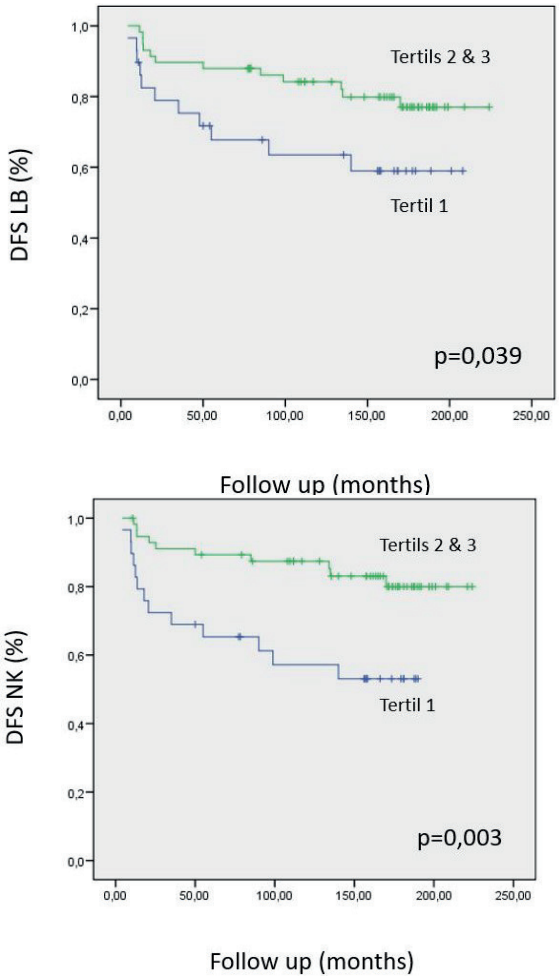
Conclusion
A mechanistic model for radiation response of normal human cells was successfully modified to simulate measured in vitro CS of 19 cancer cell lines. Independent of cancer entity, the radiobiological value ß was predictable only with known GS while the prediction of α additionally required at least knowledge of the p53 mutation status. An observed correlation of GS with the number of chromosomes and nucleus size, both clinically accessible from a biopsy prior to treatment, may facilitate individualized radiotherapy based on cell-specific survival prediction.

EP-2333 NK and B cells are novel prognostic factors for disease free survival in cervical carcinoma patients

R. Ordoñez Marmolejo[1], L.A. Henriquez-Hernandez[2], B. Pinar[3], M. Federico[3], M. Lloret[3], M. Rodriguez Ibarria[3], I. Ribeiro[3], L. Garcia-Cabrera[3], C. Rodríguez-Gallego[4], P.C. Lara[3]
[1]*Virgen de la Victoria, ONCOLOGIA RADIOTERAPICA, Málaga, Spain*
[2]*Universidad de las Palmas, Clinical Sciences Department, Las Palmas Gran Canaria, Spain*
[3]*Hospital Dr. Negrin, Radiation Oncology, Las Palmas de Gran Canaria, Spain*
[4]*Hospital Dr. Negrin, Immunology Department, Las Palmas de Gran Canaria, Spain*

Purpose or Objective
A close relationship exists between immune response and tumor behavior. Cervix carcinoma is related to HPV infection. Both innate immunity and adaptative antibody mediated immunity could have a role in this disease. This study aimed to explore the associations between NK and B cells radiation-induced apoptosis (RIA) in peripheral blood lymphocytes (PBL) as prognostic factor for survival in cervical carcinoma patients.
Material and Methods
Between February 1998 and October 2003, 94 consecutive patients with nonmetastatic, localized stage I-IV cervical carcinoma who had been treated with radiotherapy (RT) ± chemotherapy ± surgery were included in this study. Follow-up ended in September 2016.
PBL subpopulations were isolated and irradiated with 0, 1, 2 and 8 Gy, then incubated for 48 h. Apoptosis was measured by flow cytometry and the ß value, a parameter defining RIA of lymphocytes, was calculated.
Results
Mean follow-up duration was 139± 58,6 months. Patients with lower B and NK lymphocyte ß values were at a higher risk of relapse: Exp(B) = 0,873, confidence interval (CI) 95 % = 0,770-0,990, p = 0.034; and Exp(B)= 0,951, confidence interval (CI) 95 % = 0,910-0,994, p = 0.026, respectively.
To visualize these observations, ß values were segmented according to tertils. Tertil 1 (lower ß values) was found a clinically relevant cut-off in the Kaplan-Meier analysis (figure 1). In multivariate analysis, age, stage, histology, grade, primary treatment, NK and B cells data were included. Only the clinical stage (p=0,001) and NK ß values (p=0,050) were found to be of prognostic significance for disease-free survival.



Conclusion
For the first time, RIA in B and NK cells was demonstrated to be a predictive factor for disease free survival in cervical carcinoma patients with long follow-up.

EP-2334 A method to assess radiation-induced DNA damage and microenvironmental parameters in tumor spheroids

A. Menegakis[1], B. Van den Broek[2], R. Medema[1]
[1]*Netherlands Cancer Institute, Division Cell Biology, Amsterdam, The Netherlands*
[2]*Netherlands Cancer Institute, Division Cell Biology- Bio-imaging facility, Amsterdam, The Netherlands*

Purpose or Objective
Tumour cell response to radiation-induced damage shows high heterogeneity which is further influenced by local tumour microenvironmental parameters, such as the existence of tumour hypoxia. Assessment of inter-cellular variability in radiation response based on quantification of radiation-induced DNA double strand breaks (DSBs) in tumour tissue or 3D models is increasingly used. However, these methods are often hampered from the small number of cells evaluated, the applied randomization process of nuclei selection and inter-observer variability. Our aim was to develop an automated, objective, unbiased method that allows quantitative analysis of radiation-induced DSBs in tumour cells of multicellular tumour spheroids.