in the context of maternal breast cancer'. Generating a theory from the children themselves, who are experiencing their mother's cancer diagnosis and treatment, was central to this study. To date most studies relevant to this area have investigated parents perceptives of children's experiences or examined children's own experiences many years following their mother's breast cancer diagnosis.
The aim of this particular grounded theory study was to generate a theory about the experiences of 7−11 year old children whose mothers' had been diagnosed with and receiving treatment for early-stage breast cancer with a view to contributing to knowledge development. Analysis of the data revealed that the main concerns of the participants were the processes involved in navigating their lives through a period of disrupted mothering. The substantive theory of 'protecting' was conceptualised from the data to describe children's experiences in the context of maternal breast cancer. This theory had three sub-core categories of Shifting Normality, Shielding and Transitioning, with numerous properties and sub-properties. The central argument in this study is that the children were trying to protect their own lives and well as that of their mothers. The findings provide a mechanism for understanding how the perceived loss of the 'well' mother raised concerns for the children and resulted in adaptations of roles and responsibilities.
It is suggested that there is a gap in services where the needs of children, whose mother had cancer, are not adequately met in that the children had little, if any, opportunity to meet with healthcare professsionals. In addition, parents may need assistance with how they can talk with their children about the diagnosis and the changes in family life. Children's experiences of parental chronic illness is a healthcare issues for the present and the future and the healthcare system needs to identify future services and developments if society is to truly value children and listen to children's voices.

Scientific Symposium (Sat, 24 Sep, 11:15−13:15)
Improving the Therapeutic Ratio of Radiotherapy: New Concepts to Protect Normal Tissues

5 INVITED

Molecular Targeting to Protect Normal Tissues

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Strategies for 'molecular-based' modification of normal tissue responses to irradiation must be designed for the underlying pathobiology in the individual tissues and organs, and the biology of the contributing cell populations as well as their interactions. Interventions in the processing of radiation damage can be directed against any step of the pathogenetic cascade in cells, including early production of free radicals (e.g. hypoxia, radical scavenging), activation of transcription facors (such as NFkB or AP-1), modulation of intracellular and juxta-/parakrine signaling cascades (activation/deactivation of growth factor signaling pathways, inflammatory signaling and others), or modulation of the immune response. Moreover, 'tissular' reactions may be modified, such as proliferation/differentiation in early responding tissues, the chronic oxidative stress response, the vascular response, or the fibrotic tissue remodeling in late responding tissues. Examples will be presented. However, the mechanisms of action underlying the protective or ameliorative effects must be clarified in order to design optimum clinical protocols. Therefore, before clinical investigation, modification approaches should be thoroughly tested in animal models, with relevant (fractionated) irradiation protocols and relevant endpoints. Results from single-dose and fractionation studies can be divergent, as has been demonstrated in a number of preclinical investigations. In order to prove selectivity and to guarantee a therapeutic gain, any strategy, if applied during or shortly after the oncological treatment, must also be tested for potential tumour effects.
Today, most of the normal tissue targeting strategies must be considered experimental. Most promising, with first clinical studies, are the interaction with (some) growth factor signaling cascades, the interruption of chronic oxidative stress (in late tissue reactions), and the treatment (mobilization and transplantation) with stem cells, haematopoietic, mesenchymal and tissue-specific. It must, however, be assumed that strategies targeting single molecules or cascades will only result in a delayed tissue response, because of the activation of backup pathways in the cells and tissues. Hence, 'modification cocktails' may be required, but the efficacy of their individual components must first be tested under the premises outlined above.

6 INVITED

Stem Cells − Role for Radiation Response and Therapeutic Approaches

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Inevitably normal tissues are also exposed to ionizing radiation during the treatment of cancer with radiotherapy. Many factors play a role in the response of tissues to irradiation, but ultimately the ability of stem cells to reconstitute functional cells determines the onset and the severity of the radiation effects. The maintenance and repair of the tissue integrity are the primary roles of the tissue stem cell. Stem cells are undifferentiated reside between differentiated cells and are able to self-renew to prevent aging. In contrast, progenitor cells although capable of producing specialized cell types have a limited life-span as they are incapable of self-renewal. Stem cell therapy provides a potential prevention or treatment of radiation-induced normal tissue damage. Several stem cell types are being investigated for their potential use in stem/progenitor cell therapy: embryonic stem cells, induced pluripotent stem cells, tissue adult stem cells (ASC), mesenchymal stem cells (MSC) and epithelial progenitor cells (EPC). Of these the last three are used as or are close to clinical application and will be discussed in the current presentation.
Mobilisation or injection of bone marrow derived MSCs have been shown to ameliorate radiation-induced side-effects in gut, skin, oral mucosa and salivary gland, potentially through modulation of immunogenicity, apoptosis and the secretion of cytokines/growth factors that stimulate regeneration, but not through transdifferentiation. Indeed, stimulation of surviving stem/progenitor cells using growth factors has been shown to be promising in e.g. gut and salivary glands albeit only when sufficient stem/progenitor cell remain after irradiation. For tissue in which vascular damage is the main cause of organ dysfunction after irradiation, transplantation or mobilisation of bone marrow derived EPCs and vascular progenitor cells may result in inhibition of normal tissue damage, although also for such therapies survival of local stem/progenitor cells seems to be a prerequisite. Independent from the number of surviving stem/progenitor cells ASC transplantation seems to be a promising therapy. For mouse salivary gland the potential of such therapies has been shown and are currently being translated to clinical application. Recent developments will be discussed. The application of stem/progenitor cell therapy in all its forms to reduce normal tissue effects will have a tremendous impact on radiation oncology in the near future.

7 INVITED

Possible Intervention Strategies to Reduce Radiation-induced Heart Disease

S. Hoving[1], I. Seemann[1], K. Gabriels[2], N.L. Visser[1], J.A. te Poele[1], S. Heeneman[2], M.J. Daemen[2], F.A. Stewart[1]. [1]*The Netherlands Cancer Institute − Antoni van Leeuwenhoek Hospital, Experimental Therapy, Amsterdam,* [2]*Cardiovascular Research Institute Maastricht, Experimental Vascular Pathology, Maastricht, The Netherlands*

Radiotherapy is a common treatment for breast cancer and Hodgkin's lymphoma and it is associated with good long-term survival prognosis. Unfortunately, increased risk of cardiovascular disease is now recognized as a late complication when significant volumes of the heart and coronary arteries are included in the radiation field. We showed previously in mice that radiotherapy induces dose- and time-dependent changes in the heart, starting with an acute epicardial inflammation and microvascular damage and progressing to vascular leakage, myocardial fibrosis and amyloid depositions. Intervention to inhibit the progressive inflammatory changes or stimulate vascular recovery could help to prevent cardiac failure at later times after irradiation. This presentation will review the limited published data available and describe novel approaches to inhibit radiation-induced heart damage.
Pre-clinical studies in rats have investigated amifostine, pentoxifylline/tocopherol I (Vit. E) or the ACE inhibitor captopril in heart radiation models. Amifostine is able to prevent radiation-induced reduction in coronary flow and aortic flow and decreased the histopathological changes seen at 6 months post-irradiation. Pentoxifylline/Vit. E reduced TGF-β1 mRNA levels and subsequently reduced fibrosis. Captopril did not prevent cardiac dysfunction, but did reduce myocardial fibrosis and prevented left ventricular capillary density loss after local heart irradiation. In myocardial infarct models, thalidomide has been shown to attenuate the development of fibrosis and promote vessel maturation. Other experimental studies have shown that bone-marrow-derived endothelial progenitor cells (BM-EPC) can stimulate vascular recovery and improve cardiac function after myocardial infarction. We are currently setting up experiments to evaluate the potential of thalidomide or BM-EPC to stimulate vascular recovery after cardiac irradiation in mice.

With increasing attention on quality of life aspects of cancer treatments, more knowledge is required on the mechanisms of development and management of late side effects. This knowledge should enable the development of effective intervention strategies to prevent or ameliorate the development of heart damage in patients following radiation therapy.
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8 INVITED

Modern Radiotherapy Techniques to Spare Normal Tissues

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Modern radiotherapy techniques have been used to reduce the dose to radiosensitive critical structures close to tumour bearing tissues. Randomised trials have demonstrated that clinically relevent reductions in normal tissue toxicity can be achieved. This lecture will provide an overview of this topic.

Scientific Symposium (Sat, 24 Sep, 11:30−13:15) **Telethinking in Cancer Care**

9 INVITED

Technologies and Challenges for Nurses and Health Care

D. Benton[1]. [1]*International Council of Nurses, Geneva, Switzerland*

Health systems are facing unprecedented challenges at this time. Not only are the social demographics of many countries changing but also the patterns of disease. The economic crisis in 2008 has underlined the connectivity that we all can experience in our daily lives. Some aspects of globalisation are very positive others can present problems. What is certain is that the world's population is not going to require less access to nursing care in the future and technology will play an increasing role in its delivery. This paper explores a range of challenges as well as the opportunities that the nursing profession faces as we move forward through increasing use of technologies and sets out how nurses can play a more proactive role in designing our profession's destiny as well as securing increased access to quality services for citizens.

10 INVITED

Patient-Centred Techologies − the Future is Here

N. Kearney[1], R. Maguire[1]. [1]*University of Dundee, School of Nursing and Midwifery, Dundee, United Kingdom*

By 2030, there will be almost 21.4 million new cases of cancer diagnosed annually and more than 13.2 million deaths, compared to 12.7 million new cases and 7.6 million deaths in 2008, according to the International Agency for Research on Cancer (2010), with half of these likely to be in Europe. The scale of the challenge to deliver optimal care to this population means that we have to consider a different model of delivering health care that will involve a shift from hospital based care to much more care being delivered in, or close to, people's home. Changing the way we deliver healthcare outside of an acute hospital, either on an outpatient basis or in local communities has indisputable implications for patients receiving cancer treatment, and for patients with other chronic conditions, as they can experience multiple needs in relation to symptom management, self-care and support. Cancer treatment related toxicities often lead to distressing and potentially life threatening side effects (Kuderer 2006), which are associated with poor treatment adherence, impaired quality of life, increased infections, and mortality and time spent in hospital. Supporting patients, who are experiencing such morbidity, within their own home or local community will be key to ensuring optimal patient outcomes. Technology is now accepted as pivotal to future health care delivery not only to enable safe and effective evidenced based care, but also a means to delivering affordable care across populations. Virtual supportive care networks that utilise technology to enable individualised patient centred care are being used for patients with chronic conditions and allow patients to remain at home and access appropriate supportive care as and when required. Within cancer care there has been a reliance on hospital based, specialist care however there is a growing recognition that telehealth systems, for example ASyMS, have the potential to transform supportive care outcomes in patients with cancer by allowing health professionals to respond in 'real time' to a patient's actual symptoms. The ability to capture symptom data in 'real time' is now regarded as the gold standard to allow rapid clinical decision making and intervention to improve patient outcomes. Telehealth systems will mean real time patient reported outcomes become standard care and linking such systems to point of care testing devices, such as white cell monitoring, will transform the management of patients with cancer.

11 INVITED

Using Technology in Palliative Care − a Reality

B.M. Johnston[1]. [1]*University of Dundee, Department of Nursing and Midwifery, Dundee, United Kingdom*

The term given to the remote monitoring of patients through information and communication technologies is "Telecare" or 'Telehealth" and is being evidenced increasingly as a means of addressing the increased demand on health services alongside more patient-focused care. However, there is a relative lack of evidence based research in the use of Telehealth in palliative care in the UK, particularly in Scotland, in comparison to other countries such as Australia, Canada and the USA. Telehealth is, however, gaining widespread acceptance and is both usable by, and acceptable to, patients and health professionals in palliative care settings, particularly in light of the need for increased home care for palliative patients and ongoing symptom management. This paper will report on the type of telehealth applications used in palliative care, and discuss patient and Health Professional experience of using telehealth applications in palliative and end of life care.

Scientific Symposium (Sat, 24 Sep, 11:15−13:15) **Small RNAs and Cancer**

12 INVITED

MicroRNAs and Regulatory RNA Binding Proteins in Cancer

R. Agami[1]. [1]*The Netherlands Cancer Institute, Department of Gene Regulation, Amsterdam, The Netherlands*

MicroRNAs (miRNAs) are genes involved in normal development and cancer. They inhibit gene expression through interaction with 3′-untranslated regions (3′UTRs) of messenger RNAs (mRNAs), and are thought to regulate a large proportion of protein coding genes. Patterns of mis-expression of miRNAs in cancer suggest key functions of miRNAs in tumorigenesis. We performed in the past genetic screens to identify cancer functions of miRNAs. Using a library of vectors expressing human miRNAs and we identified miRNAs that cooperate with oncogenes in cellular transformation, which stimulate cellular migration, invasion and metastasis, as well as key regulators of tumour suppressor genes.
In recent years, it is becoming apparent that the miRNAs themselves are subjected to intense regulation at various levels. miRNA biogenesis and activity can be kept in pace by RNA-binding proteins (RBPs). We show that interplay between RBPs and miRNA exists that affects gene expression in processes such development and cancer.

13 INVITED

Causes and Consequences of microRNA Dysregulation

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During the past several years it has become clear that alterations in the expression of microRNA genes contribute to the pathogenesis of most, perhaps all, human malignancies. These alterations can be caused by a variety of mechanisms, including deletions, apmplifications or mutations involving microRNA loci, by epigenetic silencing or by dysregulation of transcription factors targeting specific microRNAs. Since malignant cells show dependence on the dysregulated expression of microRNA genes, which in turn control or are controled by dysregulation of multiple protein coding oncogenes or tumour suppressor genes, these small RNAs provide important opportunities for development of future microRNA based therapies.

14 INVITED

Deregulated tRNA Expression in Cancer

R.J. White[1]. [1]*Beatson Institute for Cancer Research, Glasgow, United Kingdom*

RNA polymerase (pol) III is responsible for ~10% of nuclear transcription and makes a variety of short non-coding RNAs, including tRNA. Levels of the initiator tRNA are limiting for translation in some cell types. Mild overexpression of this tRNA not only stimulates protein synthesis, but