ausser einer I-Bandverdichtungszone kaum Strukturreinzelheiten erkennbar sind (Figur 2, d). Die Gründe hierzu sind neben dem geringen Elektronenstreuvermögen des Kupfers offenbar eine relativ geringe AB-Bindung im reaktiven Strukturmaterial infolge sterischer Anlagerungsbehinderung der grossen Cu-Phthalocyaninmoleküle durch Proteinaminogruppen, wie sie lichtmikroskopisch im Falle anderer Gewebe festgestellt wurde[7,12]. Eine auf die Aldehydfixierung folgende Desaminierung bewirkt nämlich auch bei der glycerinierten Flugmuskelfibrille eine verstärkte AB-Bindung und somit elektronenoptisch sowohl eine Intensivierung als auch eine stärkere Differenzierung des Cu-Kontrastbildes[15]. Eine deutlich verstärkte AB-Bindung tritt in den I-Bändern und in der h/M-Region nach einer Desaminierungszeit von 2 bis 3 h bei 20 °C bzw. von 18 bis 20 h bei 2 °C ein. Die Bleicitrat-Schnittkontrastierung verstärkt dieses elektronenoptische Bild der Cu-Verteilung, wobei nach einfacher Formolfixierung eine Abschwächung des Z-Bandkontrastes in Abhängigkeit von der Kontrastierzeit infolge von Materialextraktion durch das alkalische Kontrastiermedium eintritt (Figuren 1, b und 2, e). Das alcianophile Strukturmaterial setzt am Z-Streifen an und erstreckt sich über das I-Band. Nach Desaminierung sind Ausläufer bis in den Anfangsbereich des benachbarten A-Bandes erkennbar (Figur 2, d und e). Die Uranylacetat-Schnittkontrastierung, ebenso die OsO_4-Nachfixierung, ergeben das Kontrastbild der glycerinierten Flugmuskelsarkomere unter Darstellung der leicht gequollenen Filamente und der Z-Streifen.

Durch Methylierung werden beide Metallbindungsreaktionen in den I- und A-Bändern licht- und elektronenmikroskopisch völlig eliminiert, während in den Z-Streifen der positive Ausfall der Fe-Colloidreaktion im Fe-Kontrastbild zwar abgeschwächt wird, aber selbst bei langer Methylierungsdauer erhalten bleibt und durch OsO_4-Nachfixierung sogar verstärkt erscheint (Figur 2, c). Die Cetylpyridiniumchlorid-Behandlung blockiert die AB-Bindung in allen Sarkomerenzonen fast völlig. Dagegen führen Inkubationen mit hypotonischem *Tris*-Puffer, mit 0,1 *N* HCl oder hydrolytischen Enzymen zu keiner eindeutigen Abnahme der Metallbindungen; der partielle tryptische Abbau liefert ein AB- und Fe-Colloid-positives Muskelfragmentgerüst und die extensive Trypsinierung ein ungeordnetes, das gleiche Metallbindungsvermögen zeigendes Netzwerk, bestehend aus einer matrixartigen Masse, in welche die Mitochondrienstränge eingelagert sind.

Chemical Modification of Genetic Damage from Continuous Irradiation in Mice

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Summary. It has been demonstrated that Adeturon (S-2-aminoethyl-iso-thiuronium adenosine triphosphate) showed a marked protective effect against translocations induction in mice germ cells after chronic γ-or neutron-irradiation.

Studies of the antiradiation potential of Adeturon (S-2-aminoethyl-iso-thiuronium adenosine triphosphate) have shown this compound to be highly effective in terms of integral measures of radiation damage[1–3]. As it was further found that this compound is clearly able to protect germ cell heritable structures in the case of acute radiation exposures[4], and to have low toxicity when given daily for a 3-week period (unpublished data), we were led to extend this line of inquiry to continuous irradiation.

In the present work spermatogonial irradiation effects were assessed cytologically by determining chromosome translocation rates in primary spermatocytes – a test of high significance in studing radiation response of premeiotic germ cell stages.

Materials and methods. Male H mice aged 3.5 months were given continuous exposure to a 900-rad mean total dose of ^{226}Ra γ-rays at 1.0 $\pm$ 0.2 rad/h. C57BL males of the same age were exposed to a plutonium-beryllium source of 4.1 MeV neutrons at a rate of 0.08 $\pm$ 0.02 rad/h, receiving a mean total dose of 30 rad. Half the animals in each irradiation group were injected i.p. with daily Adeturon doses of 300 mg/kg body wt. in 0.5 ml aqueous solutions.

Three months following irradiation, the animals were killed to prepare testis slides by the method of EVANS et al.[5]. From each animal, 200–300 diakinesis-metaphase-I primary spermatocytes were examined. At this stage of spermatogenesis, 20 bivalents are normally formed. Chromosome translocations (symmetrical interchanges) are manifest as ring (R) or chain (C) multivalents.

Results. The Table shows the results of cytogenetic analysis in primary spermatocytes from mice given chronic γ-rays or neutrons, with or without daily Adeturon administration. 8-month-old untreated controls showed no chromosome translocations. Chronic 900-rad γ-rays produced translocations, amounting, as scored in primary spermatocytes, to 1.35%. The anomalies observed were exclusively quadrivalent rings and chains, in 1:2 ratio. With daily Adeturon administration, recovery of translocated cells fell to 0.60% ($0.01 < p < 0.02$).

For 30 rad chronic neutron exposure, the translocated cell frequency observed was 1.17%. Adeturon treatment reduced this genetic damage to 0.32% ($p \sim 0.005$). In this case also, the only forms of translocation observed were ring and chain quadrivalents.

Discussion. It is well known that a determining factor in modifying genetic response to a particular radiation dose is the time distribution of this dose. Studies of this point have clearly shown the amounts of chromosome translocations to diminish with decrease in γ-[6,7] or X-ray[8] dose rates. Our figure of 1.35% for translocated cell yield also indicated low dose-rate-irradiation to be quite inefficient in producing translocations. This effect has mostly been related to the possibility of repair of premutational changes in chromosomes, with a resulting reduction in the likelihood of interchange occurence, though other factors also seem to be involved[6,9].

It has lately been shown that acute genetic damage may be effectively reduced by radioprotective compounds, applied singly[4,10,11] or in mixtures[12,13]. In the case of prolonged exposures, however, even established antira-

Results from the cytogenetical analysis of mice germ cells after chronic γ- or neutron-irradiation with and without Adeturon protection

Group	No. animals	No. analyzed cells	Cells with translocations		
			R IV	C IV	Total (% ± SE)
γ-Rays	10	2,000	9	18	1.35 ± 0.15
Adeturon + γ-rays	10	2,000	4	8	0.60 ± 0.10
Neutrons	7	1,740	9	13	1.17 ± 0.32
Adeturon + neutrons	4	1,200	4	—	0.32 ± 0.17
Control[a]	10	2,000	—	—	0.00

[a] 5 mice C57BL and 5 mice H.

diation drugs show a decrease in their ability to protect somatic as well as germ cells. Our present findings indicated that Adeturon may still be protective when administered daily to mice receiving chronic γ-exposure. Presumably, the compound reduces radiation damage to germ cells by altering the general reactivity of organism as well as by favoring repair processes. Supportive evidence for this assumption is provided by studies where Adeturon, given as pretreatment + treatment, has been observed to protect the hemopoietic system, speeding up recovery during the postradiation period[14,15].

The present study gives further evidence of the ability of Adeturon to protect germ cells from chronic neutron exposure. Biological response to high-LET radiation is generally considered to be subject to little, if any, dose-rate effect or modification by protectants acting through oxygen-effect-related mechanisms[16,17]. However, from a more critical review of the relevant literature, it appears that some reservation is needed in such a generalization, since experimental results are often diverging or incomplete. It should be pointed out that, until disproved in experiments with dose rates sufficiently low to allow repair to take place, there is no a-priori ground to rule out the possibility of occurrence of repair processes in the case of high-LET radiation exposures. Furthermore, positive evidence has been obtained that S-containing protectants may be effective against neutron-induced damage by mechanisms differing from those that underlie protection from low-LET radiation[16,18].

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Unstable L-Forms of Micrococci in Human Foetal Blood

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Summary. In human foetal blood the presence of Micrococcaceae in the unstable L-form, probably taking origin from the placental transmission of minimal reproductive units, has been recognized by means of microscopic and cultural methods.

An incorporation of ^{14}C- and ^{3}H-labelled nucleosides and amino acids in suspensions of erythrocytes[1] and platelets[2] from normal human subjects has been detected and considered to be attributable to metabolic processes of bacterial L-forms. The ubiquitous symptomless presence of unstable L-forms of Micrococcaceae in the circulating blood has been described[3–5] and further confirmed, following other authors'[6] and our own observations. This presence appears to be correlated to a state of immunological tolerance (manuscript in preparation). Previous researches[7] have demonstrated the placental transmission of *Haemobartonella muris*, a microorganism which, from the point of view of other authors and our-

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