Available online at www.sciencedirect.com

ScienceDirect

Current Opinion in Microbiology

Membrane trafficking and remodeling at the host–parasite interface

Julien Santi-Rocca and Nicolas Blanchard

Membrane shape is functionally linked with many cellular processes. The limiting membrane of vacuoles containing *Toxoplasma gondii* and *Plasmodium* apicomplexan parasites lies at the host–parasite interface. This membrane comprises intra-vacuolar and extra-vacuolar tubulo-vesicular deformations, which influence host–parasite cross-talk. Here, underscoring specificities and similarities between the *T. gondii* and *Plasmodium* contexts, we present recent findings about vacuolar membrane remodeling and its potential roles in parasite fitness and immune recognition. We review in particular the implication of tubulo-vesicular structures in trapping and/or transporting host and parasite components. Understanding how membrane remodeling influences host–pathogen interactions is expected to be critical in the battle against many intracellular pathogens beyond parasites.

Address
Centre de Physiopathologie de Toulouse Purpan (CPTP), INSERM, CNRS, Université de Toulouse, UPS, Toulouse, France

Corresponding author: Blanchard, Nicolas (nicolas.blanchard@inserm.fr)

Current Opinion in Microbiology 2017, **40**:145–151

This review comes from a themed issue on **Host-microbe interactions: parasites**

Edited by **Maryse Lebrun** and **Nicolas Blanchard**

https://doi.org/10.1016/j.mib.2017.11.013

1369-5274/© 2017 Elsevier Ltd. All rights reserved.

Introduction

Plasmodium spp. and *Toxoplasma gondii* (*T. gondii*) are unicellular organisms from the Apicomplexa phylum that are responsible for two major human diseases, namely malaria and toxoplasmosis. During their obligate intracellular cycle in mammalian hosts, *Plasmodium* and *T. gondii* dwell within a parasitophorous vacuole (PV). The PV limiting membrane (PVM) represents a physical boundary between the host cell cytoplasm and the PV lumen. Hence the PVM serves as a 'gate' for host–pathogen exchanges, which may either benefit the host (e.g. immune recognition) or the parasite (e.g. nutrient acquisition and/or transport of modulatory effectors). Rather than being stiff and smooth, the PVM is dynamically shaped into inward-extending and outward-extending membranous tubules and vesicles.

The first description of intra-vacuolar tubulo-vesicular structures in *T. gondii* PV, at the time coined 'stereocilia', dates back half a century [1•]. The first outward extensions of the PVM into the host cell cytoplasm were reported in the early 1990s in *T. gondii*-infected cells [2] and in *Plasmodium*-parasitized Red Blood Cells (pRBC) [3]. Several studies have confirmed and complemented those pioneer findings and varying terminology has been used to designate these structures (Table 1). As an attempt to introduce a unified and more inclusive nomenclature, we will respectively use iTVN and eTVN to designate the intra-vacuolar and extra-vacuolar membranous Tubulo-Vesicular Networks that are directly contiguous to the PVM. The prefix Tg- or the prefix P- distinguish structures formed by *T. gondii* or *Plasmodium* respectively (Table 1).

By underscoring relevant parallels and distinctions between *T. gondii* and *Plasmodium*, we review recent advances on the morphology, the composition, the biogenesis and the functions of these membrane structures. We first cover the iTVNs and then present the eTVN structures (Figure 1).

The *T. gondii* intra-vacuolar Tubulo-Vesicular Network (Tg-iTVN)

Morphology

Several studies using electron microscopy (EM) [1•,4,5] or helium ion microscopy [6] have revealed, firstly, that some tubules of the Tg-iTVN are attached to the PVM and to the parasite's plasma membrane and secondly, that at least two types of intertwined and connected structures co-exist, namely tubules of ~30 nm-diameter and thinner filaments with an uneven thickness of 2–10 nm [6]. The former are hollow tubes that are delimited by membranes and are in continuity with the PVM [4], suggesting a topological identity between the tubule lumen and the host cell cytosol. The latter structures are still poorly understood. A third category of tubular structures wrapping around host microtubules pushing on and invaginating the PVM, has been reported in one study [7].

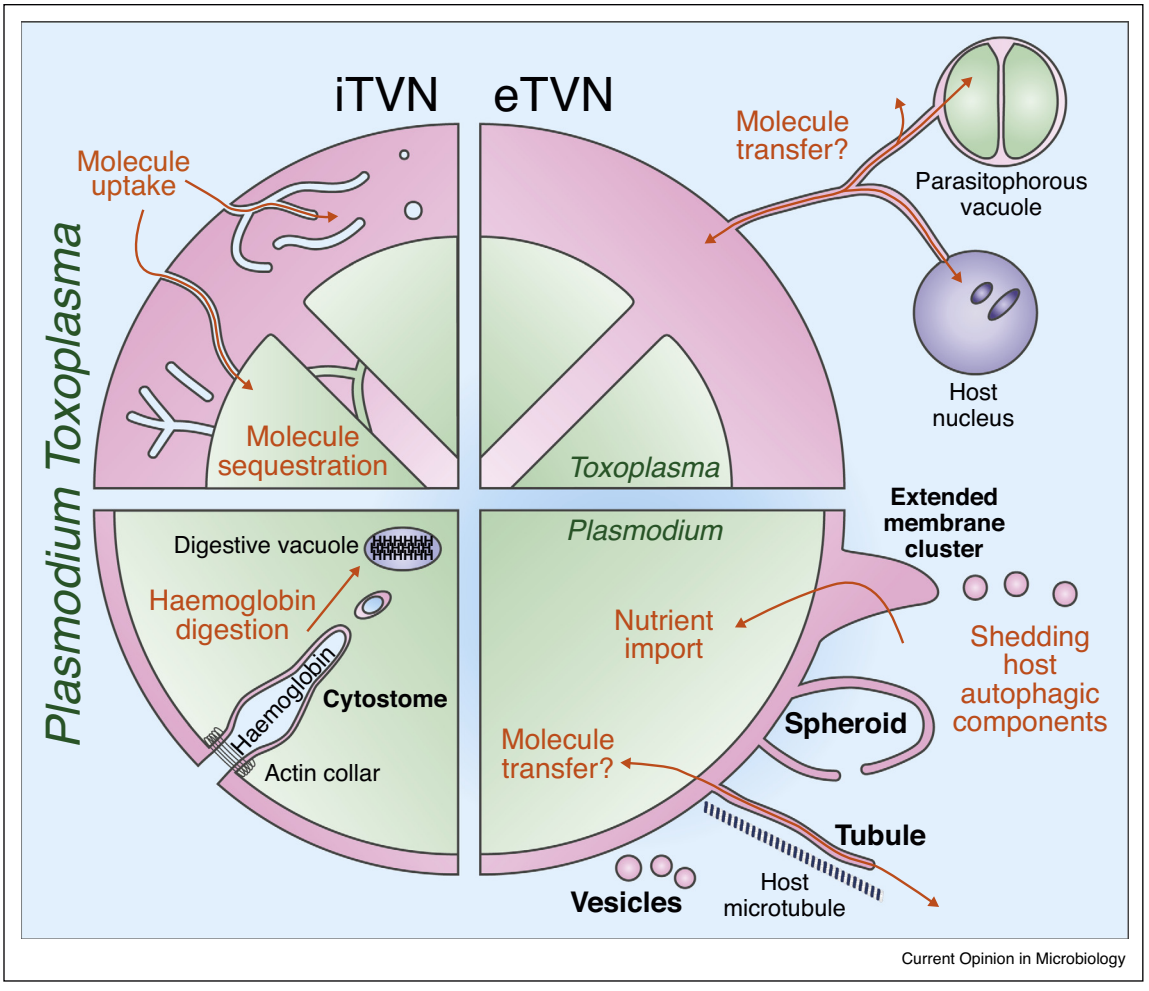
Currently however, a precise description of the 3D-morphology of the Tg-iTVN is missing. Such an approach would help address if the Tg-iTVN is one reticulated compartment with full continuity with the host cytosol or a juxtaposition of discrete networks.

Table 1

Historical names and acronyms of membrane tubulo-vesicular networks (TVN) of *T. gondii* and *Plasmodium*

Nomenclature used in this review	Organism	Name	Acronym	Host cell	Ref. or PMID
Tg-iTVN	*T. gondii*	Stereocilia	n/a	Kidney	[1•]
		Intra-Phagosomal Membrane	IPM	Macrophage	3528173
		Tubulo-Vesicular Network	TVN	Fibroblast	[4]
		Intra-Vacuolar Network	IVN	Fibroblast	9664038
		Membrane Nanotubular Network	MNN	Fibroblast	18061598
Tg-eTVN	*T. gondii*	PV-extending strands or extensions	n/a	Fibroblast	[2]
P-iTVN	*Plasmodium*	Micropyle	n/a	Hepatocyte	[27]
		Cytostome	n/a	RBC	5914696
P-eTVN	*Plasmodium*	Tubo or Tubulo-Vesicular Membrane Network	TVM or TVN	RBC	[3]
		Liver Stage TuboVesicular Network	LS-TVN	Hepatocyte	[35]

Figure 1



Schematics of iTVN and eTVN deformations at the PVM interface between host cells and *Toxoplasma gondii* or *Plasmodium*. Intra-vacuolar and extra-vacuolar tubulo-vesicular networks (iTVN and eTVN, left and right columns, respectively) in *Toxoplasma gondii* (top row) and *Plasmodium* spp. (bottom row). Parasites (green) reside within the PV lumen (pink). The names of characteristic membrane extensions are in bold. The established or putative roles for the TVNs are shown in orange, with a question mark when hypothetic.

Download English Version:

https://daneshyari.com/en/article/8745127

Download Persian Version:

https://daneshyari.com/article/8745127

Daneshyari.com