The Fat Body of the Hematophagous Insect, *Panstrongylus megistus* (Hemiptera: Reduviidae): Histological Features and Participation of the β-Chain of ATP Synthase in the Lipophorin-Mediated Lipid Transfer

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Abstract

In insects, lipid transfer to the tissues is mediated by lipophorin, the major circulating lipoprotein, mainly through a nonendocytic pathway involving docking receptors. Currently, the role of such receptors in lipid metabolism remains poorly understood. In this work, we performed a histological characterization of the fat body of the Chagas’ disease vector, *Panstrongylus megistus* (Burmeister), subjected to different nutritional conditions. In addition, we addressed the role of the β-chain of ATP synthase (β-ATPase) in the process of lipid transfer from lipophorin to the fat body. Fifth-instar nymphs in either fasting or fed condition were employed in the assays. Histological examination revealed that the fat body was composed by diverse trophocyte phenotypes. In the fasting condition, the cells were smaller and presented a homogeneous cytoplasmic content. The fat body of fed insects increased in size mainly due to the enlargement of lipid stores. In this condition, trophocytes contained abundant lipid droplets, and the rough endoplasmic reticulum was highly developed and mitochondria appeared elongated. Immunofluorescence assays showed that the β-ATPase, a putative lipophorin receptor, was located on the surface of fat body cells colocalizing partially with lipophorin, which suggests their interaction. No changes in β-ATPase expression were found in fasting and fed insects. Blocking the lipophorin–β-ATPase interaction impaired the lipophorin-mediated lipid transfer to the fat body. The results showed that the nutritional status of the insect influenced the morphohistological features of the tissue. Besides, these findings suggest that β-ATPase functions as a lipophorin docking receptor in the fat body.

Key words: fat body, lipophorin, lipid metabolism, Triatominae, docking receptor

Chagas’ disease or American trypanosomiasis is caused by the protozoan parasite, *Trypanosoma cruzi* (Chagas) (Kinetoplastida: Trypanosomatidae). This neglected tropical disease affects 6–7 million people globally, although most of infected populations are located in Latin America (WHO 2019). The transmission of *T. cruzi* in endemic areas relies on the presence of hematophagous insects belonging to the subfamily Triatominae, commonly known as kissing bugs (Schofield et al. 2006). *Panstrongylus megistus* (Burmeister), the main vector in Brazil after the control of *Triatoma infestans* (Klug) (Hemiptera: Reduviidae) populations (Coura 2015), has been employed as an experimental model to study the metabolism of lipids and lipoproteins mainly in the midgut and ovaries (Canavoso et al. 2004; Fruttero et al. 2009, 2011, 2014, 2017a). However, knowledge about the processes involved in lipid transfer to the fat body is still limited in this species.

In insects, the fat body plays an essential role in the dynamics of energy storage and mobilization. In addition, most of the biosynthetic activity and the intermediate metabolism take place there (Arrese and Soulages 2010). The fat body is distributed throughout the body of the insect, filling the spaces between the remaining organs. The adipocytes or trophocytes, characterized by abundant lipid droplets (also termed ‘adiposomes’), comprise the major cell type of the tissue (Roma et al. 2010).
Lipophorin is the main circulating insect lipoprotein (Van der Horst et al. 2009). Although its composition depends on the nutritional status, the developmental stage, and the insect species, the main lipid class transported is diacylglycerol (Soulagés and Wells 1994). The central physiological role of lipophorin is the transport and transfer of its lipid cargo to and from the sites of absorption, storage, and utilization, mostly by a docking, nonendocytic pathway (Atella et al. 2005, Van der Horst et al. 2009). During this transfer process, the lipoprotein interacts with the plasma membrane of the target cells (Canavoso et al. 2001, Van der Horst et al. 2009). In this context, we have already demonstrated that the beta subunit of the F$_{1}$F$_{0}$ adenosine triphosphate synthase complex ($\beta$-ATPase), located on the plasma membrane of the cells, acts as a docking receptor of lipophorin in the midgut and in the ovaries of P. megistus (Fruttero et al. 2014, 2017a). The ATP synthase complex is located in the inner mitochondrial membrane, where it synthesizes ATP at the expense of the transmembrane electrochemical proton potential difference (Walker 1998). However, in addition to its mitochondrial subcellular location, several reports showed that the $\beta$-ATPase, probably forming part of the ATPase complex, is also present in the plasma membrane of mammalian (Chi and Pizzo 2006) and insect cells (Fruttero et al. 2017b). The ‘ectopic’ distribution of $\beta$-ATPase allows its functioning as a receptor of several ligands as well as its participation in different physiological and pathological processes. They include the binding of the high-density lipoprotein in the reverse cholesterol transport (Vantourout et al. 2010), the binding of juvenile hormone binding protein in insect endocrine regulation (Zalewska et al. 2009), and the binding of dengue-2 virus during mosquito cell infection (Paingankar et al. 2010).

In the context of insect physiology, we propose that in P. megistus, the stimulus of bloodmeal leads to morphological changes in the fat body that will be reflected at the histological and ultrastructural levels. It is also hypothesized that the $\beta$-ATPase, located on the surface of fat body cells, functions as a lipophorin docking receptor facilitating the transfer of lipids between the main lipoprotein and the cells.

Materials and Methods

Insects and Ethic Statement

Insects were obtained from a colony of P. megistus maintained under standardized conditions of light, temperature, and humidity as described (Canavoso and Rubiolo 1995). The colony was fed on hen blood according to the recommendations of the National Institute of Parasitology (Health Ministry, Argentina; Nuñez and Segura 1987). Animal housing and manipulation followed the current protocols of the Centro de Investigaciones en Bioquímica Clínica e Inmunología (CIBICI-CONICET-Universidad Nacional de Córdoba) animal facility (Res. 1392–2016, UNC), in accordance with the guidelines published by the Canadian Council on Animal Care with the assurance number A5802-01 delivered by the Office of Laboratory Animal Welfare (National Institutes of Health).

Experimental approaches were carried out employing fifth-instar nymphs in fasting condition (7 d post-ecdysis) and fed condition (7 d after a bloodmeal).

Tissue Processing for Cryostat Sectioning

Abdominal fat bodies were obtained by careful dissection of insects placed dorsal side up. The digestive tracts were totally removed to achieve an easier and efficient collection of the fat body. The tissues were rinsed, fixed in 4% formaldehyde in phosphate-buffered saline (PBS; 6.6 mM Na$_{2}$HPO$_{4}$/KH$_{2}$PO$_{4}$, 150 mM NaCl, pH 7.4) for 60 min at room temperature, and subsequently cryoprotected in PBS solutions with increasing concentrations of sucrose at 4°C (Fruttero et al. 2009). Twelve-micrometer sections obtained with a Leica CM1510 cryostat (Leica Microsystems, Wetzlar, Germany) were placed onto slides pretreated with poly-L-lysine (Sigma–Aldrich, St. Louis, MO).

Cryostat tissue sections were stained with Oil Red O to visualize lipid droplets (Leyria et al. 2014).

Immunofluorescence Assays

To analyze the histological distribution of $\beta$-ATPase and lipophorin, cryosections were sequentially incubated with an anti-$\beta$-ATPase antibody (dilution 1:100, rabbit anti-ATP5B/$\beta$-chain of ATP synthase of human origin, sc-33618, Santa Cruz Biotechnology, Inc., Santa Cruz, CA) followed by a goat anti-rabbit IgG labeled with Alexa 568 antibody (dilution 1:400, Molecular Probes, Eugene, OR) and a polyclonal anti-lipophorin-FITC antibody (dilution 1:40) obtained in our laboratory (Fruttero et al. 2017a). Slides were mounted with Fluorsave (Calbiochem, Darmstadt, Germany) and observed with a Leica DMI 8 fluorescence microscope (Leica Microsystems, Wetzlar, Germany) or an Olympus FV300 laser scanning confocal microscope (Olympus, Tokyo, Japan), as corresponding.

Transmission Electron Microscopy

Fat bodies were dissected out and fixed in Karnovsky mixture containing 4% formaldehyde and 2% glutaraldehyde in 0.1 M cacodylate buffer for 2 h. Then, the fixed tissues were washed and treated with 1% OsO$_4$ for 1 h. After dehydration with a series of cold-graded acetone solutions, the tissues were embedded in Embed 812 resin. Following a 48-h polymerization at 60°C, semithin sections of 200 nm were obtained using a JEOL microtome equipped with a diamond blade and were stained with toluidine blue for high-resolution light microscopy analysis. For transmission electron microscopy (TEM), 90-nm ultrathin sections (JEOL JUM-7 ultramicrotome) were placed on grids which were later post-stained with uranyl acetate/lead citrate, examined using a Zeiss Leo 906-E electron microscope (Oberkochen, Germany), and photographed with a Megaview III camera (Olympus, Center Valley, PA). The size of the lipid droplets was determined by measuring their diameter in a minimum of 25 trophocytes from ultrathin sections of each experimental condition.

$\beta$-ATPase Expression Analysis

Transcriptional levels of $\beta$-ATPase were determined by qPCR using RNA extracted from the tissues with TRIzol reagent (Thermo Fisher Scientific, Waltham, MA) and a cDNA synthesized by the MMLV reverse transcriptase protocol (Promega, Heidelberg, Germany; Leyria et al. 2015). qPCR assays were carried out employing specific primers for $\beta$-ATPase (forward: 5′-CGGGCGAGTGAATGAAACC-3′, reverse: 5′-CAGTGGCCAAAGTTGGCTG-3′) and for the normalization 18S ribosomal RNA (18S rRNA; Fruttero et al. 2014, Leyria et al. 2015).

To analyze the expression of $\beta$-ATPase by western blot, fat body homogenates were subjected to subcellular fractionation as described (Fruttero et al. 2014, 2017a). The subcellular fractions were probed against the anti-$\beta$-ATPase antibody (dilution 1:400) followed by the immunodetection with a Li-Cor IRDye 800CW polyclonal goat anti-rabbit IgG secondary antibody (dilution of 1:15,000; Li-Cor Biosciences, Lincoln, NE; Fruttero et al. 2017a). After washing, blots were rinsed, fixed in 4% formaldehyde in phosphate-buffered saline (PBS; 6.6 mM Na$_{2}$HPO$_{4}$/KH$_{2}$PO$_{4}$, 150 mM NaCl, pH 7.4) for 60 min at room temperature, and subsequently cryoprotected in PBS solutions with increasing concentrations of sucrose at 4°C (Fruttero et al. 2009). Twelve-micrometer sections obtained with a Leica CM1510 cryostat (Leica Microsystems, Wetzlar, Germany) were placed onto slides pretreated with poly-L-lysine (Sigma–Aldrich, St. Louis, MO).
were scanned and analyzed with the Odyssey quantitative western blot near-infrared system (Li-Cor Biosciences) using default settings.

In Vivo Injection Experiments
For these assays, insects were immobilized and placed ventral side up under a standard stereo microscope. The injections were carried out with a Hamilton syringe at the coxa-trochanter joint of a hind leg.

To follow the fate of the injected anti-β-ATPase antibody, fed insects were inoculated into the hemocoel with 5 µl (10 µg) of this antibody, and 1 h later, the fat bodies were dissected out and processed for cryosectioning. Tissue sections were then incubated with the anti-rabbit IgG antibody conjugated to Alexa 568 (dilution 1:400), mounted, and processed for fluorescence microscopy (Fruttero et al. 2014, 2017a).

With the aim of following the fate of the lipophorin particle after blocking the β-ATPase-lipophorin interaction, the insects were injected with either 5 µl (10 µg) of an irrelevant anti-BSA-antibody (controls) or 5 µl (10 µg) of the anti-β-ATPase antibody. After 1 h, the insects were injected with lipophorin conjugated with Dil (Lp-Dil, 5 µl per insect, Sigma–Aldrich, St. Louis, MO), a nonexchangeable fluorophore. One hour later, the fat bodies were dissected out and processed for confocal laser microscopy as reported previously (Fruttero et al. 2017a).

Similarly, blocking assays were conducted using lipophorin conjugated with Bodipy-FA (Lp-Bodipy-FA, 4,4-difluoro-5,7-dimethyl-4-bora-3a,4a-diaza-s-indacene-3-hexadecanoic acid, Bodipy FL C16; Molecular Probes, Eugene, OR), an exchangeable fatty acid analog that allowed to follow the fate of lipids transferred from the lipophorin to the fat body. With the purpose of estimating the amount of fluorescent lipids transferred from lipophorin to the fat bodies, total lipids were extracted from each experimental group according to Folch et al. (1957). The fluorescence was measured by fluorometry with 485–505/528–548 nm excitation/emission filters (Multi-Mode Microplate Reader Sinergy HT, BioTek Instruments, Winooski, VT).

Statistical Analysis
The results from qPCR and fluorescence quantification are presented as the mean ± SEM from four to five independent experiments, as stated in each figure legend. Graphs and statistical tests were performed using GraphPad Prism 6.0 and GraphPad Instat 3.0. (GraphPad Software, San Diego, CA). Student’s t-test was used for comparisons and a P < 0.05 was considered statistically significant.

Results
The main histological features of the fat body assessed in semithin sections are displayed in Fig. 1. Our results showed that the fat body is organized in lobes of different thickness; the cells displayed rounded to irregular shape, vacuolated cytoplasm, and pleomorphic nuclei. When comparing nymphs from different nutritional status, in the fat body from those under fed condition, the cells were larger with rounded euchromatic nuclei, numerous vacuoles, and stained granules (Fig. 1). Instead, few and sparse granules were seen in the cytoplasm of cells from fasted samples. In the fed condition, occasional intensively stained cells were found (indicated with an asterisk in Fig. 1). On the other hand, the distribution of lipid droplets was visualized with Oil Red O staining (Fig. 1). These organelles showed variable sizes and were evenly distributed in the cell cytoplasm. In the fed condition, cell size was remarkably increased and the cytoplasm contained numerous lipid droplets, which is compatible with the high rate of lipid storage in the tissue after feeding.

The analysis of ultrathin sections by TEM for the unfed condition (Fig. 2) depicted several vesicles and/or inclusions (Figs. 2A and B) as well as fewer and generally less electron-dense lipid droplets of an average size of 3.25 ± 0.15 µm (Fig. 2A and D). The difference in electron density between lipid droplets observed in unfed and fed conditions (Figs. 2A and D and 3A–D, respectively) can be attributed to the degree of saturation of fatty acids which compose the lipid esters present in the core of the organelle (Wigglesworth 1975, Cheng et al. 2009). The trophocytes of unfed nymphs showed mostly rounded mitochondria with tubular, less defined cristae, although occasional elongated mitochondria were also present. The large nuclei observed showed peripheral regions of condensed chromatin (Fig. 2C–F). On the contrary, trophocytes from fed nymphs displayed more numerous and electron-dense lipid droplets (Fig. 3A–D), which showed an average size of 4.04 ± 0.16 µm. It was also observed that cells contained more developed rough endoplasmic reticulum, abundant elongated mitochondria with well-defined cristae, and high electron-dense matrix (Fig. 3E and F).

To elucidate the putative role of β-ATPase in lipid transfer, we examined its expression at transcriptional level in fat bodies of P. megistus from insects in fasting and fed conditions. When analyzed by qPCR, transcriptional levels of β-ATPase seemed to be slightly upregulated in the fed condition even though no significant differences were observed upon comparison with samples from the fasting condition (Fig. 4, left panel). To evaluate the expression of β-ATPase by western blot, fat body homogenates were subjected to subcellular fractionation. We focused on the β-ATPase located in the subcellular fraction containing the plasma membranes of the cells because such localization allowed both, β-ATPase-lipophorin interaction (Fig. 2B) as well as fewer and generally less electron-dense lipid droplets of an average size of 3.25 ± 0.15 µm (Fig. 2A and D). The difference in electron density between lipid droplets observed in unfed and fed conditions (Figs. 2A and D and 3A–D, respectively) can be attributed to the degree of saturation of fatty acids which compose the lipid esters present in the core of the organelle (Wigglesworth 1975, Cheng et al. 2009). The trophocytes of unfed nymphs showed mostly rounded mitochondria with tubular, less defined cristae, although occasional elongated mitochondria were also present. The large nuclei observed showed peripheral regions of condensed chromatin (Fig. 2C–F). On the contrary, trophocytes from fed nymphs displayed more numerous and electron-dense lipid droplets (Fig. 3A–D), which showed an average size of 4.04 ± 0.16 µm. It was also observed that cells contained more developed rough endoplasmic reticulum, abundant elongated mitochondria with well-defined cristae, and high electron-dense matrix (Fig. 3E and F).

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interaction and its participation on the lipid transfer, as proposed previously (Fruttero et al. 2017b). Our results showed that in fasting and feeding conditions, β-ATPase was expressed at similar levels in total homogenates, mitochondrial and microsomal fractions (Fig. 4, right panel).

Taking into account that in P. megistus, the expression of β-ATPase in the membrane fractions of fat bodies from fed and fasted insects was similar and considering that the store of lipids in the fat body is maximal after blood feeding, the following experiments were conducted with fifth-instar fed nymphs. First, we obtained evidence of the partial colocalization between lipophorin and β-ATPase in the plasma membrane of the fat body cells (Fig. 5), suggesting their interaction. On the other hand, immunofluorescence assays demonstrated that the injected anti-β-ATPase antibody was localized mainly in the plasma membrane of the fat body cells (Fig. 6A). Finally, blocking assays directed to prevent the interaction between lipophorin and β-ATPase in the fat body were carried out to explore the participation of β-ATPase in lipophorin-mediated lipid transfer. As shown in Fig. 6B, the faint fluorescent signal in the tissue indicated that lipophorin binding to the fat body was impaired when fifth-instar nymphs were treated with an anti-β-ATPase antibody before Lp-DiI injection. Similar experiments were performed by injecting Lp-Bodipy-FA to follow the fate of the lipids transported by lipophorin to the fat body. The results showed that the fluorescent signal in the lipid droplets of the tissue from treated insects substantially decreased in comparison to that from controls (Fig. 6C). Semiquantitative analyses of the fluorescent lipids transferred to fat bodies showed that blocking β-ATPase significantly impaired the transfer of Bodipy-FA from lipophorin to the tissue (Fig. 6D).

**Discussion**

In hematophagous insects, the bloodmeal triggers the secretion of neuropeptides and hormones to the circulation that, in turn, activate lipid metabolism in peripheral tissues, including the fat body (Gondim et al. 2018, Li et al. 2019). This organ accumulates lipids mostly in the form of triacylglycerol, and these reserves will help the insect to withstand fasting periods (Arrese and Soulages 2010).

In our laboratory, P. megistus has been employed as a hematophagous insect model in studies that were focused on the relevance of lipid and lipophorin metabolism in energy supply during starvation and reproduction (Canavoso et al. 2003, 2004; Fruttero et al. 2009, 2011). Feeding experiments in fifth-instar nymphs of this species with radiolabeled lipids demonstrated that triacylglycerol was hydrolyzed in the midgut lumen to glycerol and fatty acids, and that the levels of radiolabeled triacylglycerol in the fat body increased as the tissue accumulated lipids (Canavoso et al. 2004). Thus, the triacylglycerol lipid content peaked a few days after feeding and then diminished, corresponding to the transition from lipid accumulation...
to mobilization in response to fasting (Canavoso et al. 2004). In spite of the current knowledge about lipid metabolism in the fat body, the events involved in lipophorin–fat body interaction have not been completely elucidated. In addition, cellular characterization of this tissue in *P. megistus* is still missing.

In the present work, the trophocytes displayed variable phenotypes, sharing histological features with the fat body cells reported in other insect species (Dean et al. 1985, Zara and Caetano 2004, Roma et al. 2010, Martins et al. 2011). As expected, the bloodmeal lead to remarkable changes in the tissue which showed larger trophocytes with abundant lipid droplets, in agreement with the physiological role of the tissue and with its higher triacylglycerol content (Canavoso et al. 2004, Pontes et al. 2008). The differences between the two nutritional conditions were clearly visualized by TEM. Thus, the fat body of unfed insects displayed fewer and less electron-dense lipid droplets as well as round mitochondria. On the contrary, fed insects presented more elongated mitochondria and a well-developed rough endoplasmic reticulum. This ultrastructural organization was similar to what was described by Wigglesworth (1933, 1967, 1982) in *Rhodnius prolixus* (Stal) (Hemiptera: Reduviidae). Also in line with the available literature in triatomines (Juárez and Fernandez 2007, Martins and Ramalho-Ortigão 2012), the fat body of *P. megistus* did not present associated oenocytes.

Several membrane proteins that interact with lipophorin and can function as docking receptors have been reported (Fruttero et al. 2017b). Among them, the transmembrane proteins of the CD36 family Cameo2 and SCRB15 are involved in the selective transfer of carotenoids from lipophorin to the salivary glands in *Bombyx mori* (Linnaeus) (Lepidoptera: Bombyciidae) (Tsuchida and Sakudoh 2015). Moreover, a Lipophorin Receptor (LpR) belonging to the Low-Density Lipoprotein Receptor family was described for the first time in the fat body of *Locusta migratoria* (Linneaus) (Orthoptera: Acrididae), participating in lipophorin endocytosis and resecretion (Dantuma et al. 1997). However, the inhibition of endocytosis caused no effect on diacylglycerol transfer to the tissue, suggesting thus a minor role for LpR in the lipophorin-mediated transfer of

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**Fig. 4.** Expression of the beta chain of ATP synthase (β-ATPase) in the fat body of *P. megistus* fifth-instar nymphs. Left panel: Fat bodies from insects at 7 d post-ecdysis (fasting condition, PE) and at 7 d after a bloodmeal (fed condition, PBM) were dissected out, their RNA extracted and used in qPCR assays employing specific primers. Results are expressed as relative expression by 1,000 copies of 18S ribosomal RNA ± SEM. ns, no statistically significant differences (*P* = 0.095, *t* = 1.978, df = 6, *n* = 4, unpaired *t*-test). Right panel: Homogenates of fat bodies from insects at fasting and fed conditions were subjected to subcellular fractionation as described in the text. The resulting fractions were probed with the anti-β-ATPase antibody and visualized by western blot. Fifty micrograms was loaded in each lane and a homogenate of rat brain was employed as positive control. Similar results were obtained from three separate experiments.

**Fig. 5.** Localization of lipophorin (Lp; green) and β-ATPase (red) in the fat body by immunofluorescence. Merge: image showing the partial colocalization of Lp and β-ATPase (arrowheads). The insets show the corresponding DIC images. H, hemolymph. Similar results were obtained from three separate experiments. Bars: 10 µm.
neutral lipids to the fat body (Dantuma et al. 1997). In Drosophila melanogaster (Meigen) (Diptera: Drosophilidae), although the knockdown of LpR did not affect the lipid content of the fat body and hemolymph, it impaired the uptake of neutral lipids in the oocytes and imaginal discs, suggesting an organ-specific role (Parra-Peralbo and Culi 2011). In the present study, it was observed that nutritional condition influenced the histological features of the tissue, but it did not change the expression of β-ATPase in fed and fasted insects. The ATP synthase complex is composed by the hydrophilic $F_1$ portion and the hydrophobic $F_0$ portion, which is embedded in the mitochondrial membrane. Each portion is formed by different subunits, being the α- and β-subunits part of the catalytic $F_1$ portion (Meier et al. 2011). At present, there are reports demonstrating that the β-ATPase in the plasma membrane not only serves as receptor of several ligands but also participates in different cellular and metabolic processes (Zalewska et al. 2009, Paingankar et al. 2010, Vantourout et al. 2010). In P. megistus, the occurrence of β-ATPase in the microsomal fraction strongly suggested its presence in the plasma membranes of fat body cells. This subcellular location, which was also observed in the midgut and the ovarian tissue of P. megistus (Fruttero et al. 2014, 2017a), would allow the interaction of β-ATPase with lipophorin. Moreover, β-ATPase was found partially colocalizing with lipophorin in the cell surface, suggesting their interaction.

In the present investigation, anti-β-ATPase injection assays were carried out to evaluate the role of β-ATPase in the transfer of lipids to the fat body. In these experiments, it was observed that blocking the interaction between lipophorin and β-ATPase led to a diminished binding between lipophorin and the fat body and to an impairment of the fatty acids transferred from lipophorin to the tissue. Taking into account that the antibody blocks the interaction between lipophorin and the tissue, is likely that the transfer of other lipids, beside fatty acids, would also be impaired. Similar findings were reported in P. megistus nymphs, where the injection of the anti-β-ATPase antibody interfered not only with the binding of lipophorin to the midgut, but also with the bidirectional transfer of lipids from lipophorin to the tissue (Fruttero et al. 2014). Taken together, the results suggest that, at least in P. megistus, the function of β-ATPase as lipophorin receptor could be a common feature of the tissues with a high rate of lipid storage and mobilization. In D. melanogaster, it has been proposed that LpR participates in the process of lipid transfer to oocytes by a nonendocytic pathway, being its function to stabilize lipophorin in the extracellular domain of the plasma membrane of the oocyte, which in turn would allow the lipolysis and the transfer of lipids. This model also involved the participation of lipases and transfer proteins (Parra-Peralbo and Culi 2011, Rodríguez-Vázquez et al. 2015). Although it is possible that β-ATPase could function increasing the local concentration of lipophorin and facilitating lipid exchange, further experiments are needed to fully understand this issue. Once lipophorin reaches the plasma membrane, the bidirectional transfer of lipids to and from the tissue can occur either by passive diffusion or with the involvement of specific membrane transporters. In this regard, the fatty acid transport proteins, the previously mentioned members of the CD36 family Cameo2 and SCR-B15, as well as other still uncharacterized transfer candidates might account for the process (Arrese et al. 2001, Tsuchida and Sakudoh 2015, Gondim et al. 2018).

In conclusion, the findings presented here provide fruitful information on a poorly known aspect of lipid metabolism, which in turn impacts on the survival and reproduction of this hematophagous species with relevance in the epidemiology of Chagas’ disease.
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