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Summarize the findings of this study: Arendash GW. The Evidence That Brain Cancers Could Be Effectively Treated with In-Home Radiofrequency Waves. Cancers (Basel). 2025 Aug 15;17(16):2665. doi: 10.3390/cancers17162665. PMID: 40867293; PMCID: PMC12384137.

Here is a concise summary and critical take-home of the Arendash (2025) perspective article "The Evidence That Brain Cancers Could Be Effectively Treated with In-Home Radiofrequency Waves" (Cancers) PMC+1

Summary of Key Points

Background & Rationale

- Brain cancers (both primary, e.g. gliomas/glioblastomas, and brain metastases) currently have very poor long-term prognoses and limited effective treatments. PMC+1
- Conventional therapies (surgery, radiation, chemotherapy, targeted therapy, immunotherapy) are constrained by issues such as tumor invasiveness, blood-brain barrier, tumor heterogeneity, immunosuppressive microenvironments, and treatment resistance. PMC+1
- The author proposes a novel non-thermal, non-invasive approach: **Transcranial Radiofrequency Wave Treatment (TRFT)** (also called TEMT) delivered via a head-cap device (MemorEM) that patients could self-administer at home. PMC+1

Proposed Mechanisms of Action Against Brain Tumors

Arendash posits that TRFT could target brain cancers by three (potentially synergistic) "disease-modifying" mechanisms:

1. Enhancement of meningeal lymphatic (mLV) flow / immune trafficking

- The meningeal lymphatic vessels (mLVs) are pathways by which interstitial fluid and immune cells can drain from the brain toward cervical lymph nodes. PMC+1
- TRFT might modulate VEGF levels (or otherwise influence lymphatic dynamics) to increase mLV flow, improving "communication" between the brain tumor microenvironment and peripheral immune organs, facilitating tumor antigen presentation and immune activation against the tumor. PMC+1

Rebalancing cytokine / inflammatory milieu in brain / tumor microenvironment

- Brain tumors are often surrounded by dysregulated cytokine environments, promoting chronic inflammation, immunosuppression, and tumor-supportive signaling. PMC+1
- The author cites data from Alzheimer's Disease patients treated with TRFT showing that cytokine levels (in CSF and plasma) may be "rebalanced" i.e. if a cytokine is abnormally low,



- TRFT tends to raise it, and if abnormally high, tends to lower it. PMC
- Correspondingly, TRFT reduced levels of CRP (a marker of inflammation) in CSF/plasma over extended treatment in those Alzheimer's subjects, indicating anti-inflammatory effects. PMC+1
- If similar modulation occurs in tumor-bearing brains, the idea is that TRFT could make the tumor microenvironment less hospitable to tumor growth and more permissive to immune attack.

3. Potential direct anti-proliferative / cytotoxic effects on tumor cells

- In vitro studies (i.e. cell culture) have shown that exposure of glioma / glioblastoma cell lines to specific RF frequencies (e.g. 2.4 GHz) can reduce tumor cell viability or proliferation. PMC+1
- The author acknowledges that these cell studies are simplistic models (lack the complexity of in vivo tumor microenvironment, immune and vascular influences), but they provide proof-ofconcept that RF waves might directly impair tumor cells. PMC+1

Supporting Evidence & Previous Applications

- The author points out that TRFT (via the MemorEM device) has already been used in small human studies in Alzheimer's Disease, with some claimed reversal of cognitive decline and metabolic improvements (FDG-PET) over 2 months of therapy. PMC+1
- The author also references pre-clinical RF / electromagnetic wave studies in other settings (e.g. Alzheimer's mouse models) that show beneficial effects on brain pathology, mitochondrial function, etc., as supporting precedent for safe and biologically active brain RF exposure.
 PMC+2
- Further, epidemiological large studies of cell phone use (RF exposures in the 900–1800 MHz range) have generally not found increased brain cancer risk (some even hinting at lower rates), which the author uses to argue that RF in that range may not be harmful to brain health. PMC+1

Limitations & Open Questions (acknowledged)

- Importantly, **no clinical trials of TRFT have yet been conducted in patients with brain cancers** (either primary or metastatic). The entire argument is based on extrapolation from cell culture, animal studies, and Alzheimer's clinical data. PMC+1
- The author notes that more extensive biomarker studies, including immune cell changes (macrophages, T cells, dendritic cells) in CSF and tumor, are needed in cancer settings. PMC+1
- It is possible that TRFT will need to be combined with other modalities (e.g. immune checkpoint inhibitors) to achieve maximal effect. PMC+1
- The precise mechanisms of how RF waves modulate cytokines, VEGF, or immune trafficking remain mechanistic hypotheses rather than established facts. PMC+1
- Translational hurdles include skepticism from clinicians unfamiliar with this technology, regulatory acceptance, optimal dosing/frequency protocols, safety in cancer patients, and device deployment logistics. PMC+1

Conclusion & Author's Recommendation

• The author argues that the cumulative evidence (pre-clinical, in vitro, and human Alzheimer's trials) is strong enough to **justify immediate clinical trials of TRFT in brain cancer patients**, either



- alone or in combination with immunotherapies. PMC+1
- The author suggests that if TRFT can deliver even modest tumor regression or inhibit growth, its favorable safety, non-invasiveness, and ease of home administration would make it a paradigm-shifting modality for brain cancers. PMC+1

Critical Appraisal & Commentary

While Arendash's proposal is intriguing and ambitious, some caveats temper enthusiasm:

- The evidence in cancer settings is currently *indirect*. The leap from Alzheimer's RF effects to anti-tumor effects is speculative.
- In vitro cell line reductions in viability under RF exposure may not translate to meaningful in vivo effects, especially in a complex, heterogeneous tumor microenvironment with blood flow, immune suppression, host repair, and other compensatory mechanisms.
- Safety, optimal parameters (frequency, power, duration), and long-term effects of brain-wide RF exposure in cancer patients remain to be validated.
- The immune modulation hypothesis (enhanced lymphatic flow, cytokine rebalancing) is plausible, but mechanistic pathways remain to be elucidated and experimentally validated in tumor models.
- There is potential for bias: the author is inventor and patent-holder of the TRFT technology. PMC+1
- Translational challenges (device scaling, regulatory, funding, clinical trial design) may be significant barriers.

In short: this is a **perspective / hypothesis-driven** article, not a report of definitive empirical results in brain cancer. It introduces a novel idea worthy of further exploration, but the claims are ahead of demonstrated data.

If you like, I can dig up commentary, critiques, or independent validation studies of TRFT in oncology to see how the field is reacting. Would you like me to do that?



Yes

Here's a summary of what I found in follow-up literature and critiques relating to Arendash's proposal, along with caveats and gaps in the current evidence. (In short: the idea is provocative but still speculative; there's little direct support in cancer patients as yet.)

What independent or related evidence exists

1. In vitro / cellular studies of RF effects on glioma / brain cancer cells

• A recent study exposed U-118 MG glioblastoma cells to 2.4 GHz radiofrequency electromagnetic fields (RF-EMF) and observed a **significant decrease in viability** relative to controls, with a greater reduction in cancer cell lines than in a "normal" cell line (HEK-293). PubMed



- More broadly, there is literature on electromagnetic / RF exposure and its effect on cellular behavior (proliferation, gene expression, oxidative stress), though results are heterogeneous and often provocative rather than definitive.
- Some authors raise concerns that RF exposure can alter gene expression in glioblastoma cells (i.e. non-thermal biological effects). For example, there are recent claims (in less rigorous sources) that "RF-induced gene expression changes in glioblastoma" are being "ignored by regulators." (However, these should be interpreted cautiously, as they come from advocacy sources or non-peer-review summaries) rfsafe.com

Thus, there is modest supporting in vitro evidence that particular RF frequencies *may* impair viability or alter behavior of glioma cells — consistent with one of Arendash's proposed "direct anti-tumor" mechanisms. But these are preliminary and often in simplified systems.

2. Animal / in vivo RF / electromagnetic exposure and cancer incidence

- A relatively recent systematic review of in vivo studies (rodent exposure to RF in the 100 kHz –
 300 GHz range) examined tumor incidence across tissues. The meta-analysis found no consistent,
 statistically robust association between RF exposure and increased tumor incidence in most
 tissues. Only in some organs (heart, CNS/brain, intestine) were marginal associations observed, but
 overall the evidence was judged "low or inadequate" for a causal link. PubMed
- This means that, on the flip side, there is no strong existing animal evidence that RF exposure causes brain tumors (i.e. as a risk factor). That reduces one obvious safety concern, though doesn't guarantee safety in a therapeutic context.
- However, the absence of observed carcinogenicity in broad RF exposure studies is not directly reassuring for therapeutic application (since those studies are not designed to test targeted, high-dose brain RF therapy).

3. Human epidemiology / observational studies on RF exposure and brain cancer risk

- A 2024 systematic review of observational human studies concluded that exposure to RF from mobile phone use "likely does not increase the risk of brain cancer", and similarly for occupational RF exposure. ScienceDirect
- Another more recent occupational study (INTEROCC) applying a job-exposure matrix (JEM) for RF
 exposure found largely null or inverse associations for glioma and meningioma risk across
 various exposure lags and metrics. Some statistically significant positive associations arose in
 specific exposure windows, but the authors cautioned that these may reflect residual bias or
 limitations in exposure assessment. Wiley Online Library
- A broader review (EMJ Reviews) of RF field exposure and cancer risk summarized that no convincing evidence exists linking mobile phone or RF exposure to increased brain tumour risk.
 European Medical Journal

These epidemiological findings suggest that long-term, chronic low-to-moderate RF exposures (from phones, occupational sources) have not been conclusively linked to higher brain cancer risk, which may alleviate (though not eliminate) safety concerns about RF in general.



4. Established clinical use of radiofrequency in oncology (hyperthermia)

- The concept of **radiofrequency hyperthermia** (i.e. heating tissues to ~42 °C or more) is a more established adjunct in oncology. A review article describes that localized RF hyperthermia (using capacitive or inductive approaches) has been safely applied in clinical trials, often in combination with radiotherapy or chemotherapy, in superficial and deeper tumors. PubMed
- However, note that **hyperthermia uses heating (thermal effects)**, quite different biologically from what Arendash is proposing (which is non-thermal RF modulation). The mechanisms, safety profiles, and effects are not directly comparable.

Critiques, gaps, and cautionary points

Putting together Arendash's proposal and the independent literature, here are major criticisms or open questions:

1. Lack of direct clinical cancer trials

- To date, there is no published trial using TRFT (non-thermal RF) in human patients with brain cancer (primary or metastatic). Arendash acknowledges this himself. PMC+1
- Evidence from Alzheimer's disease (on which much of his translational rationale relies) may not generalize to a malignant, immunosuppressive tumor context.

2. Mechanistic speculation is relatively weak at present

- The proposed immunologic mechanisms (enhanced meningeal lymphatic flow, cytokine rebalancing) are biologically plausible but remain speculative. There is no published in vivo study showing that non-thermal RF exposure can meaningfully increase immune trafficking from brain tumors to cervical lymph nodes, or reprogram tumor-associated immune suppression.
- The detailed biophysics by which RF fields at the proposed intensities and patterns would selectively modulate VEGF, cytokines, or lymphatic flow are not well demonstrated in the literature.

3. Parameter optimization, dosing, and safety uncertainties

- What frequencies, waveforms, intensities, durations, and spatial distributions are optimal for tumor knockdown (but safe to normal brain) is entirely unspecified.
- Long-term safety is not fully established for repeated RF exposure in patients with damaged or vulnerable brain tissue (post-surgery, radiotherapy, edema).
- There is a potential for unintended effects (e.g. interference with normal neural circuitry, vascular or glial side effects, thermal hotspots) that have not been systematically studied.

4. Potential bias / conflict of interest

- Arendash holds patents related to TRFT and is affiliated with a company manufacturing the devices, which introduces a risk of bias in interpretation or enthusiasm. PMC+1
- Because the paper is a "perspective" rather than a primary empirical study, the arguments are more promotional and hypothesis-driven than rigorously integrative.



5. Generalizability from Alzheimer's to cancer is tenuous

- The immune and molecular environments differ drastically between neurodegenerative disease and aggressive malignancy.
- Effects observed in non-cancer settings (e.g. cognitive improvement, modulation of cytokines) may reflect modulation of non-malignant tissue processes; translating them to a tumor context is nontrivial.

6. Heterogeneity and replicability of RF / electromagnetic biology

- The broader literature on nonthermal RF effects is fraught with irreproducible results, small effect sizes, and methodological challenges (sham control, blind exposure, dosimetry).
- Many claimed effects depend sensitively on experimental design, frequency, exposure geometry, cell type, culture conditions, and endpoints.
- Some studies reporting gene expression or viability changes under RF exposure are from lowertier sources or advocacy-oriented outlets, which need independent replication.

Overall assessment & outlook

- Arendash's proposal is bold and intriguing: using non-thermal, self-administered RF to treat brain cancers is a radically different paradigm than standard pharmacologic or radiotherapeutic approaches.
- The supporting evidence (in vitro glioma cell effects, epidemiologic safety, Alzheimer's clinical results, literature on RF hyperthermia) provides **some grounding** for plausibility but falls far short of proof.
- The biggest gap is direct **in vivo efficacy** and **clinical safety** data in brain cancer patients. Without that, the notion remains speculative and hypothesis-generating.
- Before broad adoption, the community would need well-designed pilot clinical trials, ideally with rigorous controls, biomarker endpoints (tumor volume, immune infiltration, molecular changes), and long-term follow-up.
- Skepticism is justified given the history of many electromagnetic / bioelectronic proposals that did not scale from promising in vitro/animal findings to successful clinical translation.

If you like, I can check whether any clinical trials have been registered (e.g. in clinicaltrials.gov) for TRFT in brain cancer, or whether any independent groups are attempting replication. Would you like me to do that?



Yes

I searched clinical trial registries and the published literature, and here's what I found regarding whether any trials are registered (or underway) testing **Transcranial Radiofrequency Wave Treatment (TRFT)** or closely related radiofrequency electromagnetic interventions in brain cancer patients:



Registered / ongoing trials relevant to radiofrequency / electromagnetic fields in glioblastoma / brain tumors

- 1. NCT06140875 "Amplitude Modulated Radiofrequency Electromagnetic Field Treatment Combined With Radiochemotherapy and Maintenance Chemotherapy in Patients With Glioblastoma (Brain-RF)"
 - This is an interventional clinical trial combining standard-of-care radiochemotherapy (radiation + temozolomide) with amplitude-modulated radiofrequency electromagnetic field (RF-EMF) treatment. National Brain Tumor Society+1
 - The device uses a carrier frequency of 13.56 MHz. National Brain Tumor Society+1
 - During the radiochemotherapy phase, RF sessions are planned at 60 minutes three times per week, and during maintenance chemotherapy phase, twice per week.
 National Brain Tumor Society+1
 - The study is recruiting (or intended) and is designated as Phase 1 (i.e. focusing on feasibility / safety). National Brain Tumor Society
 - It is being conducted at Charité Universitätsmedizin Berlin (Germany).
 National Brain Tumor Society

This is probably the closest trial to Arendash's vision of combining RF with standard brain cancer therapy, though it is not exactly the same as home-administered, non-thermal TRFT (Arendash's proposal focuses more on non-thermal modulation rather than amplitude-modulated RF adjuncts).

- 2. Older clinical trials of radiofrequency hyperthermia in malignant brain tumors
 - There are some older trials using **RF hyperthermia (i.e. thermal heating approaches)** in malignant brain tumors (e.g. 13.56 MHz capacitive heating) in small cohorts (often combined with radiation or chemotherapy). PubMed+1
 - Example: In one trial with 16 patients, RF heating was applied (with efforts to limit temperature increases) and a few tumor regressions were reported on imaging. PubMed
 - Another trial in 19 patients also attempted intraoperative and external RF heating in brain tumors and reported partial responses. PubMed
 - However, these are **thermal** RF interventions (i.e. purposeful heating) rather than the non-thermal, home-device style modulation that Arendash advocates.

3. Amplituted-modulated RF in other cancers

- A phase 2 study in pancreatic cancer is ongoing using amplitude-modulated RF electromagnetic fields (AM RF EMF), though not in brain tumors. ascopubs.org
- This shows that RF-based modalities are being tested in oncology, but not specifically (yet) in brain cancers in the ARendash TRFT framework.

Interpretation & conclusions

• Yes — there *is* at least one registered trial (NCT06140875) applying RF electromagnetic fields **concurrently with chemoradiation** in newly diagnosed glioblastoma patients. This is the most



direct clinical test that approaches the idea of combining RF modulation with standard therapy.

- However, that trial is **not identical** to Arendash's home-based non-thermal TRFT paradigm (which would ideally be self-administered, noninvasive, and used in isolation or in combination). The Berlin trial uses amplitude modulation and a medical setup adjunct to standard therapy.
- No trials (based on my search) appear to be testing **home-administered non-thermal transcranial RF modulation (Arendash's full vision)** in brain cancer patients yet.
- The older RF hyperthermia trials, while relevant to the broader class of RF approaches, differ in mechanism (they rely on heating) and are older, small, and largely preliminary.

If you like, I can check whether that Berlin trial has progressed results or update you on its status (e.g. publications, outcomes). Do you want me to dig into that?



Yes

I found no published results or outcome reports so far from the **Brain-RF (NCT06140875)** trial; it appears to still be in the enrollment / early stage. Here's what I located and what that implies:

What is known about the Brain-RF trial status & design

• Registration & timeline

The trial was first submitted and posted in November 2023.

ICHGCP+2

Estimated primary completion is **May 14, 2029**, and full study completion also by **May 14, 2029**. ICHGCP+2

So as of now (2025), the trial is ongoing and not yet at the stage of reporting primary endpoints. ICHGCP+2

Design features

- It is a single-arm, open-label (no randomization, no blinding) interventional trial combining standard-of-care chemoradiotherapy + temozolomide with amplitude-modulated RF electromagnetic field sessions. ICHGCP+2
- The RF treatment is scheduled as **60-minute sessions** three times per week during the radiotherapy phase, and twice per week during maintenance temozolomide cycles, totaling about 30 RF sessions across the protocol. National Brain Tumor Society+2
- The carrier frequency used is **13.56 MHz** for the RF electromagnetic field treatment. National Brain Tumor Society+1
- Key endpoints include **progression-free survival (PFS) at 6 months**, **overall survival (OS)** over the study period, toxicity (acute and late), quality-of-life, brain function metrics, and salvage treatments. ICHGCP+1

• Recruitment / target enrollment

The trial aims to recruit ~26 participants. MedPath+2
The eligibility criteria include newly diagnosed glioblastoma (age 18–70, WHO performance ≤2),



adequate organ function, and having had at least subtotal or gross resection of tumor, among others. ICHGCP+2

• No published results yet

I did not locate any peer-reviewed paper, conference abstract, or clinical trial registry update that reports efficacy or safety results from this Brain-RF trial.

Related trials and RF / EMF oncology results

While the Brain-RF trial has no results yet, there are related lines of investigation that may inform expectations and mechanisms:

Trials in other tumor types

There is a Phase 2 study underway using amplitude-modulated RF EMF (AM RF EMF) in metastatic pancreatic cancer (NCT05776524). ascopubs.org+1

There is also a trial planned (or recruiting) in hepatocellular carcinoma combining AM RF EMF with immune checkpoint inhibitors (Immune-RF, NCT06821958). CenterWatch

These aren't brain tumor trials, but they represent the growing interest in RF/EMF modalities in oncology.

• Preclinical / mechanistic support

- A 2025 in vitro study at 2.1 GHz exposure (non-thermal RF) in U118-MG glioblastoma cells showed induction of apoptosis (increased CASP3/8/9), and alterations in gene expression (higher BAX/BCL-2 ratio) after longer exposure (48 h). PubMed
- A 2023 study (2.4 GHz) found that glioblastoma U-118 MG cells exposed to RF had decreased viability, more so than a "normal" HEK-293 cell line. PubMed
- More broadly, the concept of tumor-specific amplitude-modulated EMF has precedent: in a 2009 study, researchers identified "tumor-specific frequencies" in patients (0.1 Hz to 114 kHz range) and applied them to patients with various advanced cancers (compassionate use).
 Some patients had objective responses or stable disease, with minimal toxicities reported.
 BioMed Central

• Cellular RF gene expression / apoptosis effects beyond brain tumors

The 2025 U118-MG study is one of the more direct recent examples in glioma models. PubMed Older works have explored how amplitude modulation at tumor-specific frequencies may influence intracellular signaling pathways (e.g. IP3/DAG, calcium signaling) in cancer cell lines (e.g. hepatocellular carcinoma). ascopubs.org

What the absence of published results implies (and caveats)

- Because the trial completion is years in the future (2029 estimated), it is too early to expect publicly available outcome data. ICHGCP+2
- The design is a feasibility / early-phase trial (safety, toxicity, tolerability, preliminary signals) rather than a large randomized efficacy trial, so even when results appear, they may be more about safety and tolerability than definitive proof of efficacy. National Brain Tumor Society+2



- Because the trial is open-label and single-arm, interpretation of any tumor response or survival benefit will need cautious comparison to historical controls or matched cohorts not a randomized control.
- The lack of result reporting to date is not unusual for early-phase device/EMF trials, especially when the trial is ongoing and still within its recruitment and follow-up phases.

If you like, I can set up an alert or check future updates periodically and notify you when results are posted, or I can try to look for any interim reports (e.g. conference abstracts) that might not yet appear in journals. Do you want me to monitor that for you?