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Dear editor

In their case control study, Tsegaye et al 1 by introducing the stool antigen test (SAT) for the diagnosis of *Helicobacter* infection (*Hp-I*) concluded that *Hp-I* is linked with preeclamptic pregnant women in Ethiopia associated with metabolic syndrome (MetS) parameters such as body mass index, diabetes mellitus, dyslipidemia and arterial hypertension. Although the SAT has been introduced broadly in Africa, the limitation of this study, as also mentioned by others 2 is that only the SAT was used. By introducing several diagnostic approaches increases the detection of *Hp-I*. 2

Specifically, despite the advantages (easy to perform and rapid for *Hp* detection), SATs display several disadvantages. Because the principle of SAT is an antigen–antibody reaction, differences in antigens used for SAT in different geographic regions may cause heterogeneity of results; the diagnostic performances of different SATs are heterogeneous, and this could relate to the designs of the test like enzyme immunoassay and immunochromatographic assay (ICA) and for the selection of antibody, such as monoclonal antibody and polyclonal antibody. SAT shows a high rate of false positivity. Moreover, SAT negative results may not signify the absence of *Hp-I* because the low gastric colonization of bacteria results in low concentration of *Hp* antigen in the sample. Furthermore, concerning ICA-based methods, subjective interpretation of the results, especially for the tracing line, makes it problematic to diagnostic accuracy. The accuracy of SAT is influenced by several factors, including antibiotics, proton pump inhibitors, N-acetylcysteine, bowel movement and upper gastrointestinal tract bleeding. Preservation of the specimen, like temperature and transport time before testing, and cut-off valve also have impacts on the SAT diagnostic accuracy; if study results are not compared to the gold standard examinations for the diagnosis of active *Hp-I* (histology, rapid urease test, culture or urea breath test), positive or negative results are still controversial.

In this regard, important to note that, only active *Hp-I* induces humoral and cellular immune responses that, owing to the sharing of homologous epitopes (molecular mimicry), cross-react with components of the host, thereby contributing and possibly perpetuating *Hp*-related systemic disorders 3–5 including preeclampsia. 5 In this respect, maternal inflammatory immune reactions appear to be involved in the pathogenesis of preeclampsia; several studies of both patients and animal models have established that changes in the immune system (cellular plus humoral immunity and/or adaptive-innate immunity) could contribute to the pathophysiology of preeclampsia.

*Hp-I*, a worldwide burden with a mean global prevalence of 58% and accounting of about 4.4 billion of *Hp* infected individuals, is also related with MetS-systemic systemic pathologies such as preeclampsia, cardio-cerebrovascular, and neurodegenerative diseases, the end outcomes of MetS; 3–5 and it appears to be a critical risk factor of MetS in pregnancy and influences the occurrence of many opposing pregnancy outcomes 5 such as gestational diabetes mellitus, arterial hypertension, hyperemesis of pregnancy, preeclampsia in obese or overweight pregnant women, fetal growth restriction,
premature delivery, abortion, Hp-I of newborn, and neural tube defects. Moreover, eradicating Hp-I may reduce or delay such systemic pathologies. 

Therefore, active Hp-I-related MetS may affect pregnancy critical outcomes, Hp eradication may benefit pregnant women and their neonates and thus further studies are needed.

Disclosure
The authors report no conflicts of interest in this communication.

References