Tackeling Cancer at Molecular Level

As per the WHO stats, Cancer, the 2nd leading cause of death worldwide, happens due to mutations in functional genes. According to the American Cancer Society, about 5% to 10% of all cancer are due to inheritance of the cancer-causing mutation. For remaining cases, environmental factors such as UV radiation, exposure to a carcinogen or the lifestyle of an individual is responsible. There are multiple proteins involved in the growth of a cell. Proteins are the factors that are involved in almost every aspect of cellular function. Abberation in their function leads to either cell death or abnormal growth leading to tumour formation which is termed as a “malignant tumour” (Type of tumour that represents cancer). Proteins are manufactured in the cell through a series of events from DNA to messenger RNA (mRNA) and to Protein.

There was a theory called “one gene-one protein”, it means one gene can only code for one protein. But further research found that one gene can code for multiple proteins. Answer for this lies at the mRNA level. Matured mRNA is composed of patched of sequence that is joined together in different combination to generated multiple protein isoforms, by the process called as alternative pre-mRNA splicing. There is a bunch of proteins that carry out this function depending on modifications on mRNA. One such modification is “m6A”, which is added by a group of proteins called “m6A writers”. Further read by “m6A reader” proteins to execute the function associated with it. One such function, alternative pre-mRNA splicing is carried out by these m6A readers.

My work is mainly focused on such m6A reader to identify its role in alternative pre-mRNA splicing and can be used as a novel drug target to interfere with its function that probably will lead to reduced cell growth in malignant tumours. Mechanistically, m6A is added to GGACU consensus sequence in the mRNA, which is mostly found near splice sites in the mRNA. So the m6A reader along with other auxiliary proteins helps to splice specific factors to either include or exclude a patch of sequence in the mRNA generating different isoforms. These isoforms could be the factors that need for cell growth and survival. So if we alter the function of this m6A reader protein by targeting with drug molecule, we could avoid the abnormal cell growth in a malignant tumour.

Mr. Rajnikant Dilip Raut
(Research Scholar)
Department of Liberal Arts