

Biomarker Analysis of Mechanically Ventilated Adults using Propofol or Dexmedetomidine

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INTRODUCTION

• Increased inflammatory cytokines and biomarkers contribute toward increased permeability of the blood brain barrier, which has been postulated to contribute to acute brain dysfunction and delirium.

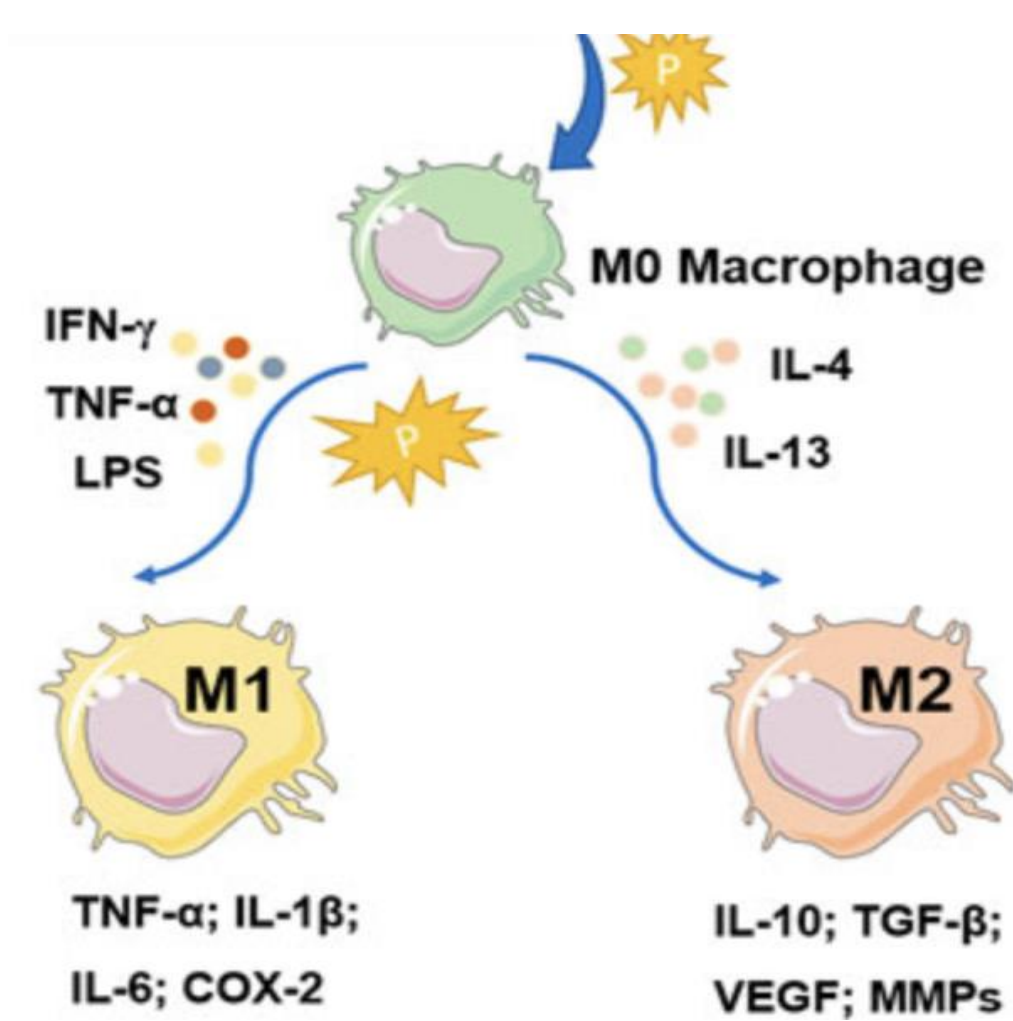
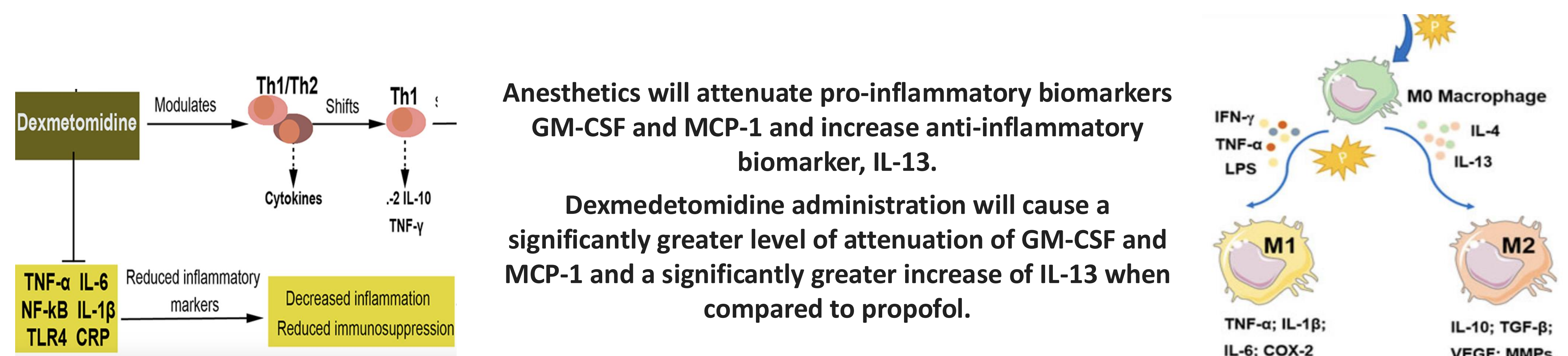
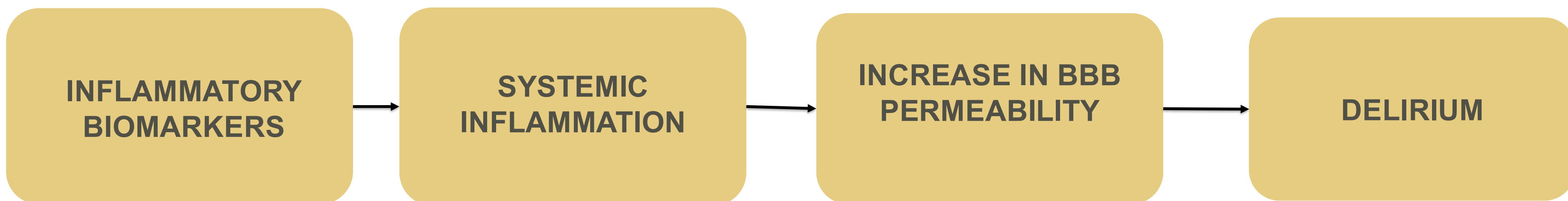
• Granulocyte-macrophage colony stimulating factor (GM-CSF) and monocyte chemoattractant protein 1 (MCP-1) both stimulate macrophages/monocytes, specifically M1 pro-inflammatory macrophages.

• Medications like propofol and dexmedetomidine have been shown to mitigate inflammation by downregulating the polarization of M1 macrophage and upregulating M2 macrophages.

- M2 macrophages produce anti-inflammatory cytokines (i.e., IL-13) which attenuate inflammation and may have a protective effect on the brain.

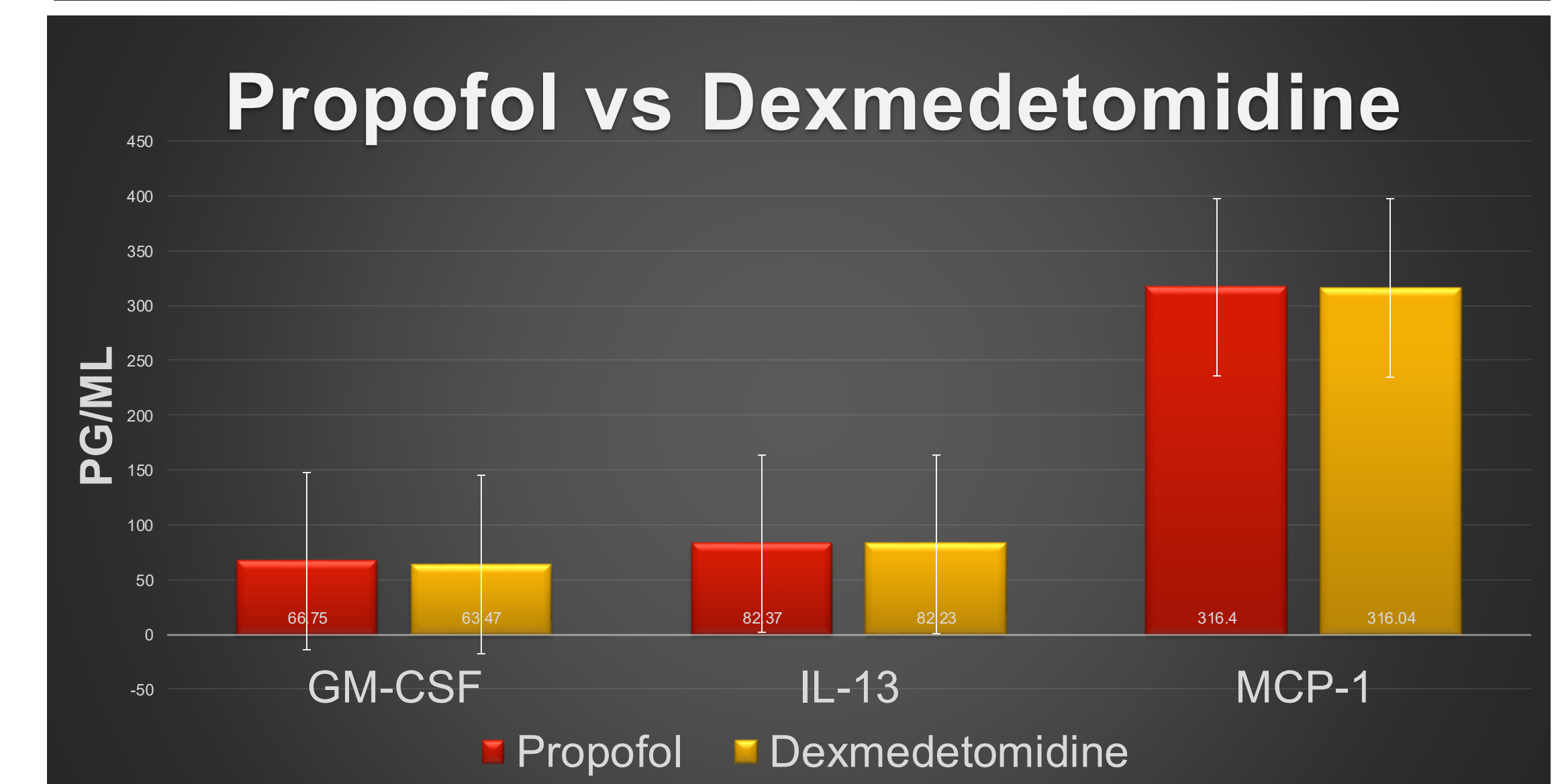
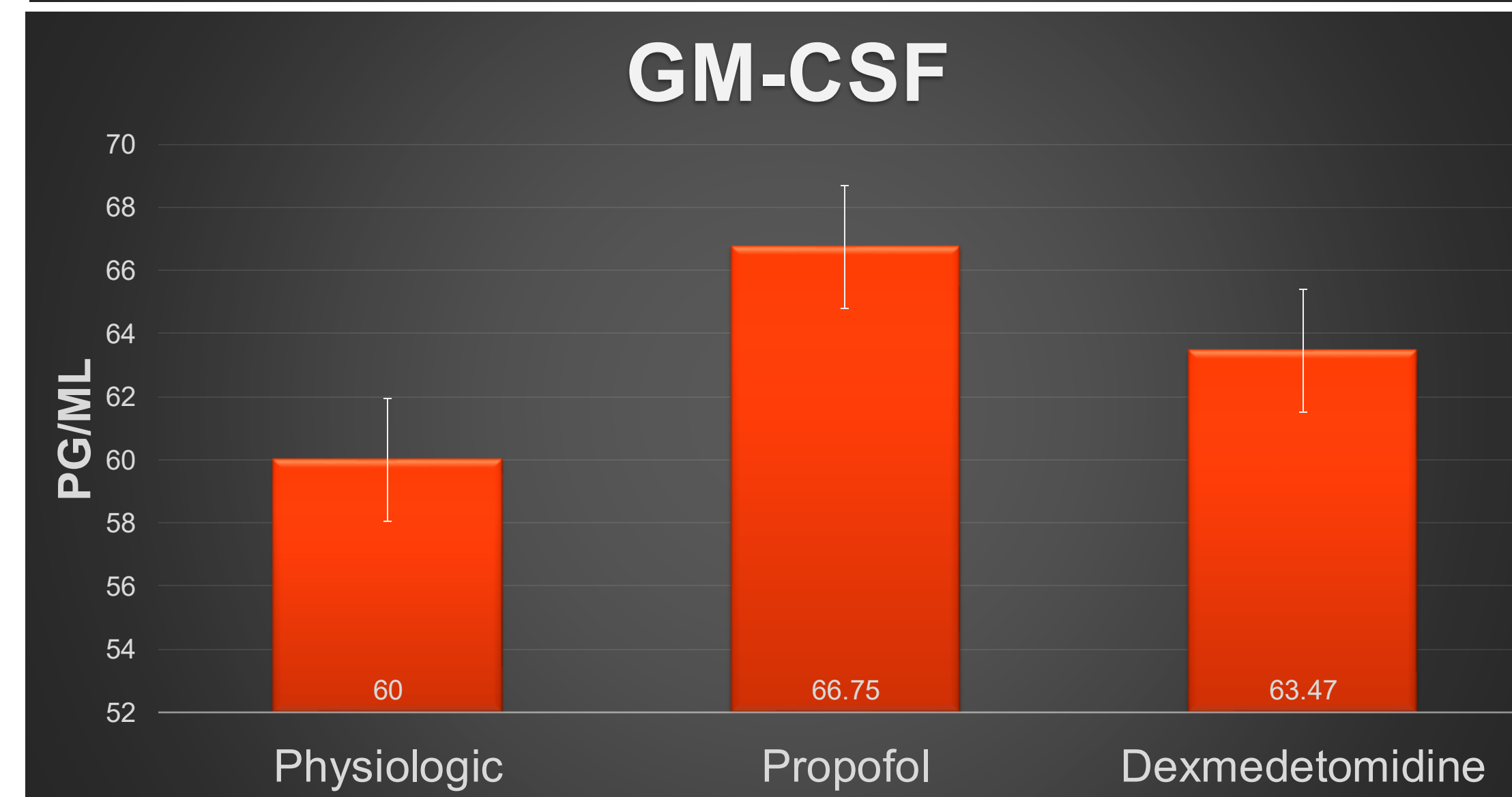
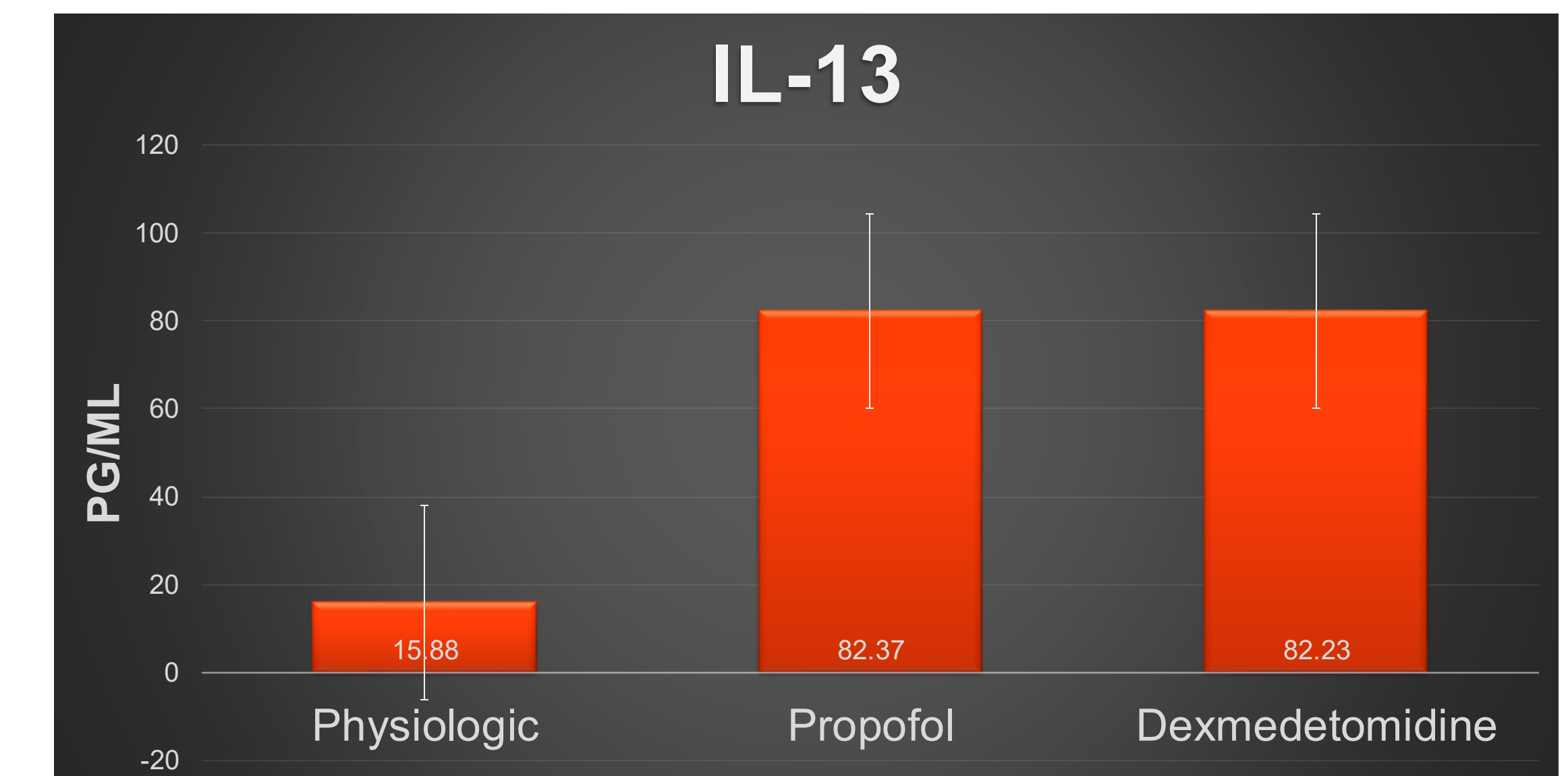
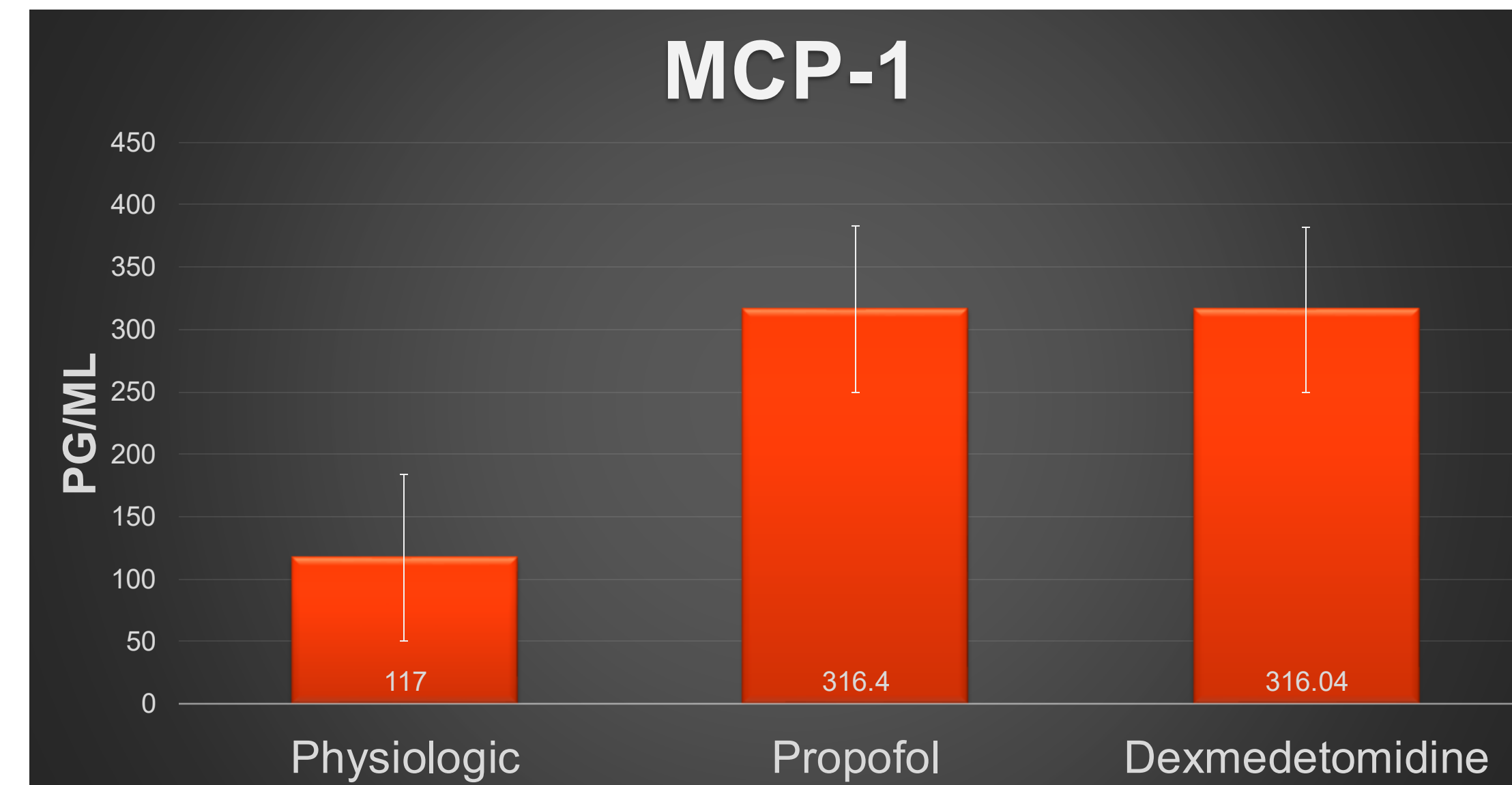
• Dexmedetomidine has been shown to cause greater attenuation of inflammation than propofol in mouse models.

HYPOTHESIS



RESULTS

- Collected 1099 samples from 422 participants on enrollment days 1, 3, 5, and 7
- No significant differences between mean biomarker and inflammatory levels of propofol or dexmedetomidine recipients



DISCUSSION

• Among critically ill adults with sepsis randomized to propofol or dexmedetomidine for continuous sedation, we did not identify a significant difference between biomarker and inflammatory cytokine levels

• These results are in agreement with primary study findings that demonstrated no difference in delirium outcomes between propofol and dexmedetomidine recipients

• Results should be interpreted with limitations including 48 hours administration limitations, relatively low medication doses to achieve light sedation, and potential significance of alternative biomarker or cytokines not investigated.

REFERENCES

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