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# The Interaction of Malnutrition and Neurologic Disability in Africa

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Malnutrition and neurodisability are both major public health problems in Africa. This review highlights key areas where they interact. This happens throughout life and starts with maternal malnutrition affecting fetal neurodevelopment with both immediate (eg, folate deficiency causing neural tube defects) and lifelong implications (eg, impaired cognitive function). Maternal malnutrition can also increase the risk of perinatal problems, including birth asphyxia, a major cause of neurologic damage and cerebral palsy. Macronutrient malnutrition can both cause and be caused by neurodisability. Mechanisms include decreased food intake, increased nutrient losses, and increased nutrient requirement. Specific micronutrient deficiencies can also lead to neurodisability, for example, blindness (vitamin A), intractable epilepsy (vitamin B6), and cognitive impairment (iodine and iron). Toxin ingestion (eg, from poorly processed cassava) can cause neurodisability including a peripheral polyneuropathy and a spastic paraparesis. We conclude that there is an urgent need for nutrition and disability programs to work more closely together.  
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## Introduction

Both malnutrition and disability are major global health problems. Over 1 billion people worldwide are malnourished<sup>1</sup> and 1 billion live with a disability.<sup>2</sup> Important links and interactions between both are attracting increasing

international attention with growing recognition of the potential for synergy and mutual benefit by approaching both the issues together.<sup>3,4</sup>

In Africa, a recent Lancet review estimated that 56 million out of a total 158 million children (United Nations population estimates) younger than 5 years are stunted (chronically malnourished, indicated by low height for age); 6 million are severely wasted (acutely malnourished, indicated by low weight for height or low midupper arm circumference); 28 million are underweight (a combination of acute and chronic malnutrition, indicated by low weight for age).<sup>5</sup> Micronutrient malnutrition is also common: 42% of children in Africa have vitamin A deficiency (serum retinol <0.70 μmol/L), 40% are iodine deficient (<100 μg/L), 24% are zinc deficient, and 20% are iron deficient (<110 g/L).<sup>5</sup> Many children are affected by multiple forms of malnutrition in varying combinations and degrees.

Childhood disability is also common in resource-poor settings: 80% of the world's disabled population, of all ages, live in low-income countries, many in Africa.<sup>2</sup> Neurologic impairment is a particular problem given struggling health services and frequently suboptimal treatment of common conditions, such as perinatal asphyxia, jaundice, and infection (meningitis and cerebral malaria). Prevalence estimates

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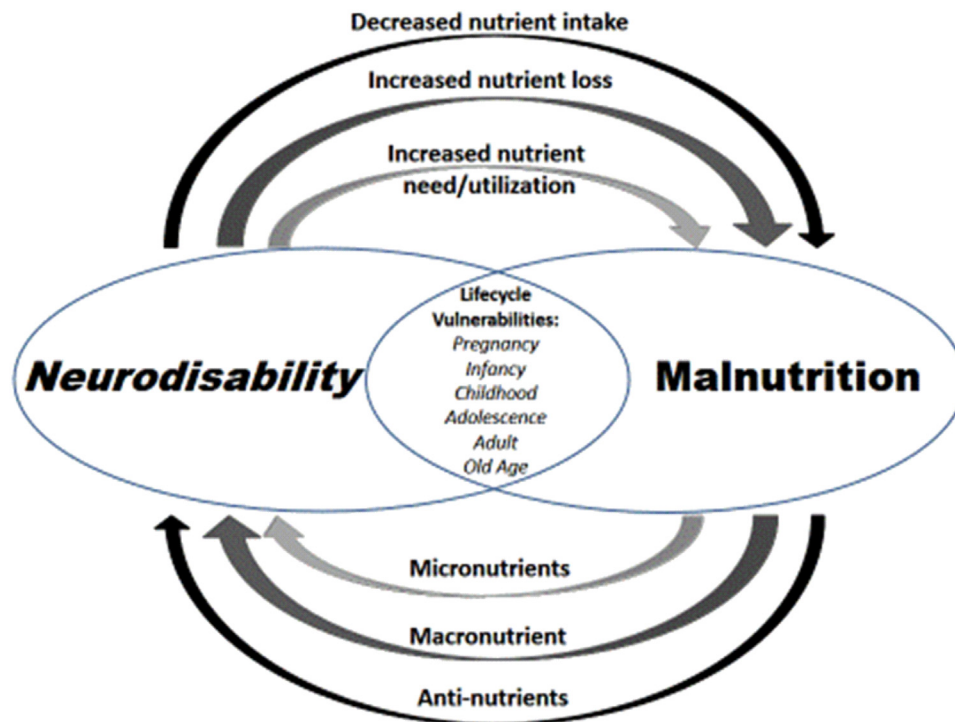
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**Figure 1** Neurodisability-malnutrition interactions: how neurodisability affects malnutrition and malnutrition affects neurodisability. (Color version of figure is available online.)

for childhood neurologic impairment range from 19–61 per 1000.<sup>6</sup>

In this review, we consider the relationship between malnutrition and neurologic disability, with a focus on Africa. Taking a life-cycle approach, we discuss how malnutrition can cause or affect neurodisability, and, in turn, how neurodisability affects nutrition (Fig. 1).

## Neurodisability and Nutrition in Pregnancy and Infancy

Maternal health and nutrition are key to optimal fetal development. Risk factors for poor nutrition are common in Africa and include poverty,<sup>7</sup> maternal ill health, and depression.<sup>8</sup> Though humans have evolved to prioritize maternal resources toward the developing fetus/infant,<sup>9</sup> maternal malnutrition can result in fetal growth restriction and low birth weight (<2500 g).<sup>5</sup> Most neurodevelopment is completed during the first 1000 days after conception, that is, during intrauterine life and infancy,<sup>10</sup> and optimizing nutrition during this period has become a global priority.<sup>11,12</sup>

Maternal malnutrition can result in both global and specific neurodevelopmental sequelae. Children of underweight women are reported to have an increased risk of delayed mental development (relative risk = 1.36, 95% CI: 1.04–1.78).<sup>13</sup> The hippocampus (memory), the cortex, and auditory development are particularly vulnerable to malnutrition in early pregnancy.<sup>14</sup> Other effects of early intrauterine malnutrition only become apparent in later life,<sup>10</sup>

and include adolescent and adult problems, such as attention-deficit disorder, conduct problems, and eventual low socioeconomic status.<sup>15–17</sup> Prenatal exposure to daytime fasting during Ramadan has been reported to increase the likelihood of adult disability by more than 20% in Iraq and Uganda, with substantially larger effects on mental and learning disabilities.<sup>18</sup> Though from a very different setting and time, important warnings for African populations at risk of maternal malnutrition can be drawn from seminal studies of individuals exposed in utero to the “Dutch famine” (resulting from severe wartime food shortages during the winter of 1944–1945). A variety of neurodevelopmental sequelae included increased risk of schizophrenia, increased response to stress, and poorer cognitive performance.<sup>19</sup> Timing of the nutritional insult is important, those exposed early in gestation being at greatest risk. Other adult disabilities are less directly linked with fetal undernutrition. Sequelae, such as coronary heart disease and diabetes,<sup>18</sup> are key risk factors underlying stroke, a major cause of adult-onset disability.<sup>20</sup>

Specific maternal nutritional deficiencies and their effects on infant neurodevelopment are shown in Table 1.

They are seldom found in isolation and it is common to have multiple deficiencies against a background of maternal undernutrition, frequent ill health, and poverty. Time-specific nutritional deficiencies during a pregnancy can alter genotype expression and induce abnormal phenotype. The developing brain between 24 and 42 weeks of gestation is particularly vulnerable to nutritional insults because of the rapid trajectory of several neurologic processes, including synapse formation and myelination. The brain’s

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