

## THE EFFECT OF DIETARY MENHADEN OIL ON ULTRA-VIOLET RADIATION INDUCED SKIN CANCER IN THE HAIRLESS MOUSE

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Previous experiments with the Skh-ultraviolet radiation (UVR) induced skin tumour model have shown a requirement for polyunsaturated fatty acids (PUFA) for the promotion of these tumours (Reeve et al. 1988). This need may be for the essential PUFA that form prostaglandins (pg's), specifically  $pgE_2$ . Through dietary intervention we hoped to alter the available essential fatty acids by feeding  $\omega_3$  rather than  $\omega_6$  PUFA and therefore altering subsequent prostaglandin formation.

Skh:HR-1 mice 6 to 8 weeks old were age matched in groups of twenty. All groups were fed a semipurified diet based on that recommended by the AIN (1977). Each group was fed a different fat type or level with the caloric difference adjusted by changing the level of carbohydrate. The diets were made fortnightly and stored in the dark at 4°C. The mice were fed isocalorically, weighing and feeding a determined quantity of diet daily to maintain normal body weight. One group was fed a 5% sunflower oil diet (control), another two groups 5% menhaden oil, a fourth group 20% sunflower oil and the fifth and sixth groups 20% menhaden oil.

After two weeks on the semipurified diet the mice were exposed to ten weeks of five day per week UVR starting with a minimal erythema dose (MED) of ten minutes increasing this time each week by 20% to maintain a MED. Tumour growth was then monitored for the next 200 days. One of each group on the menhaden oil diet were fed this oil during the ten week period of exposure to UVR and then changed to a sunflower oil diet. The second group were fed the sunflower oil during the UVR exposure period and transferred to the menhaden oil diet whilst tumour growth was monitored. The same fat quantity was maintained for each group throughout the experiment.

We found the menhaden oil, when fed at the 5% level, at either stage provided no protection against UVR-induced tumour growth. When menhaden oil was fed at the 20% level we observed tumour promotion.

At the end of the tumour monitoring period a sample of six mice from each group were age matched with unexposed mice on a standard pellet diet and their immune status measured using the contact hypersensitivity assay. All mice on a 20% fat diet were immunosuppressed compared to those on the 5% diet or unexposed mice on pellets.

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