J Cancer Sci Ther 2016, 8:9(Suppl)
http://dx.doi.org/10.4172/1948-5956.C1.085

Validity and reproducibility of a food frequency questionnaire for dietary factors related to colorectal cancer

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Diet has been suggested to play a major role in the onset of colorectal cancer. To date, the validity of a comprehensive FFQ to assess risk factors for colorectal cancer had not been assessed in an elderly population. Hence, this study evaluated the ability of a self-administered semi-quantitative Food Frequency Questionnaire (FFQ) to measure consumption of foods and nutrients related to the development of colorectal cancer in a population >50 years old in Flanders, Belgium. The FFQ was administered during two days in a period of two weeks. The FFQ approach was validated by comparing to the 3-day diary method. Spearman correlations, Wilcoxon signed rant, Kappa tests and Bland-Altman plots were used to assess measurement agreement. 162 respondents (mean age 57.5 years, 59% female) provided data for the FFQ, of whom 156 also participated in the validity assessment. Correlation and classification agreement tests showed high reproducibility of the FFQ for measuring food and nutrient intake. For most of the foods and nutrients there was no significant difference between the first and second FFQ assessment in estimating foods and nutrients at a population level. Intake of 'fresh red meat', 'total red meat', 'total red meat plus meat products' and 'alcohol drinks' however, were significantly higher based on the FFQ compared to the 3-day diary method. For nutrients, Bland-Altman statistics revealed that only the mean difference in estimated alcohol intake differed substantially between both methods. For both the validity and reproducibility, measurement agreement at individual level varied widely at higher level of foods and nutrients intake. Therefore, the FFQ is a valid tool to estimate intake of foods and nutrients at a lower intake level to assess dietary exposure to colorectal cancer in an elderly population.

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Targeting histone H3K36me3-deficient cancers

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Histone H3 lysine 36 trimethylation (H3K36me3) plays a central role in both orchestrating the DNA damage response and in suppressing tumorigenesis. Accordingly SETD2-dependent H3K36me3 is frequently lost in a number of cancer types, including high-grade pediatric gliomas (>50%) and metastatic renal carcinomas (~60%), for which prognosis is poor. These findings identify loss of this histone mark to be an important potential therapeutic target. We have identified an evolutionarily conserved synthetic lethal interaction between histone H3K36me3 deficiency and inhibition of the cell cycle regulator WEE1. We show that H3K36me3-deficient human cells are selectively killed with the WEE1 inhibitor, AZD1775. Cell death is associated with replication stress, DNA damage and apoptosis. An *in vivo* experiment in SETD2-deficient A498 xenografts showed that treatment with AZD1775 regress tumors, producing a marked reduction in tumor size compared with vehicle-treated control. We have also developed a companion biomarker to detect H3K36me3 loss in patient tissue microarrays (TMAs). As AZD1775 is already in Phase II clinical trials, we anticipate these findings will be of clinical relevance.

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