# Melanoma Incidence and Frequency Modulation (FM) Broadcasting

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**ABSTRACT. The incidence of melanoma has been increasing steadily in many countries since 1960, but the underlying mechanism causing this increase remains elusive. The incidence of melanoma has been linked to the distance to frequency modulation (FM) broadcasting towers. In the current study, the authors sought to determine if there was also a related link on a larger scale for entire countries. Exposure–time-specific incidence was extracted from exposure and incidence data from 4 different countries, and this was compared with reported age-specific incidence of melanoma. Geographic differences in melanoma incidence were compared with the magnitude of this environmental stress. The exposure–time-specific incidence from all 4 countries became almost identical, and they were approximately equal to the reported age-specific incidence of melanoma. A correlation between melanoma incidence and the number of locally receivable FM transmitters was found. The authors concluded that melanoma is associated with exposure to FM broadcasting.**
<Key words: environmental, epidemiology, FM broadcasting, malignant, melanoma, population>

DOLK ET AL.[1] found that there was significant decline in skin and bladder cancer incidence among adults in England as distance from a frequency modulation (FM) broadcasting tower increased. In the second part of the study, these investigators reported a similar trend for individuals who lived various distances from FM and TV towers, but the effect was less pronounced than in the first study.

In the current study, we sought to determine if the findings of Dolk et al.[1] on a local scale generally held true for a country as a whole. If the findings held true, our second objective was to address possible improvements that could be initiated to reduce risk.

We conducted the study in several steps. We initially addressed the survival probability of the general population to correctly account for natural deaths in the data analysis. We then analyzed independent sets of exposure and incidents data from 4 different countries to extract each individual exposure–time-specific incidence function (Etsi). Published sets of age-specific incidence data (ASI) were reviewed for different years. Finally, the numbers of radio stations that were locally receivable were correlated with the melanoma incidence for the 288 communities in Sweden.

## Method

The hypothesis we tested was that the increase in melanoma incidence over the years could be explained entirely by the expansion of the FM broadcasting network. If true, we should be able to calculate the number of cases each year on the basis of some publicly known facts: (1) the total exposure history of the population (i.e., number of individuals covered by the expanding FM broadcasting network each year); (2) the survival probability of the population (for the correct accounting of the number of exposed individuals remaining alive over time); and (3) age-specific incidence data, which we used to determine the probability of contracting melanoma vs. exposure time.

**1. Survival statistics.** As time passes, exposed individuals will die from many causes, and they will be replaced by newborns. The out-dying function was calculated based on survival data and the age distribution within the Swedish population. The function is different from that of newborn babies inasmuch as it is based on a mixed population: from 1 to 90 yr of age (Fig. 1). One must consider this function if the number of incidents by year are to be calculated correctly, given that people who die will no longer contribute to the incidence statistics.

**2. Exposure–time-specific incidence.** It is necessary to know the exposure–time-specific incidence when a possible effect from a continuous environmental stress is to be estimated. The ASI for melanoma in Sweden is shown in Figure 2. ASI increases from the age of puberty (as it also does in Norway). It is assumed that an adult population follows the ASI curve (i.e., from 12 yr of age) immediately upon start of the exposure.

If the measured incidence is a function of exposure time only and the natural high-age incidence without the environmental stress is low, then the ASI should approach a constant level at high ages. This is so because old people have not been exposed for a longer time than has elapsed since the start of the exposure, irrespective of their age. Consequently, we expected this constant level to increase by calendar time.

**3. Calculation of annual cases.** The annual numbers of the disease in a country can be calculated by multiplying each year's number of exposed people by the Etsi and by the relevant survival function. The response from all exposed people is then added to give the total number of incidents per year. The formal mathematics is provided in the Appendix.

## Results

1. A literature study revealed a link to the distance from an FM tower[1] (as was mentioned earlier). We also found a report that claimed that cancer could develop faster in a radiofrequency (RF) field.[2] Cancer incidence

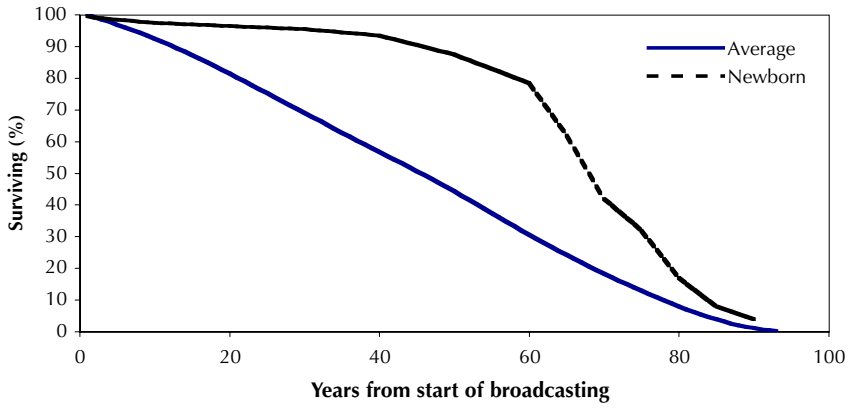


**Fig. 1. Survival function for the average population (solid line) and for newborn babies (dashed line).**

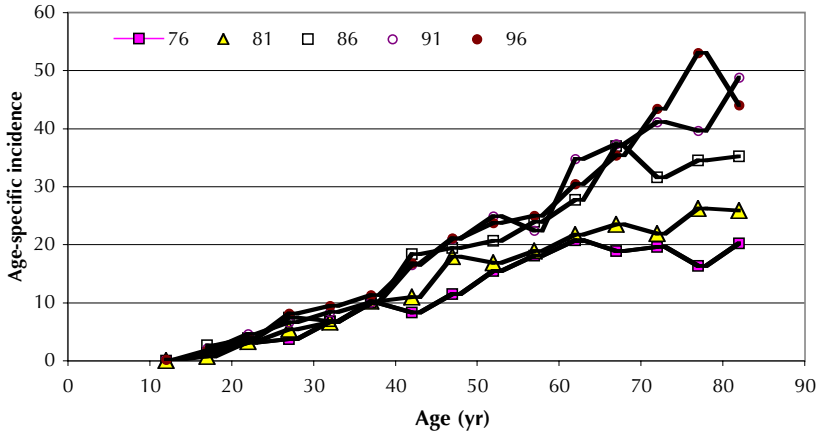


**Fig. 2. Age-specific incidence of melanoma in Sweden, 1979–1996. As was expected, the high-age flat level is increasing by each year. The top curve (1996) was used for the calculation of the response in Sweden and Denmark.**

has also been associated with proximity to television towers.[3] Other related reports were presented by Andersson et al[4] and by Westlund et al.[5]

2. Data on the expansion of the FM broadcasting networks were collected for the United States (i.e., via Internet), Norway[6] (i.e., direct contact with NRK [Norwegian Broadcasting]), Sweden,[7] and Denmark.[8] We also collected information on the frequencies used for FM in different countries. Western Europe and the United States have been using 87–108 MHz, whereas the former communist countries have used lower frequencies (i.e., approximately 70 MHz [Fig. 3]). Japan also uses a lower frequency than Western Europe and the United States. Subsequent to 1992, many Eastern Europe countries have transformed to the "Western" band of 87–108 MHz.

3. The procedure we used to calculate the melanoma incidence in Norway, Sweden, Denmark, and the United States is shown in Figure 4 for Norway.

4. The calculations were based on the age-specific incidence reported in Norway (for Norway and the United States) and Sweden (for Sweden and Denmark), and they were not the result of curve-fitting. We correlated all calculated and measured incidence of melanoma, expressed in ppm per year of the respective populations. The correlation was expressed by $R^2$ = 0.9788, $\beta = 0.989$, and $p < .0001$.

5. For all 4 countries, the cumulative Etsi could be modeled[9] by a log-normal distribution, sigma (base 10) of 0.52–0.59, and a time to 0.1% around 20 yr (Fig. 5).

6. The melanoma incidences and the average FM power densities in the 27 different counties of Sweden are shown in Figure 6.

7. It was also of interest to us to determine if the total

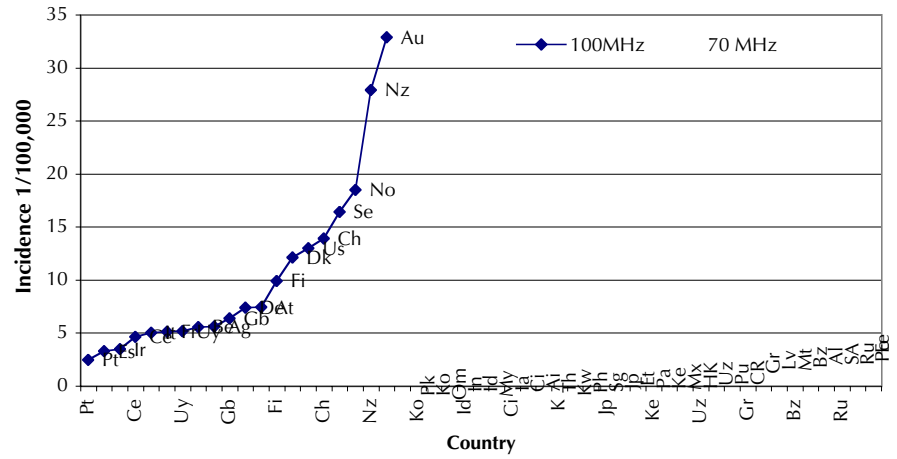


**Fig. 3. Melanoma incidences in different countries vs. frequency modulation, as was reported in 1996. Some of the countries marked as "70 MHz" have unknown frequencies.**

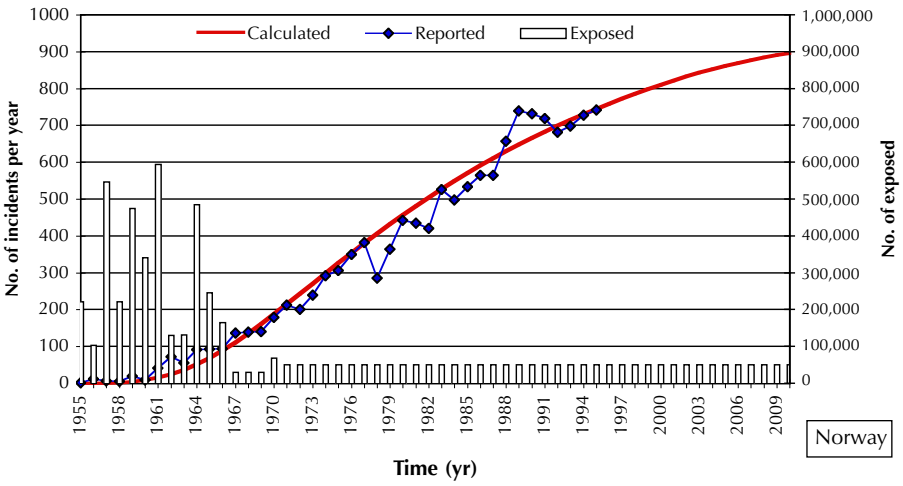


**Fig. 4. Reported and calculated numbers of melanoma cases per year in Norway. The numbers are on top of a "natural" level of 100 cases per year that is not shown in the graph.**

power density and the number of main FM stations available at a location related to the local melanoma incidence. Therefore, the incidence numbers in all 288 communities were collected[10] for 1992–1996, and the average values were established. The radiated power densities from all main FM transmitters in Sweden[11] and Norway, and for a few neighbors in Denmark and Norway, were superimposed and displayed on maps. These maps resembled maps showing melanoma incidences over Sweden and Norway.

8. The incidences of melanoma in all communities were plotted against the number of receivable FM transmitters, as described elsewhere[11] (see box plot in Fig. 7).

Using multiple regression analysis, we found a linear relationship between the incidences reported in the 288 Swedish communities and radiated power density, field strength, and the number of transmitters covering each community. The $p$ value for power density was .019; for field strength, $p = .0035$; and for number of transmitters, $p < .001$ ($R = .5592$; adjusted $R^2 = .3079$).

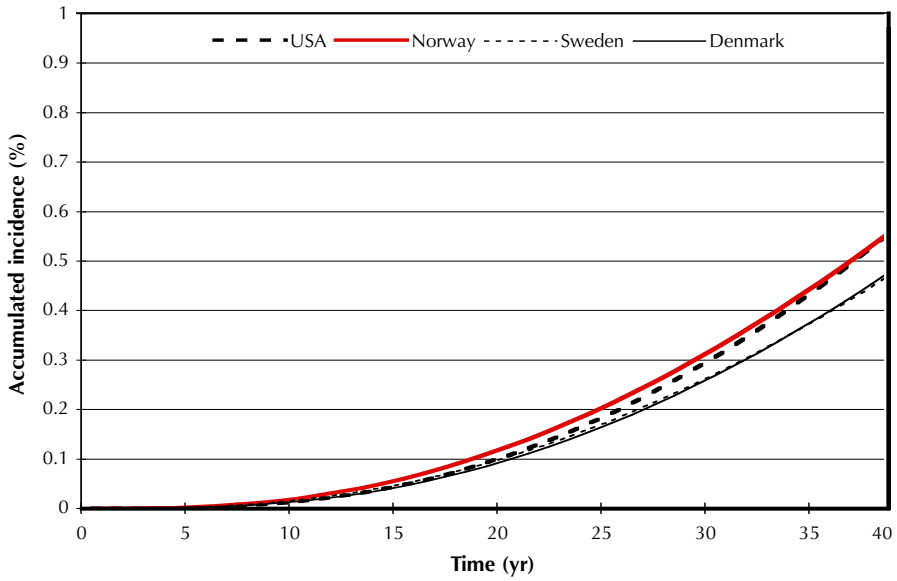


**Fig. 5. Accumulated incidence vs. exposure time in the United States, Norway, Sweden, and Denmark.**

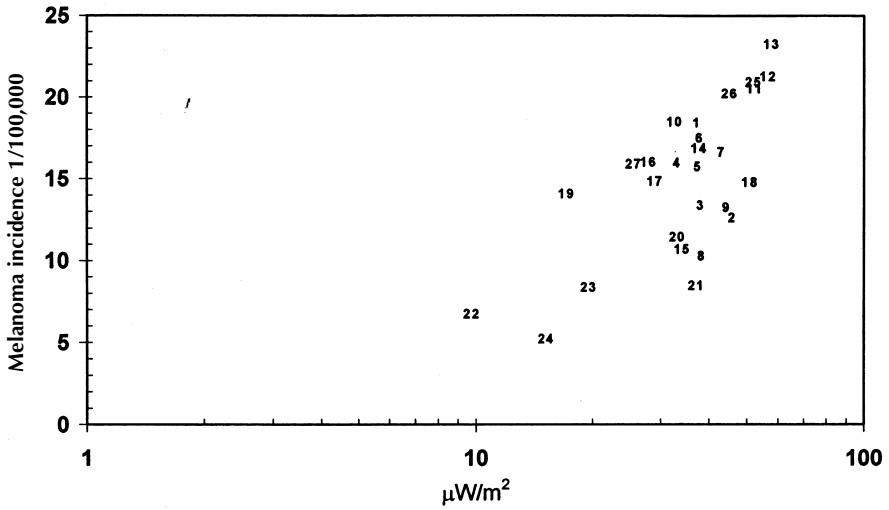


**Fig. 6. Melanoma incidence and power density from the main frequency modulation broadcasting towers in the 27 different counties of Sweden. The power density is simply calculated as the total emitted power within 1 county divided by the area of that county. Numbers refer to the county numbering used in reference no. 14.**

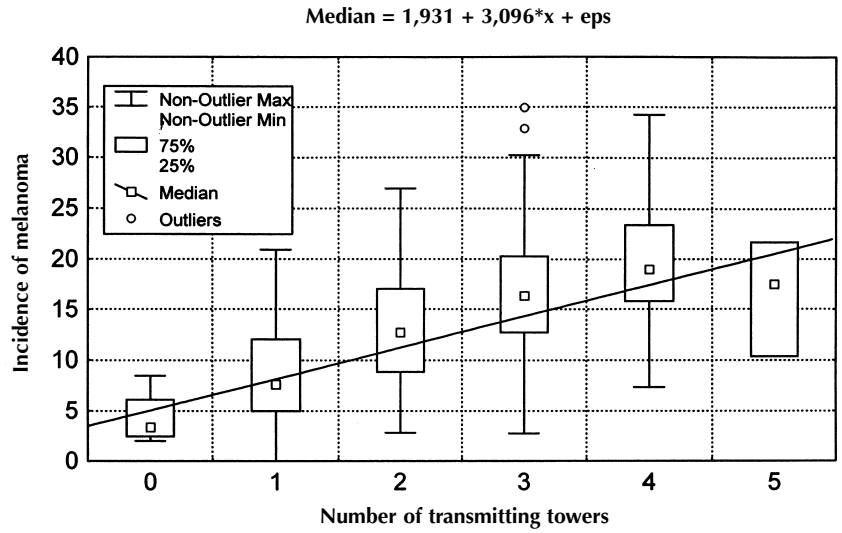


**Fig. 7. Box plot of melanoma incidence reported in 288 Swedish communities vs. number of frequency modulation (FM) transmitting towers that can be received in each community. The incidence (= 2) at zero transmitters is taken from the average incidence in Sweden before 1955 when no FM transmitters were available.**

During the study, we noticed that asthma prevalence increased[12] in a fashion similar to that of melanoma incidence in Sweden. A similar drastic increase was noticed in Finland among candidates for military conscription, with diagnosis of asthma at or before call-up medical examination.[13] The mortality in Sweden[14] also showed a trend break after 1955.

## Discussion

The human body has a resonance frequency to RF fields that can be described as follows[15]:

$$Fr = 11{,}400/L \quad [MHz],$$

where L is the effective body length in cm. The 85–108 MHz corresponds to lengths of 105–135 cm, which corresponds to arms, legs, and the main body of most adults. A lower broadcasting frequency will require longer arms, legs, and body to get into resonance. All countries we examined that have been using lower FM broadcasting bands have also reported low melanoma incidences. Given that most of the main FM transmitters use horizontally polarized transmission, the strongest reception might occur at night (i.e., during sleep). The probability of a "good" reception will increase with the number of surrounding transmitters.

The correlation between power density and incidence is less than that between the number of transmitters and incidence. Even small power densities seem detrimental, and a "safe" level has not been identified in this study. Power density levels as low as 30 $\mu W/m^2$ cannot be regarded as safe (Fig. 8). This value is much lower than the International Commission on Nonionizing Radiation Protection level of 4,500,000 $\mu W/m^2$ or the recent Swiss level of 42,000 $\mu W/m^2$.

Radio–frequency-induced currents at resonance may become highest in the central parts of the body,[15] where melanoma of the skin is predominantly found.[16] It appears that the density of moles on the body follows the same pattern.

Our model predicted that 320 children younger than 20 yr should have developed melanoma in Sweden between 1958 and 1992. The reported total number was 287.[17] The model also predicted a stable incidence rate of 13 cases of melanoma for children younger than 20 yr of age, for each year after 1975 in Sweden. The actual reported figures vary between 5 and 20.

It is postulated that the East European countries that have introduced the 88–108-MHz band will show an increasing melanoma rate for the next 10 yr. The outcome in Estonia is predicted in Figure 9. Mortality from melanoma of the skin in Estonia[18] is also plotted. The modest increase since 1968 will likely accelerate after the introduction of FM at the 88–108-MHz band in 1992.

The melanoma response in the United States was calculated for 3 cases of exposure history. In one case, it was assumed that the entire population was exposed suddenly from 1955 onward. In another, it was assumed that the whole population was exposed suddenly from 1974 onward. In the third calculation, it was assumed that the exposure was developed in accordance with the build-up of the FM broadcasting network. All 3 responses are shown in Figure 10, together with actual reported numbers. Obviously, the exposure profile according to the FM expansion matches the reported incidents.

**Cell effects.** Investigators assume that ultraviolet (UV) light is the most important factor for the initiation of malignant melanoma. Considerable controversy sur-

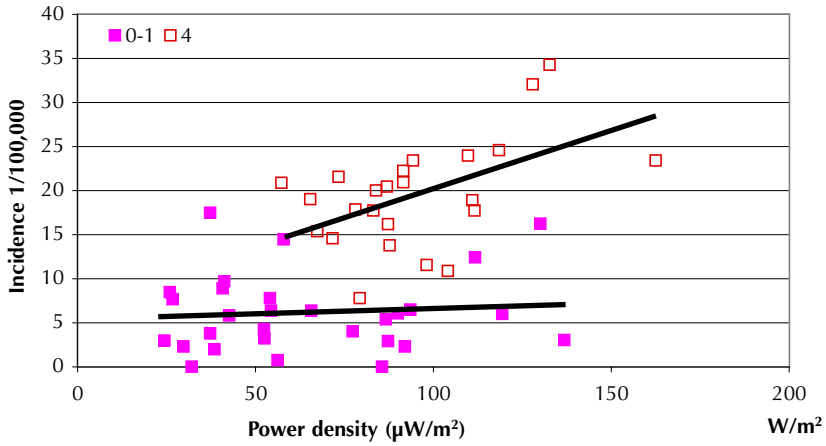


**Fig. 8. Melanoma incidences in communities having 0–1 and in communities having 4 receivable frequency modulation (FM) transmitters plotted vs. the total power density.**

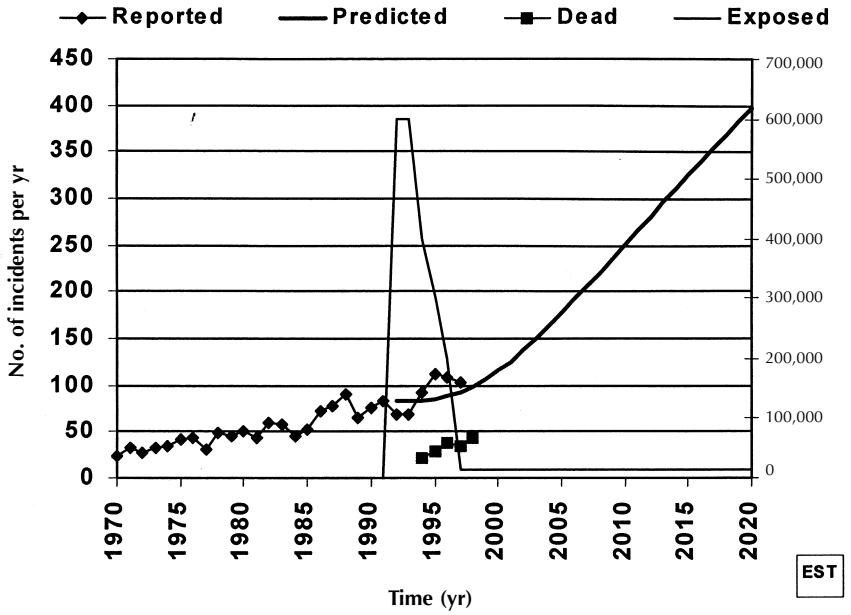


**Fig. 9. Reported and expected future development of melanoma incidents[19] in Estonia. Also included are reported deaths subsequent to 1994, as reported in reference 18. The peak in the center gives the annual number of people who became exposed, right scale.**

rounds the question of possible harmful effects of low- or high-frequency electromagnetic fields (EMFs). Animal experiments showed disturbances in endogenous opioid systems, with secondary effects on cholinergic ones, following exposure to low-frequency EMFs.[20] Pulsed EMFs imposed on developing chick embryos brought about an increase in the frequency of abnormal development.[21] Mammalian cell lines respond to extremely low-frequency EMFs, with an increase in proliferation rates,[22] as well as an increase in calcium influx.[23] EMFs also promote peripheral nerve regeneration, both in vivo and in vitro.[24–29] Lai and Singh[30] showed that brain cells exposed to pulsed (i.e., 2 ms pulse width, 500 pulses/sec) or to continuous-wave 2,450-MHz microwave electromagnetic radiation produced deoxyribonucleic acid strand breaks. Therefore, the data suggest that EMFs can directly affect biological systems.

Donnellan et al.[31] have shown clear-cut effects on a mast cell line of EMFs at 835 MHz. The rate of DNA synthesis and cell replication increased, actin distribution and cell morphology became altered, and the amount of β-hexosaminidase released in response to a calcium ionophore was enhanced significantly, compared with unexposed cultures. There were no effects

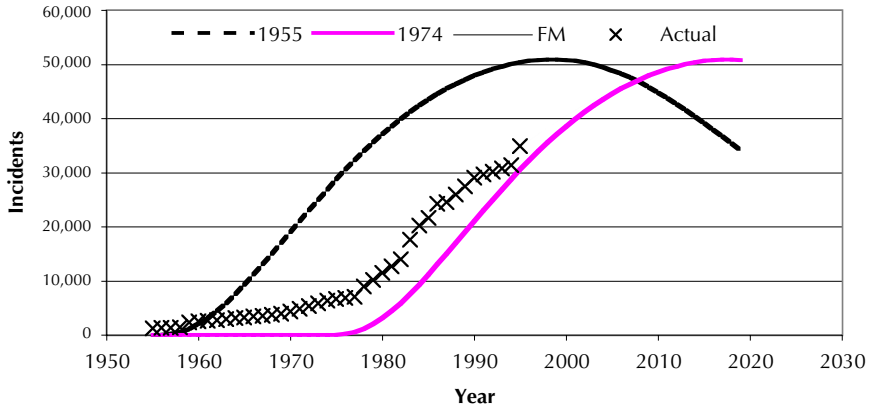


**Fig. 10. The melanoma response in the United States was calculated in accordance with 3 assumptions: (1) the entire population was exposed in 1955, (2) the entire population was exposed in 1974, or (3) exposure occurred in accordance with the frequency modulation exposure profile. The actual reported numbers were also plotted for purposes of comparison.**

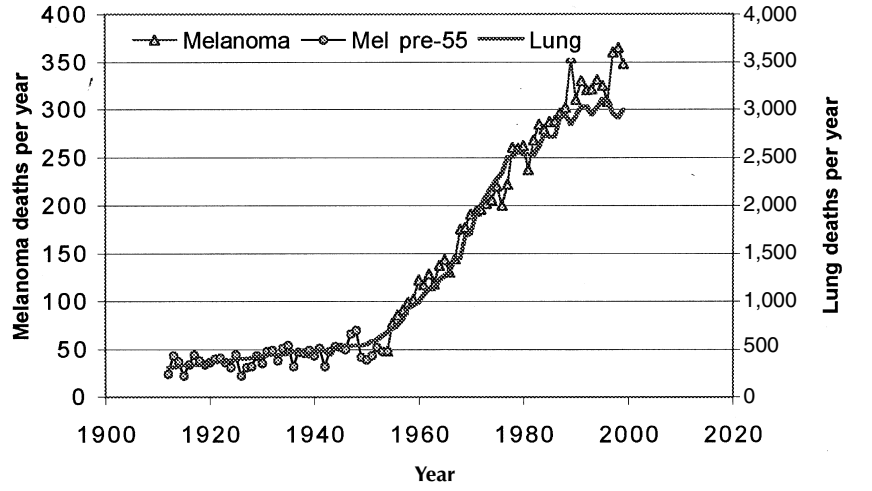


**Fig. 11. Number of individuals killed per year by melanoma of the skin and by lung cancer in Sweden subsequent to 1912.**

seen on the levels of cytoskeletal protein synthesis or β-actin messenger ribonucleic acid. However, the amount of Ras in the membrane fraction of exposed cells increased. These morphological changes persisted following subculture for at least 7 days in the absence of further exposure.[31] A recent theoretical paper has disclosed the mechanisms involved.[32]

**Possible confounders.** In both Sweden and Norway, there exists some correlation between latitude and melanoma incidence. At first glance one might relate this to differences in UV exposure. However, both the radiated power density and the number of overlapping transmitters also show a correlation to latitude. Given that Norway and Sweden are more densely populated in the southern parts than in the northern parts, this leads to higher power and transmitter density in the southern parts.

Investigators have attempted to relate melanoma incidence to traffic increases. The total car population growth since 1935 was related to reported melanoma incidence in 3 different countries. It appeared that the 3 Etsi functions were not similar, and, therefore, are dissimilar to the data in Figure 10. We found no indication that traffic expansion is causing melanoma. A similar experiment could be done easily, and one could determine if asthma correlates with an increase in traffic.

Investigators have questioned[33] whether increased surveillance of melanoma is the solution or the problem. Subsequent to 1996, the evidence gathered, including the findings of our study, gives several reasons for one to believe that the increasing incidence of melanoma is real. As an example, the annual numbers of individuals who have died from melanoma in Sweden appear in Figure 11.

## Conclusions

The results from this analysis support the findings of Dolk et al.[1] and show that melanoma incidence is cor-

related with public FM broadcasting—not only on a local scale, but also for entire countries.

The results of the analysis also show that melanoma incidence is a function of the exposure time to FM broadcasting and a function of the number of active FM stations available at each location. We predict that the East European countries that adopted the 87–108-MHz band in 1992 will show increasing melanoma rates. These frequencies should be avoided for public broadcasting.

An inexpensive precaution is to turn one's bed into the direction of the weakest reception of a horizontal FM radio antenna. Obviously, additional research on the effects of EMFs is needed.

### Appendix

The following definitions are made:

$\mu_1(j)$ = survival probability of the general population after $j$ yr,
$\mu_2(j)$ = survival probability of a newborn baby after $j$ yr,
$h_1(j)$ = incidence after $j$ yr of exposure of the general population,
$h_2(j)$ = incidence after $j$ yr of exposure of newborn babies (= age-specific incidence data),
$N_1 i$ = number of individuals in the general population who were exposed in year $i$,
$N_2 i$ = number of newborn babies who became exposed in year $i$,
$n_{i,j-i}$ = $N_i * \mu(j - i) * h(j - i)$ = number of incidents in year $j$ of those exposed from year $i$, and
${}_0\Sigma^j n_{i,j-i}$ = the total number of incidents in year $j$.

The table below shows how the numbers are calculated and added together to give the total number of incidents, by each year. One has to calculate $n_{i,j-i}$, which is based on both the sudden exposure of the general population (e.g., during the 1950s and 1960s) and the continuous supply of newborn children as defined above.

**Calculation of Annual Incidents on Top of the "Natural" Level that Was Reported before the Exposure Started**

| Year | Exposed | 1955 | 1955+1 | 1955+2 | 1955+3 | ...1955+$j$ |
|---|---|---|---|---|---|---|
| 1955 | $N_0$ | $n_{0,0}$ | $n_{0,1}$ | $n_{0,2}$ | $n_{0,3}$ | $n_{0,i}$ |
| 1956 | $N_1$ | | $n_{1,0}$ | $n_{1,1}$ | $n_{1,2}$ | $n_{1,j-1}$ |
| 1957 | $N_2$ | | | $n_{3,0}$ | $n_{3,1}$ | $n_{3,j-2}$ |
| | ... | | | | ... | ... |
| 1955+$i$ | $N_i$ | | | | | $n_{i,j-i}$ |
| | ... | | | | | |
| Incidents | $n_{1,0}$ | | ${}_0\Sigma^1 n_{i,2-i}$ | ${}_0\Sigma^2 n_{i,3-i}$ | ${}_0\Sigma^3 n_{i,3-i}$ | ${}_0\Sigma^j n_{i,j-i}$ |

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