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Genetic-metabolic advances in the study of the anthelmintic resistance in *Fasciola hepatica*

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The fasciolosis, a parasitic disease, is caused by the trematode *Fasciola hepatica* (Fluke) and its control is mainly based on the use of the anthelmintic triclabendazole (TCBZ). Resistance to TCBZ in fluke has been reported in many parts of the world and is not associated with changes in the molecule target (tubulin). The increased cellular efflux of TCBZ was related to P-glycoproteins (PGP) and a T687G SNP was initially suggested as responsible for the resistant phenotype. In our lab, a complete PGP sequence has been assembled and analyzed the transcripts from this gene region in TCBZ resistant strains (TCBZ-R) from Peru and Argentina and in susceptible strains (TCBZ-S) from Mexico and Ireland. In this study, the T687G SNP is not associated with resistance to TCBZ. Instead, we identified other SNPs, A617T and T267G, which fall in a PGP intron, 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. These results highlight the lack of clear association of SNPs in this PGP gene with resistance to TCBZ. Our previous studies over the strains TCBZ-R confirmed in the Phase I of detoxification overexpression of flavin-monooxygenases. This phenomenon should not be the only response that the trematode has and should not rule out the involvement of others process of detoxification of Phase I or II. In the TCBZ-R strains, the GST, GPx and GSR activity is higher than that in the TCBZ-S strains whereas CE activity did not differ between the different strains tested. Our work detected a multi-enzymatic response involving at all the family of enzymes GSH dependent. This results revealing the presence of multiple ABC transporters and multidrug resistance genes opens new avenues for investigating variants associated with the resistance of *F. hepatica* to TCBZ.

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