Ortiz et al., J Bacteriol Parasitol 2015, 6:4
http://dx.doi.org/10.4172/2155-9597.S1.014

OMICS International
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International Conference on
Parasitology
August 24-26, 2015 Philadelphia, USA

The T687G SNP in a P- glycoprotein gene of *F. hepatica* is not detected in different strains susceptible and resistant to triclabendazole

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Fasciolosis is a parasitic disease caused by the trematode *Fasciola hepatica*. Its control is mainly based on the use of triclabendazole (TCBZ), a halogenated benzimidazole with excellent efficacy against juvenile and adult stages. Resistance to TCBZ in *F. hepatica* has been reported in many parts of the world. Benzimidazole resistance in nematodes is mainly caused by specific amino acid substitutions in β-tubulin resulting from a nucleotide substitution in the gene. However, the resistance to TCBZ in *F. hepatica* is not associated with changes in this molecule. The increased cellular efflux of TCBZ in *F. hepatica* is related to ABC transporters as the P-glycoproteins (PGP). In this protein single nucleotides polymorphisms (SNPs) were identified, such as T687G which was associated the phenomenon of resistance. Other SNPs not yet associated with resistance were also identified (A617T and T267G). In the present work, a complete PGP sequence has been assembled based on sequences available in databases. We analyzed transcripts from this gene region in TCBZ resistant strains from Peru and Argentina and in susceptible strains from Mexico and Ireland. We observed that the T687G SNP is not detected in any of the sequences analyzed. The SNPs A617T and T267G, fall in a PGP intron, and are shared by all resistant strains, but also are present in some susceptible ones. RT-PCR from adult RNA showed no evidence of alternative splicing. Our results highlight the lack of clear association of SNPs in this PGP gene with resistance to TCBZ. The recent release of two assemblies of the *F. hepatica* genome revealing the presence of multiple ABC transporters opens new avenues for investigating variants associated with the resistance of *F. hepatica* to TCBZ.

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