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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 transmitters 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 transmitters 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.

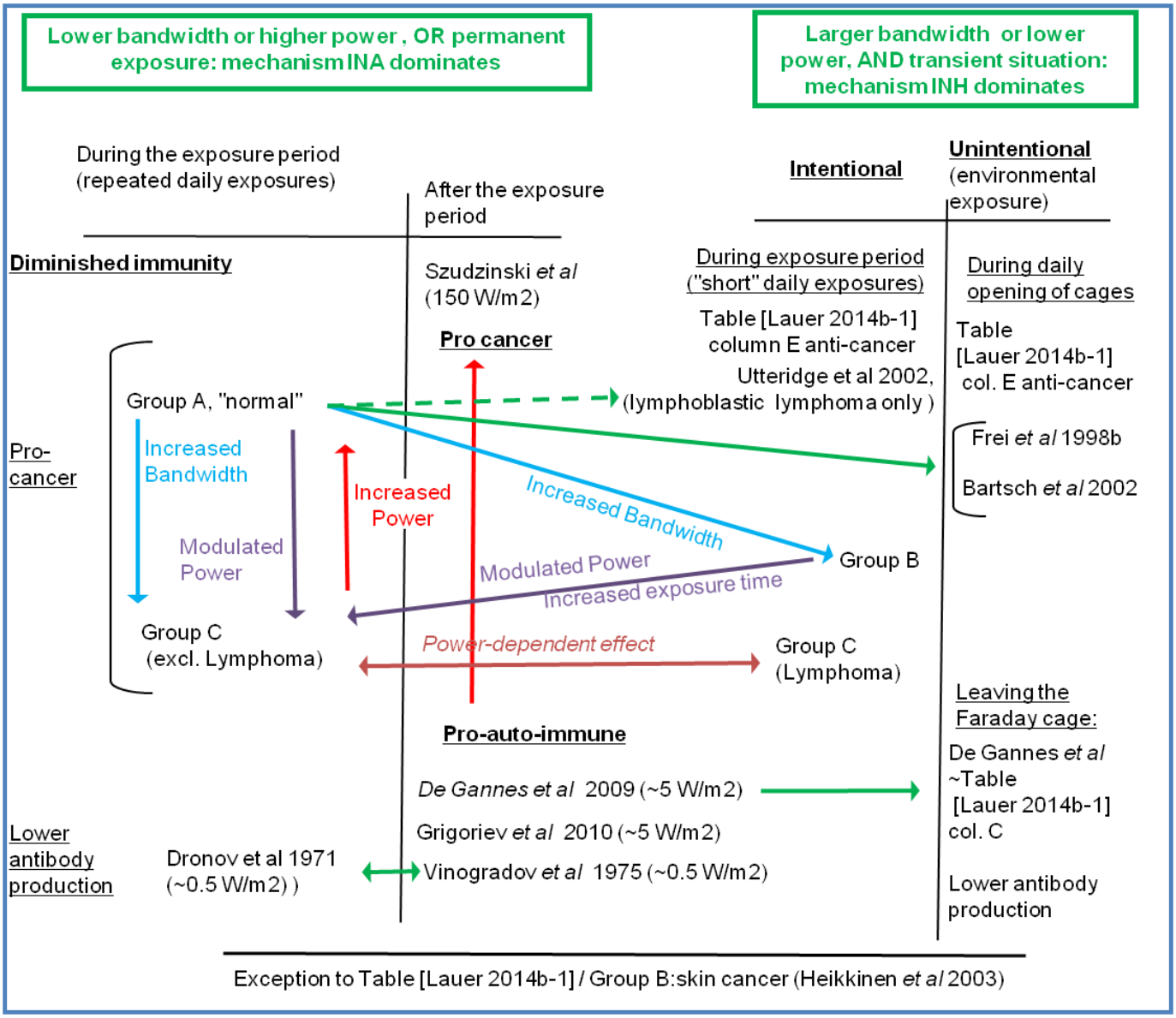

Figure 1: summary of animal experimentation.

1.1. CANCER

No cancer-related experiment employed fully permanent exposure, because exposure is always interrupted at least for daily caretaking of the animals. Thus all cancer-related experiments are part-time exposure, although exposure and non-exposure times vary widely. 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), longer exposure times of more than 20h/day and a modulated power (power control, see signal definition in (Ndoumbe M'bonjo M'bonjo 2004)). As compared to group A, the bandwidth is sufficient to trigger mechanism INH and also increase the efficiency of mechanism INA (to a lesser extent than INH). Under equation (1) of (Lauer 2014b), stimulation of mechanism INA (increase of Ω_{bd}) tends to cause oscillations between wells (c) and (d) that only marginally yield further transfers from well (d) to other quantum states needed for the further evolution of the lymphocyte to state ANR (temporary inactivation). However if large power variations exist, after transferring from state (a) to state (d) at high power the TCR-pMHC can be temporarily stabilized at low power in state (d), which favors its further evolution and thus favors mechanism INA. Therefore, as compared to both Groups A and B the modulated power in Group C favors mechanism INA. As compared to Group B, longer exposure time also reduces the efficiency of mechanism INH by reducing the non-exposure period, during which lymphocytes are efficient against cancer. If these power variations present some repetitive features, mechanism INA can present power windows as discussed in (Lauer 2014b). If power is increased above an optimal power value, mechanism INA can also become less efficient as also discussed in (Lauer 2014b). Thus effects in Group B depend on the interaction of mechanism INA (which is strongly power-dependent) with mechanism INH. They can be anti-cancer if INA is inefficient because the power is excessive or is at such position in a power window that INA is not triggered (if power windows are present), so that mechanism INA is cancelled and mechanism INH dominates. They can be pro-cancer if power is sufficient but not excessive and, if any power windows are present, properly positioned in the power window.

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, and Repacholi *et al* 1997 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, and Adey *et al* 2000 (included in Group A due to the low bandwidth of FM signals) found no pro-cancer effects nor trends.

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 4 W/kg 2 W/kg	2450 MHz	significant acceleration of skin cancer development	pro-cancer
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) anti-cancer trend (lymphomas only)
					0.25 W/kg		No effect on total tumor incidence but less lymphoblastic lymphomas (p=0.02).	neutral (global) anti-cancer (lymphomas only)
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 (bioelectro-magnetics)				0.3 W/kg		trend towards less malignant tumors (p=0.07) in exposed group.	anti-cancer (trend)
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 / anti cancer
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	No effect	neutral

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

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.

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. 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 (see Appendix A). 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 wavelenghth 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 neomplasms in the DCS sham-exposed group and only 3 in the GSM sham-exposure group (see Appendix A). 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 / 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 et al 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 et al 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 et al 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.

Experiments in Group C are expected to yield neutral, pro-cancer or anti-cancer outcomes, however neutral outcomes are less likely than in other groups because the larger bandwidth causes more lymphocytes to be affected. 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 solely 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 at 4.8 W/m2 went significantly beyond compensation of the anti-cancer for foci of hepatocellular alterations, hepatocellular adenomas, bronchio-alveolar carcinomas, and near significantly for bronchio-alveolar adenomas, all of 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 pro-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 power 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.

reference	SAR	freq.	exp. timing	exposed animals	N	description	short description
Tillmann *et al* 2010	~0.8 W/kg (48 W/m2)	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
	~0.08 W/kg (4.8 W/m2)			female mice, ENU-treated	~58	higher average percentage of neoplastic and pre-neoplastic lesions (p<0.0001) (b)	pro-cancer
Sommer et al 2007, Lerchl 2005	0.4 W/kg	2000 MHz	permanent 36w	male and female mice, AKR/J (spontaneous lymphomas)	160	Anti-cancer	Anti-cancer

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).

	48 W/m2				48 W/m2		4.8W/m2			
	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
Bronchio-alveolar carcinoma							0.01	41%		33
Bronchio-alveolar adenoma							0.07	38%		27

Table 4: outcomes of the (Tillmann *et al* 2010) experiment. p-values based on difference between proportions.

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 significantly higher than the number of surviving sham-exposed animals (Table 2 of Lerchl 2005, see Appendix A). In the absence of a pro-cancer effect under mechanism INA, mechanism INH dominated yielding an anti-cancer effect under Table [Lauer 2014b-1] column E due to lack of exposure during daily periods of animal care. A contribution of the same “accidental” over-exposure discussed above for (Bartsch et al 2002) cannot be excluded.

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 braught 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 [p<0·00001]	2 [p=0·69]
Exposed(g1) – Sham(g2)	4 [p=0·21]	3 [p=0·43]
Control(g1) - Sham(g2)	11 [p=0·00002]	1 [p=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)

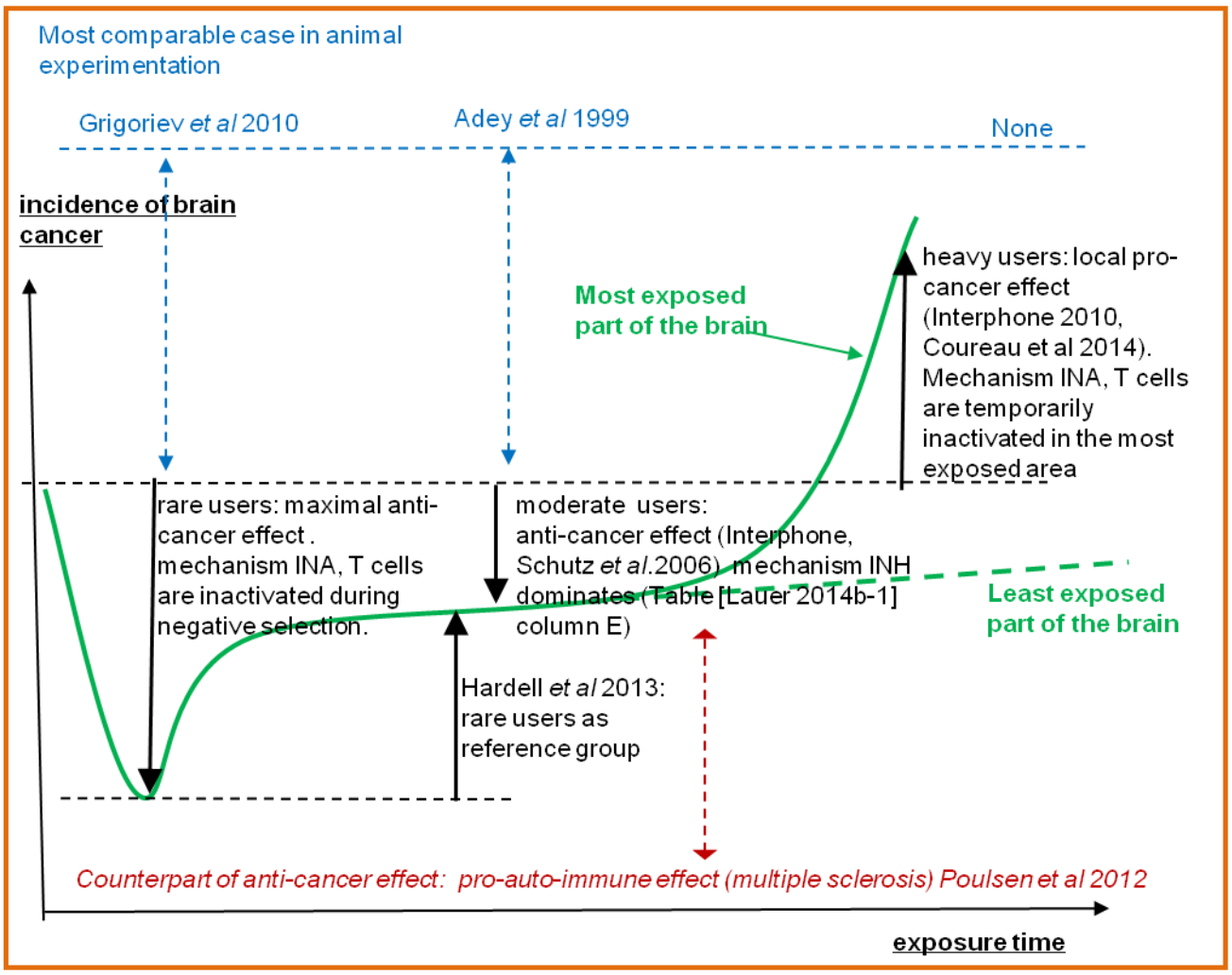

Figure 2: summary of effects on mobile phone users

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 60W/m2 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 2W/m2 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 W/m2 (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 inversed, 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).

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, see Appendix B).

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

3.1. EFFECTS OF TV TRANSMITTERS ON LEUKEMIA

Onset of Digital Video Broadcasting followed by shutdown of analog television, both on a local transmitter, 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 transmitter (Lauer 2013). DVB power was less than 5 µW/m2 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-imune 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.

In the 0-10 km around TV-only transmitters other than Crystal Palace, the incidence of Leukemia was increased by 7% relative to UK national average (Dolk et al 1997b, also see appendix B and Tables D6, D8 in appendix D). The relative incidence rate in these cases was 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 transmitter power increases over time and by the pro-cancer effect under Table [LAUER 2014b-1] column C applied to newly arrived residents. Notably, within the 0-10 km range around TV-only transmitters other than Crystal Palace the incidence of leukemia did not show any increasing trend near the transmitter, which to a certain degree confirms the dominance of an effect under mechanism INH, which is less power-dependent than mechanism INA.

The case of the Crystal Palace transmitter is specific since despite being classified as TV-only in the study it also transmitted FM radio after 1981 corresponding to the mid-term of the study. The shape of the corresponding curve (figure D2) is therefore intermediate between TV-only and mixed TV and FM emitters.

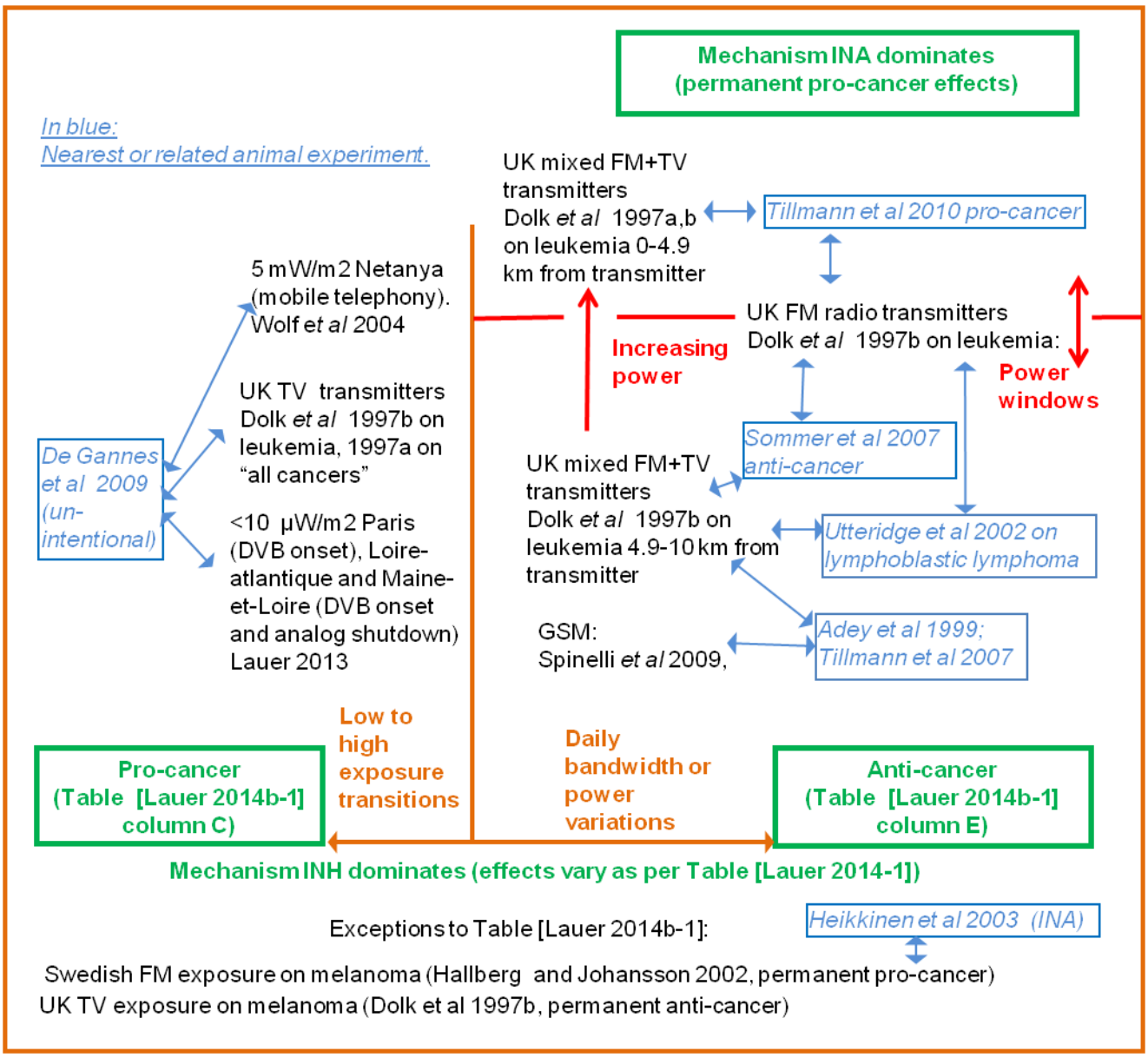

Figure 3: summary of exposure to broadcasting towers and mobile telephony base stations.

3.2. EFFECTS OF MIXED TV AND FM TRANSMITTERS ON LEUKEMIA

On the 4.9-10 km range around mixed TV and FM transmitters (6.3-10km in Sutton Coldfield) the incidence of Leukemia was significantly diminished relative to the general population except very near to the emitter (Dolk et al 1997b, also see appendix B, and Tables D11, D13 and Figure 3 of appendix D). The case of leukemia is thus specific as compared to the all-cancers case which was increased in (Dolk et al 1997a). The specificity of leukemia may be linked to the specificity of lymphoblastic lymphomas in (Utteridge et al 2002) where these lymphomas reacted to smaller bandwidth than other cancers, and with an anti-cancer effect. It is noteworthy that in the human being lymphoblastic lymphomas is essentially the same disease as acute lymphoblastic leukemia. It is known that the effects of exposure to a particular field differ in the presence of another field, see for example figure 14 and section 8 in (Lauer 2013). In the instant case, the combination of FM exposure with TV exposure may have resulted in affecting lymphocytes differently as compared to TV only or FM only exposure, in particular within a more permissive bandwidth condition than FM only. At the time FM radio stations used to shut down a few hours a day, unlike TV transmitters which were always transmitting. Lymphocytes that reacted specifically to the combination of FM and TV were thus affected part-time only, generating an anti-cancer effects under Table [LAUER 2014b-1] column E.

However in the 0-4.9 km range the incidence of leukemia was near significantly increased as compared to the general population (significantly in Sutton Coldfield in the 0-6.3 km range). The higher exposure favored effect INA, yielding a dominant pro-cancer effect due to elimination of lymphocytes by positive selection. This is also confirmed by the significant increase of leukemia in the 0-6.3 km range in Crystal Palace (intermediate between TV-only and mixed TV+FM) relative to the 6.3-10 km range (Table D7).

3.3. EFFECTS OF FM TRANSMITTERS ON LEUKEMIA (POWER WINDOWS)

Based on the reaction of lymphoblastic lymphomas in (Utteridge et al 2002), leukemia can react under mechanism INH at a lower bandwidth as compared to other cancers. It may thus have reacted to FM radio fields to which other cancers did not react. Since FM exposure was part-time, it created an anti-cancer effect as per Table [LAUER 2014b-1] column E based on mechanism INH. However, lymphocytes were also affected by mechanism INA and the observed window effect (Dolk et al 1997b, also see appendices B, D and Figure D1) results from the interaction of the two mechanisms INA and INH.

If lymphocytes are not affected under mechanism INA, the anti-cancer effect under Table [LAUER 2014b-1] column E takes place.

If lymphocytes are affected under mechanism INA, if power is sufficient elimination of lymphocytes by positive selection in exposed conditions yields a pro-cancer effect.

Thus if lymphocytes are not affected under mechanism INA, there is an anti-cancer effect, but if they are affected under mechanism INA, there is a pro-cancer effect. Since mechanism INA can be affected by power windows as discussed in (Lauer 2014b), the resulting alternance of the presence or absence of mechanism INA at different power values results in an alternance of anti-cancer and pro-cancer effects, as observed. This mechanism is analogous to the power-dependent mechanisms affecting Group C in animal experiments, although in Group C it is unclear whether power windows are implied or not.

The observed power windows can exist only if the exposure power is modulated. FM broadcasting uses “wideband FM”: the carrier frequency essentially disappears and the main frequency components are two symmetrical components at each side of the carrier. The wave integrated over a frequency band encompassing these two frequencies has time variations with characteristic times equivalent to the inverse of the difference between these frequencies. These time variations may have provided the modulation of exposure power over time which is necessary to create power windows.

3.4. EFFECTS OF MIXED TV AND FM TRANSMITTERS ON “ALL CANCERS”

In the 0-10 km around a mixed TV and FM radio transmitter, the incidence of “all cancers” was increased by 3% relative to the UK national average (Dolk et al 1997a). This pro-cancer effect also presented power windows (Figure D4) similar to those observed for FM transmitters, but fading away more rapidly with distance from the transmitter. The overall effect is likely a superposition of the temporary pro-cancer effect on persons moving into the area (similat to the effect discussed in 3.1 for TV-only emitters) and of the power windows discussed above for FM exposure.

3.5. EFFECTS OF FM TRANSMITTERS ON MELANOMA

Melanoma incidence was found to increase with the number of FM transmitters (Hallberg and Johansson 2002, see Appendix C). This pro-cancer effect is similar to the one discussed for (Heikkinen et al 2003): the 3 m wavelenghth at 100 MHz is longer than the size of the human 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 INH is likely the dominant mechanism due to the low exposure power. This effect depends on the contrast between exposure power on the skin and exposure power in the thymus, i.e. attenuation between skin and thymus. In a large range of power (wherever thymus exposure remains significantly higher than thermal power) this effect is thus substantially independent of power. This effect is based on mechanism INH and is thus strongly dependent on bandwidth. An increased number of FM

transmitters yields an increased overall bandwidth, which favors mechanism INH and therefore the pro-cancer effect. This explains the increase of the pro-cancer effect with the number of FM transmitters in (Hallberg and Johansson 2002).

3.6. EFFECTS OF TV TRANSMITTERS ON MELANOMA.

In (Dolk et al 1997b) there is an anti-melanoma overall effect, dominated by TV-only exposures (see Table D19). The cause of this effect could involve a shielding effect. The part of the skin which is opposite the RF source is shielded by the body (at sufficiently short wavelength) and thus subject to a lower exposure than the thymus, generating an anti-cancer effect. As the person moves the shielded part of the body changes, but an anti-cancer effect which is only part-time can still be efficient, so that overall there is generally an anti-cancer effect on melanoma.

3.7. EFFECTS OF MOBILE PHONE BASE STATIONS

Unlike TV transmitters, mobile phone base stations emit a time-varying signal. Generally, the following aspects may contribute to effects near a mobile phone base station:

(i) Permanent pro-cancer effects due to elimination by positive selection and temporary inhibition of lymphocytes based on mechanism INA, at very short distance from the base station, analogous to the pro-cancer effect observed in (Tillmann et al 2010) .

(ii) Temporary pro-cancer effects under Table [LAUER 2014b-1] column C also have a contribution, particularly in 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, possibly contributing to an 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.

For non-users, when far enough from the base station to avoid the high-power effect (i) or (vi), the anti-cancer effect (iv) is the sole long-term effect. Accordingly, 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 anti-cancer effect (iv) dominates.

However, in the years immediately following the onset or any power or bandwidth increase of the base station, there can be a pro-cancer effect (ii). Accordingly, 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 transmitter 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.

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 may be 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 may yield 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 or UMTS network having similar occupation/non-occupation contrast. Therefore, the result in (Spinelli et al 2010) which was obtained in a context dominated by GSM, does not necessarily apply to CDMA or UMTS base stations.

The study in (Dode et al 2011) might have been impacted by pro-cancer effects (i) and (iii) in view of measured power levels reaching 0.4 W/m2 at ground level and in view of the use of CDMA, but is unreliable due to methodological issues (Appendix C). Therefore effects (i) and (iii) cannot be considered as statistically confirmed.

In (Elliott et al 2010), cancer in children aged 0-4 years was investigated in relation to the mother's exposure during pregnancy (which in 62% of cases was the same as exposure at diagnosis), in a GSM-dominated context. This study may have been affected by an anti-cancer effect (iv) similar to (Spinelli et al 2010) but was inconclusive. This could be an indication that children are less sensitive to permanent anti-cancer effects, in good agreement with the fact that in figure 29 of (Lauer 2013) an abnormally low cancer death rate after GSM onset was found to have occurred for men in age categories above 35 years but not below 24 years.

4. A REMARK ON POWER-DEPENDENT PRO-CANCER EFFECTS.

Whether in animal experiments (Tillmann et al 2010) or in epidemiology (Dolk et al 1997b, FM exposure at pro-cancer position in power window, see appendices B, D) permanent power-dependent pro-cancer effects have been confirmed only for time-varying exposure at sufficient power, although the non-exposure time was much shorter than the exposure time. In such situations, a lymphocyte normally meets positive [resp. negative] selection both in exposed and non-exposed periods during its transfer through the positive [resp. negative] selection section of the thymus. The most demanding criteria determine the outcome, i.e. positive selection in exposed conditions and negative selection in non-exposed conditions. If power is sufficient so that all lymphocytes that are capable of surviving positive selection are eliminated by negative selection, there is a strong pro-cancer effect.

If exposure is permanent, then the negative selection criteria becomes more lenient, and lymphocytes survive positive and negative selection even if in a time-varying situation they would not have survived. The pro-cancer effect in a permanent exposure situation is thus weaker than in temporary exposures. On the other hand it is not competing with an anti-cancer effect. Overall, the magnitude of the pro-cancer effect in a permanent exposure situation is uncertain and cannot be predicted from the existing results in non-permanent exposure.

5. 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 experientally obtained results.

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APPENDICES

Appendix A-Justification of findings for experiments in Groups B and C when pro- or anti-cancer effects are not clearly stated in the conclusions of the original article.

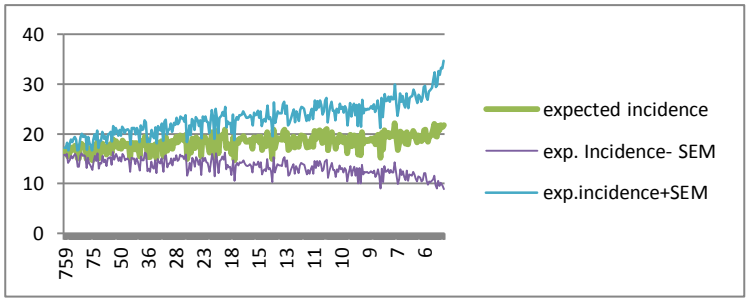
reference	conclusions of the original article	Facts (references are to the original article)	Explanations.
Heikkinen et al 2003	Indications of accelerated tumor development, particularly in non-transgenic animals, but no significant effect. *The proportion of animals with macroscopic skin tumors (non-transgenic and transgenic combined) that appeared before week 51 were 2,22,37 and 41% for the cage-control, sham-RFR, RFR(DAMPS) and RFR(GSM) groups, respectively [non-significant between sham-RFR and RFR]*	Figure 5 shows that at week 50, which is the last week for which validated results are available, there are 13% skin-tumor-bearing animals in sham-exposed non-transgenic group (N=26), but 34% in RF-exposed non-transgenic group (N=53 pooling DAMPS and GSM exposure groups), p<0.05 using difference in proportions.	The author did not pool the two exposure groups. Instead he pooled the non-transgenic animals with the transgenic animals which reacted only weakly, yielding non-significant results.
Sommer et al 2007	Chronic exposure to UMTS-modulated electromagnetic fields had no influence on lymphoma development. *There was no significant difference in the percentage of healthy animals at the end of the experiment .* *No effects on survival were attributable to the exposure (with animals censored which were still alive at the end of the study)*	Table 1 shows 28 survivals of exposed animals vs 14 survivals of sham-exposed animals, a difference which is significant as stated in (Lerchl 2005) which describes the same experience.	Limiting the analysis to animals that were still healthy at the end of the experiment, or censoring animals that were surviving, yielded non-significant results, whilst the brute results (i.e. taking into account all surviving animals) were significant as stated in (Lerchl 2005).
Smith et al 2007	*There was an incidence of 4/50 prostate adenomas in the DCS high-dose group compared to 0/50 in the sham-exposed controls (…) this is considered an isolated, incidental finding unrelated to treatment.*	Table 8 shows that the number of females with malignant neoplasms was 3 in the GSM sham-control group and 11 in the GSM low-dose group, a significant pro-cancer effect (p=0.02 using difference in proportions). Table 8 shows that the number of females with malignant neoplasms was 12 in the DCS sham-control group but 3 in the DCS low-dose group, a significant anti-cancer effect (p=0.01 using difference in proportions). Table 8 shows a significant difference between the number of females with malignant neoplasms in the GSM sham-exposure group (3) and in the DCS sham-exposure group (12).	The existence of both pro-cancer and anti-cancer effects in this experiment was not noted. The difference between different GSM and DCS sham-exposed groups was not noted.

Appendix B- Justification of epidemiological findings when effects are not clearly stated in the conclusions of the original article.

reference	conclusions of the original article	Facts	Explanations.
Poulsen et al 2012	We found little evidence for an association between mobile phone use and risk of Multiple Sclerosis (MS) or of death in MS patients (…) *We did, however, observe an increased risk of both MS symptoms and diagnosis along long-term female subscribers, although this was based on small numbers.*	(references from original article) In women: A) The risk of MS diagnosis was significantly increased in women having used a mobile phone for more than 10 years (Table 1, OR=2.08, 95%CI 1.08-4.01, based on 9 cases). B) The risk of dying from MS was significantly increased in women having 7-9 years of subscription after MS diagnosis (Table 3, OR=2.44, 95% CI 1.20-4.98, based on 8 cases) Exposure periods started in 1987 and ended in 2004 (end of the follow-up), i.e. a total of 18 years. For a woman to be both in the cases A and B she would have had to get a subscription in 1987 or 1988. Mobile phone penetration in the Danish female population in 1990 was still near zero (Figure 1), so it is unlikely that any women were simultaneously in the cases determining A and B. The finding of an increased risk of MS diagnosis or death after sufficiently long exposure is thus based on a total of 17 cases, which not a "small number". It does not reflect in the overall risk estimate for the risk of MS diagnosis because the 9 cases for this risk represent less than 10% of the total cases. In men: C) The risk of MS diagnosis was increased near significantly overall (Table 1, OR=1.11, 95%CI = 0.98-1.26, based on 406 cases). This increase was due to patients diagnosed within 1 to 9 years from subscription for which the increase was significant when averaging the log of the OR for the 3 corresponding age classes (OR=1.14, 95% CI 1.00-1.31, based on 341 cases, not shown in original paper). It was also near significant for patients diagnosed within 4 to 6 years after subscription (OR=1.21, 95% CI 0.98-1.50, based on 128 cases).	In the conclusions increases (B) and (C) were ignored; increase (A) was downplayed but not ignored.
Dolk et al 1997a	Confirmation of a reported excess of leukemias (within 2 km from) the Sutton Coldfield radio and television transmitter. Secondary findings of the study were declines in skin melanoma and bladder cancer with distance from the transmitter site.	(references from appendix D unless otherwise stated) For FM-only transmitters, there are pro-leukemia and anti-leukemia effects in alternating concentric circles (power windows) (Figure D1, Table D5) For TV-only transmitters there is a pro-leukemia effect above 4 km from the transmitter as compared to the general population (Figure D2 and Tables D7, D9) For mixed TV and FM transmitters there is an anti-leukemia effect which increases with increasing distance above 4 km from the transmitter (Figure D3, Table D11, D13). Transmitters at Sutton Coldfield (mixed TV and FM) and Crystal Palace (TV only until 1981, then mixed TV+FM – classified as TV-only in the study) are installed in populated areas, the site height being little different from the surroundings, whilst most transmitters are on small hills in less populated areas. Sutton Coldfield and Crystal Palace transmitters have a pro-leukemia effect relative to other transmitters in the areas near to the transmitter (Figures D2, D3; Tables D1, D2) which may be attributable to the lower height and thus higher power (Table D3). The 3 transmitters having individually significant overall leukemia risk alteration in the 0-10 km range (Crystal Palace, Sandy Heath, Winter Hill, see Table 3 of Dolk 1997b) were TV-only (although during only part of the study in Crystal Palace) and had an increased leukemia incidence as compared to the general population. It was argued that due to the use of the 1981 census the OE ratios may have been "overestimated by 1.6%, and by 4% for crystal palace". However the claimed over-estimation would not cancel the significance of the increased leukemia incidence in Sandy Health and Winter Hill, and it is also unlikely that the use of the 1981 census would have selectively affected TV transmitters as compared to mixed FM and TV transmitters. In Sutton Coldfield there was a pro-cancer effect as compared to the general population (figure D4; Table D15) which unlike the effect on Leukemia did not decrease with distance above 4 km. The "all cancers" figure was not reported in other locations. For all transmitters pooled (dominated by TV-only transmitters) there was an anti-cancer effect on skin melanoma (Table D19). There are often (non-significant) low cancer death rates very near to the transmitter, and part of the explanation may be a lower received power (Table D3).	For the Leukemia analysis all groups comprised at least some mixed TV and FM transmitters. This did not make it possible to distinguish properties of TV-only and FM-only transmitters. The "all cancers" increase in Sutton Coldfield was igored in the conclusions and the "all cancers" parameter was not reported in other sites. Variations of leukemia risk relative to the general population were inappropriately attributed to the census year used for the analysis.
Dolk et al 1997 b	Whilst *there is evidence for a decline of leukemia risk with distance from transmitters*, the pattern and magnitude of risk associated with residence near the Sutton Coldfield transmitter does not appear to be replicated around other transmitters.		

reference	conclusions of the original article	Facts (references are from original article)	Explanations.
Interphone 2010	Overall, no increase in risk of either glioma or meningioma was observed in association with use of mobile phones. *There were <u>suggestions</u> of an increased risk of glioma,(…)* However,biases and errors limit the strength of the conclusions we can draw from these analyses and prevent a causal interpretation.	Heavy short-term users having used their phone more than 1640h in the last 4 years had more chances of developing a menngioma (OR 4.8, 95% CI 1.49-15.4, based on 22 cases, Table 3) or a glioma (OR=3.77, 95% CI 1.25-11.4, based on 23 cases, Table 3). These conclusions are highly significant, and based on a reasonable number of cases. They cannot reasonably be attributed to "biases and errors". Mobile phone users had less chances of having a meningioa (OR=0.79, 95% CI=0.68-0.91, Table 2) or a glioma (OR=0.81, 95% CI=0.70-0.94, Table 2). This is also highly significant. It was also noted that for gliomas (as opposed to meningiomas which are ordinarily benign) the anti-cancer effect is stronger for rare users (appendix 2 to Interphone 2010) but the strongest correlation was found with time since first use rather than with time since start of regular use (significant increase above 2 years of use relative to less than 2 years of use). Thus there was a significant pro-cancer effect in heavy users, and a significant anti-cancer effect for average use, with the anti-cancer effect being stronger for recent or rare users.	Pro-cancer effects which were highly significant were downplayed. Anti-cancer effects were ignored in the conclusions but not in the text.
Schuz et al 2006	In conclusion, we found no increased risk of brain tumors, acoustic neuromas, salivary gland tumors, eye tumors, leukemias, or overall cancer in this large, nationwide cohort study of cellular telephone subscribers in Denmark.	Page 1710: (…) which allowed us to investigate brain tumor risk in subjects having a subscription for 10 years or more, and the number of brain tumors among such subscribers was lower than expected on the basis of the incidence rates in the general population. (….) <u>We have found no biologically plausible evidence in the literature to support an inverse association between radio frequency electromagnetic fields and brain tumor development</u> Page 1711: We observed statistically significantly reduced risks among men for all cancers and for smoking-related cancers in particular.	Anti-cancer effects were found but the authors looked for alternative explanations due to the perceived lack of "biologically plausible evidence". Anti-cancer effects were ignored in the conclusions.

Appendix C- Methodological issues with some studies

reference	Main issue	facts and comments (references are to the original papers)	Conclusion of the analysis
Dode et al 2011	No correction for confounding factors including age distribution.	A) The population within each distance range (for example less than 100 m from the antennas) increased over time. For example the number of base stations almost doubled between 2003 and 2006 (section 2.2.2.) so the population residing less than 100m from an antenna may also have doubled. However, for estimating the relative risk (Table 5), the deaths having occurred less than 100m from an antenna are divided by a single population estimate which does not depend on time. This estimate is probably based on the distribution of antennas in 2006, yielding an over-estimation of the denominator for previous years and an under-estimation of the pro-cancer effect. B) The population at risk is also over-estimated for the reason mentioned in section 2.2.5., end of first paragraph: for estimating the population within a certain distance from an antenna, Census Tracts which are only in part within that distance are taken into account in their entirety. Because the number of Census Tracts (2563) is commensurate with the number of antennas (856) this also yields a non-negligible over-estimation of the denominator and under-estimation of the pro-cancer effect. C) The city of Belo Horizonte has an older population in the Centro Sul district than in other districts (based on the IBGE census 2000), which is the dominant explanatory factor for the almost 3 times higher cancer death rate in Centro Sul than in Barreiro (Table 4). The Centro Sul district also has much more antennas than other districts (see Figure 9), so being near an antenna is associated with being in Centro Sul and thus with a stronger cancer death rate, but this stronger death rate is dominantly due to the older population, not to the presence of the antenna. Generally, differences in the age distribution are the dominant explanatory factor for strong contrasts in cancer death rates between districts. The study did not compensate these differences in age distribution, yielding an over-estimation of the pro-cancer effects. The overall result of the study is largely determined by a balance between under-estimating (issues A, B) and over-estimating (issue C). The magnitudes of the under-estimation and over-estimation are higher than the net pro-cancer effect found in the study.	The conclusion of a pro-cancer effect in (Dode et al 2011) is unreliable because it is dominantly determined by the balance between over-estimation and under-estimation (issues A,B,C). This does not exclude the possibility of a pro-cancer effect having taken place in Belo Horizonte during the period of the study, but such effect is not reliably reflected in the study.
Hallberg and Johansson 2002	No correction for confounding factors including age distribution.	This paper presents a graph (Figure 7) in which melanoma incidence was plotted against the number of received FM transmitting towers in each Swedish municipality. In this graph, melanoma incidence is not age-standardized nor corrected for any confounding factor. However the expected incidence (figure C1) is largely independent of the size of the municipality and even increases with decreasing number of inhabitants. Since a large number of inhabitants is likely associated with having more FM transmitters, non-correction of the age and sex distribution is likely to yield a slight under-estimation of the pro-cancer effect, rather than over-estimation. The large dispersion of incidences in Figure 7 is commensurate with the evaluation of the SEM on 5 years corresponding to the 1992-1996 averaging period and it thus due to the small size of municipalities rather than to any hidden confounding factor. Non-correction of UV exposure as a confounding factor is a potential problem but effects of UV exposure are usually attributed to travel abroad, which is likely to affect different geographic areas of Sweden reasonably homogeneously, so that there is not much chance of it having caused the relatively large observed variations of the Figure 7 graph.  Figure C1: expected incidence of melanoma in each Swedish municipality; calculated from the age and sex distribution of the population in 2002 in each municipality and from the age and sex-specific melanoma incidence in 1996 for Sweden. The number of inhabitants (in thousands) is on the horizontal axis. The expected standard error of the means for a five years period is also shown.	Methodological issues can be raised but are not supported by facts. The study appears reliable, although the influence of confounding factors other than age cannot be entirely excluded.

Appendix D – Figures and Tables supporting the analysis of (Dolk 1997a,b) in appendix B.

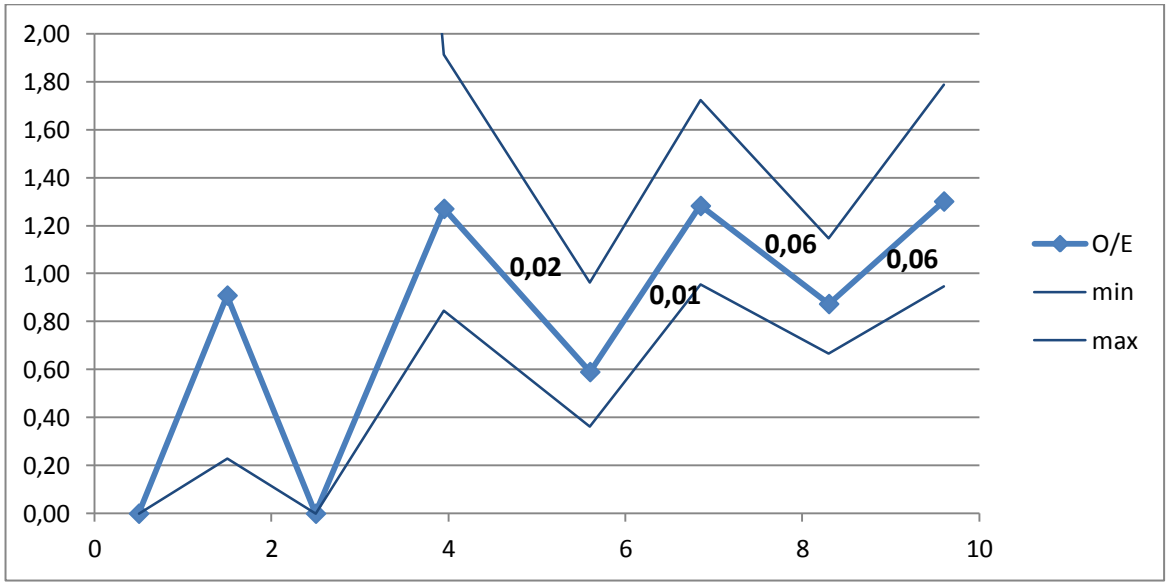


Figure D1: Observed/Expected ratios for leukemia, FM transmitters only, rearranged circles, with 95% confidence interval limited by "min" and "max" , p-values of transitions shown. FM transmitters only is Group 3 – Group 4 in Table 2 of (Dolk et al 1997b). Abscissa is in km from the transmitter.

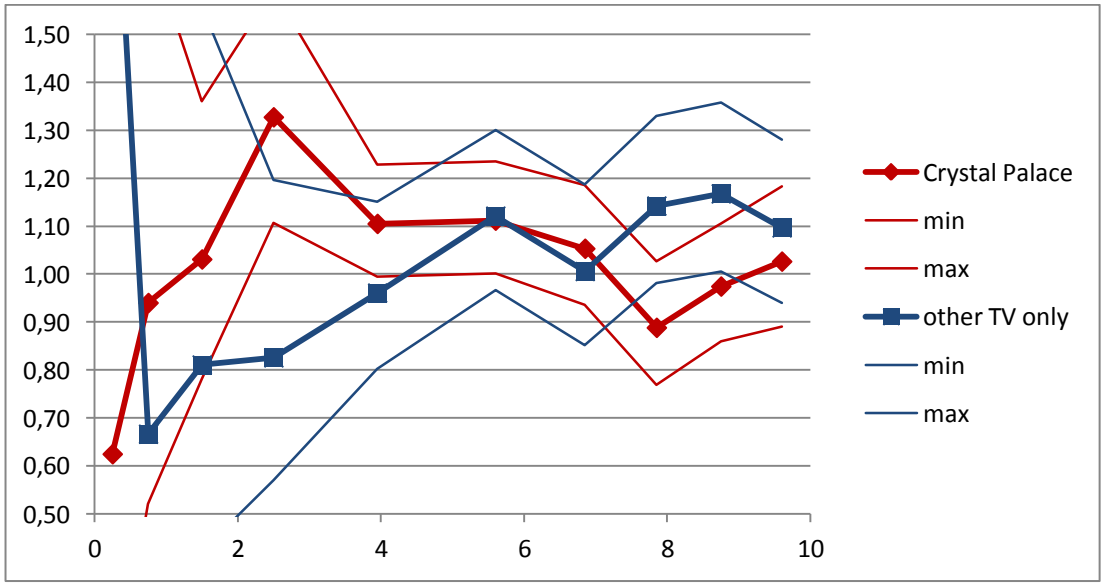


Figure D2: Observed/expected ratios for leukemia for Crystal Palace (TV only until 1981 then mixed TV and FM) and for other TV only transmitters, original circles. Other TV transmitters only is Group 2 – Group 4 – Crystal Palace in Table 2 of (Dolk et al 1997b). Also showing 95% confidence intervals as thin lines. Abscissa is in km from the transmitter.

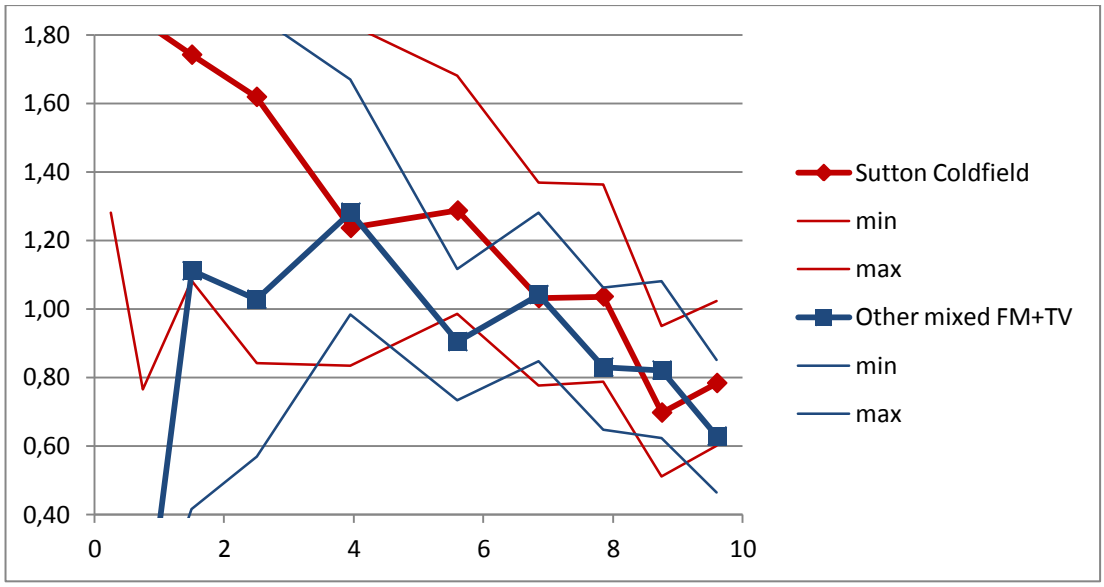


Figure D3: observed/expected ratios for leukemia for Sutton Coldfield and for other mixed TV/FM transmitters (group 4 of (Dolk et al 1997b)).). Also showing 95% confidence intervals as thin lines. Abscissa is in km from the transmitter.

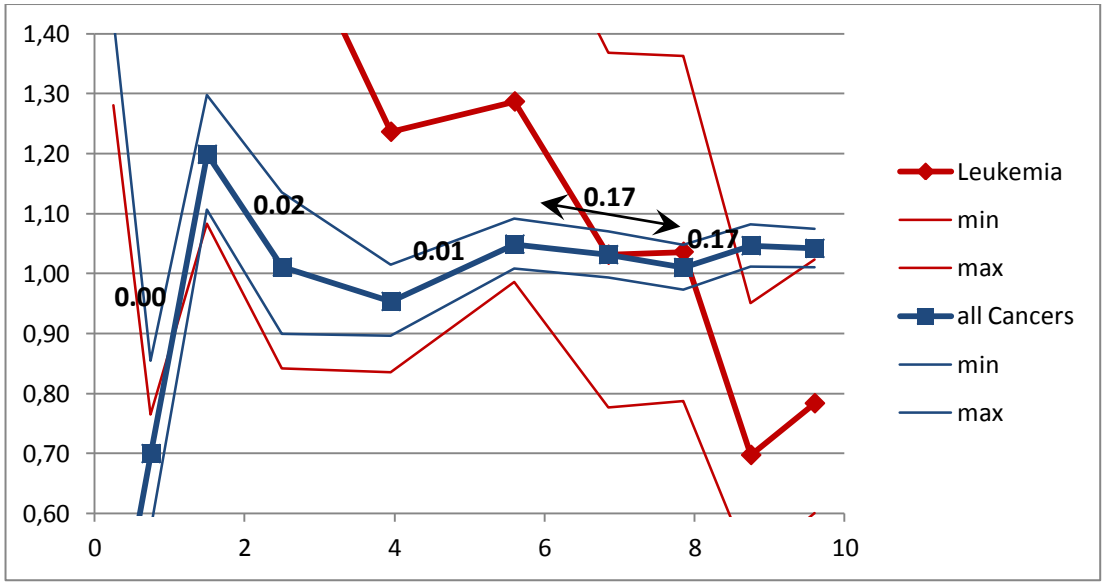


Figure D4: incidence of Leukemia and all cancers in Sutton Coldfield. From Table 2 of (Dolk et al 1997a).). Also showing 95% confidence intervals as thin lines. Abscissa is in km from the transmitter. P-values shown for transitions of interest.

Sutton Coldfield vs other mixed TV and FM									
	inner	outer	width		observed	expected	O/E	z (relative)	p (relative)
Suton Coldfield	0	3	3		32	18,15	1,76		
Other mixed TV and FM transmitters	0	3	3		15	14,6	1,02	1,75	0,08

Table D1 significance of the higher incidence of Leukemia in the first 3 km in Sutton Coldfield as compared to other mixed TV and FM transmitters.

Crystal Palace vs other TV only									
	inner	outer	width		observed	expected	O/E	z (relative)	p (relative)
Crystal Palace	0	4,9	4,9		521	459,6	1,13		
Other TV only transmitters	0	4,9	4,9		157	169,8	0,92	2,24	0,02

Table D2: significance of the higher incidence of Leukemia in the first 4.9 km in Crystal Palace as compared to other TV transmitters.

	Sutton Coldfield vs other		Crystal Palace vs other		Sutton Coldfield (different distances from transmitter)			
ERP (TV)	4,00E+06	4,00E+06	4,00E+06	4,00E+06	4,00E+06	4,00E+06	4,00E+06	4,00E+06
height above housings	**220**	**420**	**220**	**420**	220	220	220	220
distance	1500	1500	4000	4000	**750**	**1500**	**2500**	**5600**
angle, degrees	8,3	15,6	3,1	6,0	16,3	8,3	5,0	2,2
attenuation (TV)	0,15	0,05	0,6	0,2	0,025	0,15	0,25	0,25
surface of sphere	2,83E+07	2,83E+07	2,01E+08	2,01E+08	7,07E+06	2,83E+07	7,85E+07	3,94E+08
received power mW/m2 (TV)	**21,2**	**7,1**	**11,9**	**4,0**	**14,1**	**21,2**	**12,7**	**2,5**

Table D3 evaluation of received TV power in areas of higher incidence in Sutton Coldfield and Crystal Palace as compared to other transmitters, taking into account lower height above housings, and of received power at different distances from the Sutton Coldfield transmitter, based on radiation patterns in (Millard 1967,1968).

Following tables: observed/expected ratios and two-tailed p-values for leukemia for several transmitter types and diseases, for original and rearranged circles around transmitter. Relative=relative to previous circle. Absolute = relative to general population. Values are for the area between concentric circles having inner and outer diameter (in km) from transmitter, as shown. Last line of each table is for the overall 0-10km area around transmitter. Note that since difference in proportion is used, p-values are indicative only when numbers are too small (less than 5 expected or observed). Values taken from rearranged groups in Table 2 of (Dolk et al 1997b) and Table 2 of (Dolk et al 1997a).

Table D4: Leukemia, FM only: original data

inner	outer	width	Observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	0,5	0,5	0	0,1	0,00			0,32	0,75
0,5	1,0	0,5	0	0,4	0,00			0,63	0,53
1,0	2,0	1,0	2	2,2	0,91	-0,60	0,55	0,13	0,89
2,0	3,0	1,0	0	3,2	0,00	1,71	0,09	1,79	***0,07***
3,0	4,9	1,9	23	18,1	1,27	-2,02	0,04	-1,15	0,25
4,9	6,3	1,4	16	27,1	0,59	2,41	0,02	2,13	**0,03**
6,3	7,4	1,1	44	34,3	1,28	-2,73	0,01	-1,66	***0,10***
7,4	8,3	0,9	20	24,1	0,83	1,63	0,10	0,84	0,40
8,3	9,2	0,9	32	35,4	0,90	-0,30	0,76	0,57	0,57
9,2	10,0	0,8	38	29,2	1,30	-1,53	0,13	-1,63	***0,10***
0,00	10,00	10,00	175	174,1	1,01			-0,07	0,95

Table D5: Leukemia, FM only: rearranged circles

inner	outer	width	Observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	1,0	1,0	0	0,5	0,00			0,71	0,48
1,0	2,0	1,0	2	2,2	0,91	-0,67	0,50	0,13	0,89
2,0	3,0	1,0	0	3,2	0,00	1,71	***0,09***	1,79	***0,07***
3,0	4,9	1,9	23	18,1	1,27	-2,02	**0,04**	-1,15	0,25
4,9	6,3	1,4	16	27,1	0,59	2,41	**0,02**	2,13	**0,03**
6,3	7,4	1,1	44	34,3	1,28	-2,73	**0,01**	-1,66	***0,10***
7,4	9,2	1,8	52	59,5	0,87	1,89	***0,06***	0,97	0,33
9,2	10,0	0,8	38	29,2	1,30	-1,88	***0,06***	-1,63	***0,10***
0,00	10,00	10,00	175	174,1	1,01			-0,07	0,95

Table D6: Leukemia, Crystal Palace (TV only until 1981 then mixed FM+TV): original data

inner	outer	width	Observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	0,5	0,5	1	1,6	0,63			0,47	0,64
0,5	1,0	0,5	11	11,7	0,94	-0,39		0,20	0,84
1,0	2,0	1,0	50	48,5	1,03	-0,28	0,78	-0,22	0,83
2,0	3,0	1,0	116	87,4	1,33	-1,50	0,13	-3,06	**0,002**
3,0	4,9	1,9	343	310,4	1,11	1,71	0,09	-1,85	***0,06***
4,9	6,3	1,4	346	311,2	1,11	-0,08	0,94	-1,97	**0,05**
6,3	7,4	1,1	273	259,2	1,05	0,67	0,50	-0,86	0,39
7,4	8,3	0,9	184	207,1	0,89	1,79	0,07	1,61	0,11
8,3	9,2	0,9	244	250,4	0,97	-0,95	0,34	0,40	0,69
9,2	10,0	0,8	190	185,1	1,03	-0,54	0,59	-0,36	0,72
0,00	10,00	10,00	1758	1672,6	1,05			-2,09	**0,04**

Table D7: Leukemia, Crystal Palace (TV only until 1981 then mixed FM+TV): rearranged circles									
inner	outer	width	Observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	6,3	6,3	867	770,8	1,12			-3,46	**0,0005**
6,3	10,0	3,7	891	901,8	0,99	2,72	**0,007**	0,36	0,72
0,00	10,00	10,00	1758	1672,6	1,05			-2,09	**0,04**

Table D8; Leukemia, TV only except Crystal Palace: original data									
inner	outer	width	Observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	0,5	0,5	1	0,5	2,00			-0,71	0,48
0,5	1,0	0,5	1	1,5	0,67	0,82		0,41	0,68
1,0	2,0	1,0	9	11,1	0,81	-0,19	0,85	0,63	0,53
2,0	3,0	1,0	28	33,9	0,83	-0,05	0,96	1,01	0,311
3,0	4,9	1,9	118	122,8	0,96	-0,72	0,47	0,43	0,66
4,9	6,3	1,4	174	155,2	1,12	-1,29	0,20	-1,51	0,13
6,3	7,4	1,1	140	139,2	1,01	0,96	0,34	-0,07	0,95
7,4	8,3	0,9	167	146,2	1,14	-1,11	0,27	-1,72	0,09
8,3	9,2	0,9	170	145,6	1,17	-0,20	0,84	-2,02	0,04
9,2	10,0	0,8	161	146,8	1,10	0,57	0,57	-1,17	0,24
0,00	10,00	10,00	969	902,8	1,07			-2,20	**0,03**

Table D9: Leukemia, TV only except Crystal Palace:rearranged circles									
inner	outer	width	Observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	4,9	4,9	157	169,8	0,92			0,98	0,33
4,9	10,0	5,1	812	733	1,11	-2,08	**0,04**	-2,92	**0,004**
0,00	10,00	10,00	969	902,8	1,07			-2,20	**0,03**

Table D10 Leukemia, mixed TV and FM except Suton Coldfield (group 4): original data									
inner	outer	width	Observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	0,5	0,5	0	0,1	0,00			0,32	0,75
0,5	1,0	0,5	0	0,2	0,00			0,45	0,65
1,0	2,0	1,0	4	3,6	1,11	-0,47	0,64	-0,21	0,83
2,0	3,0	1,0	11	10,7	1,03	0,13	0,89	-0,09	0,927
3,0	4,9	1,9	55	42,9	1,28	-0,67	0,50	-1,85	***0,06***
4,9	6,3	1,4	87	96,2	0,90	2,04	**0,04**	0,94	0,35
6,3	7,4	1,1	90	86,4	1,04	-0,94	0,35	-0,39	0,70
7,4	8,3	0,9	63	75,9	0,83	1,39	0,17	1,48	0,14
8,3	9,2	0,9	51	62,1	0,82	0,06	0,95	1,41	0,16
9,2	10,0	0,8	42	66,8	0,63	1,29	0,20	3,03	**0,002**
0,00	10,00	10,00	403	444,9	0,91			1,99	**0,05**

Table D11 Leukemia, mixed TV and FM except Sutton Coldfield (group 4) : rearranged circles									
inner	outer	width	observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	4,9	4,9	70	57,5	1,22			-1,65	0,10
4,9	10,0	5,1	333	387,4	0,86	2,66	0,01	2,76	0,01
0,00	10,00	10,00	403	444,9	0,91			1,99	**0,05**

Table D12 Leukemia, Sutton Coldfield (mixed TV and FM) : original data									
inner	outer	width	observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	0,5	0,5	1	0,11	9,09			-2,68	0,01
0,5	1,0	0,5	5	2,72	1,84			-1,38	0,17
1,0	2,0	1,0	17	9,76	1,74	0,11	0,92	-2,32	**0,02**
2,0	3,0	1,0	9	5,56	1,62	0,18	0,86	-1,46	0,14
3,0	4,9	1,9	25	20,22	1,24	0,70	0,49	-1,06	0,29
4,9	6,3	1,4	54	41,96	1,29	-0,17	0,87	-1,86	***0,06***
6,3	7,4	1,1	48	46,54	1,03	1,12	0,26	-0,21	0,83
7,4	8,3	0,9	51	49,22	1,04	-0,02	0,98	-0,25	0,80
8,3	9,2	0,9	40	57,35	0,70	1,89	0,06	2,29	**0,02**
9,2	10,0	0,8	54	68,90	0,78	-0,56	0,58	1,80	**0,07**
0,00	10,00	10,00	304	302,34	1,01			-0,10	**0,92**

Table D13: Leukemia, Sutton Coldfield (mixed TV and FM) : rearranged circles									
inner	outer	width	observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	6,3	6,3	111	80,33	1,38			-3,42	0,0006
6,3	10,0	3,7	193	222,01	0,87	3,93	0,0001	1,95	0,05
0,00	10,00	10,00	304	302,34	1,01			-0,10	**0,92**

Table D14 : all cancers, Sutton Coldfield (mixed TV and FM): original data									
inner	outer	width	observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	0,5	0,5	2	5,61	0,36			1,52	0,13
0,5	1,0	0,5	96	137,19	0,70			3,52	**0,00**
1,0	2,0	1,0	605	504,69	1,20	-4,96	**0,00**	-4,46	**0,00**
2,0	3,0	1,0	282	279,01	1,01	2,37	**0,02**	-0,18	0,858
3,0	4,9	1,9	1002	1050,86	0,95	0,86	0,39	1,51	0,13
4,9	6,3	1,4	2414	2301,25	1,05	-2,54	**0,01**	-2,35	0,02
6,3	7,4	1,1	2734	2650,62	1,03	0,60	0,55	-1,62	0,11
7,4	8,3	0,9	2827	2798,65	1,01	0,78	0,44	-0,54	0,59
8,3	9,2	0,9	3363	3213,75	1,05	-1,38	0,17	-2,63	0,01
9,2	10,0	0,8	4084	3919,59	1,04	0,18	0,85	-2,63	**0,009**
0,00	10,00	10,00	17409	16861,22	1,03			-4,21	**0,00**

Table D15: all cancers, Sutton Coldfield (mixed TV and FM): rearranged circles									
inner	outer	width	observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	1,0	1,0	98	142,8	0,69			3,75	0,00
1,0	2,0	1,0	605	504,69	1,20	-5,19	**0,00000**	-4,46	**0,00**
2,0	4,9	2,9	1284	1329,87	0,97	4,40	**0,00001**	1,26	**0,21**
4,9	7,4	2,5	5148	4951,87	1,04	-2,37	**0,02**	-2,79	0,005
7,4	8,3	0,9	2827	2798,65	1,01	1,23	0,22	-0,54	0,59
8,3	10,0	1,7	7447	7133,34	1,04	-1,49	**0,14**	-3,71	0,00
0,00	10,00	10,00	17409	16861,22	1,03			-4,21	**0,00002**

Table D16: skin melanoma, Sutton Coldfield (mixed TV and FM): original data									
inner	outer	width	observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	0,5	0,5	0	0,09	0,00			0,30	0,76
0,5	1,0	0,5	2	2,02	0,99	-0,30	0,77	0,01	0,99
1,0	2,0	1,0	11	6,99	1,57	-0,61	0,54	-1,52	0,13
2,0	3,0	1,0	12	5,03	2,39	-1,00	0,32	-3,11	0,002
3,0	4,9	1,9	16	16,16	0,99	2,38	0,02	0,04	0,97
4,9	6,3	1,4	26	28,77	0,90	0,29	0,77	0,52	0,61
6,3	7,4	1,1	28	27,93	1,00	-0,38	0,70	-0,01	0,99
7,4	8,3	0,9	32	30,9	1,04	-0,13	0,90	-0,20	0,84
8,3	9,2	0,9	28	35,66	0,79	1,07	0,28	1,28	0,20
9,2	10,0	0,8	34	43,08	0,79	-0,02	0,98	1,38	0,167
0,00	10,00	10,00	189	196,63	0,96			0,54	0,59

Table D17: skin melanoma, Sutton Coldfield (mixed TV and FM): rearranged circles									
inner	outer	width	observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	1,0	1,0	2	2,11	0,95			0,08	0,94
1,0	3,0	2,0	23	12,02	1,91	-0,97	0,33	-3,17	**0,002**
3,0	7,4	4,4	70	72,86	0,96	2,92	**0,003**	0,34	0,74
7,4	8,3	0,9	32	30,9	1,04	-0,35	0,73	-0,20	0,843
8,3	10,0	1,7	62	78,74	0,79	1,26	0,21	1,89	***0,06***
0,00	10,00	10,00	189	196,63	0,96			0,54	0,59

Table D19: skin melanoma, all sites except Sutton Coldfield: original data									
inner	outer	width	observed	expected	O/E	z (relative)	p (relative)	z (absolute)	p (absolute)
0,0	2,0	2,0	51	45,95	1,11			-0,75	0,46
2,0	4,9	2,9	297	345,35	0,86	1,69	0,09	2,60	**0,01**
4,9	7,4	2,5	5508	6404,65	0,86	0,00	1,00	11,20	**0,00**
7,4	10,0	2,6	673	715,96	0,94	-2,18	**0,03**	1,61	0,11
0,00	10,00	10,00	6529	7511,90	0,87			11,34	0,00000000