C.N.R. Rao Prize winner Muntaser Ibrahim of Sudan spots genetic differences that explain why some people are more susceptible to infections than others.

Why are some populations more prone to malaria, cancer and other diseases? The answer is encrypted in our genes, and molecular biologist Muntaser Ibrahim, from the University of Khartoum in Sudan, is working to decipher this hidden message.

Muntaser Ibrahim – a founder of the Sudanese Academy of Sciences and an expert on population genetics – is the winner of the 2014 C.N.R. Rao Prize for Scientific Research. He received the prize during the opening ceremony of TWAS’s 25th General Meeting in Oman for his life-long commitment to understanding the role of human genetic variations and populations’ structure in disease susceptibility. His discoveries bring scientists closer to developing effective vaccines and novel treatments against common diseases.

TWAS awards the C.N.R. Rao prize every year, to reflect the innovative spirit and accomplishments of Indian chemist C.N.R. Rao, a TWAS Founding Fellow, former Academy president and a leading scientist in the field of solid-state chemistry and materials science.

Ibrahim’s investigations address malaria, cancer and infectious diseases that are common in Africa. Malaria, in particular, is a global burden, killing about a million people worldwide every year, 90% of them in Sub-Saharan Africa. Effective vaccines are still missing because of the complexity of the disease.

Malaria is caused by the parasite Plasmodium. To complete its life cycle, Plasmodium needs two hosts: the mosquito Anopheles, a vector that hosts and spreads Plasmodium; and humans, who are the final recipients that Plasmodium invades via a mosquito’s bites.

"Malaria is very interesting. Different populations show different susceptibility to the disease, both at the immunological and genetic levels," Ibrahim explained. “They do not respond in the same way to the infection – some never develop the disease, while others do – because of variations in their genomic settings.”

This unusual susceptibility prompted Ibrahim to investigate 15 ethnic groups across Africa, looking for gene variants that could explain differences in the infection pattern. In the study, carried out with colleagues from the consortium MalariaGen, the scientists found DNA mutations in two genes coding for proteins that Plasmodium uses to bind human red blood cells, where its life cycle is completed.

These mutations change the final shape of the protein that the parasite uses to enter red blood cells. Different populations show different rates of protective mutations in these DNA hot spots, and also variations in their susceptibility to the infection.

By tracking how local communities have evolved protective mechanisms against parasites, and how the parasites are co-evolving with humans, Ibrahim is writing a chapter in the book of ancient migrations across Northeastern Africa.

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