



St. Jude Global Scholars Projects: Yusuf Danasabe Jobbi



Unveiling the Diversities of Beta Globin Gene and Associated Defective MBL2 Gene Variants: as Predictors of Bacteremia Among Sickle Cell Disease Children in Kano, Nigeria

This project aimed to investigate gene polymorphisms that are associated with Mannose Binding Lectin Gene2 (MBL2G) and haplotypes associated with Human Beta Globin (HBB gene) and their association with clinical outcomes including bacteremia among children with sickle cell disease (SCD) in Kano, Nigeria.

This study improved understanding of the clinical, demographic, and laboratory associations between implicating genes and certain clinical outcomes. It also supplied benchmark data to support innovative experimental protocol related to genomic studies in SCD in the future. The project is now transitioning into a new phase focused on institutional integration and collaboration.

Discussions are underway to expand the work to provide biobanking services to study subject samples for other genomic surveillance and profiling studies. The data infrastructure, stakeholder networks, and implementation frameworks established during the initial phase will serve as the foundation for broader genomic surveillance and pediatric health care initiatives.

Project Details



Location
Kano, Nigeria



Setting
Multiple Centers
within a country



Health Condition
Sickle Cell Disease



Focus Areas
Equity, Quality,
Timeliness



Target Age Group
Birth to 12 years

Systems-Level Change

Participants

561 Children

24 Health Care Workers

585 Caregivers

UN Sustainable Development Goal

Goal 3: Good Health and Well-Being

Goal 4: Quality Education

Goal 8: Decent Work and Economic Growth

Goal 9: Industry, Innovation and Infrastructure

Goal 10: Reduced Inequalities

Goal 17: Partnerships for the Goal



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Key Findings

The following are key findings from this Global Scholars Project which aimed to identify genetic diversities related to polymorphisms linked to bacteremia among children with sickle cell disease in Kano, Nigeria.

- **Genetic Predisposition to Infection:** Variants of the MBL2 gene, particularly low-function polymorphisms, impair opsonization and complement activation, thereby increasing susceptibility to bacteraemia in children with sickle cell disease.
- **Regional Genetic Influence:** The Benin HBB haplotype and SS+F genotypes reflect a regional genetic signature that modulates inflammatory responses and haemolysis.
- **Age-Dependent HbF Protection:** Children under 5 years of age maintained HbF levels of ~18%, compared with $\leq 14\%$ among children aged 5-12 years. This decline in HbF marks a critical window of heightened infection risk as children age.
- **Hematologic Markers of Inflammation:** Elevated white blood cell and neutrophil counts in older children serve as indicators of chronic subclinical infection or persistent inflammation.

WHO Health System Building Blocks

Health service delivery

Health workforce



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Key Findings

- **Impact on Immunization Efficacy:** A significant association ($p < 0.01$) was observed between MBL2 single nucleotide polymorphisms and immunization status, suggesting that genetic deficits may diminish vaccine-induced protection.
- **Socioeconomic Risk Amplification:** Environmental determinants, such as low household income, exacerbate biological vulnerabilities by delaying diagnosis and limiting access to prophylaxis.
- **Translational Therapeutic Targets:** BCL11A was identified as a key regulator of HbF persistence, supporting its potential as a target for gene therapy to reduce infection-related complications.
- **Clinical Recommendations:** Early genomic mapping should be integrated into SCD management to enable proactive antibiotic prophylaxis and tailored immunization strategies.

The data reveal that bacteraemia in children with SCD is not merely a clinical complication but a genetically mediated vulnerability. MBL2 and HBB variants interact with age-dependent hematologic changes to shape infection risk. Early genomic profiling can identify children at highest risk, enabling proactive immunization, antibiotic prophylaxis, and future gene therapy targeting BCL11A.



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