Kære ATU'er,

Vedhæftet er 3 abstracts som vi vil bruge på Introduktion til Sundhedsvidenskab. Vi forventer at I læser dem og får en idé om det overordnede emne, men ikke at I forstår dem i detaljer.

Med venlig hilsen,
Jenny, Anne-Mette, Stine og Jakob

Abstract 1

Lancet. 2003 Sep 20;362(9388):933-40.

Five compared with six fractions per week of conventional radiotherapy of squamous-cell carcinoma of head and neck: DAHANCA 6 and 7 randomised controlled trial.

Overgaard J, Hansen HS, Specht L, Overgaard M, Grau C, Andersen E, Bentzen J, Bastholt L, Hansen O, Johansen J, Andersen L, Evensen JF.

Erratum in
Lancet. 2003 Nov 8;362(9395):1588.

Abstract

BACKGROUND:
Although head and neck cancer can be cured by radiotherapy, the optimum treatment time for locoregional control is unclear. We aimed to find out whether shortening of treatment time by use of six instead of five radiotherapy fractions per week improves the tumour response in squamous-cell carcinoma.

METHODS:
We did a multicentre, controlled, randomised trial. Between January, 1992, and December, 1999, of 1485 patients treated with primary radiotherapy alone, 1476 eligible patients were randomly assigned five (n=726) or six (n=750) fractions per week at the same total dose and fraction number (66-68 Gy in 33-34 fractions to all tumour sites except well-differentiated T1 glottic tumours, which were treated with 62 Gy). All patients, except those with glottic cancers, also received the hypoxic radiosensitiser nimorazole. Analysis was by intention to treat.

FINDINGS:
More than 97% of the patients received the planned total dose. Median overall treatment times were 39 days (six-fraction group) and 46 days (five-fraction group). Overall 5-year locoregional control rates were 70% and 60% for the six-fraction and five-fraction groups, respectively (p=0.0005). The whole benefit of shortening of

treatment time was seen for primary tumour control (76 vs 64% for six and five fractions, p=0.0001), but was non-significant for neck-node control. Six compared with five fractions per week improved preservation of the voice among patients with laryngeal cancer (80 vs 68%, p=0.007). Disease-specific survival improved (73 vs 66% for six and five fractions, p=0.01) but not overall survival. Acute morbidity was significantly more frequent with six than with five fractions, but was transient.
INTERPRETATION:
The shortening of overall treatment time by increase of the weekly number of fractions is beneficial in patients with head and neck cancer. The six-fractions weekly regimen has become the standard treatment in Denmark.

Abstract 2

Am J Epidemiol. 2007 Jun 15;165(12):1372-9. Epub 2007 Mar 16.

Is prenatal exposure to tobacco smoking a cause of poor semen quality? A follow-up study.

Ramlau-Hansen CH1, Thulstrup AM, Storgaard L, Toft G, Olsen J, Bonde JP.

Abstract

A few studies indicate that exposure to maternal smoking during fetal life decreases semen quality in adult life, but the results are inconsistent and retrospectively collected smoking data were used in most studies. From a Danish pregnancy cohort established in 1984-1987, 347 of 5,109 sons were selected according to their exposure to tobacco smoke in fetal life. From February 2005 to January 2006, a semen sample from the 347 men was analyzed for conventional semen characteristics according to standardized criteria by using a mobile laboratory. The authors found an inverse association between maternal smoking during pregnancy and total sperm count (p = 0.002). Men exposed to more than 19 cigarettes daily during pregnancy had approximately 19% lower semen volume (p = 0.04), 38% lower total sperm count (p = 0.11), and 17% lower sperm concentration (p = 0.47) compared with unexposed men. The odds ratio for oligospermia was 2.16 (95% confidence interval: 0.68, 6.87) among exposed men compared with the unexposed. No associations were found for sperm motility or morphology. These results indicate that prenatal exposure to tobacco smoke may have an adverse effect on semen quality and, if these associations are causal, they could explain some of the reported differences between populations and secular changes in semen quality.

Abstract 3

Chem Biol Interact. 2015 Feb 5;227:37-44. doi: 10.1016/j.cbi.2014.12.032. Epub 2015 Jan 2.

Betanin reduces the accumulation and cross-links of collagen in high-fructose-fed rat heart through inhibiting non-enzymatic glycation.

Han J, Tan C, Wang Y, Yang S, Tan D.

Abstract

We attempted to determine whether betanin (from natural pigments) that has antioxidant properties would be protective against fructose-induced diabetic cardiac fibrosis in Sprague-Dawley rats. Fructose water solution (30%) was accessed freely, and betanin (25 and 100 mg/kg/d) was administered by intra-gastric gavage continuously for 60 d. Rats were sacrificed after overnight fast. The rat blood and left ventricle were collected. In vitro antiglycation assay in bovine serum albumin/fructose system was also performed. In rats treated only with fructose, levels of plasma markers: glucose, insulin, HOMA and glycated hemoglobin rised, left ventricle collagen accumulated and cross-linked, profibrotic factor-transforming growth factor (TGF)-β1 and connective tissue growth factor (CTGF) protein expression increased, and soluble collagen decreased, compared with those in normal rats, showing fructose induces diabetic cardiac fibrosis. Treatment with betanin antagonized the changes of these parameters, demonstrating the antifibrotic role of betanin in the selected diabetic models. In further mechanistic study, betanin decreased protein glycation indicated by the decreased levels of protein glycation reactive intermediate (methylglyoxal), advanced glycation end product (N(ε)-(carboxymethyl) lysine) and receptors for advanced glycation end products (AGEs), antagonized oxidative stress and nuclear factor-κB activation elicited by fructose feeding, suggesting inhibition of glycation, oxidative stress and nuclear factor-κB activation may be involved in the antifibrotic mechanisms. Betanin also showed anitglycative effect in BSA/fructose system, which supported that anitglycation was involved in betanin's protective roles in vivo. Taken together, the potential for using betanin as an auxillary therapy for diabetic cardiomyopathy deserves to be explored further.