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Analysis molecular mimicry between protozoa Giardia intestinalis and autoantigens implicated in autoimmune reactive arthritis: potential cross-reactivity

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Abstract

Introduction: *Giardia intestinalis (G. intestinalis)* is an intestinal parasite that infected between 200 and 300 million people worldwide. Some patients developed reactive arthritis after infection clearance. Molecular mimicry could induce cross reactivity to generate autoimmune disease.

Objective: To evaluate the potential molecular mimicry between autoantigens implicated in in reactive arthritis and *G. intestinalis* antigens using *in silico* analysis.

Methodology: Similarity between autoantigens and protozoa antigens was analyzed using PSI-BLAST. After, shared regions were identified performing binary alignments. Using 3D structures retrieved from protein data bank or modelled B cell epitopes were predicted with Ellipro tool. Epitopes predicted were visualized on 3D structure with Pymol software.

Results: In total, six of thirty-nine autoantigens report for reactive arthritis-associated showed similarity with proteome reported for G. intestinalis. autoantigens were included in the study and compared against the *G. intestinalis* proteome. These autoantigens shared up 30% with homologues from protozoa.

We have that the autoantigens that showed the greatest identity were Alpha enolase with Enolase and HSP 60 Chaperonin 60, Putative TCP-1/cpn60 chaperonin family protein, 60 kDa of groel chaperonin. Sixty-eight epitopes with scores greater than 0.7 were identified from *G. intestinalis* antigens.

Conclusion: Six *G. intestinalis* antigens are identified that mimic reactive arthritis autoantigens, with moderate levels of identity that could explain a cross-reactivity involved in the development of the autoimmune response reported in patients who suffered from the infection.

1. Introduction

G. intestinalis is a protozoan parasite with a worldwide distribution. Its infection develops in the human small intestine, with over 200 million infected individuals reported around the world (1). It is more prevalent in developing countries with limited access to clean water and inadequate sanitation facilities. In Colombia, poor hygienic conditions facilitate its transmission, with an estimated prevalence of 12% in the general population and 28% among children aged 1 to 4 years (2). Giardiasis is a self-limiting disease; however, it is associated with the development of gastroenteritis and, in some cases, complications at the digestive system level. Evidence suggests an association between giardiasis and reactive arthritis (REA) or joint pain in up to 51% of cases, with symptoms lasting approximately six months. Some infected individuals exhibited arthritis symptoms, in some cases consistent with REA (3) (4). In addition to *G. intestinalis*, other pathogens such as *Clostridium difficile*, *Mycoplasma pneumoniae*, *Leptospira spp*., and *Babesia spp*. have also been associated with this disease (5)(6)(8).

Evidence such as the study of the immune response against the bacterial protein aquaporin-Z (AqpZ) could trigger an autoimmune reaction against human aquaporin-4 (Aqp4), a key protein in the function of astrocytes in the central nervous system. This is given that there are regions of structural similarity

between AqpZ and Aqp4 where antibodies can occur with cross-reactivity. Likewise, there is a perspective that certain bacterial infections could trigger autoimmunity in neuromyelitys optica through this mechanism (9).

The pathogenesis of RA is not well characterized. However, the presence of the HLA-B27 allele has been reported as a genetic factor that increases susceptibility to reactive arthritis, being found in up to 80% of affected individuals, though its precise role remains unclear (10, 11). Additionally, HLA-B27 contributes to the chronicity of REA by promoting cross-reactivity between microbial and host antigens (12).

Although there is evidence of the relationship between HLA and the development of autoimmune diseases, other immunopathological mechanisms that may be involved remain unknown (13, 14). Some studies suggest that pathogens associated with mucosal lymphoid tissues can be transported to anatomical sites such as the joints, where they trigger immune responses leading to chronic inflammatory processes. This, in turn, may cause lymphocytes to recognize antigens within these anatomical regions (15, 16).

Among the human autoantigen involved as autoantigens in REA are heat shock proteins, peptidyl arginine deiminase 4 (PAD4), protein kinase Cβ1 (PKCβ1), phosphatidylinositol 4-phosphate 5-kinase type II γ (PIP4K2C), and the catalytic domain B1 homolog of the murine sarcoma viral oncogene v-raf (BRAF) (17, 18). Since infections play a crucial role in the development of autoimmune responses, this study aims to identify molecular mimicry and its potential implication in cross-reactivity between *G. intestinalis* and REA autoantigens using bioinformatics approaches to analyze molecular mimicry.

2. MATERIALS AND METHODS

2.1 Antigen search and comparison

A comprehensive literature review was performed to identify antigens related to reactive arthritis. Also, autoantigens reported in AAgAtlas (https://pubmed.ncbi.nlm.nih.gov/32162267/) were retrieved to get aminoacid sequences of each one using Uniprot database. Each amino acid sequences was used as input in PSI-BLAST to identify similarities with *G. intestinalis* proteome. Antigens with similarity > 30% were used for further analyses. A binary alignment between autoantigen and its homologous was done in order to determine conserved regions, for this, IBIVU Praline (https://pubmed.ncbi.nlm.nih.gov/24170407/) tool was used.

2.2 Homology-Based Modeling

Tridimensional models were used to identify exposed and conserved surface residues to located predicted B cell epitopes. Antigens with experimentally resolved 3D structures were retrieved from the Protein Data Bank (https://pubmed.ncbi.nlm.nih.gov/10592235/). 3D structures no reported were modelled based on homology with Swiss model server (https://pubmed.ncbi.nlm.nih.gov/29788355/) and Alphafold (https://pubmed.ncbi.nlm.nih.gov/34757056/) The predicted proteins were refined using

Deep View to minimize energy. Their quality was evaluated using multiple tools, including Ramachandran plots, WHATIF, QMEAN4 index, and energy values (GROMOS96 force field). All models were visualized using PyMOL 2.3 (https://pubmed.ncbi.nlm.nih.gov/33999189/).

2.3Homology structural analysis with protein superposition was performed using the Pymol 2.3 software (https://pymol.org/), to calculate RMSD (Root Mean Square Deviation) values between ReA autoantigens and *G. intestinalis* selected antigens.

2.4Epitope Prediction

Model were used to B-cell epitope prediction with the ElliPro server (http://tools.iedb.org/ellipro/). Parameters were set up as default.. Epitopes with a score equal or higher than 0.7 were selected to explore molecular mimicry.

3. RESULTS

3.1 Sequence Alignment

A total of thirty-nine autoantigens associated with REA were found to share similarity with proteins reported in *G. intestinalis* proteome. Seven antigens from parasite shared identity values greater than 30%. The highest percentage of identity found was for alpha-enolase with enolase, at 51.50% **(Table 1).**

The *G. intestinalis* antigens homologous to HSP60 were conserved, as shown in Fig. 1. Amino acid residues corresponding to potential linear and conformational epitopes, likely recognized by the adaptive immune system, were identified in **Fig. 2.**

On the other hand, autoantigens such as the platelet-derived growth factor beta receptor and the serine interaction receptor and their homologs did not show conserved regions or allow for the identification of antigenic patches.

FIGURE 1. Structure-based alignments of human proteins and G. intestinalis antigens with their Praline identity values.

FIGURE 2: Antigenic patches of linear and conformational epitopes identified in the alignment between reactive arthritis autoantigens and G. intestinalis antigens.

LINEAR

CONFORMATIONAL

3.2 Prediction of Linear and Conformational Epitopes

Linear epitope prediction was performed using ElliPro. A total of 68 epitopes with scores above 0.7 were identified from *G. intestinalis* antigens. These epitopes were highlighted with representative colors on the 3D protein structures **(Table 2 and Fig. 3).**

For the serine/threonine-protein kinase, two epitopes were identified (LE1: 281–376, LE2: 1–15); for the mitogen-activated protein kinase, four epitopes were found (LE1: 1–20, LE2: 233–281, LE3: 82–88, LE4: 30–40); for a kinase protein, five epitopes (LE1: 329–374, LE2: 1–38, LE3: 638–709, LE4: 388–417, LE5: 430–448); for enolase, five epitopes (LE1: 264–295, LE2: 52–88, LE3: 1–17, LE4: 195–217, LE5: 237–252); for chaperonin 60, seven epitopes (LE1: 1–20, LE2: 527–539, LE3: 432–453, LE4: 298–323, LE5: 339–374, LE6: 106–122, LE7: 225–248); for the putative TCP-1 protein, three epitopes (LE1: 1–20, LE2: 101–149, LE3: 300–321); and for the 60 kDa GroEL homolog, two epitopes (LE1: 134–152, LE2: 42–56). Of these several coincidences with conserved regions that can be observed in the binary alignments boxed in **Fig. 2** along with their exposure score given by Ellipro, for Enolase of G. intestinalis it could be seen that of the predicted linear epitopes an epitope with a score of 0.761 coincides with the conserved regions at position 52–54 and 63–65, in turn the Hsp60 with Chaperonin 60 coincided conserved regions with the linear epitope with a score of 0.779 at position 112–117, that of 0.726 at position 226–230, another of 0.801 450–453 and an epitope of 0.814 at position 534–537, finally the Hsp60 with the putative Tcp-1 at position 112–117.

The epitopes derived from chaperone proteins and enolase were found to be conserved when compared to their human homologs, as shown in **Fig. 3.** This finding could help explain the extraintestinal manifestations secondary to *G. intestinalis* infection. Conformational epitope prediction was also performed with ElliPro for proteins where conserved regions and antigenic patches could be recovered, identifying a total of seven conformational epitopes. These were visualized on the 3D structures using PyMOL **(Table 3 and Fig. 3).** *G. intestinalis* enolase presented one conformational epitope (CE1: 2–136); the putative TCP-1 protein presented two (CE1: 5–137, CE2: 39–153); and the chaperonin protein presented four conformational epitopes (CE1: 351–382, CE2: 4–539, CE3: 219–323, CE4: 39–55). Likewise, in **Fig. 2**, these conformational epitopes that, in proportion to their size, covered conserved regions of the binary alignments with greater identity were marked in boxes.

Table 4 outlines the sequences, residues, and scores of linear epitopes derived from autoantigens associated with reactive arthritis, while **Table 5** corresponds to the conformational epitopes identified in enolase, the putative TCP-1 protein, and chaperonin of *G. intestinalis*. These linear epitopes represent specific protein segments recognized by the immune system and may contribute to the autoimmune responses observed in REA. Antigenic regions within these autoantigens are highlighted in **Figs. 3 and 4**, indicating segments recognized as foreign by the immune system. These antigenic regions may include linear epitopes or regions involved in molecular mimicry with parasitic antigens. Blue, purple, green, and orange colors represent epitopes with the highest scores as described in **Tables 2, 3, 4, and 5.**

3.3 Homology Modeling

Using SWISS-MODEL, we generated an overview of the autoantigens and their corresponding homologous template proteins, including Ramachandran plot statistics and sequence identities. **Table 6** highlights human Bruton's tyrosine kinase (BTK), alpha-enolase, and HSP60 as having Ramachandran values above 95%, reflecting the predominant secondary structure configurations of their homologous templates. This structural insight supports a better understanding of their potential immunogenicity.

3.4 Protein Superposition

Using the PyMOL tool, 3D structural superimpositions were performed between human proteins associated with reactive arthritis (REA) and their *G. intestinalis* homologs. The serine interaction receptor showed a root-mean-square deviation (RMSD) value of 7.22. The platelet-derived growth factor beta receptor exhibited RMSD values ranging from 6.024 to 9.606. HSP60 showed RMSD values of 32.066 with the chaperonin protein, 69.563 with the putative TCP-1 protein, and 1.168 with the 60 kDa GroEL chaperonin. These values, being distant or relatively distant from zero, suggest low structural homology.

In contrast, alpha-enolase demonstrated an RMSD value of 0.554, which is notably close to zero and indicates a high degree of structural similarity, as illustrated in **Fig. 5.**

FIGURE 3. Linear and conformational antigenic regions of G. intestinalis proteins.

FIGURE 4. Linear and conformational antigenic regions of autoantigens involved in reactive arthritis.

The epitopes highlighted in different colors correspond to the scores obtained from the ElliPro epitope prediction platform. Colors such as purple-blue represent the highest scoring epitopes across all autoantigen structures, followed by green for the second-highest scores and orange for the third, in a hierarchical order from highest to lowest. This ranking is based on the values presented in **Tables 2, 3, 4, and 5**: purple-blue, green, orange, red, purple, yellow, cyan, hot pink, lime green, and olive green.

FIGURE 5. Structure-based alignments of human proteins and G. intestinalis antigens with their RMSD value per Pymol

4. DISCUSSION

Reactive arthritis has a low global prevalence and primarily affects young adults between 20 and 40 years of age, with an incidence of up to 30 cases per 100.000 individuals. This inflammatory joint disease typically develops 2 to 4 weeks after a gastrointestinal or genitourinary infection, without the detection of pathogens in synovial samples. Due to these characteristics, the World Health Organization (WHO) and the Arthritis and Rheumatism Research Council have classified ReA as a category III condition (6, 7).

G. intestinalis, It has been recently identified in patients diagnosed with ReA. In ReA, autoantibodies against PAD4, PKCβ1, PIP4K2C and BRAF are generated (19, 20). We here report for the first time molecular mimicry between antigens involved in ReA and homologous proteins in intestinal parasites such as *G. intestinalis*. Remarkably, conserved antigenic regions were identified in proteins such as HSP60, alpha-enolase, and kinases. Previous reports of molecular mimicry between *Trypanosoma* proteins and human autoantigens related to systemic lupus erythematosus (SLE), 260 SLE-associated autoantigens were analyzed, of which 93 candidates with molecular mimicry potential were selected.

Ultimately, only 36 of those 93 autoantigens—each with 30% identity with *Trypanosoma* antigens—were used for epitope prediction (21).

The analysis employed bioinformatic tools such as PSI-BLAST to identify sequence similarities, homology modeling for antigens lacking reported three-dimensional structures, and epitope prediction via the IEDB database and the ElliPro server. A total of 139 shared epitopes were identified between Trypanosoma and the selected autoantigens, some with prediction scores above 0.9; notably, one autoantigen showing 62% identity was an enolase—implicated in cross-reactivity due to molecular mimicry as a possible trigger of autoimmune diseases following *Trypanosoma* infections. Similarly, other studies linking Leishmania to potential SLE autoimmunity, using the same methodology, provided preliminary evidence that *Leishmania sp.* could induce an autoimmune response in susceptible patients via the same mechanism involving human antigens.

These findings may help to elucidate the relationship between parasitic infections and autoimmune diseases, thereby improving diagnostic methods and therapeutic approaches (21, 22).

Of the 39 human autoantigens analyzed, seven *G. intestinalis* homologs exhibited sequence identities exceeding 30%, including serine/threonine kinases, mitogen-activated protein kinases, enolase, chaperonin 60, the putative TCP-1/cpn60 protein, and the GroEL chaperonin. These levels of identity, coupled with structural conservation revealed through RMSD analysis and epitope mapping, provide strong computational evidence supporting potential cross-reactive immune responses. Significantly, alpha enpolase displayed an identity value of 51% with G. intestinalis enolase and yielded a particularly low RMSD value (0.554), indicating a high degree of structural similarity and reinforcing its candidacy as a molecular mimic.

In line with our results, a study by Téllez et al. (23). found that alpha-enolase was among the *G. intestinalis* antigens recognized by secretory antibodies in breastfeeding women from an endemic population (23). In that study, secretory immune responses to *G. intestinalis* were evaluated in the breast milk and saliva of Nicaraguan lactating women to determine antibody levels against this protozoan and the antigens recognized.

Milk and saliva samples from 100 lactating women in León, Nicaragua, were analyzed. Indirect immunofluorescence (IIF) and one- and two-dimensional Western blot assays were performed in vitro. Immunoreactive proteins were identified using recombinant antigens and antigenic pattern analysis. It was found that 59% of breast milk samples and 52% of saliva samples contained antibodies against *G. intestinalis*. These antibodies reacted with membrane antigens, flagellar components, and cytoplasmic proteins of the parasite. Up to 16 immunoreactive proteins ranging from 20 to 165 kDa were identified. Subsequent analysis confirmed reactivity against variant surface proteins (VSPs) and highlighted α-enolase as a prominent antigen in your results. It was also demonstrated that *Giardia* specifically secretes ADI, OCT, and α-enolase upon interacting with human intestinal cells (23).

Another study characterized an enolase, Plaa6, from the recently identified allergenic source of Platanus acerifolia (London plane) pollen (24). Although only four allergens had previously been recognized in this pollen, researchers detected an IgE-binding protein band in crude pollen extracts, leading to its identification as an enolase. Pla a 6 was purified via chromatographic strategies, its gene sequence determined, and it was expressed recombinantly in *E. coli*. Its allergenic potential was assessed by ELISA, Western blot, and basophil activation tests (BAT). The protein showed strong IgE-binding activity in Western blot assays; in ELISA analyses, 45.95% of allergic patients exhibited specific IgE against rPla a 6. IgE binding to pollen extract could be inhibited by up to 45.77% using recombinant Pla a 6, confirming its immunological relevance. It also activated patients' basophils, reaffirming its role as a significant allergen (24). These data further support the notion of immune recognition of *G. intestinalis* antigens, which may contribute to autoimmune responses.

Moreover, antibodies against *G. intestinalis* antigens specific to the trophozoite stage—characterized as variant (VSP) and invariant proteins—have been described as potential causative agents of ReA (25, 26). This further supports the idea that *G. intestinalis* antigens are immunologically recognized and could contribute to autoimmune responses. Additionally, it has been reported that *G. intestinalis* antigens specific to the trophozoite stage—comprising variant-specific and invariant proteins—could be potential triggers of ReA (25, 26).

The evaluation of heat shock proteins (HSPs) it was done by their high association with autoimmune diseases and cancer. HSP27, HSP60, HSP70, and HSP90 have all been linked to rheumatoid arthritis, acting as autoantigens, with structural alterations in these proteins potentially promoting inflammatory processes that lead to autoimmune conditions (27). In our study, and in parallel in silico work by other researchers, significant similarities were found between α-enolase and human γ-enolase and two *Helicobacter pylori* proteins (phosphopyruvate hydratase), with 48% identity, suggesting high sequence conservation. Epitope predictión of these proteins revealed the most relevant α-enolase epitope spanning residues 49–106 with 45.6% identity to the H. pylori protein, and for γ-enolase, residues 47–105 with 45.76% identity. These findings led the authors to propose that molecular mimicry between H. pylori and human proteins may play a role in the development of type 1 gastric neuroendocrine tumors (28).

Subsequent comparisons revealed significant similarity between human HSP60 and the *H. pylori* GroEL chaperonin—proteins implicated in the genesis of post-infection reactive arthritis by *G. intestinalis*, as found in our study—showing 54% identity and 92% coverage. Potential HSP60 epitopes capable of eliciting cross-reactive immune responses were also analyzed: eight linear epitopes and three discontinuous epitopes were identified in the human protein, with highly conserved regions compared to the *H. pylori* GroEL. Displaying remarkable conservation in the sequence alignment. HSP60 is a key protein in immune regulation and cellular homeostasis; its involvement in type 1 gastric neuroendocrine tumor pathogenesis may likewise be driven by the same autoimmune mechanism (28).

In silico identification of antigenic regions has been employed in other studies to identify novel immunogenic patches and molecular mimics between human and pathogenic antigens implicated in autoimmune diseases (21, 22). Such approaches are useful in understanding the etiology of autoimmune disorders. However, they must be complemented by experimental studies to clarify host-pathogen interactions and to precisely define antigenic determinants and their functional implications.

5. CONCLUSION

Among the 39 autoantigens associated with reactive arthritis, *G. intestinalis*-derived enolase showed the highest sequence identity, followed by heat shock proteins and kinase proteins. The epitopes identified in this study are located in linear and structural regions shared between human alpha-enolase and *G. intestinalis* enolase, as well as in HSP60. These findings support the hypothesis of molecular mimicry as a potential mechanism for cross-reactivity in ReA.

6. RECOMMENDATIONS

In line with our research and its development using in silico tools, and given that the results obtained suggest a possible immunopathological association between *G. intestinalis* and reactive arthritis via a molecular mimicry mechanism, it is recommended to pursue this line of investigation through in vitro and in vivo studies that allow experimental validation of the proposed hypothesis.

As a suggestion, it is considered necessary to perform structural and functional studies of the immunogenic epitopes identified computationally, using techniques such as ELISA and epitope recognition assays with sera from patients sensitized by giardiasis. Additionally, the use of animal models immunized with mimetic peptides will enable evaluation of cross-reactivity induction and the onset of joint symptoms.

These steps will help elevate the level of evidence from the exploratory stage to experimental phases, which is essential to support the development of immunotherapeutic strategies—such as vaccines tailored for endemic populations affected by giardiasis and at risk of post-infectious reactive arthritis complications.

Furthermore, researchers are encouraged to form interdisciplinary teams—integrating biologists, clinicians, and other specialists—to take the appropriate actions.

Abbreviations

Rea (Reactive arthritis), *G. intestinalis* (*Giardia intestinalis*), AqpZ (Aquaporin-Z), Aqp4 (Aquaporin-4), HSP (heat shock proteins), PAD4 (peptidyl arginine deiminase 4), PKCβ1 (protein kinase Cβ1), PIP4K2C (phosphatidylinositol 4-phosphate 5-kinase type II γ), BRAF (catalytic domain B1 homolog of the murine sarcoma viral oncogene v-raf), BTK (Bruton's tyrosine kinase), LE (linear epitope), CE (conformational epitope), RMSD (Root Mean Square Deviation)

Declarations

1. **Conflicts of interest**

No conflicts of interest declared

2. **Declaration of generative AI in scientific writing**

Generative AI (ChatGPT, developed by OpenAI) was used to assist with the following tasks: -Refinement of language and grammar in the manuscript to ensure clarity and adherence to British English conventions. Assisting with summarization, formatting, and improving the readability of the content while maintaining the integrity of the scientific findings and data. The authors confirm that all scientific content, data interpretation, and conclusions were independently determined and verified by the authors. Generative AI was utilized strictly as tool to streamline the writing process and ensure professional presentation.

3. **Source of financing**

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Tables

Tables are available in the Supplementary Files section.

Table 6 is not available with this version.

Figures

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Supplementary Files

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