UGM FK-KMK Doctoral Program Student Conducts Research on Anticancer Compounds

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Cancer is the second most dangerous illness leading to the cause of death in the world, which is responsible for 9.6 million deaths in 2018. One of the most common factors causing cancer is a mutation in the wild-type p53 gene.

Student of the Doctoral Program of the UGM Faculty of Medicine, Public Health, and Nursing (FK-KMK), Jeffry Julianus, M.Sc., raised a topic to be presented in his dissertation entitled "Synthesis, Anticancer Activity Testing, and Computational Study of Cardiene Compounds and Their Derivatives as Reactivating Pathways p53 in the Cell line Carrying Mutant p53". He presented this dissertation in the online open examination of the doctoral program on Thursday (5/11).

"This research includes several fields of science to produce a new anticancer compound that selectively targets mutant p53," explained Jeffry.

In his dissertation, he also explained that cancer is a disease caused by cancer cells' presence. These cells have characteristics including the ability to increase continuously, insensitivity to growth-inhibiting signals, and the immune system cannot damage it, replicates continuously, occurs

inflammation, and deregulation of cellular energy.

"Cancer has an ability that is difficult to treat until now," he said.

Jeffry also explained that the fields of organic compound synthesis, molecular pharmacology, and computational studies are directly involved in discovering these new anticancer compounds. This study's results are beneficial for promoting this field of science in discovering an anticancer compound.

P53 itself is a protein transcription factor that significantly takes a stand in maintaining genome stability. Therefore, it is called the genome's guardian. This protein is stable at physiological temperatures, has a melting point of 44 ° C, a half-life of 9 minutes, and requires Zn2 + ions to have the right conformation.

The application of anticancer compounds targeting mutant p53 may increase therapeutic efficacy. The success of converting the mutant p53 conformation to the p53 wt conformation can treat 50 percent of cancers. The difference between the mutant p53 and p53 wt conformation gives the anticancer compound a chance to target mutant p53 selectively.

"The selectivity of the anticancer compounds decreases the toxic effect on normal cells," he added.

Author: Gloria
Translator: Natasa A

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