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Editorial

Evolution of Thioredoxin and Redox Signaling Research: Viewpoint

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LIFE ON EARTH is constantly challenged by life-threatening environmental stressors including nitric oxide (NO), reactive oxygen species (ROS), and various other oxidative stress inducers such as ultraviolet rays, X ray, xenobiotics and a variety of infectious agents. At the same time, these stressors are considered to induce various host cell responses, leading to the adaptation and evolution of living organisms. Redox regulation constitutes a fundamental principle that modulates signal transduction, regulation and repair of genes, and cell growth and death. Redox signaling represents a new frontier in biomedical research that cuts through most physiological as well as pathophysiological processes.

In the 1960s Reichard *et al.* discovered thioredoxin (TRX) in *Escherichia coli*. Since then, numerous reports have documented the role of TRX in plants and microbes. More recently, numerous laboratories have turned to elucidate the significance of oxido-reductive processes in various aspects of biomedical research. In the later part of 1980s, human TRX was identified as adult T-cell leukemia (ATL)-derived factor (ADF), which we had reported as a soluble mediator inducing interleukin-2 (IL-2) receptor, which in turn has cell-activating properties. ADF is produced by transformed T cells from ATL patients. ATL is a peculiar helper T-cell leukemia/lymphoma of late onset first reported by Takatsuki, Uchiyama, and myself as a clinical entity in the mid 1970s in Japan. The causative agent, the human T-cell lymphotropic virus type I (HTLV-I) retrovirus, was discovered in around 1980 in United States as well as in Japan. A fraction of HTLV-I infected carriers develop various diseases, including ATL and nonmalignant autoimmune-like disorders in the later part of life.

We identified that ADF or human TRX has numerous intracellular as well as extracellular activities. Other laboratories have also reported TRX or related proteins may function as cytokine-like factors; these include 3B6-IL1, MP6-BSF, eosinophil cytotoxicity enhancing factor (ECEF), and early pregnancy factor (EPF). Intracellular roles of TRX in signal transduction, gene activation and repair, as well as cytoprotection have been studied extensively in the recent past years.

In 1989, we started our first domestic ADF workshop. The workshop continued successfully until 1992. Given the rapidly growing interest in the field, the time was then ripe for an international meeting. In 1993 and 1994, we organized the International Workshop for Redox Signals in Kyoto, Japan. This highly successful meeting led to the International Symposium for Oxidative Stress and Redox Regulation in Paris in 1996 organized by Dr. Luc Montagnier and others. By then the field of redox regulation of signal transduction had become even more active, and direct links to clinical implications were established. In 1998, Dr. Chandan K. Sen of Berkeley founded the present journal, *Antioxidants & Redox Signaling*, with support from Dr. Dipak Das of Connecticut, several of us in Japan, and numerous key figures from all over the world who joined the truly international editorial board. These events may be arguably viewed as landmarks in the evolution of the

field of redox biology as it relates to molecular and cell biology.

The Redox 99 International Symposium in Kyoto, Japan, took place November 4–6th 1999. The meeting was attended by over 160 scientists, and was a venue at which several late-breaking advances were reported. The current issue contains several select peer-reviewed papers from that meeting. Presentation of this collection of outstanding papers was made possible because of the intensive editorial efforts and steadfast enthusiasm of Drs. Chandan K. Sen and Hajime Nakamura.

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