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CC –BY **G6PD deficiency and ABO incompatibility are far greater causes of severe jaundice than rhesus disease in Nigerian neonates**

DOI:<http://dx.doi.org/10.4314/njp.v45i3.7>

Accepted: 17th September 2018

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Dear Editor,

We read with interest the Conference summary report of the 10th Annual General and Scientific Meeting of the Nigerian Society of Neonatal Medicine (NISONM) held in July 2017 at Ibadan, published in a prior issue of this journal.¹ We congratulate the leadership of NISONM for disseminating the key messages from this important conference for the benefit of the readers of the journal and other stakeholders who could not attend the meeting. We also commend the concerted efforts on many fronts to advance the care of newborns in Nigeria through global initiatives and collaborations.

However, we were deeply disturbed to read the suggestion by one of the invited speakers at the conference, Dr. Vinod Bhutani from Stanford University, USA and co-founder of CURhE (Consortium for Universal Rhesus Elimination), attributing the high rates of exchange transfusions in Nigeria to the high prevalence of rhesus incompatibility among newborns. This claim is misleading and unfounded based on the extensive body of literature on the burden of neonatal jaundice published in Nigeria since 1960.² Rather, available evidence clearly shows that glucose-6-phosphate dehydrogenase (G6PD) deficiency, (frequently exacerbated by exposure to icterogenic agents or oxidant stressors such as insecticides, menthol-based, naphthalene-camphor products, sulfonamides or sulfa-containing drugs and herbal concoctions), ABO incompatibility and sepsis are the far leading causes of haemolytic hyperbilirubinaemia requiring exchange transfusion.² Besides, there is no evi-

dence in this journal or elsewhere to suggest any epidemiological shift in the leading clinical risk factors for neonatal hyperbilirubinaemia in Nigeria.

More crucial is the contribution of, are non-clinical risk factors linked to the three levels of delay commonly associated with bilirubin-induced mortality and neurodevelopmental disorders, including kernicterus namely: i) the timeliness of the decision to seek appropriate care by mothers at the onset of jaundice, ii) reaching an appropriate health facility promptly, and iii) receiving adequate/appropriate care at the health facility.³ This is corroborated by our experience in prospectively investigating the pattern and burden of acute bilirubin encephalopathy in a foremost referral children's hospital in inner-city Lagos since 2008 which has been reported in several reputable journals.⁴⁻⁶ While we do not dispute the importance of rhesus incompatibility for severe neonatal jaundice in general,⁷ we feel constrained to draw attention to the exaggeration in Dr. Bhutani's presentation as to the scale of the problem among newborns in Nigeria, compared to G6PD deficiency with an estimated national prevalence of at least 15.0%. In fact, routine screening for G6PD deficiency is considered a priority for all malaria-endemic populations, and Nigeria should not be an exception.⁸ Besides, neonatologists have a far more limited role to play in the effective elimination of rhesus disease as championed by CURhE compared to obstetricians and public health practitioners. While the potential severity of haemolytic jaundice should be recognised in all clinical settings, it is important for paediatricians and child health specialists to disseminate and commit to evidence-based practices in advancing the interest of every newborn child in Nigeria building on our local expertise generated over many decades of dedicated research work.

Conflict of interests: None

Funding: None

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