# Radio frequency radiation-related cancer: assessing causation in the occupational/military setting

Michael Peleg[a,*], Or Nativ[b], Elihu D. Richter[b]

[a] Dept. Electrical Engineering, Technion – Israel Institute of Technology, Technion City, Haifa 3200003, Israel and Rafael Ltd. POB 2250, Haifa 3102102, Israel
[b] Unit of Occupational and Environmental Medicine, Hebrew University-Hadassah School of Public Health and Community Medicine, POB 12272 Jerusalem Israel

## ARTICLE INFO

## ABSTRACT

*Background and aim:* We reexamine whether radio frequency radiation (RFR) in the occupational and military settings is a human carcinogen.

*Methods:* We extended an analysis of an already-reported case series of patients with cancer previously exposed to whole-body prolonged RFR, mainly from communication equipment and radar. We focused on hematolymphatic (HL) cancers. We used analysis by percentage frequency (PF) of a cancer type, which is the proportion of a specific cancer type relative to the total number of cancer cases. We also examined and analyzed the published data on three other cohort studies from similar military settings from different countries.

*Results:* The PF of HL cancers in the case series was very high, at 40% with only 23% expected for the series age and gender profile, confidence interval CI95%: 26–56%, p < 0.01, 19 out of 47 patients had HL cancers. We also found high PF for multiple primaries. As for the three other cohort studies: In the Polish military sector, the PF of HL cancers was 36% in the exposed population as compared to 12% in the unexposed population, p < 0.001. In a small group of employees exposed to RFR in Israeli defense industry, the PF of HL cancers was 60% versus 17% expected for the group age and gender profile, p < 0.05. In Belgian radar battalions the HL PF was 8.3% versus 1.4% in the control battalions as shown in a causes of deaths study and HL cancer mortality rate ratio was 7.2 and statistically significant. Similar findings were reported on radio amateurs and Korean war technicians. Elevated risk ratios were previously reported in most of the above studies.

*Conclusions:* The consistent association of RFR and highly elevated HL cancer risk in the four groups spread over three countries, operating different RFR equipment types and analyzed by different research protocols, suggests a cause-effect relationship between RFR and HL cancers in military/occupational settings. While complete measurements of RFR exposures were not available and rough exposure assessments from patients interviews and from partial exposure data were used instead, we have demonstrated increased HL cancers in occupational groups with relatively high RFR exposures. Our findings, combined with other studies, indicate that exposures incurred in the military settings evaluated here significantly increased the risk of HL cancers. Accordingly, the RFR military exposures in these occupations should be substantially reduced and further efforts should be undertaken to monitor and measure those exposures and to follow cohorts exposed to RFR for cancers and other health effects. Overall, the epidemiological studies on excess risk for HL and other cancers together with brain tumors in cellphone users and experimental studies on RFR and carcinogenicity make a coherent case for a cause-effect relationship and classifying RFR exposure as a human carcinogen (IARC group 1).

## 1. Introduction and background

### 1.1. The scope

This paper examines whether exposure to radio frequency radiation (RFR) is a human carcinogen. We focus on occupational/military settings.

### 1.2. Types of exposure

Radio frequencies comprise the band of 30 kHz to 300 GHz. This includes microwaves covering the 1–100 GHz band. The major uses of RFR in the military are radio communications, radar for surveillance and weapon guidance, and electronic warfare transmitted to disrupt communications and radar. Exposures to the whole body or major parts of the body of operators and bystanders occur from a normal operation

or from lapses in safety control at near or intermediate distances at varying intensities and wavelengths. Interaction with other exposures (e.g., ionizing radiation or toxins) is possible.

The exposure levels are usually regulated within the International Commission on Non-Ionizing Radiation Protection (ICNIRP) occupational limits (ICNIRP, 1998) and exceed them occasionally due to organizational and technical shortcomings and human errors. The ICNIRP limits are designed mainly to prevent thermal damage and permit extreme peak power of pulses, provided the average power does not exceed the thermal-based limits. The high peak power may have additional biological consequences. Assessment of exposure requires considerable resources and is difficult in many cases as reported in (Paljanos et al, 2015).

### 1.3. Past findings and IARC classification

Most studies in humans have focused on brain cancers from cell phone use because hundreds of millions of people experience these exposures. The possible carcinogenicity of RFR was studied extensively by epidemiology of humans — for example in (Hardell et al., 2013; Carlberg and Hardell, 2017; Coureau et al., 2013), by animal studies with carcinogenicity possibly indicated in Chou et al. (1992); and Wyde et al. (2016); physical mechanisms such as influence on the radical oxide species in Barnes et al., 2015; and Friedman et al. (2007); and physical principles (e.g. Vistnes et al., 2001, and Peleg, 2012). In 2011, the International Agency for Research on Cancer (IARC) classified RFR as a possible human carcinogen (IARC group 2B), see IARC (2013). Hardell and Carlberg, 2013, and Carlberg and Hardell, 2017, suggested reclassifying RFR as a human carcinogen (IARC group 1) based on elevated risk ratios of mobile phone users and discussed in depth Bradford Hill's nine viewpoints from 1965 (Hill, 1965), the classical framework for causal inference in epidemiologic studies. Some reports did not detect RFR's carcinogenicity (e.g., Frei et al., 2011), which is an open and important question; see the discussion in IARC (2013).

Studies on cancers in the occupational/military setting from whole-body exposures are much fewer because the number of individuals exposed is far fewer than population-wide exposure to cellphones.

### 1.4. Context of this paper

In this paper, we extend our analysis of data previously reported in a case series of 47 self-referred patients with cancer by Stein et al. (2011). The patients were previously exposed to radiation from radio and radar in the occupational and military settings characterized in the "Types of exposure" section above.

We previously reported (Stein et al., 2011) that latent periods for cancer of the testes were very short, that latencies for HL cancers were longer, and that latent periods for solid cancers were even longer, suggesting a coherent and biologically plausible pattern of latency in relation to the onset of exposure to electromagnetic fields and other agents. Our approach (Stein et al., 2011) included a case versus case analysis in which the group of patients provides its own reference data. The case-case approach has been used by other researchers to rule out or remove biases possibly present in case-control studies. For example, Hardell et al. (2013), used meningioma cases as controls for other cancer cases to rule out bias from the analysis, particularly bias related to reporting the exposure by the patients. The case-case methodology has the advantage of relative freedom from many types of bias, including patient-reporting bias.

ln the current paper, we perform a quantitative statistical analysis of the same case series of patients with cancer (Stein et al., 2011), focusing on characteristics of the cancers in the exposed patients group rather than on the usually reported risk ratios (RR) relative to the general population. We mainly analyze the percentage (relative) frequency (PF) of hemolymphatic cancers following the approach of Boyle and Parkin (1991) (Eq. 11. 27); see the "Materials and Methods" section for details.

We will show that such an analysis is rigorous (i.e., that by using this method we can compute the probabilities of the observed cancer characteristics to occur at random by chance under the hypothesis of no causation by the exposure [p-values]). We test consistency with similar HL cancer characteristics in three other groups of patients in the military/occupational setting in three different countries using the above analysis of PF augmented by the usual RR.

## 2. Materials and methods

### 2.1. Case presentation

In the case series Stein et al. (2011), patients were referred and came to the unit of occupational and environmental medicine for the evaluation of cause-effect relationship between their cancer and their military/occupational exposure to RFR emitted by communications equipment and radar and to Extremely Low Frequency (ELF) electromagnetic fields, mainly powerlines. Most of the cancer diagnoses were during the years 1987–2007. All patients were included in the analysis and no further selection was performed. See Stein et al. (2011) for the details of patient recruitment methods. The exposure to RFR was assessed from patient's interviews and from all documentation which was available. The authors consulted with experts and the intensity range and type of exposure of each patient was estimated by an engineer based on transmitters powers and distances from antennas as available per case, see the individual exposure assessment in Stein et al. (2011).

### 2.2. Characterization of the patient's group

The patients were previously exposed to radiation from radio and radar in the occupational and military settings as presented in the "Types of exposure" section above. The exposures often involved the whole or many parts of the body, and not just the head. In certain cases, exposure came from equipment with direct contact – on the user's back or lap, such as in the case of radio transmitters with antenna. These exposures were often intense and irregular, varying in duration and target organs.

The patients came from the military, during or after the service, and from the electronics industry. They were in general younger than the average Israeli cancer patients as can be seen from the range of ages at diagnosis which was 18.5–64 years and from the much lower average age at diagnosis which is presented in Table 1. The individual ages at diagnosis are tabulated and analyzed in Stein et al. (2011).

The age profile of the population the patients came from is mostly the characteristic military one; more quantitative information is not available. Thus the analysis as presented below is based on ages at diagnosis which are known for all the patients.

Some patients were exposed also to ELF. Twenty three patients were exposed to RFR only, twenty one to both RFR and ELF and three to ELF only. This issue of mixed exposures is addressed in the 'Results' section below.

Some of the patients were presumably exposed to various chemicals, such as fuels and solvents, as typical in the military service and in the industry. Since the patients came from many different military units and workplaces, such an exposure is similar to that of all servicemen and workers. We expect the population of servicemen and workers to be

**Table 1**
Average ages at diagnosis.

| Average ages at diagnosis, years | | |
| --- | --- | --- |
| | Patient's group in Stein et al. (2011) | Israeli general population |
| All cancers | 33.3 | 65.4 |
| HL cancers | 28.9 | 61.9 |

somewhat healthier than the general public, i. e. the 'healthy soldier' and the 'healthy worker' effect. We have no evidence to indicate that there were other exposures which could account for the results we present.

### 2.3. Analysis of causation by percentage frequency of a cancer type

In our case series (Stein et al., 2011), it was not possible to obtain the data on the number of individuals ever exposed (i.e., the denominator in the cancer rate expression) and to carry out RR analysis. Problems of access to data such as denominators are common in studies in the occupational/military settings (see Milham et al., 2008). Therefore, we used a recognized epidemiological parameter to analyze such data sets proposed by Boyle and Parkin (1991) — the PF of different cancer types. PF is the proportion of a specific cancer type relative to the total number of cancer cases in the group of patients. We use the abbreviation PF for both the "relative" and "percentage" definitions and mark by "%" the values expressed as percentage.

Consistent and statistically significant association of unusual PF of a definite cancer type with some agent such as RFR exposure suggest an association of the cancer with this agent. We studied hematolymphatic (HL) and testicular cancers.

We denote by $H$ the cancer type, in this paper HL or testes. The observed PF, denoted $PF_{obs}$ (the subscript stands for "Observed"), is calculated by dividing the number $N_H$ of patients with cancer of type $H$ by the total number $N$ of cancer patients in this group.

$$PF_{obs} = \frac{N_H}{N} \qquad (1)$$

See Boyle and Parkin (1991) (Eq. 11. 27). When $PF_{obs}$ is computed in a group of patients selected randomly from the general population, the results are concentrated around the typical mean value of the general population denoted $PF_M$, and the deviation from the mean is usually within the typical statistical deviation, which becomes small for a large number $N$ of patients. If a special group of patients with cancer is selected based on specific circumstances such as previous exposure to some agent and the $PF_{obs}$ for this group deviates noticeably from the typical, the tool to decide whether this deviation can be due to random variation is the p-value — that is, the probability for such an $PF_{obs}$ to occur in a randomly selected group of patients from the general unexposed population, and equivalently, under the hypothesis of no causation by the exposure. The p-value can be calculated simply and rigorously as presented in the next section. P-values smaller than 0.05 or 0.01 are considered an indication of association between the cancer of this type and the circumstances under which the group of patients was selected, such as previous exposure to RFR.

Analyses by PF and by RR test somewhat different attributes of the cancer occurrence. The RR is the ratio by which a carcinogen increases cancer risk. Some carcinogens might increase the risk for many cancer types, as noted in animal (Chou et al., 1992) and human (Szmigielski, 1996) studies. Consider a hypothetical carcinogen with an RR high and equal for all cancer types. Such a carcinogen would affect strongly the group of the exposed people, but the observed PF for the cancer types would not change. In such a scenario, an RR-based study would detect the elevated risks unlike our PF-based approach, which is effective only if the RR of some cancer types are significantly higher than others. Indeed, Szmigielski, 1996, reports an approximate two-fold increase of brain cancer risk and no noticeable change in PF. Thus, while PF-based study can detect causation of cancer, it should be complemented by a RR study to assess the actual personal risks. Such RR studies complementing ours are available in Szmigielski (1996), Peleg (2009), and Degrave et al. (2009), indicating severely elevated RR associated with RFR.

### 2.4. The statistical analysis methods

#### 2.4.1. About this section

The "statistical analysis methods" section presents the procedures of computing PF, its confidence interval and its p-value. A statistical model of multiple primaries is cited and a consequent link between multiple primaries and RR is analyzed. The reader might skip this section on first reading of the paper.

#### 2.4.2. Notation

Random variables are denoted by italicized capital letters such as $X$. $P(X)$ denotes probability of $X$, and $P(X|Y)$ denotes the conditional probability, that is, probability of $X$ when $Y$ is known. $P(HL)$ and $P(C)$ denote the probability of HL and of any cancer, respectively.

#### 2.4.3. Percentage frequency of cancer types in the general population

We used the Israeli cancer registry (CR) (Israeli cancer database, 2016), using tables derived from Israeli Jews in 2001, 2002, and 2003, summing up the 3 years of numbers of cases for each category (gender, cancer type, and age of diagnosis group). The number of all patients with cancer was taken from the table denoted "all cancers types," "all sites combined." For HL cancers, we summed leukemia, Hodgkin's and non-Hodgkin's lymphomas, multiple myeloma, and plasma cell tumors.

The data we used are plotted in Fig. 1, summed over the two genders for clarity.

The classical statistical model is random occurrence of cancer with rates documented in the CR and determined by cancer type, age, and gender.

The probability of each patient selected at random from the patients in the general population to develop a cancer of a given type ($H$) is, by the rules of conditional probability

$$PF_M \overset{def}{=} P(H|C) = \frac{P(H)}{P(C)} \qquad (2)$$

where $PF_M$ stands for the mean PF, it is equal to the probability $P(H|C)$ of having a cancer of type $H$ conditioned on being a patient with cancer. When the studied group comprises categories of age and gender, as in this paper, the mean PF is computed as a weighted average among those categories following Boyle and Parkin (1991) (Eq. 11.29); see Eq. (6) in Appendix A. We use pie charts to present PF in Fig. 3 below.
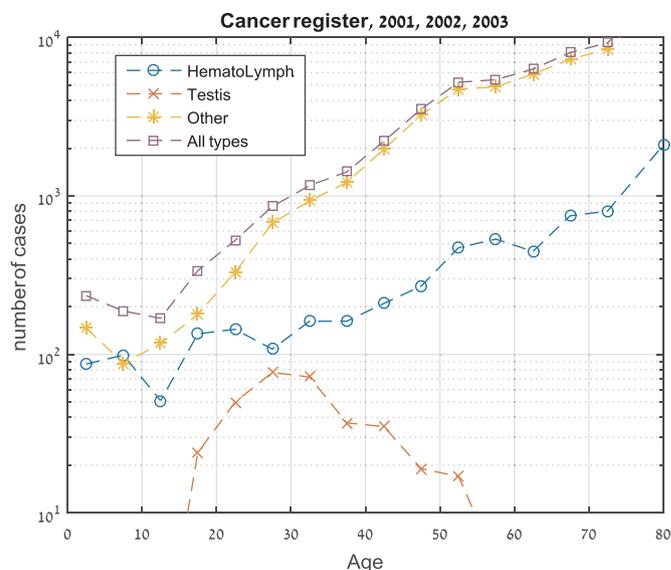


**Fig. 1.** *Number of cases from the Israeli CR* (Israeli cancer database, 2016). Years 2001–2003. Each point represents a 5-year range of ages of diagnosis. "Other" denotes non-HL and non-testicular cancer.

### 2.4.4. The p-value definition

The observed PF ($PF_{obs}$) of a cancer type (see Eq. (1), is a characteristic of a group of patients with cancer. The corresponding p-value is the probability that PF equal or larger than the observed $PF_{obs}$ would occur under the "null hypothesis," which is at random in a general population with gender and age profile identical to the exposed group and not influenced by radiation exposure.

Given our group of patients with cancer, the null-hypothesis model is as follows:

1. A hypothetical comparison group of $N$ patients is formed with ages of diagnosis and genders the same as those of the group under study.
2. The probability distribution function (PDF) of cancer types for each patient is as it appears in the CR, dependent on age of diagnosis and gender.

The p-value is the probability of at least $N_H$ cancer cases of type $H$ (in this paper usually $H$ = HL) among the $N$ patients, where $PF_{obs} = N_H / N$.

The model above is applied to our group of patients denoted $S$ being selected by the self-referral. Self-referral might be influenced by age at diagnosis $A$ and gender $G$ but not directly by the cancer type $H$. The cancer type probability is influenced by age at diagnosis and gender. Equivalently, the random variables

$$S–(A, G)–H$$

form a Markov chain implying:.

$$P(H|A, G, S) = P(H|A, G) \qquad (3)$$

where conditioning on $S$ denotes that the patient belongs to our group $S$. This Markov chain property enables the application of equation (11. 27) from Boyle and Parkin (1991) to our group of patients.

### 2.4.5. Computing p-values of PF

Our method belongs to the class of exact tests because the p-value is derived exactly, avoiding the Gaussian or similar approximations. The p-value defined above is the probability of $N_H$ reaching at least $PF_{obs} \cdot N$. Thus, we need to derive $P(N_H)$. To do so, we divide the patients into the categories of the CR (Israeli cancer database, 2016), each defined by its 5-years age of diagnosis range and gender; the categories are indexed by $k$. The PDF of number $N_k^H$ of patients with cancer type $H$ in CR category $k$ is the binomial distribution parametrized by the number of patients from the group belonging to this category and by the PF of cancer type $H$ in this category denoted $PF_k^H$. The $PF_k^H$ is obtained from the CR by dividing the number of type $H$ cancer cases in the category, by the total number of CR cancer cases in this category. Because $N_H$ is the sum of $N_k^H$, the $P(N_H)$ is the standard convolution of the binomial distributions in all the $(A,G)$ categories. The p-value is the sum of the terms of $P(N_H)$ corresponding to $N_H \geq PF_{obs} \cdot N$. The corresponding equations are presented explicitly in Appendix A. As a precaution against errors, the analysis was verified by a Monte Carlo simulation.

When computing p-value on the data in Szmigielski (1996), we use a single age-gender category comprising all the patients following the data structure of Szmigielski (1996). Our results are then identical to the single-tailed Binomial test.

### 2.4.6. Confidence interval of PF

*PF* denotes the probability of a patient randomly chosen from the exposed group having an HL cancer. Our standard confidence interval (CI) procedure estimates *PF* as the proportion of patients with HL cancer applying (Boyle and Parkin, 1991, Eq. 11.27) and then calculates its CI based on the binomial distribution of the number of HL cases parametrized by the *PF* to be estimated and by the number of patients *N*. See Appendix A for details.

The PFs and their corresponding CIs are influenced by age and gender profile and by the RFR exposure characteristics, which differ between the

groups; thus, the p-value, not the PF and its CI, is the parameter appropriate for joint evaluation of different cohorts of patients.

### 2.4.7. Analyzing multiple primaries

Some of the patients in Stein et al. (2011) reported with multiple primary tumors. The PF of multiple primaries is the observed proportion of cases with multiple primaries relative to the total number of patients with cancer. We analyzed the multiple primaries PF by the same method as we analyzed the PF of cancer type. The expected PF, denoted $PF_M$, for the age and gender profile of the group of patients under the no-causation hypothesis and the corresponding p-value were computed as in the section above. The only difference was the source and reliability of the reference data.

Data are not readily available on the incidence of multiple cancers by age and gender subgroups, so we used the model proposed by Spratt and Hoag (1966) Fig. 1 and Spratt (1977), Fig. 1. Spratt and colleagues concluded from clinical studies that the first primary and the other primaries occur mostly independently. This approximation is equivalent to a Poisson distribution for the number of primaries in each human, namely the probability of zero, one and two primaries is

$$P(0) = e^{-\lambda}$$
$$P(1) = \lambda e^{-\lambda}$$
$$P(2) = 0.5\lambda^2 e^{-\lambda} \qquad (4)$$

where $\lambda$ is the expected number of primaries in the group of initially healthy people. Now $P(0)$ is available in all CRs as the probability of not developing cancer; therefore, the probability of one, two, and more primaries in a single human can be calculated from standard CR data. Thus, Eq. (4) enables us to model the age-dependent statistics of multiple primaries as in Spratt and Hoag (1966), Fig. 1.

We verified that the Poisson distribution reproduces Spratt's results in Spratt and Hoag (1966), Fig. 1. Next, we derived similar multiple primaries statistics from the Israeli CR; see Fig. 2 below. The plot is similar to (Spratt and Hoag 1966; Figure 1).

The appropriate age parameter for each patient would be the age of self-referral. Because we do not have this statistic on record, we overestimated it conservatively by the age at diagnosis with 10 years added to it (the typical period from diagnosis to self-referral in this group of patients is 6.5 years) with an upper limit of 72.5 years (the maximal age of self-referral recorded is 68).

The ratio of $P(2)/P(1)$ from Eq. (4) shows that the proportion of double primaries relative to single primaries equals half of the expected number $\lambda$ of primaries, as in Fig. 2. Thus, the overall cancer risk $P_C$ in a
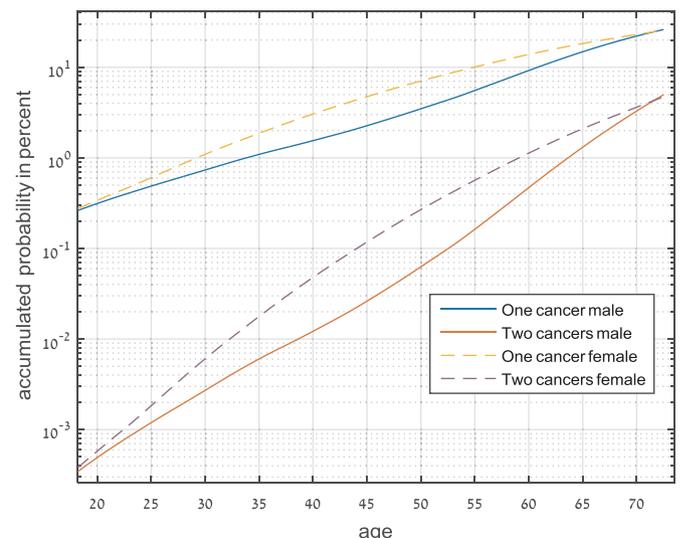


**Fig. 2.** *Probability of two primaries based on Israeli CR. Probability of one and two cancers from birth to the age on the horizontal axis. Used CR data from 2003.*

population is predicted well by twice the *PF* of multiple primaries in a patient group coming from this population; see details in Appendix A.

If the PF of multiple primaries in a group of patients with cancer is larger than the expected value by a factor *F*, then the RR, according to the Poisson model, is approximately *F* as shown quantitatively in Fig. A.1 in Appendix A.

Our analysis of multiple primaries is an approximation due to the use of Spratt's Poisson model and to the non-rigorous assignment of age to each patient; no such limitations were encountered in the analysis of the cancer types.

## 3. Results

### 3.1. The relevant data from Stein et al. (2011)

The study by Stein et al. (2011) reported an atypical distribution of cancers by types and latencies in a group of 47 patients:

1. Non-typical occurrence of cancer types diagnosed: 19 HL, 6 testicular, 14 head and neck (comprising 8 brain cancers, 3 bone cancers of the head, acoustic nerve, melanoma and carcinoma of the cheek mucosa), gastrointestinal tract, 2 breast, lung, kidney, melanoma, bone and liver. The brain cancers were 2 glioblastomas, glioma, astrocytoma, medulloblastoma, malignant neoplasm of the meninges, of the pituitary gland and of the pineal gland. We listed here the first primary of each patient.
2. A coherent and biologically plausible pattern of latency was noted in relation to the onset of exposure to RF: patients with testicular and HL tumors had shorter latencies than those with solid tumors.
3. 6 patients (12.7%) had multiple primary tumors.

Full information on all the cases is tabulated in (Stein et al., 2011). Our present work is focused on the PF of the HL cancer types which are summarized in Table 2.

### 3.2. Percentage frequency in the case series of Stein et al. (2011)

We found that the proportion PF of HL cancer in the group of

**Table 2**
HL cancer cases in (Stein et al., 2011), the first primaries.

| Classification[a] | HL cancer type | Number of cases in Stein et al. (2011) |
|---|---|---|
| C81 | Hodgkin lymphoma | 4 |
| *C83 Non-follicular lymphomas:* | | |
| C83.3 | Diffuse large B cell lymphoma | 1 |
| C83.5 | Lymphoblastic (diffuse) lymphoma | 1 |
| *C84 Mature T/NK-cell lymphomas:* | | |
| C84.4 | Peripheral T-cell lymphoma, not classified | 1 |
| *C85 Other and unspecified types of non-Hodgkin lymphomas:* | | |
| C85.9 | Non-Hodgkin lymphoma, unspecified | 2 |
| *C90 Multiple myeloma and malignant plasma cell neoplasms:* | | |
| C90.2 | Extramedullary plasmacytoma | 1 |
| *C92 Myeloid leukaemias:* | | |
| C92.0 | Acute myeloblastic (myeloid) leukemia [AML] | 2 |
| C92.9 | Myeloid leukemia, unspecified | 1 |
| *C91 Lymphoid leukaemias:* | | |
| C91.0 | Acute lymphoblastic leukemia [ALL] | 3 |
| C91.1 | Chronic lymphocytic leukemia of B-cell type | 1 |
| C91.4 | Hairy-cell leukemia | 1 |
| C91.5 | Adult T-cell lymphoma/ leukemia (HTLV−1 associated) | 1 |

a The classification system is as in http://apps.who.int/classifications/icd10/browse/2010/en#C91.

patients was 40%, while the expected PF for this age and gender profile is 23.4%. The p-value, that is, the chance that at least 19 patients with HL cancers in Stein et al. (2011), were afflicted at random in the group of 47 patients under the hypothesis of no causation by radiation, is smaller than 1% (p < 0.01). Thus, the chance of such PF increase occurring at random is small. See Table 3.

Influence of the exposure to ELF: Three of the 47 patients were not exposed to RFR but to ELF alone, none of those had HL cancer. To verify the link between the high HL PF and the RFR exposure we repeated the HL PF analysis on the 23 patients who were exposed to RFR only and not to ELF. The results showed the same characteristics as the whole group reported above: 10 patients out of 23 had HL cancers, HL PF = 43%, HL PF expected for the age and gender profile 25%, p-value = 0.037 < 0.05. We infer that the results of the whole group are a good estimate of the specific RFR influence. The data do not reveal whether ELF is carcinogenic or not since not enough patients were exposed to ELF alone to perform the PF calculation.

Applying the same analysis on all male patients and testicular cancer in Stein et al. (2011), yielded $PF_{obs}$ very similar to the one expected in the unexposed general population (p-value of 0.55). The normal $PF_{obs}$ and non-significant p-value of testicular cancers compared to the highly elevated PF for HL adds a check on our procedure: a method error increasing the PF of the HL cancers while not affecting the PF of the testicular cancers is less likely. This type of comparison is similar to the use of normal results on meningioma to verify abnormal results on other cancer types in Hardell et al. (2013).

Note that normal PF indicates only that the RR of testicular cancers is similar to the RR of all cancers in the exposed group; the absolute risk may be still raised relative to the general population, as explained in the "Materials and Methods" section above.

### 3.3. Multiple primaries in the Stein et al. (2011), group

Six out of the 47 patients with cancer joined the self-referral with a diagnosis of multiple primary tumors. In only one patient the second cancer was hematolymphatic (lymphoma) and so was his initial diagnosis. Our analysis results of the multiple primaries are

Observed PF: 6/47 = 12.8% (CI95: 4.8–26%)
Expected PF: 2.8% based on age and gender profile of the group
p-value: p < 0.005 (p = 0.0011)

Thus, the ratio of observed to expected (O/E) multiple primaries is OE = 12.8/2.8 = 4.5. As explained in the "Materials and methods" section above, this O/E ratio predicts an overall cancer RR of 4.5 relying on the model by Spratt.

Influence of exposure to ELF: Three of the 47 patients were not exposed to RFR but to ELF only, one of them reported with multiple primaries. To check the link between the high multiple primaries PF and the RFR exposure we repeated the analysis on 44 patients while excluding the three patients with no RFR exposure and also on the 23 patients who were exposed to RFR only and not to ELF. The results of both the analyses are presented in Table 4. Both results are similar or stronger than the results of the whole group above. We infer that the results of the whole group are a fair estimate the specific RFR influence.

### 3.4. Results from other studies

The results above are those of a single study and may be influenced to a limited degree by the self-referral process (see the "Discussion" section below) or by some hidden influence or error. Therefore, verification by similar occurrences in other independent and unrelated groups of patients is required. To this end, we analyzed and reviewed past reports by us and by other researchers on different and independent groups of patients. The groups were as reported in Peleg (2009), Szmigielski (1996), and Degrave et al. (2009). These reports use

**Table 3**
Percentage frequencies from four groups of patients.

| Cancer type | Patients group ref. | $PF_M$ general population according to age and gender profile of the exposed group.[*] | Group size $N$ | Number of patients with cancer of this type in the group | $PF_{obs}$ in the group and its 95% CI | p-value of $PF_{obs}$ |
|---|---|---|---|---|---|---|
| HL | Stein et al. (2011) | 23.4% | 47 | 19 | 40% (26% − 56%) | 0.0055 |
| HL | Peleg (2009) | 17.5% | 5 | 3 | 60% (15% − 95%) CI90: (19% − 92%) | 0.0384 |
| HL | Szmigielski (1996) | 12% (control group) | 66 | 24 | 36% (24.9% − 49%) | < 0.001 |
| Testis | Stein et al. (2011) | 14.7% | 40 | 6 | 15% (6% − 30%) | 0.55 |
| HL[**] | Degrave et al. (2009) | 1.4% (control group) | 133 | 11 | 8.3% | [**] RR = 7.2,CI95% = (1.09–47.9) |

[*] The age and gender adjustment in the $PF_M$ column is to that of the groups of patients specified in the Patients group ref. column.

[**] Our particular analysis method is not applicable to computing p-value and CI of the results in Degrave et al. (2009), as explained in the "Belgian radar technicians" section below. The rate ratio and its CI, copied from the table from Degrave et al. (2009), are statistically significant.

**Table 4**
RFR and ELF influence on multiple primaries.

| Exposure type: | All combinations of ELF and RFR | RFR with ELF and RFR only | RFR only |
|---|---|---|---|
| Group size | 47 patients | 44 patients | 23 patients |
| Observed PF | 6/47 = 12.8% | 5/44 = 11.4% | 4/23 = 17% |
| Expected PF | 2.8% | 2.1% | 1.7% |
| p-value | p < 0.005 | p < 0.005 | p < 0.001 |
| O/E ratio | 4.5 | 5.3 | 10 |

diverse research and analysis methods and are clearly not influenced by our current choice of cancer types. The results on HL cancer risks being associated with RFR exposure in each of the three groups, as demonstrated by high PF and/or high RR, are consistent with our findings. Further findings by Milham (1988), and Groves et al. (2002), provide more data on HL cancers and RFR. The details follow.

### 3.4.1. Defense industry personnel in Israel

Peleg, 2009, reported five cancer cases linked to radio and radar exposure in the occupational setting of antenna ranges. There was a frequent and long term exposure to diverse forms of RFR at the site. The exposure was controlled to be within the occupational ICNIRP limits. The RR for all cancers was 8.3, p < 0.005. Out of those, three were HL (leukemia, lymphoma and plasmacytoma). We calculated PF of HL cancers as 60%, while 17% is expected for this group age and gender profile, p = 0.04. The new independent PF analysis supports the hypothesis of causation stated in Peleg (2009), based on high RR of all cancer types. Importantly, Peleg (2009), did not use self-referral, did not classify cancer types, and did not calculate PF; hence, bias related to self-referral or to selection of HL is not relevant to the new analysis of the (Peleg, 2009) data.

### 3.4.2. The Polish military sector

Szmigielski, 1996, reports a larger-scale research, encompassing the whole Polish military sector over 15 years. The exposure, as described in Szmigielski, 1996, was similar to that in the Stein et al. (2011) report; both cover a defense sector of a technologically-mature country. There were about 66 patients from the exposed group, as estimated by us (from Szmigielski, 1996 Table 1 and its "Results and discussion" section, $3700 \times 15 \times 119/100000 = 66$), out of which about 24 are HL (lymphomas of several types, leukemia of several types, myeloma and plasmacytoma). Szmigielski reports RR for all cancers as 2, p < 0.05, and RR = 6.3 for HL cancers, p < 0.001. We calculated that the proportion of HL cancers among exposed military personnel is PF = 36% out of the total cancers, while among all unexposed military personnel it is only 12%, which is similar to the value of the Israeli general population. The PF is calculated from Szmigielski (1996), table 1, by dividing the incidence of the HL cancers (the line before last in the table)

by that of all malignancies, last line in the table. We calculated the corresponding p-value of the HL PF as p < 0.001. Interestingly:

1. The PF in Szmigielski (1996), is similar to the 40% value in the Stein et al. (2011), group from a similar military/occupational setting.
2. The results in Szmigielski (1996), are roughly age and gender-adjusted because both the exposed and comparison groups are Polish military personnel. Furthermore, possible small differences in the age profiles between the exposed and the comparison groups cannot explain the extreme PF = 36% of HL cancers observed.
3. Our new PF analysis of the Szmigielski (1996), data provides an additional evidence against bias. Indeed, IARC (IARC, 2013) presented critics on Szmigielski (1996), based on age categories not being sufficiently detailed; Szmigielski (1996), stated that the full information was classified. Such critics are not applicable to the new PF result since the PF is calculated without layering into age categories relying on the age adjustment described in the point above.

### 3.4.3. Belgian radar technicians

Degrave et al., 2009, carried out a mortality study of cancer among Belgian radar operators. The exposure is estimated in (Degrave et al., 2009) and varies greatly with the exact position of the servicemen relative to radar transmitter in the range of 10 V/meter to 1300Volts/meter, thus exceeding the ICNIRP standards in some placements. Degrave did not calculate the exact individual exposures of the servicemen due to lack of records. Degrave identified the possible association of HL cancers and RFR. The results of Degrave et al., 2009, include rate ratios of HL cancer mortality rate of 7.2, CI95% = (1.09–47.9) among the studied radar operators relative to a similar non-exposed group of service personnel. There were 11 deaths from HL cancers and 133 deaths from all cancers in the radar battalions versus 1 and 72 respectively in the control battalions. Degrave analyzed causes of death many years after the exposure rather than incidence rates at diagnosis in the other three studies described above, so the resulting PF is different and not straightforward to compare to our results. From table IV in Degrave et al., 2009, the HL PF in the exposed group was 11/133 = 8.3%, while in the comparison group it was 1/72 = 1.4% where 11, 133, 1 and 72 are the numbers of deaths. Our analysis method, which is based on large CR-type control groups, cannot compute the p-value of the PF due to different type of control group in Degrave et al., 2009. The statistical significance is evident from the above RR and its CI as presented in Degrave et al., 2009.

Degrave et al. considered also the possibility of causation of the cancers by ionizing radiation emitted by the radar equipment. The correspondence (Telle-Lamberton, 2010),the reply (Degrave et al., 2010) and (Beyea et al., 2014) show that the confounding influence of ionizing radiation there was probably very weak.

#### 3.4.4. U.S.Navy, Korean War

Groves et al. (2002), reported mortality rates in three occupational subgroups of U.S. Navy veterans from the Korean conflict. In one highly exposed subgroup, namely electronics technicians in aviation squadrons, they found nonlymphocytic leukemia standardized mortality ratio (SMR) = 2.2, 95% CI:(1.3, 3.7). There was no increase in other cancer types. Robinette et al. (1980), table 6, reported a mortality ratio of 1.4 in fire control and in aviation electronics technicians from the same war much earlier, before it gained the statistical significance reported in Groves et al. (2002). Both studies identified elevated cancer rates in the groups with the high RFR exposure and not in the lower exposure groups. This finding suggests a dose-response relationship and therefore strengthens the case for causality with respect to occupational exposure.

#### 3.4.5. U.S. radio amateurs

Milham (1988), and Milham (1985), studied mortality rates among American radio amateurs using SMR and proportionate mortality ratio (PMR). Milham reported in the two papers a statistically significant increase in some HL cancers (leukemia, types of leukemia and of lymphoma, and possibly multiple myeloma). The radio amateurs are subject to whole-body exposure similarly to the military/occupational setting but with probably lower level and no radar pulses.

#### 3.4.6. Graphic summary of the results

Fig. 3 presents the main data described above. Each row of pie charts represents a different group of patients: Stein et al. (2011), Szmigielski (1996), Peleg (2009), and Degrave et al. (2009). The left pie chart in each row presents the PF expected in a group of patients from an unexposed population, with the age and gender profile matched to that of the exposed patients group. The right side shows the PF pie charts of the exposed group. The increase in PF indicates that the radiation exposure elevated the $PF_{obs}$ considerably; the low probability of this to occur by chance is quantified by the significant p-values. The RR of all cancers and of HL cancer, where available, are listed on the right.

## 4. Discussion

### 4.1. Association and causation

The data presented graphically in Fig. 3 show high HL cancer risk in four independent groups of people in three different countries exposed to RFR in the military/occupational setting. The risk is identified by a high conventional RR, high PF or both. The four groups were exposed to RFR by different types of equipment and were studied by different methods. The association of RFR with increased risk of HL cancers is evident in each group separately. Additionally, the trend of increase in HL cancers associated with whole-body exposure to RFR is also consistent with a report on radio amateurs and with a report on Korean war veterans (Groves et al. (2002)). In short, the findings are robust.

We are not aware of any hidden bias or influence other than causation that could explain the association of RFR and increased cancer
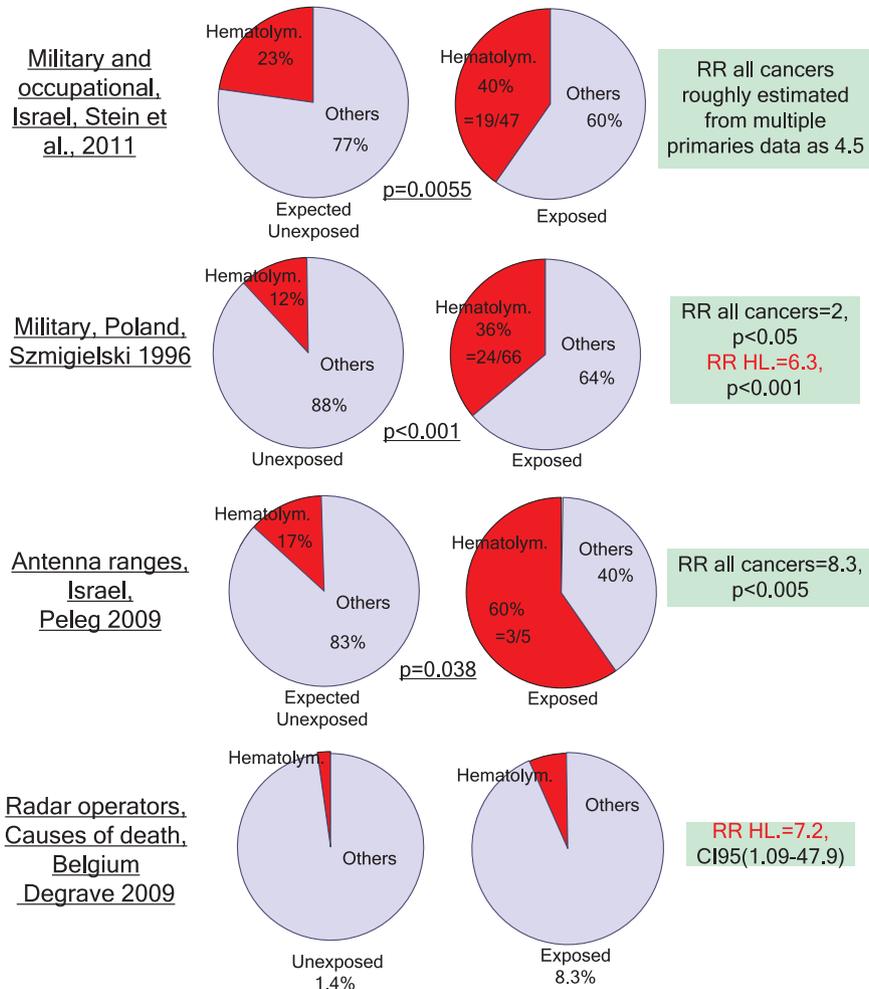


**Fig. 3.** *Overview of the main data.* PFs of HL cancers in the general population and in the patient groups: Stein et al. (2011), Szmigielski (1996), Peleg (2009), and Degrave et al. (2009). Left side: unexposed, age, and gender matched to the patients group; right side: exposed. Each of the four rows relates to a different patient group. The RR of all cancers and of HL cancer, where available, are listed on the right.

risk in at least four groups of patients — Stein et al. (2011), Peleg (2009), Degrave et al. (2009), Szmigielski (1996). This is because these groups were spread over three countries, were separated by many years, and used different radiation-generating equipment. Further, emission of some unknown carcinogenic chemical by the transmitters in all four places is unlikely. We are left with causation as the sole viable explanation of the association that we, and many before us — Stein et al. (2011), Szmigielski (1996), Degrave et al. (2009), Peleg (2009), Milham (1988) – have shown.

### 4.2. Assumptions and limitations

Our analysis accounts for the selection of patients being influenced by age and gender, but not by cancer type. The inclusion of the individual patients in the research (Stein et al., 2011) involved their personal decision to report their cases as possibly caused by RFR exposure. Such personal decision might affect, in principle, the type of cancer in this research; some may have been inclined to report some cancer types more than others. We think that such an influence would be possible but unlikely to produce strong effects because the fact that is important to the patient is the diagnoses and severity and not the type of cancer. We suggest that the non-elevated PF results on testicular cancers indicate that our method does not produce sporadic abnormal results. Furthermore, this problem would be relevant only to the Stein et al., 2011, study. Such a bias is impossible in Peleg (2009), Degrave et al. (2009), and Szmigielski (1996), in which there was no self-referral.

The multiple primaries result predicting overall RR of 4.5 in the Stein et al. (2011), case series is an approximation as explained in the "Materials and Methods" section above, since it uses the Poisson distribution following the Spratt model. Future refinements of the model that we used for the general population may modify somewhat the O/E ratio and the p-value; however, multiple primaries are not common in a normal young population; Jena et al. (2016), reported only 13 cases in a 5-year study. We surmise that the PF of multiple primaries observed in the case-series group is atypically high and suggests an increased cancer risk.

### 4.3. The patterns of cancer types in the occupational/military setting

The exposure characteristics in terms of power density, radio frequency, and waveform (pulses and modulations) varied considerably from group to group. It is known from research on rats (Chou et al., 1992) (Wyde et al., 2016) that the outcome in terms of cancer types is influenced by the waveforms. In Wyde et al., 2016, radiation-related cancer types were somewhat different for the CDMA (code-division multiple access) and GSM (Global System of Global Communications) waveforms and a similar earlier experiment (Chou et al., 1992, see table 2), using a different radar-like waveform, was associated with many cancer types, suggesting universal carcinogenicity. Therefore, the cancer characteristics may vary across groups. It is possible that different groups of patients with cancer from the military/occupational setting will exhibit different proportions of cancer types because of some variation in the RF exposure characteristics such as frequency, pulse-shape, peak to average power ratio (PAPR), duty cycle, or modulation. Additional cancer types were reported as possibly associated with RFR whole-body exposure, e.g., head and neck cancers indicated but not classified in Stein et al. (2011), and Peleg (2009); testes in Davis and Mostofi (1993), and Finkelstein (1998); melanoma skin cancer in Finkelstein (1998); breast cancer in Kliukiene et al. (2003); and esophageal, stomach, colorectal, nerve and brain cancers in Szmigielski (1996).

The cancer types prevalent with the use of mobile phones are those of the brain (Hardell and Carlberg, 2013), of the acoustic nerve, and some others. Those cancer types may be a consequence of the mobile phone RFR being stronger in the user's head region. In the

occupational/military setting, whole-body exposure is common, with all organs and tissue types affected; therefore, additional cancer types may be expected as the HL cancers studied in this work.

### 4.4. Relation to brain, head and neck cancers in cell-phones users

Our findings on increased risks for HL cancers from whole-body exposure to RFR complement the findings of Hardell and Carlberg, 2013, Sadetzki et al. (2008), Momoli et al. (2017) and references within, on increased risks for head and neck cancers from cell phones. In summary, the two sets are pieces of the same puzzle. A similar pattern of electromagnetic fields causing both HL and brain cancers is emerging also at ELF. Magnetic fields at ELF were classified as a possible carcinogen by IARC in 2002 mainly due to leukemia in children; Carlberg. et al., 2017 recently reported an increased risk of glioblastoma multiforme linked to ELF electromagnetic fields.

The scenario for cell phone users (Hardell et al., 2013; Carlberg and Hardell, 2017) is a small increase in individual risk applied to many users, whereas in occupational settings, the scenario appears to be a large increase in individual risk applied to relatively small numbers of individuals exposed, as explained by Szmigielski.

## 5. Conclusion

We have presented evidence supporting the case for a cause-effect relationship between radio and radar radiation and HL cancers in occupational/military settings.

Our case series showed an increased PF for HL cancers relative to all cancers. The high PF for multiple primaries adds to the case for a cause-effect relationship in those occupationally exposed. Three previous observational population-based cohort studies showed a uniquely high increased risk for HL cancers in three countries. Similar outcomes were reported among radio amateurs. Supporting evidence comes from epidemiological studies on brain and salivary gland cancers in humans such as (Coureau et al., 2013), (Carlberg and Hardell, 2017), (Hardell et al., 2015), (Sadetzki et al., 2008); animal experiments such as (Chou et al., 1992) and (Wyde et al., 2016); experiments on human cells such as (Friedman et al., 2007); and physical principles e.g. (Vistnes et al., 2001), (Barnes et al., 2015), and (Peleg, 2012). Our findings on occupational exposures and HL appear to satisfy the view-points and suggestions of causality by Bradford Hill (Hill, 1965), as was the case of cell phone and brain cancer reviewed by Carlberg and Hardell, 2017.

While complete measurements of RFR exposures were not available and rough exposure assessments from patients interviews and from partial exposure data were used instead, we have demonstrated increased HL cancers in occupational groups with relatively high RFR exposures. Our findings, combined with other studies, indicate that exposures incurred in the military settings evaluated here significantly increased the risk of HL cancers. Accordingly, the RFR military exposures in these occupations should be substantially reduced and further efforts should be undertaken to monitor and measure those exposures and to follow cohorts exposed to RFR for cancers and other health effects.

The HL cancer risks from the four patient groups reported here show that the ICNIRP radiation limits do not guarantee human safety in the occupational/ military settings and should be replaced by biologically based guidelines accounting for the accumulated knowledge.

Overall, the excess risk for HL and other cancers in occupational groups complements the findings of brain tumors in cellphone users. These epidemiologic findings together with experimental studies on RFR and carcinogenicity make a coherent case for a cause-effect relationship. We are unable to find alternative explanations. We endorse Hardell's call for classifying this exposure as an IARC group 1 carcinogen and for updating the exposure standards.

## Appendix A

This appendix is intended for those interested in verifying and extending the analysis. The appendix includes equations not written explicitly in the "Materials and methods" section and a reference to a standard MATLAB program used to compute CI.

### A.1 Notation

See the "Notation" subsection at the beginning of the "Statistical analysis methods" section above. The additional notation used in this appendix follows.

The number of age-gender categories is denoted $K$, the categories are indexed by $k$. Number of cancer patients in the cancer register is $N^C$, out of which $N^{CH}$ have HL cancers. The corresponding numbers in each age-gender category $k$ are $N_k^C$ and $N_k^{CH}$, respectively.

The numbers of patients in the case series is $N$, out of which $N_k$ are in category $k$. $N_k^H$ patients in category $k$ have cancer of the HL type. The number of patients with HL cancer in the case series is denoted $N^H$ in this appendix and $N_H$ in the paper body.

The binomial PDF is denoted

$$P_b = B(n_1, n, p) \tag{5}$$

Where $P_b$ is the probability that $n_1$ out of $n$ Bernoulli trials will yield a "yes" result where $p$ is the probability of the "yes" result in each Bernoulli trial.

### A.2 Mean percentage frequency (PF$_M$) of cancer types expected in the case series group of patients

Given the patient data of the case series and the CR data, the PF$_M$ is computed following Boyle and Parkin (1991) (eq. 11.29):

$$PF_M = \sum_{k=1}^{K} \frac{N_k^{CH}}{N_k^C} \frac{N_k}{N} \tag{6}$$

### A.3 Computing p-values

The p-values are computed under the null hypothesis.

The PDF of having $N_k^H$ patients with HL cancer type in the $k$ category of the case series group is binomial:

$$P(N_k^H) = B\left(N_k^H, N_k, \frac{N_k^{CH}}{N_k^C}\right); \quad N_k^H = 0, 1, \dots N_K \tag{7}$$

Now since

$$N^H = \sum_{k=1}^{K} N_k^H \tag{8}$$

the PDF of having $N^H$ patients with HL cancer type in the whole case-series group is convolution of $P(N_k^H)$ from all the categories

$$P_t(N^H) = P(N_1^H) \otimes P(N_2^H) \otimes \dots P(N_K^H) \tag{9}$$

The convolution operator $\otimes$ is applied sequentially, starting with $P(N_{k=1}^H)$, convolving it with $P(N_{k=2}^H)$, convolving the result with $P(N_{k=3}^H)$, and so on. The convolution operation between a pair of PDFs $P_1(M_1)$ and $P_2(M_2)$ with non-negative integer arguments $M_1$ and $M_2$ is:

$$P_1(M_1) \otimes P_2(M_2) = P(M_1 + M_2 = M)$$
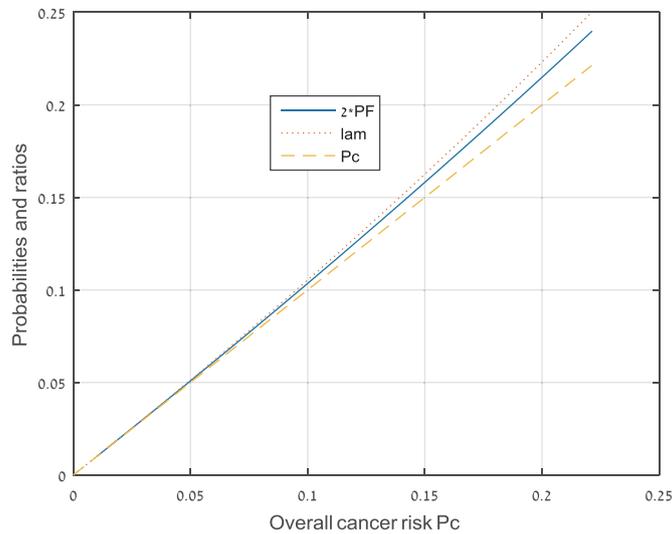$$= \sum_{M_1=0}^{M} P_1(M_1)P_2(M - M_1) \tag{10}$$

Fig. A.1. The accuracy of prediction of the overall cancer risk $P_C$ in a population by twice the PF of multiple primaries. $\lambda$ is denoted by "lam".

The p-value is the probability of having at least $N_1$ patients with the HL cancer type in the case series. The p-value is then a sum of the terms in (9) as follows:

$$pvalue \overset{def}{=} P(N^H \geq N_1) = \sum_{N^H=N_1}^{N} P_t(N^H)$$

(11)

When computing p-value on the data in Szmigielski (1996), we use a single age-gender category comprising all the patients following the data structure of Szmigielski (1996). With $K=1$ the convolution (9) leaves the PDF unmodified, and our results are identical to the single-tailed Binomial test.

Note that the $PF_M$ in (6) was not used in the calculation of the p-value. One may wonder why the following simpler method to compute the p-values was not used:

1. Compute the $PF_M$ expected in the case-series group using (6).
2. Compute the p-value using a standard single-tailed binomial test on the whole case series using only $PF_M$, $N$ and $N^H$.

We tested this simpler approach, and it yields p-values very similar to those obtained by the full procedure we presented and used. The problem with the simpler approach is that to be rigorous, $PF_M$ in (6) should be a real probability. The term $\frac{N_k}{N}$ in (6) must then be assumed to be not only a ratio of two numbers, but also a probability that a patient in the case series belongs to category $k$. This is a good approximation, but it had to be validated by a Monte Carlo test; hence, the simpler approach, while valid, was more cumbersome to present rigorously. Therefore, we preferred the full procedure above.

*A.4 The confidence Interval (CI) of PF*

Recall that $PF$ is the probability of a patient with cancer from the exposed population to have a HL cancer type. In principle, it is a weighted average of the unknown PFs in the age categories in the exposed population similarly to Eq. (6) which describes the unexposed population. We estimate $PF$ and compute its CI from $N^H$ and $N$ and do not attempt to estimate the PF in each age category of the exposed group since not enough data exists for such a detailed study. Under this view, the cancer type of any patient from the exposed population, HL or not HL, is the result of a Bernoulli trial with probability of the HL outcome being $PF$. Thus, the PDF of the number $N^H$ of patients with HL cancer in the case series is binomial

$$P(N^H) = B(N^H, N, PF); \quad N^H = 0, 1, \dots N$$

$PF$ is estimated by applying Eq. (1), and its CI is computed by standard procedure based on the binomial PDF above. We used the program berconfint.m from the Matlab communications toolbox. We verified that berconfint.m reproduces the results of Peleg (2009), which did not use this program.

Note that the CI analysis is done without conditioning on the ages and genders of the patients leading to binomial PDF of $N^H$, while the p-value analysis is performed with conditioning on age and genders leading to PDF which is a convolution of binomial distributions. This is compatible with the definitions of the p-value and of the CI here and in the "Materials and methods" section.

*A.5 Analyzing multiple primaries*

We explain here why the PF of multiple primaries is a predictor of overall cancer risk. Recall Eq. (4):

$$P(0) = e^{-\lambda}$$
$$P(1) = \lambda e^{-\lambda}$$
$$P(2) = 0.5\lambda^2 e^{-\lambda}$$

where λ is the expected number of all primaries in the group of initially healthy people.

From the above

$$\frac{P(2)}{P(1)} = 0.5\lambda$$

We start with an intuitive reasoning. The left-hand side of the last equation is an approximation of the PF of multiple primaries, and λ on the right-hand side of the equation is a fair approximation of the overall cancer risk. Hence, 2*PF* should be a good predictor of the overall cancer risk.

The exact relationships are derived easily from Eq. (4) as follows. The overall cancer risk is $P_C = 1 - P(0)$, and the PF of multiple primaries is PF = $[P_C - P(1)]/P_C$. We plotted these relationships in Fig. A.1. It is evident from the figure that the overall cancer risk $P_C$ in a population is predicted well by twice the *PF* of multiple primaries in a patient group coming from this population.

## Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.envres.2018.01.003.

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