

Infectious Diseases 2015 :Evaluation of interaction mechanisms between *Acinetobacter baumannii* bacteria and lipidic nanocapsules by flow cytometry -Angelique Montagu -University of Angers

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Acinetobacter baumannii is an important nosocomial pathogen, resistant to many commonly-used antibiotics. Considering the limited number of antibiotics in development, interesting strategies could lay on the use of natural resources especially essential oils (EOs). An interesting strategy underlined the antibacterial potential of EO components (carvacrol, cinnamaldehyde) loaded LNCs against *A. baumannii*. The aim of this study was to realize and characterize DiO-LNCs loaded with antibacterial actives. The antibacterial activity of these nano-carriers was evaluated in vitro against *A. baumannii*. Finally, we determine the interactions between bacteria and LNCs over time thanks to the fluorescence of DiO-LNCs and the properties of trypan blue in order to precise the physicochemical mechanisms occurring at the level of the biological membrane. The results underlined the attractiveness of the encapsulated actives compared to unloaded-LNCs. These results demonstrated the capacity of carvacrol-loaded-LNCs to interact and penetrate the bacterial membrane in comparison with cinnamaldehyde-LNCs and unloaded-LNCs. Moreover, the fluorescence of bacteria remained constant after contact with carvacrol-loaded LNCs and cinnamaldehyde-LNCs whereas the fluorescence of the blank-LNCs decreased over time. This phenomenon could be explained by the release of these blank-LNCs by efflux pumps. Thereafter, modifications of carvacrol after substitution of hydroxyl functions by fatty acids (acetic acid, palmitic acid) demonstrated the crucial role of these latter for antibacterial activity. Finally, after contact with an efflux pump inhibitor CCCP (carbonylcyanide-3-chlorophenyl hydrazine), the results underlined a total synergistic effect for Car-LNCs showing that the CCCP is associated with action mechanism of carvacrol especially at the level of efflux pump mechanism. *Acinetobacter baumannii* may be a typically short, almost round, rod-shaped (coccobacillus) Gram-negative bacterium. It's named after the bacteriologist Paul Baumann. It are often an opportunistic pathogen in humans, affecting people with compromised immune systems, and is becoming increasingly important as a hospital-derived (nosocomial) infection. This might flow from the activity of type IV pili, pole-like structures which will be extended and retracted. Motility during a *A. baumannii* can also flow from the excretion of exopolysaccharide, creating a movie of high-molecular-weight sugar chains behind the bacterium to maneuver forward. Clinical microbiologists typically differentiate members of the genus *Acinetobacter* from other Moraxellaceae by performing an oxidase test, as *Acinetobacter* spp. are the sole members of the Moraxellaceae to lack cytochrome oxidases. *A. baumannii* is a component of the ACB complex (*A. baumannii*, *A. calcoaceticus*, and *Acinetobacter* genomic species 13TU). It's difficult to work out the precise species of members of the ACB complex and that they comprise the foremost clinically

relevant members of the genus. *A. baumannii* has also been identified as an ESKAPE pathogen (*Enterococcus faecium*, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Enterobacter* species), a gaggle of pathogens with a high rate of antibiotic resistance that are liable for the bulk of nosocomial infections. Adhesion are often a critical determinant of virulence for bacteria. The power to connect to host cells allows bacteria to interact with them in various ways, whether by type III secretion system or just by holding on against the prevailing movement of fluids. Outer membrane protein A (OmpA) has been shown to be involved within the adherence of *A. baumannii* to epithelial cells. This enables the bacteria to invade the cells through the zipper mechanism. The protein was also shown to localize to the mitochondria of epithelial cells and cause necrosis by stimulating the assembly of reactive oxygen species. Pathogenicity islands, relatively common genetic structures in bacterial pathogens, are composed of two or more adjacent genes that increase a pathogen's virulence. They'll contain genes that encode toxins, coagulate blood, or as during this case, allow the bacteria to resist antibiotics. AbaR-type resistance islands are typical of drug-resistant *A. baumannii*, and different variations could also be present during a given strain. Each consists of a transposon backbone of about 16.3 Kb that facilitates horizontal gene transfer. Transposons allow portions of genetic material to be excised from one spot within the genome and integrate into another. This makes horizontal gene transfer of this and similar pathogenicity islands more likely because, when genetic material is haunted by a replacement bacterium, the transposons allow the pathogenicity island to integrate into the new microorganism's genome. during this case, it might grant the new microorganism the potential to resist certain antibiotics. AbaRs contain several genes for antibiotic resistance, all flanked by insertion sequences. These genes provide resistance to aminoglycosides, aminocyclitols, tetracycline, and chloramphenicol. The first, AdeB, has been shown to be liable for aminoglycoside resistance. Bacterial small RNAs are noncoding RNAs that regulate various cellular processes. Three sRNAs, AbsR11, AbsR25, and AbsR28, are experimentally validated within the MTCC 1425 (ATCC15308) strain, which may be a (multidrug-resistant) strain showing resistance to 12 antibiotics. AbsR25 sRNA could play a task within the efflux pump regulation and drug resistance. *A. baumannii* has been noted for its apparent ability to survive on artificial surfaces for an extended period of your time, therefore allowing it to continue the hospital environment. This is often thought to flow from to its ability to make biofilms. For several biofilm-forming bacteria, the method is mediated by flagella. However, for *A. baumannii*, this process seems to be mediated by pili. Further, disruption of the putative pili chaperone and usher genes *csuC* and *csuE* were shown to inhibit biofilm formation.

The formation of biofilms has been shown to change the metabolism of microorganisms within the biofilm, consequently reducing their sensitivity to antibiotics. This might be because fewer nutrients are available deeper within the biofilm. A slower metabolism can prevent the bacteria from taking over an antibiotic or performing an important function fast enough for particular antibiotics to possess an impact. They also provide a physical barrier against larger molecules and should prevent desiccation of the bacteria.

Biography

Angelique Montagu is a PhD student at the University of Angers through a CIFRE thesis with Eydo pharma, dealing with essential oils encapsulation for the treatment of nosocomial infections under the direction of Pr. Marie-Laure Joly-Guillou and Pr. Patrick Saulnier. She studied biology at the University of Angers and she obtained master's degree in biology whose the subject was the use of mesenchymal stem cells as vehicles for lipid nanocapsules for the treatment of glioma.

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