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EDITORIAL

In focus in HCB

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Several review articles are published in this issue as part of the 60th anniversary of *Histochemistry and Cell Biology*.

The review by Schatten and Sun (2018) focuses on **the mammalian centrosome**, an organelle not only known as a primary microtubule organizing center, but also as a major communication center for signal transduction pathways and as a center for proteolytic activities. The current review is based on information presented in the 2008 review (written in commemoration of the 50th anniversary of the journal; Schatten 2008) and highlights new information on centrosome structure and function in normal and diseased conditions that have become available through the use of new experimental tools including advanced genetic manipulation, as well as live cell imaging and super-resolution fluorescence microscopy. Six sections are presented to address (1) centrosome structure and functions, and new insights into the role of centrosomes in cell cycle progression; (2) the role of centrosomes in tumor initiation and progression; (3) primary cilia, centrosome-primary cilia interactions and consequences for cell cycle functions in health and disease; (4) transitions from centrosome to non-centrosome functions during cellular polarization; (5) other centrosome dysfunctions associated with the pathogenesis of human disease; and (6) centrosome functions in oocyte germ cells and dysfunctions in reproductive disorders and reproductive aging.

The review by Steinbacher and Ebnet (2018) concentrates on **the regulation of junctional actin dynamics by cell adhesion receptors** and covers recent developments on the role of cell adhesion molecules at epithelial and endothelial cell–cell junctions in the regulation of junctional actin dynamics. One focus is on actin and the dynamic recruitment of actin regulators at adherens junctions and actin at tight junctions. It is emphasized that the junctional actin is highly dynamic, even after the maturation of intercellular junctions and the development of apico-basal polarity. Another focus of the review is on the vertebrate homologs of Drosophila Crumbs and the role of Crumbs3 in the regulation of the cortical actin cytoskeleton and the formation of mature, barrier-forming cell–cell junctions in Sertoli cells. Furthermore, the role of the cell adhesion molecule JAM-A in the regulation of the actin cytoskeleton reorganization in epithelial cells during cell division is discussed. Together with additional findings during collective cell migration and on VE-cadherin fingers, all observations point to the highly dynamic association of actin and cell adhesion molecules.

In their review on **the role of connexins during early embryonic development**, Wörsdörfer, Wagner and Ergün (2018) summarize current knowledge on the possible role(s) of the gap junction proteins during preimplantation development and in embryonic stem cells. After recapitulating the function of connexins as gap junction proteins, additional, rather unexpected roles are listed and the importance of specific connexin proteins at different early embryonic stages is discussed. Moreover, the complexity of the connexin expression patterns and its interspecies variation during early embryogenesis is underscored. The controversy surrounding the debate of whether connexins are essential key regulators of development and what their precise role may be in this process is thoroughly considered. Finally, in an attempt to provide more definite information concerning the functional implications of connexin protein expression, the authors consider and discuss the use of pluripotent stem cells and trophoblast stem cells, as well as artificial embryo-like structures and organoid cultures in combination with multiplex CRISPR/Cas9-based genome editing.

Yi et al. (2018) provide an overview of **the role of keratins in the digestive system** in health and disease. Keratins belong to the intermediate filament protein family and are expressed in a tissue-specific pattern as heteropolymers. It is now well established that mutant keratins predispose to, or cause various diseases in humans. This review summarizes

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the current state of knowledge obtained through the use of transgenic mice on the understanding of mutant keratin-associated diseases of the liver, pancreas and colon. Depending on the position of the keratin mutation, disruption of the keratin filament organization, keratin expression levels, and effects on post-translational modifications of keratins such as phosphorylation and O-GlcNAcylation may result. Starting with an overview on tissue-specific keratin heteropolymer expression patterns, the consequences of keratin8/keratin18 mutations in liver are detailed, which may include increased hepatocyte fragility, Mallory body formation or chronic hepatitis, as well as enhanced susceptibility to liver injury by various types of agents. Further, various pancreas phenotypes, such as occurence of epithelial dysplasia or loss of acinar architecture and pancreas atrophy, as well as the absence of a phenotype associated with keratin8/keratin18 mutations are specified. Various keratins are expressed in the intestine, and keratin8-null mice showed chronic spontaneous colitis, epithelial hyperplasia, decreased rate of apoptosis and higher susceptibility to drug-induced injury or carcinogenesis.

Human trophoblast invasion: new and unexpected routes and functions is the topic of the review by Moser and colleagues (2018). The established view of trophoblast invasion during human placentation posits the invasion of uterine connective tissue and of uterine spiral arteries to guarantee the blood supply from the mother to the placenta through the spiral arteries to the intervillous placental space. The newly added routes and functions include the invasion of endoglandular trophoblasts into uterine glands beginning at the time of implantation, enabling histiotrophic nutrition of the embryo prior to perfusion of the placenta with maternal blood. The subsequent invasion of trophoblasts into uterine veins (and lymphatics, the function of which is still not clear) guarantees the drainage of fluids from the placenta back into the maternal circulation throughout pregnancy. Therefore, and because of additional findings, it seems as if there is no restriction on trophoblast invasion in terms of specificity of invaded structures. The possible, currently largely unknown consequences of these additional invasive routes of trophoblasts for pregnancy pathologies, such as recurrent spontaneous abortions and tubular pregnancies will likely represent a hot topic for future studies. It is also imagined that the migration of extravillous trophoblasts towards the cervix may provide new strategies for noninvasive prenatal testing or even diagnosis.

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