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Polymorphisms in the *ABCB1* gene and effect on outcome and toxicity in childhood acute lymphoblastic leukemia

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The membrane transporter P-glycoprotein, encoded by the *ABCB1* gene, influences the pharmacokinetics of several anti-cancer drugs. We hypothesized that 1199G>A, 1236C>T, 2677G>A/T and 3435C>T variants of *ABCB1* affect outcome and toxicity in childhood acute lymphoblastic leukemia (ALL), since the treatment includes known P-glycoprotein substrates and 3435C/T may affect methotrexate effects.

We studied 522 Danish children with ALL treated according to NOPHO ALL92 and ALL2000 protocols, 93% of all those eligible during 1992-2007. Risk of relapse was increased 2.9-fold for 41 patients with the 1199GA variant compared to 477 with 1199GG (p=0.001), and reduced by 61% and 40%, respectively for 421 patients with the 3435CT or 3435TT variants compared to 96 patients with 3435CC (overall p=0.02).

The degree of bone marrow toxicity during doxorubicin, vincristine and prednisolone induction therapy was higher in 71 patients with 3435TT variant (median nadirs: hemoglobin 3% and platelets 34/37% lower in 3435CT/3435CC) compared to 160 patients with 3435CT/3435CC (Hemoglobin p=0.01 and platelets p<0.0001).

We observed more liver toxicity after high-dose methotrexate in 109 patients with 3435CC variant versus 3435CT/TT (Median max alanine aminotransferase: 280 versus 142/111 U/L, p=0.03).

In conclusion, there is a statistically significant association between *ABCB1* polymorphisms and efficacy and toxicity in childhood ALL.

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Sexual frustration as the cause of Breast cancer in women: How correlations and cultural blind spots conceal causal effects

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The main premise of this hypothesis is that breast cancer is caused by sexual frustration. Sexual frustration is triggered by multiple forms of dissonance between the absence or lack of sexual reward and the (un)conscious motivation to obtain these sexual rewards. Study assumes that neural and hormonal processes are capable of adjusting or distorting biologically active forms of specific sex hormones depending on experienced sexual stimuli. It is hypothesized that prolonged sexual frustration will ultimately lead via aberrantly metabolized sex hormones to the development of breast cancer. Human female sexual behavior research links sexual frustration with breast cancer risk. The distinction between human female sexual behavior and reproduction is crucial to understand breast cancer risk. Current explanations are focused on reproduction.

However, human female sexual behavior is causal in breast cancer development and androgens rather than estrogens are crucial for sexual behaviors in women. Social learning is the main determinant of human sexual behaviors that is why cultural and social processes are very important to understand breast cancer risk. Epidemiologists should evaluate breast cancer risk based on cultural female attitudes towards sexually related issues. Female mate choices should be examined for (un)conscious cultural, ethnic, religious, and socioeconomic pressure to make a thorough assessment of breast cancer risk. Closer examination of (un)conscious female copulation strategies reveal that they are potential sources of sexual frustration in specific groups of women. Postmenopausal women seem vulnerable for self-fulfilling prophecies about post reproductive sexuality, body image, and negative perceptions of menopause which may cause sexual frustrations.

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