S3-14. Evaluation of optical techniques for microbiological inactivation of blood

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Blood can be the target of bacterial, viral and parasitic contamination, which can trigger serious diseases. In this study, photodynamic inactivation and ultraviolet radiation were evaluated in the in vitro decontamination of whole blood, erythrocytes, and platelet-rich plasma with S. aureus. For PDI, Photogem® and 630nm light were evaluated, and risks of toxicity of the treatment were determined by hemolysis and cell viability assays. The reductions of S. aureus in whole blood, erythrocytes, and platelet-rich plasma at 15 J/cm2 and 50 µg/mL porphyrin were 1.0 log, 1.3 logs and 0.4 log CFU/mL, respectively. Hemolysis rate for erythrocytes in whole blood was 10.7%. However, erythrocytes hemolysis was 100% when in the absence of plasma. The cell viability assay showed 14% apoptosis rates in isolated erythrocytes, indicating damaging action of PDI, and no damage in platelet. For UVC radiation (254nm), different light doses were analyzed, and the cell viability assay determined the toxicity of technique. The reductions of S. aureus in whole blood, erythrocytes and platelet-rich plasma at 23 J/cm2 were 1.7 logs, 1.1 logs and 2.5 logs CFU/mL, respectively. Relatively small differences were observed in plasma as a function of irradiation time, suggesting some degradation of plasma proteins with 23 J/cm2. The cell viability assay showed normal rates for erythrocytes, however, in the platelets, a high apoptosis rate was observed (74%). Therefore, the optical techniques showed opposite damage effects in each blood component, and the use of one or another technique should be evaluated considering the better microbial inactivation and blood components preservation conditions.

S3-15. Antimicrobial Photodynamic Therapy Challenges Microbial Drug-Resistance

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Background: The rising challenge of microbial resistance to chemotherapy demands immediate implementation of global policies and therapeutic alternatives. Antimicrobial photodynamic therapy (APDT) combines the administration of a photosensitizer (PS) compound with low-intensity monochromatic light to induce photochemical reactions that yield high amounts of reactive oxygen species (ROS). Since some PS molecular frameworks can be selectively incorporated by pathogens and ROS react with virtually all biomolecules, APDT offers a powerful strategy to challenge microbial resistance of local infections. Methods: In this study we assayed the APDT efficacy, using methylene blue (MB) as PS and red light provided by LED, against planktonic suspensions of high-risk representative fungal and bacterial species. The species tested include A. baumannii (OXA-23 and 143), E. aerogenes (NDM-1), E. faecalis (VAN-B), E. faecium (VAN-A), E. coli (MCR-1, CTX-M8 and 15), K. pneumoniae (KPC-2, IMP-1, OXA-48), S. aureus (MRSA, VISA), P. aeruginosa (VIM-1, SPM-1, GES-5), C. albicans and C. neoformans. For all species, we tested standard control strains compared to azole-resistant veast, or bacteria resistant to nearly all commercially available antimicrobials, in attempt to observe any crossresistance in between APDT and standard chemotherapy. Results: More than 5log10 reduction was observed within less than a minute of illumination for non-capsulated bacteria and within less than 5 minutes for yeast and capsulated bacteria. Regardless of resistance phenotype MB-APDT presented species-specific dose-response kinetics suggesting that similar therapeutic protocols may bring successful outcomes in clinical practice. Conclusions: Our study proposes that MB-APDT can efficiently inactivate a broad-spectrum of drug-resistant microorganisms and impair drug-resistance genes selection and dissemination.

S3-16. Photodynamic inactivation using curcumin applying in biofilm developed at polymeric surfaces.

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Infectious disease is a continuous treatment challenge for the hospitals. Ventilator Associated Pneumonia (VAP) is one of the most dangerous respiratory disease, because can lead the patient to death. The majority of the antibiotics can not destroy the cells from the developed biofilm at a surface of the endotracheal tube. The