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Epigenetic modification associated with type ii interferon gamma (IFNG) in chalenge virus standard strain (CVS) and nigerian street rabies virus (SRV) infected mice.

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Statement of Research Problem: Rabies is one of the most feared known human diseases recognized since the early period of civilization with 55,000 and 24,000 deaths recorded annually worldwide and in Africa respectively. Despite extensive investigation in the past 10 decades, rabies remains a public health threat around the globe. Rabies virus (RABV) has evolved specific mechanisms to escape early immune system recognition and avoid premature death of infected neurons. RABV relies on mechanisms, such as suppression of interferon response. The Purpose of this study is to establish whether or not, Rabies Virus adopts Epigenetic Mechanism for the suppression of the interferon system.

Methodology & Theoretical Orientation: Mouse inoculation test (MIT) and direct fluorescent antibody test (DFAT) methods were used to inoculate and check for the presence of RABV in the brain of infected mice. A real time quantitative PCR was used to check the expression level of interferon gamma (IFNG). A methylation specific quantitative PCR was used to check the methylation status at the promoter region of IFNG. ELISA was carried out to check DNMT and HAT activity. Findings: From the result, the expression level of IFNG was shown to increase at early stages of infection and decrease as disease progressed. There was no significant difference in the methylation status of the test group compared to the control group, an almost similar result was obtained for the ELISA with no changes in the level of DNMT and HAT from the normal.

Conclusion & significance: Rabies virus affects the level of IFNG production by interfering with its production in a diseased state. However, Epigenetic mechanism (DNA methylation) cannot be categorically linked to Rabies virus infection, thus, its mechanism of action is not fully understood with recommendations of exploring other mechanisms it uses to suppress immune system.