Researches on the area of apoptosis have achieved explosive progress over the past two decades. Two major regulating mechanisms of apoptosis have been uncovered in great details. One is called extrinsic apoptosis pathway, also known as the death-receptor pathway. The extrinsic pathway is usually activated from outside the cell by TNF and other cytokines with their corresponding death receptors, such as TNF receptor, Fas, and TRAIL. The other is named intrinsic apoptosis pathway, also called as the mitochondrial pathway. The intrinsic pathway is activated responding to cell stresses or intracellular damage, such as DNA damage. So far, hundreds of proteins have been identified to be involved in the regulation of both extrinsic and intrinsic apoptosis pathways. And the collection of four research articles in this issue will provide new factors to this long list of apoptosis.

In the first article, Dr Jiang and colleagues analyzed the pathogenesis of a new H1N1 influenza virus strain, which was collected from China in 2009. Dr Jiang’s lab showed that viral infection by this H1N1 strain induced significant apoptotic cell death in two human carcinoma cell lines, and then concluded that the 2009 pandemic H1N1 strain can induce apoptosis in epithelial cells of the human respiratory tract. In the research area of infection diseases, people have established a link between apoptosis and virus infection, for example, HIV infection results in the apoptosis of T cells; while, by this study, Dr Jiang incorporates the concept of apoptosis with the pathogenesis of H1N1 influenza virus, which might provide a clue for developing new therapeutic approaches against influenza virus.

Cancerous cells usually gain the apoptosis-suppressing properties. In the second article, Dr Li and colleagues investigated the tumorigenesis role of SOX2, which is known as a key regulator in embryonic stem cells. They found that SOX2 promoted cell growth and increased the apoptosis-resistant properties of prostate cancer cell line. Their results indicate that SOX2 might be a molecular biomarker to evaluate the progression of prostate cancer and also serve as a potential drug target.

Apoptosis plays an important role in neuronal degeneration. In this issue, Dr He and his laboratory reported that nitrated α-synuclein involved in neurodegenerative disorders through induction of cell death. Dr He revealed a novel molecular mechanism on this nitrated-protein induced apoptosis, by which nitrated α-synuclein might mediate the activation of inducible NO synthase and inhibition of focal adhesion kinase. The authors argue that the induced apoptosis of nitrated α-synuclein is mediated via an integrin-iNOS/-FAK signaling pathway.

Acetylcholinesterase (AChE) identified in various cell types, such as neuronal cells, epithelial cells, blood cells, and vascular endothelial cells, has been implied the involvement of apoptosis. In this issue, Dr Zhang and his colleagues present a detailed analysis on an AChE-derived protein during apoptotic process. They showed that a 55 kDa AChE protein resulting from the cleavage of 68 kDa AChE was induced during apoptosis, and the formation of this AChE-derived protein was negatively regulated by the Akt pathway. The authors suggest that an alternative form of AChE may play a role in apoptosis.

We hope that these research articles will provide more information for understanding the molecular mechanism of apoptosis and its roles in physiological and pathological processes.