

**Exercise in colon cancer modulation: an experimental approach # 20**

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Carcinogenesis is a complex process best characterized as an accumulation of alterations in genes regulating cellular homeostasis. The whole process can be divided into three main stages: initiation, promotion, and progression. It has been observed that moderate and regular physical activity (PA) may prevent cancer, mainly colon cancer (CC), up to 50% in humans. The evidence that PA protects against colon cancer is convincing. Also, PA probably protects against postmenopausal breast cancer and cancer of the endometrium. On the other hand, exhaustive exercise increases free radical DNA oxidative damage, inflammation and depresses immune function, events also related to the increased risk for cancer development. Nevertheless, the mechanisms involved in both PA effects remain largely unknown and poorly studied. Understanding the mechanisms that link PA with cancer is useful to identify plausible mechanisms and associations between PA and cancer; to provide evidence for implementing interventions on clinical and public-health levels; to define exercise prescription for people without and with cancer; and to identify new clues to cancer biology, which might help in designing other cancer prevention and treatment modalities. Because of the complexity and heterogeneity of activity in people, animal models for carcinogenesis and PA present the opportunity to study the amounts and types of PA and biomarkers that influence carcinogenesis in controlled environments. Epithelial cell proliferation and aberrant crypt foci (ACF) have been used for early detection of factors that influence colorectal carcinogenesis in rats and can be induced by the colon carcinogen dimethyl-hydrazine (DMH). This interesting animal-tumor model is possible a useful approach for studying the influence of exercise during the initiation and post initiation period, and has already contributed to the current understanding of colon carcinogenesis and PA relationship.

**Key words:** Colon cancer; chemical colon carcinogenesis model in rats; epithelial cell proliferation; physical activity.