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URINARY EXCRETION OF DEOXYCYTIDINE: A POTENTIAL BIOCHEMICAL RADIATION DOSIMETER

by
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excretion occurs within the first 24 hours following irradiation, and therefore parallels the time at which the maximum loss of DNA from spleen occurs (19,21). A correlation between DNA degradation and CdR excretion is also suggested by the similar temporal sequence in the urine CdR increase and the appearance of polydeoxynucleotide (PDN) in spleen (19). As recently demonstrated, the appearance of PDN represents a catabolic process in the radiation induced loss of cellular and tissue DNA and is probably mediated through the action of specific DNases (22). In an analogous manner the elevated excretion of the nucleoside, pseudouridine, after irradiation has also been attributed to the degradation of nucleic acid (soluble RNA) in the spleen (5).

While this evidence implies that the catabolism of DNA is a prominent factor in the urinary CdR increase, the role of an additional impairment in DNA synthesis must be considered. Parizek (1) speculated that the specificity of CdR as an indicator of DNA degradation was in part related to the normal deficiency of enzymatic pathways for its utilization. However, CdR has been shown to be not only actively deaminated after phosphorylation, with the subsequent accumulation of deoxyuridine, thymidine (TdR) and uracil, but to be incorporated as well into DNA (23,24). Further, the incorporation of labeled CdR into DNA has been observed to be depressed in irradiated thymus and regenerating liver (24,25). While the detailed mechanisms involved are beyond the scope of this discussion, the extensive literature on the radiation-induced damage of DNA priming ability and/or its enzymatic activation indirectly imply some inherent alteration in CdR utilization (26,27). Moreover, the fall in tissue levels of the crucial enzymes deoxycytidylate deaminase and thymidylate synthetase indicates that the block in DNA synthesis may reside at an early stage in the pathway of CdR incorporation into DNA (24). A schema summarizing the possible biochemical sources of the increased CdR excretion after irradiation, is given in Figure 4.

The physiologic significance of tissue CdR in relation to the intermediary metabolism of the pyrimidine compounds is at present highly conjectural. The possibility that urinary CdR might function biologically in a manner analogous to uric acid in purine metabolism would appear unlikely in view of the active utilization of the former in DNA synthesis. A relationship between CdR and DNA turnover has been noted, however. The tissue levels of this compound are increased not only in regenerating liver but also in various tumors (28-30). Our present finding that the excretion of CdR bears also some relationship to the growth status of these animals in terms of age is of interest, and is consistent with similar associations at a tissue level. Thus the urinary excretion of CdR might be useful as an indicator of tissue growth of a cellular (DNA) nature in a manner similar to other urine metabolites (i.e., hydroxyproline for bone turnover). The present

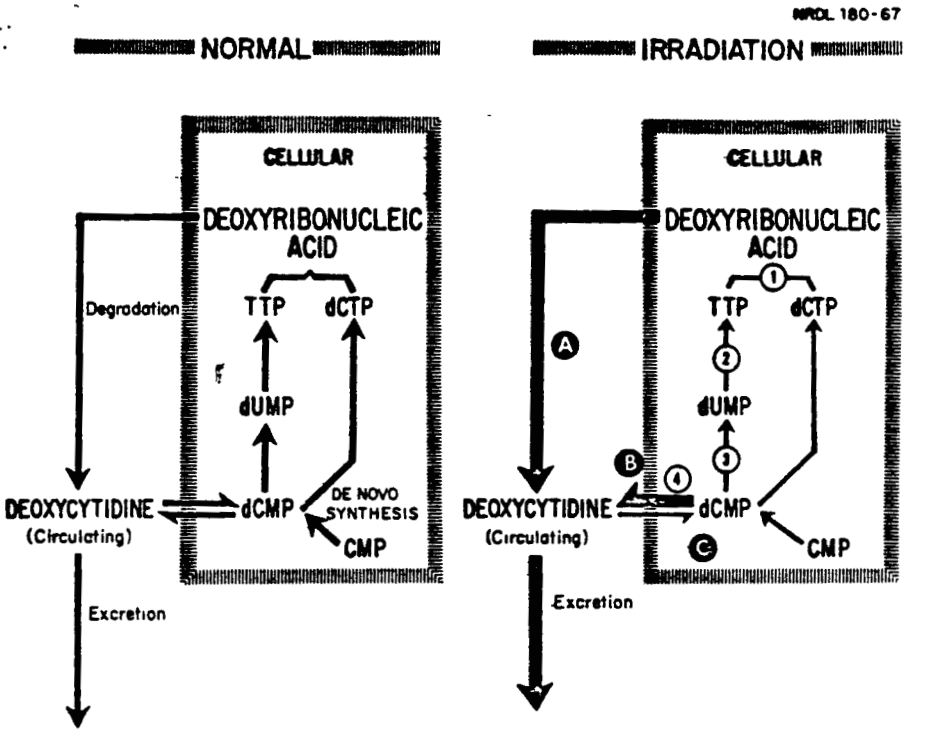

Figure 4. Schema of biochemical events leading to the urinary excre of deoxycytidine in the normal and X-irradiated rat.

observation that the increase in the excretion of CdR after irradiation bears a relationship to the age of the animals, requires further study although the relationship of age and radiation susceptibility has been studied (31).

In theory, an elevation in the excretion of CdR might also represent an increased synthesis of DNA related to tissue regeneration or cellular repair. The definite response observed at doses (10 to 50 R), which are borderline doses in terms of visible morphologic or biochemical (i.e., DNA loss) effects, may thus be an expression of alterations oriented towards the repair of nucleic acids after radiation damage. Skurikhina and Tereshchenko (4) have reported that the increase in urinary CdR after a minimal radiation dose (7 R) occurs at a time suggestive of a repair process; and other work on excretion of Dische-positive substances is also consistent with this possibility (9). More recent studies on thymidine, another urinary metabolite of DNA, after variable doses of radiation have been similarly interpreted (32). Further studies along these lines are therefore definitely indicated.

Finally, the demonstration of these alterations in the urinary excretion of CdR after irradiation is of practical significance. While this phenomenon has been reported predominantly in rats, there has been some suggestion of a similar finding in man. Saenger, indeed, has noted increased excretions of CdR in humans after therapeutic radiation doses of 237 R and 263 R (10). Present studies are being conducted along these lines, with appropriate modifications of the analytical methods being adopted for routine use, and the possible application of CdR as a biochemical radiation dosimeter.

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