribution of biomarkers.

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