GDV1 induces sexual commitment of malaria parasites by antagonizing HP1-dependent gene silencing

ID 4482093
Author(s) Filarsky, M.; Fraschka, S. A.; Niederwieser, I.; Brancucci, N. M. B.; Carrington, E.; Carrió, E.; Moes, S.; Jenoe, P.; Bartfai, R.; Voss, Till S.
Author(s) at UniBasel Filarsky, Michael ; Niederwieser, Igor ; Brancucci, Nicolas ; Carrington, Eilidh ; Carrio Gaspar, Elvira ; Voss, Till ;
Year 2018
Title GDV1 induces sexual commitment of malaria parasites by antagonizing HP1-dependent gene silencing
Journal Science
Volume 359
Number 6381
Pages / **Article-Number** 1259-1263
Mesh terms Animals; Chromosomal Proteins, Non-Histone, metabolism; Gametogenesis, genetics; Gene Silencing; Malaria, Falciparum, parasitology; Plasmodium falciparum, growth & development; Sex Differentiation, genetics
Malaria is caused by Plasmodium parasites that proliferate in the bloodstream. During each replication cycle, some parasites differentiate into gametocytes, the only forms able to infect the mosquito vector and transmit malaria. Sexual commitment is triggered by activation of AP2-G, the master transcriptional regulator of gametocytogenesis. Heterochromatin protein 1 (HP1)-dependent silencing of ap2-g prevents sexual conversion in proliferating parasites. In this study, we identified Plasmodium falciparum gametocyte development 1 (GDV1) as an upstream activator of sexual commitment. We found that GDV1 targeted heterochromatin and triggered HP1 eviction, thus derepressing ap2-g. Expression of GDV1 was responsive to environmental triggers of sexual conversion and controlled via a gdv1 antisense RNA. Hence, GDV1 appears to act as an effector protein that induces sexual differentiation by antagonizing HP1-dependent gene silencing.
Publisher American Association for the Advancement of Science
ISSN/ISBN 0036-8075
edoc-URL https://edoc.unibas.ch/65295/
Full Text on edoc Available;
Digital Object Identifier DOI 10.1126/science.aan6042
PubMed ID http://www.ncbi.nlm.nih.gov/pubmed/29590075
ISI-Number WOS:000427504900041
Document type (ISI) Journal Article