Table 1 gives average values obtained for sections taken from the cortical portion of the femur and from trabecular bone obtained from rib, vertebrae and the head of the femur. The perimeter, measured in centimetres on the two dimensional microradiograph, includes periosteal, endosteal and Haversian canal surfaces, but does not include lacunar or canalicular surfaces. The bone area includes all bone within the periosteal and endosteal surfaces exclusive of the Haversian canals.

Table 1. PERIMETER/AREA (EXPRESSED IN CM/CM²) OF DIFFERENT BONES IN THE HUMAN SKELETON

Cortical bone	
Mid femur	30
Trabecular bone	
Head of femur	75
Vertebra (thoracic)	90
Vertebra (lumbar)	90
Rib	90

The values reported here for the vertebrae agree with those reported by Bromley *et al.*[5] and Dunnil *et al.*[6]. Values given by other authors, however, both for cortical and trabecular bone, vary by a factor of 20 (refs. 7 and 8).

From the figures in Table 1, we can estimate the surface/volume ratio (S/V) for the whole skeleton if we assume that the skeleton is composed of 80 per cent cortical and 20 per cent trabecular bone:

$$S/V = \frac{80}{100} \cdot 30 + \frac{20}{100} \cdot 90 = 42 \text{ cm}^2/\text{cm}^3$$

This can be converted to total skeletal surface if we assume that bone density is 2 g/cm³ and the weight of the fat-free skeleton is 4 kg. The total surface is then 8.4 m².

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by Mallard & Whittaker Lam

Dielectric Absorption of Microwaves in Human Tissues

An earlier publication from this laboratory[1] presented the results of calculations on the propagation of 3 cm plane microwaves in a semi-infinite plane stratified slab of tissues to simulate, in a simple way, the effect of the human body on the beam.

In that communication the results of attenuation coefficient measurements on various tissues reported by England and by England and Sharples[3] were used. It has since been realized that these attenuation coefficients were amplitude attenuation coefficients and not power attenuation coefficients as had previously been assumed. As a result, the numerical results of the calculations are incorrect but the essence of the conclusions made remains the same.

The calculations have been repeated by an improved method, using a digital computer and the correct attenuation coefficients. In addition, the data published by England[3] were used to extend the calculations to two other frequencies.

The model used consisted of a slab of normal tissue bounded by a 1 mm layer of skin on each side. A slab of tumour tissue (1 cm) was situated at various positions inside the normal tissue.

Because the model is so simple, the results are intended only to provide a very rough indication of the results that could be expected *in vivo*. In particular, it must be emphasized that the plane nature of the model produces interference effects arising from multiple reflexions between parallel interfaces which can hardly be expected *in vivo*; diffraction and refraction effects would be very important for small tumours in a real anatomical situation.

The values of propagation constant (β radians/cm) and amplitude attenuation coefficient (α nepers/cm), which were used in the calculation, are given in Table 1.

Table 1. VALUES OF PROPAGATION CONSTANT AND AMPLITUDE ATTENUATION CONSTANT USED IN THE CALCULATIONS

λ	Constant	Fat	Muscle	Skin	Tumour
3·18 cm	α Nepers/cm	0·49	2·6	1·65	[illegible]
	β Radians/cm	4·0	11·7	12·1	12·5
10 cm	α Nepers/cm	0·20	Not available	0·73	0·67
	β Radians/cm	1·58	but taken	[illegible]	[illegible]
1·27 cm	α Nepers/cm	1·5	as the same	[illegible]	[illegible]
	β Radians/cm	8·8	as skin	24·8	[illegible]

Table 2. EFFECT OF TUMOUR ON TRANSMITTED POWER

Model	λ	1·27 cm	3·18 cm	10 cm
10 cm total thickness, 1 mm thick skin, 1 cm tumour centrally placed in fat	T	2×10^{-4}	[illegible]	[illegible]
	Q_T	3×10^{-2}	5×10^{-2}	[illegible]
10 cm total thickness, 1 mm thick skin, 1 cm tumour centrally placed in muscle	T	5×10^{-21}	[illegible]	[illegible]
	Q_T	1×10^{0}	[illegible]	[illegible]
5 cm total thickness, 1 mm thick skin, 1 cm tumour, centrally placed in fat	T	5×10^{-2}	[illegible]	[illegible]
5 cm total thickness, 1 mm thick skin, 1 cm tumour centrally placed in muscle	T	1×10^{-10}	[illegible]	[illegible]

λ = Free space wavelength; T = power transmitted/power incident, without tumour; Q_T = T with 1 cm tumour/T without tumour.

Table 2 summarizes the results for transmitted power. The presence of tumour tissue is seen to have a large effect on the transmitted power, but attenuation is high. Only a few centimetres of muscle reduce the transmitted power to detector noise level, if the incident power is the accepted safe value of 10 mW/cm². This could perhaps be improved by using more intense, pulsed microwave illumination.

The results for the same models, without a skin layer on back and front, are very similar except for tumours within a centimetre of the surface, when large variations in transmitted power are caused by interference effects.

The ratio of power transmitted with tumour and without (Q_T) is quoted in Table 2 for one thickness only, because it varies only slightly with total model thickness. The variation is largest when absorption is least, that is, for larger wavelengths and fatty tissues. Thus Q_T for a centrally placed tumour in fatty tissue, using 10 cm waves, changes by less than 5 per cent as the total model thickness is increased from 10 cm to infinity.

Q_T also varies in an oscillatory manner as the tumour is moved from the centre of the model to the surface. This variation is also largest when absorption is least. For example, the variations are within 50 per cent of the central tumour position value for a 10 cm fat model using 10 cm waves.

These variations are caused by multiple reflexions from the air–tissue interface, and are reduced if the impedance match at this interface is improved.

The results calculated from the same basic model, but considering the reflected microwave radiation, instead of transmitted, are given in Table 3.

Table 3. EFFECT OF TUMOUR ON [illegible]

Model		1·27	3·18	10
10 cm fat (thickness), 1 mm skin layer	R	0·49	0·67	0·40
[illegible] 1 cm from front in fat	Q_R	0·99	0·95	1·15
	D cm	1	4	10
10 cm fat (thickness), 1 mm skin layer	R	0·46	0·53	0·57
	Q_R	1·00	1·00	0·99
1 cm tumour, 1 cm from front in muscle	D cm	—	—	1

R = Power reflected/power incident; Q_R = R with 1 cm tumour, 1 cm front of model/R without tumour; D = distance of tumour from front of model when Q_R is within 1 per cent of unity.

It is seen that the ratio, with tumour to without, Q_R, is much less significant for reflected waves than for transmitted waves.

The effect of the skin layer is very important because the tumour only gives an appreciable effect when close to the front surface. The effect of removing the skin layer from a 10 cm model of fatty tissue, illuminated by 3 cm microwaves, is to change: R from 0·67 with skin to 0·12 without skin; Q_R from 0·98 to 1·33; D from 4 cm to 6 cm.

Thus the skinless model gives a much improved chance of tumour detection, principally because the impedance match at the air-tissue interface is better.

Impedance matching at skin-air interfaces may make it possible to detect tumours within a few centimetres of the surface of fatty tissue by measuring reflected power. It would probably be too difficult to detect a tumour in muscle by this method, because of the small reflexion coefficient at a muscle-tumour interface.

Transmission measurements in fat should indicate the presence of a tumour using 3 cm microwaves; but larger wavelengths, with consequent loss of resolution, would be needed to give measurable output through more than a few centimetres of muscle.

Perhaps the closest anatomical situation to the model discussed is the human breast, although the attenuation *in vivo* would probably be intermediate between that of fat and muscle.

The conclusions of the previous publication are still valid—that it may be possible, using microwaves, to demonstrate differences of dielectric properties from point to point in the body and perhaps outline and localize tissues, body cavities, abnormal regions of organs or interfaces and to observe the movement of interfaces. It is not impossible that it may be of some use for tumour detection and localization. For this, much more precise data on dielectric constant values are required and work is in progress to provide this.

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GENETICS

Cytogenetic Study of the Offspring of Atom Bomb Survivors

EXTENSIVE studies of offspring conceived after the exposure of their parents to the atom bomb have revealed no increased frequency of stillbirths, neonatal deaths or gross malformations[1]. Furthermore, there has been no overall increase in the mortality rate in the F_1 generation[2], and initial impressions of an alteration in the sex ratio have not been substantiated[3]. These essentially negative findings suggest that no effects were produced, that the sample size was too small to allow detection of genetic effects, or that the indicators used were insufficiently sensitive.

This cytogenetic study was designed to evaluate the sensitivity to radiation of human germ cell chromosomes, on the assumption that subtle but detectable chromosomal rearrangements induced in the meiotic chromosomes of an exposed parent would be reflected in the somatic cells of the progeny, even if the offspring were phenotypically normal. It has been well established, for example, that heterozygous carriers of balanced translocations may be clinically unremarkable[4-6].

The selection of a relatively small number of children of heavily exposed survivors was planned as a preliminary approach to an anticipated effort on a larger scale. Families were selected from the fertility study sample of Sawada *et al.*, the criteria being (1) that at least one parent had had a minimum exposure dose of more than 100 rads[7] and (2) that there were children born both before and after parental exposure.

Peripheral blood leucocytes were cultured for 46–50 h (ref. 8). Slides were coded and analysed without knowledge of the age of the patients. Twenty-five well spread metaphases were examined microscopically, and an additional five cells were independently selected and photographed for karyotype analysis.

As Table 1 shows, leucocytes were successfully cultured from 185 children from ninety-eight families with one or more exposed parent. Of the 185, 128 individuals—sixty-two males and sixty-six females—were conceived after parental exposure, and fifty-seven children—twenty-six males and thirty-one females—were conceived before exposure. Of the 128, 103 came from families in which the maternal dose exceeded 150 rads, the father having been out of the city at the time of the bombing (eighty-seven cases) or exposed to less than 150 rads (sixteen cases). The remaining twenty-five of the 128 children were born to parents exposed to lower doses, but in all instances, at least one parent received more than 100 rads.

Although all families included children born both before and after parental exposure, in only one-third of the families contacted were we able to obtain blood samples from children born before August 1945.

Table 1. NUMBER OF CYTOGENETICALLY EXAMINED CHILDREN OF SURVIVORS OF THE ATOM BOMB BY TIME OF BIRTH, CITY AND SEX

Period of birth	Hiroshima			Nagasaki			Combined		
	Male	Female	Total	Male	Female	Total	Male	Female	Total
1940–1946	21	24	45	6	7	12	26	31	57
1946–1950	37	40	77	11	15	26	48 } 62	65 } 66	103 } 128
1951–1963	—	—	—	14	11	25	14	11	25
Total	58	64	122	30	33	63	88	97	185
No. of families		69			29			98	

Of the 185 children studied, 182 were karyotypically normal. Among the other three, one female was found to have a deleted short-arm of one of the *G* group chromosomes in all cells analysed (Fig. 1). This girl, born in 1948, was clinically normal. Her sister, born in 1941, and sampled as a family control for the proband, also had the same chromosomal abnormality. The third karyotypically abnormal patient was a female, born in 1949. She showed a sex chromosomal mosaicism of 45, X/47, XXX. Ninety-

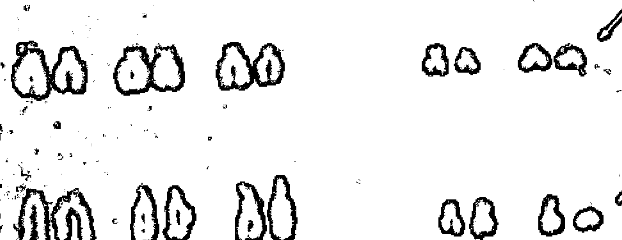

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Fig. 1. Partial karyotypes showing the short-arm deletion of a *G* group chromosome in a female born in 1948 (upper row) and in her sister born in 1941 (lower row). This abnormality was also found in the mother and was therefore familial.