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ÉCOSCOPE 2/20 – Tableau 1 relatif à l'article du Dr Stefan Zbornik
www.aefu.ch/ecoscope/zbornik_tableau1.pdf

Tableau 1: Compilation de paramètres choisis des signaux radio mobile de la 5G pouvant occasionner des effets thermiques et non thermiques sur l'organisme.

1. Fréquences porteuses

Toutes les bandes de fréquences requises jusqu'à maintenant pour la téléphonie mobile situées entre 700 MHz et 2600 MHz peuvent, en principe, être également utilisées pour la «5G». Les bandes de fréquence récemment mises aux enchères, situées entre 3500 et 3800 MHz, sont aussi employées pour la «5G». En outre, le Conseil fédéral a réservé les bandes ISM 24 GHz, 61 GHz, 122 GHz et 245 GHz pour la téléphonie mobile [1]. Ces fréquences élevées sont qualifiées d'ondes millimétriques Il est bien connu qu'en présence de fréquences plus élevées, la profondeur de pénétration du rayonnement radio diminue dans les tissus et qu'en présence d'ondes millimétriques presque tout le rayonnement est absorbé par les couches supérieures de la peau et par les yeux, ce qui, en soi, est déjà problématique. Cela peut être potentiellement le cas pour des ondes continues non modulées mais non pour les signaux pulsés pilotés en phase tels qu'ils sont générés pour la «5G» [2]. Le rayonnement des couches supérieures de la peau peut également causer des effets dans des organes internes. La recherche médico-biologique civile sur les effets des ondes millimétriques de la «5G» sur l'homme, les animaux et les plantes est, jusqu'ici, quasiment inexistante et c'est pourquoi, il serait imprudent d'affirmer que le rayonnement à couverture globale dû à la «5G» n'est pas sans risque.

2. Fréquences de modulation et fréquences à modulation par impulsions

Pour que les signaux de télécommunication soient transmis aux fréquences porteuses, ceux-ci sont modulés avec certaines fréquences [3]. Dans le cas de la «5G», des procédés de modulation numériques complexes sont utilisés dans ce but. Des blocs de données sont alors rassemblés en paquets et transmis à des moments définis. Si cela a lieu de manière continue, on parle de fréquences d'impulsions ou de fréquences à modulation par impulsions. En présence de configuration correspondante, les mâts d'antennes «5G» émettent sans interruption un bloc de synchronisation pour signaler la disponibilité à la réception à tous les appareils mobiles se trouvant dans la zone de couverture. Des mesures actuelles de champs montrent qu'un tel signal est envoyé toutes les 20 millisecondes, ce qui donne une fréquence d'impulsions de 50 Hz qui, jusqu'à maintenant, n'existait pas pour la téléphonie mobile. La régulation dynamique de la puissance et, notamment, la formation de faisceaux (beamforming) adaptative rapide occasionnent une impulsion supplémentaire des signaux radio au niveau du récepteur. Il existe des études, fruit de nombreuses années de recherche, qui prouvent que les fréquences comparativement basses des signaux pulsés et modulés par impulsions peuvent induire des effets autant positifs que négatifs sur les organismes biologiques [4, 5, 6]. La fréquence d'impulsion en question, l'intensité de champ générée et le modèle d'exposition en question semblent être particulièrement critiques. Ainsi, par exemple, l'impulsion citée de 50 Hz de la «5G» se situe dans la plage des ondes électromagnétiques gamma du cerveau humain et pourrait altérer le fonctionnement de ce dernier. Jusqu'à maintenant, il y a eu, par comparaison, très peu de recherches et de publications sur les effets des différentes fréquences à modulation par impulsions sur les organismes biologiques. Cela est vrai, notamment, pour la «5G». C'est pourquoi des effets nocifs ne peuvent pas être exclus.

3. Beamforming adaptatif (formation de faisceaux adaptative)

Lors du beamforming, les ondes émises sont fortement groupées en faisceaux au moyen de la technique du régulateur de phase [7]. Les faisceaux ainsi générés peuvent, combinés à la technique MIMO massif des mâts d'antennes, être orientés et concentrés horizontalement et verticalement sur les appareils mobiles et leur environnement et, ainsi, transmettre plus de données avec une puissance surhaussée. Cela a lieu de manière adaptative et très vite, toutes les millisecondes. C'est pourquoi, on peut parler d'une quasi-impulsion. Ainsi, toutes les personnes se trouvant dans un secteur à faisceaux sont également exposées toutes les millisecondes à un rayonnement en faisceaux focalisé. Jusqu'ici, presque rien n'a été publié par la recherche civile sur les effets du beamforming de la «5G» et la technique du MIMO massif sur les organismes biologiques. Des rapports de personnes qui se sont trouvées, par hasard, dans des installations de tests de faisceaux de la «5G» le long de la voie ferrée sur les bords du lac de Walen, ont indiqué des risques sanitaires. C'est pourquoi on ne peut pas exclure des effets pathogènes.

4. Largeur de bande de signal et formes de signal

Pour la «5G», la largeur de bande de signal de 100 MHz à 2000 MHz peut être configurée pour pouvoir atteindre des taux variables de transmission des données. De ce fait, cette largeur de bande peut être largement plus importante que celle de la téléphonie mobile habituelle. Des travaux de recherche prouvent que, d'un point de vue médico-biologique, les grandes largeurs de bande peuvent être plus problématiques que les signaux à bande étroite [5]. Les formes de signaux pour la «5G» résultent d'autres paramètres les plus divers comme le niveau de modulation, le facteur de crête, la relation problématique de phase, la pente du signal, les techniques de modulations/multiplex, l'état de polarisation etc. Presque aucun de ces paramètres n'a été pris en compte dans le cadre de la maigre recherche sur les risques de la «5G». Il en est de même pour les études sur les autres générations. C'est pourquoi des effets nocifs différenciés sur les organismes ne peuvent pas être exclus.

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ÉCOSCOPE 2/20 – Tableau 2 relatif à l'article du Dr Stefan Zbornik
www.aefu.ch/ecoscope/zbornik_tableau2

Tableau 2: Paramètres typiques d'exposition et objets exposés qui doivent être pris en compte lors de l'évaluation des effets thermiques et non thermiques sur des organismes.

Paramètres d'exposition typiques des champs électromagnétiques (CEM)

1. Intensité de champ électrique (V/m)

2. Densité surfacique de puissance électrique (W/m^2)

3. Intensité de champ magnétique (A/m)

4. Induction magnétique (T)

5. Le débit d'absorption spécifique (DAS) qui en résulte

   Pour toutes les mesures de grandeurs physiques, il faut saisir des valeurs de crête et des valeurs moyennes pour permettre une évaluation transparente des résultats. En outre, en plus des mesures instantanées, il faut recenser la variation dans le temps, à long terme.

6. Exposition au champ lointain ou au champ proche

7. Des signaux de téléphonie mobile originaux et non des signaux simulés provenant de générateurs

8. Durée d'exposition (brève et longue)

9. Modèle d'exposition temporel (continu ou intermittent)

10. Interférences avec

    - d'autres CEM à haute fréquence (par ex. d'appareils de laboratoire, de téléphones portables, du WLAN),

    - des CEM à basse fréquence (par ex. de lignes électriques, de transformateurs, de moteurs),

    - des champs électriques statiques (par ex. de revêtements en matériaux synthétiques),

    - des champs magnétiques statiques (par ex. du champ magnétique terrestre)

11. Résonances

12. Autres facteurs environnementaux/facteurs de stress (par ex. lumière, bruit, CO_2)

13. entre autres.

Objets typiques exposés lors d'études sur les effets de la CEM

- cellules (procaryotes, eucaryotes)

- virus (ADN, ARN)

- plantes (arbres, plantes vivaces, plantes grimpantes, fleurs, légumes ...)

- animaux (mammifères, insectes, oiseaux, reptiles, également génétiquement modifiés)

- homme (âge, sexe, poids, appartenance ethnique, …)

ÉCOSCOPE 2/20 – références relatives à l'article du Dr Stefan Zbornik
www.aefu.ch/ecoscope/zbornik_références.pdf

Téléphonie mobile : La nouvelle norme passée au crible

Stefan Zbornik, Kreuzlingen/TG

Depuis environ trois ans, la 5G est sur toutes les lèvres. Peu de gens sont au fait des techniques et des risques que recèle cette nouvelle norme. Les opérateurs l'associent à de belles promesses et des émotions.

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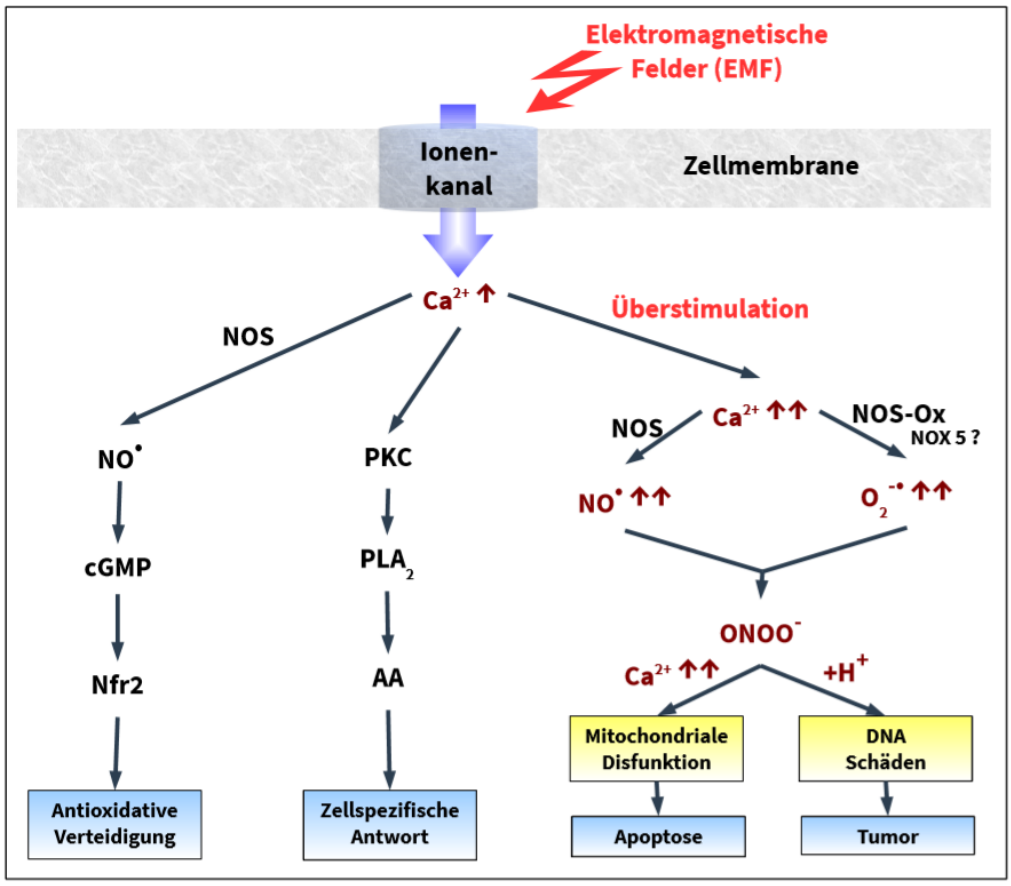
Alle aufgeführten Homepages wurden letztmals am 23. Mai 2020 abgerufen.

ÉCOSCOPE 2/20 – Graphique relative à l'article du Prof. Dr Volker Ullrich
www.aefu.ch/ecoscope/ullrich_graphique.pdf

Grafique : Nouvelle voie de l'afflux de calcium dans la cellule via le canal ionique dépendant du voltage (CADV).



Des signaux de calcium

Électo-hypersensibilité: nouvelles explications neurobiochimiques

Volker Ullrich,
Université de Constance (D)

Les symptômes dus au rayonnement de téléphonie mobile sont souvent niés. Un mécanisme biochimique de l'afflux de calcium dans les cellules fournit des explications, aussi pour l'électrosensibilité.

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Nachstehend englisches Original dieses Artikels

Relevance of bioassays

Animal studies: prediction of carcinogenicity in humans

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Substances demonstrated to be carcinogenic in animal studies have always subsequently appeared to be carcinogenic for humans too. It becomes apparent that this is also true for mobile phone radiation.

There are three major factors at the origin of the increased incidence, and therefore increased mortality, of cancer over the last 50-60 years: 1) the increase in life expectancy (about 10 years for males and 15 years for females); 2) the increase in the diffusion of agents and situations presenting carcinogenic risks in both the occupational and general environment; and 3) last but not least, it is also important in the carcinogenetic process genetic predisposition; however, it is unlikely that this factor has significantly changed during the last decades.

As a whole, the process of carcinogenesis may be expressed, albeit simplistically, by the following formula: C = f (P+E+A) , where cancer is a function (f) of predisposition (P), exposure (E) and age (A), the last of which when increasing, allows an environmental carcinogen to more fully express its effects, in particular those which are attributable to the sum/synergy of low and extremely low dose exposure to carcinogenic agents. Of the three factors which condition the carcinogenetic process, it is difficult to imagine changing predisposition (P) by modifying the genetic profile, even more so regarding age (A).

The possibility to modify the trend in cancer incidence and mortality therefore depends on the capacity to identify agents and situations of carcinogenic risk in the environment related to occupation and/or lifestyle (E). Once identified, it follows that exposure to these risks should then be eliminated or at least reduced as much as possible. If it is true that the carcinogenetic process depends in large part on the role of genetic predisposition, environment and age, and that in order to modify the present epidemiological dimension of cancer and other degenerative diseases the most compatible solution is intervening to reduce exposure to agents and situations of

environmental cancer risk, it is also true that our knowledge of these risks is still unfortunately extremely limited. The identification of carcinogenic agents may be pursued today using three types of research, all of which offer various possibilities and advantages, but each of which are limited in different ways.

1. Short and medium term studies

Short and medium term studies include research on toxic effects (acute, subacute and subcronic), on mutagenic effects (in bacterial and animal cells) and on bio-molecular mechanisms which may be at the origin of these effects. These types of research may be completed in a relatively short time frame, but with regard to carcinogenicity, these studies provide only indirect data. Since these data are not based on end points, they cannot reveal a specific final event such as the presence or absence of a tumour. The utility of these studies therefore has a precise limit, even if in some cases they can provide, in addition to an initial orientation, useful data about the mechanistic aspects of the action of agents on biological systems (related or unrelated to carcinogenesis).

2. Long term carcinogenesis bioassays on experimental animals

When well planned and conducted using adequate animal models (as close as possible to the human equivalent) and reproducing as much as possible human exposure scenarios, long term carcinogenesis bioassays may provide specific indications on carcinogenicity which may then be extrapolated to the human in both qualitative and quantitative terms.

Much has been done by interested parties to discredit these bioassays and associated scientists. However, there are three important considerations which support their use: 1) all agents demonstrated via epidemiological studies to be carcinogenic in humans also resulted carcinogenic in experimental animals. The carcinogenicity of one third of the agents recognized to be carcinogenic by the International Agency for Research of Cancer (IARC) was first demonstrated in rodent studies and only subsequently in humans; 2) no agent demonstrated to be carcinogenic in animals has been found to be non-carcinogenic in humans when adequate epidemiological studies were performed; 3) the type of relationship between exposure to a carcinogenic agent and the neoplastic response and evolution of the carcinogenesis process are comparable in animals and humans [1].

3. Oncological epidemiological surveys

Oncological epidemiological surveys, when conducted with adequate information on the exposure scenarios and correct methodology, produce clear results and represent the most direct proof of the carcinogenicity of an agent, factor or situation. Often however, these surveys have precise limitations: 1) their feasibility, in that the dimension of the exposed or follow-up populations may be insufficient and/or in that the data related to the level of exposure may be limited due to the difficulty of retracing the universe of exposed persons or of a representative sample; 2) the capacity to produce clear results due to confounding factors or, in the case of low or medium-low exposure, the difficulty of finding a large enough sample of exposed persons and adequate controls; methodological inadequacies may also impede clear results; 3) the inherent delay in epidemiological results due to the long period of tumour latency in humans (on average 10-30 years), during which time agents may continue to produce their grave effects; 4) the widespread exposure (diffuse exposure) to carcinogenic agents, and as a consequence the difficulties related to find a large control group, not exposed at all.

Bioassays are quick and reliable

Since the period of latency is proportional to the average lifespan of an organism, latency is proportionally shorter in rodents commonly used in the laboratories for long term bioassays. An example is that of Sprague Dawley rats used in our laboratory, the laboratory of the Cesare Maltoni

Cancer Research Centre of the Ramazzini Institute. Our strain of rat has a lifetime of 160 weeks which corresponds to 95-100 human years. A latency time of one year is therefore equivalent to slightly more than 30 years of latency in humans. In other words, long term bioassays allow for a very rapid surrogate response, avoiding the frequent exposure of humans to unknown agents posing carcinogenic risk. In conclusion, it may be affirmed that carcinogenesis bioassays, if adequately conducted, are relatively rapid and highly predictive of effects on humans (it is also true that studies of this type which are not correctly planned and conducted represent a factor of confusion). Epidemiological surveys could be those which technically provide the most direct (albeit delayed), information, but are often impeded by obstacles to their feasibility and frequently produce non conclusive results due to methodological and non- methodological inadequacies.

The International Agency for Research on Cancer evaluated more than 1000 agents for carcinogenicity in humans, using animal data together with epidemiological studies and mechanistic studies, and provide the specific classification (https://monographs.iarc.fr/monographs-available/#24). The Preamble to the IARC Monographs describes the objective and scope of the programme, the scientific principles and procedures used in developing a Monograph, the types of evidence considered, and the scientific criteria that guide the evaluations(https://monographs.iarc.fr/iarc-monographs-preamble-preamble-to-the-iarc-monographs/), with the following classification.

Group 1	Carcinogenic to humans	120 agents
Group 2A	Probably carcinogenic to humans	83 agents
Group 2B	Possibly carcinogenic to humans	314 agents
Group 3	Not classifiable as to its carcinogenicity to humans	500 agents

Agents Classified by the IARC Monographs, Volumes 1–125.

Proof of transferability on human beings

From IARC criteria of evaluation it is clear the important role of experimental bioassays for the identification of hazards for humans. One fact remains abundantly clear: for every known human carcinogen that has been tested adequately in laboratory animals, the findings of carcinogenicity are concordant [1, 2, 3]. In the following table I would like to give to the readers some example of the magnitude of prediction for the human counterpart regarding only few of the compounds, out of the more than 200 we studied performed at the Cesare Maltoni Cancer Research Centre of the Ramazzini Institute (CMCRC-RI) in more than 40 years of research. Our year of publication of data is compared to the year of publication of the IARC Monograph as Group 1, carcinogenic to humans and multipotential carcinogen.

Agent	**CMCRC-RI* cancerogenicity pubblication**	**IARC Group 1** [< 1](sufficient in humans, or mechanistic upgrade*), vol, yr	Time to Group 1 (yrs)/multipote-ntial carcinogen	IARC Group 1 and **additional** tumour sites in humans/ Volume, yr
Vinyl Chloride	1974	7/1974 liver angiosarcoma	0/**38**	100F, 2012,S Hepatocellular Cancer
Chromium VI compounds	1974	2/1972 only lung	0/**38**	100C, 2012 nasal cavity & paranasal sinuses
Benzene	1979	S1, 1979 AML	0/**39**	120, 2018 L several LH tumours
Erionite	1982	42/1986 meso	4/**26**	100c, 2012, none
Trichloro-ethylene	1986	106/2012 kidney	26/**28**	106, 2014, L NHL, liver
Silica dust	1988	68/1996 Lung	8/**24**	100c, 2012, none
Asbestos (all forms)	1989	2/1973 Lung, meso	0/**22**	100C, 2012, S larynx, ovary L colorectum, pharynx, stomach
Formaldehyde	1989	88/2006 NPC	17/**23**	100F 2012, S leukemia, L sinonasal
Tamoxifen	1997 Chemopreven-tion breast cancer	66/1996	0/**15**	100A, 2012(reduced risk contralateral breast cancer)
Diesel (engine exhaust)	1997	105/2012 lung	15/**15**	105, 2012 L bladder
Ethanol (in alcoholic beverages)	2002	96/2007	5/**10**	100E, 2012, none
Acetaldehyde (in alcoholic beverages)	2002	100E/2009	7/**10**	100E, 2012
Fluoroedenite	2004	111/2014 mesothelioma	10/**10**	111

Note: S = Supplement; # some variation of AML vs ANLL; several additional sites with limited evidence (100F, 120)
* The CMCRC evaluations, apart fibres like asbestos, silica and fluoroedenite, that were regarding mesothelioma, were of multiple site carcinogens.

Long term studies on RFR urgently required

Long term carcinogenesis experiments are fundamental for the identification of carcinogenic agents, both for evaluating products which are already on the market and for studying those which have yet to be commercialized. The reliability of these studies is such that their use is constantly growing, especially for the evaluation of the efficacy and tolerability of pharmaceuticals/active principles, including those which could potentially be used for the chemoprevention of tumours. Also of great importance is their use for the identification and validation of biological markers and medical devices.

In this perspective, it appears not understandable the fact that, on the contrary of what happens in Europe for all chemicals and pharmaceutical compounds before their commercialization, long term studies to exclude the adverse effect of devices for telecommunication, like mobile phones and radio-base stations antennas emitting different frequencies, are not requested to the Companies by

worldwide regulators. Early warnings on the potential carcinogenic risks of mobile phone radiofrequency radiation (RFR) raised in the early 2000 when, for the first time, it was published that people using mobile phones had a significant increased risk to develop vestibular Schwannoma and brain tumours.

In 2011, the International Agency for Research on Cancer (IARC) classified RFR as possible human carcinogen (Group 2B) based on limited evidence both in humans and experimental animals [5].

Preliminary results show causal link

In 2018 the Ramazzini Institute published the first important findings of the RI experimental study [6]. The communication of preliminary results was urged by different factors: 1) the fact that also a small increase of the incidence of tumours induced by the exposure to RFR could have great impact for public health because the exposed people are billions; 2) The RI findings on far field exposure to RFR are consistent with the results of the NTP study [7, 8] on near field exposure to RFR, as both reported an increase in the incidence of tumours of the brain and Shwannomas of the heart in RFR-exposed Sprague-Dawley rats; and 3) because the tumours of the brain and heart observed at increased incidence in rats exposed to RFR generated by an 1.8 GHz GSM antenna in our study and the one of NTP are of the same cytological origin of those observed in some epidemiological studies [10, 11, 12, 13] of cell phone users. Glioblastoma Multiforme (GBM, an associated rapidly progressive fatal brain cancer) and acoustic neuroma, satisfy the Hill criteria for causality from RFR exposure based on human epidemiological studies [14]. For all these scientific considerations we are asking for adopting the precautionary principle.

Precautionary protection of public health

The application of the Precautionary Principle has been called for over many years, by multiple, credible, professional organisations and most recently by the European Parliament [15, 16, 17]. This is now crucial in order to protect both public health and the economy given the already apparent escalating health costs. RFR has been proven to damage biological systems at levels well below those claimed to be safe within the ICNIRP guideline levels [18]. Public exposures to existing levels of RFR are already harmful and will rise substantially with the deployment of 5G.

From the results of our study we consider the ICNIRP guidelines for limiting exposure to electromagnetic fields as insufficient. They should be adapted, like many countries as Italy and Switzerland already have realized, specifically intending to minimise the possible risks in their country.

In Italy 20V/m are considered as a limit of exposure, 6V/m as a limit of attention for residents and employees, and 6V/m as a so-called quality limit. In 2012, the limit of attention has been massively relaxed [19]; 6V/m have been considered since as an average in 24 hours. In the light of the precautionary principle, this threshold value should thus not be exceeded at any time.

Furthermore, Companies should be called to produce lesser invasive devices, able to reduce the exposures from mobile phone itself. New experimental research should be soon performed to evaluate the 5G frequencies alone, frequencies not yet adequately investigated, and the possible synergistic effects of exposure to concurrent different frequencies, that is exactly what happens to the human population.

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Wooden exposition system for experimental animals of the Ramazzini study[1], reproducing the situation in a condominium. The animals' exposition lasted from the 12th day of pregnancy of the mother animal until the sudden death.

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Wooden exposition system for experimental animals of the Ramazzini study, see above.

[1] Falcioni, L. et al. 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. Environ. Res. 2018, 165, 496-503.


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In the Ramazzini study[12] exposition system, every room had shielded walls to inhibit reflexion of waves.

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ÉCOSCOPE 2/20 – Tableau rélatif à l'article du Dr Fiorella Belpoggi
www.aefu.ch/ecoscope/belpoggi_tableau.pdf

Tableau: chaque carcinogène humain connu, testé de manière adéquate sur des animaux de laboratoire, a montré ici des résultats concordants en matière de carcinogénicité pour l'homme. Au centre de recherche sur le cancer Cesare-Maltoni de l'Institut Ramazzini de Bologne (I), des chercheurs ont analysé plus de 200 substances au cours des 40 dernières années. Le tableau rassemble certains exemples.

Agent	**CMCRC-RI* cancerogenicity pubblication**	**IARC Group 1** [< 1](sufficient in humans, or mechanistic upgrade*), vol, yr	Time to Group 1 (yrs)/multipote-ntial carcinogen	IARC Group 1 and **additional** tumour sites in humans/ Volume, yr
Vinyl Chloride	1974	7/1974 liver angiosarcoma	0/**38**	100F, 2012,S Hepatocellular Cancer
Chromium VI compounds	1974	2/1972 only lung	0/**38**	100C, 2012 nasal cavity & paranasal sinuses
Benzene	1979	S1, 1979 AML	0/**39**	120, 2018 L several LH tumours
Erionite	1982	42/1986 meso	4/**26**	100c, 2012, none
Trichloro-ethylene	1986	106/2012 kidney	26/**28**	106, 2014, L NHL, liver
Silica dust	1988	68/1996 Lung	8/**24**	100c, 2012, none
Asbestos (all forms)	1989	2/1973 Lung, meso	0/**22**	100C, 2012, S larynx, ovary L colorectum, pharynx, stomach
Formaldehyde	1989	88/2006 NPC	17/**23**	100F 2012, S leukemia, L sinonasal
Tamoxifen	1997 Chemopreven-tion breast cancer	66/1996	0/**15**	100A, 2012(reduced risk contralateral breast cancer)
Diesel (engine exhaust)	1997	105/2012 lung	15/**15**	105, 2012 L bladder
Ethanol (in alcoholic beverages)	2002	96/2007	5/**10**	100E, 2012, none
Acetaldehyde (in alcoholic beverages)	2002	100E/2009	7/**10**	100E, 2012
Fluoroedenite	2004	111/2014 mesothelioma	10/**10**	111

Note: S = Supplement; # some variation of AML vs ANLL; several additional sites with limited evidence (100F, 120).

* The CMCRC evaluations, apart fibres like asbestos, silica and fluoroedenite, that were regarding mesothelioma, were of multiple site carcinogens.

Études animales: des pronostics de la carcinogénicité chez l'homme

Fiorella Belpoggi, Bologne (I)

Ce qui s'est avéré cancérigène dans des études animales, s'est, a posteriori, toujours révélé cancérigène pour l'homme. Il semble que cela s'applique aussi au rayonnement de téléphonie mobile.

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ÉCOSCOPE 2/20 – références relatives à l'article de Cornel Wick, Dr en méd.
www.aefu.ch/ecoscope/wick_références.pdf

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