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（For JSPS Fellow）

Form B-5

Date（日付）
12/12/2017

Activity Report -Science Dialogue Program-
（サイエンス・ダイアログ事業　実施報告書）

- Fellow's name（講師氏名）: Md. Morshedul Alam (ID No.P17116)

- Participating school（学校名）: Akita Prefectural Yokote High School, Yokote, Akita.

- Date （実施日時）: 07/12/2017

- Lecture title（講演題目）: Antioxidants and Aging Science

- Name and title of your company（同行者 職・氏名）
Dept. Of Gene Expression Regulation, Institute of Development, Aging, and Cancer (IDAC), Tohoku University, Sendai, Japan.

- Lecture format（講演形式）:
◆Lecture time（講演時間） 80 min（分）, Q&A time（質疑応答時間） 30 min（分）
◆Lecture style（ex.: used projector, conducted experiments）
（講演方法（例：プロジェクター使用による講演、実験・実習の有無など））
Used projector

- Lecture summary（講演概要）: Please summary your lecture 200-500 words.

'Aging' is the term meaning the gradual declination of our life span to death. More simply, it is the term depicting the gradual changes from younger age to older age. Aging and aging associated disorders (such as diabetes, cancer, autoimmune diseases, neuronal disorders etc) are the burning question to scientists in the medical as well as life sciences. The dream of cheating death has evolved into a scientific desire to extend healthy life span. Scientists and doctors are looking for ways to maximize the number of years that we live free of chronic diseases, cancer, and cognitive decline. But before we can remedy, we have to understand the cellular and molecular mechanisms that drive aging and senescence.

Some clues can be mentioned that causes organismal aging, for example, shortening of telomeres, mitochondrial dysfunction, altered metabolism, cellular senescence, reactive oxygen species, aged stem cells and so on. Other research points to the decreased or aberrant antioxidant capacity. Current aging science initiatives aim to explain biological

mechanisms of aging, have provided insights into molecular processes that underlie biological aging and perhaps more importantly, potential interventions to delay aging and promote healthy longevity. Here I am describing some of the advanced approaches along with efforts to move aging science from the bench to the clinic. I am emphasizing on the cellular antioxidant system that play very important role in cytoprotection from oxidative stress.

NRF2 (nuclear factor erythroid 2- related factor 2) is a key transcriptional activator that mediates the inducible expression of antioxidant genes under oxidative stressed condition through the induction of its target genes that are involved in the detoxification or elimination of various toxic insults. Recent studies have unveiled the functional contributions of the KEAP1-NRF2 system and defined its broader involvement in biological processes, including cell proliferation and differentiation as well as cytoprotection.

Various studies using mouse model clarifies that *Nrf2*-null mice reflected susceptibility to xenobiotics. NRF2 plays cytoprotective role in case of liver damage. Pharmacologic activation of NRF2 signaling effectively attenuated cigarette smoke-induced emphysema, among many toxicological studies. NRF2 protects cells from DNA damaging insults, such as reactive oxygen species (ROS) and electrophilic toxicants, thereby inhibiting cancer initiation.

Aging associated disorders or metabolic disorders, such as obesity, diabetes and its complications, are closely related to dysregulation of oxidative stress and effectively treated with NRF2 inducers. NRF2 activation in each organ appears to orchestrate the anti-diabetic effects. Ionizing radiation, including UV and X-rays, is another important cause of oxidative stress. NRF2 inducers protect skin from the irradiation-induced dermatitis. Beside these, NRF2 also ameliorates chronic inflammation, which is beneficial to the treatment of autoimmune disease such as rheumatoid arthritis.

Based on profound contributions of NRF2 to prevention and alleviation of a wide variety of pathological conditions, worldwide efforts have been made to develop potent and potentially specific NRF2 inducing chemicals and few of the examples can be mentioned such as dimethyl fumarate (Tecfidera®), CDDO-Me, sulforaphane etc.

As the progression of aging associated disorders are executed at the cellular level due to increased oxidative stress, DNA damage, increased apoptosis-senescence, so that inhibition or prevention of these cytotoxic effects could be alleviated by the induction of NRF2-dependent antioxidant response pathway.

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- Overall advice or comments to future participants in the program（今後の講師へのアドバイス）:
Speaker should speak slowly by using simple sentences and easy words. Displying more cartoons in slides would be better.

- Other noteworthy information（その他特筆すべき事項）:
N/A

- Impressions and opinions from a company（同行者の方から、本事業に対する意見・感想等がありましたら、お願いいたします。）
N/A