Health protection deserves clear answers

RF and EMF exposure: a selective review

20 October 2026 | Version 4

People should be able to rely on health protections without having to become scientists. Research on radiofrequency (RF) signals and other electromagnetic fields (EMF) raises questions about cancer, reproductive health and how living systems respond to exposure. Understanding what those findings mean matters to the protection people rely on every day.

Start with rare heart tumours in male rats. The US National Toxicology Program classified its findings after prolonged whole-body exposure as 'clear evidence' of cancer-causing activity. A separate lifetime study reported a significant increase in the same tumour type at its highest exposure. Laboratory studies also found sperm damage, including in human sperm studied outside the body. Cancer prevention and reproductive health give these findings a human importance that deserves serious attention. The experiments alone do not tell us the risk from everyday phone use or establish infertility in people.

This updated selective review brings published adverse findings, exposure-associated symptoms, susceptibility and biological responses potentially relevant to harm more readily into view. It makes original evidence accessible while keeping its limits visible. It is neither a comprehensive systematic review nor a balanced assessment of the entire literature. We hope it helps researchers, health professionals, public authorities and interested readers give these findings the careful consideration their stakes deserve. A central question runs through the review: do existing exposure protections adequately address the outcomes identified here?

Begin with the chronic-pathology chapter and records RF-001 and RF-002, then reproductive findings RF-006 to RF-008. The coloured 'Why this matters' panels offer a short way into every record; its technical detail shows what was found and what remains uncertain.

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Finding your way

The chapters introduce the evidence; linked identifiers lead to the detailed records. Start with chronic pathology, then reproductive findings: their disease or functional endpoints make the stakes especially concrete. Neurological, cellular and ecological chapters raise different questions and need their own exposure conditions.

Every record opens with a coloured 'Why this matters' panel. Its verification group distinguishes original-text review, abstract-level evidence, replication context, retrieval leads and editorial notices. These are 79 publication records, not 79 independent experiments. Contents, bookmarks and author, topic and exposure indexes help you find a route through them. Fuller source and verification methods follow the annex.

Chronic pathology: the cancer question

NTP's controlled rat bioassay RF-001 found malignant cardiac schwannomas, tumours of nerve-supporting cells in the heart. Male-rat findings were classified as clear evidence of carcinogenic activity under both GSM and CDMA modulation, using survival-adjusted analysis. Prolonged whole-body exposures of 1.5, 3 and 6 W/kg are experimental hazard conditions, not ordinary handset use.

The Ramazzini lifetime study RF-002 reported the same rare tumour type in a separate far-field programme. Male cases were 0/412, 3/401, 1/209 and 3/207 at 0, 5, 25 and 50 V/m; only the highest exposed group differed significantly from sham. The authors reported no survival difference. Sparse events, one room per intensity, litter assignment and tests without NTP's survival adjustment qualify the result. The top estimated rat whole-body SAR, about 0.1 W/kg, does not certify compliance with a human limit.

Convergence across different arrangements matters, but these were not identical-protocol replications and involved pathology collaboration. Later molecular profiling RF-025, with a 2026 correction, reuses Ramazzini tissues; it adds tumour biology, not another cohort or proof of RF-caused mutations. Chou's older programme RF-026 adds a pooled-malignancy signal without a significant single-site or overall-neoplasm increase.

Reproducibility remains unresolved. Japanese and Korean follow-ups at 4 W/kg CDMA CTX-001 CTX-002 did not reproduce significant glioma or cardiac-schwannoma increases. Japanese sham survival was lower; Korean survival was comparable, so that caveat cannot explain both results. Neither recreates the Ramazzini lifetime far-field protocol. Together, these findings make chronic cancer protection a serious question while leaving ordinary-use human risk unquantified.

Susceptibility and combined exposures

Tillmann RF-004 and Lerchl RF-003 ask whether RF can modify tumour development in mice given the cancer-causing chemical ENU. Tillmann reported lung carcinoma in 45/58 combined-exposure mice versus 33/60 given ENU alone. Liver findings were excluded from the authors' interpretation because of evidence of Helicobacter infection. Lerchl reported increases in selected tumours, including at nominal whole-body SAR 0.04 W/kg, with one-tailed tests, no monotonic dose-response and substantial position-dependent SAR variation. Different biological laboratories shared exposure-engineering investigators: this is a connected programme, not wholly independent corroboration.

These experiments concern an initiated host; they do not establish RF-alone initiation or a clinical category of vulnerable people. A fetal follow-up did not establish the proposed early DNA-adduct mechanism CTX-003. The original lymphoma finding in genetically susceptible mice RF-024 was not confirmed in independent attempted replications CTX-004.

Ding RF-035 reported transformation-related cell changes and tumours after exposed cells were transplanted into immunodeficient mice. The recipients were not RF-exposed. High culture SAR, chemical-promoter use in one assay and unclear donor-cell histories prevent an RF-alone initiation claim. A separate power-frequency plus gamma-radiation experiment ELF-009 raises a coexposure question in another field regime. Susceptibility is worth understanding, but each model and comparator determines what it can tell us about protection.

Reproductive findings: damage and its consequences

Sperm movement and genetic integrity are meaningful reproductive endpoints. De Iuliis RF-006 found reduced movement and survival in isolated human sperm, with oxidative DNA damage at higher culture exposures. The published correction changes biological replication to three and corrects Figure 1D. Houston's whole-body mouse experiment RF-007 found sperm functional and DNA damage, while fertilisation and early embryo development were not impaired. Related culture work RF-008 found cell-stage and time-dependent responses; a transient comet-assay signal and a negative halo assay must remain together. These papers involve related Newcastle investigators, not three independent laboratories establishing infertility.

Separate rat and human-semen experiments RF-018 RF-019 broaden the laboratory evidence, with room, temperature and dose uncertainties. Other entries remain retrieval leads. Rahban's study RF-038 found an association between frequent phone use and lower total sperm count in young men; self-reported use was not measured RF dose. A newer rat experiment RF-022 explicitly lacked animal-position dose and organ-temperature measurements.

The protection question is consequential: do reproducible sperm changes persist and affect fertility, pregnancy or offspring? The distinction between cellular damage and demonstrated reproductive impairment gives that question precision rather than diminishing its importance.

The brain: tissue findings, function and symptoms

Salford RF-020 reported higher blinded dark-neuron scores in rat brains about 50 days after a two-hour exposure. Comparisons were significant at 0.02 and 0.2 W/kg, not 0.002 W/kg. Small mixed-sex groups, ordinal scoring, an embedded figure correction and nonconfirming independent replication CTX-006 constrain this pathology signal. It does not establish permanent human injury or a low-dose human threshold.

Lund follow-up RF-028 found delayed leakage of a blood protein into brain tissue without increased dark neurons. Cognitive and histological papers RF-029 CTX-005 used the same rats: poorer object-order memory was not explained by demonstrated neuronal loss. Handling and test experience complicate interpretation. Other institutions' barrier and cognition findings remain distinguishable where only abstracts were verified.

Blinded human experiments provide a different kind of evidence. EEG changes RF-010 RF-011 and increased regional brain glucose use RF-049 are objective responses with uncertain adverse significance. Lustenberger RF-032 found slightly less sleep and less overnight improvement in one motor-task measure in 16 men. Its unusual pulses and distinct peak, burst and overnight-average doses limit generalisation. Reanalyses of the same participants are not independent experiments.

Foerster's adolescent memory association RF-033 was significant in the operator-data subgroup, while the full-sample confidence interval included zero; an earlier paper overlaps the cohort. Hutter RF-013 reported an adjusted headache association, without a significant sleep-quality association or multiple-testing correction. Functional changes and reported symptoms deserve attention, while observational bias, exposure uncertainty and clinical meaning remain open questions.

Cellular responses: when does a change become harmful?

NTP-associated work RF-005 found tissue- and scoring-specific comet-assay DNA damage, with negative micronucleus tests. It belongs to the same programme as the tumour bioassay. Choi and Min RF-009, from a separate Korean programme, reported chemical stress, reduced growth and senescence in human cell cultures during continuous LTE exposure. Measured DNA-double-strand-break and apoptosis markers did not increase. Arrested growth has different implications in normal and cancer cells.

Xu RF-039 found DNA-damage signalling in two cell types without corresponding sustained abnormalities in other assays. Souffi RF-037 found altered sound-evoked brain responses in inflammatory-challenged rats, making biological state a specific susceptibility question; it did not demonstrate human hearing loss. Broom RF-036 reported persistent early-life-exposure behavioural changes in small mouse groups, with differing directions and uncertain adversity. Lameth RF-021 found altered cortical gene expression under a genuine 5G-modulated 3.5-GHz signal, with behaviour unchanged.

Such responses can help explain how exposure interacts with living tissue. Their protection significance depends on persistence, reproducibility and a link to impaired function or disease. The classic DNA-break abstracts remain methods-pending. Schwarz's Expression of Concern RF-016 prevents that paper from carrying the central mechanism argument.

Wildlife and other electromagnetic exposures

Finding the way home is essential to a foraging bee. An engineered experiment ECO-004 found reduced homing after prolonged 2.4/5.8-GHz exposure. Brood development and overall survival were not significantly different; a partial survival-window result was exploratory. Continuous transmission represents a heavily loaded condition, not every Wi-Fi setting. A queen-development experiment ECO-003 found reduced hatching, with only two rearing-colony pairs and an unusual handset arrangement; mating among surviving queens was unchanged.

Blinded bird experiments ECO-006 ECO-007 found disruption of magnetic-compass orientation under particular noise bands, including 75-85 MHz. They concern a specialised sense and related research programmes, not demonstrated migration losses, population decline or effects from GHz phone signals. These functional endpoints show why human exposure restrictions alone cannot establish wildlife protection.

Other field regimes raise their own questions. A single-person power-frequency electric-field challenge ELF-007 cannot estimate RF symptom prevalence. The intermediate-frequency memory experiment IF-001 required a separate high-exposure cohort after equipment problems. MRI static-field balance and dizziness records SF-001 to SF-004 remain retrieval leads. Findings cannot be transferred between these exposures simply because all are non-ionising fields. Ecological associations and unverified leads likewise remain distinct from established causal effects.

What the findings can tell us about protection

The US RF rule, 47 CFR 1.1310, distinguishes general-public whole-body SAR (0.08 W/kg), peak local SAR (1.6 W/kg over 1 g) and frequency-dependent external-field quantities. SAR describes absorbed power per unit mass. The rule specifies averaging and evaluation conditions and expressly applies to continuous exposure for indefinite periods: a 30-minute averaging window is not a limit on its duration of application. ICNIRP 2020 is a separate framework.

Animal whole-body SAR, local human SAR, culture SAR and external electric field are different quantities. Frequency, geometry, spatial distribution, averaging time, duty cycle and temperature affect interpretation. A lower numerical value in one experiment neither certifies human-limit compliance nor proves that a standard fails. The records preserve those distinctions rather than deriving a replacement exposure limit.

The central public question remains whether the evidence underlying current protection adequately addresses chronic disease, reproductive function and susceptibility. Direct adverse outcomes deserve close consideration; physiological and molecular responses need an additional link to harm. Replication failures, shared cohorts and unresolved methods change the confidence a finding can carry. Making that evidence accessible can support scientific understanding and well-founded protection, while leaving the overall balance of the literature to a broader assessment.

Annex A Original text reviewed

Methods and results in an original article body, accepted manuscript, proof or government report were inspected during this or earlier source verification. The source form is identified in each record. This label does not mean every supplement, figure, dataset or underlying analysis was audited. Some well-characterised biological responses have uncertain adverse significance.

Records in this section: RF-001, RF-002, RF-003, RF-004, RF-005, RF-006, RF-007, RF-008, RF-009, RF-010, RF-011, RF-013, RF-014, RF-018, RF-019, RF-020, RF-021, RF-022, RF-025, RF-026, RF-028, RF-032, RF-033, RF-035, RF-036, RF-037, RF-038, RF-039, RF-040, RF-041, RF-049, ELF-007, ELF-009, IF-001, ECO-003, ECO-004, ECO-006, ECO-007

RF-001 Malignant heart tumours after long term exposure

> **Why this matters**
> Male rats developed malignant tumours in cells that support nerves in the heart. The US government's finding raises a concrete question about long-term cancer protection. Translating this animal hazard to human exposure needs careful assessment; the experiment does not measure anyone's personal cancer risk.

Original text reviewed | Source checks to 20 October 2026

National Toxicology Program. NTP Technical Report on the Toxicology and Carcinogenesis Studies in Hsd Sprague Dawley SD Rats Exposed to Whole-Body Radio Frequency Radiation at a Frequency of 900 MHz and Modulations GSM and CDMA Used by Cell Phones. Research Triangle Park NC: NIEHS; 2018. NTP TR 595. doi:10.22427/NTP-TR-595.

Exposure. 900 MHz; whole-body SAR 1.5, 3 or 6 W/kg; 10 minutes on/10 minutes off; about 9 hours 10 minutes RF-on daily, prenatal to two years.

Design and controls. 105 offspring per sex/group initially; 90 per sex/group in two-year pathology cohorts; shared sham controls.

Reported finding. NTP classified male-rat heart schwannoma findings as clear evidence of carcinogenic activity for both GSM and CDMA. Counts were 0/90 sham versus 2/90, 1/90, 5/90 GSM and 2/90, 3/90, 6/90 CDMA.

Interpretive limits. Animal disease endpoints; these whole-body exposures are not ordinary handset use. Control and exposed survival differed; NTP used survival-adjusted analyses.

Programme and cohort links. RF-002 is a separate laboratory programme. RF-005 uses interim animals from the NTP programme.

The protection question raised. How should a controlled animal finding of malignant heart tumours inform long-term human protection, given the differences in exposure, survival and species? It identifies a cancer hazard under these experimental conditions, not an everyday-use risk estimate.

Source verification. Official full report consulted for current status, funding, contractor roles, pathology and peer review. Original final conclusions use clear evidence for male cardiac malignant schwannomas under both modulations; female findings are equivocal. Appendix L documents external peer review; the final categories replace draft categories, without implying a suppressed or secretly changed result.

Source locations. Report pp 66 and 103, Tables 20 and 47; pp xxiii-xxiv conclusions; p viii formatting statement; pp ix-xiii contributor roles; p xvi peer-review remit; p xviii acknowledgments; pp xxiii-xxiv/124 conclusions; Appendix L peer review.

Editorial status. Current official PDF p viii states it was reformatted to meet new NTP publishing requirements and its content has not changed. Official report remains TR 595, November 2018; PubMed indexing in 2021 is not a new experiment. No correction, retraction or expression of concern located in official/PubMed and targeted DOI/title notice checks on 2026-10-20. The report's publication-details page prints NTP-TOX-595 in one DOI field; use NTP-TR-595 confirmed by official landing-page citation and PubMed.

Funding. NIEHS/NIH Intramural Research Program ES103316, ES103318 and ES103319; performed for NTP/HHS under federal contracts listed in the original report acknowledgments, p xviii. IIT Research Institute conducted studies; IT'IS constructed/maintained exposure system; NIST independently validated that system. Affiliations and contractor roles are documented, not inferred financial conflicts.

Conflicts. Not established from retrieved material.

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RF-002 Heart tumours in a separate lifetime rat programme

> **Why this matters**
> A separate lifetime study found more of the same rare heart tumour in male rats at its highest exposure. That convergence deserves attention when considering cancer protection, even though the experiments differed and shared pathology expertise. The finding cannot establish a safe or unsafe human exposure level.

Original text reviewed | Source checks to 20 October 2026

Falcioni L, Bua L, Tibaldi E, Lauriola M, De Angelis L, Gnudi F, Mandrioli D, Manservigi M, Manservisi F, Manzoli I, Menghetti I, Montella R, Panzacchi S, Sgargi D, Strollo V, Vornoli A, Belpoggi F. Report of final results regarding brain and heart tumors in Sprague-Dawley rats exposed from prenatal life until natural death to mobile phone radiofrequency field representative of a 1.8 GHz GSM base station environmental emission. Environmental Research. 2018; 165: 496-503. doi:10.1016/j.envres.2018.01.037.

Exposure. 1835 MHz GSM/GMSK, full time-slot assignment; far-field 0/5/25/50 V/m, 19 h/day, gestational day 12 to natural death. Estimated whole-body SAR 0/0.001/0.03/0.1 W/kg; coupling-factor estimate, not individual/organ dosimetry.

Design and controls. 2448 Sprague-Dawley rats; male n=412/401/209/207, female n=405/410/202/202 in ascending field groups. Sham; blinded pathology, blinded NTP second-opinion review. Tumour incidence Fisher/chi-square; Cochran-Armitage trend; survival separately Kaplan-Meier/log-rank/Cox.

Reported finding. Male cardiac schwannomas: 0/412, 3/401, 1/209, 3/207; highest group p≤.05, Fisher. Female cardiac schwannomas: 4/405, 9/410, 1/202, 2/202. Female malignant glial tumours: 2/405, 3/410, 2/202, 3/202; nonsignificant. Hyperplasia increases were nonsignificant. Authors reported no survival difference.

Interpretive limits. Sparse events; highest-dose male comparison drives significance. Tumour tests not survival-adjusted; one room/intensity; litter assignment. Rat SAR cannot certify human-limit compliance.

Programme and cohort links. Separate exposure/animal programme from NTP RF-001, with NTP pathology collaboration; Brooks 2024 reuses these tissues. Soffritti 2019 mini-review repeats results, not another bioassay.

The protection question raised. What does convergence on a rare heart tumour across different lifetime exposure arrangements mean for chronic protection? Sparse events, shared pathology expertise and estimated rat doses limit the inference; numerical SAR alone cannot answer it.

Source verification. Original published 8-page PDF: methods, results, Tables 1-3, Fig 3 and funding reviewed; metadata independently checked with Scite/PubMed.

Source locations. pp 497-499 §§2.1, 2.3-2.6; p 500 Fig 3; p 501 Table 2; p 502 Table 3/funding.

Editorial status. Scite metadata and PubMed relations checked 2026-10-20: no correction/retraction/concern located. PubMed links 2020 statistical-reappraisal comment (10.1016/j.envres.2020.110233) and 2021 response (10.1016/j.envres.2021.111067); these are comments, not notices.

Funding. RI; ARPA Emilia-Romagna; Children With Cancer UK; Cassa di Risparmio Bologna; INAIL; P.E.I.; Fondazione del Monte Bologna/Ravenna; Environmental Health Trust. Authors report no conflict; funders no direct study/publication role.

Conflicts. Authors declare no conflicts or competing interests, as documented in the retrieved source disclosure.

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RF-003 More tumours in chemically initiated mice

> **Why this matters**
> In mice given a cancer-causing chemical, some RF-exposed groups developed more tumours. The study raises a health-protection question about combined exposures and susceptibility: could the response depend on an existing biological vulnerability? It cannot show what RF alone would do, and the pattern did not rise steadily with dose.

Original text reviewed | Source checks to 20 October 2026

Lerchl A, Klose M, Grote K, Wilhelm AFX, Spathmann O, Fiedler T, Streckert J, Hansen V, Clemens M. Tumor promotion by exposure to radiofrequency electromagnetic fields below exposure limits for humans. Biochemical and Biophysical Research Communications. 2015; 459(4): 585-590. doi:10.1016/j.bbrc.2015.02.151.

Exposure. UMTS; nominal whole-body SAR 0/0.04/0.4/2 W/kg, gestational day 6 onward; maternal ENU 40 mg/kg injection on day 14; 23.5 h/day for 72 weeks. Freely moving three-mouse cages; reference 20 g/mouse.

Design and controls. 96 female B6C3F1 offspring/group; four ENU groups plus 96 untreated cage controls. Examined organ denominators 91-96. Blinded exposure codes; 107 pathology slides cross-checked externally; Helicobacter PCR negative.

Reported finding. At 0.04 W/kg, lung adenomas 43/94 vs 22/96 sham ($p<.001$), liver carcinomas 28/93 vs 13/96 ($p<.01$). At 0.4 W/kg, lymphoma 23/96 vs 9/96 ($p<.01$), lung carcinoma 92/96 vs 81/96 ($p<.01$). One-tailed Fisher tests; no monotonic dose-response; survival unaffected by RF.

Interpretive limits. ENU coexposure; no RF-only experimental group. SAR variability 36% in reference configuration, 30-91% within cages; local maxima 3-5 times whole-body SAR. High-dose amplifier problem for 20 days; terminal sacrifices differed 1-2 weeks. Not a human compliance test.

Programme and cohort links. Replication/extension of RF-004 in different biology laboratory, with shared engineering investigators Streckert/Hansen and similar waveguide programme. Klose 2015 dissertation is related reporting. Lerchl 2020 adduct follow-up is a distinct mechanistic experiment, not tumour replication.

The protection question raised. Could RF exposure modify tumour development in an already initiated animal? The nonmonotonic findings and variable absorbed dose leave that question distinct from RF-alone cancer initiation or a human exposure threshold.

Source verification. Six-page original proof dated 10 March 2015 reviewed, including Table 1; final citation confirmed with Scite/PubMed. Supplements not independently retrieved.

Source locations. Proof pp 2-3, sections 2.1-2.4/Table 1; p 4 results; p 5 limitations/funding.

Editorial status. No correction/retraction/concern located in Scite/PubMed checks 2026-10-20. Nesslany 2015 DOI 10.1016/j.bbrc.2015.09.114 is a comment.

Funding. German Federal Office for Radiation Protection (BfS), which defined principal design with author; authors declare no conflicts.

Conflicts. Authors declare no conflicts or competing interests, as documented in the retrieved source disclosure.

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RF-004 Earlier experiment behind the tumour promotion finding

> **Why this matters**
> The lung findings helped prompt the later experiment in RF-003. They matter because protecting health includes understanding how exposures act together. Evidence of infection complicated interpretation of the liver results, so the strongest point here is the lung-cancer comparison in chemically treated mice.

Original text reviewed | Source checks to 20 October 2026

Tillmann T, Ernst H, Streckert J, Zhou Y, Taugner F, Hansen V, Dasenbrock C. Indication of cocarcinogenic potential of chronic UMTS-modulated radiofrequency exposure in an ethylnitrosourea mouse model. International Journal of Radiation Biology. 2010; 86(7): 529-541. doi:10.3109/09553001003734501.

Exposure. 1966 MHz generic UMTS; 4.8 W/m² plus maternal ENU 40 mg/kg on gestational day 14; RF from gestational day 6, 20 h/day, 7 days/week. Separate 48 W/m² RF-only group. Whole-body SAR varies with size/posture/cage; a single 0.4 W/kg value requires qualification.

Design and controls. Female B6C3F1 offspring: 60 ENU, 60 ENU+RF, 60 cage controls, 54 sham, 57 RF-only. ENU groups terminated at week 75 after mortality exceeded 75%; other groups continued up to 24 months.

Reported finding. ENU+RF lung carcinoma 45/58 vs 33/60 ENU-only ($p<.05$); multiple lung carcinoma 39/58 vs 18/60 ($p<.001$), two-tailed Fisher. Lung adenoma/carcinoma combined 50/58 vs 42/60 ($p<.05$). ENU-group survival comparable.

Interpretive limits. Pilot: one sex/one ENU+RF intensity, limited protocol organs. Retrospective Helicobacter evidence led authors to exclude liver findings from interpretation. Metastases 15/58 vs 7/60 were not marked significant. Body-temperature checks were prestudy, not throughout pregnant/pup exposure.

Programme and cohort links. Fraunhofer ITEM/Hannover-Wuppertal experiment behind RF-003; the 2008 Reproductive Toxicology abstract (10.1016/j.reprotox.2008.05.039) appears to be precursor reporting, not another study.

The protection question raised. Does the lung-tumour increase reflect a reproducible interaction with chemical initiation? The infection-limited liver interpretation cannot supply equivalent support. This is a combined-exposure question, not evidence of RF-alone tumour causation.

Source verification. Full 13-page original reviewed: Tables I-VII, methods/results/limitations and funding.

Source locations. pp 531-533 methods; p 534 survival; p 535 Tables I-II; pp 536-537 Tables IV-VII; pp 538-539 limitations.

Editorial status. Scite/PubMed and targeted title/DOI notice searches 2026-10-20: no correction/retraction/concern located.

Funding. Compagnia di San Paolo, grant 11581/23718; no conflicts declared.

Conflicts. Authors declare no conflicts or competing interests, as documented in the retrieved source disclosure.

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RF-005 DNA damage in selected rodent tissues

> **Why this matters**
> DNA carries the instructions cells need to function. This NTP experiment found more DNA damage in particular rodent tissues using one test, while another test was negative. Whether these changes persist and help explain disease remains important. This shared programme is distinct from independent confirmation.

Original text reviewed | Source checks to 20 October 2026

Smith-Roe SL, Wyde ME, Stout MD, Winters JW, Hobbs CA, Shepard KG, Green AS, Kissling GE, Shockley KR, Tice RR, Bucher JR, Witt KL. Evaluation of the genotoxicity of cell phone radiofrequency radiation in male and female rats and mice following subchronic exposure. Environmental and Molecular Mutagenesis. 2020;61(2):276-290. doi:10.1002/em.22343.

Exposure. Rats: 900 MHz, 1.5-6 W/kg, about 19 weeks; mice: 1900 MHz, 2.5-10 W/kg, 14 weeks; GSM/CDMA.

Design and controls. Five animals per sex/dose/modulation for genotoxicity; 100- and 150-cell analyses reported.

Reported finding. Comet assays found increased DNA damage in male-mouse frontal cortex, female-mouse blood and male-rat hippocampus under specified conditions. Micronucleus assays were negative.

Interpretive limits. Hippocampal significance depended on cell-scoring approach. Pilot work informed temperature limits; temperature was not monitored during this genotoxicity experiment.

Programme and cohort links. NTP interim cohort, not an independent laboratory replication of RF-001.

The protection question raised. Do the tissue-specific DNA-test changes persist or connect to disease? Their significance depends on assay agreement and downstream function. Shared NTP animals and programme links do not provide independent confirmation of the cancer finding.

Source verification. Original indexed methods/results reviewed in earlier verification work; discussion/disclosures rechecked

Source locations. Results and Discussion; figure captions; statistical methods

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. NTP contracts HHSN291200555544, N01-ES-35514 and HHSN273201300009C; NIH NIEHS intramural programme ZIA ES103316-04.

Conflicts. Authors state no competing financial interests.

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RF-006 Reduced sperm function and oxidative DNA damage in vitro

Why this matters

Movement and DNA integrity matter to sperm function. This laboratory study of isolated human sperm reported impaired movement and survival, with DNA damage at higher exposures. It identifies specific reproductive endpoints worth examining, while leaving open whether comparable injury occurs in the body or affects fertility.

Original text reviewed | Source checks to 20 October 2026

De Iuliis GN, Newey RJ, King BV, Aitken RJ. Mobile Phone Radiation Induces Reactive Oxygen Species Production and DNA Damage in Human Spermatozoa In Vitro. PLoS ONE. 2009;4(7):e6446. doi:10.1371/journal.pone.0006446.

Exposure. 1.8 GHz; 0.4-27.5 W/kg in vitro; 16 hours at 21 C.

Design and controls. 22 normozoospermic donors supplied the sample pool. Figures 1-5 each use three biological replicates, per 2013 correction, not 22 independent replicates per SAR. Controls outside the waveguide;16 h at 21°C; separate heating experiment.

Reported finding. At 1 W/kg, human sperm vitality fell from 89% to 65% and motility from 86% to 68%. Oxidative DNA damage and fragmentation became significant at 2.8 W/kg and above.

Interpretive limits. An in-vitro functional and DNA endpoint, not a human infertility incidence study. Culture SAR cannot be treated as a human local-SAR compliance test.

Programme and cohort links. RF-007 and RF-008 extend the Newcastle research lineage; not independent research networks.

The protection question raised. Do the sperm changes seen in these culture conditions occur within the body and affect reproductive outcomes? Movement, viability and DNA integrity are relevant endpoints, but this isolated-cell experiment cannot establish infertility.

Source verification. Original full text and publisher correction independently rechecked.

Source locations. Figures 2, 4 and 5; correction to Figure 1 and all figure legends

Editorial status. 2013 correction DOI 10.1371/annotation/9a8a0172-3850-4059-b852-72c330769c1b: Figures 1-5 n=3, not 4. Figure 1D mitochondrial ROS 28±1% exposed versus 5±1% control, P=.0056.

Funding. ARC Centre of Excellence in Biotechnology and Development CE0348239 and NHMRC Program Grant 494802; funders reported no role.

Conflicts. Authors declare no competing interests.

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RF-007 Sperm damage after whole body exposure of mice

> **Why this matters**
> This experiment moved beyond sperm in a dish: mice received whole-body exposure for weeks. Their sperm showed functional and DNA damage, yet fertilisation and early embryo development were not impaired. That combination makes sperm injury worth examining carefully without treating it as demonstrated infertility.

Original text reviewed | Source checks to 20 October 2026

Houston BJ, Nixon B, McEwan KE, Martin JH, King BV, Aitken RJ, De Iuliis GN. Whole-body exposures to radiofrequency-electromagnetic energy can cause DNA damage in mouse spermatozoa via an oxidative mechanism. Scientific Reports. 2019;9(1):17478. doi:10.1038/s41598-019-53983-9.

Exposure. 905 MHz; reported whole-body SAR 2.2 W/kg; 12 hours/day for 1, 3 or 5 weeks.

Design and controls. Sham, untreated and RF groups. Figure 5 sperm function n=5-8 mice/group; Figure 7 fragmentation n=5-8, comet n=3, 8-OHdG n=3-5; testis histology n=3. Exposure was 905 MHz, whole-body SAR 2.2 W/kg, 12 h/day for 1/3/5 weeks.

Reported finding. After five weeks, exposed mice had lower sperm vitality and motility and increased sperm DNA fragmentation. Oxidative DNA-base damage increased across exposure periods.

Interpretive limits. Fertilisation and blastocyst development were not impaired in the study. The affirmative claim concerns sperm damage, not a demonstrated loss of fertility.

Programme and cohort links. Same Newcastle research network as RF-006/RF-008; distinct animal experiment.

The protection question raised. Can sperm functional and DNA changes have consequences beyond the measured period? Fertilisation and early embryo development were not impaired here, so cellular injury and demonstrated reproductive impairment remain separate questions.

Source verification. Original full text through Scite and PMC XML rechecked for replicates/disclosures.

Source locations. Figures 5,7,8; Methods; Acknowledgments.

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. NHMRC Australia APP1156997 to G.N.De Iuliis,B.Nixon,B.V.King and R.J.Aitken; B.Houston Australian Postgraduate Award scholarship.

Conflicts. Authors declare no competing interests.

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RF-008 Cell-stage differences plus transient mature-sperm DNA damage and motility loss

> **Why this matters**
> Sperm at different stages did not respond alike. In mature mouse sperm, one DNA test changed temporarily and movement declined, while another DNA test was negative. For health protection, the important question is which responses persist and affect reproduction under realistic conditions, rather than whether any laboratory marker changes.

Original text reviewed | Source checks to 20 October 2026

Houston BJ, Nixon B, King BV, Aitken RJ, De Iuliis GN. Probing the Origins of 1,800 MHz Radio Frequency Electromagnetic Radiation Induced Damage in Mouse Immortalized Germ Cells and Spermatozoa in vitro. Frontiers in Public Health. 2018;6:270. doi:10.3389/fpubh.2018.00270.

Exposure. Continuous-wave 1.8 GHz; main protocol 0.15 W/kg, with 1.5 W/kg supplementary comparison. Germ cells up to 6 h; mature sperm up to 4 h; culture maintained at 23°C. Thermal-probe/numerical SAR calibration.

Design and controls. Cell lines and primary spermatogonia versus somatic cells; mature caudal sperm from three mice per assay, at least three independent replicates. Electron-transport inhibitors and complementary ROS/DNA/function assays.

Reported finding. At0.15 W/kg, mitochondrial ROS rose in GC1/GC2 cells and primary spermatogonia. Mature sperm did not show elevated real-time ROS, but alkaline comet fragmentation increased approximately 20% at 3 h (P<.05; no longer significant 4 h),8-OHdG increased 4 h, and total motility fell from 46% to 35% at 4 h (P<.05). Halo assay was negative.

Interpretive limits. Cell-stage/assay/time dependence is material: no significant exposure-induced germ-cell-line DNA fragmentation, no exposure-related mature-sperm real-time ROS/lipid peroxidation or vitality effect; comet signal transient and halo negative. Small n and nonphysiological culture; no clinical fertility endpoint or human-limit compliance inference.

Programme and cohort links. Newcastle programme with RF-006/007; distinct experiments but one research network.

The protection question raised. Which reproductive-cell stages and exposure durations are vulnerable, and do the measured changes persist? Differences between assays and time points matter; this related laboratory programme does not establish a human fertility effect.

Source verification. Full original methods/results, Figures 1-8, supplements and disclosures rechecked through PMC XML.

Source locations. Results/Figure 1; mature-sperm Results and Figures 5-8; SupplementalFigure 1; Funding.

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Australian Research Council Discovery Project DP110103951 to R.J.Aitken and B.V.King; B.Houston Australian Postgraduate Award PhD scholarship.

Conflicts. Authors declare no commercial/financial relationships construed as conflicts.

Retrieve. DOI or primary source | Original document or related source 1 | PubMed record

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RF-009 Oxidative stress and reduced growth in human cell cultures

> **Why this matters**
> Human cells grown in the laboratory showed chemical stress and multiplied less readily during prolonged LTE exposure. They also showed signs of senescence, a state of arrested cell growth. Whether such responses can impair normal tissue function remains unresolved; the study did not demonstrate disease in people.

Original text reviewed | Source checks to 20 October 2026

Choi J, Min K, Jeon S, Kim N, Pack JK, Song K. Continuous Exposure to 1.7 GHz LTE Electromagnetic Fields Increases Intracellular Reactive Oxygen Species to Decrease Human Cell Proliferation and Induce Senescence. Scientific Reports. 2020;10(1):9238. doi:10.1038/s41598-020-65732-4.

Exposure. 1.7 GHz LTE; culture SAR 1 or 2 W/kg; 72 hours.

Design and controls. Several human cell types; generally at least three independent experiments; calibrated radial-transmission exposure system and refrigerated water cooling.

Reported finding. Continuous 72-h LTE exposure reduced proliferation across normal and cancer-derived human cultures. ROS and senescence increased; antioxidant NAC partly restored proliferation. Assayed DNA-double-strand-break and apoptotic markers did not increase.

Interpretive limits. Culture response, not clinical injury. Proliferation inhibition in cancer and normal cells has different implications. Culture SAR is not a human compliance test. Experimental blinding was not established in the retrieved text.

Programme and cohort links. Separate Korean research network from Newcastle sperm work.

The protection question raised. Could persistent cellular stress and arrested growth impair normal tissue function? The different responses of normal and cancer cells, and the unchanged measured DNA-break and apoptosis markers, constrain a general injury claim.

Source verification. Original indexed results/methods and figure captions rechecked; publisher funding disclosure located

Source locations. Results and Figures 3-8; Materials and Methods exposure calibration; Acknowledgements

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Korean NRF Bio and Medical Technology Development Program NRF-2016M3A9C6918275; Korea Mobile EMF consortium; Choi partially supported by BK21 PLUS.

Conflicts. Not established from retrieved disclosure material.

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RF-010 Exposure related changes in sleep EEG

> **Why this matters**
> Electrical recordings can reveal brain responses that a person may not feel. In this small blinded study, the clearest sleep-brainwave change occurred at the higher exposure; sleep structure was unchanged. Its contribution is a measurable response whose significance for sleep and health still needs to be established.

Original text reviewed | Source checks to 20 October 2026

REGEL SJ, TINGUELY G, SCHUDERER J, ADAM M, KUSTER N, LANDOLT HP, ACHERMANN P. Pulsed radio-frequency electromagnetic fields: dose-dependent effects on sleep, the sleep EEG and cognitive performance. Journal of Sleep Research. 2007;16(3):253-258. doi:10.1111/j.1365-2869.2007.00603.x.

Exposure. 900 MHz pulse-modulated signal; 0.2 or 5 W/kg peak SAR averaged over 10 g; 30 minutes before sleep.

Design and controls. 15 healthy men; randomised double-blind crossover, one week between sessions.

Reported finding. A significant exposure-level trend occurred in fast spindle EEG power and 1-back task speed. Only the 5 W/kg EEG condition differed significantly from sham in the separate post-hoc comparison.

Interpretive limits. The 0.2 W/kg condition alone is not a demonstrated harmful threshold. Sleep architecture was unchanged; some cognitive outcomes improved.

Programme and cohort links. Zurich programme; RF-012 related. A physiological response is not equivalent to diagnosed disease.

The protection question raised. Does a reproducible change in sleeping brain activity affect sleep quality or next-day function? A dose-related EEG response without demonstrated sleep impairment does not itself identify a harmful exposure level.

Source verification. Full indexed article body reviewed

Source locations. Results; Figures 1-3; Table 1 dosimetry caption

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

Retrieve. DOI or primary source | PubMed record

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RF-011 Brainwave changes in a blinded human experiment

> **Why this matters**
> The brain's response differed depending on whether participants' eyes were open or closed. Because exposure and sham sessions were blinded, this is a useful objective observation. Whether these brainwave changes affect function remains unresolved; the recording itself does not show injury.

Original text reviewed | Source checks to 20 October 2026

Wallace J, Shang W, Gitton C, Hugueville L, Yahia-Cherif L, Selmaoui B. Theta band brainwaves in human resting EEG modulated by mobile phone radiofrequency. International Journal of Radiation Biology. 2023;99(10):1639-1647. doi:10.1080/09553002.2023.2187477.

Exposure. 900 MHz GSM; maximum reported 0.70 W/kg over 1 g, 0.49 W/kg over 10 g; about 25.5 minutes.

Design and controls. 21 healthy adults; randomised counterbalanced double-blind crossover; interference checks.

Reported finding. Theta EEG power decreased with eyes open and increased with eyes closed during real versus sham exposure. Theta effects survived cluster-based multiple-comparison testing.

Interpretive limits. Objective electrophysiology, not evidence of clinical injury. Formal FCC-compliance equivalence still requires the complete exposure definition.

Programme and cohort links. Secondary analysis of the earlier Wallace programme; publications from the same participants are one experiment.

The protection question raised. What does this state-dependent brainwave response mean for function? Objective physiology can help define the question, but independent confirmation and a link to an adverse outcome remain missing.

Source verification. Full indexed article body reviewed

Source locations. Methods b-c; Results b; Figures 5-6

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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RF-013 Symptoms associated with measured bedroom fields

> **Why this matters**
> A study of people's homes linked higher estimated base-station fields with more reported headaches, even after adjustment for concern about exposure. This is a reason to investigate symptoms with repeated measurements over time. A single household visit cannot establish whether RF caused the headaches or identify a symptom threshold.

Original text reviewed | Source checks to 20 October 2026

Hutter HP, Moshammer H, Wallner P, Kundi M. Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations. Occupational and Environmental Medicine. 2006;63(5):307-313. doi:10.1136/oem.2005.020784.

Exposure. Predominantly 900-MHz GSM base stations; measured 80 MHz-2 GHz spectrum. Maximum base-station exposure estimated from broadcast channels; categories 0.5 mW/m2; maximum estimate 4.1 mW/m2.

Design and controls. Austrian cross-sectional study; 365 adults, 336 with usable household measurements near ten selected base stations. Logistic regression adjusted for age, sex, region, phone use and fear of adverse effects.

Reported finding. Headache prevalence odds were elevated in the highest estimated bedroom base-station category (>0.5 versus <0.1 mW/m2): adjusted estimate 3.06 (95% CI 1.22-7.67). Sleep quality was not significantly associated with measured exposure.

Interpretive limits. Association based on symptom reports and one bedroom measurement visit; temporality, residual confounding and selection remain unresolved. No multiple-testing correction. Table calls these relative risks, but methods use logistic regression; do not interpret as prospective incidence risk.

Programme and cohort links. Measured environmental fields, distinct from handset call-history studies and from controlled provocation.

The protection question raised. Are the reported headaches related to exposure over time, or to factors this cross-sectional design could not resolve? Adjusted symptom associations matter, but a single measurement visit cannot establish a causal threshold.

Source verification. Original published PDF methods, Table 4, discussion and disclosures reviewed

Source locations. pp.309-310 methods and Table 4; p.312 acknowledgements and competing interests

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Scientific Medical Funds of the Mayor of Vienna; Government of Carinthia.

Conflicts. Authors state none.

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RF-014 Heavy phone use categories in CERENAT

> **Why this matters**
> People reporting the heaviest lifetime phone use had higher odds of two types of diagnosed brain tumour in this French study. Regular use overall showed no significant association. Better exposure histories could help clarify the heavy-use finding, because memory and participation biases can distort such comparisons.

Original text reviewed | Source checks to 20 October 2026

Coureau G, Bouvier G, Lebailly P, Fabbro-Peray P, Gruber A, Leffondre K, Guillamo JS, Loiseau H, Mathoulin-Pélissier S, Salamon R, Baldi I. Mobile phone use and brain tumours in the CERENAT case-control study. Occupational and Environmental Medicine. 2014;71(7):514-522. doi:10.1136/oemed-2013-101754.

Exposure. Recalled mobile-phone use; regular use >=one call weekly for >=six months. Main analyses exclude use in the year before diagnosis; categories based on control distribution, highest >=90th percentile.

Design and controls. France 2004-2006; 253 gliomas, 194 meningiomas, 892 matched controls. Conditional logistic models: glioma adjusted for education and ionising radiation; meningioma for education. Exposure-variable sample sizes vary.

Reported finding. In the >=896-h recalled cumulative-call category, adjusted ORs were 2.89 (95% CI 1.41-5.93) for glioma (24 cases/22 controls in category) and 2.57 (1.02-6.44) for meningioma (13/9). Regular use overall was not significantly associated with either tumour.

Interpretive limits. Retrospective case-control association, with recall/selection bias, proxy interviews, small extreme-use cells and multiple analyses. Call duration is not absorbed dose; no harmful SAR threshold follows. Main Table 3 estimates are distinct from later laterality recalculations.

The protection question raised. Does the heavy-use association reflect a real cancer risk or biases in recalled use and participation? It raises a long-term exposure question without converting reported call time into absorbed dose.

Source verification. Original article PDF methods, Tables 2-4 and disclosures reviewed; journal final metadata verified

Source locations. Original online-first PDF pp.2-3 methods; p.5 Table 3 with covariates; p.6 Table 4; p.8 funding

Editorial status. No formal retraction/concern located in Scite and targeted publisher/DOI search. Author response DOI 10.1136/oemed-2014-102649 revisits laterality analysis; no revised laterality estimate is used here.

Funding. Fondation de France; AFSSET; Association pour la Recherche contre le Cancer; Ligue contre le Cancer; INSERM ATC Environnement et Sante.

Conflicts. Authors state none.

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RF-018 Rat sperm motility and oxidative markers

> **Why this matters**
> A separate laboratory reported poorer sperm movement in exposed rats, alongside signs of chemical stress in reproductive tissues. This broadens the reproductive question beyond one research group. Small samples, separate exposure rooms and missing dose measurements limit conclusions about the cause or relevance to human exposure.

Original text reviewed | Source checks to 20 October 2026

Mailankot M,Kunnath AP,Jayalekshmi H,Koduru B,Valsalan R. Radio Frequency Electromagnetic Radiation (RF-EMR) from GSM(0.9/1.8GHZ) Mobile Phones Induces Oxidative Stress and Reduces Sperm Motility in Rats. Clinics.2009;64(6):561-565. DOI10.1590/S1807-59322009000600011.

Exposure. Dual-band 0.9/1.8 GHz commercial handset; no animal SAR or incident field reported. Pre/post facial temperatures were not significantly different.

Design and controls. Male Wistar rats,n=6/group. Active GSM phone or battery-free phone 1 h/day for 28 days inside a wooden-bottom nested cage; separate similar exposure rooms; freely moving animals.

Reported finding. Motile sperm 43.08±10.03% exposed versus 71.97±8.7% control,P<.002. Testis/epididymis MDA increased and GSH decreased. Total sperm count was not significantly different,P=.052.

Interpretive limits. Small groups; rooms confounded with treatment; facial thermometry does not exclude organ heating; no dose assessment or mating/fertility outcome.

The protection question raised. Does this separate laboratory sperm finding persist when exposure-room and thermal uncertainties are resolved? The missing absorbed dose prevents a quantitative comparison with human protection.

Source verification. Original PMC XML full methods/results reviewed; upgraded from record only.

Source locations. Animals; Mobile Phone Exposure; Results; Figures 1-2.

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. No specific funding statement located; technical assistance acknowledged from Clinical Embryology,Kasturba Medical College.

Conflicts. No declaration located in retrieved original; absence of conflicts not inferred.

Retrieve. DOI or primary source | Original document or related source 1 | PubMed record

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RF-019 Sperm motility and fragmentation in paired samples

> **Why this matters**
> By dividing each semen sample, researchers compared exposed and control portions from the same donor. They reported poorer forward movement and more fragmented sperm DNA after exposure. That design is useful for investigating sperm function, but different temperature-controlled chambers and unmeasured sample dose leave important alternative explanations.

Original text reviewed | Source checks to 20 October 2026

Gorpinchenko I, Nikitin O, Banyra O, Shulyak A. The influence of direct mobile phone radiation on sperm quality. Central European Journal of Urology. 2014;67(1):65-71. doi:10.5173/ceju.2014.01.art14.

Exposure. GSM900/1800 MHz phone,5 cm,5 h; no sample SAR or incident-field measurement reported.

Design and controls. Paired aliquots from 32 normozoospermic men,aged 27-40;5 h in separate thermostats at 27°C. GSM900/1800 MHz phone 5 cm from exposed samples,standby with calls every 10 min.

Reported finding. After 5 h,progressive motility 66.5±6.3% exposed versus 81.3±7.2% control; SCD fragmentation 8.8±2.2% versus 4.2±1.8%,both P<.05. Vitality,count,morphology and immotile fraction unchanged.

Interpretive limits. Separate thermostats may confound treatment; no sample dose or continuous temperature monitoring; nonphysiological 27°C culture. Ejaculate exposure is not in-vivo testis exposure or clinical fertility.

The protection question raised. Are the reported semen effects attributable to RF under adequately characterised chamber and temperature conditions? This independent human-sample strand remains limited by the original exposure information available.

Source verification. Original full PMC XML,Table 2 and results reviewed; upgraded from abstract.

Source locations. Methods; Table 2; Figures 4-5.

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. No funding statement located in original.

Conflicts. Not established from original.

Retrieve. DOI or primary source | Original document or related source 1 | PubMed record

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RF-020 Blinded rat histology with significant findings at two of three exposure levels

Why this matters

When brain tissue was examined weeks after a two-hour RF exposure, a blinded examiner found higher scores for dark-staining nerve cells in some rat groups. This makes delayed tissue changes worth examining. The lowest dose was nonsignificant and independent replication did not confirm the result; permanent human injury was not established.

Original text reviewed | Source checks to 20 October 2026

Salford LG, Brun AE, Eberhardt JL, Malmgren L, Persson BRR. Nerve cell damage in mammalian brain after exposure to microwaves from GSM mobile phones. Environ Health Perspect. 2003;111(7):881-883. doi:10.1289/ehp.6039.

Exposure. GSM telephone fed into TEM cells, no voice modulation; nominal 915-MHz programme. Average whole-body SAR 0.002, 0.02, or 0.2 W/kg for 2 h; corresponding peak power densities 0.24, 2.4, 24 W/m². Brain SAR distribution simulated by FDTD, varying <6 dB. Room/cell air temperature controlled.

Design and controls. 32 male and female Fischer 344 rats aged 12-26 weeks, 8/group including sham; awake, movable within chamber. Perfusion fixation; cresyl-violet staining and albumin immunohistochemistry. Neuropathologist blinded; dark-neuron score 0/1/2 (none/occasional, moderate, abundant).

Reported finding. After one 2-h exposure and approximately 50 days' recovery, the blinded semiquantitative dark-neuron score differed across four groups (Kruskal-Wallis $p<0.002$). Individual comparisons with sham: 0.002 W/kg $p<0.2$; 0.02 W/kg $p=0.01$; 0.2 W/kg $p=0.03$. The lowest group was not significant. Changes occurred particularly in cortex, hippocampus and basal ganglia.

Interpretive limits. Small mixed-sex, broad-age sample and subjective ordinal endpoint. Histology is a reported pathological endpoint, but permanent functional injury, apoptosis and human disease were not established. Direct replication did not confirm the outcome (Masuda 2009). Rat whole-body SAR cannot certify human local-SAR compliance.

Programme and cohort links. Same Lund programme as RF-027, RF-028, RF-029 and CTX-005. CTX-006 is a specific independent replication attempt, not a general safety review.

The protection question raised. Is the delayed dark-neuron signal reproducible and specific to injury rather than tissue handling or scoring? Its small sample, nonsignificant lowest dose and nonconfirming replication prevent a harmful human exposure threshold.

Source verification. Original three-page journal PDF read, including embedded correction; Scite supplied abstract only

Source locations. pp. 881-882 Materials and Methods; p.882 Results, Figure 3 and caption; p.883 Correction

Editorial status. Embedded Correction on p.883 replaces Figure 1 from earlier immediately-sacrificed animals with present-study specimens (sham and 2 mW/kg). No separate notice DOI identified. Scite metadata did not surface this correction. No retraction/concern found in targeted search.

Funding. Swedish Council for Work Life Research grant (p.881).

Conflicts. Authors declare no conflict of interest (p.881).

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RF-021 Changed cortical gene expression after a 5G signal

> **Why this matters**
> Genes help cells respond and carry out their work; measuring which genes are active can reveal a biological response. Repeated exposure to a 5G signal changed gene activity in mouse brain tissue without altering behaviour. The health question is whether those changes have lasting consequences.

Original text reviewed | Source checks to 20 October 2026

Lameth J, Royer J, Martin A, Marie C, Arnaud-Cormos D, Lévêque P, Poirier R, Edeline JM, Mallat M. Repeated Head Exposures to a 5G-3.5 GHz Signal Do Not Alter Behavior but Modify Intracortical Gene Expression in Adult Male Mice. International Journal of Molecular Sciences. 2025;26(6):2459. doi:10.3390/ijms26062459.

Exposure. 5G-modulated 3.5 GHz; one hour/day, five days/week; 27 exposure hours over six weeks for transcriptomics. Whole-brain average SAR 0.19 W/kg.

Design and controls. 32 male mice overall; exposed, pseudo-exposed and unrestrained controls. Transcriptomics n=7 exposed and n=8 pseudo-exposed.

Reported finding. RNA sequencing detected 77 differentially expressed genes in the right sampled cortex and 84 in the left after repeated exposure, using false-discovery-rate correction.

Interpretive limits. Behaviour was not altered. Gene-expression change is a biological response, not demonstrated brain injury. Preprint and journal paper are the same work.

Programme and cohort links. Distinct French research programme.

The protection question raised. Do the gene-expression changes persist or alter brain function? Behaviour was unchanged. A signal-specific biological response is useful to scientific understanding without establishing injury or measuring population cumulative exposure.

Source verification. Full indexed article body reviewed

Source locations. Results RNA sequencing; Figure 1; protocol Figure 2

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Crossref publisher-deposited metadata lists French ANSES environmental and occupational health programme grant 2020/2 RF/14. Complete original funding/disclosure statement not rechecked.

Conflicts. Not established from retrieved material.

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RF-022 Reproductive endpoints at 3.5 and 24 GHz

> **Why this matters**
> This rat study brings reproductive endpoints into research at 3.5 and 24 GHz. It reported changes in sperm function and biological signals in the testes. Yet dose at the animals and reproductive-organ temperature were not measured, preventing a confident separation of RF effects from heating or a comparison with human limits.

Original text reviewed | Source checks to 20 October 2026

Syed Taha SMA, Jaffar FHF, Hairulazam A, Vijay S, Jamaludin N, Zulkifli AF, Mat Ros MF, Osman K, Zakaria Z, Mohd Bahar MAA, Ibrahim SF. Prolonged 3.5 GHz and 24 GHz RF-EMF Exposure Alters Testicular Immune Balance, Apoptotic Gene Expression, and Sperm Function in Rats. Biomedicines. 2025;13(10):2471. doi:10.3390/biomedicines13102471.

Exposure. 3.5 or 24 GHz; one or seven hours/day for 60 days.

Design and controls. Six rat groups, n=6 per group, including corresponding controls.

Reported finding. Authors report frequency/duration-specific sperm-function, cytokine and apoptosis-gene changes.

Interpretive limits. The methods explicitly state that animal-position SAR/power density and scrotal/core temperatures were not measured. No below-limit or nonthermal attribution is justified.

The protection question raised. Can the reported reproductive effects be separated from heating and quantified exposure? Without verified absorbed dose or organ temperature, neither a nonthermal mechanism nor harm under human-limit conditions follows.

Source verification. Full indexed article body reviewed

Source locations. Exposure Experimental Setup; Conclusions

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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RF-025 Genetic profiling of rat gliomas and cardiac schwannomas from life-time radiofrequency radiation exposure study using a targeted next-generation sequencing gene panel

> **Why this matters**
> Looking inside rat tumours can help researchers ask how closely the disease resembles human cancer. This paper examines tissues from the Ramazzini animals, so it adds biological detail rather than a new cancer experiment. Its corrected findings do not show that RF caused the mutations.

Original text reviewed | Source checks to 20 October 2026

Brooks AM, Vornoli A, Kovi RC, Ton TVT, Xu M, Mashal A, Tibaldi E, Gnudi F, Li JL, Sills RC, Bucher JR, Mandrioli D, Belpoggi F, Pandiri AR. Genetic profiling of rat gliomas and cardiac schwannomas from life-time radiofrequency radiation exposure study using a targeted next-generation sequencing gene panel. PLOS ONE. 2024;19(1):e0296699. Correction: PLOS ONE. 2026;21(4):e0346498. doi:10.1371/journal.pone.0296699.

Exposure. Reuses RI 1.8 GHz lifetime tissues from RF-002 and one-year interim exposed non-tumour brain samples; 0/5/25/50 V/m. The 2024 methods print 1.001 W/kg, inconsistent with RF-002's 0.001 W/kg for the lowest exposed group. No published correction to that SAR detail was verified.

Design and controls. 14 gliomas (12 exposed, 2 spontaneous), 9 cardiac schwannomas (8 exposed, 1 spontaneous); 30 interim exposed non-tumour brains; age-matched non-tumour controls; matched kidney genomic controls.

Reported finding. Targeted 23-gene panel found some rat tumour variants homologous to human cancer variants; canonical human IDH1 R132/IDH2 R172 hotspots were absent. Morphology and mutation profiles inform translation, not proof that RF caused the mutations.

Interpretive limits. Same bioassay, not independent cancer replication; small spontaneous-tumour comparator; targeted panel, not whole genome; no dose-related interim non-tumour mutation frequency.

Programme and cohort links. RF-002 tissue follow-up; RI/NIEHS collaboration. Count once as cohort.

The protection question raised. How closely do these rat tumours resemble human cancers? Molecular comparisons can inform translation, while species differences and reused tissues prevent a new incidence finding or proof that RF caused particular mutations.

Source verification. Publisher full original methods/results/discussion, funding and 2026 correction reviewed; Scite flags correction.

Source locations. Methods: Tissue samples, NGS analysis; Results: brain tumours, interim group, cardiac schwannomas; 2026 correction main text/Fig 4.

Editorial status. Correction dated 2026-04-02 (10.1371/journal.pone.0346498) replaces Fig 4 and changes interim-sample variant counts to at least 15/30 (50%) and 11/30 (37%). Not retracted.

Funding. NIEHS/DNTP intramural support and Children with Cancer UK; no competing interests declared; funders no role.

Conflicts. Authors declare no conflicts or competing interests, as documented in the retrieved source disclosure.

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RF-026 Long-term, low-level microwave irradiation of rats

> **Why this matters**
> An older, long-running rat experiment found more animals with malignant tumours when different cancer types were combined. No single tumour site or overall tumour total increased significantly. Its value is historical evidence for a fresh look at the underlying pathology, without calling it another confirmation of the heart-tumour finding.

Original text reviewed | Source checks to 20 October 2026

Chou CK, Guy AW, Kunz LL, Johnson RB, Crowley JJ, Krupp JH. Long-term, low-level microwave irradiation of rats. Bioelectromagnetics. 1992;13(6):469-496. doi:10.1002/bem.2250130605.

Exposure. 2450 MHz pulsed microwaves: 800 pulses/s, 10 µs width, 8 Hz square-wave modulation; whole-body SAR approximately 0.4 to 0.15 W/kg as animals grew; 21.5 h/day, 7 days/week, 25 months from 8 weeks of age. Individual circularly polarised waveguides.

Design and controls. 100 male Sprague-Dawley rats exposed, 100 sham; 10/group interim sacrifice at 13 months; 200 animals examined pathologically.

Reported finding. Primary malignant lesions in 18/100 exposed vs 5/100 sham. Age-stratified Mantel-Haenszel OR 4.27, p=.006; death-time surrogate analysis p=.006. No significant specific tumour-site increase or all-neoplasms increase; longevity comparable.

Interpretive limits. Pooling across histologically different organs/tumours and numerous endpoints limits inference; one intensity/one sex; authors considered biological meaning uncertain. SAR declines with growth. Not a demonstrated schwannoma replication.

Programme and cohort links. University of Washington/USAF programme. Guy et al. 1985, USAFSAM-TR-85-64, Volume 9/AD-A159512, and component reports describe the same animals; count once.

The protection question raised. How much weight can the pooled malignant-lesion increase carry when single-site and overall-neoplasm results were not significant? It remains relevant chronic-pathology history, with uncertainty about biological meaning.

Source verification. Original 28-page published article reviewed, including Tables 2-4, survival and discussion; 1985 USAF summary checked as same programme.

Source locations. pp 469-470 abstract; p 478 exposure; pp 487-488 survival/pathology; p 490 Table 2; p 491 statistics; p 492 Table 4; pp 493-494 interpretation/funding.

Editorial status. Scite metadata/publisher and targeted DOI-notice search 2026-10-20: no correction/retraction/concern located.

Funding. USAF School of Aerospace Medicine contracts F33615-78-C0631 and F33615-80-C-0612; partial NCI grant CA33752. No modern conflict declaration located.

Conflicts. Not established from retrieved material.

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RF-028 Delayed albumin leakage, with a specific significant exposure comparison

> **Why this matters**
> Researchers detected a blood protein outside brain vessels a week after exposure, suggesting altered control of passage into brain tissue. They did not find an increase in dark-staining nerve cells. The question is whether the reported barrier change is reproducible and has consequences for brain function.

Original text reviewed | Source checks to 20 October 2026

Nittby H, Brun A, Eberhardt J, Malmgren L, Persson BRR, Salford LG. Increased blood-brain barrier permeability in mammalian brain 7 days after exposure to the radiation from a GSM-900 mobile phone. Pathophysiology. 2009;16(2-3):103-112. doi:10.1016/j.pathophys.2009.01.001.

Exposure. GSM-900; 2 h in TEM cells; whole-body SAR sham, 0.12, 1.2, 12, 120 mW/kg; 7-day recovery.

Design and controls. 48 male/female Fischer 344 rats aged 2-3 months: 8/group at four active levels, 16 sham. Awake, movable; 7-day recovery; perfusion fixation, albumin immunohistochemistry/cresyl-violet; pathologist blinded; ordinal scoring. Brain-SAR orientation variability 56% standard deviation around mean.

Reported finding. At 7 days, focal albumin leakage occurred in 25% of exposed versus 0% sham rats (Fisher exact p=0.04); five-group score comparison p=0.012. The 12 mW/kg comparison was significant (p=0.007); 0.12 and 120 mW/kg were trends (p=0.1). Neuronal albumin uptake differed globally (p=0.03) and at 1.2 mW/kg versus sham (p=0.02). Dark-neuron occurrence did not differ.

Interpretive limits. Original article-in-press proof reviewed. These are BBB/albumin endpoints, without increased dark-neuron pathology at 7 days. Trends are not significant findings. No claim of human-limit compliance from rat whole-body SAR.

Programme and cohort links. Same Lund research programme as RF-020 and RF-027; separate 7-day experiment, not the 14/28-day animals.

The protection question raised. Is the delayed barrier change reproducible, and does it affect brain function? Passage of a blood protein into tissue is distinct from neuronal injury; dark neurons did not increase here.

Source verification. Original 10-page article-in-press proof hosted by California Council on Science and Technology; methods, results and funding reviewed

Source locations. Proof pp.5-7 sections 3.1-3.4 and 4; p.8 Acknowledgements; final pp.103-112

Editorial status. Scite metadata checked 20 Oct 2026; no editorial notice returned. Primary bibliographic records and targeted notices were checked where retrievable; no retraction or expression of concern located. This is not certification that no notice exists.

Funding. Hans and Märit Rausing Charitable Foundation (proof p.8).

Conflicts. No competing-interest declaration located in original proof; no inference.

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RF-032 Blinded experiment joins sleep physiology to reduced task improvement

> **Why this matters**
> A good night's sleep is something people value for how they function the next day. Under this study's unusual pulsed exposure, young men slept slightly less and showed less overnight improvement on one tapping-task measure. That functional result deserves independent testing; it does not establish a sleep disorder.

Original text reviewed | Source checks to 20 October 2026

Lustenberger C, Murbach M, Dürr R, Schmid MR, Kuster N, Achermann P, Huber R. Stimulation of the brain with radiofrequency electromagnetic field pulses affects sleep-dependent performance improvement. Brain Stimul. 2013;6(5):805-811. doi:10.1016/j.brs.2013.01.017.

Exposure. 900 MHz; 7.1-ms pulses at 10 W/kg peak spatial SAR over 10g, arranged in bursts (1 W/kg burst average); all-night average 0.15 W/kg. Repeated 18-min on/off pattern; six-minute average never exceeded 0.34 W/kg.

Design and controls. 16 healthy men aged 18-21; randomized double-blind crossover, adaptation before each of two nights; objective sleep EEG and evening/morning tapping task.

Reported finding. Overnight reduction in motor-task response-time variance was smaller during exposure (28.97%) than sham (49.04%; adjusted condition p=0.03). Sleep time fell 9.23 min (p=0.04); slow-wave activity increased in fourth NREM episode.

Interpretive limits. Small experimental study; response-time variance is a task endpoint, not diagnosed memory disease. Unusual pulse pattern and different SAR averaging periods must be preserved; no automatic inference of human-limit compliance.

Programme and cohort links. Zurich sleep/dosimetry programme overlaps investigators in RF-010/RF-012; this is a distinct experiment, not an independent institution's replication.

The protection question raised. Could exposure-related changes in sleep and overnight task improvement matter to daily function? The small experiment and unusual pulse/averaging conditions leave clinical significance and a general protective threshold unresolved.

Source verification. Original author/institution-hosted uncorrected proof reviewed; citation/result direction checked against PubMed publication record

Source locations. Proof pp.2-4 Methods, Statistics, Results; Figures 1-3; p.1 funding/conflicts; final pp.805-811

Editorial status. Scite metadata checked 20 Oct 2026; no editorial notice returned. Primary bibliographic records and targeted notices were checked where retrievable; no retraction or expression of concern located. This is not certification that no notice exists. Source is an uncorrected proof, not an independently counted publication.

Funding. Swiss Research Foundation on Mobile Communication (FSM A2008-08); Swiss National Science Foundation (P00A-114923).

Conflicts. Authors declared no competing interests (proof p.1).

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RF-033 Prospective cognitive-test association with modeled brain dose

Why this matters

This study followed adolescents and measured changes in memory rather than asking only how much they used phones. A negative association appeared in the subgroup with operator records, while the full-sample estimate was not significant. Developmental function matters; this qualified pattern deserves independent scrutiny.

Original text reviewed | Source checks to 20 October 2026

Foerster M, Thielens A, Joseph W, Eeftens M, Röösli M. A Prospective Cohort Study of Adolescents' Memory Performance and Individual Brain Dose of Microwave Radiation from Wireless Communication. Environ Health Perspect. 2018;126(7):077007. doi:10.1289/EHP2427.

Exposure. Individually modeled RF brain dose combining device/network use and absorption estimates; operator records calibrate call time; personal measurements/geospatial modeling for far-field sources. Dose is modeled, not directly measured absorbed energy; typical use dominated by GSM calling.

Design and controls. HERMES Swiss school cohort, two sampling waves 2012-2016; 843 followed; 670 completed figural tests, 669 in full adjusted model. Standardized computerized memory testing approximately 1 year apart; regression adjusted for demographic, lifestyle and baseline-score factors.

Reported finding. Adjusted figural-memory change per IQR brain dose: full sample n=669, -0.22 points (95% CI -0.47 to 0.03; IQR 953 mJ/kg/day), not statistically significant. Operator-data subgroup n=274: -0.26 (-0.42 to -0.10; IQR 341 mJ/kg/day). Right-side users n=532: -0.39 (-0.67 to -0.10).

Interpretive limits. Observational association, residual confounding and exposure-model uncertainty. Full-sample estimate crosses zero. No clinical injury or causal inference established. This expands/reanalyzes the first wave reported by Schoeni 2015 plus newly recruited wave; not wholly independent replication.

Programme and cohort links. Swiss TPH/Basel with Ghent/IMEC dosimetry; separate from Zurich sleep experiments. Schoeni 2015 first-wave DOI 10.1016/j.envint.2015.09.025 is not an independent cohort.

The protection question raised. Could developmental cognition be associated with exposure? The operator-data subgroup finding deserves scrutiny alongside the nonsignificant full-sample estimate, cohort overlap and exposure-model uncertainty; it does not establish a harmful dose.

Source verification. Original indexed body read for methods/results/limitations, primary publisher searchable record and original journal PDF checked for math/funding/COI

Source locations. Methods; Table 3 and Figure 2; laterality Figure 3; Discussion 'Comparison with Previous Analysis'; Acknowledgments; PDF first page conflicts

Editorial status. Scite metadata checked 20 Oct 2026; no editorial notice returned. Primary bibliographic records and targeted notices were checked where retrievable; no retraction or expression of concern located. This is not certification that no notice exists.

Funding. Swiss National Science Foundation project 138190; European Community FP7 GERONIMO grant 603794.

Conflicts. Authors declared no actual or potential competing financial interests.

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RF-035 Transformation phenotypes and tumors after transplanting exposed fibroblasts

Why this matters

Cells given prolonged, high-dose laboratory exposure later formed tumours when transplanted into mice with weakened immune defences. This is a consequential endpoint to investigate. One assay used a tumour-promoting chemical, and unclear cell histories prevent an RF-alone claim. The recipient mice were not RF-exposed.

Original text reviewed | Source checks to 20 October 2026

Ding Z, Xiang X, Li J, Wu S. Molecular Mechanism of Malignant Transformation of Balb/c-3T3 Cells Induced by Long-Term Exposure to 1800 MHz Radiofrequency Electromagnetic Radiation (RF-EMR). Bioengineering. 2022;9(2):43. doi:10.3390/bioengineering9020043.

Exposure. 1.8-GHz continuous wave; culture average SAR 8 W/kg, 4 h/day for 40 or 60 days, sXc-1800 waveguide system. Authors selected high culture SAR to accelerate transformation. Fans/air-temperature monitoring and medium-response assessment described; alarm threshold for temperature difference 0.1°C. This is not a measurement of human-limit compliance.

Design and controls. Sham/exposed Balb/c-3T3 cultures passaged together; experiment and analysis blinded. Reporting describes triplicate experiments with triplicate samples but does not clearly distinguish independent biological batches from subsamples. Focus assay: 30-day post-exposure culture; TPA 0.1 μg/mL for 24 h on days 1, 4 and 7 in sham and exposed cultures. Transplantation: 2×10^6 cells/mouse, 4-week observation; HeLa positive control. Soft agar: 4 weeks; HeLa positive control. Student t tests; no multiple-testing adjustment stated.

Reported finding. Original results report transformation foci, soft-agar colonies, and visible tumors four weeks after transplanting 40- or 60-day exposed Balb/c-3T3 cells into SCID mice; sham cultures lacked foci/colonies and mice receiving sham cells lacked tumors. The focus assay included TPA treatment of all groups. Exact effect sizes, mouse denominators and culture-lineage independence were not adequately specified for a quantitative risk estimate.

Interpretive limits. Distinct endpoint beyond a transient biomarker, but donor-cell passage/TPA history before transplantation and soft agar is not explicit, and mouse group size/biological replicate structure are unclear. Thus describe reported tumorigenicity of exposed-cell preparations, not proved RF-alone cancer initiation or spontaneous cancer in RF-exposed mice. Only one high culture SAR; no dose-response series or independent replication established. Pathway enrichment does not establish causal mediation.

Programme and cohort links. Separate Shenzhen/Beijing programme from NTP, Ramazzini, Newcastle and Lund. Culture phenotyping and transplanted-cell tumors are components of the same study, not independent replications. No linked preprint/duplicate version located.

The protection question raised. Would reproducible transformation and tumour formation from exposed-cell preparations strengthen a cancer-hazard inference? High culture dose, chemical-promoter use and unclear donor-cell histories prevent an RF-alone initiation claim here.

Source verification. Original open-access publisher/PMC article passages retrieved via indexed primary records; Methods 2.1-2.6 and 2.10, Results 3.1-3.4 and disclosures checked.

Source locations. Methods sections 2.1-2.3 exposure/sham/TPA; 2.5-2.6 transplantation/soft agar; 2.10 statistics; Results sections 3.1, 3.3, 3.4 and Figures 2, 4, 5; Funding and Conflicts of Interest.

Editorial status. PubMed, PMC/publisher primary records and targeted DOI/title notice search checked 20 Oct 2026; no correction, retraction or concern found. This does not certify that no notice exists.

Funding. Shenzhen High-level Hospital Construction Fund; Shenzhen Key Medical Discipline Construction Fund SZXK013; Sanming Project of Medicine in Shenzhen SZSM201612063.

Conflicts. Authors declare no conflict of interest. Ethics approval: North China University of Technology (2017014, 7 March 2017). Data available from corresponding author on reasonable request.

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RF-036 Persistent mouse behavioural changes after early-life LTE exposure

> **Why this matters**
> Early development is a period when lasting changes deserve particular attention. Mice exposed before and shortly after birth later behaved differently in several activity measures. The directions varied between exposure groups and the samples were small. Their significance for developmental function remains uncertain; they do not establish a human diagnosis.

Original text reviewed | Source checks to 20 October 2026

Kerry A. Broom; Richard Findlay; Darren S. Addison; Cristian Goiceanu; Zenon Sienkiewicz. Early-Life Exposure to Pulsed LTE Radiofrequency Fields Causes Persistent Changes in Activity and Behavior in C57BL/6 J Mice. Bioelectromagnetics. 2019;40(7):498-511. doi:10.1002/bem.22217.

Exposure. 1,846 MHz realistic LTE downlink; nominal whole-body average SAR 0.5 or 1 W/kg; 30 min/day, 5 days/week on gestational days 13.5-18.5 and postnatal days 3-21. Numerical mother/fetus/pup dosimetry; fetal SAR varied substantially (0.125-1.39 W/kg at maternal target 0.5 W/kg).

Design and controls. Random allocation of pregnant C57BL/6J mice; sham exposure with field set to zero. Only male offspring, at most one per litter per group. Two experimental repetitions; behavioural n=5-6/group at most timepoints, n=3 at weeks 14,21,28 after equipment data loss; body mass n=3/group.

Reported finding. Persistent changes in locomotion, feeding/drinking and shelter use were recorded through adulthood. At 0.5 W/kg distance moved and movement duration were reduced; at 1 W/kg wheel revolutions and movement duration increased. Both exposed groups spent longer in shelter.

Statistical detail. Distance moved: exposure ANOVA $F(2,212)=10.09$, $P<.001$; 0.5 W/kg versus sham $P\leq.001$. Movement duration: $F(2,212)=14.45$,$P<.001$. Wheel revolutions: 1W/kg versus sham $P\leq.006$.

Interpretive limits. Small male-only groups, numerous repeated endpoints and no consistent monotonic exposure-response. Changes are biological/behavioural responses of uncertain adverse significance, not diagnosed neurodevelopmental disease. Nominal whole-body exposures exceeded human public whole-body restrictions; no inference of ordinary handset risk or human compliance follows.

Programme and cohort links. UK Public Health England/ELERaFF programme, distinct from Yale Aldad and Newcastle sperm work. BIOEM2016 conference presentation is preliminary reporting of this research, not a separate experiment.

The protection question raised. Can early-life exposure produce persistent changes that impair function? Small samples and effects in different directions leave adverse significance uncertain; these behavioural responses are not a human developmental diagnosis.

Source verification. Full original methods/results, Table 1, figures and disclosures reviewed via Scite fulltext and PMC original XML.

Source locations. Methods/Animals and Exposures; Table 1; Results/Figures 2-4; Discussion; article footnote and Acknowledgments.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. UK Department of Health and Social Care Policy Research Programme, ELERaFF 091/0211.

Conflicts. Richard Findlay works for an independent EMF safety organization; Zenon Sienkiewicz was an ICNIRP main-commission member; both declare no competing interests (article footnote).

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RF-037 LTE effects on sound-evoked cortical activity during experimental inflammation

> **Why this matters**
> An inflammatory challenge changed how rats' hearing-related brain cells responded to LTE exposure. In the challenged animals, sound responses weakened and stronger sounds were needed to evoke them. This gives susceptibility a concrete experimental form; recordings from anaesthetised rats do not establish hearing loss in people.

Original text reviewed | Source checks to 20 October 2026

Samira Souffi; Julie Lameth; Quentin Gaucher; Délia Arnaud-Cormos; Philippe Lévêque; Jean-Marc Edeline; Michel Mallat. Exposure to 1800 MHz LTE electromagnetic fields under proinflammatory conditions decreases the response strength and increases the acoustic threshold of auditory cortical neurons. Scientific Reports. 2022;12(1):4063. doi:10.1038/s41598-022-07923-9.

Exposure. Single 2-hour head-only LTE-1800 MHz exposure under ketamine/xylazine anaesthesia. Mean brain SAR 0.37±0.23 W/kg; auditory cortex 0.50±0.08 W/kg (model SD). FDTD and homogeneous-phantom temperature-rise dosimetry differed by <30%. Heating pad maintained body temperature around 37°C.

Design and controls. Adult male Wistar rats. Electrophysiology in four groups of five rats: LTE/sham with and without LPS250µg/kg i.p.24 h earlier. Separate LPS histology n=5-6/group. Exposure-zero sham; waveform trigger and histological analyses blinded.

Reported finding. In LPS-treated rats, LTE exposure reduced sound-evoked response strength and increased cortical sound thresholds. The same principal response-strength and threshold effects were not seen in healthy rats. Microglial morphology was not significantly changed.

Statistical detail. LPS groups: total tone-evoked response strength P=.0017; response at best frequency P=.0445; vocalization-response strength/reliability P=.043. Analyses used electrode-array positions to address nesting, with 25-26 positions but only 5 animals/group.

Interpretive limits. An acute anaesthetized inflammatory animal model; cortical physiology does not demonstrate clinical deafness or human EHS. Small animal-level n and analyses based on multiple electrode positions leave uncertainty over full between-animal generalizability. No human-limit compliance conclusion from cortex SAR.

Programme and cohort links. French ANSES/Paris-Sud-Limoges neuroinflammation programme; linked to earlier GSM experiments and Lameth 2025 RF-021. Journal DOI and preprint 10.1101/2022.01.13.476168 are one work, not independent evidence.

The protection question raised. Can an inflammatory state change vulnerability to exposure? The contrast with healthy rats makes susceptibility testable, while anaesthesia, small animal numbers and electrode-level analyses limit inference about people or hearing loss.

Source verification. Original full methods/results, Table 1,Figures 2,4-5 and disclosures reviewed via Scite fulltext and PMC original XML.

Source locations. Methods/Animals,LTE head exposure,Electrophysiological recordings,Statistical analysis;Table 1;Fig 2;Fig 4 versus Fig 5.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. ANSES French National Research Program for Environmental and Occupational Health 2018/2RF/16; Investissements d'avenir ANR-10-IAIHU-06.

Conflicts. Authors declare no competing interests in original article.

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RF-038 Mobile-phone-use associations with objectively measured semen count

Why this matters

This study measured semen in young men and found an association between frequent phone use and lower total sperm count. It brings a human reproductive endpoint into the discussion. Because phone use was self-reported and RF dose was unmeasured, it cannot identify radiation as the cause or diagnose infertility.

Original text reviewed | Source checks to 20 October 2026

Rita Rahban; Alfred Senn; Serge Nef; Martin Röösli. Association between self-reported mobile phone use and the semen quality of young men. Fertility and Sterility. 2023;120(6):1181-1192. doi:10.1016/j.fertnstert.2023.09.009.

Exposure. Self-reported mobile-phone-use frequency and phone placement,2005-2018. No personal RF field or testicular absorbed-dose measurements; network generation/output power not individually established.

Design and controls. Nationwide Swiss cross-sectional conscription sample:2,886 men aged 18-22;2,759 reported usage frequency. Standardized semen analysis near 6 recruitment centres; adjusted regression, multiple imputation.

Reported finding. Higher usage frequency associated with lower total sperm count and a concentration trend. For >20 uses/day versus 1-5/day, adjusted total-count coefficient was negative; the corresponding concentration confidence interval included zero. No consistent adverse motility/morphology association, and pocket carriage was not associated with lower semen measures.

Statistical detail. Table 2, >20/day: cubic-root-transformed total-count β=-0.271(95%CI-0.515,-0.027),P=.030; concentration β=-0.152(CI-0.316,0.011),P=.067. Continuous frequency trends were significant for concentration(P=.032) and count(P=.012),Table 3.

Interpretive limits. Cross-sectional association with phone-use behaviour, not established RF causation or measured infertility. Self-selection (~3% contributed biological material), residual lifestyle confounding, one semen sample and substantial exposure misclassification. Cubic-root coefficients are not percentage reductions. Temporal attenuation does not identify a causal mechanism.

Programme and cohort links. Swiss SCAHT/University of Geneva reproductive programme with Swiss TPH. Same conscription collection later used in geographical/semen studies; do not count those as independent RF replications.

The protection question raised. Is the association with total sperm count related to RF or to other aspects of phone use and lifestyle? Unmeasured dose and cross-sectional sampling leave causation and fertility consequences unresolved.

Source verification. Original published 13-page PDF retrieved from University of Geneva archive; methods,Tables 2-3,Discussion and declarations reviewed. Scite metadata confirmed; no readable Scite fulltext.

Source locations. Original pp 1182-1183/Methods;Table 2 p1185;Table 3 p1186;limitationsp 1189;declarationsp 1190.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. Swiss Centre for Applied Human Toxicology(SCAHT) and Département de l'Instruction Publique,state of Geneva(toS.N.).

Conflicts. R.R./A.S.:nothing to disclose. S.N.:SCAHT/University of Geneva funding for work. M.R.:funding from Swiss environment/health offices,WHO,ANSES outside work and consulting fees from Swedish Radiation Authority.

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RF-039 Cell-specific γH2AX response with no sustained functional injury

> **Why this matters**
> In laboratory cultures, a DNA-related signal changed in two cell types, while additional tests did not find corresponding sustained damage or loss of function. This study helps distinguish an early cellular response from an adverse outcome, and shows why several complementary tests are needed before interpreting a positive marker.

Original text reviewed | Source checks to 20 October 2026

Shanshan Xu; Guangdi Chen; Chunjing Chen; Chuan Sun; Danying Zhang; Manuel Murbach; Niels Kuster; Qunli Zeng; Zhengping Xu. Cell Type-Dependent Induction of DNA Damage by 1800 MHz Radiofrequency Electromagnetic Fields Does Not Result in Significant Cellular Dysfunctions. PLoS ONE. 2013;8(1):e54906. doi:10.1371/journal.pone.0054906.

Exposure. 1,800 MHz GSM-like 217 Hz modulation; cell-monolayer SAR 3.0 W/kg for 1 or 24 h, 5 min on/10 min off. Dual R18 waveguides; sham isolation >60 dB; medium-specific dosimetry; air 37±0.1°C and culture temperature rise <0.1°C.

Design and controls. Six cell types; blinded exposure and assay teams; approximately 200 cells/sample; at least four independent experiments for foci; 4NQO positive controls. Orthogonal DNA-damage and functional assays in human skin fibroblasts.

Reported finding. After 24 h, γH2AX foci increased in Chinese hamster lung and human skin fibroblasts, but not four other cell types. Comet, TUNEL, proliferation and viability did not show corresponding sustained abnormalities in fibroblasts.

Statistical detail. Mean foci/cell: CHL 4.30±0.37 versus 3.10±0.30, P=.022; fibroblasts 6.21±0.44 versus 4.54±0.22, P=.011. A small cell-cycle shift at 6 h was absent at 12 h; ROS increase was nonsignificant.

Interpretive limits. γH2AX marks DNA-damage signalling; it does not establish irreversible strand breakage or clinical injury. Multiple assays and small replicate counts; downstream functional effects were largely absent.

Programme and cohort links. Zhejiang University/IT'IS study, distinct from Vienna REFLEX RF-016. ETH repository DOI 10.3929/ethz-b-000064177 is a version of the same work.

The protection question raised. When does DNA-damage signalling indicate lasting injury? The lack of corresponding sustained abnormalities in other assays makes this a cell-specific response question rather than an established genotoxic disease mechanism.

Source verification. Original full methods, results and supplemental descriptions reviewed through PMC XML; Scite abstract and metadata corroborated.

Source locations. Exposure System; Cell Exposure Protocol; Figure 1; Figures 2-6; Figure S1; Funding Statement.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. Chinese Ministry of Science and Technology 2011CB503702; NSFC 81102094/30872082; Zhejiang 5Y205458; doctoral and central-university grants; author career awards. Funders reported no role.

Conflicts. Authors declare no competing interests. IT'IS/Zurich dosimetry coauthors participated.

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RF-040 Sperm apoptosis and oxidative markers in a separate Chinese rat experiment

Why this matters

More sperm cells entered programmed cell death in exposed rats, alongside chemical-stress changes. That makes cell survival a reproductive-protection question for a laboratory outside the Newcastle programme. The study did not test fertility, and a separate sperm-count result has a reporting inconsistency that should be clarified.

Original text reviewed | Source checks to 20 October 2026

Qi Liu; Tianlei Si; Xiaoyun Xu; Fuqiang Liang; Lufeng Wang; Siyi Pan. Electromagnetic radiation at 900 MHz induces sperm apoptosis through bcl-2, bax and caspase-3 signaling pathways in rats. Reproductive Health. 2015;12(1):65. doi:10.1186/s12978-015-0062-3.

Exposure. 900 MHz, 50 Hz sinusoidal amplitude modulation, 100% index; 2 h/day for 50 days; power density 1±0.03 mW/cm² at 7 cm. Simplified prolate-spheroid whole-body SAR estimate 0.66±0.01 W/kg. Holding-unit temperature rise at most 0.07°C.

Design and controls. 48 male Sprague-Dawley rats randomized 24 exposed/24 sham; identical holding units without RF for sham. Epididymal sperm assays; protein and mRNA assays n=15/group.

Reported finding. Sperm apoptosis increased from 11.51±0.53% to 22.47±1.83%; ROS rose and antioxidant capacity fell. Apoptosis-related proteins/transcripts changed. Overall sperm malformation percentage was not significantly different.

Statistical detail. Table 4, n=24/group, mean±SEM: ROS 53.00±1.89 versus 36.51±3.64; antioxidant capacity 4.80±0.12 versus 6.82±0.21. Authors mark P<.05 using t-tests.

Interpretive limits. No mating or pregnancy outcome; sperm injury does not establish infertility. Holding-unit temperature is not organ/core thermometry; SAR uses a simplified model; blinding unestablished. Table 3 sperm-count significance appears inconsistent with its means and SEMs: do not rely on that count claim without clarification.

Programme and cohort links. Huazhong Agricultural University/Wuhan experiment, separate from Newcastle sperm studies; not a standardized replication.

The protection question raised. Does the sperm-injury signal persist with stronger dose and temperature characterisation, and does it affect fertility? The unresolved sperm-count reporting inconsistency cannot strengthen the case without clarification.

Source verification. Original full methods, results, tables and disclosures reviewed using Scite and PMC XML.

Source locations. Exposure methods; Table 4; Figures 4-6. Table 3 needs reporting clarification.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. China National citrus industry technology system programme CARS-27-05B, as reported.

Conflicts. Authors declare no competing interests. Biofavor assisted Western blot analyses.

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RF-041 Yale prenatal handset mouse experiment with acknowledged dosimetry limits

> **Why this matters**
> After exposure during pregnancy, mouse offspring showed altered activity and object recognition. Developmental outcomes deserve attention, but the experiment did not measure how much RF energy the mothers or fetuses absorbed. Its published hormone correction is retained; these results do not establish ADHD or a human prenatal risk level.

Original text reviewed | Source checks to 20 October 2026

Aldad TS, Gan G, Gao XB, Taylor HS. Fetal Radiofrequency Radiation Exposure From 800-1900 Mhz-Rated Cellular Telephones Affects Neurodevelopment and Behavior in Mice. Scientific Reports. 2012;2(1):312. doi:10.1038/srep00312.

Exposure. Muted/silenced 800-1900 MHz-rated phones on active calls during gestational days 1-17, 4.5-22.3 cm from mice; deactivated-phone controls. The stated 1.6 W/kg is the phone rating, not measured maternal/fetal SAR.

Design and controls. 33 exposed and 42 control pregnant mice. Object-recognition testing in 161 offspring (82 exposed,79 control); blinded behavioural observers. Prefrontal electrophysiology used prenatal protocols of 0,9,15 or 24 h/day.

Reported finding. Exposure-associated hyperactivity, reduced object recognition and altered prefrontal glutamatergic synaptic transmission were reported.

Statistical detail. Original Results: cumulative object-recognition preference 63.0% exposed versus 69.9% control, P=.003,n=161. Light/dark transitions 24.4 versus 16.4,P<.001.

Interpretive limits. Authors explicitly acknowledged unmeasured exposure and no traditional calibrated EMF generator. Maternal/fetal dosimetry absent; offspring/neuron counts are not independent dam counts. Not proof of human ADHD or below-limit prenatal injury.

Programme and cohort links. Yale programme, independent from UK Broom 2019 but substantially different exposure protocol.

The protection question raised. Are the developmental changes reproducible under measured maternal and fetal exposure? Uncalibrated handset exposure and litter-level uncertainty prevent a quantitative prenatal protection claim or a human ADHD inference.

Source verification. Original eight-page PDF, including appended 2013 corrigendum, reviewed. Scite notice confirmed.

Source locations. Results/Figure 1; Methods; Discussion on exposure limitations; appended corrigendum.

Editorial status. 2013 corrigendum DOI 10.1038/srep01320: corticosterone ELISA repeated using stored serum; control 5.4 versus exposed 6.1 ng/ml, P=.786, n=6/group. Authors state conclusions unchanged.

Funding. EHHI and NICHD HD052668, original acknowledgments.

Conflicts. Authors declare no competing financial interests.

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RF-049 Regional brain glucose metabolism in a blinded crossover experiment

> **Why this matters**
> A brain scan detected increased glucose use in the region nearest the active phone's antenna in a blinded human experiment. Glucose use measures brain metabolism, so this provides evidence of an objective response under that protocol. Whether the response affects health or function remains unresolved; the scan did not demonstrate injury.

Original text reviewed | Source checks to 20 October 2026

Volkow ND, Tomasi D, Wang GJ, Vaska P, Fowler JS, Telang F, Alexoff D, Logan J, Wong C. Effects of cell phone radiofrequency signal exposure on brain glucose metabolism. JAMA. 2011;305(8):808-813. doi:10.1001/jama.2011.186.

Exposure. 837.8 MHz CDMA handset; right phone active and muted for 50 min versus both phones off. Manufacturer model maximum head SAR 0.901 W/kg, not participant-specific measured SAR.

Design and controls. Randomized crossover, 47 healthy participants analysed, each with two FDG-PET sessions; participants blinded; field signal checked throughout calls.

Reported finding. Near-antenna regional metabolism was 35.7 versus 33.3 micromol/100 g/min; mean difference 2.4 (95% CI 0.67-4.2), p=.004. Whole-brain metabolism did not differ.

Interpretive limits. Objective physiological response of unknown clinical significance; neither brain injury nor cancer demonstrated. No individual compliance certification from handset model SAR.

Programme and cohort links. US NIH/Brookhaven programme; independent of Swiss and French EEG experiments.

The protection question raised. Does the local metabolic response have consequences for health or function? This blinded human observation establishes an objective response under its protocol, while clinical significance and individual delivered dose remain unresolved.

Source verification. Original accepted manuscript methods, results and disclosures reviewed; Scite abstract and notices checked

Source locations. Author manuscript pp.3-5 methods/results; p.7 funding; p.1 conflict statement

Editorial status. No correction, concern or retraction identified in Scite/primary PubMed and targeted DOI search; commentary is not an erratum.

Funding. NIH intramural research programme and US Department of Energy infrastructure support; manuscript states funders had no role in conduct or interpretation.

Conflicts. Authors reported none on ICMJE disclosures.

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ELF-007 Symptoms during blinded electric field transitions

> **Why this matters**
> Symptoms deserve careful investigation even when they are difficult to explain. This blinded challenge recorded one person's responses to a power-frequency electric field. Its value is a testable individual observation; it does not tell us how common such responses are or establish a diagnosis for other people.

Original text reviewed | Source checks to 20 October 2026

McCarty DE, Carrubba S, Chesson AL, Frilot C, Gonzalez-Toledo E, Marino AA. Electromagnetic Hypersensitivity: Evidence for a Novel Neurological Syndrome. International Journal of Neuroscience. 2011;121(12):670-676. doi:10.3109/00207454.2011.608139.

Exposure. 60 Hz electric field, average over head 300 V/m; repeated 100-second challenge intervals.

Design and controls. Clinical case and controlled provocation; n=1; accepted manuscript methods inspected.

Reported finding. One participant reported symptoms in all 10 pulsed-field intervals, versus mild symptoms in five and none in five sham intervals. A separate behavioural task found no above-chance conscious field identification.

Interpretive limits. Individual response, not prevalence or general RF causation. Continuous versus pulsed conditions and symptom scoring must remain distinct.

Programme and cohort links. Different field regime from RF-013.

The protection question raised. Is this person's symptom response reproducible under comparable blinded conditions? A controlled individual observation is worth understanding without turning it into a prevalence estimate, general RF effect or diagnostic test.

Source verification. Original accepted manuscript methods and results inspected

Source locations. Accepted manuscript Tables 2-5; symptoms and field-identification tasks are separate

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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ELF-009 Life-span exposure to sinusoidal-50 Hz magnetic field and acute low-dose γ radiation induce carcinogenic effects in Sprague-Dawley rats

> **Why this matters**
> Rats given both a power-frequency magnetic field and a small gamma-radiation dose developed more of some cancers than rats receiving gamma radiation alone. It makes combined exposures worth examining. The comparison matters: the heart-tumour result was not significant against gamma-only animals, and this was not an RF experiment.

Original text reviewed | Source checks to 20 October 2026

Soffritti M, Tibaldi E, Padovani M, Hoel DG, Giuliani L, Bua L, Lauriola M, Falcioni L, Manservigi M, Manservisi F, Panzacchi S, Belpoggi F. Life-span exposure to sinusoidal-50 Hz magnetic field and acute low-dose γ radiation induce carcinogenic effects in Sprague-Dawley rats. International Journal of Radiation Biology. 2016;92(4):202-214. doi:10.3109/09553002.2016.1144942.

Exposure. 50 Hz sinusoidal magnetic field, 20 or 1000 μT, 19 h/day, gestational day 12 to natural death; additional whole-body 0.1 Gy gamma dose at 6 weeks. ELF plus ionising coexposure, not RF.

Design and controls. 1658 rats including 1001 shared untreated controls; gamma-only 118 M/105 F; 20 μT+gamma 105 M/107 F; 1000 μT+gamma 110 M/112 F. Cox survival-adjusted models; Mantel-Haenszel for some zero-case endpoints. Survival unchanged. Same pathology team/supervisor review; masking not documented in reviewed methods.

Reported finding. At 1000 μT+gamma, female mammary adenocarcinoma 18/112 vs 8/105 gamma-only ($p \leq .05$); male haemolymphoreticular neoplasia 28/110 vs 18/118 ($p \leq .05$). Male cardiac schwannomas 2/105 and 3/110 vs 1/500 untreated were significant; comparison against gamma-only 0/118 was nonsignificant.

Interpretive limits. Coexposure; large shared untreated control and smaller gamma-only comparator; not an RF-alone or below-limit cancer demonstration; combined atypia/carcinoma endpoints distinct from carcinoma alone.

Programme and cohort links. RI ELF megaexperiment; 1001 untreated controls shared with ELF-alone and formaldehyde arms. Distinct animals/exposure from RF-002; Soffritti 2019 mini-review repeats data.

The protection question raised. Can combined exposures modify cancer outcomes? Comparisons with gamma-only animals are essential: selected mammary and haemolymphoid findings differed, whereas the heart-schwannoma comparison did not. This is a power-frequency coexposure experiment.

Source verification. Original 14-page early online journal PDF reviewed: methods, Tables 2-6, results, funding; final citation checked with Scite/PubMed.

Source locations. PDF pp 3-4 treatment/pathology/statistics; p 7 Tables 4-6; p 9 heart comparison; p 12 funding. Final journal pp 202-214.

Editorial status. No editorial notice located in Scite/PubMed/targeted title searches 2026-10-20; not certification of no possible future notice.

Funding. RI; ARPA Emilia-Romagna; UK Children With Cancer; Cassa di Risparmio Bologna; INAIL; Fondazione del Monte Bologna/Ravenna; Municipality of Bologna. No conflicts declared; funders no direct role.

Conflicts. Authors declare no conflicts or competing interests, as documented in the retrieved source disclosure.

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IF-001 Learning and memory endpoints at 7.5 kHz

> **Why this matters**
> The higher-exposure mice had difficulty learning and retaining parts of a task in this intermediate-frequency experiment. These functional findings give the assessment a different focus from isolated cell markers. Equipment problems forced a separate high-exposure cohort, so a carefully matched repeat is needed before applying the result to public exposures.

Original text reviewed | Source checks to 20 October 2026

Kumari K, Koivisto H, Viluksela M, Paldanius KMA, Marttinen M, Hiltunen M, Naarala J, Tanila H, Juutilainen J. Behavioral testing of mice exposed to intermediate frequency magnetic fields indicates mild memory impairment. PLOS ONE. 2017;12(12):e0188880. doi:10.1371/journal.pone.0188880.

Exposure. 7.5 kHz magnetic field; sham, 12 or 120 microtesla; continuous exposure for five weeks.

Design and controls. 70 male mice: 30 sham and 20 per exposed group.

Reported finding. The 120-microtesla group showed impaired task acquisition and passive-avoidance retention, with elevated hippocampal TNF-alpha expression.

Interpretive limits. Equipment problems required a separate high-exposure cohort. Many other endpoints were unchanged. The high field exceeded the occupational reference level cited by the authors.

The protection question raised. Do the memory and inflammatory findings persist with concurrent comparison groups? The separate high-exposure cohort limits inference, and intermediate-frequency conditions cannot be transferred directly to public RF exposures.

Source verification. Full indexed article body reviewed

Source locations. Materials and methods; Conclusions; supplementary test summary

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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ECO-003 Bee queen development under GSM exposure

> **Why this matters**
> Fewer queen larvae reached hatching in the exposed group. Because queen development is central to a bee colony, this is a meaningful outcome for environmental assessment. Only two pairs of rearing colonies were studied under an unusual handset-exposure arrangement; the result cannot establish a cause of wider colony losses.

Original text reviewed | Source checks to 20 October 2026

Odemer R,Odemer F. Effects of radiofrequency electromagnetic radiation(RF-EMF) on honey bee queen development and mating success. Science of the Total Environment.2019;661:553-562. DOI10.1016/j.scitotenv.2019.01.154.

Exposure. 900 MHz GSM AEG handset attached to grafting frame;15 two-minute calls/day for about 14 days; queen cups within~21 cm. Off-phone controls. Human phantom handset SAR0.59/1.16 W/kg is not bee SAR.

Design and controls. Two paired queen-rearing colony replicates;26 grafted larvae per treatment/replicate,52 per treatment total. Hohenheim 2018; surviving queens tested for mating and 9 subsequent colonies followed to day 88.

Reported finding. Reduction from original grafts to hatching:30.8% controls versus 61.5% exposed,LogRank P=.0065. Overall mortality HR2.3,P=.003; mating among hatched queens and later colony sizes not significantly different.

Interpretive limits. Only two rearing-colony pairs; colony nesting remains a limitation. Handset exposure is explicitly an unrepresentative worst-case apiary scenario; human SAR rating does not measure bee absorption. No established colony-collapse cause.

Programme and cohort links. Hohenheim programme, distinct experiment/setup from Treder 2023. Shared institution does not establish shared colonies.

The protection question raised. Is the reduced queen hatching reproducible under well-characterised, environmentally relevant exposure? Two colony pairs and an unusual handset arrangement limit translation to bee populations or wider colony losses.

Source verification. Original author-hosted 10-page published PDF methods/results/disclosures reviewed; upgraded from retrieval lead.

Source locations. Sections 2.2-2.3,3.1-3.3; Figures 3-6; Conclusion; Funding/Declarations.

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Authors report no specific public,commercial or nonprofit grant.

Conflicts. Declarations of interest:none.

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ECO-004 Homing ability of exposed honey bees

Why this matters

Finding the way home is essential to a foraging bee. After prolonged exposure to a continuously transmitting test signal, fewer bees returned successfully. This gives environmental assessment a functional endpoint beyond brood development or overall survival, which were not significantly changed. It does not establish colony collapse or effects in every Wi-Fi setting.

Original text reviewed | Source checks to 20 October 2026

Treder M,Müller M,Fellner L,Traynor K,Rosenkranz P. Defined exposure of honey bee colonies to simulated radiofrequency electromagnetic fields(RF-EMF): Negative effects on the homing ability,but not on brood development or longevity. Science of the Total Environment.2023;896:165211. DOI10.1016/j.scitotenv.2023.165211.

Exposure. Simultaneous 2.450/5.805 GHz IEEE802.11 a OFDM packets,100% duty,continuous 12 weeks in 2020/14 weeks in 2021; homing after~7 weeks plus 40 min outside hives. EIRP177.83/10 mW per side; colonies 2 m from emitter; controls>100-fold lower. No bee SAR.

Design and controls. Hohenheim field experiments 2020/2021;16 colonies randomly split 8/8. Long-term homing:4 hives/group,8 paired trials,~160 bees. Short-term:9 additional colonies,11 trials,~200 bees. GLMM includes hive/trial random factors.

Reported finding. Long-term return 78.6% exposed versus 95.2% control, GLMM P=.0064; return time nonsignificant. Short-term 40 min return 90.0% versus 86.6%, P=.4696. Brood development and overall 28-day survival were not significantly different (survival Log-Rank P=.102). A partial day 11-29 foraging-age-window survival analysis differed (Wilcoxon P=.042; exposed n=280, control n=217).

Interpretive limits. Fitness-related field endpoint with engineered signal; 100% duty is not all typical Wi-Fi. Exposure geometry confounded with location; observer blinding unestablished; independent replication absent. The partial survival-window result is exploratory and does not establish a shortened overall lifespan. No inference of colony collapse, human risk or cellular 5G effects.

Programme and cohort links. Hohenheim/KIT programme; different protocol from Odemer 2019. Shared network is not independent laboratory replication.

The protection question raised. Could prolonged exposure impair navigation under field-relevant conditions? Reduced homing is functionally important, but site confounding, absent independent replication and unchanged overall survival limit an ecological risk conclusion.

Source verification. Original nine-page KIT repository PDF reviewed; methods,Tables 1-3,results and disclosures checked; upgraded from retrieval lead.

Source locations. Sections 2.1-2.6; Tables 1-3; Sections 3.2-3.3; Figures 6-7; Funding/Declaration.

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Ministerium für Ernährung,Ländlichen Raum und Verbraucherschutz Baden-Württemberg.

Conflicts. Authors declare no known competing financial interests or personal relationships.

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ECO-006 Double-blind broadband RF disruption of a bird magnetic compass

Why this matters

Migratory songbirds can use the Earth's magnetic field as a compass. In this blinded experiment, electromagnetic noise disrupted that orientation. It asks environmental assessors to consider sensory function. These were laboratory tests with kilohertz-to-megahertz noise, not a study of migration losses or an experiment with GHz phone signals.

Original text reviewed | Source checks to 20 October 2026

Susanne Schwarze; Nils-Lasse Schneider; Thomas Reichl; David Dreyer; Nele Lefeldt; Svenja Engels; Neville Baker; P. J. Hore; Henrik Mouritsen. Weak Broadband Electromagnetic Fields are More Disruptive to Magnetic Compass Orientation in a Night-Migratory Songbird (Erithacus rubecula) than Strong Narrow-Band Fields. Frontiers in Behavioral Neuroscience. 2016;10:55. doi:10.3389/fnbeh.2016.00055.

Exposure. European robins in shielded nonmagnetic chambers; broadband noise approximately 2 kHz-9 MHz versus dummy and narrow-band conditions, under normal and 120°-rotated geomagnetic fields. Table 1 reports broadband summed average magnetic intensity 35 nT; inspect the original spectra rather than treating accumulated max-hold values as instantaneous exposure.

Design and controls. Double-blind exposure and independent orientation-paper assessment. Four migratory seasons, 2012-2014. Broadband comparison used the same 16 birds across conditions in autumn 2013. Prespecified inactivity/ambiguity exclusions; 4,098 individual tests across the programme.

Reported finding. Birds were disoriented under broadband noise but oriented under dummy and selected narrow-band conditions. The earlier claimed highly specific Larmor-frequency resonance was not replicated.

Statistical detail. Broadband: normal-field vector r=.17, P≈.60; rotated-field r=.19, P≈.56, n=16 each. Dummy: r=.54, P<.01 and rotated r=.49, P<.05.

Interpretive limits. Captive-bird compass interference, not demonstrated population decline or human injury. MHz noise does not establish effects of GHz cellular signals. Within-condition Rayleigh tests alone do not quantify between-condition effects; some control orientation shifts differed from the expected 120°.

Programme and cohort links. Oldenburg/Oxford programme with Engels 2014 and Leberecht 2022. These are not three independent laboratories. Frankfurt/Ritz narrow-band experiments are an earlier separate programme.

The protection question raised. Could exposure interfere with specialised wildlife senses under environmental conditions? Laboratory compass disruption is a functional endpoint, not population decline; this frequency range cannot establish effects from GHz phone signals.

Source verification. Original full methods, Table 1, Figures 5-7 and disclosures reviewed via Scite and PMC XML.

Source locations. Table 1; Time-dependent Fields; Data Collection; Figure 6; Discussion; Funding.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. DARPA QuBE; ERC 340451; USAF AF9550-14-1-0095; German BMBF Varying Tunes; DFG MO1408/1-2 and GRK1885; Volkswagenstiftung.

Conflicts. Authors declare no commercial or financial relationships constituting potential conflicts.

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ECO-007 Magnetic-compass disruption extends to 75-85 MHz in blackcaps

> **Why this matters**
> A later study in another bird species found disrupted magnetic-compass orientation at 75-85 MHz. It extends the programme to a different frequency band and functional test. Whether such exposure affects wild populations remains unanswered, and this related laboratory programme is not independent confirmation across research groups.

Original text reviewed | Source checks to 20 October 2026

Bo Leberecht; Dmitry Kobylkov; Thiemo Karwinkel; Sara Döge; Lars Burnus; Siu Ying Wong; Shambhavi Apte; Katrin Haase; Isabelle Musielak; Raisa Chetverikova; Glen Dautaj; Marco Bassetto; Michael Winklhofer; P. J. Hore; Henrik Mouritsen. Broadband 75-85 MHz radiofrequency fields disrupt magnetic compass orientation in night-migratory songbirds consistent with a flavin-based radical pair magnetoreceptor. Journal of Comparative Physiology A. 2022;208(1):97-106. doi:10.1007/s00359-021-01537-8.

Exposure. 75-85 MHz broadband noise, 2.53 pT/√Hz reported max-hold spectral density. Three aluminium-shielded chambers; normal and 120°-rotated static magnetic fields. Magnetic/electric spectra measured daily. Spectral density is not total magnetic field.

Design and controls. Eurasian blackcaps in two seasonal cohorts, spring 2019 and 2021; same individuals tested across four conditions, n=27-32/condition after activity exclusions. Blinded orientation assessment; control pre-screening and subsequent fresh test series.

Reported finding. Controls oriented and shifted with the rotated geomagnetic field. Under RF noise, headings were indistinguishable from random and significantly more dispersed than controls.

Statistical detail. Normal control: n=32, r=.4018, P=.005; RF: n=27, r=.1237, P=.6655. Rotated control: n=31, r=.4122, P=.0044; RF: n=29, r=.0740, P=.8552. Bootstrap directedness comparisons P=.00854 and .00902.

Interpretive limits. Laboratory orientation is a functional endpoint, not observed migration mortality, population decline or human health risk. Radical-pair compatibility does not identify the in-vivo sensor conclusively. Same Oldenburg/Oxford research network as 2014/2016.

Programme and cohort links. Online 2021, issue 2022: one paper. No independent laboratory replication of this 75-85 MHz protocol established in this pass.

The protection question raised. Do environmental spectra overlap conditions that disrupt this bird compass, and are there population consequences? The later frequency-band result extends a related programme rather than providing independent multispecies confirmation.

Source verification. Original full methods, results and disclosures reviewed in PMC XML; Scite forward graph traced the programme.

Source locations. Generation and Measurement of Time-dependent Fields; Behavioural Experiments; Figures 2-3; Results.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. DFG SFB1372/GRK1885; DAAD; Lower Saxony Nano-Energieforschung stipend; ERC QuantumBirds 810002; Projekt DEAL open access.

Conflicts. Authors declare no conflict of interests.

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Annex B Original abstracts with methods still to audit

The original abstract supports the stated finding, but complete original methods/results remain unreviewed or partly unresolved. These records are useful retrieval routes and qualified observations. An abstract-level result is not promoted into a fully verified mechanism, dose threshold or population risk.

Records in this section: RF-012, RF-015, RF-024, RF-027, RF-029, RF-030, RF-031, RF-034, RF-042, RF-043, RF-044, RF-045, RF-046, RF-047, RF-048, RF-050, RF-051, ELF-010, ECO-005

RF-012 Pulse modulation and regional blood flow

> **Why this matters**
> The way a radio signal is delivered may matter as well as its frequency. This human study's abstract reports different brainwave responses to pulsed and continuous signals, alongside blood-flow changes. Full methods still need review before drawing dose conclusions; the observation points to waveform as a health-assessment question.

Original abstract verified | Source checks to 20 October 2026

Huber R, Treyer V, Borbély AA, Schuderer J, Gottselig JM, Landolt HP, Werth E, Berthold T, Kuster N, Buck A, Achermann P. Electromagnetic fields, such as those from mobile phones, alter regional cerebral blood flow and sleep and waking EEG. Journal of Sleep Research. 2002;11(4):289-295. doi:10.1046/j.1365-2869.2002.00314.x.

Exposure. 900 MHz; 30-minute head exposure.

Design and controls. Human PET and sleep-EEG experiments.

Reported finding. The original abstract reports pulse-modulated exposure altered regional cerebral blood flow and waking/sleep EEG; continuous-wave exposure did not produce the same EEG result.

Interpretive limits. Retrieve full methods before quoting precise sample/dosimetry details; physiological outcomes are not disease endpoints.

Programme and cohort links. RF-010 and other Zurich publications require cohort linkage

The protection question raised. Could signal modulation help explain differences in brain responses? Full methods and delivered-dose details remain necessary before the abstract-level result can inform a protection conclusion.

Source verification. Original abstract reviewed

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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RF-015 Highest cumulative use category in INTERPHONE

> **Why this matters**
> The highest reported call-time group had elevated odds of glioma, a type of brain tumour, in this international study. The authors could not interpret that association as causal because of bias and reporting errors. It highlights why the heaviest-use histories need close scrutiny and better exposure verification.

Original abstract verified | Source checks to 20 October 2026

The INTERPHONE Study Group. Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case-control study. International Journal of Epidemiology. 2010;39(3):675-694. doi:10.1093/ije/dyq079.

Exposure. Interview-based lifetime handset-use histories across 13 countries; no measured individual SAR.

Design and controls. 2708 glioma and 2409 meningioma cases with matched controls; original abstract and PubMed linked-notice record checked.

Reported finding. Original abstract: highest recalled call-time decile (>=1640 h) had glioma OR 1.40 (95% CI 1.03-1.89), while regular use overall and >=10 years since first use did not show increased risk. Authors state biases and errors prevent causal interpretation.

Interpretive limits. Implausible reported use in the highest decile, participation bias and recall error materially qualify the positive stratum. Do not count country analyses as independent cohorts.

The protection question raised. Can the highest-use association be separated from reporting error and selection bias? Shared national and pooled participants must remain visible when judging corroboration; this record does not establish a causal human cancer estimate.

Source verification. Original abstract and primary PubMed publication/erratum linkage reviewed; full methods not re-audited

Source locations. Original abstract Results and Conclusions; PubMed PMID 20483835 Erratum in

Editorial status. PubMed lists a 2012 erratum correcting collaborator Montestrucq's name. Scite incorrectly labels dyq082 as an erratum: dyq082 is Saracci/Samet commentary; its own correction is dyq135. No quantitative tumour correction inferred.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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RF-024 Lymphoma in genetically susceptible mice

> **Why this matters**
> These mice were genetically prone to lymphoma, a cancer of immune cells. The reported increase makes inherited susceptibility a serious question, but later experiments did not reproduce it under their different protocols. That history limits what this model can tell us about who might be especially vulnerable.

Original abstract verified | Source checks to 20 October 2026

Repacholi MH, Basten A, Gebski V, Noonan D, Finnie J, Harris AW. Lymphomas in Eμ-Pim1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields. Radiation Research. 1997;147(5):631-640. doi:10.2307/3579630.

Exposure. 900 MHz plane-wave fields, 217 Hz pulse repetition, 0.6 ms pulse width; two 30-minute periods/day for up to 18 months. Incident power density 2.6-13 W/m²; SAR 0.008-4.2 W/kg, averaging 0.13-1.4 W/kg. Freely moving animals. Seven days/week is reported in IARC Table 3.1; original abstract does not give weekly frequency.

Design and controls. 101 exposed and 100 sham-exposed female Eμ-Pim1 transgenic mice predisposed to spontaneous lymphoma. Sham exposure; original full methods/pathology tables not obtained during this check.

Reported finding. Original abstract reports lymphoma odds ratio 2.4 (95% CI 1.3-4.5), p=.006; follicular lymphomas made the main contribution. IARC Table 3.1 extracts all-lymphoma counts as 43/101 exposed vs 22/100 sham. Counts are a government-assessment extraction, not a newly checked original table.

Interpretive limits. Genetically predisposed strain; wide individual SAR range. IARC notes no standardized necropsy-selection criteria and that clinically surviving 18-month animals were discarded without necropsy; survival data were not reported. Oberto 2007 and Utteridge 2002 did not reproduce the lymphoma finding under modified dosimetry/restraint protocols. Not an established independently replicated susceptibility effect.

Programme and cohort links. Original Adelaide/Australian lymphoma-prone mouse experiment. Utteridge 2002 and Oberto 2007 are later attempts at replication, not additional affirmative cohorts; keep their protocol differences explicit.

The protection question raised. Is the lymphoma finding a reproducible feature of this genetically susceptible model? Nonconfirming attempted replications limit both the original inference and any general claim about inherited human vulnerability.

Source verification. Original-abstract content available in literature records and government abstract compilation; full citation/DOI independently confirmed by publisher JSTOR issue and coauthor UNSW publication record. IARC original assessment Table 3.1 and narrative reviewed for counts/limitations. Full original journal article not retrieved; retain abstract-level verification.

Source locations. Original abstract, Radiation Research 147(5):631; IARC Monographs volume 102, section 3.1.3(a) and Table 3.1 (page numbering differs between chapter versions).

Editorial status. Targeted DOI/title notice search 2026-10-20 found no formal correction/retraction/concern. Replication criticism is not an editorial retraction. Full publisher notice-history audit incomplete.

Funding. Original funding/conflict statement not verified. A BfS-hosted 2008 Australian research presentation attributes funding to Telstra Research Laboratories; do not substitute that secondary attribution for original disclosure.

Conflicts. Not established from retrieved material.

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RF-027 Lund time-course follow-up

> **Why this matters**
> The blood-brain barrier normally controls what passes from the bloodstream into brain tissue. This follow-up reported leakage and nerve-cell changes at different times after exposure. It makes the timing of tissue examinations important; the abstract alone cannot show which individual exposure levels drove each result.

Original abstract verified | Source checks to 20 October 2026

Eberhardt JL, Persson BRR, Brun AE, Salford LG, Malmgren LOG. Blood-brain barrier permeability and nerve cell damage in rat brain 14 and 28 days after exposure to microwaves from GSM mobile phones. Electromagn Biol Med. 2008;27(3):215-229. doi:10.1080/15368370802344037.

Exposure. Real programmable 900-MHz GSM phone, TEM cells; 2 h; average whole-body SAR 0.12, 1.2, 12 or 120 mW/kg.

Design and controls. 96 non-anesthetized rats exposed or sham-exposed, examined 14 or 28 days later. Exact allocation, masking and dose-specific estimates require full paper.

Reported finding. Original abstract: after 14 days, albumin extravasation and neuronal albumin uptake increased (Kruskal-Wallis p=0.02 and 0.002); those findings were not increased after 28 days. Dark neurons increased at 28 days (p=0.02); neuronal albumin uptake correlated with damaged neurons at 28 days (Spearman r=0.41, p<0.01).

Interpretive limits. Original abstract verified; full methods/results not obtained. These are global/time-course findings, not proof that every exposure group differed from sham. Same programme as RF-020; not independent corroboration.

Programme and cohort links. Distinct experiment within Lund programme. Do not combine with 48 rats at 7 days (RF-028).

The protection question raised. Could the timing of tissue examination explain different barrier and neuronal findings? The abstract supports a time-course question, but dose-specific results and full methods remain unverified.

Source verification. Scite original abstract and Lund institutional record

Source locations. Original abstract; original pages 215-229 not fully audited

Editorial status. Scite metadata checked 20 Oct 2026; no editorial notice returned. Primary bibliographic records and targeted notices were checked where retrievable; no retraction or expression of concern located. This is not certification that no notice exists.

Funding. Not established from retrieved original abstract.

Conflicts. Not established from retrieved original abstract.

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RF-029 Temporal-order memory after 55 weeks of weekly exposure

> **Why this matters**
> Remembering the order of events is a specific mental function. Exposed rats performed less well on an object-order task after a long exposure programme, although other tests were unchanged. Tissue examination of the same animals did not demonstrate neuronal injury, so the behavioural finding needs its own explanation.

Original abstract verified | Source checks to 20 October 2026

Nittby H, Grafström G, Tian DP, Malmgren L, Brun A, Persson BRR, Salford LG, Eberhardt J. Cognitive impairment in rats after long-term exposure to GSM-900 mobile phone radiation. Bioelectromagnetics. 2008;29(3):219-232. doi:10.1002/bem.20386. Online 28 November 2007.

Exposure. GSM-900, 2 h once weekly for 55 weeks; whole-body SAR initially 0.6 and 60 mW/kg, declining with growth (companion methods: approximately 0.4 and 40 mW/kg at end).

Design and controls. 56 Fischer 344 rats: 16 in each active group, 16 sham, 8 cage controls. Companion original methods describe randomized groups, equal sexes, 4-6 months old at start; behavioral testing 3-7 weeks after exposure; 54 survived to testing/pathology.

Reported finding. Original abstract reports poorer memory for objects and their temporal presentation order in exposed rats versus sham (p=0.02). Open-field behavior and memory of object location did not differ.

Interpretive limits. Cognitive article abstract verified; exposure/design linkage checked in original companion methods. Same animals as CTX-005, not an independent replication. Companion notes cage controls performed even worse than exposed rats, indicating handling/experience matters. Histological injury was not demonstrated in these animals.

Programme and cohort links. Same 56-rat experiment as CTX-005; journal year 2008, online year 2007.

The protection question raised. Why did object-order memory differ without demonstrated neuronal injury in the same animals? Handling and test experience complicate interpretation, leaving the functional observation separate from a claim of brain-cell loss.

Source verification. Original abstract; original full companion article reviewed

Source locations. Original abstract; companion Grafström 2008 GSM exposure/Animals and Individual correlation sections

Editorial status. Scite metadata checked 20 Oct 2026; no editorial notice returned. Primary bibliographic records and targeted notices were checked where retrievable; no retraction or expression of concern located. This is not certification that no notice exists.

Funding. Not established in retrieved cognitive abstract/companion indexed body.

Conflicts. Not established in retrieved cognitive abstract/companion indexed body.

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RF-030 Separate Gazi laboratory reports sex-dependent barrier response

> **Why this matters**
> A separate laboratory reported a change in passage from blood into brain tissue in male rats, with no corresponding finding in females. This suggests a question about biological differences worth testing directly. The abstract does not establish a statistical difference between sexes, and full methods remain to be checked.

Original abstract verified | Source checks to 20 October 2026

Sırav B, Seyhan N. Effects of radiofrequency radiation exposure on blood-brain barrier permeability in male and female rats. Electromagn Biol Med. 2011;30(4):253-260. doi:10.3109/15368378.2011.600167.

Exposure. 0.9- or 1.8-GHz continuous wave for 20 min under anesthesia; reported SAR 4.26 and 1.46 mW/kg, respectively (averaging definition to verify in full methods).

Design and controls. Male/female Wistar albino rats; sham controls; spectrophotometric Evans-blue assay in right brain, left brain, cerebellum and total brain. Sample sizes and masking require full paper.

Reported finding. Original abstract reports increased albumin/Evans-blue permeability in male rats after exposure; female rats showed no albumin extravasation. Exact region-level statistics not available in retrieved original abstract.

Interpretive limits. Separate institution and assay from Lund, not a direct replication of the dark-neuron finding. Anesthesia and sex are relevant; no claim of human injury or compliance with a human exposure limit.

Programme and cohort links. Gazi University, Ankara programme; related pulse-modulated experiment RF-031, not independent of that programme.

The protection question raised. Is there a genuine sex difference in barrier response? A significant result in males and a nonsignificant result in females do not themselves establish a between-sex interaction; original data remain important.

Source verification. Original PubMed abstract

Source locations. PMID 22047463 abstract

Editorial status. Scite metadata checked 20 Oct 2026; no editorial notice returned. Primary bibliographic records and targeted notices were checked where retrievable; no retraction or expression of concern located. This is not certification that no notice exists.

Funding. PubMed classifies non-US government research support; specific funder not documented in retrieved abstract.

Conflicts. Not reported in retrieved abstract.

Retrieve. DOI or primary source | PubMed record

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RF-031 Pulse-modulated follow-up in a separate institutional programme

> **Why this matters**
> This follow-up used pulsed signals and reported different patterns by sex and frequency in how much blood-borne dye entered brain tissue. It helps define more specific experiments on the brain's protective barrier. Dose and temperature details remain unverified, limiting what the findings reveal about relevant exposure conditions.

Original abstract verified | Source checks to 20 October 2026

Sırav B, Seyhan N. Effects of GSM modulated radio-frequency electromagnetic radiation on permeability of blood-brain barrier in male & female rats. J Chem Neuroanat. 2016;75(Pt B):123-127. doi:10.1016/j.jchemneu.2015.12.010. Online 23 December 2015.

Exposure. Pulse-modulated 900 and 1800 MHz, 20 min. Absorbed dose, pulse parameters and temperature measurement not verified; no low-dose or nonthermal claim adopted.

Design and controls. Six groups of young adult male/female Wistar albino rats; injected Evans-blue, quantitative whole-brain assay; group n/masking to verify.

Reported finding. Original abstract: male whole-brain Evans-blue content rose from 0.08±0.01 mg% control to 0.13±0.03 at 900 MHz and 0.26±0.05 at 1800 MHz (both $p<0.01$). Female response reported at 900 MHz, but not 1800 MHz. The abstract does not define the ± measure.

Interpretive limits. Original abstract only; not an independent programme from RF-030 or replication of dark-neuron pathology. Absorbed-dose details remain unverified.

Programme and cohort links. Gazi University programme; journal year 2016, online year 2015.

The protection question raised. Do frequency, pulse format and sex modify barrier responses? Unverified absorbed-dose and temperature details prevent this abstract-level record from establishing the adequacy of an exposure limit.

Source verification. Original PubMed abstract and EPA HERO primary citation record

Source locations. PMID 26723545 abstract

Editorial status. Scite metadata checked 20 Oct 2026; no editorial notice returned. Primary bibliographic records and targeted notices were checked where retrievable; no retraction or expression of concern located. This is not certification that no notice exists.

Funding. PubMed lists non-US government support; exact grant/funder unavailable from retrieved original abstract.

Conflicts. Not reported in retrieved abstract.

Retrieve. DOI or primary source | PubMed record | Original document or related source 2

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RF-034 Separate Chinese programme with functional, morphological and molecular endpoints

> **Why this matters**
> This Chinese experiment reports memory-task difficulty alongside tissue and brain-barrier changes in rats. That combination is worth examining across independent programmes. Only the original abstract has been verified here; the whole-body and local head doses differ sharply and must remain separate when considering relevance.

Original abstract verified | Source checks to 20 October 2026

Tang J, Zhang Y, Yang L, Chen Q, Tan L, Zuo S, Feng H, Chen Z, Zhu G. Exposure to 900 MHz electromagnetic fields activates the mkp-1/ERK pathway and causes blood-brain barrier damage and cognitive impairment in rats. Brain Res. 2015;1601:92-101. doi:10.1016/j.brainres.2015.01.019.

Exposure. 900 MHz, 1 mW/cm²; 3 h/day for 14 or 28 days. Original abstract reports 0.016 W/kg whole-body and 2 W/kg local head SAR; preserve both, and do not present whole-body value as head dose.

Design and controls. 108 male Sprague-Dawley rats, active/sham; Morris water maze, ultrastructure, Evans blue, immunostaining, Western blot. Full masking/exposure-temperature audit outstanding.

Reported finding. Original abstract reports reduced water-maze platform crossing/target-quadrant time after 28-day exposure, cellular edema/organelle degeneration, altered BBB/albumin endpoints, increased mkp-1 and ERK dephosphorylation. Exact contrasts, n per assay and effect statistics remain unverified.

Interpretive limits. Original abstract verified but full methods and results inaccessible. Mechanistic measurements do not alone establish that mkp-1/ERK causally mediated injury. Distinct regimen from Salford, not direct replication.

Programme and cohort links. Southwest Hospital, Third Military Medical University, Chongqing; distinct from Lund/Gazi programme. Separate animals and methods.

The protection question raised. Do the combined cognitive, tissue and barrier findings hold up in the original methods and independent experiments? Abstract-only verification and distinct whole-body and head doses limit their use in protection assessment.

Source verification. Scite bibliographic record; original PubMed abstract; ARPANSA government primary-citation summary used only to locate original

Source locations. PMID 25598203 abstract; original pages 92-101 require retrieval

Editorial status. Scite metadata checked 20 Oct 2026; no editorial notice returned. Primary bibliographic records and targeted notices were checked where retrievable; no retraction or expression of concern located. This is not certification that no notice exists.

Funding. Not established from retrieved abstract.

Conflicts. Not established from retrieved abstract.

Retrieve. DOI or primary source | PubMed record

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RF-042 3.5 GHz skin-cell/UV-B mitochondrial response

> **Why this matters**
> Skin cells encounter more than one stressor. This study's abstract reports that a 5G signal changed some chemical responses involving ultraviolet light, with different patterns in different cell types. The finding makes combined exposures worth examining; full methods and functional consequences must be checked before drawing a health-protection conclusion.

Original abstract verified | Source checks to 20 October 2026

Lorenza Patrignoni; Annabelle Hurtier; Rosa Orlacchio; Alexandre Joushomme; Florence Poulletier de Gannes; Philippe Lévêque; Delia Arnaud-Cormos; Hamid Reza Revzani; Walid Mahfouf; André Garenne; Yann Percherancier; Isabelle Lagroye. Evaluation of mitochondrial stress following ultraviolet radiation and 5G radiofrequency field exposure in human skin cells. Bioelectromagnetics. 2024;45(3):110-129. doi:10.1002/bem.22495.

Exposure. 5G-modulated 3.5 GHz at 0.25,1 or 4 W/kg for 24 h in fibroblasts/keratinocytes, including UV-B co-exposure.

Design and controls. Original in-vitro experiment; full methods, independent replicate counts and temperature controls remain to retrieve.

Reported finding. Abstract reports reduced mitochondrial ROS in fibroblasts at 1 W/kg and enhanced UV-B-related ROS in keratinocytes at 0.25 and 1 W/kg. No membrane-potential or apoptosis effect.

Interpretive limits. A biochemical response, not demonstrated injury. No independently checked effect size or complete protocol; exclude from central claims.

Programme and cohort links. Bordeaux/Limoges skin-cell programme. Haidar 2025, DOI10.1038/s41598-025-15090-w, uses different ROS biosensors and is not an exact independent replication.

The protection question raised. Could RF modify a skin-cell response to ultraviolet exposure? Different cell responses and absent measured apoptosis leave this a biochemical co-stressor hypothesis; full methods and functional consequences remain unverified.

Source verification. Primary publisher abstract and first PDF page reviewed; Scite abstract only. Retrieval lead.

Source locations. Abstract; publisher PDF first page 110.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. ANSES2019/2RF/018; New Aquitaine AAPR2020A-2019-8140010, as listed on original PDF first page.

Conflicts. Not established from retrieved material.

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RF-043 2026 GSM-modulated 3.5 GHz reproductive study: retrieval lead

> **Why this matters**
> The abstract reports changes in rat reproductive hormones and testicular tissue, partly reduced by an antioxidant treatment. It offers a chemical-stress hypothesis to verify. Although the frequency was 3.5 GHz, the signal format was GSM, not the format used by 5G networks. Full methods remain unreviewed.

Original abstract verified | Source checks to 20 October 2026

Hava Bektas; Seval Yildirim; Serife Cakir; Semih Dogu; Fikret Altindag. Ameliorative Role of Coenzyme Q10 in RF Radiation-Associated Testicular and Oxidative Impairments in a 3.5-GHz Exposure Model. Bioelectromagnetics. 2026;47(1):e70043. doi:10.1002/bem.70043.

Exposure. GSM-modulated 3.5 GHz,2 h/day for 30 days. Simulated whole-body SAR0.16995 W/kg; testis 0.02669 W/kg. CoQ10 10 mg/kg/day intraperitoneally.

Design and controls. 28 adult male Wistar rats in four groups: control, RF, CoQ10, RF+CoQ10; nominal n=7/group. Full methods and thermometry not retrieved.

Reported finding. Primary abstract reports lower gonadotropins/testosterone, increased oxidative markers and testicular degeneration, partially ameliorated by CoQ10.

Interpretive limits. GSM modulation at 3.5 GHz is not 5G NR. Full methods/statistics and actual fertility outcomes unverified. The SAR numbers do not establish human-limit compliance.

Programme and cohort links. Bektas/Van programme, with authors already represented; does not independently confirm other Bektas papers.

The protection question raised. Are the reported reproductive-tissue changes robust under verified thermal and exposure conditions? Full methods remain unreviewed, and GSM modulation at 3.5 GHz cannot be treated as a 5G NR experiment.

Source verification. Primary PubMed/publisher abstract and Scite abstract only. Retrieval lead.

Source locations. Abstract.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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RF-044 Prenatal 3.5 GHz exposure and delayed testicular endpoints: retrieval lead

> **Why this matters**
> A study of prenatal exposure looked for reproductive changes in male rats a year after birth. The abstract's delayed findings make follow-up beyond infancy an important question. Original dose information and an analysis accounting for offspring from the same litter remain essential to interpretation.

Original abstract verified | Source checks to 20 October 2026

Elif Gelenli Dolanbay; Tugay Mert; Gozde Caliskan Bender; Hava Bektas; Unal Uslu; Claudio Enrique Fernandez-Rodriguez; Suleyman Dasdag. Male Reproductive and Cellular Damage After Prenatal 3.5 GHz Radiation Exposure: One-Year Postnatal Effects. Annals of the New York Academy of Sciences. 2025;1554(1):140-152. doi:10.1111/nyas.70116.

Exposure. 3.5 GHz,2 h/day during full or late gestation; male offspring studied at 12 months. Dosimetry and waveform details absent from retrieved abstract.

Design and controls. Pregnant Wistar Hannover rats in sham, full-gestation and late-gestation groups; animal/litter counts and full methods not retrieved.

Reported finding. Primary abstract reports altered testicular tubule/epithelium morphology, Johnsen score, sperm morphology, γH2AX, Beclin 1 and TUNEL at 12 months.

Statistical detail. Abstract states Holm-Bonferroni correction; full-gestation Johnsen score adjusted P=.022 and sperm morphology P=.036. Extremely small tissue-marker P values need experimental-unit audit.

Interpretive limits. Abstract verified only. Dose, blinding, litter nesting and thermal controls unverified. Numerical significance cannot resolve those limits; no central causal use.

Programme and cohort links. Istanbul Medeniyet/Van programme with Bektas/Dasdag. Relationship to prenatal kidney paper 10.1007/s00418-026-02504-7 needs cohort checking.

The protection question raised. Could prenatal exposure affect reproductive development much later in life? The delayed endpoints are worth examining, but unverified dose, thermal controls and litter structure prevent a central causal inference.

Source verification. Original publisher abstract, conflict statement and Crossref metadata reviewed. Retrieval lead.

Source locations. Abstract and conflict declaration.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. Not established from retrieved material.

Conflicts. Authors declare no conflicts of interest.

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RF-045 Oxidative DNA-base damage in mouse spermatocyte-derived cells

> **Why this matters**
> DNA's chemical building blocks can be damaged even when a test for broken strands is negative. The abstract reports this distinction in a laboratory line of mouse reproductive cells. It points to a damage pathway for verification; full exposure controls and evidence about reproductive function remain missing.

Original abstract verified | Source checks to 20 October 2026

Chuan Liu; Weixia Duan; Shangcheng Xu; Chunhai Chen; Mindi He; Lei Zhang; Zhengping Yu; Zhou Zhou. Exposure to 1800MHz radiofrequency electromagnetic radiation induces oxidative DNA base damage in a mouse spermatocyte-derived cell line. Toxicology Letters. 2013;218(1):2-9. doi:10.1016/j.toxlet.2013.01.003.

Exposure. 1,800 MHz GSM-talk mode,1/2/4 W/kg,24 h intermittent exposure,5 min on/10 min off.

Design and controls. GC-2 mouse spermatocyte-derived cell line; FPG-modified comet,8-oxoG and ROS assays; α-tocopherol co-treatment.

Reported finding. Original abstract reports oxidative-base-damage and ROS signals at 4 W/kg, mitigated by antioxidant. Ordinary alkaline comet did not detect strand breakage.

Interpretive limits. Full methods, replicate counts and temperature controls not retrieved. Cell line is not mature sperm or a fertility endpoint. Preserve the different assay outcomes.

Programme and cohort links. Chongqing Third Military Medical University programme with Xu2010; distinct from Zhejiang Xu2013.

The protection question raised. Does the oxidative-base signal persist and affect reproductive-cell function? The negative ordinary comet result must remain alongside it. Full methods and temperature controls are still needed to interpret this abstract-level observation.

Source verification. Original PubMed abstract reviewed. Retrieval lead.

Source locations. Abstract.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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RF-046 Oxidative mitochondrial DNA markers in primary cortical neurons

> **Why this matters**
> Mitochondria supply cells with energy and contain their own DNA. In rat nerve cells grown in the laboratory, the abstract reports changes in that DNA after RF exposure. It identifies a possible dysfunction pathway; incomplete methods and temperature information prevent a conclusion about neurological harm in people.

Original abstract verified | Source checks to 20 October 2026

Shangcheng Xu; Zhou Zhou; Lei Zhang; Zhengping Yu; Wei Zhang; Yuan Wang; Xubu Wang; Maoquan Li; Yang Chen; Chunhai Chen; Mindi He; Guangbin Zhang; Min Zhong. Exposure to 1800 MHz radiofrequency radiation induces oxidative damage to mitochondrial DNA in primary cultured neurons. Brain Research. 2010;1311:189-196. doi:10.1016/j.brainres.2009.10.062.

Exposure. 1,800 MHz pulsed RF with 217 Hz modulation,average SAR2 W/kg; primary rat cortical neurons assessed 24 h after exposure.

Design and controls. Primary culture experiment with melatonin pretreatment; full methods, replicate counts and thermometry unverified.

Reported finding. Original abstract reports increased mitochondrial 8-OHdG and reduced mtDNA copy number/mtRNA transcripts, reversed by melatonin pretreatment.

Interpretive limits. No demonstrated clinical neurotoxicity. Abstract does not establish complete exposure duration or temperature controls. Antioxidant rescue alone does not establish RF specificity.

Programme and cohort links. Chongqing Third Military Medical University programme, linked to Liu 2013 oxidative germ-cell research.

The protection question raised. Could the mitochondrial changes impair neuronal function? Incomplete exposure and thermal information, and the limits of antioxidant rescue, leave the proposed pathway provisional rather than demonstrated neurological harm.

Source verification. Original PubMed abstract reviewed. Retrieval lead.

Source locations. Abstract.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. PubMed labels non-US government support; detailed funder unverified.

Conflicts. Not established from retrieved material.

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RF-047 Earlier Newcastle male-germline mouse experiment

> **Why this matters**
> An earlier Newcastle experiment reported changes in selected DNA targets in exposed mice without changes in sperm function. It helps trace how the later reproductive studies developed. The full methods still need review, and several papers from this group must not be mistaken for several independent laboratory confirmations.

Original abstract verified | Source checks to 20 October 2026

R. J. AITKEN; L. E. BENNETTS; D. SAWYER; A. M. WIKLENDT; B. V. KING. Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline. International Journal of Andrology. 2005;28(3):171-179. doi:10.1111/j.1365-2605.2005.00531.x.

Exposure. 900 MHz,12 h/day for 7 days; reported whole-body SAR approximately 0.09 W/kg still needs full-method verification.

Design and controls. Mouse exposure; gene-specific quantitative PCR, electrophoresis and sperm-function assays.

Reported finding. Original abstract reports damage at mitochondrial and nuclear β-globin PCR loci, without gross electrophoresis strand breaks or sperm-function changes.

Interpretive limits. PCR amplification endpoints are not interchangeable with comet/TUNEL or infertility. SAR calculation and thermal controls not rechecked in full.

Programme and cohort links. Foundational Newcastle/Aitken-King programme behind RF-006/007/008; not an independent network.

The protection question raised. How do the selected DNA-amplification endpoints relate to later sperm findings? The unchanged sperm function and related research lineage prevent counting this as independent confirmation of reproductive injury.

Source verification. Original PubMed abstract reviewed. Retrieval lead.

Source locations. Abstract.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. Not verified from original full text.

Conflicts. Not established from retrieved material.

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RF-048 Marine-radar workers and biomarkers: retrieval lead

> **Why this matters**
> People working near radar bring a different exposure setting into the evidence. This study's abstract reports more DNA-related and chemical-stress markers in exposed workers. Personal dose histories and other workplace exposures still need examination; these measurements alone do not show that RF caused disease or that limits were exceeded.

Original abstract verified | Source checks to 20 October 2026

Vera Garaj-Vrhovac; Goran Gajski; Senijo Pažanin; Antonio Šarolić; Ana-Marija Domijan; Dubravka Flajs; Maja Peraica. Assessment of cytogenetic damage and oxidative stress in personnel occupationally exposed to the pulsed microwave radiation of marine radar equipment. International Journal of Hygiene and Environmental Health. 2011;214(1):59-65. doi:10.1016/j.ijheh.2010.08.003.

Exposure. Pulsed marine-radar RF at 3,5.5 and 9.4 GHz. Authors measured fields and determined SAR, but numerical personal dose/history not retrieved.

Design and controls. Workers versus unexposed subjects; comet, micronucleus, glutathione and MDA assays. Full sample/confounder/blinding details unverified.

Reported finding. Original abstract reports higher comet/micronucleus damage and MDA, and lower glutathione in exposed workers.

Interpretive limits. Observational biomarkers do not establish RF causation, disease or exposure-limit compliance. Personal dose, covariates and participant overlap require full audit.

Programme and cohort links. Zagreb Institute for Medical Research and Occupational Health programme. Earlier radar micronucleus/conference reports may share participants; independence unverified.

The protection question raised. Do the worker biomarkers track RF exposure independently of other workplace factors? Original dose, covariate and participant-overlap checks remain necessary before a causal or clinical protection conclusion.

Source verification. Original PubMed/publisher abstract and institutional CroRIS publication record reviewed. Retrieval lead.

Source locations. Abstract; institutional publication record.

Editorial status. No editorial notice surfaced in the Scite/primary-record checks used for this record; this is not a comprehensive clean-bill certification.

Funding. Not verified from original full text.

Conflicts. Not established from retrieved material.

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RF-050 Early microwave brain DNA experiment

> **Why this matters**
> Some DNA effects may be missed if researchers look only immediately after exposure. This rat study's abstract reported a delayed increase in single-strand breaks after pulsed microwaves. Full methods and replication comparisons remain unfinished, so its contribution is a defined timing question rather than an established human hazard.

Original abstract verified | Source checks to 20 October 2026

Lai H, Singh NP. Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. Bioelectromagnetics. 1995;16(3):207-210. doi:10.1002/bem.2250160309.

Exposure. 2450 MHz, two hours; pulsed 2 microsecond pulses at 500/s, whole-body SAR 0.6 or 1.2 W/kg; continuous wave 1.2 W/kg.

Design and controls. Rat-brain alkaline microgel electrophoresis; original abstract verified. Animal counts and complete controls require full paper.

Reported finding. Pulsed exposure increased single-strand breaks at four hours after exposure, with no significant immediate effect. Continuous-wave exposure at 1.2 W/kg increased breaks immediately and at four hours.

Interpretive limits. Original abstract only; assay controls and independent replication history not fully audited. No below-limit or established injury inference.

Programme and cohort links. RF-051 extends same University of Washington programme; not independent laboratory replication.

The protection question raised. Does delayed DNA strand breakage persist across validated assays and independent experiments? The abstract identifies a timing question, but unfinished methods and replication checks prevent an established injury or human-limit claim.

Source verification. Scite original abstract and Wiley primary record verified

Source locations. Original abstract; pp.207-210 full methods pending

Editorial status. No notice surfaced in Scite or targeted title/DOI search; full publisher status audit incomplete.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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RF-051 Follow-up acute microwave DNA strand-break experiment

> **Why this matters**
> A follow-up reported breaks affecting one or both strands of DNA in rat brain cells after exposure. It broadens the laboratory endpoint examined in RF-050, while remaining part of the same research programme. Full assay controls and replication history remain necessary before treating this as confirmed injury.

Original abstract verified | Source checks to 20 October 2026

Lai H, Singh NP. Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation. Int J Radiat Biol. 1996;69(4):513-521. doi:10.1080/095530096145814.

Exposure. 2450 MHz pulsed (2 microseconds, 500/s) or continuous-wave; two hours; spatial-average incident power density 2 mW/cm2 and whole-body SAR 1.2 W/kg.

Design and controls. Rat-brain microgel electrophoresis at four hours post-exposure; original abstract checked; exact n and assay quality-control procedures pending.

Reported finding. Original abstract reports increased single- and double-strand breaks after either signal, with no significant difference between pulsed and continuous-wave effects.

Interpretive limits. Mechanistic research lead; full methods and conflicting replication protocols require comparison before a causal generalisation. Whole-body animal exposure does not establish below-limit human injury.

Programme and cohort links. Same programme as RF-050; count the publications separately from independent laboratories.

The protection question raised. Are the reported strand-break findings reproducible across comparable assays and exposure conditions? Related investigators and unresolved replication differences limit a general mechanism or human protection inference.

Source verification. Scite original abstract and metadata verified

Source locations. Original abstract; pp.513-521 methods pending

Editorial status. No notice surfaced in Scite or targeted title/DOI search; not a complete status certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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ELF-010 Power-frequency brain DNA response and chemical interventions

> **Why this matters**
> The abstract reports DNA strand breaks in rat brain cells after prolonged power-frequency magnetic-field exposure, with several chemical interventions blocking the response. It suggests a pathway to test, rather than a settled explanation of harm. Full methods and independent replication remain to be assessed, separately from radiofrequency studies.

Original abstract verified | Source checks to 20 October 2026

Lai H, Singh NP. Magnetic-field-induced DNA strand breaks in brain cells of the rat. Environ Health Perspect. 2004;112(6):687-694. doi:10.1289/ehp.6355.

Exposure. 60 Hz magnetic field at 0.01 mT for 24 or 48 h; additional acute experiments described in original abstract.

Design and controls. Rat-brain assays with antioxidant, nitric-oxide-synthase inhibitor and iron-chelator interventions; exact n and full methods pending.

Reported finding. Original abstract reports strand-break increases after 24 h, larger after 48 h, blocked by Trolox, 7-nitroindazole and deferiprone; apoptosis/necrosis also reported in acute experiments.

Interpretive limits. Power-frequency experiment, distinct from GHz RF. Interventions support hypotheses, not a settled general mechanism; full methods/replication audit incomplete.

Programme and cohort links. Same Washington investigators as RF-050 and RF-051; different field regime.

The protection question raised. Do the chemical interventions identify a reproducible pathway for the reported DNA response? Abstract-only verification and incomplete replication checks leave that mechanism provisional and separate from GHz RF evidence.

Source verification. Original abstract retrieved through Scite

Source locations. Original abstract; pp.687-694 full methods pending

Editorial status. No notice surfaced in Scite metadata; targeted status audit incomplete.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

Retrieve. DOI or primary source | PubMed record

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ECO-005 Fly reproduction and exposure intensity windows

> **Why this matters**
> The reported effects concern fly reproduction, a meaningful outcome for an organism, rather than a cell marker alone. Main-study methods still need checking. A published correction fixes figure labels and must accompany the paper; it does not by itself establish or invalidate the reported reproductive effect.

Original abstract verified | Source checks to 20 October 2026

Panagopoulos DJ, Chavdoula ED, Margaritis LH. Bioeffects of mobile telephony radiation in relation to its intensity or distance from the antenna. International Journal of Radiation Biology. 2010;86(5):345-357. doi:10.3109/09553000903567961.

Exposure. See original source for calibrated field and modulation conditions.

Design and controls. Observational or experimental ecology; original methods pending.

Reported finding. Discovery dossier reports reduced reproduction and gonadal cell death across selected handset-exposure conditions.

Interpretive limits. A retrieval entry, not a verified ecological causal conclusion.

Programme and cohort links. Athens Panagopoulos/Chavdoula/Margaritis programme. Companion intensity-window paper 10.3109/09553000903567979 is related research and has a separate September 2010 erratum; do not conflate notices.

The protection question raised. Do the original exposure conditions and analyses support the reported fly reproductive effects? Methods remain to be checked, and the figure-label correction belongs with any interpretation of this retrieval lead.

Source verification. Original PubMed abstract and complete publisher erratum reviewed; full main-study methods still outstanding.

Source locations. Original abstract; erratum p612/Figure 5 c.

Editorial status. Publisher erratum 1July 2010, DOI10.3109/09553002.2010.497612, corrects incompletely labelled Figure 5 c; original online figure corrected. This is a figure-label correction,not a retraction.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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Annex C Replication and shared cohort context

These records supply specific context needed to describe an included adverse finding accurately. They identify nonconfirming replication or shared-cohort results. They are not an assembled sample of the broader reassuring literature and should not be counted as additional affirmative experiments.

Records in this section: CTX-001, CTX-002, CTX-003, CTX-004, CTX-005, CTX-006

CTX-001 The International Collaborative Animal Study of mobile phone radiofrequency radiation carcinogenicity and genotoxicity: the Japanese study

> **Why this matters**
> This Japanese follow-up did not find significant increases in the brain and heart tumours being investigated. That limits the consistency of the cancer evidence. Survival differed between exposed and comparison animals, and an equipment failure altered part of the exposure; those details matter when comparing the studies.

Replication and cohort context | Source checks to 20 October 2026

Imaida K, Kawabe M, Wang J, Yokohira M, Imai N, Han KH, Kim YB, Jeon SB, Kim HS, Ahn YH. The International Collaborative Animal Study of mobile phone radiofrequency radiation carcinogenicity and genotoxicity: the Japanese study. Toxicological Sciences. 2026;209(3):kfag002. doi:10.1093/toxsci/kfag002.

Exposure. 900 MHz CDMA, target whole-body SAR 4 W/kg in reverberation chambers; daily 18 h20 with 10-min on/off cycles (about 9 h10 RF-on), from maternal gestational day 5 through lactation to postnatal day 21 and then 104 weeks after weaning. Approximately four weeks ran at about 2 W/kg because one amplifier failed.

Design and controls. 70 male Hsd:Sprague Dawley SD offspring/group selected at postnatal day 21 from three maternal groups (30 pregnant females/group): RF, chamber sham and cage control. RF pathology denominator 68 after two equipment-malfunction deaths were excluded.

Reported finding. Male-rat follow-up at 4 W/kg, 900 MHz CDMA did not reproduce significant glioma/cardiac-schwannoma increases. Heart schwannoma: one in each cage/sham/RF group. Survival at 105 weeks: cage 34.3% (24/70), sham 42.9% (30/70), RF 64.7% (44/68); two equipment-malfunction deaths excluded. Retain sham comparison.

Interpretive limits. Specific replication context for RF-001. Lower sham than exposed survival, the amplifier interruption and denominator exclusions require attention. This protocol does not reproduce the Ramazzini lifetime far-field experiment. The Japanese survival issue cannot be generalised to the separate Korean arm.

The protection question raised. What explains the different tumour findings across protocols? Japanese survival and equipment issues matter, but cannot explain the separate Korean result or substitute for comparison with the Ramazzini exposure arrangement.

Source verification. Primary full-text results/survival and publisher/PubMed records reviewed; not a complete supplement audit.

Source locations. Original accepted manuscript Materials and Methods pp.6-7 (exposure/amplifier); 2-Year Carcinogenicity Study p.12; Figure 2; Results survival p.18. Final Table 2 confirms group denominators 70/70/68.

Editorial status. No editorial notice located in DOI/title checks on 2026-10-20; publisher notice-history audit not exhaustive.

Funding. Japan Ministry of Internal Affairs and Communications grant 0155-0094, with Korean-government MSIT/IITP grants RS-2024-00466966 and 2019-0-00102; authors declare no conflicts. Funding text checked in accessible original accepted-manuscript PDF, p 31; final funding-page recheck not completed.

Conflicts. Authors declare no conflicts or competing interests, as documented in the retrieved source disclosure.

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CTX-002 The International Collaborative Animal Study of the carcinogenicity and genotoxicity of mobile phone radiofrequency radiation: the Korean study

> **Why this matters**
> The Korean arm also did not confirm the tumour findings, and its exposed and comparison animals had similar survival. That distinction prevents the Japanese survival issue from becoming an explanation for both results. Interpretation must account for this independent arm while preserving the differences from the original positive experiments.

Replication and cohort context | Source checks to 20 October 2026

Kim HS, Han KH, Kim YB, Jeon SB, Lee AK, Moon JI, Choi HD, Imaida K, Yokohira M, Kawabe M, Imai N, Wang J, Ahn YH. The International Collaborative Animal Study of the carcinogenicity and genotoxicity of mobile phone radiofrequency radiation: the Korean study. Toxicological Sciences. 2026;209(3):kfag001. doi:10.1093/toxsci/kfag001.

Exposure. 900 MHz CDMA-IS95A (1.25-MHz bandwidth), whole-body average SAR 4 W/kg in a reverberation chamber; gestational days 5-21, postnatal days 1-21, then 104 weeks post-weaning (postnatal days 22-749). Daily 18 h20 in 10-min on/off cycles, seven days/week (about 9 h10 RF-on).

Design and controls. Three groups: RF, identical chamber sham without RF, and standard cage control; 70 male Hsd:Sprague Dawley SD rats/group for the two-year study. Shared ICAS protocol with Japan; separately conducted and analysed.

Reported finding. Separate Korean arm: 70 male rats/group; 900 MHz CDMA, 4 W/kg did not reproduce significant tumour/genotoxic changes. Survival 58.6% RF vs 54.3% sham at 104 weeks was comparable. Shared protocol with Japan; independently conducted/analysed.

Interpretive limits. Important reproducibility boundary; Japanese differential-survival caveat cannot be applied to Korean arm. No sweeping human-safety inference.

The protection question raised. How do the nonconfirming Korean results constrain the positive NTP inference? Comparable group survival rules out borrowing the Japanese survival explanation; exposure and protocol differences still require careful interpretation.

Source verification. Primary methods/abstract/results and government BfS discussion reviewed; not full supplemental audit.

Source locations. Original Materials and Methods: RF exposure system; Animal assignment; RF exposure schedule and Figure 2B; 2-year study. Original results survival and tumour tables; final PDF p.14 disclosures.

Editorial status. No editorial notice located in DOI/title checks on 2026-10-20; publisher notice-history audit not exhaustive.

Funding. Korean-government MSIT/IITP grants RS-2024-00466966 and 2019-0-00102, with Japan MIC grant 0155-0094. Original final PDF p 14 states no actual/potential conflicts, no RF-technology commercial funding and no funder role in design, collection, analysis, interpretation, writing or submission decision.

Conflicts. Authors declare no actual or potential conflicts; no RF-technology commercial funding.

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CTX-003 No Increased DNA Damage Observed in the Brain, Liver, and Lung of Fetal Mice Treated With Ethylnitrosourea and Exposed to UMTS Radiofrequency Electromagnetic Fields

> **Why this matters**
> Researchers tested whether RF increased an early form of chemical damage to fetal DNA after a cancer-causing chemical. They did not find the proposed increase. That result limits one explanation of the tumour-promotion findings; it does not repeat the lifetime cancer experiments or settle every possible mechanism.

Replication and cohort context | Source checks to 20 October 2026

Lerchl A, Klose M, Drees K. No Increased DNA Damage Observed in the Brain, Liver, and Lung of Fetal Mice Treated With Ethylnitrosourea and Exposed to UMTS Radiofrequency Electromagnetic Fields. Bioelectromagnetics. 2020;41(8):611-616. doi:10.1002/bem.22301.

Exposure. Approximately 1960 MHz UMTS from gestational day 7, sham/0.04/0.4 W/kg; ENU 40 mg/kg on day 14; fetal brain/liver/lung examined 24, 36 and 72 hours later. Primary abstract gives 24-57 fetuses per subgroup.

Design and controls. ENU-treated pregnancies with sham or UMTS exposure; fetal brain, liver and lung assessed at 24, 36 and 72 hours after ENU. Primary abstract gives 24-57 fetuses per subgroup; fetal counts do not establish independent dam counts.

Reported finding. Fetal follow-up at 0.04/0.4 W/kg did not increase adenyl-DNA-adduct signal after ENU at 24/36/72 hours. Does not retest lifetime tumours or rule out every genotoxic mechanism.

Interpretive limits. Keep mechanism unresolved; do not present early ENU-adduct potentiation as established.

Programme and cohort links. 2018 BfS-RESFOR-130/18, urn:nbn:de:0221-2018011014465, reports the same mechanistic project; count once.

The protection question raised. What mechanism could account for the lifetime promotion findings? This result leaves early fetal DNA-adduct potentiation unsupported, without retesting tumours or excluding every possible interaction pathway.

Source verification. Original abstract plus 2018 government report methods/results reviewed; not whole 2020 article.

Source locations. Original 2020 abstract; Table 1 and Experimental Design/RF-EMF Exposure; 2018 government project methods/results (same project). Full 2020 article not completely audited.

Editorial status. No editorial notice located in DOI/title checks on 2026-10-20; publisher notice-history audit not exhaustive.

Funding. BfS project 3615S82431 is documented in the linked 2018 government report; the 2020 original acknowledgment notes Projekt DEAL open-access support. Full original journal conflict/funding statement not completely audited.

Conflicts. Not established from retrieved material.

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CTX-004 Carcinogenicity Study of 217 Hz Pulsed 900 MHz Electromagnetic Fields inPim1Transgenic Mice

> **Why this matters**
> An independent attempt did not reproduce the lymphoma increase seen in genetically susceptible mice. This keeps a striking original finding from being presented as a settled susceptibility effect. Differences in dose and restraint need comparison, but they cannot simply be assumed to explain the failure to replicate.

Replication and cohort context | Source checks to 20 October 2026

Oberto G, Rolfo K, Yu P, Carbonatto M, Peano S, Kuster N, Ebert S, Tofani S. Carcinogenicity Study of 217 Hz Pulsed 900 MHz Electromagnetic Fields inPim1Transgenic Mice. Radiation Research. 2007;168(3):316-326. doi:10.1667/rr0425.1.

Exposure. 900 MHz, 217-Hz pulsed fields; whole-body SAR 0.5, 1.4 or 4 W/kg; one hour/day for 18 months.

Design and controls. E-mu-Pim1 transgenic mice; 50 males and 50 females/group, with RF groups versus sham and cage controls. Blinded attempted replication of RF-024. Tubes restrained animals, unlike the original free-movement arrangement.

Reported finding. Blinded attempted RF-024 replication: 50/sex/group, 0.5/1.4/4 W/kg, one hour/day for 18 months; no tumour-site increase. Tubes restrained animals, unlike original free movement.

Interpretive limits. RF-024 should not be described as independently established. Utteridge 2002 also did not confirm; DOI 10.1667/0033-7587(2002)158[0357:LTEOEP]2.0.CO;2.

The protection question raised. Is the original lymphoma signal reproducible in the susceptible model? Differences in restraint and exposure remain relevant, but nonconfirming attempts prevent treating the effect as independently established.

Source verification. Primary PubMed abstract/method summary checked.

Source locations. Original abstract, Radiation Research 2007;168(3):316-326; full-method audit incomplete.

Editorial status. No additional editorial notice established in the retrieved original record.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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CTX-005 Essential same-cohort boundary for Nittby's cognitive finding

> **Why this matters**
> These are the same rats used for the memory paper RF-029. Their brain-tissue examination did not demonstrate neuronal injury. Reading both papers together prevents a memory-test result from becoming an unsupported claim of brain-cell loss, and prevents one experiment from being counted twice.

Replication and cohort context | Source checks to 20 October 2026

Grafström G, Nittby H, Brun A, Malmgren L, Persson BRR, Salford LG, Eberhardt J. Histopathological examinations of rat brains after long-term exposure to GSM-900 mobile phone radiation. Brain Research Bulletin. 2008;77(5):257-263. doi:10.1016/j.brainresbull.2008.08.004.

Exposure. Same weekly GSM-900 exposures for 55 weeks as RF-029; initial whole-body SAR 0.0006 or 0.06 W/kg; final approximately 0.0004 or 0.04 W/kg. Animals examined 6-7 weeks after last exposure.

Design and controls. Original methods: 56 randomized mixed-sex Fischer 344 rats, four groups (16 low,16 high,16 sham,8 cage); 54 brains available; neuropathologist blind, cresyl violet/albumin/GFAP/Sudan Black B/Gallyas staining.

Reported finding. Blinded histology found no significant group differences in albumin leakage, dark neurons, glial reactions, lipofuscin or cytoskeletal/neuritic changes. No correlation joined albumin leakage/dark neurons to cognitive performance. Thus the cognitive observation was not explained by demonstrated neuronal damage.

Interpretive limits. Included solely as a necessary same-cohort interpretive boundary, not as a separately recruited reassuring-literature sample. Brain-SAR orientation variability was substantial (56% standard deviation around average). Later review quoted an erroneous 0.06-0.6 W/kg range; use original units.

Programme and cohort links. Identical animals to RF-029; count once. Earlier acute Salford experiments differ in regimen and age.

The protection question raised. Can a memory-test difference be explained by neuronal damage in these rats? The companion histology did not demonstrate it. Functional and tissue endpoints remain distinct, and these publications represent the same cohort.

Source verification. Original indexed body, all 35,875 characters read through Scite

Source locations. GSM exposure; Animals; Histopathology and methods; Histopathological examinations; Individual correlation; Discussion

Editorial status. Scite metadata checked 20 Oct 2026; no editorial notice returned. Primary bibliographic records and targeted notices were checked where retrievable; no retraction or expression of concern located. This is not certification that no notice exists.

Funding. Not stated in indexed body obtained; not inferred.

Conflicts. Not stated in indexed body obtained; not inferred.

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CTX-006 Independent test limiting the certainty of the Salford finding

> **Why this matters**
> An independent laboratory did not confirm Salford's reported brain-tissue changes. The exposure range and tissue staining were not identical, so the comparison needs care. It keeps reproducibility central. A common blinded protocol could help clarify the difference before conclusions about harmful exposure levels.

Replication and cohort context | Source checks to 20 October 2026

Masuda H, Ushiyama A, Takahashi M, Wang J, Fujiwara O, Hikage T, Nojima T, Fujita K, Kudo M, Ohkubo C. Effects of 915 MHz Electromagnetic-Field Radiation in TEM Cell on the Blood-Brain Barrier and Neurons in the Rat Brain. Radiation Research. 2009;172(1):66-73. doi:10.1667/rr1542.1.

Exposure. 915 MHz TEM cells, one 2-h exposure; whole-body average SAR 0,0.02,0.2,2 W/kg; examination 14 and 50 days later.

Design and controls. 64 male F344 rats aged 12 weeks; histology and immunohistochemistry; hematoxylin/eosin dark-neuron assessment. Full methodological audit incomplete.

Reported finding. Original abstract: no albumin immunoreactivity in exposed groups; dark neurons rare and not significantly different between active and sham groups. The authors explicitly failed to confirm Salford's result.

Interpretive limits. Specific replication context required for faithful RF-020 interpretation. Does not test Salford's lowest 0.002-W/kg group, and differs in sex/age selection and staining. Not used as a general safety conclusion.

Programme and cohort links. Independent National Institute of Public Health/Japanese collaborators; targeted RF-020 replication rather than related Lund follow-up.

The protection question raised. How do assay, animal and exposure differences affect the original pathology signal? Nonconfirmation limits certainty; neither small study alone can establish a harmful threshold or a general human safety conclusion.

Source verification. Original abstract through Scite/PubMed; Kyoto repository link identified but did not return readable PDF

Source locations. Original abstract; article pp.66-73 full-methods retrieval outstanding

Editorial status. Scite metadata checked 20 Oct 2026; no editorial notice returned. Primary bibliographic records and targeted notices were checked where retrievable; no retraction or expression of concern located. This is not certification that no notice exists.

Funding. PubMed classifies non-US government support; specific grant not verified.

Conflicts. Not reported in retrieved abstract.

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Annex D Retrieval leads

Bibliographic identities have been checked, but central details remain dependent on the discovery dossier or incomplete retrieval. These entries do not underpin unqualified findings in the report. The record states the retrieval gap rather than supplying an unsupported numerical claim.

Records in this section: RF-017, RF-023, ELF-001, ELF-002, ELF-003, ELF-005, ELF-006, ELF-008, SF-001, SF-002, SF-003, SF-004, ECO-001, ECO-002

RF-017 Phone exposure and human semen in vitro

> **Why this matters**
> This pilot study may help compare how sperm samples behave near an active phone. The original methods still need retrieval, so its reported changes remain a lead, not verified evidence in this package. A device's exposure rating does not establish the dose received by a semen sample.

Retrieval lead | Source checks to 20 October 2026

Agarwal A, Desai NR, Makker K, Varghese A, Mouradi R, Sabanegh E, Sharma R. Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an in vitro pilot study. Fertility and Sterility. 2009;92(4):1318-1325. doi:10.1016/j.fertnstert.2008.08.022.

Exposure. 850 MHz talk-mode phone for one hour; reported device SAR 1.46 W/kg.

Design and controls. Paired semen aliquots.

Reported finding. Discovery dossier reports lower motility/viability and higher ROS in exposed aliquots.

Interpretive limits. Device rating is not established sample SAR. Do not label this below FCC limits from that number.

The protection question raised. Could the reported semen changes persist under verified sample exposure and temperature conditions? Original methods remain to be checked; a handset rating does not establish the absorbed dose in semen.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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RF-023 New hippocampal study awaiting original methods

> **Why this matters**
> This is a retrieval lead: the title points to changes in a rat brain region involved in memory. The original paper is worth obtaining to examine the exposure and tissue findings together. Only its bibliographic identity is verified here; methods and numerical claims cannot yet support a conclusion about injury.

Retrieval lead | Source checks to 20 October 2026

Bektas H, Cakir S, Alizade A, Dogu S, Altindag F. Chronic 3.5 GHz radiofrequency exposure is associated with behavioral, molecular, and histopathological changes in the rat hippocampus. International Journal of Radiation Biology. 2026;:1-14. doi:10.1080/09553002.2026.2708930.

Exposure. 3.5 GHz; discovery dosimetry values not independently verified.

Design and controls. Rat experiment; publication date 7 August 2026.

Reported finding. Title identifies behavioural, molecular and histopathological hippocampal changes; discovery dossier contains detailed claims requiring original-source checking.

Interpretive limits. Metadata only. Do not use its numerical results or dose as a featured claim until primary text is obtained.

The protection question raised. The title identifies a potentially important brain-tissue question. Until the original exposure, methods and results are obtained, this retrieval lead cannot support a conclusion about injury or protection.

Source verification. Title DOI authors and date verified; no readable article text served

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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ELF-001 Residential magnetic fields and childhood cancer

> **Why this matters**
> A report about childhood leukaemia deserves careful attention, particularly when it concerns where children live. This Swedish power-line study remains a retrieval lead here: the original exposure estimates and results need checking. It concerns power-frequency magnetic fields, a different exposure from mobile-phone signals.

Retrieval lead | Source checks to 20 October 2026

Feychting M, Alhbom M. Magnetic Fields and Cancer in Children Residing Near Swedish High-voltage Power Lines. American Journal of Epidemiology. 1993;138(7):467-481. doi:10.1093/oxfordjournals.aje.a116881.

Exposure. Calculated residential power-line magnetic fields.

Design and controls. Swedish case-control study.

Reported finding. Affirmative childhood-leukaemia strand identified in discovery dossier.

Interpretive limits. Retrieve original exposure categories and estimates before numerical use. ELF findings are not RF findings.

The protection question raised. Does the original residential magnetic-field analysis support the reported childhood-leukaemia association? Exposure categories and estimates remain to be verified; this power-frequency retrieval lead cannot establish an RF hazard.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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ELF-002 Childhood leukaemia and distance from power lines

> **Why this matters**
> This retrieval lead concerns childhood leukaemia and the distance between a birth address and high-voltage lines. Location is easy to describe, but it does not tell us the magnetic field a child experienced. The original analyses need checking before this observation can inform a protection assessment.

Retrieval lead | Source checks to 20 October 2026

Draper G, Vincent T, Kroll ME, Swanson J. Childhood cancer in relation to distance from high voltage power lines in England and Wales: a case-control study. BMJ. 2005;330(7503):1290. doi:10.1136/bmj.330.7503.1290.

Exposure. Distance from high-voltage lines rather than measured fields.

Design and controls. England and Wales case-control study.

Reported finding. Discovery dossier reports increased leukaemia risk in birth-address distance categories near power lines.

Interpretive limits. Distance association is not an absorbed-dose measurement; not evidence about mobile-phone RF.

The protection question raised. What does distance from a power line tell us about the reported leukaemia association? Original analyses remain unreviewed, and location alone does not establish a child's magnetic-field exposure.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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ELF-003 Personal magnetic measurements during pregnancy

Why this matters

This retrieval lead asks whether short peaks in magnetic-field exposure during pregnancy relate to miscarriage. The reported association used the maximum reading rather than the daily average. Checking the original measurement-day selection and analyses is essential before drawing a health conclusion from such a sensitive outcome.

Retrieval lead | Source checks to 20 October 2026

Li DK, Odouli R, Wi S, Janevic T, Golditch I, Bracken TD, Senior R, Rankin R, Iriye R. A Population-Based Prospective Cohort Study of Personal Exposure to Magnetic Fields during Pregnancy and the Risk of Miscarriage. Epidemiology. 2002;13(1):9-20. doi:10.1097/00001648-200201000-00004.

Exposure. 24-hour personal monitoring during early pregnancy.

Design and controls. Prospective cohort; 969 participants reported in discovery dossier.

Reported finding. Discovery dossier reports a miscarriage association with maximum, rather than average, measured magnetic field.

Interpretive limits. Original methods and exposure-day selection need checking before featuring.

The protection question raised. Does a maximum magnetic-field reading capture a relevant pregnancy exposure or a measurement-day artefact? Original methods and exposure-day selection remain to be checked before this retrieval lead informs a health conclusion.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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ELF-005 Long residential duration and dementia mortality

> **Why this matters**
> This retrieval lead links a long residential history with a reported dementia-mortality association. Its particular question is whether duration matters, rather than only where someone lives at one moment. Original analyses and overlapping cohorts still need checking; proximity alone cannot establish what caused an individual's illness.

Retrieval lead | Source checks to 20 October 2026

Huss A, Spoerri A, Egger M, Roosli M, for the Swiss National Cohort Study. Residence Near Power Lines and Mortality From Neurodegenerative Diseases: Longitudinal Study of the Swiss Population. American Journal of Epidemiology. 2008;169(2):167-175. doi:10.1093/aje/kwn297.

Exposure. Residential proximity and duration; power-frequency regime.

Design and controls. Swiss national cohort.

Reported finding. Discovery dossier reports increasing Alzheimer mortality association with longer residence near high-voltage lines.

Interpretive limits. Observational mortality association; newer overlapping cohorts are extensions, not independent replications.

The protection question raised. Does duration near high-voltage lines add a credible dementia-mortality signal? Original analyses and overlapping cohorts remain to be checked; proximity cannot establish the cause of an individual illness.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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ELF-006 Tumour promotion under a power frequency magnetic field

> **Why this matters**
> Rats exposed to a cancer-causing chemical reportedly developed more and larger mammary tumours under a power-frequency magnetic field. This remains a retrieval lead. It may help examine combined exposures, provided the original results and follow-up experiments are checked and related Hannover publications are not counted as independent laboratories.

Retrieval lead | Source checks to 20 October 2026

Löscher W, Mevissen M, Lehmacher W, Stamm A. Tumor promotion in a breast cancer model by exposure to a weak alternating magnetic field. Cancer Letters. 1993;71(1-3):75-81. doi:10.1016/0304-3835(93)90100-n.

Exposure. 50 Hz; 100 microtesla; 91 days reported in discovery dossier.

Design and controls. DMBA-treated rats; 99 exposed and 99 sham reported.

Reported finding. Discovery dossier reports more and larger mammary tumours in a chemically initiated rat model.

Interpretive limits. Retrieve original and follow-up experiments; repeated Hannover papers are not separate laboratories.

The protection question raised. Is the reported mammary-tumour promotion reproducible in this chemically initiated model? Original and follow-up papers remain retrieval work, and related Hannover publications are not independent laboratory confirmations.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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ELF-008 Intermittency and fibroblast DNA strand breaks

> **Why this matters**
> This retrieval lead suggests that switching a power-frequency magnetic field on and off may produce a different cell response from continuous exposure. That distinction could matter to experimental design. The original methods and publication history still need review before the reported DNA-test result can carry a health-protection claim.

Retrieval lead | Source checks to 20 October 2026

Ivancsits S, Diem E, Pilger A, Rüdiger HW, Jahn O. Induction of DNA strand breaks by intermittent exposure to extremely-low-frequency electromagnetic fields in human diploid fibroblasts. Mutation Research/Genetic Toxicology and Environmental Mutagenesis. 2002;519(1-2):1-13. doi:10.1016/s1383-5718(02)00109-2.

Exposure. 50 Hz; 1 millitesla; human fibroblasts.

Design and controls. In-vitro experiment.

Reported finding. Discovery dossier reports comet-assay increases under intermittent but not continuous exposure.

Interpretive limits. Publication-status and original methods audit incomplete; no claim of below-limit residential injury.

The protection question raised. Could intermittent and continuous power-frequency fields produce different cellular responses? The original methods and publication-status checks remain incomplete, preventing a residential injury or protective-limit inference.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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SF-001 Transient balance changes at 7 tesla

> **Why this matters**
> This retrieval lead concerns temporary balance effects around a very strong MRI magnet. Such effects could matter to the experience of a scan and to work near scanners. The original experiment still needs review; an MRI static field is physically different from a phone or Wi-Fi signal.

Retrieval lead | Source checks to 20 October 2026

Theysohn JM, Kraff O, Eilers K, Andrade D, Gerwig M, Timmann D, Schmitt F, Ladd ME, Ladd SC, Bitz AK. Vestibular Effects of a 7 Tesla MRI Examination Compared to 1.5 T and 0 T in Healthy Volunteers. PLoS ONE. 2014;9(3):e92104. doi:10.1371/journal.pone.0092104.

Exposure. Tesla-scale MRI static magnetic fields and motion through gradients.

Design and controls. Human experiment.

Reported finding. Affirmative vestibular or neurobehavioural finding described in discovery dossier.

Interpretive limits. Retained in a separate regime-specific catalogue. No inference to Wi-Fi or mobile-phone exposures.

The protection question raised. How might temporary balance changes affect scanning or work near strong MRI magnets? The original experiment remains to be reviewed; this static-field lead cannot support a phone or Wi-Fi protection claim.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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SF-002 Performance during movement in MRI stray fields

> **Why this matters**
> Work near an MRI scanner raises a question about movement through the field as well as field strength. This human-experiment retrieval lead concerns short-term performance. Its original methods and results still need verification before it can inform workplace practice; it does not assess mobile-phone exposure.

Retrieval lead | Source checks to 20 October 2026

de Vocht F, Stevens T, van Wendel-de-Joode B, Engels H, Kromhout H. Acute neurobehavioral effects of exposure to static magnetic fields: Analyses of exposure-response relations. Journal of Magnetic Resonance Imaging. 2006;23(3):291-297. doi:10.1002/jmri.20510.

Exposure. Tesla-scale MRI static magnetic fields and motion through gradients.

Design and controls. Human experiment.

Reported finding. Affirmative vestibular or neurobehavioural finding described in discovery dossier.

Interpretive limits. Retained in a separate regime-specific catalogue. No inference to Wi-Fi or mobile-phone exposures.

The protection question raised. Does movement through an MRI stray field affect short-term performance? Verification of the original methods and results remains necessary before this lead can inform workplace protection.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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SF-003 Sex differences in MRI associated dizziness

> **Why this matters**
> This retrieval lead raises a specific question about whether women and men experience MRI-related dizziness differently. Understanding any reproducible difference could help assessments account for individual susceptibility. The original experiment must be checked before treating the title's claim as established or generalising it to other electromagnetic exposures.

Retrieval lead | Source checks to 20 October 2026

Heinrich A, Szostek A, Meyer P, Reinhard I, Gilles M, Paslakis G, Rauschenberg J, Gröbner J, Semmler W, Deuschle M, Meyer-Lindenberg A, Flor H, Nees F. Women are more strongly affected by dizziness in static magnetic fields of magnetic resonance imaging scanners. NeuroReport. 2014;25(14):1081-1084. doi:10.1097/wnr.0000000000000225.

Exposure. Tesla-scale MRI static magnetic fields and motion through gradients.

Design and controls. Human experiment.

Reported finding. Affirmative vestibular or neurobehavioural finding described in discovery dossier.

Interpretive limits. Retained in a separate regime-specific catalogue. No inference to Wi-Fi or mobile-phone exposures.

The protection question raised. Is any sex difference in MRI-associated dizziness reproducible and relevant to susceptibility? Original results remain unverified, so the title alone cannot establish the effect or justify wider extrapolation.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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SF-004 Slower MRI entry and vestibular responses

> **Why this matters**
> This retrieval lead asks a practical question: whether entering a strong MRI magnet more slowly reduces dizziness and involuntary eye movements. Those eye movements are called nystagmus. Full methods and results need review before any clinical recommendation, but the proposed link between movement and symptoms is directly testable.

Retrieval lead | Source checks to 20 October 2026

Pogson JM, Shemesh A, Roberts DC, Zee DS, Otero-Milan J, Ward BK. Longer duration entry mitigates nystagmus and vertigo in 7-Tesla MRI. Frontiers in Neurology. 2023;14:1255105. doi:10.3389/fneur.2023.1255105.

Exposure. Tesla-scale MRI static magnetic fields and motion through gradients.

Design and controls. Human experiment.

Reported finding. Affirmative vestibular or neurobehavioural finding described in discovery dossier.

Interpretive limits. Retained in a separate regime-specific catalogue. No inference to Wi-Fi or mobile-phone exposures.

The protection question raised. Could entry speed influence dizziness and involuntary eye movements near a strong MRI magnet? The lead raises a practical question, but its unreviewed original results do not yet support a clinical recommendation.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

Retrieve. DOI or primary source | PubMed record

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ECO-001 Sparrow abundance and measured base station fields

> **Why this matters**
> This retrieval lead concerns whether house-sparrow abundance varies with measured base-station fields. It brings free-living wildlife into the assessment, where habitat and other local conditions can also shape results. The original design and analyses need checking before an observed association can support a causal ecological claim.

Retrieval lead | Source checks to 20 October 2026

Everaert J, Bauwens D. A Possible Effect of Electromagnetic Radiation from Mobile Phone Base Stations on the Number of Breeding House Sparrows (Passer domesticus). Electromagnetic Biology and Medicine. 2007;26(1):63-72. doi:10.1080/15368370701205693.

Exposure. See original source for calibrated field and modulation conditions.

Design and controls. Observational or experimental ecology; original methods pending.

Reported finding. Discovery dossier reports negative association between house-sparrow abundance and measured GSM fields.

Interpretive limits. A retrieval entry, not a verified ecological causal conclusion.

The protection question raised. Does the sparrow-abundance association persist after accounting for habitat and other local conditions? Until original methods and analyses are checked, this is a field-observation lead rather than an ecological causal finding.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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ECO-002 Tree damage and spatial exposure observations

Why this matters

Tree-crown damage is a visible ecological endpoint, and this retrieval lead describes its spatial relationship with measured RF fields. The observation deserves examination alongside other causes of tree damage. Original methods and comparison sites remain unreviewed here, so the paper's causal title is not adopted as a verified conclusion.

Retrieval lead | Source checks to 20 October 2026

Waldmann-Selsam C, Balmori-de la Puente A, Breunig H, Balmori A. Radiofrequency radiation injures trees around mobile phone base stations. Science of The Total Environment. 2016;572:554-569. doi:10.1016/j.scitotenv.2016.08.045.

Exposure. See original source for calibrated field and modulation conditions.

Design and controls. Observational or experimental ecology; original methods pending.

Reported finding. Discovery dossier describes spatial correspondence between crown damage and measured RF exposure.

Interpretive limits. A retrieval entry, not a verified ecological causal conclusion.

The protection question raised. Does the spatial observation distinguish RF from other causes of tree damage? Original comparison sites and methods remain unreviewed; the paper's causal title cannot substitute for verified evidence.

Source verification. Record verified; full text still required

Editorial status. Primary PubMed linked-notice record checked 20 Oct 2026; no correction, concern or retraction linked at check. This is not an exhaustive publisher-history certification.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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Annex E Editorial notices requiring caution

Use these papers with the identified notices. They remain discoverable for assessment, but do not carry the report's central adverse-evidence argument. An expression of concern is distinguished from a retraction; no conduct allegation is inferred.

Records in this section: RF-016, ELF-004

RF-016 Different genotoxic responses across human cell types

> **Why this matters**
> This paper carries an editorial Expression of Concern. Its reported cell-DNA findings therefore cannot serve as a firm foundation for a health claim here. The record keeps the result and warning together so readers can inspect the publication history; the notice's full explanation was not obtained.

Editorial notice flagged | Source checks to 20 October 2026

Schwarz C, Kratochvil E, Pilger A, Kuster N, Adlkofer F, Rüdiger HW. Radiofrequency electromagnetic fields (UMTS, 1,950 MHz) induce genotoxic effects in vitro in human fibroblasts but not in lymphocytes. International Archives of Occupational and Environmental Health. 2008;81(6):755-767. doi:10.1007/s00420-008-0305-5.

Exposure. 1950 MHz UMTS; detailed low-SAR values in discovery dossier not yet rechecked.

Design and controls. In-vitro human cells.

Reported finding. Publisher abstract reports comet/micronucleus increases in human fibroblasts, but no lymphocyte effect. Quantitative dose claims are not used in the report pending a full methods and editorial-history audit.

Interpretive limits. Publisher explicitly links an Expression of concern. Do not use as central evidence of genotoxicity, human injury or below-limit effects; no allegation about individual conduct is inferred.

The protection question raised. The unresolved editorial concern limits the confidence this cell-DNA result can carry. Its publication history remains available for scrutiny, but it cannot underpin the central health-protection argument.

Source verification. Original publisher abstract and linked editorial-notice metadata checked; full original and notice body not obtained

Source locations. Publisher article page linked Editorial and abstract; notice page title, date and original-article link

Editorial status. Drexler H, Schaller KH. Expression of concern. Int Arch Occup Environ Health. 2009;82:143-144; published online 11 Oct 2008. DOI 10.1007/s00420-008-0360-y. Publisher links notice to this paper; Scite did not surface it.

Funding. Publisher acknowledgements: Austrian Workers Compensation Board, Verum Foundation and Austrian Science Fund FWF P18984-B09.

Conflicts. Not established from retrieved material.

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ELF-004 Later miscarriage cohort with editorial notice

> **Why this matters**
> The reported miscarriage association concerns power-frequency magnetic fields, not wireless RF. An editorial Expression of Concern states that participant consent did not allow data sharing, limiting independent reanalysis. This is not a retraction, but the restriction affects how much weight the finding can carry.

Editorial notice flagged | Source checks to 20 October 2026

Li DK, Chen H, Ferber JR, Odouli R, Quesenberry C. Exposure to Magnetic Field Non-Ionizing Radiation and the Risk of Miscarriage: A Prospective Cohort Study. Scientific Reports. 2017;7(1):17541. doi:10.1038/s41598-017-16623-8.

Exposure. 24-h EMDEX Lite power-frequency magnetic-field monitoring; 99th-percentile exposure classification. The meter does not establish RF wireless exposure.

Design and controls. 913 pregnancies reported in discovery dossier.

Reported finding. Prospective miscarriage association reported in 913 pregnancies; retained with the linked Expression of Concern, without a featured numerical risk claim.

Interpretive limits. Underlying data cannot be shared because retrospective participant consent is unavailable, per notice. This is an Expression of Concern, not a retraction. No RF causal inference; not used centrally.

The protection question raised. How much weight can the miscarriage association carry without independent reanalysis of the data? The consent-related Expression of Concern remains material, and this power-frequency observation is not an RF causal finding.

Source verification. Original indexed methods and official 2021 notice reviewed

Source locations. Original Materials and Methods exposure monitoring; official notice full text

Editorial status. Editorial Expression of Concern, 10 March 2021, DOI 10.1038/s41598-021-85479-w. Notice says data unavailable for readers wishing to repeat analyses because consent for sharing was not obtained.

Funding. Not established from retrieved material.

Conflicts. Not established from retrieved material.

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How the evidence was assembled

This is an updated selective review of adverse findings and biological responses potentially relevant to harm. It incorporates earlier discovery dossiers and evidence records, followed by targeted source verification and broader discovery. It applies no publication-date cutoff or ceiling inherited from the original collection. Scite discovery and citation tracing were combined with publisher records, government reports, PubMed, institutional repositories and accessible original manuscripts. This editorial edition carries forward that verified research; it adds no new literature search.

Original methods, results and identified tables or figures were examined wherever obtainable. Abstract fallbacks are labelled. The annex contains 38 original-text records, 19 original-abstract records, six replication/shared-cohort context records, 14 retrieval leads and two notice-flagged papers. These are publication records, not independent experiment counts. Original-text review can include a manuscript or proof and does not certify every supplement or underlying dataset.

Linked PubMed notices were checked across retrieved records; important publisher notices were examined separately. Corrections, comments, expressions of concern and retractions remain distinct. Shared tissues, participant cohorts and preprint/journal versions are identified. Documented funding and conflicts are retained; unknown disclosures remain unknown, and funding alone is not evidence of misconduct or a finding's validity.

Reading priority reflects endpoint directness, controls, exposure characterisation, statistical support and independent corroboration, without a numerical quality score. Nonconfirming records supply necessary context for included positive findings, rather than a systematic sample of reassuring literature. Retrieval leads and notice-flagged papers do not underpin central claims.

Repeated searches around the selected tumour, reproductive, neurological and ecological programmes increasingly returned the same experiments, derivative reports and reviews. Other programmes supplied leads still needing original methods. This marks diminishing returns in those search lanes, not exhaustion of the literature. Extensive working notes and retrieval logs remain outside the public narrative. Source checks were carried forward to 20 October 2026; the record gives the source form and location.

Regulatory framework sources used in the protection chapter:

FCC exposure rule 47 CFR 1.1310

ICNIRP 2020 guidelines

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Names are indexed from complete author metadata where available; group authors remain group authors. Multiple records under a name do not establish independent cohorts.

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