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Extended Abstract
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Infection Control 2018: Association of Helicobacter pylori biofilm with enterovirus 71 prolongs viral viability and survival - Vincent TK Chow - National University of Singapore

Vincent TK Chow
National University of Singapore

Viruses are unable to replicate outside their hosts, and thus the transition time during which a virus leaves its host and infects the next susceptible host is critical for virus survival. Closely associated with hand, foot and mouth disease with occasional neurologic complications, enterovirus 71 (EV71) is stable in aqueous environments. However, its survival in the environment and interactions with bacteria are not well-established. On the other hand, Helicobacter pylori is a well-known and highly successful gut bacterial pathogen that infects 50% of individuals, with its capacity to form biofilms being linked with its transmission. We hypothesized that bacterial biofilm may play a significant role in the survival of EV71 in the external environment. In this study, we examined the interactions of EV71 with the biofilm of H. pylori. The results reveal that EV71 associated with H. pylori biofilm as observed under confocal and scanning electron microscopy. Furthermore, the presence of biofilm prolonged viral viability as demonstrated by virus plaque assays. Interestingly, the viability of the virus was dependent on the quantity of H. pylori biofilm formation. Taken together, the ability of bacterial biofilm in extending EV71 viability for prolonged periods may partially contribute to EV71 outbreaks, and implies that the association of the virus with bacterial biofilm may serve as a potential pathway of EV71 transmission.

In an underlying examination of biofilm development by H. pylori, two investigations described biofilm arrangement by this living being. As the primary showing of the in vitro capacity to shape biofilms by H. pylori, Stark et al. detailed that a water-insoluble polysaccharide-containing biofilm has been seen at the air-fluid interface when H. pylori strain NCTC 11637 was ceaselessly developed in a glass fermenter. In this way, Cole et al. announced that the entirety of the H. pylori strains utilized in their investigation, including clinical separates, research facility strains, and a mouse-adjusted strain, had the option to shape biofilms on glass surfaces. They likewise announced that H. pylori could frame a biofilm just at the air-fluid interface, which is in all likelihood characteristic of its microaerobic. In any case, at present, biofilm development by H. pylori has not been broadly described. Subsequently, we broke down the capacity of H. pylori strains to frame biofilms and portrayed the fundamental components included. At first, we set up an attainable and stable model for biofilm development by this microorganism. Quickly, disinfected glass coverslips were plated into 12-well microtiter plates. Each very much was loaded up with 2 mL of Brucella stock enhanced with 7% fetal calf serum (FCS) to permit adherence of H. pylori at the air-fluid interface. The arrangement of biofilms was started by immunizing around 5 × 105 cells into each well. The way of life was brooded under microaerobic conditions at 37°C for 3 to 5 days with shaking. Utilizing this model, the biofilm-shaping capacity of eight H. pylori strains including standard SS1, ATCC 43579, ATCC 43579, and NCTC11638 strains and clinical confines from Japanese patients were dissected. Under these conditions, the entirety of the strains framed biofilms at the fluid gas interface of the way of life. In particular, strain TK1402, which was secluded from a Japanese patient with duodenal and gastric ulcers, demonstrated fundamentally more significant levels of biofilm arrangement comparative with different strains. The solid biofilm-framing capacity of TK1402 was reflected in the overall thickness of the biofilms. To explain the engineering attributes of H. pylori biofilms, we looked at TK1402 and SS1 biofilms by examining electron microscopy (SEM). In the SS1 biofilms, the microscopic organisms appended to glass surfaces in slight layers, and the biofilms comprised mostly of bleb-like or indistinct structures. Then again, the TK1402 biofilms were made essentially out of cells with bacillary morphology which were plainly delineated. We additionally examined the biofilm cells of different strains utilizing SEM. In any case, most of these biofilm cells comprised of autolyzed cells, proposing that the solid biofilm-framing capacity of TK402 may have come about because of a functioning metabolic state for a moderately lengthy timespan without displaying morphological changes or autolysis. Likewise, the biofilms of the TK1402 strain

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demonstrated the nearness of numerous external film vesicles (OMVs) on the glass surfaces just as on the bacterial cell surfaces. These structures were not distinguished in the biofilms of different strains. OMVs were all the more firmly saw in the slender separated biofilms utilizing transmission electron microscopy (TEM) and the OMVs were situated at the base bacterium interface and in the extracellular spaces. Furthermore, biofilm arrangement by strain TK1402 has unequivocally corresponded with the creation of OMV. These outcomes proposed that the OMV delivered by strain TK1402 may fill in as an EPS lattice for these biofilms. OMV creation is a physiologically ordinary capacity of gram-negative microscopic organisms. In Pseudomonas aeruginosa, OMVs have multifunctional natural jobs including microbial communication and host contamination just as upkeep of the structure of biofilm. In Porphyromonas gingivalis, OMVs advance connection, conglomeration, and biofilm arrangement, and the elements of OMVs in biofilms have been talked about. Like most gram-negative microbes, H. pylori discharged OMV into the extracellular space. Significant protein and phospholipid parts related to the OMVs were distinguished. We broke down the protein profile of the OMV delivered by strain TK1402 to figure out which segments of the OMV add to biofilm development in H. pylori. The outcomes showed that a particular around 22 kDa protein may be engaged with the biofilm-shaping capacity of this strain. Extra exploration is currently in progress to figure out what components are straightforwardly associated with biofilm development by strain TK1402.

Pathogenic bacteria including H. pylori within biofilms can shake both host immune responses and therefore the effects of antimicrobial agents. Consequently, chronic infections by biofilm-forming bacteria become troublesome and difficult to treat. Some of the previous studies have shown that H. pylori forms a biofilm on the human gastric mucosa. Nevertheless, assessment of H. pylori strains susceptibility to antibiotics in vitro has traditionally been evaluated using planktonic cells, so that MICs are not reliable predictors of the antibiotic effects in the human stomach. The assessment of the ability to form biofilms in H. pylori could play a crucial role in preventing and controlling the generation of antibiotic resistance. It is expected that enhancing our knowledge of H. pylori biofilm formation will cause new treatment therapies for preventing H. pylori infections. However, it is recognized that our understanding of H. pylori biofilm formation is still in its infancy. Further studies of the mechanism of H. pylori biofilm formation need to be performed.