**Instructions for abstract submission**

1. Font: Arial, 12 pt.
2. Abstract size is 8.5" x 5.5" (19 cm x 14 cm) with 1 cm margins.
3. Title should be in bold letters, capitalized and centered across the page
4. Author names are centered and identified with numerical superscripts with the presenting author's name underlined.
5. Author affiliations are centered and identified with numberical superscripts that correspond to the respective author's names; include the email address for the presenting author
6. Please see below for an example.

**Phospholipases and matrix vesicles release**

1,2Najwa Skafi, 1Saida Mebarek, 1Leyre Brizuela, 3Nicolas Vitale, 2Eva Hamade, 2Badran Bassam, 1Rene Buchet

1Université Lyon 1, INSA de Lyon, CPE, Université de Lyon and UMR-5246 CNRS, F-69622 Villeurbanne, France (rbuchet@univ-lyon1.fr)

2Université Libanaise, Faculté des Sciences, Beirut Lebanon

3Institut des Neurosciences Cellulaires et Intégratives, UPR-3212 CNRS and 3Université de Strasbourg, F-67084 Strasbourg, France

Phospholipase D (PLD) catalyzes the hydrolysis of phospholipids, forming phosphatidate and a head group. The products of phospholipid hydrolysis affect cell signaling, differentiation, proliferation and maturation. In addition, phosphatidate induces membrane curvature and is suspected to facilitate exocytosis or endocytosis of vesicles. We reasoned that secretion of matrix vesicles (MVs) would increase upon activation of PLD due to alteration of plasma membranes or of actin cytoskeleton of mineralizing cells. MVs are vesicles secreted by mineralizing cells, initiating apatite formation. Here, we will report the effects of PLD on mineralization induced by chondrocytes. The presence of two PLD isoforms (PLD1 and PLD2) was ascertained by measuring their RNA level expression in primary chondrocytes. Mineralization process induced by primary chondrocytes isolated from wild type and from KO PLD mouse models were compared. As probed by cresolphtaline assay, calcium deposition decreased slightly in primary chondrocytes extracted from KO PLD1 and from KO PLD2 mouse model. These findings were correlated with a decrease in TNAP activity, as well as a decrease of RNA expression levels of *runx2* and *ocn* for KO PLD2 mouse model. MVs extracted from primary chondrocytes of KO PLD1 mouse were compared to MVs extracted from primary chondrocytes of wild types. Taken together these findings suggest that the activity of PLD regulates finely the mineralization process and may influence secretion of functional MVs.