Table 1. Average values of blood count (n/mm^3) and megakaryocyte bone marrow counts (n/mm^3) in C3H/J and C57BL/6 mice - controls, 9th day and 8th week after low-dose irradiation

	Controls				9 days				56 days
	MegaKC	Thr	Ery	Leuko	MegaKC	Thr	Ery	Leuko	Thr
C3H/J									
mean value	9.5	676600	6113000	3200	17.7	226600	5462000	1580	611200
SD	2.64	110182	435200	718	6.5[a]	49300[a]	865300[b]	769[a]	66092[b]
C57BL/6									
mean value	14.8	374200	4733333	4200	13.2	108200	5224000	1320	590600
SD	2.3	198436	1225126	1122	3.2[b]	56500[a]	690750[b]	379[a]	100200[b]

[a] $P < 0.01$; [b] $P > 0.1$
Ery = erythrocyte, Leuko = leukocyte, MegaKC = megakaryocyte, Thr = thrombocyte

from the very beginning and even under the condition when low-dose radiation was used (7 Gy). This finging suggests that not only the TGF-β1 production by a variety of cells leads to postirradiation fibrotic changes, but serious tissue damage is also necessary.

In the C57BL/6 mice strain, TGF-β1 production occurs with a considerably reduced number of thrombocytes in lung vessels. Thus, it is obvious that the TGF-β1 tissue production also occurs without correlation with the thrombocyte trapping in lung capillaries, which was even reduced in irradiated mice of both strains studied. Thus, we consider that the total TGF-β1 production in lung tissues is mostly induced by an even different mechanism from the primary thrombocyte aggregation. From this standpoint, the selectively enhanced trapping of thrombocytes in lung vessels after intravenous administration of bleomycin seems to be only a secondary reaction to primarily selective damage to the vessel barrier of the lung tissue. Thus, in spite of the fact that thrombocytes are selectively captured in the lung tissue after administering bleomycin, the main cause of this process is a change at the level of endothelial cells of lung capillaries, which is reflected in the expression of CD11a and CD11b integrins (Piguet and Vesin, 1994).

A decrease in the white blood cell and thrombocyte numbers in the acute postirradiation period was observed in both mouse strains. In spite of the fact that this phenomenon was expected (Sadílková et al., 1981), it was assumed that particularly thrombocytes would also be aggregated in an enhanced extent in lung capillaries (see above). As a matter of fact, in the case of a damage different from radiation (e.g. after thermal damage using high-frequency current), the initial thrombocytopenia peak at 3.5 h is followed by a reactive thrombocytemia with a maximum achieved on the third day (Nakayama and Nakamura, 1984). In our experiment, this reaction, however, did not occur, probably due to low functional radiation damage of endothelial cells. TGF-β1 is synthesized in irradiated lungs independently of the total amount of platelets in lung capillaries. The observed decrease in platelet numbers in blood count and also in relative counts in lung capillaries probably depends on functional radiation damage of megakaryocytes, whose numbers in the bone marrow remained stable or surprisingly increased in C3H/J mice in the acute stage (see Table 1). These observations suggest that enhanced platelet trapping alone does not seem to be an initiator of TGF-β1 mRNA production, and subsequently of fibrosis production. This is in disagreement with previous articles suggesting the key role of platelets in lung fibrosis (Piguet and Vesin, 1994). Furthermore, our previous observation shows a consequent decrease in platelet count from day 1 to day 9 after irradiation using the dose of 7 Gy in the C3H/J strain (unpublished data).

Parallel electron-microscopic observation, particularly of changes on endothelial cells, only supports the older observations describing the enhanced electrodensity of endothelial cells. This reversible change, together with the dilatation of cisternae of the rough endoplasmic reticulum, seems to be the consequence of the reactive synthesis and disturbance of the trans-Golgi transportation related to the radiation damage of cells (Thomas, 1989). More prominent changes of irradiated lung parenchyma presented in the literature, such as interstitial oedema, lymphatic dilatation, apoptosis and arterial proliferation, etc. (Weir and Miachaelson, 1971; Ward et al., 1993), were observed neither in the acute nor in the subacute stage in our experiment. We explain this situation by a lower radiation dose used in the presented study (7 Gy), which is about one half of the dose (about 15 Gy) that caused more intensive pathological changes within the lung tissue in previous experiments (Guerry-Force et al., 1988; Waters et al., 1996). However, although the aim of the presented study was not to demonstrate the blood barrier disruption, surprisingly, the reversible endothelial damage was evident.

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