In this article, we discuss an alternative explanation for the fact that the production and plasma levels of IL-17 are diminished in patients chronically infected with Trypanosoma cruzi, suffering from congestive heart failure. This alternate hypothesis considers the inhibitory pharmacological action of digoxin, a drug commonly used to treat heart failure. We believe this is a significant point to be further studied as IL-17 was initially considered to be heart-protective. Therefore, our argument has profound therapeutic implications.

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autoimmune disease. Recently, TH-17 could not be associated with inflammatory but rather with a homeostatic/regulatory signature\(^2^0\). This observation would favor an alternative hypothesis, and one should consider different biological functions for TH-17, depending on the producer cell-type or even the location (organ) these cells are parked. However, Chagas’ disease may include another particular situation since one of the most common drugs used to treat chagasic heart failure is digoxin, a drug reported to be a potent inhibitor of IL-17 production either in mice or humans\(^2^1\). Information about drugs taken by chagasic patients is missed from both clinical studies mentioned above.

Conclusion

In this opinion letter, we have briefly discussed the role of IL-17 in Chagas’ disease, proposing an alternative explanation to the fact that this interleukin was diminished in chagasic patients with established heart disease, especially in patients with congestive heart failure\(^2^0\). We argue that lower levels of IL-17 could be due to the use of digoxin intake by patients rather than a particular immune response that could favor or not the production of IL-17. Therefore, an alternative interpretation of these results may well be considered, and further studies/information is needed to clarify this point.

Conflicts of interest: The authors declare no conflicts of interest.

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