chemotherapy, surgery, radiotherapy, or with metastatic cancer constituting a 'high risk' group. All others were considered 'average risk'.
Results: 16 studies, published in 20 papers, were identified for inclusion. The data from the included studies consistently reported annual VTE incidence rates of between 2−10%, depending on the populations studied. The highest incidence cancer types for average risk patients were bone (75.47/1000; 95% CI: 33.91, 167.99) and brain malignancy (64.27/1000; 95% CI: 45.92, 89.95), and for high risk patients were pancreatic cancer (100.74/1000; 95% CI: 66.95, 151.60) and brain malignancy (96.93/1000; 95% CI: 36.28, 258.96).
Conclusions: Venous thromboembolism is common among cancer patients, and there is considerable variation in risk by cancer type and in those with additional risk factors for VTE. Venous thromboembolism is a devastating complication which, with adequate treatment, may be prevented from occurring to a reasonable degree. This review highlights patient groups at particular risk who may well benefit from targeted strategies to deliver antithrombotic interventions.

64 Subtype-specific risk of testicular germ-cell tumours among immigrants and their descendants in Sweden, 1960–2007

O. Beiki[1], F. Granath[2], P. Allebeck[3], O. Akre[2], T. Moradi[1]. [1]*Karolinska Institutet, Institute of Environmental Medicine, Stockholm, Sweden,* [2]*Karolinska Institutet, Department of Medicine, Stockholm, Sweden,* [3]*Karolinska Institutet, Department of Public Health Sciences, Stockholm, Sweden*

Background: Testicular cancer is the most common cancer among young male adults in many populations and there has been an unexplained dramatic increase in its occurrence in several populations over the past decades. To elucidate the importance of genetic and environmental factors in testicular cancer etiology and to gain insight into the potential timing of exposures; we compared histo-pathological subtype-specific risk of testicular cancer among migrants and their descendants to that of Swedish-born men.
Material and Methods: A nation-wide cohort of 3.6 million men aged 15−54 years was followed between 1960 and 2007 through the linkage between Swedish National Registers including Total Population Register, Cancer Register, Cause of Death, and Multi-Generation Register. Incidence rate ratio (IRR) adjusted for age and calendar period of follow-up with 95% confidence intervals (CIs) was estimated using Poisson regression models.
Results: 5,801 cases of testicular cancer occurred during 80 million person-years of follow-up. First-generation immigrants had a lower risk compared with Swedish-born men (IRR = 0. 66; 95% CI = 0.60–0.72). The risk among first-generation immigrants varied remarkably by birthplace, reflecting the risk in their countries of birth. The risk of testicular seminomas was statistically significantly modified by age at immigration and duration of residence among immigrants born in high-risk areas ($P_{homogeneity}$ = 0.004 and 0.05, respectively). We observed a statistically significantly convergence of risk among second-generation immigrants toward the risk in Sweden (RR = 1.02; 95% CI = 0.93–1.12). This convergence was regardless of the risk level (high or low) in the parental country of birth. The risk among second-generation immigrants was not affected by duration of stay of their mothers in Sweden before pregnancy.
Conclusions: Our study provides evidence that interaction between exposures in uterus and after birth might be important in the development of testicular cancer.

65 A noble melanoma discrimination index based on hyperspectral data

H. Okutani[1], T. Nagaoka[2], A. Nakamura[2], Y. Kiyohara[3], T. Sota[1]. [1]*Waseda University, Department of Electrical Engineering and Bioscience, Tokyo, Japan,* [2]*Waseda University, Waseda Research Institute For Science and Engineering, Tokyo, Japan,* [3]*Shizuoka Cancer Center Hospital, Dermatology Division, Shizuoka, Japan*

Backgrounds: It is well known that noninvasive and untouchable diagnosis of malignant melanoma at an early-stage is very important to reduce melanoma-related mortality rate. Therefore a lot of automated melanoma screening systems have been studied. Objective of this study is to develop a melanoma discrimination index based on hyperspcetral data (HSD), which consist of both spatial and spectral information.
Material and Methods: 157 HSD (52 HSD from 5 melanoma patients, 95 HSD from 11 seborrheic keratosis (SK) patients and 10 HSD from 4 volunteers with nevus: The patients and volunteers were all Japanese) were measured using a newly designed hyperspectral imager. A spectrum of each pixel was considered as a multi-dimensional vector, and a spectral angle between the vector and a reference vector was calculated. Here the reference vector was defined by an average spectrum of typical normal skin. An entropy index was calculated every HSD using the probability of finding a spectral angle and regarded as a melanoma discrimination index. Statistical tests were performed to verify the effectiveness of the proposed index. Statistical significance was set to be 5%. Receiver operating characteristic (ROC) analysis was also made. The present study was approved from the Institutional review board at Shizuoka Cancer Center.
Results: Mann–Whitney U test revealed that the present index was useful to discriminate melanomas from SK and nevus. An area under the ROC curve was 0.93 with the present index, while it was 0.77 with the pseudo-fractal dimension based index which we had proposed previously. A linear discrimination analysis gave an accuracy of 91.1%, a sensitivity of 92.3% (95% CI: 85.0–99.6%), and a specificity of 90.5%(95% CI: 84.9–96.1%).
Conclusions: We have proposed a noble melanoma discrimination index derived from spectra which vary from site to site. Although the sample size is still small, the index has been considered to be useful for discriminating melanomas from SK and nevus. The present result suggests that disordered nature of pigment skin lesions may be important in melanoma screening system.

66 Publish or perish in cancer – but where?

R.W. Glynn[1], J.Z. Chin[1], N. Miller[1], M.J. Kerin[1], K.J. Sweeney[1]. [1]*Clinical Sciences Institute, Department of Surgery, Galway, Ireland*

Background: Bibliometric analysis has previously been employed as a method of correlating research productivity in oncology with geographic variation in output and funding, and the development of translational research. Investigation of output across a range of disciplines within oncology has not been undertaken previously. The aims of this study are to measure the proportion, quality and relevance of articles relating to common malignancies in the medical press.
Materials and Methods: Both PubMed and the WoS databases were consulted for the reference period 01/01/2007 to 31/12/2007. Publications were retrieved by searching for each malignancy using its medical subject heading (MeSH) term in PubMed. The subheadings encompassed by each MeSH term were then employed to perform an equivalent search in the WoS database. The 26 malignancies with the highest incidence as defined by the Surveillance, Epidemiology, and End Results (SEER) database of the National Cancer Institute (NCI) in 2006 were included in the study. The top twenty journals by impact factor (IF) and eigenfactor (EF) in general medicine and oncology journals, and the presence of each malignancy within these titles was then analysed. The journals publishing most prolifically on each neoplasm were also identified and their impact assessed.
Results: The two databases generated 63260 (PubMed) and 126845 (WoS) entries, respectively. The 26 neoplasms accounted for 25% of total output from the top medical publications. 5 malignancies dominated the first quartile of output in the top oncology journals; breast, prostate, lung, and intestinal cancer, and leukaemia. Journals publishing most frequently on these neoplasms are associated with much higher IFs and EFs, though these measures are not equivalent across all sub-specialties. The EF and IF correlated strongly in the general medical ($r = 0.854$, $p = 0.000$) but not in the oncology literature ($r = 0.289$, $p = 0.217$).
Conclusions: Oncology enjoys a disproportionately large representation in what are traditionally regarded as the more prestigious medical journals. 5 malignancies receive the majority of this attention however, and there is a need to delineate between proxy measures of quality and the relevance of output when assessing its relative merit. Our results also suggest that the most relevant information for those working in many of the oncologic sub-specialties is not necessarily to be found in the most prestigious journals as delineated by proxy indicators of quality. These findings raise significant questions regarding the best method of assessment of research and scientific output in the field of oncology.

67 A case-control study on the effect of ApoE genotypes on head and neck cancer risk

E. De Feo[1], G. Cadoni[2], J. Rowell[1], N. Nicolotti[1], M. Volante[2], G. Paludetti[2], D. Arzani[1], R. Amore[1], G. Ricciardi[1], S. Boccia[1]. [1]*Catholic University of the Sacred Heart, Institute of Hygiene, Rome, Italy*, [2]*Catholic University of the Sacred Heart, Institute of Otorhinolaryngology, Rome, Italy*

Background: The Apolipoprotein E gene (19q13.2) which is involved in the clearance of lipoproteins from plasma has three major isoforms encoded by ε2, ε3 and ε4 alleles with different receptor-binding abilities. Since a nearly linear relationship between A*poE* genotypes and levels of total and low-density lipoprotein serum cholesterol (LDL-C) has been reported, the ε4 allele is associated with hypercholesterolemia whereas the ε2 allele relates with the reverse effect if compared to the reference ε3 allele. An inverse relationship between serum cholesterol and head and neck cancer has been previously suggested but the role of *apoE* genotypes on HNC etiology has never been investigated. Since the question on the role of hypocholesterolemia as a predisposing factor, or result of the preclinical stage of HNC itself, remains still under debate, our hospital-based case-control study aimed to overcome

this issue by directly looking at the relationship between APOE and HNC, as well as the interaction with potential effect modifiers.
Materials and Methods: Four hundred seventeen HNC cases and 436 hospital controls were genotyped for *apoE* polymorphism. The relationship between HNC and putative risk factors was measured using the adjusted odds ratios (ORs) and their 95% confidence intervals (CIs) derived from logistic regression analysis. Finally a gene-environment interaction analyses were performed.
Results: A nearly significant 40% decreased HNC risk (OR = 0.58, 95% CI: 0.31–1.05) was observed for individuals carrying at least one apoE ε2 allele while no effect was shown for those with one ε4 allele. A statistically significant interaction resulted between alcohol drinking and the ε4 allele (p-value for interaction = 0.044) with alcohol drinkers carrying at least one ε4 allele having a 2-fold HNC increased risk respect to non drinkers with ε3/ε3 genotype.
Conclusions: Our study provides for the first time evidence of a possible protective effect of the ε2 allele against HNC, thus suggesting a direct relationship between cholesterol levels and HNC risk. Larger studies are needed to confirm these findings to further investigate the role of *apoE* genotype in HNC etiology and give insight into the causal role of cholesterol on carcinogenesis as well.

68 Examination of GST and CCR5 gene polymorphisms in children with acute lymphoid leukemia in Hungarian population

O. Csorba[1], A.F. Semsei[1], D.J. Erdélyi[1,4], E. Barabás[1], M.S. Vángor[1], G. Schermann[1], E. Hadadi[1], V. Virág[1,2], A. Falus[1], C.S. Szalai[1,2,3]. *[1]Semmelweis University, Department of Genetics Cell- and Immunobiology, Budapest, Hungary, [2]Csertex Research Laboratory, Budapest, Hungary, [3]Heim Pal Children Hospital, Budapest, Hungary, [4]2nd Department of Paediatrics, Semmelweis University, Budapest, Hungary*

The glutation-S-transferase (GST) genes are involved in the metabolism of carcinogens and chemotherapy drugs, while the C-C chemokine receptor type 5 (CCR5) gene is a member of the beta chemokine receptor family of integral membrane proteins, which plays an important role in the tissue infiltration of lymphoblasts. These genes are polymorphic in humans and may affect the risk of acute lymphoid leukemia (ALL) and the outcome of the antileukemia treatment. The GSTT1 and GSTM1 genes have frequent inactive (null) variants and the CCR5D32 is a deletion mutation which makes the CCR5 receptor unfunctional.
Previous studies showed inconsistent results about the role of GST and CCR5 variants in childhood leukemia. To investigate the putative role of the inactive variants of these genes in the risk of ALL, we performed an analysis on 455 ALL children (diagnosed 1980–2005) and 359 controls using polymerase chain reaction (PCR) and gel electrophoresis.
Based on our data, we found that the homozygous frequencies of GSTM1 null genotype in the control group and in patients with ALL, did not show significant difference (51.6% vs. 54.6%; p = 0.399; OR = 1.13 (95% CI (0.85–1.49)). Comparison of deletion allele frequencies of the *CCR5*D32 in control patient and ALL cases (8.8% vs. 8.8%; p = 0.996; OR = 1.00 (0.70–1.42)) showed neither significant difference. The frequencies of patients homozygous for the inactive GSTT1 variant differed significantly between ALL children and controls (18.6% vs. 25.0%; p = 0.035; OR = 0.70 (0.50–0.98)), indicating that children without GSTT1 had a reduced risk against developing ALL in Hungarian children. GSTM1 or CCR5 deletions or deletion both in GSTT1 and GSTM1, or in GSTM1 and CCR5 genes, were not associated with ALL.
Our data suggest that the absence of GSTT1 gene may decrease the risk of developing ALL in the Hungarian population, however the inactive variants of the GSTM1 or CCR5 genes or deletions in both GSTT1 and GSTM1, or in CCR5 and GSTM1 do not affect of the risk of childhood ALL.

69 Post-GWAS pancreatic cancer susceptibility loci and their importance in survival

D. Campa[1], C. Rizzato[1], A. Bauer[2], S. Rachakonda[3], N. Giese[4], R. Kumar[3], J. Lorenzo-Bermejo[5], M. Büchler[6], J. Hoheisel[2], F. Canzian[1]. *[1]German Cancer Research Center, Genomic Epidemiology, Heidelberg, Germany, [2]German Cancer Research Center, Functional Genome Analysis, Heidelberg, Germany, [3]German Cancer Research Center, Molecolar Genetic Epidemiology, Heidelberg, Germany, [4]UNI-KlinikumHeidelberg, Klinik für Allgemeine Viszerale und Transplanationschirurgie, Heidelberg, Germany, [5]Institut für Medizinische Ruprecht-Karls Universität Heidelberg, Biometrie und Informatik (IMBI), Heidelberg, Germany, [6]UNI-Klinikum-Heidelberg, Klinik für Allgemeine Chirurgie, Heidelberg, Germany*

Background: Pancreatic cancer is one of the neoplasms with the worst mortality rate (5-year survival: 4.3%). Relatively little is known about etiology (tobacco smoking, obesity, diabetes, chronic pancreatitis, family history are the only established risk factors, and pancreatic cancer is part of some genetic cancer syndromes). There is no effective screening test for the malignancy and metastatic disease is commonly present at initial diagnosis. In 2009 the PanScan project, a genome-wide association study, identified various loci affecting susceptibility to pancreatic cancer. The aims of this study are: to replicate the association between loci identified by PanScan and pancreatic cancer risk, and to evaluate the possible associations between the inter-individual variation and overall survival time of pancreatic cancer patients.
Material and Methods: Fifteen SNPs mapping to six regions identified by PanScan were typed in a population of 700 patients and 2200 healthy controls recruited from Heidelberg and Liverpool. Genotyping was performed using the TaqMan real time PCR assay. Association between SNPs and pancreatic cancer risk was performed with logistic regression; survival analysis was performed with Cox regression models.
Results: Most of the PanScan SNPs were significantly associated with pancreatic cancer risk in our population. In addition, we found that a SNP in chromosome 15 may also play an important role in overall survival of pancreatic cancer patients.
Conclusions: The majority of the associations found in PanScan were replicated in this population and one SNP was found to be associated with a different survival time of the patients.

70 Genetic polymorphisms and risk of familial non-medullary thyroid cancer

F. Canzian[1], F. Lesueur[2], E. Bonora[3], T. Oudot[4], J. McKay[5], D. Campa[1], C. Rizzato[1], G. Romeo[6]. *[1]Deutsches Krebsforschungszentrum, Genomic Epidemiology, Heidelberg, Germany, [2]International Agency for Research on Cancer, Genetic Cancer Susceptibility, Lyon, France, [3]Policlinico S. Orsola-Malpighi, Medical Genetics, Bologna, Italy, [4]Faculté de Médecine Pitié-Salpêtrière, INSERM U525, Paris, France, [5]International Agency for Research on Cancer, Genetic Epidemiology, Lyon, France, [6]University of Bologna, Medical Genetics, Bologna, Italy*

Background: Thyroid cancer is the most common endocrine malignancy. Non-medullary thyroid cancer (NMTC), with its main subtypes papillary and follicular thyroid cancer, represents about 90% of all cases. Epidemiological data show that risk of NMTC in the ?rst-degree relatives of affected cases is elevated five- to ninefold. Familial NMTC (FNMTC) accounts for about 3−7% of all thyroid tumours and is a clinical entity characterized by a more aggressive phenotype than its sporadic counterparts. Characterization of genetic susceptibility to FNMTC was attempted a decade ago with microsatellite genotyping and linkage analysis methods, and led to the mapping of a few susceptibility genes, but identification of specific variants was not possible at that time. The advent of high-throughput SNP genotyping recently led to the identification of polymorphisms affecting NMTC risk in its sporadic and familial forms. In particular, a genome-wide association study (GWAS) identified common SNPs at 9q22.33 and 14q13.3, which increase the risk of sporadic NMTC. We have investigated if these newly identified genetic risk factors for NMTC are relevant for FNMTC as well.
Material and Methods: We genotyped 13 SNPs from recent studies, reported to affect the risk of NMTC, in 805 subjects belonging to 133 pedigrees with at least two observed cases of NMTC. Genotyping was performed with amplification refractory mutation system (KASPar) technology. Data were analyzed for both linkage and association. Single-SNP analysis of the candidate loci was performed either on the nuclear families (N = 239) using Family-Based Association Tests (FBAT), or on the whole set of pedigrees (N = 133) using Modified Quasi-Likelihood Score (MQLS).
Results: SNPs on chromosomes 9q22.33 and 14q13.3 showed convincing evidence of association with NMTC risk in these families using both methods, whereas the other tested markers resulted negative. Haplotype analyses for the loci where more than one SNP was tested confirmed the results of the single SNP analyses.
Conclusions: Consistent with findings from a recent GWAS, SNPs on chromosomes 9q22.33 and 14q13.3 appear to be associated with the familial form of NMTC as well as the sporadic form. We will perform further genotyping in the familial samples of SNPs located in *FOXE1* (9q22.33) and *NKX2* (14q13.3), as well as additional SNPs reported to be associated with risk of NMTC.