between 30 pg/mL (group 2) and 56 pg/mL (group 3), with standard deviations ranging from 39% to 60%. They did not find any difference between the groups. Single measurements of 17β-estradiol without further specification of phase of the estrous cycle do not necessarily reflect differences in exposure of estrogen-sensitive tissue to estrogen. It is, therefore, not warranted to draw any conclusion from these data with respect to the effects of dietary fiber on estrogen metabolism.

In our study (2), mean plasma 17β-estradiol levels were 10 pg/mL and 12 pg/mL in the high-fiber and low-fiber groups, respectively, during the basal period of the cycle (estru, metestrus, and 1st day of diestrus). During the peak period (2nd day of diestrus and proestrus), a significantly higher plasma 17β-estradiol level was found in the high-fiber group (mean level, 42 pg/mL) than in the low-fiber group (mean level, 29 pg/mL). We also measured a threefold higher fecal estrogen excretion and a significantly lower urinary estrone excretion in the high-fiber group compared with the low-fiber group. We concluded that dietary fiber affects the estrogen balance by interrupting the enterohepatic circulation.

We conclude that in studies using different diets, differences in energy intake between groups must be taken into account and that more detailed studies are necessary to elucidate the intermediate role of dietary fiber on estrogen metabolism.

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References


Response

It is very difficult to compare our study with that of Arts et al., based on the information supplied, since a number of key variables, such as the source of wheat bran and the time of diet initiation, differed in the two studies. Nonetheless, it is agreed that lengthening the duration of the experiment may be useful as a means to determine the long-term protective effects of wheat bran fiber on mammary tumorigenesis. It is not clear from the letter whether the high-fat group with or without fiber exhibited lower mean plasma 17β-estradiol levels, but a decrease in plasma 17β-estradiol in the fiber-supplemented groups would be consistent with results obtained in earlier human studies. We also found a significant decrease in urinary estrogen excretion in the fiber-supplemented groups; however, we found no changes in either cytosolic and nuclear hepatic estrogen receptors or the estrogen-inducible hepatic enzyme alanine aminotransferase (data presented by M. E. Kendall at the June 1991 meeting of The Endocrine Society, in Washington, D.C.). Hence, the hypothesis that dietary fiber exerts its protective effects on breast cancer by altering estrogen balance via disruption of the enterohepatic recirculation of estrogens remains to be convincingly demonstrated.

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References


Response

While Dr. Wein’s comparison between tumor suppression and Dawe’s growth-regulated replicators hypothesis is valid to a degree, we believe that the two phenomena are probably not completely analogous. Individual cells within a multicellular organism are not subjected to natural selection pressures in the same way that the whole organism is, although parallels between these two ecosystems certainly do exist. Since we are by no means experts on evolutionary biology, however, we feel it would be irresponsible for us to comment further on Dr. Wein’s stimulating interpretation.

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