Symposium on Prostate Cancer Clark Atlanta University

September 14-16, 2025

SAMPLE ABSTRACT FORMAT

(Read all of the instructions carefully before preparing your abstract.)

- 1. Abstract submissions should resemble the example below and emailed to ccrtd@cau.edu as a word (or equivalent) file to assist with abstract booklet printing.
- 2. Font size should be 12 points, Times New Roman with Justify Alignment.
- 3. Title of the abstract should be in caps.
- 4. Name of the presenting author should be in bold and underlined.
- 5. Posterboards are sized at 48 width x 36 height (in.)
- 6. Abstract should not exceed 325 words that include title, authors and authors' affiliation(s), and acknowledgments (grant support only).
- 7. Your abstract should include background, methods, results, and conclusion.
- Please submit your abstract in .docx format (or equivalent) to ccrtd@cau.edu
 Deadline for Abstracts: August 1, 2025 @ 11:59 PM EDT August 15th, 2025

EFFECTS OF TGF-β ON SURVIVAL OF SELECTED PROSTATE CANCER CELLS

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TGF-ß is a growth factor known to elicit a growth inhibitory effect in the normal and benign prostate cells but induces cell proliferation in malignant cells. However, little is known on its effects on apoptosis and its regulation. The objective of the study is to determine if TGF-β can induce apoptosis in prostate cancer cells and use DAPI (4'-6-diamidino-2-phenylindole) staining as the procedure to measure apoptosis index. In this study, prostate cell lines DU145, PC3 and C-33 were treated with two doses of TGF-β (1 and 10 ng/ml) for 72 hours. After treatment, cells were fixed and stained with DAPI to visualize the cells under UV light. Apoptosis was determined if there was visible fragmented nuclei/condensed chromatin. PI-3 kinase and MAP kinase inhibitors, LY294002 and PD 98059 respectively, were used as positive controls for apoptosis. Among the four cell lines, DU145 and C-33 showed significant level of cell death (up to 50%) when treated with the higher dose of TGF-β (10 ng/ml). PC3 cells were more sensitive to the lower dose of TGF-β (lng/ml) showing 38% apoptotic cells. LY and PD inhibitors induced apoptosis in DU145 (LY 78%, PD 56%), PC3 (LY 38%, PD 21%), and C33 (LY 40%, PD 26%). Based on the results, we were able to demonstrate that TGF-β can induce apoptosis in these cells and that apoptosis can be measured using DAPI staining. Some minor technical difficulties were encountered such as length of treatment and normalized cell number. These observations will be considered in the next set of experiments.

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