Infection of a surgical or traumatic wound is one of the most common complications associated with elective and trauma surgery because of the inherent bacterial skin colonia break in the skin. Under the right conditions normally docile bacterial whose growth are normally kept in check may proliferate until there may be damage to the local soft tissues and bone (even expedite death). This damage can be aided with the right metabolic requirements for bacterial growth, cularity to deliver antibiotics, and the lack of skin coverage thesis, may prevent or impair healing leaving the prosthesis loose and or the fracture ends atrophic.[1] In either case, the remaining bone will resorb due to a variety of mechanical and physiologic causes expediting its failure.

However, in some instances, and for an unknown reason, extra skeletal osseous formation and remodeling may occur. This change in skeletal geometry is typically a hallmark of a deep infection or osteomyelitis. Therefore the same organism in on instance will cause new bone to be formed and in the other old bone will resorb. Though reports demonstrated that there is a macrophage mediated osteogenic effect which troduction of bacterial Lipopolysaccharide (LPS), a molecule typically found in the wall of gram negative bacteria.[2] Liof the infections do not come from gram negative bacteria, but from gram positive bacteria and mainly the Staph and Strep species. Because of the questionable utility of previous studies, further validation is necessary to validate the earlier reports by bacterial strains that are relatively more clinically relevant.

The purpose of this study was to measure the effect of a clinically important infectious agent (*staphylococcus aureus*

(continued)

and 72 hr time points. (Figure 1) Of the two Bac/Mac groups, 72h group had greater amount of alkaline phosphatase activ-

MSCs supplemented with Mac preparations at both time -
trol group. (Figures 2 & 3) The MSC cells supplemented with

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The results of this preliminary study suggest that rat pulmonary alveolar macrophages induce osteogenic response on the rat MSCs. However, this osteogenic effect on rat MSCs is inhibited with the addition of *staphylococcus aureus*. These

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The results also establish a time table for inhibition such that the osteogenic effect of macrophages emerges as soon as 6 hours and persists for up to 72 hours. While there was a trend for a temporal reduction in osteogenicity by macrophages,

power; therefore, current results need to be supplemented with further samples. It was also observed that Bac/Mac-72hr group had greater alkaline phosphatase activity than controls,

days. It is also likely that Bac/Mac-72hr group had undergone osteoblastic differentiation in a delayed manner and it may be possible that a time point later than 72hr may display

time durations to assess full recovery from this osteogenic inhibition.

From the results included here we were able to conclude that rat pulmonary alveolar macrophages induce osteogenic response on the rat MSCs. Also, when rat pulmonary alveolar macrophages are challenged by *Staphylococcus Aureus*, -
sponse on rat MSC. Lastly, *Staphylococcus aureus* inhibits macrophagemediated osteogenic effects in a time-dependent fashion.