



עמותת רגישות לקרינה ישראל

ע.ר. 580630150

רשימת מקורות

מאמרים, מחקרים וסקירות מדעיות על השפעות בריאותיות של חשיפה לקרינה בלתי מייננת

מקורות מדעיים אלה מראים כי חשיפות EMF לא-תרמיות מייצרות את ההשפעות המזיקות לבריאות.

הנחיות ICNIRP, FCC והבטיחות הבינלאומיות, המבוססות אך ורק על השפעות תרמיות, אינן מדעיות ואינן מספקות, כך שלא ניתן לסמוך עליהן כדי להגן על בריאות הציבור.

היו עתירות והצהרות אחרות של קבוצות מדענים בינלאומיות המביעות דאגה רבה ממצב זה.

א. השפעות ניורולוגיות / ניורופסיכיאטריות:

1. Marha K. 1966 Biological Effects of High-Frequency Electromagnetic Fields (Translation). ATD Report 66-92. July 13, 1966 (ATD Work Assignment No. 78, Task 11)
<https://apps.dtic.mil/dtic/tr/fulltext/u2/642029.pdf>
2. Glaser ZR, PhD. 1971 Naval Medical Research Institute Research Report, June 1971. Bibliography of Reported Biological Phenomena ("Effects") and Clinical Manifestations Attributed to Microwave and Radio-Frequency Radiation. Report No. 2 Revised.
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19. Hecht, Karl. 2016 Health Implications of Long-Term Exposures to Electrosmog. Brochure 6 of A Brochure Series of the Competence Initiative for the Protection of Humanity, the Environment and Democracy.
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ב. המערכת האנדוקרינית והשפעות הרמוניות

1. Glaser ZR, PhD. 1971 Naval Medical Research Institute Research Report, June 1971. Bibliography of Reported Biological Phenomena ("Effects") and Clinical Manifestations Attributed to Microwave and Radio-Frequency Radiation. Report No. 2 Revised. <https://apps.dtic.mil/docs/citations/AD0750271>
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ג. לחץ חמצוני / נזק של רדיקלים חופשיים (מנגנונים המעורבים כמעט בכל המחלות הכרוניות); גורם ישיר לנזק ב-DNA הסלולרי):

1. Raines, J. K. 1981. Electromagnetic Field Interactions with the Human Body: Observed Effects and Theories. Greenbelt, Maryland: National Aeronautics and Space Administration 1981; 116 p.
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ד. פגיעה ונזק ל DNA הסולרי

שברי גדיל בודד ושכפול כפול ב- DNA התא והבסיסים המחומצנים ב- DNA התא, מה שמוביל לשינויים כרומוזומליים ואחרים:

1. Glaser ZR, PhD. 1971 Naval Medical Research Institute Research Report, June 1971. Bibliography of Reported Biological Phenomena ("Effects") and Clinical Manifestations Attributed to Microwave and Radio-Frequency Radiation. Report No. 2 Revised. <https://apps.dtic.mil/docs/citations/AD0750271>
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ה. אפופטוזיס / מוות תאים (תהליך מרכזי בהיווצרות מחלות ניווניות, והיווצרות תגובות אי-פוריות):

1. Glaser ZR, PhD. 1971 Naval Medical Research Institute Research Report, June 1971. Bibliography of Reported Biological Phenomena ("Effects") and Clinical Manifestations Attributed to Microwave and Radio-Frequency Radiation. Report No. 2 Revised. <https://apps.dtic.mil/docs/citations/AD0750271>
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א. פגיעה בפוריות אצל נשים וגברים

פוריות ירודה, כולל שינויים מחדש ברקמות באשך, הורדת כמות הזרע ואיכות הזרע, פגיעה בפוריות הנשית לרבות בשחלות, אובדן ביציות (זקי), ירידה ברמת האסטרוגן, פרוגסטרוגן וטסטוסטרון (כלומר רמות הורמוני המין), שהעלתה את שכיחות ההפלה הספונטנית, ירידה בלבדו:

1. Glaser ZR, PhD. 1971 Naval Medical Research Institute Research Report, June 1971. Bibliography of Reported Biological Phenomena ("Effects") and Clinical Manifestations Attributed to Microwave and Radio-Frequency Radiation. Report No. 2 Revised. <https://apps.dtic.mil/docs/citations/AD0750271>
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12. Adams JA, Galloway TS, Mondal D, Esteves SC, Mathews F. 2014 Effect of mobile telephones on sperm quality: a systematic review and meta-analysis. *Environ Int* 70:106-112.
13. Liu K, Li Y, Zhang G, Liu J, Cao J, Ao L, Zhang S. 2014 Association between mobile phone use and semen quality: a systematic review and meta-analysis. *Andrology* 2:491-501.
14. K Sri N. 2015 Mobile phone radiation: physiological & pathophysiological considerations. *Indian J Physiol Pharmacol* 59:125-135.
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ז. סידן תאי מוגבר:

סידן תוך תאי נשמר ברמות נמוכות מאוד, פרט לעליות קצרות המשמשות לייצור תגובות רגולטוריות, כך שהעלייה מתמשכת של רמות סידן תוך תאי מייצרת פתופיזיולוגיות רבות.

1. Adey WR. 1988 Cell membranes: the electromagnetic environment and cancer promotion. *Neurochem Res*.13:671-677.
2. Walleczek, J. 1992. Electromagnetic field effects on cells of the immune system: the role of calcium signaling. *FASEB J*. 6, 3177-3185.
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ה. סרטון:

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2. Marino AA, Morris DH. 1985 Chronic electromagnetic stressors in the environment. A risk factor in human cancer. *J environ sci health C3*:189-219.
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פעימות או פולסים של EMFs (Pulsed EMFs) הם 'יותר' פעילות ביולוגית.

זה חשוב להבנה כל המכשירים האלחוטיים מתקשרים באמצעות פולסים כאלה וככל שהם יותר 'חכמים' ומעבירים ביניהם מידע באופן זה, כך ההשפעה על התפקודים הביולוגיים חריפה יותר.

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מחקרים רבים מראים כי חשיפות ל-EMFs מייצרות השפעות לא תרמיות מגוונות באמצעות הפעלת מנגנון VGCC בתאים של בני אדם ובעלי חיים ואף בתאים צמחיים בהם מעורבים מיוצרות על ידי השפעות במורד הזרם של הפעלת VGCC, באמצעות סידן תוך-תאי מוגבר $[Ca^{2+}]_i$.

המנגנונים שבאמצעותם ניתן ליצור אפקטים שונים על ידי הפעלת VGCC מתוארים באיור להלן:

EMF effect	Probable mechanism(s)
Oxidative stress	Produced by elevated levels of peroxynitrite and the free radical breakdown products of peroxynitrite and its CO_2 adduct. Four studies of EMF exposure, cited in [4] showed that oxidative stress following exposure was associated with major elevation of 3-nitrotyrosine, a marker of peroxynitrite, thus confirming this interpretation. Two other studies each found 3-nitrotyrosine elevation, both following 35 GHz exposures [19,20].
Lowered male/female fertility, elevated spontaneous abortion, lowered libido	Both the lowered male fertility and lowered female fertility are associated with and presumably caused by the oxidative stress in the male and female reproductive organs. Spontaneous abortion is often caused by chromosomal mutations, so the germ line mutations may have a causal role. Lowered libido may be caused by lowered estrogen, progesterone and testosterone levels. It seems likely that these explanations may be oversimplified. One additional mechanism that may be important in producing lowered fertility is that VGCC activation and consequent high $[Ca^{2+}]_i$ levels is known to have a key role in avoiding polyspermy. Consequently, if this response is triggered before any fertilization of an egg has occurred, it may prevent any sperm from fertilizing and egg.
Neurological/neuropsychiatric effects	Of all cells in the body, the neurons have the highest densities of VGCCs, due in part to the VGCC role and $[Ca^{2+}]_i$ role in the release of every neurotransmitter in the nervous system. Calcium signaling regulates synaptic structure and function in 5 different ways, each likely to be involved here. Oxidative stress and apoptosis are both thought to have important roles. Lowered sleep and increased fatigue are likely to involve lowered nocturnal melatonin and increased nocturnal norepinephrine.
Apoptosis	Apoptosis can be produced by excessive Ca^{2+} levels in the mitochondria and by double strand breaks in cellular DNA; it seems likely that both of these mechanisms are involved following EMF exposure. A third mechanism for triggering apoptosis, endoplasmic reticulum stress (see bottom row in this Table), may also be involved.
Cellular DNA damage	Cellular DNA damage is produced by the free radical breakdown products of peroxynitrite directly attacking the DNA [7].
Changes in non-steroid hormone levels	The release of non-steroid hormones is produced by VGCC activation and $[Ca^{2+}]_i$ elevation. The immediate effects of EMF exposures is to increase hormone release and to raise, therefore, hormone levels. However many hormone systems become "exhausted" as a consequence of chronic EMF exposures. The mechanism of exhaustion is still uncertain, but it may involve oxidative stress and inflammation.
Lowered steroid hormone	Steroid hormones are synthesized through the action of cytochrome P450 enzymes; activity of these hormones is inhibited by binding of high levels of nitric oxide (NO) leading to lowered hormone synthesis.

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EMF effect	Probable mechanism(s)
Calcium overload	Produced by excessive activity of the VGCCs; secondary calcium overload is produced by oxidative stress activation of TRPV1, TRPM2 and possibly some other TRP receptors, opening the calcium channel of these receptors.
Heat shock protein induction	There is a large literature showing that excessive $[Ca^{2+}]_i$ induces very large increases in heat shock proteins. This is thought to be produced by complex calcium signaling changes involving the endoplasmic reticulum, mitochondria and the cytosol and also involving excessive $[Ca^{2+}]_i$ producing increasing protein misfolding [21-23]. It should be noted that some calcium is essential for proper protein folding in the endoplasmic reticulum such that only excessive calcium leads to misfolding and consequent endoplasmic reticulum stress.

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