The second case of leukemia-like disease in a gene therapy patient in France appears to have the same basic cause as the first one, a scientist investigating the incident said yesterday.

In both cases, it appears the gene inserted into the boys’ cells to cure their disease accidentally landed on or near a cancer-causing gene and switched it on, said the scientist, Dr. Christof von Kalle of Cincinnati Children’s Hospital Medical Center, who is collaborating with the French researchers.

Dr. von Kalle said his preliminary assessment, which is still to be verified, suggested that the problem might be restricted to the particular gene therapy regimen in France. “This is something that is so specific and unique I would say it would not have been possible to predict this,” he said.

The Food and Drug Administration on Tuesday suspended 27 clinical trials of gene therapy after learning of the second case of leukemia. The halt was another setback to the fledgling field of gene therapy because the French experiment, in which 9 of 11 boys were essentially cured of a fatal immune deficiency, had been considered the first unequivocal success for the technique.

But three years after their treatment, two of those nine boys have developed uncontrolled growth of blood cells characteristic of leukemia. They are responding to chemotherapy, according to the National Institutes of Health.

In both cases, the gene inserted into the boys’ blood-forming stem cells landed on or near an oncogene, or cancer-promoting gene, called LMO-2, which can spur childhood leukemia. In the first case the gene landed inside LMO-2 and the second landed near enough to turn on the gene, Dr. von Kalle said.

The gene therapy used viruses to carry the therapeutic cells into the children’s blood-forming stem cells. The virus lands at random on the cell’s DNA. So scientists have long known there was a risk that the virus could land on a cancer-spurring gene, but it was thought that the chances of its happening were small.

Dr. von Kalle said the chance of a virus landing on LMO-2 was about 1 in 100,000. But since each child was given about one million cells, the probability is very high that a child received at least one cell in which the virus landed on the gene. That makes it important to monitor the other children closely, he said.

But Dr. von Kalle said scientists believed that turning on just one oncogene would not be a problem because it usually requires multiple genetic changes to turn a cell cancerous.

“There’s no human cancer model where you can see that one gene got turned on and it gave you a cancer,” he said.

Dr. von Kalle’s hypothesis is that a second genetic change came from the therapeutic gene itself. The gene put into the French children was designed to spur growth of infection-fighting blood cells, since the boys suffered from a lack of such cells, meaning they would be killed by infections. It is possible that the therapeutic gene combined with the activated oncogene to cause a surge of such blood cells, Dr. von Kalle said.

If this hypothesis is confirmed, it would indicate that gene therapy treatments in which the inserted gene is not a growth-promoting one would not face the same risk, he said.