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RESOLVING APPARENT INCONSISTENCIES IN EFFECTS OF ELECTROMAGNETIC WAVES ON CANCER AND AUTO-IMMUNITY

Vincent Lauer*

Abstract:

Experimental and statistical results on the impact of electromagnetic waves on cancer and auto-immune diseases are reviewed and their consistency with a previously published model is discussed. Experimental results on cancer are classified in three groups according to timing and bandwidth of exposure, with each of the groups yielding different results consistent with the model. Experimental results on auto-immunity are interpreted within the same frameworks as results on cancer. Statistical observations on exposure of the human population to temporary emissions from mobile phones and to permanent emissions from TV emitters or mobile telephony base stations are classified and shown to be consistent both with the model and with the experimental results.

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1 - INTRODUCTION:

A review of expert opinions does not reveal any clear answers to questions regarding the health effects of radiofrequency electromagnetic waves, particularly concerning potential carcinogenic effects. According to the WHO, "results of animal studies consistently show no increased cancer risk for long-term exposure to radiofrequency fields." (WHO 2014). The IARC concluded that there is "limited evidence" in humans or animals for the carcinogenicity of radiofrequency radiation (Baan et al 2011), based essentially on (Interphone 2010) but not on experiences on animal models. Generally, experimental and statistical results on effects of electromagnetic waves on the immune system are often viewed as inconsistent (Szmigielski 2013).

In this paper, existing studies on cancer and auto-immunity are reviewed and analyzed based on a previously published model (Lauer 2014a,b). Experimental results on cancer on animal models are classified in three groups according to timing and bandwidth of exposure, with each of the groups yielding different results consistent with the model. Experimental results on auto-immunity are interpreted within the same frameworks as results on cancer. Statistical observations on exposure of the human population to temporary emissions from mobile phones and to permanent emissions from TV emitters or mobile telephony base stations are classified and shown to be consistent both with the model and with the experimental results.

Figure 1 of (Lauer 2014b) is useful to keep as a reference to understand the experimental results, together with Table 1 (**hereafter Table [Lauer 2014b-1]**) of (Lauer 2014b) which is useful when mechanism INH dominates. Seemingly contradictory experimental results will appear mutually coherent when re-examined within this frame.

1. ANIMAL EXPERIMENTATION (FIGURE 1).

Figure 1 summarizes the findings concerning animal experimentation, discussed hereafter.

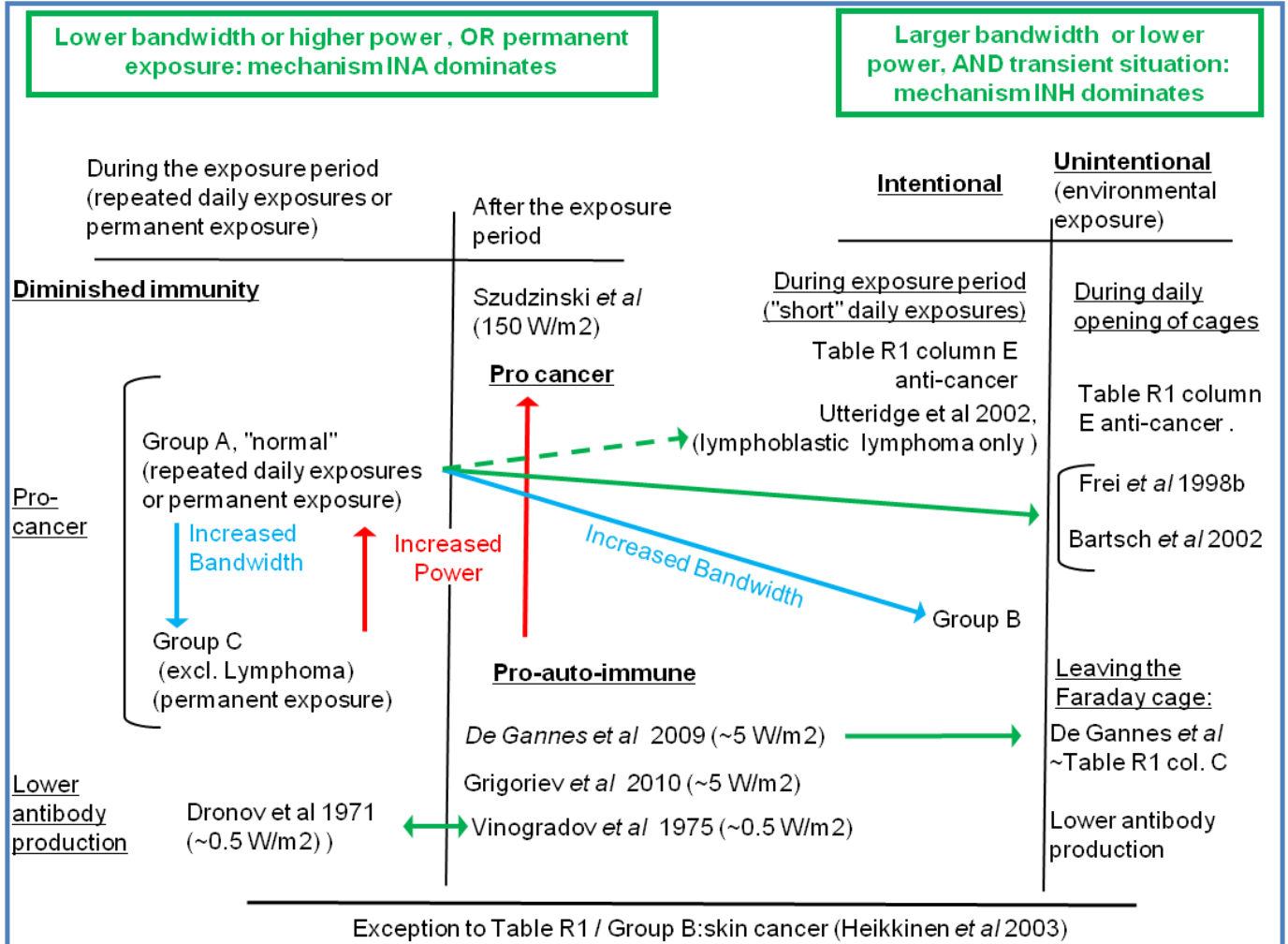


Figure 1: summary of animal experimentation.

1.1. CANCER

Cancer-related experiments were classified as follows:

Group A corresponding to very low bandwidth. Low bandwidth includes any repetitive signals with a short period, which result in several spectral lines possibly spread over a large bandwidth but not in the continuous occupation of a large bandwidth. In Group A, the low bandwidth and high instantaneous power favor mechanism INA over mechanism INH. Mechanism INA yields elimination of T lymphocytes undergoing thymus transit by positive selection and temporary inactivation of pre-existing T lymphocytes, yielding a pro-cancer effect. FM modulation (Adey et al 2000) is in group A due to its low bandwidth.

Group B, corresponding to signals having a bandwidth in the order of 100 kHz or more (which implies a modulation with random data) and exposure times of 1 to 2 hour/day. In Group B, there is a sufficient bandwidth to trigger mechanism INH, and due to the short exposure time there is an anti-cancer effect as per Table [LAUER 2014b-1] column E.

Group C, concerning signals having a bandwidth in the order of 5 MHz (also implying a modulation with random data) and near permanent (at least 20h/day) exposure. If exposure was fully permanent, under Table [LAUER 2014b-1] column B mechanism INH would not affect cancer at all. Thus, ignoring the periods of non-exposure, a pro-cancer effect is expected determined by mechanism INA. But due to the large bandwidth, mechanism INA is more efficient than in Group A yielding a significant pro-cancer effect at a lower power than in Group A. If non-exposure periods are taken into account, the overall effect is determined by competition between the pro-cancer effect and an anti-cancer effect under Table [LAUER 2014b-1] column E, and may become anti-cancer at sufficiently low exposure power or if effects of mechanism INA are cancelled for some reason.

These pro-cancer and anti-cancer effects are not systematic, because different lymphocytes may have different bandwidth, frequency and power conditions (as defined in Lauer 2014a,b). Thus pro and anti-cancer effects vary with qualitative exposure characteristics, types of cancer, animal strains, and chance since lymphocyte precursors differentiate according to a random process. When a pro-cancer (resp. anti-cancer) effect is expected by theory, the outcome on a specific animal strain and type of cancer may be either neutral or pro-cancer (resp. anti-cancer).

Experimental outcomes are further complicated by details of experimental conditions.

All experimental results reviewed in (IARC 2013) and explicitly comprising modulation with pseudo-random data of a digital transmission signal have been taken into account and classified in Group B (Adey *et al* 1999, Heikkinen *et al* 2003, Tillmann *et al* 2007, Smith *et al* 2007, see Table 2) or Group C (Tillmann *et al* 2010, Sommer *et al* 2007, see Table 3) according to the timing of exposure.

Most experimental results reviewed in (IARC 2013) fall in group A or are ambiguously termed, but only a limited number of studies were examined in detail (Table 1): Szudzinski *et al* 1982, Chou *et al* 1992, Repacholi *et al* 1997, and Adey *et al* 2000 (included in Group A due to the low bandwidth of FM signals). found significant pro-cancer effects. Utteridge *et al* 2002, Zook and Siemens 2001, Frei *et al* 1998a, and Toler *et al* 1997 found pro-cancer trends. Sommer *et al* 2004, Bartsch *et al* 2002, Frei *et al* 1998b found no pro-cancer effects nor trends.

There was no statistically significant overall anti-cancer effect in group A but there were anti-cancer aspects. In (Utteridge *et al* 2002) exposure time is 1 h/day, so that the effect could be an effect under Table [LAUER 2014b-1] column E if lymphocytes reacting to this specific cancer type have an unusually low bandwidth requirement for triggering mechanism INH. In (Bartsch *et al* 2002) and (Frei *et al* 1998a) the rats are exposed to the environmental background of artificial electromagnetic waves a few hours per day when the cages are opened for cleaning, which generates an anti-cancer effect as per Table [LAUER 2014b-1] column E, which affects both the sham-exposed and exposed groups. However if the Faraday cage for the sham-exposed group is less efficient than the Faraday cage for the exposed group (which happens easily because efficiency is essentially dependent on the quality of the door, and which is not normally checked because there are no emitted waves in the cage of the sham group that could leak into the environment) then there is an anti-cancer effect in the exposed group (which has a lower exposure to environmental waves when the cage is closed) relative to the sham group. This accidental nature of the anti-cancer effect is most evident in (Bartsch *et al* 2002) where it was not reproduced in two "identical" replications of the experiment.

Concerning lymphomas, the strength of the pro-cancer effect in (Repacholi *et al* 1997) is unusual, and explanations are uncertain. The time between exposures is 12 hours instead of 24 hours in (Utteridge *et al* 2002), which is more compatible with the roughly 12h reported duration of the temporary inactivation of lymphocytes (Lyle *et al* 1983, Lauer 2014a), and power peaks are likely higher than in (Sommer *et al* 2004) which uses permanent exposure.

Neutral results in group A are often explainable by an insufficient power: (Bartsch *et al* 2002) uses the lowest power in Group A, (Utteridge *et al* 2002) at less than 4 W/kg and (Frei *et al* 1998b) are low power replications of experiments that showed pro-cancer trends at higher power.

modulation	reference	timing	animal	N	SAR	freq.	description	short desc.
cw (Lucz generator)	Szudzinski et al 1982	2h/d	male mice	100	6 W/kg	2450 MHz	significant acceleration of skin cancer development	pro-cancer
					4 W/kg			
					2 W/kg			
GSM-like repetitive (pulse width 0.6 ms, 217 Hz)	Utteridge et al 2002	1h/d 5 d/w 104 w	female mice	120	4 W/kg	898.4 MHz	trend towards higher tumor incidence (p=0.11)	pro-cancer (trend)
					2 W/kg		no effect	neutral
					1 W/kg		No effect on total tumor incidence but trend towards less lymphoblastic lymphomas (p=0.07).	neutral (global) <u>anti-cancer trend (lymphomas only)</u>
					0.25 W/kg		No effect on total tumor incidence but less lymphoblastic lymphomas (p=0.02).	neutral (global) <u>anti-cancer (lymphomas only)</u>
repetitive pulses ? ("MiRS source")	Zook and Siemens 2001 (PRF)	6 h/d, 5d/w, 88w	male and female rats	60	1 W/kg	860 MHz	trend towards more brain tumors in exposed group (p=0.14)	pro-cancer (trend)
FM modulation with random data	Zook and Siemens 2001 (CWRF)					860 MHz	no effect	neutral
cw, non-stabilized signal generator HP8616A.	Frei et al 1998 (radiation research)	20h/d, 7d/w, 78 w	female mice	100	1 W/kg	2450 MHz	trend towards more malignant tumors (p=0.32), mammary tumors (p=0.21), animals with multiple tumors (p=0.19) in exposed group.	pro-cancer (trend)
	Frei et al 1998b (bioelectromagnetics)				0.3 W/kg		trend towards less malignant tumors (p=0.07) in exposed group.	<u>anti-cancer (trend)</u>
GSM-like repetitive (pulse width 0.6 ms, 217 Hz)	Repacholi et al 1997	30 mn each 12 hrs 80w	female mice	100	0.008-4.2 averaging 0.13-1.4 W/kg	900 MHz	more lymphomas (x2) p<0.01	pro-cancer
repetitive pulsed "uplink + downlink"	Sommer et al 2004	full time 40w	female mice	160	0.4 W/kg	900 MHz	no significant effect (but non-significantly earlier tumor onset in exposed group)	neutral
repetitive pulsed 1µs pulse width, 1 kHz pulse rate	Toler et al 1997	22h/d 7d/w 88 w	mice	200	0.32 W/kg	435 MHz	trend to earlier tumor onset in exposed group (p=0.091 one tailed).	pro-cancer (trend)
10µs pulse width, 800 pps, square modulated 8 Hz	Chou et al 1992	21.5h//d 108w	male rats	100	0.4 to 0.15 W/kg	2450 MHz	increase of primary malignant neoplasms (x4) p=0.006	pro-cancer
GSM-like repetitive (pulse width 0.6 ms, 217 Hz)	Bartsch et al 2002	52 w except 20 min/day +4 h/w	rats	60	<0.07 W/kg	900 MHz	No effect in 2 runs of same experiment and increased latency of malignant mammary tumor in exposed group in 3rd run	neutral / <u>anti cancer</u>
FM-modulated (12 kHz bandwidth, pseudo-random, 2 mn period)	Adey et al 2000	2h/d 4d/w 104w	male and female rats	90	0.27-1.6 W/kg.	836 MHz	more ENU-induced tumors in rats dying during assay.(p=0.03)	pro-cancer

Table 1: group A: CW/low bandwidth/repetitive signals (no random data), permanent or non-permanent exposure

The global teaching of Group A results is that neutral effects, pro-cancer effects and pro-cancer trends co-exist as expected, with the limit between different outcomes being uncertain, with stronger power favoring pro-cancer effects. Anti-cancer effects in Group A are mostly accidental as explained above.

Experiments in group B are expected to yield neutral or anti-cancer effects. In (Tillmann *et al* 2007), there were significant pro-cancer effects at 1.3 W/kg and 4 W/kg and a pro-cancer trend at 0.4 W/kg in male mice for exposure to GSM at 1747 MHz carrier frequency. In female mice there was a global pro-cancer effect at 1747 MHz carrier frequency, which was not significant in individual exposure groups but was significant over all averaged 1747 MHz exposure groups as compared to the average of the 902 MHz (non-affected) exposure groups. At 902 MHz there was no significant anti-cancer effect. In (Adey *et al* 1999) there was a significant anti-cancer effect (when limiting the analysis to rats that died before the end of the experiment). The anti-cancer effect in (Adey *et al* 1999) is of particular interest because the experience was exactly the same as in (Adey *et al* 2000) except for the modulation and thus the bandwidth, so that it is a particularly direct evidence of the importance of modulation and bandwidth in the experimental outcome.

reference / modulation	exp timing	N	SAR	frequency	exposed animals	description	short description
Tillmann 2007 GSM including pseudo-random data	2h/d, 5d/w, 104w	50	4 W/kg	902 MHz	male mice	no effect on total number of animals with tumors. Less hepatocellular adenoma.	anti-cancer (1 cancer type)
					female mice	no effect	neutral
				1747 MHz	male mice	less animals with tumors in exposed group in males (p<0.05). Less hepatocellular adenoma in males.	anti-cancer
					female mice	no effect	neutral
			1.3 W/kg	902 MHz	male mice	no effect	neutral
					female mice	no effect	neutral
			1747 MHz	male mice	less animals with tumors in exposed group (p<0.05)	anti-cancer	
				female mice	no effect	neutral	
			0.4 W/kg	902 MHz	male mice	no effect	neutral
					female mice	no effect	neutral
			1747 MHz	male mice	trend towards less animals with tumors (p=0.20)	anti-cancer (trend)	
				female mice	no effect	neutral	
all exp	1747 vs 902 MHz	female mice	less animals with tumors at 1747 MHz than at 902 MHz (p<0.05)	anti-cancer at 1747 MHz (a)			
Adey <i>et al</i> 1999 NADC, with random data	2h/d 4d/w 104w	60	0.27- 1.6 W/kg	836 MHz	male and female rats	less ENU-induced tumors in rats dying during assay.(p=0.03)	anti-cancer
Heikkinen <i>et al</i> 2003 Randomly modulated GSM or DAMPS	1.5 h/d 50w	26	0.5 W/kg	849 MHz (DAMPS) 902 MHz (GSM)	Female mice	More skin-tumor-bearing animals at week 50.	Pro-cancer
Smith <i>et al</i> 2007	2h/d 5d/w 104w	65	1 to 4 W/kg	902 MHz (GSM) 1747 MHz (DCS)	Male rats Female rats	Non-interpretable results (RF leaks from exposed to sham exposed groups)	Not interpretable

Table 2 : Group B: bandwidth in the order of 100 kHz, random data transmitted, part-time exposure. (a) anti-cancer effect at 1747 MHz relative to 902 MHz reveals anti-cancer effect at 1747 MHz relative to non-exposure which was not apparent due to small group size.

In (Heikkinen *et al* 2003) there were about 13% skin-tumor-bearing animals in sham-exposed non-transgenic group at week 50, 34% in RF-exposed non-transgenic group, p<0.05 using difference in proportions. Pooling the DAMPS and GSM-exposed

groups, the number of distinct skin tumor types per animal increased by 12%, $p < 0.05$ using difference in proportions and assuming distinct tumor types occur independently. The wavelength at 900 MHz is longer than the size of the mice's body, so charge carriers accumulate on the surface, canceling the field inside the body and yielding stronger fields on the skin than inside the body. When reaching the skin during exposure, lymphocytes are thus subject to mechanisms INA and INH at a stronger field than occurred during thymus selection, yielding a pro-cancer effect. Mechanism INA is likely the dominant mechanism due to its long-lasting effects (in the order of 12 h). This is an exception to the expected outcome in Group B.

In (Smith et al 2007) there were 12 females with malignant neoplasms in the DCS sham-exposed group and only 3 in the GSM sham-exposure group. This difference is significant ($p = 0.012$) and is only explainable if the RF waves from the exposed groups leaked into the sham-exposed groups, possibly due to the adopted wired termination of waveguides. The experimental outcomes thus depend on exact placement of each group in the rooms, and a detailed interpretation would be hazardous.

Experiments in Group B thus yielded the expected anti-cancer effects except for skin cancers, for the reasons explained above, and except for one non-interpretable result due to RF leaks.

reference	SAR	freq.	exp. timing	exposed animals	N	description	short description
Tillmann <i>et al</i> 2010	~0.8 W/kg (48 W/m ²)	2000 MHz	20h/day, 104 w	female mice, untreated	~54	higher average percentage of neoplastic and pre-neoplastic lesions ($p = 0.01$) (a)	pro-cancer
	female mice, ENU-treated			~58	higher average percentage of neoplastic and pre-neoplastic lesions ($p < 0.0001$) (b)	pro-cancer	
Lerchl 2005	0.4 W/kg	2000 MHz	permanent 36w	male and female mice, AKR/J (spontaneous lymphomas)	160	no effect	neutral

Table 3: group C: UMTS simulation, permanent exposure, bandwidth about 5 MHz. (a) (b): average on all reported kinds of lesions, p-value calculated from the data in Table III of (Tillmann *et al* 2010), UMTS versus sham (a) or UMTS+ENU versus ENU (b).

Experiments in Group C are expected to yield pro-cancer or neutral outcomes whenever a significant effect under mechanism INA occurs. However, in (Tillmann *et al* 2010) other aspects can also be observed (see Table 4). The sham control group shows a (near) significant anti-cancer effect relative to the cage control group, resulting from the anti-cancer effect under Table [LAUER 2014b-1] column E of exposure to environmental artificial waves during daily caretaking of the animals. The pro-cancer effect in the UMTS group relative to the sham group may result in part from a mitigation of the anti-cancer effect affecting the sham group relative to the cage controls: if the exposure in the UMTS group had been exactly equal to the environmental exposure, an apparent pro-cancer effect relative to the sham group would have resulted exclusively from cancellation, due to permanent exposure, of the anti-cancer effect affecting the sham group under Table [LAUER 2014b-1] column E, based on mechanism INH. Indeed, for hepatocellular carcinoma and hepatocellular adenoma the pro-cancer effect on the UMTS group at 48 W/m² does not go beyond the compensation of the anti-cancer effect on the sham group, so that a mitigation effect based on mechanism INH cannot be excluded. For foci of hepatocellular alteration, there was no anti-cancer effect in the sham group but there was a pro-cancer effect in the UMTS group at 48 W/m² which is thus necessarily under mechanism INA. The pro-cancer effect in the ENU+UMTS group went beyond compensation of the anti-cancer for both the foci of hepatocellular alterations and the hepatocellular adenomas, which were thus affected under mechanism INA.

The observation that hepatocellular adenomas were affected under mechanism INA at 4.8 W/m² but not at 48 W/m² is not due to an increase of the number of cases by ENU administration at 4.8 W/m², since for this lesion the number of cases was reduced by ENU administration. This observation could thus reflect a stronger anti-cancer effect at 4.8 W/m² than at 48 W/m² on Hepatocellular Adenomas. The possibility that the pro-cancer effect was lower at the higher exposure than at the lower exposure may result from the existence of an optimal power above which the efficiency of mechanism INA diminishes (Lauer 2014b). It may also result from a window effect (Lauer 2014b). in view of the fact that the signal comprised repetitive power

variations (Mbonjo Mbonjo *et al* 2004). Thus it is uncertain whether the repetitive power variations of the signal had an essential impact on the outcome or not.

	cage to UMTS (no ENU)		cage to sham (no ENU)		sham to UMTS (no ENU)		cage+ENU to UMTS+ENU		cases (cage)	cases (cage + ENU)
	p	increase	p	increase	P	increase	p	increase		
hepatocellular carcinoma	0.24	-36%	0.06	-56%	0.45	45%	1.00	0%	15	31
hepatocellular adenoma	0.47	7%	0.33	-11%	0.10	20%	<0.01	69%	46	30
foci of hepatocellular alteration	<0.01	82%	1.00	0%	<0.01	82%	0.01	83%	20	17
lymphoma	0.20	-29%	0.34	-21%	0.74	-9%	0.96	3%	24	4
other neoplastic and pre-neoplastic lesions	0.98	-1%	0.54	-23%	0.56	29%	0.02 (b)	29% (a)	13	71
total	0.51	6%	0.06	-18%	0.01	30%	<0.01(b)	36% (a)	118	153

Table 4: outcomes of the (Tillmann *et al* 2010) experiment. p-values based on difference between proportions. (a) for the average number of different neoplasms per animal (b): assuming each type of lesion occurs independently.

In (Tillmann *et al* 2010) pro-cancer effects did not affect lymphomas at 48 W/m² UMTS (about 0.8 W/kg) exposure, which is unlikely to be due to chance because there was a sufficient number of cases to detect at least a trend. Therefore, the lack of observed pro-cancer effect in a lymphoma-only experiment at 0.4 W/kg (Sommer *et al* 2007) is likely due to the fact that lymphomas were not affected under mechanism INA, possibly because the power was too high or due to a window effect. In (Sommer *et al* 2007) the number of surviving exposed animals was in fact significantly higher than the number of surviving sham-exposed animals (Table 2 of Lerchl 2005). In the absence of a pro-cancer effect under mechanism INA, this anti-cancer effect may have been due to an anti-cancer effect under Table [Lauer 2014b-1] column E due either to lack of exposure during daily periods of animal care or to the same “accidental” over-exposure discussed above for (Bartsch *et al* 2002).

Experiments in Group C thus yielded pro-cancer effects for cancers other than lymphomas. The lack of a pro-cancer effect for lymphomas in (Sommer *et al* 2007) and (Tillmann *et al* 2010) were mutually consistent. The anti-cancer effect in (Sommer *et al* 2007) on lymphomas is explainable.

Only (Szudzinski *et al* 1982) (in Group A) examined the effects of exposure on a cancer initiated after the end of the exposure period. He found a pro-cancer effect at 200 W/m² (6W/kg), due to the fact that, based on mechanism INA, positive selection in the thymus had eliminated a significant proportion of lymphocytes during the exposure period, resulting in a lower number of lymphocytes when the cancer was initiated, and thus a pro-cancer effect.

1.2. AUTO-IMMUNITY.

When rats were exposed 7 hours/day 5 days/week during 30 days to 2450 MHz at 5W/m² in a Faraday cage in Russia, a pro-auto-immune effect was found 7 and 14 days after the end of the exposure period (Grigoriev *et al* 2010). Due to the lower power, more T lymphocytes survived positive selection as compared to (Szudzinski *et al* 1982). Some of these were temporarily inactivated during their transit through the negative selection portion of the thymus, thus escaping most negative selection steps. Abnormally aggressive T lymphocytes (which should have been eliminated by negative selection) survived in a temporarily inactivated state, yielding the increased antibody production after definitive cessation of exposure when these T lymphocytes went out of the temporarily inactivated state and started auto-immune reactions. Thus, the effect post-exposure was pro-autoimmune (and impliedly anti-cancer), unlike (Szudzinski *et al* 1982) in which the effect post-exposure was pro-

cancer. However, during the exposure period, the immunity was diminished as in (Szudzinski *et al* 1982), as was verified in a similar experience (Dronov and Kiritseva 1971).

This experience was replicated in France but the pro-auto-immune effect was not observed (De Gannes & al 2009). Instead, in the exposed and sham group (both of which had stayed 30 days in the Faraday cage) a significant number of antibodies were present in significantly lesser amounts at day 14 than in the control group (which did not stay in the Faraday cage) (Table 5). An environmental exposure to a wideband electromagnetic wave was likely present, resulting in a dominant anti-auto-immune effect as per Table [LAUER 2014b-1] column C after the rats were brought out of the Faraday cage, and thus a lower antibody production.

	Number of Elisa test results in which the optical density for group (g1) is significantly higher ($p < 0.05$, single-sided) than the optical density for group (g2) [p-value]	Number of Elisa test results in which the optical density for group (g1) is significantly lower ($p < 0.05$, single-sided) than the optical density for group (g2) [p-value]
Control (g1) - exposed(g2)	13 [< 0.00001]	2 [0.69]
Exposed(g1) – Sham(g2)	4 [0.21]	3 [0.43]
Control(g1) - Sham(g2)	11 [0.00002]	1 [0.91]

Table 5: Elisa test results in (De Gannes *et al* 2009). Significance of the differences in optical densities between groups in each ELISA test result is assessed based on a single-sided z-test. Significance of the numbers shown is assessed using a cumulative distribution function of a binomial law with parameters $p=0.05$ and $N=48$ (total number of Elisa tests per group: 48).

2. USE OF MOBILE PHONES (FIGURE 2)

Effects on mobile phone users can be classified as follows.

(i) a local pro-cancer effect due to mechanism INA causing temporary inactivation of lymphocytes in the most exposed part of the head, where the instantaneous power can reach more than $60\text{W}/\text{m}^2$ for a GSM phone.

(ii) an anti-cancer, pro-auto-immune effect as per Table [LAUER 2014b-1] column E [mechanism INH].

(iii) an effect on thymus selection based on mechanism INA at a peak instantaneous power of about $2\text{W}/\text{m}^2$ for GSM. For rare exposures this effect is anti-cancer due to temporary inhibition of lymphocytes in the negative selection part of the thymus, corresponding to the pro-auto-immune effect observed in rats after cessation of exposure between 0.5 and $5\text{W}/\text{m}^2$ (Vinogradov and Dumanski 1975; Grigoriev *et al* 2010). This anti-cancer effect is attenuated by elimination during the positive selection step of T lymphocytes which otherwise would have been inactivated during the negative selection step. In the case of rare exposures (occurring at intervals longer than thymus transit time), there is no attenuation because mature lymphocytes that are temporarily eliminated in the negative selection part of the thymus during exposure were not exposed during their transit through the positive selection part. As exposures become more frequent effect (iii) is attenuated and possibly inverted, corresponding to the diminished immunity in rabbits during a period of repeated exposures (Dronov and Kiritseva 1971).

The overall effect on mobile phone users results from the combination of these effects. For rare users effect (iii) is anti-cancer and likely dominating over effect (ii) because each exposure inactivates lymphocytes for more than 12 hours, whilst inhibition under mechanism INH does not last. For moderate and heavy users effect (iii) is overcome by effect (ii) due to the relatively high bandwidth as is the case in Group B of animal exposure experiments. Indeed, the effect is found anti-cancer for rare and moderate users (Muscat *et al* 2000; Inskip *et al* 2001; Schuz *et al* 2006; Lakhola *et al* 2007; Interphone study group 2010;

Coureau *et al* 2014) with a trend towards a peak anti-cancer effect for rare users (Lakhola *et al* 2007; Interphone study group 2010) corresponding to effect (iii), which does not reach significance due to low numbers. When rare users (corresponding to maximal anti-cancer effect) are taken as the reference group the effect appears pro-cancer (Hardell *et al* 2013).

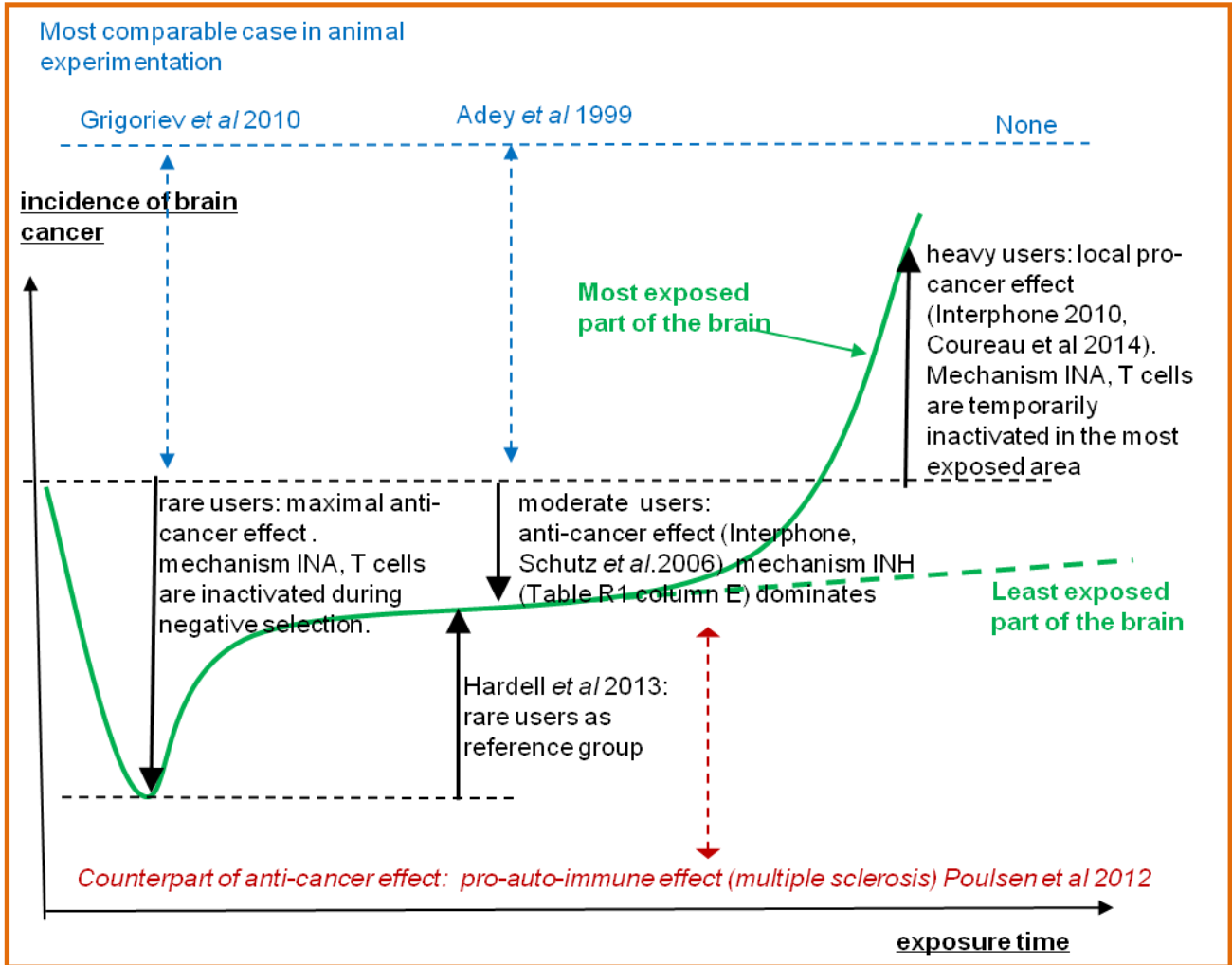


Figure 2: summary of effects on mobile phone users

Locally in the most exposed part of the brain effect (i) is found to dominate for heavy users (more than 1 hour/day) (Interphone study group 2010, Coureau *et al* 2014). The ratio of the power in the brain to the power in the thymus is stronger than in (Adey *et al* 1999) due to longer brain to thymus distance in man than in rat, so the anti-cancer effect (ii) is of a lesser amplitude than in (Adey *et al* 1999) and does not overcome the local pro-cancer effect under (i) for heavy users, obtained at a higher power.

The anti-cancer effect of moderate mobile phone use is matched by a corresponding pro-auto-immune effect yielding a higher risk of being diagnosed with multiple sclerosis for women after 10 years of mobile phone use an increased risk of death for women having multiple sclerosis if they use a mobile for 7-9 years after diagnosis of multiple sclerosis (Poulsen *et al* 2012).

3. EXPOSURE TO BROADCASTING TOWERS OR MOBILE TELEPHONY BASE STATIONS (FIGURE 3)

Onset of Digital Video Broadcasting followed by shutdown of analog television, both on a local emitter, resulted in transient variations of the percentage of deaths in the 35-54 years age class in small cities of Loire-Atlantique and Maine-et-Loire, which did not occur in comparable cities which did not have a local emitter (Lauer 2013). DVB power was less than $5 \mu\text{W}/\text{m}^2$ in at least one of these cities. These variations are attributable to the transient effects as per Table [LAUER 2014b-1] columns C and D. The fact that specific age classes are affected is attributable to corresponding stages in thymus involution. Examination of causes of death in Paris for men aged 35-44 years reveals a comparable effect (Lauer 2013) for men aged 35-44 years at the onset of DVB (which was not followed by a shutdown of analog TV), including a one-year shift between diminished mortality by heart and liver diseases (likely attributable to an anti-auto-immune effect under Table [LAUER 2014b-1] column C) and increased mortality by neoplasms (attributable to a pro-cancer effect under Table [LAUER 2014b-1] column C). This explains only partly the observations in Loire-Atlantique and Maine-et-Loire, where the affected age categories are wider. However, whilst the observed effects in Paris could arguably be due to coincidences and other causes than DVB onset, the observations in Loire-Atlantique and Maine-et-Loire cannot easily be excluded on this ground since they selectively affected cities having a change in exposure conditions.

A higher incidence of leukemia (Dolk *et al* 1997 (a), (b)) and, where evaluated, a higher cancer incidence, was found near TV and radio emitters in the UK over a 12 years period as compared to UK national average. The relative incidence rates in these cases were in the order of 5% above UK national average, much lower than in Paris following the onset of DVB, low enough to be explainable by the repeated effect of transitions as per Table [LAUER 2014b-1] column E as emitter power increases over time and by the pro-cancer effect under Table [LAUER 2014b-1] column C applied to newly arrived residents.

There are two families of standards in mobile telephony: in CDMA and UMTS the occupied bandwidth is independent of network load, whilst in GSM the occupied bandwidth varies roughly from 200 kHz to 30 MHz. Where GSM is used, lymphocytes that respond to bandwidths above a threshold which is between 200 kHz and 30 MHz do respond during high load period and do not respond during low load period. The response/non-response contrast is stronger than in the case of power variations which occur in CDMA networks, because in the simplified model of (Lauer 2014a,b) lymphocytes do not respond at all when the bandwidth of the wave is lower than their bandwidth condition, whilst their power response does not exhibit as brutal a threshold. This strong response/non-response contrast yields a stronger anti-cancer effect under Table [LAUER 2014b-1] column E for a GSM network having strong contrast between peak occupation and minimal occupation, than for a CDMA network having similar occupation/non-occupation contrast.

In Belo Horizonte in a context dominated by CDMA a 35% increase of cancer death rate was observed near mobile telephony base stations on a 10 years period relative to the null hypothesis (Dode *et al* 2011). Several effects contributed to this finding:

(i) Elimination by positive selection and temporary inhibition of lymphocytes based on mechanism INA may be the dominant cause at short distance from base stations at exposure levels of up to $0.4 \text{ W}/\text{m}^2$ at ground level and higher above ground. The existence of such pro-cancer effects of exposure to a large bandwidth wave is consistent with the findings on rodents at about $4.8 \text{ W}/\text{m}^2$ (Tillmann *et al* 2010). The power in (Dode *et al* 2011) is lower than in (Tillmann *et al* 2010), but there are strong indications in (Tillmann *et al* 2010) that the pro-cancer effect may be stronger at $4.8 \text{ W}/\text{m}^2$ than at $48 \text{ W}/\text{m}^2$, so that at $0.4 \text{ W}/\text{m}^2$ it may be comparable to what it is at $4.8 \text{ W}/\text{m}^2$. The existence of significant effects around $0.5 \text{ W}/\text{m}^2$ is also indirectly confirmed by the diminished antibody response of rabbits during the exposure period at $0.5 \text{ W}/\text{m}^2$ cw (Dronov and Kiritseva 1971) and by the experimentally observed pro-auto-immune effect after the end of the exposure period at $0.5 \text{ W}/\text{m}^2$ (Vinogradov and Dumanski 1975).

(ii) Temporary pro-cancer effects under Table [LAUER 2014b-1] column C also have a contribution, particularly as the period of the study was a period of network extension.

(iii) For a mobile phone user, the difference between exposure when calling and exposure when not calling, integrated on a bandwidth comprising both uplink and downlink emissions, is lower near to the base station. Therefore the anti-cancer effects of individual mobile phone use may be lower near to the base station, which contributes to the apparent pro-cancer effect near base stations.

(iv) The anti-cancer effect under Table [LAUER 2014b-1] column E due to the daily contrast between high and low network load had a balancing effect but essentially remained negligible as compared to the pro-cancer effects.

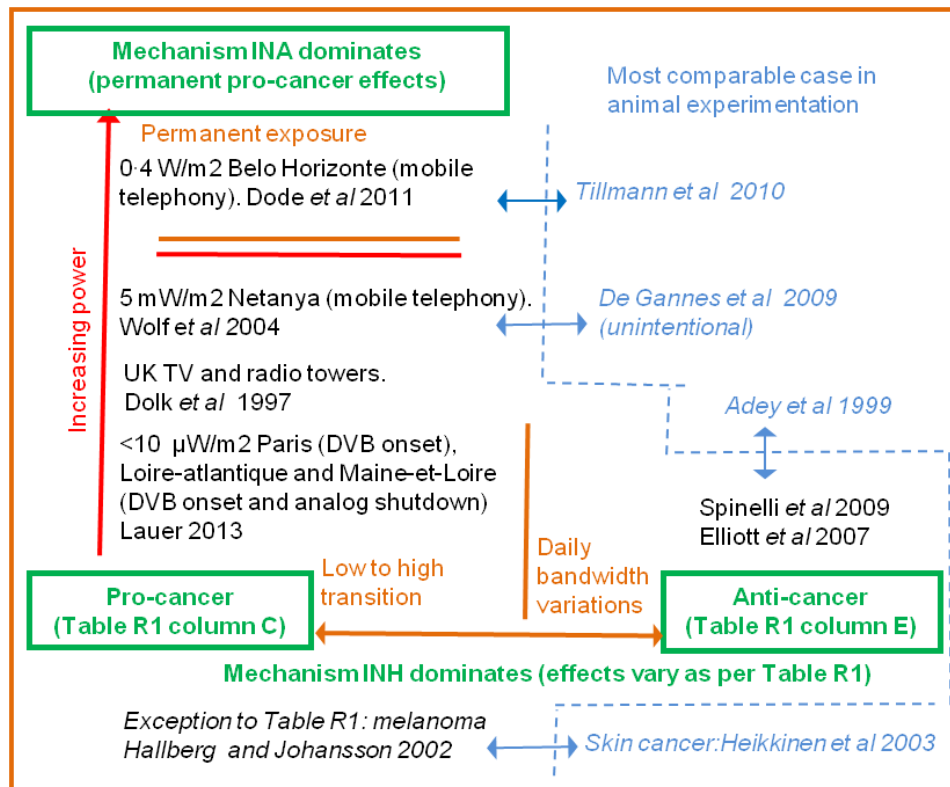


Figure 3: summary of exposure to broadcasting towers and mobile telephony base stations.

In (Spinelli *et al* 2010) in a context dominated by GSM it was found that patients diagnosed with malignant primary brain tumors in 2005 in a neurosurgery hospital lived less often within 500 m from a mobile telephony base station than other patients in neurosurgery. Other patients in neurosurgery likely comprise patients hospitalized for diseases having auto-immune aspects, so that both an anti-cancer effect of living near a mobile telephony base station and/or its corresponding pro-auto-immune effect contributed to this observation. The same contributions can be listed as in (Dode *et al* 2011) but the anti-cancer effect (iv) dominates because of the dominance of the GSM standard. This finding is confirmed, although only as a trend, in (Elliott *et al* 2010) for all cancers and particularly for leukaemia and non-Hodgkin lymphoma ($p=0.2$ for distance to base station), but not for brain cancer. The reverse trend for brain cancer in (Elliott *et al* 2010) may be attributed to there being more mobile phone users near base stations, these users having a pro-cancer trend for brain cancer due to individual mobile phone use.

In (Wolf *et al* 2004), exposure to GSM from a base station resulted in cancer incidence being multiplied by 4 in the second year after start-up of the emitter in Netanya, at 5 mW/m2 corresponding to the transient pro-cancer effect of Table [LAUER 2014b-1] column C. Most victims were women, which is attributable to a more sedentary lifestyle: men working outside the exposed area had their immune system efficiently fighting cancer when at work. Any anti-cancer effect as per Table [LAUER 2014b-1] column E was lower than the pro-cancer effect, at least during a transition period.

Epidemiological findings suggest a link between increase of melanoma incidence and exposure to FM broadcasting radio-frequencies (Hallberg and Johansson 2002), which would go farther than a temporary effect. The mechanism is analogous to the pro-cancer effect of (Heikkinen *et al* 2003) except that mechanism INH dominates due to the lower power and permanent exposure. This is an exception to Table [LAUER 2014b-1].

4. CONCLUSIONS:

Experimental and statistical results on cancer and auto-immune diseases are in agreement with the model disclosed in (Lauer 2014a,b). Whilst the model may be a crude approximation of reality, it has a strong explanatory value.

Some proposed analyses of individual experiments are open to discussion, which is unavoidable due to the many effects that participate in an experimental or statistical result. Future experimental protocols will need to be modified to avoid interference of artificial electromagnetic waves present in the environment, which impacted a number of past experimental results, and whenever possible to examine separately the effects under mechanisms INH and INA. This should make experimental results more easily interpretable and more reproducible, and will help in improving or correcting the model.

With regards to experimental results on the animal, the inconclusive findings in (IARC 2013) are in part attributable to the lack of a distinction between experiments that used randomly modulated data and experiments that used cw or pulsed signals. Since the majority of studies used cw or pulsed waves (group A), the overall impression was that no effect on cancer arose at power levels compatible with exposure of the human population. These inconclusive findings on animal models certainly impacted the perceived value of statistical results on the human being. The existence of both pro-cancer and anti-cancer effects also troubled experts who were expecting either no effect or a systematically pro-cancer effect. Indeed, whilst both the pro-cancer and the anti-cancer effects are clearly established based both on available data on animal models and on statistical observations on the human being and largely independently from the model of (Lauer 2014a,b), the lack of understanding yielded an overall impression of inconsistency and precluded the recognition and acceptance of the experimentally obtained results.

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