Results: For a scan in the head region going from Head1 to Head2 protocol the dose to lens. For the 5 years old child the dose is reduced from 6,6mGy to 1,7mGy. For the 4 years old child form 6,6mGy to 1,4mGy. For a scan in the Pelvis region changing the protocol from Thorax to Pelvis increased the dose to the Breast from 0,2 to 0,7mGy and Gonads from 13,6 to 57,8mGy for a 5 years old child. For a 10 years old child the breast dose is increased from 0,1 to 0,4 mGy and gonads from 11,8 to 46,0 mGy. 

Conclusion: It matters what protocol is used for the kVCBCT uptake. It is possible to reduce the dose remarkably when choosing the most optimized protocol. Changing the scan range for head to avoid the lens reduce the lens dose with 471%. Another area where the scan range could be of great interest is the thorax region for girls. The bone match is just as good with the thorax protocol. As seen on figure 1 the image quality drops going from pelvis to thorax protocol in the pelvic areas, but the opportunity for bone match is just as good with the thorax protocol.

Purpose or Objective: To investigate if VMAT shows any advantage in terms of reduction of second cancer risk (SCR) compared to 3DCRT using different high dose fractionation schemes in patients treated with RT for rectal cancer (RC).

Material and Methods: Twenty-five patients with stage I–III RC and pre- or postoperative RT were included in this ethics-approved retrospective study. Planning CT data prior to RT were used. CTV for rectal cancer was delineated using RTOG contouring atlas. Organs at risk (OAR) (ICRP 2007) contoured on each CT data set were bladder, colon, sigmoid, bone, gonads, uterus, skin, small intestine, muscle, anus. PTV=C-CTV+5 mm. 3-field technique 6/15 MV 3DCRT and 6 MV VMAT plans were created (Eclipse, v.10, AAA-algorithm). Doses prescribed were 25x1.8 Gy and 5x5 Gy, respectively.

Carcinogenesis model to estimate SCR emphasizes cell kinetics of radiation-induced cancer by mutational processes was used, integrating cell sterilization processes described by the LC model and repopulation effects. Model parameters were obtained by fits to epidemiological, cancer specific carcinogenesis data for carcinoma and sarcoma induction. From DVHs of structures of interest SCR in relation to organ equivalent dose (OED) was calculated. OED was converted to excess absolute risk for a western population for each organ as well as for all organs together. Resulting lifetime SCR from specific radiotherapy treatment was determined by lifetime attributable risk (LAR) by an integration of excess absolute risk from age at RT to lifetime expectancy (90 years).

Results: Mean LAR was highest for organs adjacent or close to PTV. Total LAR for VMAT and 3DCRT was 2.4-3.0% and 2.0-2.7%, respectively. For 5x5 Gy LAR was 1.4-1.9% for VMAT and 1.2-1.6% for 3DCRT and half as high as using 25x1.8 Gy. Median percentage LAR difference for OAR was significantly higher for VMAT irrespective of fractionation, and highest for bladder and colon. Individual differences in LAR ranged from 0.2-15.9% for 25x1.8 Gy and 0.1-9.6% for 5x5 Gy. Size and shape of PTV influenced SCR, and was highest for the largest PTV (RC). Median percentage SCR was 1.2-3.4% for VMAT and 1.0-1.7% for 3DCRT.

Conclusion: For bladder and colon LAR is lower using 3DCRT, however difference is small. Compared to epidemiological data (Birgisson J Clin Oncol 2005) SCR is smaller when using a hypofractionated schedule treating RC. Total SCR is 2% at normal life expectancy. Risk is highest for young patients.