controls although the HLXB9 gene is localised in the inner part of the nucleus in the cancer cells. These findings suggest that HLXB9 expression in breast cancer might be regulated by other factors and is not dependent on nuclear positioning. Further studies aimed to investigate the structure of the nuclear envelop in breast cancer cells might shed some light into the mechanisms of nuclear gene posi- tioning and expression in these diseased cells.

2.P27

Neuroblastoma by Fluorescence in situ Hybridization Experience of a Single Center in Singapore

Sharon Ai Chien Ling [1], Derrick Wen Quan Lian [1], Kenneth Tou En Chang [1], Joyce Ching Mei Lam [2], Bhattacharyya Rajat [2], Min Hwee Yong [1]
[1]Kk Women's And Children's Hospital, Department of Pathology & Laboratory Medicine, Singapore, Singapore; [2]Kk Women's And Children's Hospital, Haem/onc Services, Singapore, Singapore
Correspondence: Sharon Ai Chien Ling
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Introduction
Neuroblastomas are one of the most common solid tumours in children. Molecular cytogenetic studies are of paramount import- ance in the prognosis and treatment stratification of patients with this tumour, and are recommended by The International Neuroblastoma Risk Group Staging System (INRGSS). In particular, tumour cell ploidy, MYCN amplification, deletion of chromosome 1p and 11q, and gain of chromosome 17q portend an unfavor- able prognosis. In this study, we report 16-years' of experience at the Cytogenetics Laboratory, KK Women's and Children's Hospital (KKH), Singapore.
Methods
Interphase fluorescence in situ hybridization (FISH) studies of neuroblastoma specimens from 2001 to 2016 were reviewed. These studies were performed on tumour imprints, formalin-fixed paraffin- embedded tissues (FFPE), fresh frozen tissues and bone marrow specimens, using locus-specific (MYCN, 1p and 11q or 17q) commer- cial fish probes.
Results
Our work summarises the results of 404 neuroblastoma samples, of which 32 were primary tumours and the other 372 were post-treatment tumours. A total of 166 neuroblastoma tumours (18 primary and 148 post-treatment tumours) exhibited one or more of the three genetics parameters being investigated. Less than 20% of these 166 tumours showed two or more of the three genetics parameters investigated. Of the 18 primary tumours, 14 correlated with unfavorable histology.
Conclusion
Our data suggests that MYCN amplification, 1p and/or 11q deletion are reliable independent parameters correlating with an unfavour- able histology and poor prognosis of patients for primary tumour. Furthermore, our data also suggests that detection of these genetic parameters via FISH is also useful in follow up of post-treatment neuroblastomas.

2.P28

1p36 Abnormalities In Hematological Malignancies

R. Dilhan Kuru [1], Ayşe Çirakoğlu [1], Şükriye Yilmaz [1], Yelda Tarkan-Argüden [1], Şeniz Öngören [2], M.Cem Ar [2], Ayhan Deviren [1], Zafer Başlar [2], Teoman Soysal [2], Seniha Hacihanefioğlu [1]
[1]Cerrahpaşa Faculty of Medicine, Istanbul University, Medical Biology, Istanbul, Turkey; [2]Cerrahpaşa Faculty of Medicine, Istanbul University, Internal Medicine, Division of Hematology, Istanbul, Turkey
Correspondence: R. Dilhan Kuru
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Structural aberrations of chromosome 1p36 involving in tranlocations, inversions, deletions and duplications are very common in most hematologic malignancies. They are observed in myeloprolifera- tive disorders (MPD), myelodysplastic syndrome (MDS) and multiple myeloma (MM). Also, 1q36 abnormalities are rare in other myeloid malignancies, acute lymphoblastic leukemia (ALL), chronic lymphocytic leukemia (CLL), and non-Hodgkin's lymphoma.
We present deletions and rearrangements of breakpoint 1p36 in 18 patients with hematologic malignancies in this study.
Rearrangements occur via translocations with diffrent chromosomes, additions and inversions. Our cases consist of 3 MDS, 2 AML, 2MM, 6 CML, 2 Lymphoma, 1 B-ALL, 1 ITP and 1 Aplastic anemia patients. We have shown t(1;3)(p36;q21) at 3 patients (1AML M2 and 2 CML). The t(1;3)(p36;q21) leads to fusion of PRDM16 (containing 16 gene) at 1p36 with RPN1 at 3q21. AML and MDS with t(1;3)(p36;q21) were first described by Moir et al. (1984). Translocations of PRDM16 lead to its overexpression, and those patients have a poor prognosis. Also 7 patients (1MDS, 2 MM, 2 CML, 1 AML and 1 Aplastic anemia) have translocation 1q36 with other chromosomes or rearrangements. One ITP patient has der(1)t(1;1)(p36;q21) and another CML patient has der(1)t(1;1)(p36;q12). 1p36 region consists of genes which are important in oncogenesis. der(1)t(1;1)(p36;q11-q32) are reported in litera- ture for both myeloid and lymphoid neoplasias.
23 candidate tumor suppressor genes have been mapped in the 1p36 region, and it is the smallest region of deletion (SRD). Terminal 1p36 deletions are frequent in both myeloid and lymphoid neopla- sias. We have also demostrated several terminal deletions from 1p11 to 1p36 at five patients, that are 2 MDS, 2 Lymphoma, and 1 B-ALL.

2.P29

The Use of Multitarget FISH on Formalin Fixed Parafin Embedded Tissue in Bladder Cancer

Ayşe Çirakoğlu [1], R. Dilhan Kuru [1], Sultan Rana Ramazanoğlu [2], Süleyman Ataus [3], Bülent Alici [3], Ayhan Deviren [1], Seniha Hacihanefioğlu [1]
[1]Cerrahpaşa Faculty of Medicine, Istanbul University, Dept. of Medical Biology, Istanbul-Turkey; [2]Kanuni Sultan Süleyman Training And Research Hospital, Dept. of Pathology, Istanbul-Turkey; [3]Forte Urology, Urology, Istanbul-Turkey
Correspondence: Ayşe Çirakoğlu
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Bladder cancer is one of the common cancers in the world. The majority (70%) of bladder cancers (urothelial carcinoma) are superficial including papillary pTa (40%), pT1 (30%) and carcinoma in situ (Cis) (2-5%), at the time of diagnosis.
Genetic studies of bladder cancers observed that chromosomal changes are one of the important factors that act in tumorigenesis and behaviour of the tumor. Polysomies of chromosomes 3, 7, and 17 and deletions of p16 gene located at 9p21 are used as biomarkers in diagnosis of bladder tumors. Loss of both alleles of p16 has been shown that occur at early stages whereas polysomies of chromo- somes 3, 7, and 17 are associated with progression of the tumor. A multicolor fluorescence in situ hybridization (FISH) assay, UroVysionTM, was developed to detect these abnormalities in urine specimens for diagnosis of bladder cancer. It can also be applied to parafin-embedded tissues.
In this study, we applied UroVysionTM on archival paraffin-embedded sections from 21 patients with superficial grade T1 tumors. All cases were FISH-positive. In 14 cases, polysomy of at least two chromosomes, in similar frequencies for all combinations, with biallelic deletion of p16 were observed. Six cases had only biallelic deletion of p16 and one case had polysomy of at least two chromosomes.

2.P30

Analysis of NFκB1 gen ins del polymorphisms using PfLMI restriction enzyme on breast cancer

Onur Eroglu [1], Esin Guvenir Celik [2], Hacer Kaya [2], Merve Celen [2], Ceylan Demirci [2]
[1]Bilecik Seyh Edebali University, Biotechnology Application And Research Center, Bilecik, Turkey; [2]Bilecik Seyh Edebali University, Molecular Biology And Genetics, Bilecik,Turkey
Correspondence: Onur Eroglu
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