THE MECHANISMS BY WHICH FRACTIONS OF *Ocimum gratissimum* (*Og*) LEAF EXTRACTS INHIBIT THE PROLIFERATION OF PROSTATE CANCER (PC-3) CELLS AND COLON CANCER (HT-29) CELLS

Michael A. Webb¹, Stephen I. N. Ekunwe¹, Brittney-Shea Herbert² and Gregorio Begonia¹

¹Jackson State University Bridges to Doctorate Program, Jackson, MS, USA
²Indiana University School of Medicine and Melvin and Bren Simon Cancer Center, Indianapolis, IN, USA

**Abstract:** *Ocimum Gratissimum* (*Og*) also called *Eb’amwonkho*, is a member of the genus *Ocimum* L. (*Lamiaceae*) is native to Southern Nigeria. The leaves or whole plant of *Og* have been reported to aid in the treatment of diarrhea, upset stomach, and hemorrhoids, suggesting that *Og* possesses anti-inflammatory and anti-angiogenesis properties. This led us to hypothesize that *Og* contains compounds that may be useful in fighting cancer. In previous studies, done in Dr. Ekunwe’s lab, *Og* has been shown to inhibit the proliferation of colon cancer (HT-29; p53 null) cells as well as prostate cancer (PC-3, p53 null) cells in a dose-dependent manner. The objective of this study was to begin to elucidate the mechanism of action of several fractions (aqueous, P 2, P 3-2, and P 4-2) of *Ocimum gratissimum* leaf extracts. Thymidine incorporation assay, Western immunoblotting, cell cycle analyses by flow cytometry, MAPK antibody arrays, and plating efficiency assay were performed to determine the anti-proliferative effects, cell cycle arrest, and effects on cell cycle proteins cyclin D1, and p21 after *Og* treatment. Thymidine incorporation assays results indicated that the P 2, P 3-2 were the most potent fractions for reducing cancer cell DNA synthesis/proliferation with an IC₅₀ of 200 µg/ml after 24 hr. *Og* also reduced cyclin D1 and induced p21 expression in HT-29 and PC-3 cancer cells after 24 hr. Therefore, *Og* has the potential to reduce cancer cell proliferation by several signaling mechanisms. Results from these studies will provide insight into the mechanism of action of a potential new cancer therapeutic.

**Key words:** *Og*, cyclin d1, PC-3 cells, and HT-29 cells