# PROF. DR. AZIZ SANCAR'S STUDIES ON CANCER

Казанский федеральный
УНИВЕРСИТЕТ

# PROF. DR. AZIZ SANCAR'S STUDIES ON CANCER


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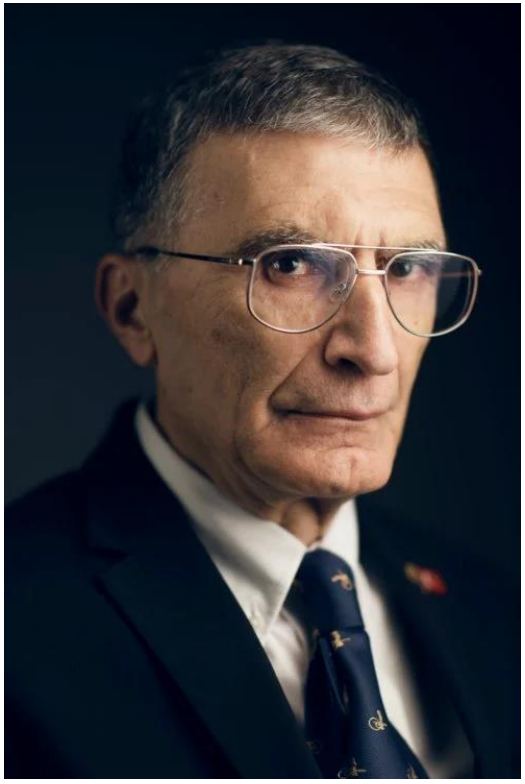
## Short Biography



Https://www.nobelprize.org/prizes/chemistry/2015/sancar/facts/

- He graduated from Istanbul University Faculty of Medicine, which he entered in 1963, with first place in 1969.
- He went to Johns Hopkins University with a NATO-TUBITAK scholarship and continued his research there for 1.5 years.
- Aziz Sancar was in the medical faculty of Yale University between 1977-1982. In this period, he stopped his studies of photolyase enzyme and started researches on nucleotide cut repair.
- He completed his associate professorship thesis in DNA repair. Since 1997, he has been continuing his research in the biochemistry and biophysics department of the University of North Carolina in Chapel Hill, North Carolina, USA, which is known for his studies in biochemistry and biophysics.
- Aziz SANCAR has been working for more than 30 years and has found direct causal relationships between nucleotide cut repair disorders and colon cancer and leukemia diseases and was awarded the 2015 Nobel Prize in Chemistry by the Royal Swedish Academy of Sciences in the field of "mechanistic studies of DNA repair".

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- Turkish scientists from Koç University conducted a study that could destroy this protein with "medicine". The research conducted by scientists from Koç University, Istanbul University, Istanbul Faculty of Pharmacy, Medipol University and Gebze Technical University was chaired by Koç University Professor of Biological Engineering and Molecular Biology and Genetics. Dr. Halil KAVAKLI is one of the main names of this research.

Stating that successful treatments are carried out in Türkiye in this regard, Aziz Sancar continued as follows:

The father of one patient showed me a summary of the interventions performed on his daughter Our Turkish doctors are far ahead of many countries of Europe and many states of the USA in the field of surgery, very successful and modern surgeries can be performed and can be successful results can be obtained. In this regard, continue to be optimistic but this EdU is not at the stage to be used at the moment, I hope we will get positive results, we will have done something useful for Türkiye, our people and humanity."

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- Türkiye is looking for solutions to defeat cancer and is doing serious work in this field, and ı want to say that Türkiye's prestige is increasing in the field of medicine. This cancer-preventing 'p53' gene, which we consider as an example, causes cancer in some people because it is low that is why new studies have been done, new molecules with no side effects were found, when tested on mice, it was found to have 25% more life. In the next 5-6 years, it will be sold as a medicine in pharmacies and other countries

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- This project, which was carried out with the support of Prof. Dr. Aziz SANCAR, contributed to the development of this scientific discovery in universities in the USA in the past years and Türkiye still working on this scientific discovery, Russia also working on this scientific discovery, Russian scientists at the Saint Petersburg State Institute of Technology recently began work on the p53 gene Russian scientists have also studied another protein, MDM2, which could lead to safer chemotheraphy drugs in the future.

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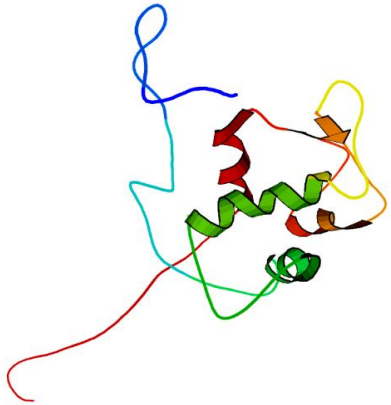
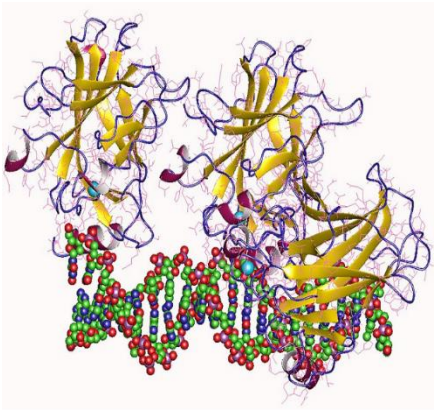
- The p53 suppressor gene was found to be the most frequently mutated gene in human cancers. Under normal conditions, p53 is found in the cell in an inactive state, at low concentration and as a short half-life. The protein called MDM2 plays a role in the regulation of stabilization. MDM2 binds to the amino end of p53, suppressing its transcriptional activity, and directing it to complexes called proteasomes, where it provides degradation (proteolysis). Interestingly, p53 activates transcription of the MDM2 gene.



**MDM2**

https://en.wikipedia.org/wiki/Mdm2



**p53**

https://tr.wikipedia.org/wiki/P53

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What about p53 and cancer treatment

- Interventions for the MDM2 protein: It is aimed to reduce the expression of MDM2, a protein that keeps the intracellular level of p53 under control, with anti-sense oligonucleotides. This method has been shown to have an "in vivo" effect in breast cancer.
- One of the other treatments is, Benefiting from the cytolytic effect of adenoviruses by proliferating in p53-deficient tumor cells: Actually, there is no interference with p53 function here. In order for adenoviruses to multiply in the cell they enter, p53 must be out of action. Since p53 function is abolished in tumor cells, it was hoped that by injecting this virus into the tumor (direct injection or systematic administration), the tumor could be reduced as a result of cytolysis. The virus itself encodes a protein called E1B that binds to p53 and neutralizes it. It was thought that the genetically modified (ie non-E1B-encoding) strain of the virus, called dl 1520 (or ONYX-015), would not be able to replicate in intact tissue and would therefore be tumor-selective. Indeed, in Phase I-II studies in head and neck tumors, pancreatic and ovarian cancers, a partial necrosis of the tumor was observed while intact tissue was preserved. However, it appears to be ineffective as a single agent. However, when combined with chemotherapy, it gives more favorable results than chemotherapy alone.

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## Referencess

For the preparation of this work, materials from the sites were used:


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## Thank you for your attention!

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