A CYTOGENETIC FOLLOW-UP OF SOME HIGHLY IRRADIATED VICTIMS OF THE CHERNOBYL ACCIDENT

A.V. Sevan'kaev[1], D.C. Lloyd[2], A.A. Edwards[2], I.K. Khvostunov[1], G.F. Mikhailova[1], E.V. Golub[1], N.N. Shepel[1], N.M. Nadejina[3], I.A. Galstian[3], V.Yu. Nugis[3], L Barrios[4], M. R. Caballin[4] and J. F. Barquinero[4]

[1]Medical Radiological Research Centre of RAMS, Koroliov St. 4, Obninsk, Kaluga Region, 249036, Russia
[2] National Radiological Protection Board, Chilton, Didcot, Oxon, OX11 0RQ, UK
[3] State Scientific Centre Institute of Biophysics, Zhivopisnaya 46, 123182, Moscow, Russia
[4] Autonomous University of Barcelona, 08193 Bellaterra (Barcelona), Spain

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Contact author:
David Lloyd
Tel: +44 (0)1235 822700
Fax: +44 (0)1235 833891
E-mail: david.lloyd@nrpb.org

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Abstract A follow-up of ten highly irradiated men, mostly reactor crew, from the Chernobyl accident is described. Their pre-accident medical conditions, and relevant medical status approximately 10-13y later are listed. A comparison is made between estimates, derived from several biological parameters, of their average whole-body penetrating radiation doses. First estimates were based on their presenting severity of prodromal sickness, early changes in blood cell counts, and dicentric chromosome aberrations in lymphocytes. In three cases ESR measurements on tooth enamel were also made. Retrospective dosimetry using FISH translocations was attempted 10-13 y later. This showed good agreement for those patients with the lower earlier dose estimates; up to about 3 Gy. For the others, extending up to about 12 Gy, the translocations indicated lower values, suggesting that in these cases translocations had somewhat declined. Repeated chromosomal examinations during the follow-up period showed an expected decline in dicentric frequencies. The pattern of decline was biphasic with a more rapid first phase, with a half-life of about 4 months followed by a slower decline with half-lives around 2-4 y. The rapid phase persisted for longer in those patients who had received the highest doses. 10-13 y later dicentric levels were still above normal background but well below the translocation frequencies.

INTRODUCTION

There is great interest in the use of chromosomal translocations in blood lymphocytes to measure the dose received by a person exposed to ionising radiation many years previously. The crucial question relates to the stability with time of the yield of translocations and how this compares with the disappearance of the dicentric which is the unstable type of aberration routinely used in the shorter term for biological dosimetry. The follow-up of persons involved in radiation accidents provides an opportunity to obtain such data and the present study reports on ten men who exhibited acute radiation syndrome (ARS) from the 1986 reactor accident at Chernobyl.

MATERIALS AND METHODS

The study group

This comprised eight reactor crew members who were present at the time of the accident, one of the first fire-fighters on the scene and one of the early clean-up workers. These 10 men were among the patients evacuated to Moscow for medical treatment of ARS. They were chosen for this follow-up because, despite their high radiation doses, none of their treatments involved procedures, such as blood transfusion or marrow engrafting, that might have prejudiced on-going cytogenetic analyses. In two patients, after the first blood sampling for cytogenetics, marrow transplantation was attempted but resulted in rejection and therefore lymphocytes examined for further cytogenetics were also their own, rather than derived from the donors. A brief summary of each person follows noting any pre-existing medical features additional to ARS and any medical conditions present approximately 10 y after the accident. The ages given are those at the time of the accident:

1. An engineer aged 46y who spent 1h 50-60m from the reactor followed by 3 hours in other parts of the damaged reactor unit no.4. On medical examination he had high blood pressure, coronary artery and cerebral vascular diseases. He presented with ARS grade I. In 1996 ocular disease (retinal arteriosclerosis and hypertension angiopathy) was noted.
2. A fire-truck driver, aged 36y, who stayed in the driver's cab parked very close (10m) to unit 4 for 3 hours. He had high blood pressure, type 2 diabetes, cerebral vascular disease and obesity. He presented with ARS grade II. In 1996 he was noted to have telangiectasia of the skin on the hands and lower legs, nodular goitre, and retinal angiopathy. In 1999 he underwent thyroidectomy for a cyst goitre on the right side and he had a cataract in one eye.
3. An engineer aged 36y who stayed inside reactor unit 4 for 3.5 hours. Medical examination showed hepatitis C and ARS grade II. By 1997 he had developed bilateral cataracts.
4. A reactor operator, aged 26y, who was close (20m) to the reactor for 20 minutes. He had no pre-existing health problems and presented with prodromal symptoms indicative of ARS grade III plus skin burns. By 1996 he still had moderate leucopenia, a cataract in one eye and then in the other eye by 1999. About 80% of his body surface exhibited scarring, fibrotic atrophy and hyperkeratosis and there was a late skin ulcer on a toe.
5. An engineer aged 48y who took part in the clean-up operation during the first 10 days. However almost all his exposure was received when he worked for 40 min about 200m from the reactor. Medical examination showed high blood pressure, hepatitis, type 2 diabetes, coronary heart disease and angina. He presented with

ARS grade II. Examination in 1996 showed diffuse nodular goitre and retinal angiopathy.

6. An engineer aged 35y, who was 60-70m from the reactor for 40 minutes. Medical examination showed high blood pressure. He presented with ARS grade III plus burns. A sibling bone marrow graft was given, but the transplant was rejected. In 1997 he still had extensive skin scarring and bilateral cataracts.
7. A reactor operator aged 28y, who was 20m from the reactor for 1 hour. Medical examination showed hepatitis and ARS grade II plus skin burns. In 1996 the long-term skin lesions were most apparent on the feet, thighs and hands.
8. An engineer aged 38y who stayed in unit 4 for 5 hours. Medical examination showed a coronary artery problem and a kidney cancer, which was removed surgically without radiotherapy or cytotoxic drugs. He presented with ARS grade II. In 1998 he had moderate thrombocytosis and bilateral senile cataracts which were not considered to be radiogenic.
9. A reactor operator, aged 25y who remained about 100m from the reactor for 3 hours. Medical examination showed symptoms of ARS grade IV and skin burns. A bone marrow transplant was rejected. In 1996 there were posterior fibrotic changes to the lenses of both eyes and the long term consequences of the burns were fibrosis, atrophy, scarring and a ulcerated buttock. He had also undergone repeated plastic surgery for radiation ulcers.
10. An engineer aged 25y, who was 60-100m from the reactor for 3 hours. Medical examination showed symptoms of ARS grade III plus burns. In 1996 he had moderate thrombocytosis, a nodule in the thyroid and the consequences of the radiation burns were fibrosis and scarring and some joint contractures.

In summary, 10-13y after the accident there have been no cancers detected in these 10 men, apart from one pre-existing tumour that was successfully treated. Cataracts have been a common feature, detected in six of the men, although in one case they were diagnosed as the type associated with senility despite the man being only aged 50y. Cataracts in these men, together with other Chernobyl victims have been more fully addressed elsewhere [1,2]. The prevalence of cataracts here is perhaps not surprising as the incidence reported from a 10y follow-up of patients given total body irradiation prior to marrow transplantation was 50% [3]. Five men developed late, often intractable, skin lesions mainly on their limbs that have necessitated continuing treatment. Skin burns in these and other Chernobyl victims have also been described elsewhere [4]. Some of the present study group have been mentioned in other publications and Table 1 shows cross references to them.

Cytogenetic analysis

Blood samples were taken from each person in the study group at various times following the accident and cultured to produce first division metaphases by standard methods[5]. Slides were stained with fluorescence plus Giemsa (FPG) and scored for dicentrics, excess acentrics and centric rings using standard criteria[5]. In addition, samples taken from 1996 onwards were 'painted' using the fluorescence in situ hybridisation (FISH) technique. Chromosomes 2, 3 and 8 were selectively highlighted with fluorescein isothiocyanate labelled probes and the remainder counterstained with 4',6-diamidino-2-phenylindole. A complete (two-way) translocation was scored when two bicoloured monocentric chromosomes (with exchanged counterparts) were present in the cell. An incomplete (one-way) translocation was scored when only one bicoloured chromosome with a single centromere was present. A dicentric was scored

when one bicoloured chromosome bearing two centromeres was present and the accompanying acentric was either bicoloured or monocoloured. Excess acentrics were scored when a coloured or bicoloured chromosome contained no centromere. A chromosome with an atypical centromere position was recorded as an inversion if it appeared to be of the right length or a terminal deletion if the chromosome was too short compared with its normal homologue. An insertion was scored when a painted length of material was present inside a counterstained chromosome or vice versa. Cells containing visibly complex rearrangements were recorded separately but not included in the specified aberration frequencies.

Dose calculations by cytogenetics

Dicentric chromosome aberration yields from blood samples taken soon after exposure were used to derive dose by reference to a linear/quadratic in vitro calibration curve for acute Co-60 gamma rays[6]. The equation is given in Table 1 footnote. A correction, G, to the square law term to take account of the duration of exposure has also been used. This used the G-function of Lea and Catcheside[7] shown in equation (1) where x = t/T, t is the duration of the irradiation in hours and T is the time constant for repair; assumed to be 2 hours.

$$G = 2(x-1+\exp(-x))/x^2 \quad \ldots\ldots (1)$$

The doses derived from the initial dicentric data are shown in Table 1 where two values are listed for each man. One is derived from the acute dose response curve and represents minimal values indicated by the dicentric yields. The other is based on the G-function using the exposure times as recollected by the men.

The conversion of FISH translocation yield to dose used calibration curves obtained with acute cobalt-60 γ rays, in two of the collaborating laboratories[8,9]. The same dose

rate correction to the square law term as for dicentrics was used and the equations are shown in the footnotes to Table 2. The first equation[8] includes a factor of 0.339 to correct to the full genome because chromosomes 2, 3 and 5 were painted and in the second [9] where chromosomes 1,4 and 11 were painted the factor is 0.314.

RESULTS

Table 1 shows each person's dose estimates made from medical observations, early neutrophil counts and from the earliest available measurement of dicentric yield. The first blood samplings took place from 1 to 55 days after the accident. Estimates of dose from electron spin resonance measurements in a tooth are given in three cases. Table 2 shows the measurements of translocation yields in blood samples taken 10-13 years after the accident and the resulting estimates of dose based on dose response curves from two laboratories. Changes in dicentric yields measured in later blood samples are plotted in Figs 1-3 where they show decreases with time which have been fitted to the sum of two decaying exponentials. The final dicentric measurements made 10-13 y later have been used (Table 3) to estimate dose retrospectively by the contaminated Poisson and Qdr methods.

DISCUSSION

On admission to hospital the initial assessment of the patients included determining their grade of ARS. In the former Soviet Union, ARS was classified into 4 grades based on the severity and timing of the early deterministic responses that constitute the prodromal reaction[10]. Based on previous experience with irradiated patients, these grades can be assigned to approximate ranges of penetrating whole-body radiation dose. These are I : 1-2 Gy; II : 2-4 Gy; III : 4-6 Gy and IV : > 6Gy.

Part of the assessment of patients included measuring haematological parameters, in particular the early changes in peripheral blood cell counts. These too may be related to absorbed radiation dose[11], and so also constitute an approximate biodosimeter. Data on the cytogenetic dose estimates based on the dicentric assay, then produced the third biological estimates of doses and experience with this assay, since its inception in the early 1960s, has shown that it provides the most quantitatively reliable biological dosimetry[12,13]. Finally, 10 or more years after the accident, the FISH translocation assay[14] was used to derive retrospective estimates of doses and to compare these with the values assigned to each patient in 1986.

On discharge from hospital the patients were advised that if, for dental care reasons, they should have a tooth removed, it should be preserved for electron spin resonance (ESR) dosimetry measurements on the enamel[15]. Patients 1, 6 and 10 did later provide a tooth for analysis.

In Table 1 there are a few anomalies between the diagnosis of the grade of ARS and the dose estimated from haematology. In patient 8 the haematological dose estimate of 1.4 Gy does not correspond to the 2-4 Gy of ARS II. However, the derivation of dose from blood count levels assumes acute exposure. A larger protracted exposure is necessary to cause a similar reduction in blood cell counts and for this patient the dose was received over 5h. In all other cases the periods of exposure were a few hours or less. Patients 6 and 10, graded ARS III, (4-6 Gy) were assessed by haematology as 4.6 and 4.3 Gy. However their higher ESR measurements of 8.9 and 6.7 Gy are more suggestive of grade IV. The ESR signal in teeth is an estimate of dose at a single point, which in an inhomogeneous irradiation field may not accurately reflect average whole body dose. However, as discussed later, the indications from dicentrics data are that in most cases the absorbed doses due to penetrating radiation were more or less

homogeneous in the whole-body. The other tooth measurement, available for patient 1 who was graded ARS I, registered a lower dose (0.77 Gy), than the corresponding haematological estimate (1.6 Gy), but this time the ARS grading and haematological estimates agree.

The ARS gradings and haematological estimates are based on the assumption of acute exposures and the ESR signals do not vary with dose rate. The dicentric dose estimates given in Table 1 have also been assessed against an acute dose response curve. However there is strong experimental evidence that exposures of a few hours duration do influence the dicentric yield and hence dose estimates [16]. Therefore the exposure durations, as given by the men's accounts, have been taken into consideration and in each case the time-dependent G-factor (eqn 1) was used to adjust the quadratic term of the acute dose response curve. In Table 1 it may be seen that this has resulted in higher values.

A complication of the cytogenetic method of dose estimation is the possibility of non-uniform irradiation. This may be indicated by overdispersion of the dicentrics among the cells compared with that expected from the Poisson distribution. Patients 2-5 show positive dispersion typified by a ratio of variance to mean (σ^2/Y in Table 1) greater than 1.0 but none of the values are significantly different from unity. Only case 3 is approaching significance. Therefore for these patients their first dicentric analyses suggest that their exposures to penetrating radiation were essentially uniform. Some of the patients presented with localised skin burns, which suggest partial body irradiation. If, however, these were caused by contamination with β-emitters on the skin surface their poor penetration into the body would be insufficient to irradiate mature lymphocytes and thus would not contribute to the cytogenetic dose estimates.

The most surprising result is patient 9 where the dicentric yield in 1986 was about 5 per cell converting to a dose in excess of 10 Gy. He also has the highest of the doses shown in Table 1 as assessed by blood counts and severity of ARS. It is remarkable that this patient survived bearing in mind that the accident occurred prior to the availability of cytokine treatment to aid restoration of marrow function, and that an attempt to give a marrow transplant failed. With the medical and nursing skills then available the LD_{50} was estimated to be around 4.5 Gy[17,18] and the steepness of the sigmoid curve for mortality would indicate no chance of survival beyond a few months at 10 Gy. A year after exposure his dicentric yield was still about 1 per cell and by 10 years it had declined to 0.01 dicentrics per cell, still an order of magnitude above normal control levels[5].

The person with the next highest cytogenetic estimate of dose, case 6, had an initial dicentric yield of about 2 per cell indicating a whole body dose of 6-7 Gy. This would seem to put him into the ARS IV category instead of the category III assessment made from the prodromal symptoms. Cases 3 and 10 have a dicentric yield of about 1 per cell indicating ARS category III. Cases 2 and 4 have a yield of 0.6 dicentrics per cell and this should correspond to ARS II. In both these cases there was a delay of about 1.5 to 2 months between exposure and blood sampling in which time the dicentric yield could have fallen. The remainder (1, 5, 7 and 8) have lower yields corresponding to ARS I or possibly II. Overall the measured dicentric yields led to dose estimates that correlated quite well with initial medical assessments.

It is usually assumed that for biological dosimetry purposes, where delayed sampling requires an extrapolation to zero time, the yield of dicentrics decreases with a half-life of about 3 years[5]. However, this seems to apply only to persons with normal haematology and there is evidence that following a high acute exposure that

the reduction is biphasic, that is initially fast and then slower[19,20]. The 10 patients here also show this pattern and for presentation purposes in Figures 1-3 they have been divided into highest, medium and lowest dose groups based on their initial dicentric frequencies. In all cases the initial fast reduction had a half-life of around 4 months and for the patients who had the highest doses (Figure 3) this phase persisted for about 2 years. In the other cases (Figures 1 and 2) the less steep second phase commenced at about 1 year after exposure. The slower phase had a half-life from 2-4 years.

Despite the very much lower dicentric yields, some of the samples scored many years after exposure did show overdispersion. This is probably caused by the replacement of lymphocytes from the stem cells, resulting in a Poisson distribution 'contaminated' with cells containing no unstable aberrations. There are two calculational approaches, the contaminated Poisson and the Qdr methods, that have been developed for application to overdispersion, usually as a result of recent partial body exposure[5]. With the present data it was possible to use these methods on some of the final blood samples 10-13y later. The contaminated Poisson method was possible in 5 cases where overdispersion was evident and Qdr in all but one case where there were no dicentrics. The data are shown in Table 3. The best agreement with dose estimates shown in Tables 1 and 2 is for patients 2-4 where the residual dicentric yields are still high but throughout Table 3 the very wide confidence limits suggest that these approaches to retrospective dosimetry are of little practical use.

The results of FISH scoring, shown in Table 2, have been converted to dose. In the early days of using this technique it was customary to list separately one-way and two-way translocations because it was thought that two-way translocations were more stable. It was later discovered that many of the apparently one-way translocations

were in reality reciprocal[21] and thus should be included with the two-way translocations. Now that the idea of scoring translocations in stable cells has been introduced [14], it is no longer necessary to distinguish them and doses derived here have been based on total translocation yields. Great care needs to be taken in choosing a calibration curve applicable to high doses because, many years after the accident, lymphocytes originally exposed have been replaced from the dividing stem cells pool. This involves a strong selection process because cells containing unstable aberrations will be largely eliminated. Strictly, translocation yields should be referred to a dose response curve obtained from scoring stable cells only. On the assumption that stable cells are those that do not contain a dicentric, acentric or centric ring, Finnon et al[8] and Rodriguez et al [9] produced such calibration curves, as given in the footnotes of Table 2. These have been converted to full genome curves using the appropriate conversion factors described earlier. It is interesting that despite the wide divergence of the coefficients in the calibration curves the estimates of doses based on translocation yields are quite close. This is because within the range of translocation yields measured here the two curves are similar; the high alpha and low beta in one curve are compensated by a low alpha and high beta in the other. Regarding stability of translocation yield with time, the cases with the higher dicentric yields (3, 6 9, and10) show that translocation yield at about 11 years is around a factor 4 lower than the initial dicentric yield. Such a large reduction has been seen following the accident in Goiania[22]. For intermediate doses, cases 2 and 4, the reduction is in the range 2-2.5. For the lowest doses, cases 1, 5, 7and 8, the reductions are 1.3, 2.2, 1.3 and 2.1 respectively. These last values may be compared with decreases of factors 1.4 to 1.6 seen in the victims of the Tammiku (Estonia) accident where the translocation yields remained stable from 4 years post-irradiation[23].

For the exceptional survivor, case 9, at 10 years post-irradiation the translocation yield converted to full genome equivalence was just above 1 translocation per cell and this converts to a dose in the region of 5-7 Gy (Table 2). This estimate must be regarded as unreliable because of the large amount of cell replacement that has taken place eliminating many of the cells containing dicentrics. This particular case has, nevertheless, shown the remarkable stability of translocations, bearing in mind that the dicentric yield has reduced by a factor of 500 in the same time and that only a very small fraction of his stem cells present at the time of irradiation would have produced viable daughter cells.

There are systematic trends for translocation measurements. For first dicentric yields greater than 1 per cell, cases 3,6,9 and 10 with high acute doses, the corresponding translocation yield produces a dose estimate lower by about a factor 2. For dicentric yields between 0.5 and 1 per cell, cases 2 and 4, translocation yield produces a dose about 30 % lower. For all other cases the doses obtained by dicentrics and translocations agree very well.

Thus it appears that the measurement of translocation yield using the FISH technique does not work very well for high acute doses usually classed as life threatening; a point that has been made previously[22]. For doses below that level (less than 3 Gy) the technique gives good dose indications, improving at the lower doses.

CONCLUSIONS

The purpose of this study was to present further evidence for the rate of decline of dicentric yield with time following irradiation and to investigate how well, by comparison with the original estimates, the measurement of translocations using FISH could estimate doses received many years previously. Patients with high near-fatal

doses showed a fast decrease of dicentric yield with time with a half-life of about 4 months. This decrease continued to about 2 years when it became slower. For the lower doses the initial half-life for dicentrics was just as rapid but became slower sooner.

The translocation technique works well as a retrospective dosemeter particularly at acute doses up to about 3 Gy. The doses need to have been delivered approximately uniformly over the body. Higher acute doses seem not to be so well measured probably because many stem cells are unable to produce progeny. In such cases clinical symptoms are immediately evident and so in practice patients should come rapidly to medical attention and biological dosimetry by the simpler dicentric analysis is quite adequate without the need to resort to FISH.

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Subject number				Exposure period, h (G-value)	ARS grade	Haematology dose, Gy	ESR Gy	Initial Dicentric Evaluation				
a	b	c	d					Yield of dicentrics/cell	Days after exposure	σ^2/y (±SE)	Dose[1] (95% C.L.)Gy	Dose[2] (95% C.L.)Gy
1	1091	11	-	1.0 (0.85)	I	1.6	0.77	21/175	45	0.98±.10	1.1(0.8,1.5)	1.2(0.8;1.6)
2	1073	4	-	3.0 (0.64)	II	2.7	-	87/120	45	1.02±.13	3.5(3.1,3.9)	4.2 (3.6;4.7)
3	1039	2	-	3.5 (0.60)	II	2.8	-	105/100	19	1.26±.14	4.3(3.8,4.8)	5.3 (4.7;6.0)
4	1047	5	11	0.33 (0.95)	III	3.5	-	188/300	52	1.08±.08	3.2(2.9,3.5)	3.3 (3.0;3.5)
5	1143	8	-	0.67 (0.90)	II	1.9	-	25/100	37	1.16±.14	1.8(1.4,2.3)	1.9(1.4;2.4)
6	1011	-	2	0.67 (0.90)	III	4.6	8.9	327/154	1	0.93±.11	6.3(6.0,6.7)	6.7 (6.3;7.1)
7	1049	7	8	1.0 (0.85)	II	2.8	-	11/35	55	0.71±.23	2.1(1.3,3.0)	2.2 (1.4;3.2)
8	1089	9	-	5.0 (0.51)	II	1.4	-	21/100	8	0.80±.14	1.6(1.2,2.1)	2.0 (1.4;2.6)
9	1029	1	9	3.0 (0.64)	IV	8.5	-	492/100	1	0.68±.14	10.0(9.5,10.5)	12.3 (11.7;12.9)
10	1052	3	7	3.0 (0.64)	III	4.3	6.7	120/115	5	0.96±.13	4.3(3.9,4.7)	5.2(4.6,5.7)

Table 1 A comparison of the absorbed doses estimated by various biological indicators.
For dicentrics the yield equation $Y=0.0003+ 0.059D+ 0.043GD^2$ was used, see text.
a –this work, b -Nugis[24], c –Sevan'kaev et al[25], d -Salassidis et al[26].
[1] Doses estimated with G=1. [2] Doses estimated using the values of G in column 5

Subject	Age,[1] y	c [2]	Cells [3] scored	2-way trs	Total trs	Dose, Gy[4]	Dose, Gy[5]
1	56	0.008	1000 (321)	19	29	1.2	1.3
2	46	0.006	1000 (321)	69	101	2.7	3.3
3	47	0.006	500 (160)	17	36	2.3	2.7
4	37	0.005	1000 (321)	54	82	2.0	2.5
5	59	0.008	500 (160)	10	18	1.3	1.5
6	48	0.006	398 (128)	44	62	2.9	3.7
7	38	0.005	499 (160)	28	40	2.1	2.5
8	49	0.006	500 (160)	12	16	1.6	1.6
9	35	0.005	200 (64)	47	75	5.3	7.1
10	35	0.005	500 (160)	32	46	2.6	3.1

Table 2. Doses estimated from translocations (trs) scored by FISH in samples taken 10-13 y after exposure.

[1] Age at time of blood sampling
[2] Assumed age related background frequency of total translocations based on Sorokine-Durm et al [(27)]
[3] Genome equivalent cell number in brackets
[4] Doses estimated from using $Y= c + 0.0077D + 0.062GD^2$ (Finnon et al,[(8)])
[5] Doses estimated from using $Y= c+ 0.033D + 0.029GD^2$ (Rodriguez et al [(9)])
Values of G are given in Table 1

Subject	Delay, y	Dicentrics	Cells	Damaged cells	Dose estimates in Gy with 95% C.L using:	
					contaminated Poisson	Qdr
1	12	4	500	10	-	0.1 (0,0.4)
2	11	22	500	23	4.0 (1.3-5.5)	4.9 (2.6,7.2)
3	11	12	500	12	5.8 (1.9-8.2)	5.2 (2.0-8.4)
4	12	11	500	15	4.2 (2.2-5.5)	2.7 (0-6.2)
5	11	6	500	12	2.4 (0-5.4)	0.2 (0-1.4)
6	13	3	500	5	-	1.3 (0-12.8)
7	10	0	500	2	-	-
8	11	5	500	7	-	3.5 (0-10.7)
9	10	5	370	12	3.2 (0-5.7)	0.1 (0-0.5)
10	10	8	500	17	-	0.2 (0-0.8)

Table 3. Estimates of dose made by using the contaminated Poisson and Qdr methods on dicentric yields observed 10-13 years after irradiation.

Captions to figures

Fig. 1. The dicentric yields as a function of time after exposure for a subgroup of the subjects with low doses. The solid line is a least squares fit producing Y = 0.095*exp(-t / 0.36) + 0.035*exp(-t / 5.58), where Y = dicentric yield, $cell^{-1}$, t = time (y) after exposure.

Fig. 2. The dicentric yields as a function of time after exposure for a subgroup of the subjects with intermediate doses. The solid line is a least squares fit producing Y = 0.61*exp(-t / 0.43) + 0.23*exp(-t / 2.7), where Y = dicentric yield, cell-1, t = time (y) after exposure.

Fig. 3. The dicentric yields as a function of time after exposure for a subgroup of the subjects with high doses. The solid line is a least squares fit producing Y = 1.67*exp(-t / 0.34) + 0.17*exp(-t / 4.02), where Y = dicentric yield, cell-1, t = time (y) after exposure.

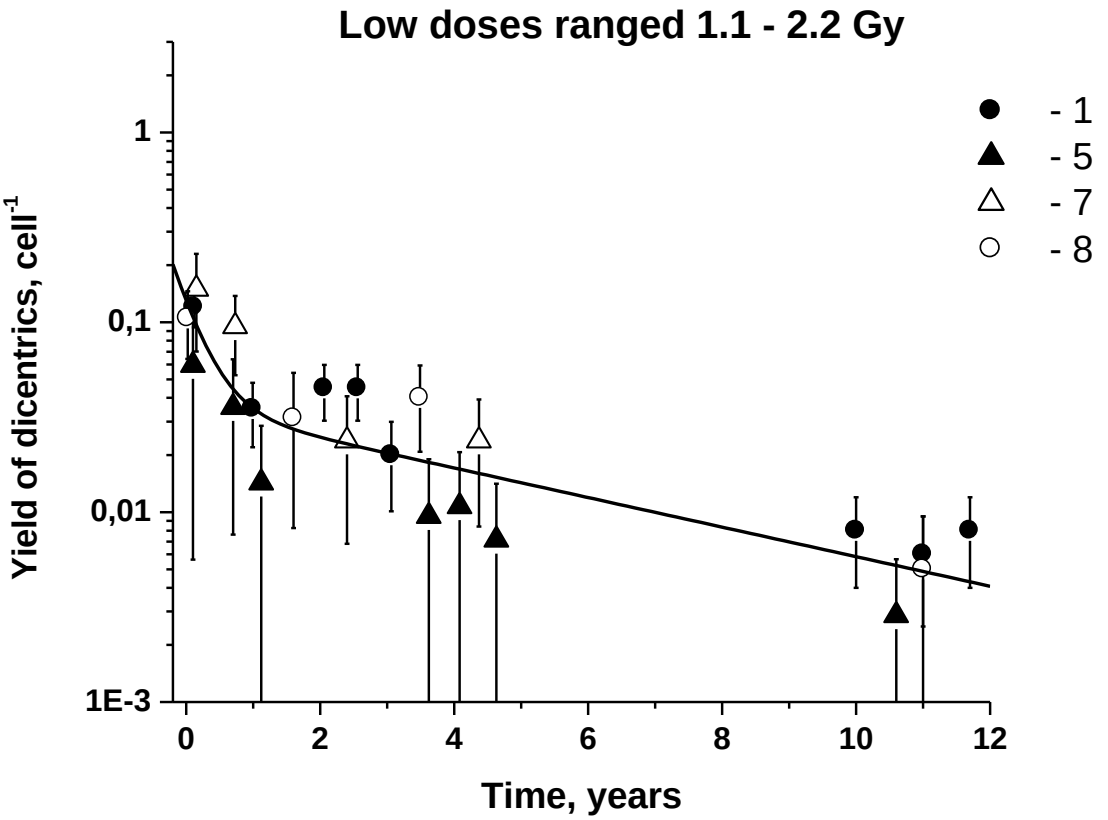
Low doses ranged 1.1 - 2.2 Gy
- 1
- 5
- 7
- 8
Yield of dicentrics, cell-1
1
0,1
0,01
1E-3
0
2
4
6
8
10
12
Time, years

Fig 1

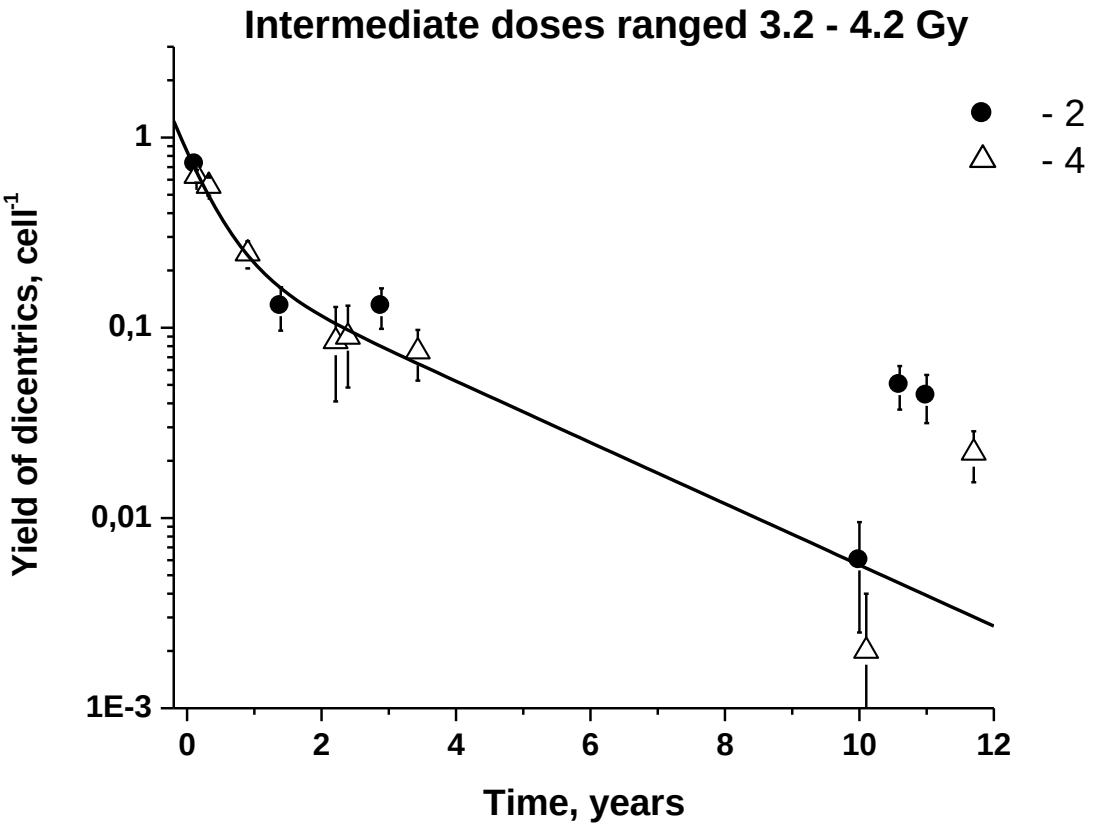
Intermediate doses ranged 3.2 - 4.2 Gy
- 2
- 4
Yield of dicentrics, cell-1
1
0,1
0,01
1E-3
0
2
4
6
8
10
12
Time, years

Fig 2

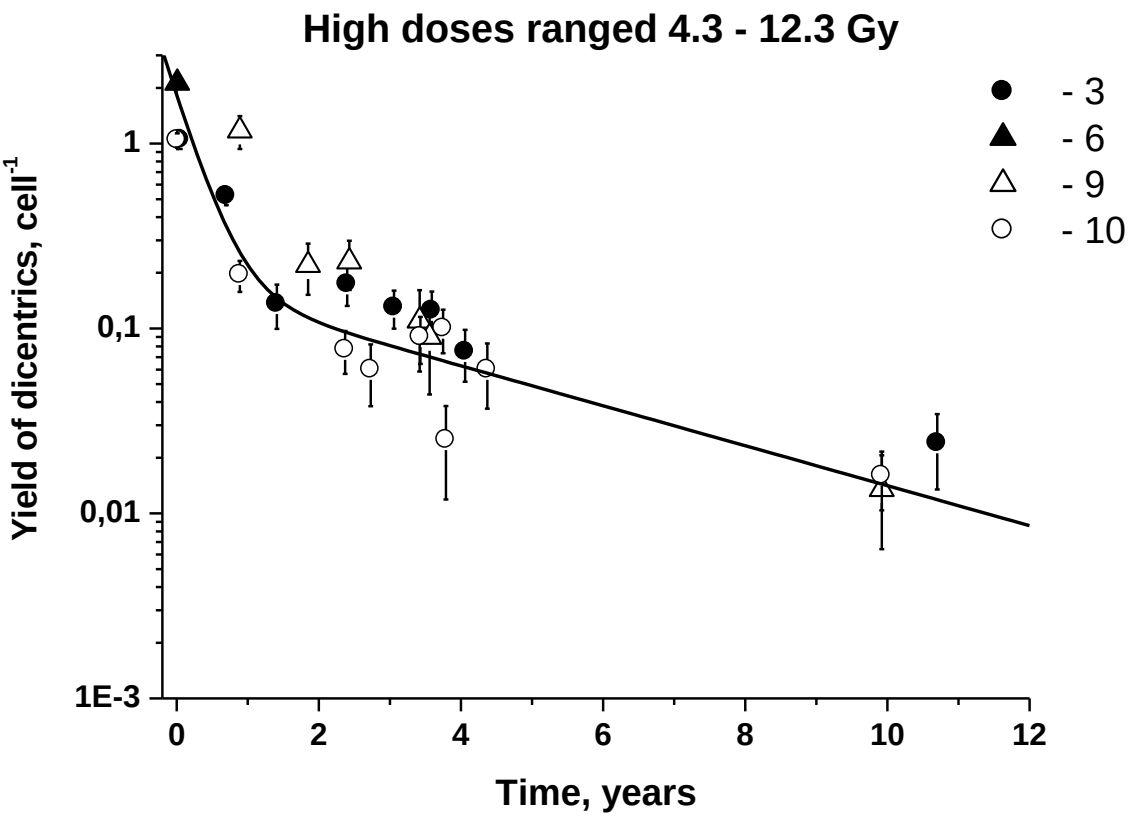
High doses ranged 4.3 - 12.3 Gy
- 3
- 6
- 9
- 10
Yield of dicentrics, cell-1
1
0,1
0,01
1E-3
0
2
4
6
8
10
12
Time, years

Fig 3