

一般演題：P2-01～P2-29、P3-30～P3-56

P2-01**Clinical study of proteinuria affected by menstrual blood**

(社会人大学院博士課程3年腎臓内科学)

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【Objective】 On latest clinical practice, urinalysis is not conducted for several days at menstrual period because it disturbs an accurate evaluation of the protein urine affected by menstrual blood. However, it is not clear how menstrual blood gives any influence on proteinuria. As adult women generally have menstrual cycles about every month, it is not rare case that the days of the menstrual cycle hit on date of consultation at the hospital. In this study, we investigated how hemoglobinuria caused by the hemolysis influenced protein urine.

【Methods】 For seven years from April 2011 to March 2017, clinical surveys were conducted in 746 general adult women volunteers among patients of Tokyo Medical University Hospital. They had quantitative or qualitative examination of urine in the timing of both monthly and non-monthly periods. These data were analyzed by the correlative and regression analysis. This study was approved by our ethics committees.

【Results】 There is no effect on proteinuria by the examination for urine fixed-quantity in a monthly and non-monthly periods. If there is little occult blood in the examination for urine qualitative analysis at menstrual period, it may not affect proteinuria.

【Conclusion】 This study highlighted the effect of menstrual blood on proteinuria. This is the first retrospective study having been examined how menstruation gives an influence on proteinuria. It may contribute to showing that a little occult blood has no influence on a

quantity of protein urine. However, the result has not yet sufficiently clear. A further study is necessary to analyze the effect of menstrual blood to proteinuria.

P2-02**ESI-09 and HJC0197, known EPAC inhibitors, sensitize lung cancer cells to glucose starvation by uncoupling mitochondrial electron transport, leading to bioenergetic crisis**

(社会人大学院博士課程3年呼吸器内科)

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Cancer cells utilize aerobic glycolysis and proliferate continuously beyond the capacity of their blood supply, leading to microenvironmental stresses such as hypoxia, nutrient (glucose) deprivation and extracellular acidosis. We have previously showed that extracellular acidosis prevents glucose starvation-induced death of lung cancer cells by reducing glycolytic energy production and de novo protein/RNA synthesis that consume ATP (AACR 2018). This acidosis-dependent, glucose starvation-resistant, and ATP-saving phenotype is thought to serve as an adaptive response to glucose deprivation in cancer cells to survive an energy-restricted tumor microenvironment. In the present study, we screened small-molecule inhibitors to explore therapeutic reagents that can exert cytotoxicity against the glucose starvation-resistant phenotype in the lung cancer cell lines, A549 and H1299 cells. Among the various inhibitors tested, we found that ESI-09 and HJC0197, known as inhibitors of the exchanger protein directly activated by cAMP (EPAC), reduced the cellular ATP levels and survival under glucose deprivation stress under both acidic (pH 6.8) and neutral (pH 7.4) conditions. Interestingly, the ATP-reducing effect of ESI-09 and HJC0197 was not