An African Herbal Extract Affects Immune Response in Type 1 Diabetic Mouse Model

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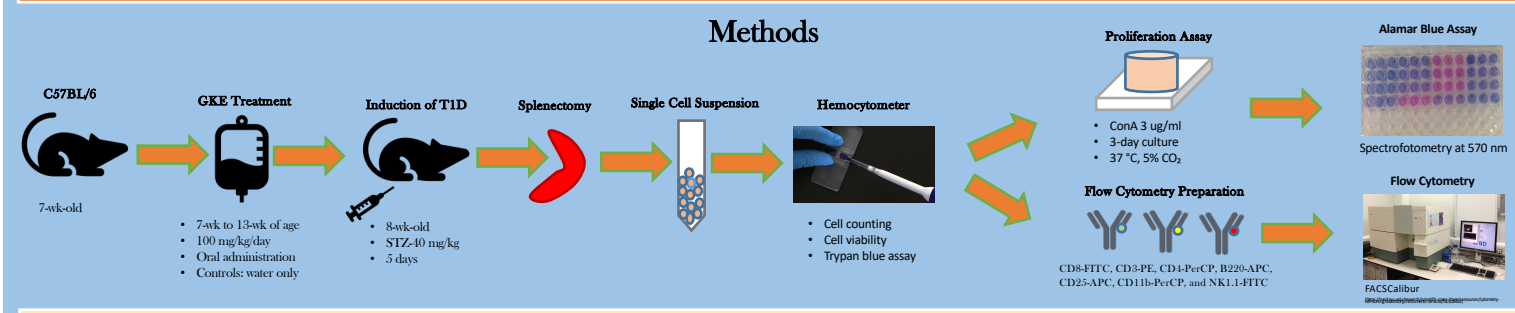
Abstract

Type 1 diabetes (T1D) is an autoimmune disease that results from the attack of auto-reactive lymphocytes (T-cells) on pancreatic beta cells causing hyperglycemia. *Garcinia kola* (GK), an African herb, is believed to have anti-inflammatory abilities. We hypothesized that GK extract (GKE) will reduce the activity of pathogenic T-cells, thus having a potential for prevention of T1D in a T1D mouse model. C57BL/6 mice were treated daily by GKE (100 mg/kg) added to their drinking water from 7 to 13 weeks of age, and diabetes was induced by the administration of streptozotocin. The GKE effects were tested on T-cell composition (by using flow cytometry) and T-cell function (by performing T-cell proliferation assays) in the treated and control mice. At the completion of the experiment, both control and treatment mice were euthanized, single cell suspension was prepared from their spleens, and cell counts and viability obtained. T -cell proliferation was tested by culturing splenocytes with the addition of T-cell-specific mitogen concanavalin A, while identification of T-cells and their respective subpopulations was performed by exposing splenic cells to fluorochrome-labeled antibodies that detect specific T-cell markers, and their quantification was completed by laser-based flow cytometry. Our results showed that GKE treatment inhibited the proliferative capability of T-cells and decreased the percentage of T-cells, as well as their subpopulations. In conclusion, our results show that GKE has a potential to act as an immunosuppressive T-cell agent that might positively affect T1D development.

Objective

To study the effects of the Garcinia kola extract (GKE) treatment on T-cell composition and function in an experimental T1D mouse model.

Methods



Results

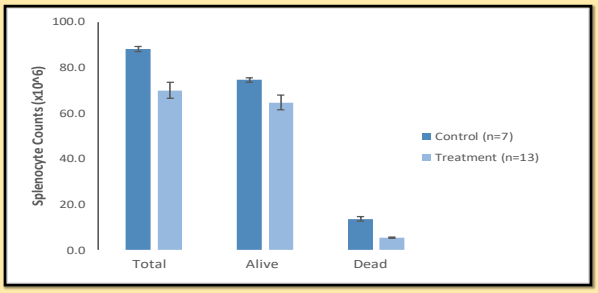


Figure 1. Cell counts (Trypan blue exclusion assay) presented as average ± SEM.

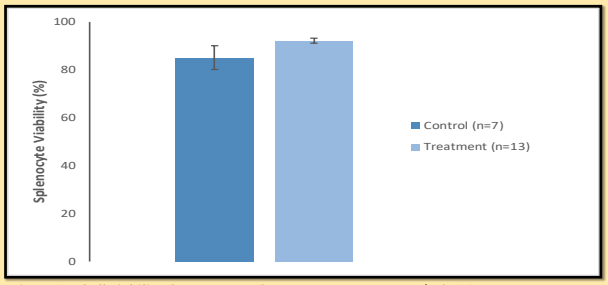


Figure 2. Cell viability data presented as average percentage ± SEM.

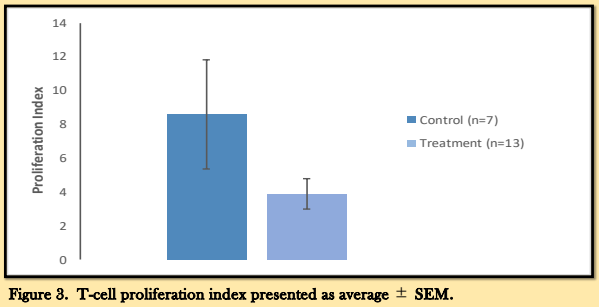


Figure 3. T-cell proliferation index presented as average ± SEM.

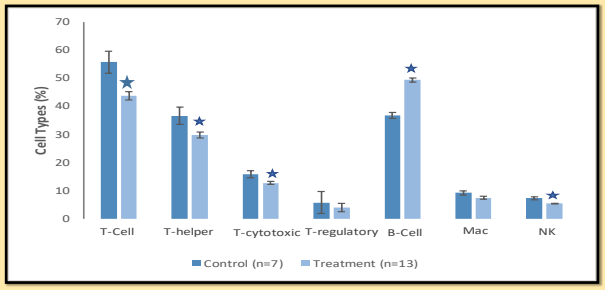


Figure 4. Immunophenotyping of splenocytes; average percentage ± SEM (*p<0.05).

Summary

GKE treatment's effects:

- No significant change in cell counts and viability (Fig. 1 and 2)
- A trend of a decreased proliferation of T-cells (Fig. 3)
- Reduced numbers of T-cells, T-helpers and T-cytotoxic cells (Fig. 4)

Conclusion

GKE treatment suppressed T-cell proliferative capacity and reduced the numbers of T-cells as well as their subpopulations, supporting our initial hypothesis. Our data suggest that GKE treatment might prevent T1D development, however, ongoing experiments in our laboratory did not confirm it.

Acknowledgements

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