Foreword

International Congress on Hormonal Steroids and Hormones and Cancer

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International Congress provide the impetus to facilitate the presentation and discussion of a wide spectrum of opinions, which then lead to the development of a broad perspective of understanding. The format chosen is usually designed to accomplish three general aims: the critical review of rapidly advancing and important fields, the integration of cutting edge information considered to represent new pathways of understanding, and the presentation of exciting and provocative preliminary data. The meeting in Fukuoka, Japan held on October 21–25 2002 served these purposes well. In addition, this meeting created unique synergy imparted by the joining of two previously independent congresses, the International Congress on Hormonal Steroids (ICHS) and the International Congress on Hormones and Cancer (ICHC). This joint congress continued a long tradition since it represented the 11th ICHS and 7th ICHC meetings. The marriage of these two congresses was timely and reflected the increasing understanding that hormones, growth factors, cytokines, and chemokines all work through receptors and influence cancer cell proliferation and apoptosis, as well as differentiated function. By providing a forum to summarize recent data from several disparate areas, this combined congress weaved a fabric of interlocking information based upon evolving principles of cellular biology and clinical intervention.

The data presented at the meeting highlighted the extraordinary advances in technology which enable the generation of new information at an increasingly rapid pace. Examples include the methods now available for dissecting out the role of specific genes in causality of cancer, the demonstration of dynamic regulation of genes through c-DNA array and proteomic techniques, and the molecular genetic methods available to identify new transcription regulatory factors, receptors, and cell signaling pathways. This issue of Endocrine-Related Cancer contains selected manuscripts based upon talks at the joint congress which specifically address the issue of cancer. Topics from the meeting directed toward more general aspects of steroidal action will be published in the Journal of Steroid Biochemistry and Molecular Biology in an upcoming issue.

Key areas of focus to be published in this issue of Endocrine-Related Cancer involve cancer susceptibility genes, mutations in receptors, identification of novel receptors, use of co-activators and co-repressors in regulation of proliferation and apoptosis, epigenetic methods of adaptation to selective pressures induced by hormonal therapies, cooperative effects of hormonal pairs, and hormonal mechanisms of regulating cell cycle activity. One specific area of focus was directed toward a greater understanding of the molecular mechanisms whereby cancer cells respond to hormones with stimulation of cellular proliferation and inhibition of programmed cell death. Much emphasis was directed toward the interactions between signaling pathways once thought to be disparate but now known to be integrally interconnected. As an example, the mitogen activated protein kinase (MAPK) and phosphoinositol-3-kinase pathways are stimulated by hormones and participate in the regulation of cell proliferation and apoptosis. Alterations in the degree of cross talk between these pathways provide clues as to the development of resistance to hormonal therapies such as tamoxifen.

A general theme of the manuscripts in this issue of Endocrine-Related Cancer is the emphasis on the degree of complexity of regulatory processes. Stimulation of growth factor pathways can occur via input from steroid hormones, from mutations of inactivating enzymes such as PTEN, from ligand independent activation of hormonal receptors, and from over-expression of receptor co-activators. The number of hormone receptors identified continues to increase as exemplified by the various forms of ERβ. Receptors can serve to amplify or to squelch transcriptional effects as exemplified by the positive regulatory effects of ERα and the negative effects of ERβ isoforms such as ERβ cx. Receptor sites of action vary as demonstrated by the actions of steroid hormone receptors at both the nuclear and cell membrane levels. The co-activators and co-repressors add to the complexity by mediating both steroidal and non-steroidal transcriptional events and facilitating cross talk between growth factor and steroidal signaling pathways. This evolving complexity highlights the emerging requirement that investigators who study classical steroid hormone actions must interact with investigators examining the broad range of effects of hormones on cancer.

In summary, this issue of Endocrine-Related Cancer provides cutting edge information regarding the multiplicity of
actions of hormones and growth factors and their exquisite coordination for regulation of proliferation and prevention of cell death. Interwoven among these manuscripts is the important concept that new technologies such as use of cDNA micro-array, restriction length genomic scanning, dynamic confocal microscopy and others will provide more powerful tools with which to dissect out complex inter-related pathways. A careful reading of the studies published in this issue will provide a sense of the rapidity of progress in our understanding of hormone dependent cancer and the advances we can anticipate over the next few years.

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