Gonadotropin responses to *Plasmodium vivax* infection: Proximate mechanisms of reproductive suppression during illness in Honduran men

Michael P. Muehlenbein and Richard G. Bribiescas

Indiana University, Yale University

**Introduction**

Suggestion of current reproduction in order to increase the likelihood of successful future reproduction should function as a basic aspect of male phenotypic plasticity and an adaptive response that facilitates the allocation of metabolic resources according to available energy and disease risk in a stochastic environment. Assuming testosterone’s immunosuppressory actions are primarily suppressive (for review see Muehlenbein and Bribiescas, 2005), depressed testosterone levels during illness or injury could function to prevent immunosuppression by otherwise higher testosterone levels (Zlotnik and Last, 1988). Augmenting steroidogenesis could thus increase the amount of energy and nutrients available for somatic repair and the maintenance and elicitation of immune responses. (Muehlenbein and Bribiescas, 2005; Shelton and Verhulst, 1996; Wedekind and Folstad, 1994). Peripherial testosterone levels may vary as a function of altered production and secretion, conversion to other hormones, uptake into tissues, and altered metabolic clearance. Estrogens, thyroid hormones, insulin, growth hormone, and/or new hormones binding globally can all alter testosterone levels. How we provide evidence that FSH, LH and inhibin B levels are not significantly altered in this sample of men infected with *Plasmodium vivax* reveals much about the route of testosterone variation as a function of disease risk in a stochastic environment. Assuming testosterone’s immunosuppressory actions are primarily suppressive (for review see Muehlenbein and Bribiescas, 2005), depressed testosterone levels during illness or injury could function to prevent immunosuppression by otherwise higher testosterone levels (Zlotnik and Last, 1988). Augmenting steroidogenesis could thus increase the amount of energy and nutrients available for somatic repair and the maintenance and elicitation of immune responses. (Muehlenbein and Bribiescas, 2005; Shelton and Verhulst, 1996; Wedekind and Folstad, 1994). Peripherial testosterone levels may vary as a function of altered production and secretion, conversion to other hormones, uptake into tissues, and altered metabolic clearance. Estrogens, thyroid hormones, insulin, growth hormone, and/or new hormones binding globally can all alter testosterone levels. How we provide evidence that FSH, LH and inhibin B levels are not significantly altered in this sample of men infected with *Plasmodium vivax* reveals much about the route of testosterone variation as a function of disease risk in a stochastic environment. 

**Methods**

**Where:** Tocoa, Honduras.

**Who:** 8 adult men diagnosed with *Plasmodium vivax*. Exclusion criteria included history of known endocrine disorder, current or recent use of hormone therapy, history of infectious disease, minor surgery, and current prescription treatment for any disease. 19 healthy age-matched controls were also sampled.

How: Those seeking treatment of malaria-like symptoms at Tocoa Hospital were diagnosed via examination of thick and thin blood smears and were recruited into the study following a signed informed consent and health and demographic questionnaire. Permission to conduct this research was granted by the Ministry of Health, and the Research Ethics Committee de Colon, Honduras. The project was approved by the Yale University Human Subjects Committee.

**Results**

An previously reported, testosterone levels were significantly lower during infection compared to after recovery.

**Punch line:** Although power was less than 0.05, these analyses because of small sample size, data suggest that hypogonadism in response to malaria in men may result from cytokine, glucocorticoid or other non-androgenic hormone effects directly in the testes rather than the pituitary.

**Why should we care?**

Similar to the adaptability of female ovarian function, changes in testosterone levels throughout the range of physiological variation may function as a basic aspect of male phenotypic plasticity and an adaptive response that facilitates the allocation of metabolic resources according to available energy and disease risk in a stochastic environment. Assuming testosterone’s immunosuppressory actions are primarily suppressive (for review see Muehlenbein and Bribiescas, 2005), depressed testosterone levels during illness or injury could function to prevent immunosuppression by otherwise higher testosterone levels (Zlotnik and Last, 1988). Augmenting steroidogenesis could thus increase the amount of energy and nutrients available for somatic repair and the maintenance and elicitation of immune responses. (Muehlenbein and Bribiescas, 2005; Shelton and Verhulst, 1996; Wedekind and Folstad, 1994). Peripherial testosterone levels may vary as a function of altered production and secretion, conversion to other hormones, uptake into tissues, and altered metabolic clearance. Estrogens, thyroid hormones, insulin, growth hormone, and/or new hormones binding globally can all alter testosterone levels. How we provide evidence that FSH, LH and inhibin B levels are not significantly altered in this sample of men infected with *Plasmodium vivax* reveals much about the route of testosterone variation as a function of disease risk in a stochastic environment. Assuming testosterone’s immunosuppressory actions are primarily suppressive (for review see Muehlenbein and Bribiescas, 2005), depressed testosterone levels during illness or injury could function to prevent immunosuppression by otherwise higher testosterone levels (Zlotnik and Last, 1988). Augmenting steroidogenesis could thus increase the amount of energy and nutrients available for somatic repair and the maintenance and elicitation of immune responses. (Muehlenbein and Bribiescas, 2005; Shelton and Verhulst, 1996; Wedekind and Folstad, 1994). Peripherial testosterone levels may vary as a function of altered production and secretion, conversion to other hormones, uptake into tissues, and altered metabolic clearance. Estrogens, thyroid hormones, insulin, growth hormone, and/or new hormones binding globally can all alter testosterone levels. How we provide evidence that FSH, LH and inhibin B levels are not significantly altered in this sample of men infected with *Plasmodium vivax* reveals much about the route of testosterone variation as a function of disease risk in a stochastic environment. 

**Literature cited**

For further information