



Localization of Fulicin-like Immunoreactivity in the Central Nervous System and Periphery of *Biomphalaria glabrata*, an Intermediate Host for Schistosomiasis

## Journal of Health Disparities Research and Practice

Volume 9  
Issue 5 *Special Issue - NIDDK STEP UP*

Article 90

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2016

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Anthony Hernandez-Vasquez

MW Miller, PhD , *University of Puerto Rico*

S Rolon-Martinez, BS , *University of Puerto Rico*

*See next page for additional authors*

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### Recommended Citation

Hernandez-Vasquez, Anthony; Miller, PhD, MW; Rolon-Martinez, BS, S; Vaasjo, BS, LO; and Rodriguez, MB (2016) "Localization of Fulicin-like Immunoreactivity in the Central Nervous System and Periphery of *Biomphalaria glabrata*, an Intermediate Host for Schistosomiasis," *Journal of Health Disparities Research and Practice*: Vol. 9: Iss. 5, Article 90.

Available at: <https://digitalscholarship.unlv.edu/jhdrp/vol9/iss5/90>

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## Abstract

An estimate of about ten percent of the population worldwide live at risk of contracting the parasitic disease schistosomiasis, or “snail fever”. The digenetic trematode worm species *Schistosoma mansoni* that is responsible for causing the most common form of intestinal schistosomiasis requires the freshwater snail *Biomphalaria glabrata* to serve as its primary intermediate host, where it multiplies and develops into its cercarial form that is infectious to humans. Parasitic castration and parasitic gigantism are among some of the profound behavioral changes that the infection of *B. glabrata* by *S. mansoni* is known to cause. For this reason, a neural transcriptomics approach was undertaken to determine precursor prohormones that could encode neuropeptides implicated in *Biomphalaria* reproductive and feeding behaviors. A transcript (1616 nucleotides) was found to encode a putative precursor polypeptide (316 aminoacids) that could give way to the neuropeptide fulicin (Phe–D-Asn-Glu-Phe-Val-NH<sub>2</sub>; Ohta et al. 1991; Yasuma Kamatani et al. 1995) and five additional related peptides. For this investigation, affinity purified polyclonal antibodies (rabbit) were developed against the anticipated fulicin neuropeptide. Fulicin-like immunoreactivity was observed throughout the central nervous system (CNS) with distinct neurons and clusters on the ventral and dorsal surfaces, as well as in peripheral tissues. Fulicin-like cells of both large and small diameter were present on the dorsal and ventral surfaces of the buccal ganglion. In addition dispersed clusters of small diameter cells were observed in the cerebral and pedal ganglia. However, in the right pleural ganglion no fulicin-like neurons were present, although it was rich in immunoreactive fibers. Within the left parietal and visceral ganglia, clusters of large prominent cells appeared to give rise to axons projecting to the anal and intestinal nerves. Additionally, peripheral tissue of *B. glabrata*, specifically regions of the mantle, lip and tentacle were rich in fulicin-like immunoreactive fibers and cell bodies. These results suggest that fulicin and other peptides derived from the fulicin precursor could regulate behaviors related to food intake, reproduction, and growth that are altered during the course of infection in this host-parasite system.

## Keywords

*Biomphalaria Glabrata*; Schistosomiasis; Snail Fever

## Authors

Anthony Hernandez-Vasquez; MW Miller, PhD; S Rolon-Martinez, BS; LO Vaasjo, BS; and MB Rodriguez



**Journal of Health Disparities Research and Practice**  
**Volume 9, Special Edition 1, Summer 2016, pp. 128-129**  
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School of Community Health Sciences  
University of Nevada, Las Vegas

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Anthony Hernandez-Vasquez  
M.W. Miller, University of Puerto Rico  
S. Rolon-Martinez, University of Puerto Rico  
L.O. Vaasjo, University of Puerto Rico  
M.B. Rodriguez, University of Puerto Rico  
**Coordinating Center:** University of Nevada Las Vegas

### **ABSTRACT**

An estimate of about ten percent of the population worldwide live at risk of contracting the parasitic disease schistosomiasis, or “snail fever”. The digenetic trematode worm species *Schistosoma mansoni* that is responsible for causing the most common form of intestinal schistosomiasis requires the freshwater snail *Biomphalaria glabrata* to serve as its primary intermediate host, where it multiplies and develops into its cercarial form that is infectious to humans. Parasitic castration and parasitic gigantism are among some of the profound behavioral changes that the infection of *B. glabrata* by *S. mansoni* is known to cause. For this reason, a neural transcriptomics approach was undertaken to determine precursor prohormones that could encode neuropeptides implicated in *Biomphalaria* reproductive and feeding behaviors. A transcript (1616 nucleotides) was found to encode a putative precursor polypeptide (316 aminoacids) that could give way to the neuropeptide fulicin (Phe–D-Asn–Glu–Phe–Val–NH<sub>2</sub>; Ohta et al. 1991; Yasuma Kamatani et al. 1995) and five additional related peptides. For this investigation, affinity purified polyclonal antibodies (rabbit) were developed against the anticipated fulicin neuropeptide. Fulicin-like immunoreactivity was observed throughout the central nervous system (CNS) with distinct neurons and clusters on the ventral and dorsal surfaces, as well as in peripheral tissues. Fulicin-like cells of both large and small diameter were present on the dorsal and ventral surfaces of the buccal ganglion. In addition dispersed clusters of small diameter cells were observed in the cerebral and pedal ganglia. However, in the right pleural ganglion no fulicin-like neurons were present, although it was rich in immunoreactive fibers. Within the left parietal and visceral ganglia, clusters of large prominent cells appeared to give rise to axons projecting to the anal and intestinal nerves. Additionally, peripheral tissue of *B. glabrata*, specifically regions of the mantle, lip and tentacle were rich in fulicin-like immunoreactive fibers and cell bodies. These results suggest that fulicin and other peptides

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derived from the fulicin precursor could regulate behaviors related to food intake, reproduction, and growth that are altered during the course of infection in this host-parasite system.

**Keywords:** Biomphalaria Glabrata, Schistosomiasis, Snail Fever

#### **ACKNOWLEDGEMENTS**

The STEP-UP HS program is supported by the National Institute of Diabetes and Digestive and Kidney Diseases of the National Institutes of Health, Grant number: 1R25DK098067-01.