

Research article

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Diet and body constitution in relation to subgroups of breast cancer defined by tumour grade, proliferation and key cell cycle regulatorsSigne Borgquist^{1,2}, Elisabet Wirfält³, Karin Jirstrom¹, Lola Anagnostaki¹, Bo Gullberg³, Göran Berglund³, Jonas Manjer⁴ and Göran Landberg¹¹Division of Pathology, Department of Laboratory Medicine, Malmö University Hospital, S-205 02 Malmö, Sweden²Division of Oncology, Department of Clinical Sciences, Lund University Hospital, S-221 85 Lund, Sweden³Department of Clinical Sciences, Malmö University Hospital, S-205 02 Malmö, Sweden⁴Department of Surgery, Malmö University Hospital, S-205 02 Malmö, SwedenCorresponding author: Göran Landberg, goran.landberg@med.lu.se

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Breast Cancer Research 2007, **9**:R11 (doi:10.1186/bcr1644)This article is online at: <http://breast-cancer-research.com/content/9/1/R11>© 2007 Borgquist *et al.*; licensee BioMed Central Ltd.This is an open access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/2.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.**Abstract**

Background The general lack of clear associations between diet and breast cancer in epidemiological studies may partly be explained by the fact that breast cancer is a heterogeneous disease that may have disparate genetic associations and different aetiological bases.

Method A total of 346 incident breast cancers in a prospective cohort of 17,035 women enrolled in the Malmö Diet and Cancer study (Sweden) were subcategorized according to conventional pathology parameters, proliferation and expression of key cell cycle regulators. Subcategories were compared with prediagnostic diet and body measurements using analysis of variance.

Results A large hip circumference and high body mass index were associated with high grade tumours ($P = 0.03$ and 0.009 , respectively), whereas low energy and unadjusted fat intakes were associated with high proliferation ($P = 0.03$ and 0.004 ,

respectively). Low intakes of saturated, monounsaturated and polyunsaturated fatty acids were also associated with high proliferation ($P = 0.02$, 0.004 and 0.003 , respectively). Low energy and unadjusted fat intakes were associated with cyclin D₁ overexpression ($P = 0.02$ and 0.007 , respectively), whereas cyclin E overexpression was positively correlated with fat intake. Oestrogen receptor status and expression of the tumour suppressor gene p27 were not associated with either diet or body constitution.

Conclusion Low energy and low total fat (polyunsaturated fatty acids in particular) intakes, and high body mass index were associated with relatively more malignant breast tumours. Dietary behaviours and body constitution may be associated with specific types of breast cancer defined by conventional pathology parameters and cyclin D₁ and cyclin E expression. Further studies including healthy control individuals are needed to confirm our results.

Introduction

The aetiology of breast cancer is complex, but a number of risk factors have been identified. In human studies body mass index (BMI) and hip and waist circumferences have been described as positively associated with risk for developing breast cancer [1]. Animal experiments support these findings and report that obesity enhances the development of mammary tumours [2]. To our knowledge, no studies have been

conducted to evaluate whether there is an eventual association between obesity and tumour characteristics in animals.

The association between dietary intake and body composition is still under debate [3,4], and the biological link between diet and breast cancer is unclear. Dietary studies indicate that diet may contribute factors that either promote or protect against breast cancer [5]. High alcohol intake, for example, has been

BMI = body mass index; ER = oestrogen receptor; MDCS = Malmö Diet and Cancer Study; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids; SFA = saturated fatty acids; TMA = tissue microarray.