ABSTRACT SUBMISSION PROCESS
22nd Workshop on Vitamin D, New York, NY

ABSTRACT FORMATTING GUIDELINES

1. File Format: **Word files only. No PDFs.**
2. Font: **Arial 10 pt. Do not use text boxes!**
3. Word Count: **Total 400 words. This includes title, authors and institutions.**
4. Title: **Capitalize all words in the title.**
5. Authors: **Place initials first followed by last name. Underline all author names.**
6. Spacing: **Use single spacing throughout.**
7. Justification: **Left.**
8. Content: **Single paragraph beginning with a three space indentation.**
9. Margins: **1 inch (left, right, top)**

Example of an appropriately formatted abstract:

VITAMIN D INHIBITS HYALURONAN SYNTHESIS IN BREAST CANCER CELLS AND TUMOR-DERIVED FIBROBLASTS. E LaPorta, S Robilotto, JE Welsh. Cancer Research Center, University at Albany, NY

Human breast tumors with abundant hyaluronan (HA) and high expression of the HA synthesizing enzyme HAS2 are more aggressive and exhibit poorer survival than tumors with low HA/HAS2 content. We identified HAS2 as a 1,25D repressed gene via genomic profiling, and examined the regulation of HA synthesis and turnover by 1,25D in the context of breast cancer. 1,25D inhibited HAS2 expression and HA synthesis in VDR positive murine mammary tumor cells, tumor derived fibroblasts and human breast cancer cell lines (MCF7, Hs578T). 1,25D had no consistent effects on expression of HAS1 or HAS3 or HA degrading enzymes (hyaluronidases HYAL1, HYAL2, HYAL3). HAS2 expression was unaffected by 1,25D in tumor cells and fibroblasts derived from VDRKO mice, however stable expression of hVDR into VDRKO cells was sufficient to confer HAS2 inhibition by 1,25D. Consistent with reduced HAS2 expression, 1,25D reduced the accumulation of HA in the peri-cellular matrix and in the culture media in VDR+ cells. Exogenous HA enhanced cell proliferation in the absence of 1,25D and partially rescued cells from growth inhibition in the presence of 1,25D. In MCF7, Hs578T and MCFDCIS cells, 1,25D downregulated expression of CD44 (a pro-survival receptor that is activated by HA) and HBEGF, POSTN and PLAU (ECM proteins that facilitate HA-CD44 signaling). These data suggest that HA-CD44 signaling is a novel target of 1,25D-VDR in aggressive breast cancer. Supported by NIH grants R01CA194500 and R21CA166434 to J Welsh.

10. Complete the online Abstract Submission information (submitting author's name and contact info, abstract title and subject category) on the abstract submission webpage at http://www.mpi-evv.com/2019VDW/2019VDWabstract.asp**.**

11. After registering your abstract email your abstract file to rhall@mpi-evv.com**. You will receive a confirmation email once the abstract has been downloaded by the organizers (typically within 24 hours).**

12. Abstracts received by March 15 that are not chosen for oral or plenary presentations will be slotted into general poster sessions on Thursday or Friday (May 30-31).

13. Abstracts received after March 15, 2019 will not be ranked or included in the program book but may be considered for presentation in the general poster sessions as space permits.

14. Multiple abstract submissions must be registered and submitted individually. One abstract only per email.