Biosurfactants Change the Thinning of Contaminated Bubbles at Bacteria-Laden Water Interfaces

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Bubbles reside at the water surface before bursting, emitting droplets that can contain chemicals and pathogens linked to disease and contamination. We discover that bacterial secretions enhance the lifetime of bubbles. We also reveal and elucidate two distinct regimes of thinning for such contaminated bubbles. Initially, marginal regeneration governs their thinning rate, similarly to clean water bubbles. However, due to their enhanced lifetime, it is eventually evaporation that governs their thinning, thus also dramatically decreasing their thickness at burst. We derive and experimentally validate the expression for the critical timescale at which the transition between the two regimes occurs. The shift in thinning law makes the droplets produced by contaminated bubbles smaller, faster, and more numerous than those produced by clean bubbles. Our findings suggest that microorganisms can manipulate the aging physics of surface bubbles to enhance their own water-to-air dispersal.

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Air bubbles are ubiquitous at water surfaces [1,2] of pools, hot tubs, recreational facilities [3-5], toilets [6-8], and wastewater plants [9], and involve sloshing, impacts, and plunging jets [10]. They are also the outcome of natural processes such as breaking waves and rain-drop impacts [10,11]. Surface bubbles eventually burst and fragment into droplets that contain chemicals and pathogens that have important consequences on climate [12-15] and air contamination, indoor and outdoor. Bubbles are a major public health concern: the droplets they generate in contaminated water carry infectious payloads [16] that can remain suspended in the air and cause airborne disease transmission [17]. A direct link between health hazards and bursting bubbles was first reported for the transport of aerosols containing neurotoxins associated with algal blooms [18-21]. Inhalation of droplets from bubbles containing bacteria is also a recognized route of infectious disease transmission [22], including for heavy burden pathogens such as Clostridium difficile [6,23], Legionella [24], and nontuberculous Mycobacteria [25,26]. Droplets from bubbles also contribute to the large scale dispersal of marine viruses [27,28]. Yet, despite their significance for climate, ecology, and public health, the interplay between bubbles and pathogens remains unknown and the ability of bacteria to manipulate the bubble physics has so far been ignored.

Upon burst, large surface bubbles ($R \gtrsim 1$ mm, with *R* the bubble cap radius) produce film droplets via fragmentation of their cap (Fig. 1). To understand the details of film droplet properties, it is important to first characterize the basic physical processes at their origin. Indeed, the number, size, and speed of film drops depend on the bubble cap

thickness at burst [29], itself a function of the time that the bubble spent at the water surface, its surface lifetime: older bubbles are thinner, and produce more, smaller, and faster droplets (Fig. 1). The bubble cap thickness evolution has been characterized for *clean* water [29,30], but discrepancies on film drops reported in the literature persist, even from experiments on individual bubbles in controlled laboratory settings. Specifically, the effect of the water composition on the bubble physics and on the number and sizes of film drops is unclear [12,31–33], and in particular the effect of bacterial contamination on surface bubbles remains unknown.

In Fig. 2 we reveal bacteria on a thin contaminated bubble cap using interferometry imaging. This novel visualization of pathogens directly shows their presence on the cap throughout the bubble life and that they do not



FIG. 1. Bubbles bursting after (a) 4 s and (b) 55 s at the surface of water with 0.61 and 0.21 ms between frames, respectively. The burst of the older bubble produces many more and smaller droplets. Scale bars are 1 mm.



FIG. 2. Fifty second-old bubble observed using monochromatic light (sodium lamp, 589 nm) at the surface of water contaminated with *E. coli*: the bright spots are only observed when bacteria are present and reveal their location and persistence on the cap. Scale bar is 1 mm.

readily drain out into the bulk. Understanding the thickness of contaminated bubble caps that governs film drop numbers, sizes, speeds [29], but also their bacterial load is thus critical. Our experimental setup is a vertical tube filled with liquid [30]. We generate bubbles one by one below the surface with a tip connected to an air pump. Once bubbles reach the surface, we monitor their lifetime and obtain the time evolution of bubble cap thickness by filming bursts with high-speed imaging. Indeed, upon rupture of a bubble, a hole forms on the cap and grows at constant speed v. We measure this speed, as explained in details in [30], and relate it to the cap thickness h using the Taylor-Culick relation [34,35]: $h = 2\sigma/\rho_w v^2$, with σ the surface tension of the bulk water and ρ_w its density.

Prior studies [30] and the present Letter show that bubbles at clean water interfaces are short lived, with lifetimes seldom longer than 10 s (Fig. 3). However, we discovered that their lifetimes can reach 70 s when the water has been stagnant and exposed to air in an uncontrolled manner for weeks [Fig. 3(a)]. We also discovered that the cap of these long-lived bubbles has a peculiar thickness evolution after 30 s, with a faster thinning [Fig. 3(a)] never reported for *clean* water bubbles. Upon *a posteriori* investigation of water-composition for such peculiar bubbles, we identified natural bacterial contamination, possibly explaining such observations. The remainder of this study focuses on elucidating the robustness of the existence of this additional thinning regime and how bacterial contamination leads to its creation, thereby altering the physics of thinning of bubble caps.

To study bacterial contamination in a controlled setting we use *E. coli* O157:H7 (strain EDL933 Δ stx), bacteria



FIG. 3. (a) Thickness evolution of bubbles with $R = 5.5 \pm 0.2$ mm at ambient humidity in fresh deionized water, M9 medium [36,37] diluted 20 times, and stagnant water. The diluted M9 culture medium does not significantly affect the lifetime nor thinning evolution of bubbles. (b) Thickness evolution of bubbles with $R = 5.4 \pm 0.2$ mm derived from solutions of *E. coli* at $c_1 = 3 \times 10^7$ and $c_2 = 6 \times 10^6$ cells mL⁻¹, with or without filtration of the bacteria. Solutions of filtered bacteria are obtained using a 0.22 μ m pore-size filter. The ambient humidity is $H = 22 \pm 5\%$, and H > 95% for saturated air conditions. All experiments in this study are conducted at ambient temperature $T = 24 \pm 1$ °C. In this figure and the next, filled and open symbols show natural and manual bursts, respectively: due to their more deterministic lifetimes, bubbles from concentrated solutions of surfactants or bacterial secretions must be pierced manually with a needle to obtain their thickness at an early time. (c) Probability distribution function of bubble lifetimes t_b in ambient air, with deionized water. Bubbles in pure water scarcely live longer than 10 s, while bubbles in filtered or unfiltered solutions of bacteria can live up to 90 s. (d) Similar results are obtained with tap water, for which bubbles have the same thickness evolution as with deionized water [30]. Each curve in (c) and (d) represents one dataset consisting of an average of 1600 bubbles per curve.

involved in waterborne outbreaks [38] and that may also be airborne [39]. We grow them in minimal medium, M9 [36] and dilute the culture in deionized water [37]. We selected this protocol to limit the possible influence of surface active materials or electrolytes present in rich growth media (e.g., lysogeny broth) on the bubble lifetime and drainage [Fig. 3(a)]. Figure 3(b) shows the time evolution of the cap thickness h(t) of contaminated bubbles. When young, the bubble cap thickness follows a power law consistent with $h(t) \sim t^{-2/3}$ as predicted by a drainage model derived and verified in prior work for clean water [29,30]. Contaminated bubbles clearly live longer than in noncontaminated water as shown in Figs. 3(c) and 3(d). Indeed, although the lifetime of water bubbles in noncontaminated water can vary when using the same initial water and same experimental setup and protocol, their lifetime always follows the same underlying physics governed by a unimodal distribution of lifetimes, with a mean lifetime ranging from 1 to 10 seconds [29,30]. Yet, when adding bacteria, that underlying physics appears to change and the bubbles can live much longer [Figs. 3(c) and 3(d)], up to 80s for the bubble size considered herein.

This shift in maximum lifetime and shape of lifetime probability density function is robust to change of water type, deionized or tap water, and turns out to have profound consequences on the thinning of the bubbles prior to burst. As seen in Fig. 3(b), when reaching approximately 30 s of age, bubbles thin extremely fast, no longer following the classical drainage law $h(t) \sim t^{-2/3}$. The transition between the two regimes appears as a kink on the curve of thickness temporal evolution. When bacteria are filtered out of the solutions, these long lifetimes and kink in the curve of thickness evolution persist. The comparison of thinning laws between filtered and unfiltered contaminated water [Fig. 3(b)] shows that *bacterial secretions* [40,41], *not directly bacteria*, are responsible for the observed kink in thinning law.

To rationalize this radical shift in thinning law for old contaminated bubbles, we hypothesize that the kink is induced by the evaporation of the cap film. We test this mechanism by comparing the thinning of contaminated bubbles in saturated and unsaturated air [Fig. 3(b)]. Without evaporation, in saturated air, the bubble cap thickness remains governed by $h(t) \sim t^{-2/3}$ even for long lived contaminated bubbles, which could live up to 2 minutes: *the kink is indeed induced by evaporative thinning*. We showed in prior work on clean water bubbles that evaporative cooling leads to replenishing Marangoni dynamics on the cap [30]: this leads to thicker bubbles in unsaturated ambient, but has little effect on the exponent of the thinning power law, $h(t) \sim t^{-2/3}$. Next, we quantify the effect of cap evaporative water loss on bubble cap thinning.

The bubble cap drainage $h(t) \sim t^{-2/3}$ is set by a competition between viscous stresses localized at the bubble foot and curvature pressure [29] as well as Marangoni

stresses arising from surface tension differences between the cap and the bulk if present [30]. This leads to an evolution of the cap thickness h(t) set by a drainage speed $u(t) \sim (\sigma/\mu)(h/R)^{3/2}$, with μ the dynamic viscosity of the liquid. In addition, evaporation is characterized by the mass flux J of vapor at the bubble surface. When neglecting convection and the influence of the surrounding water bath, this is given by the following [42]:

$$J = \frac{1}{R} \rho_a D_v \frac{M_v}{M_a} \frac{p_v^{\text{sat}}}{p_a} (1 - H), \qquad (1)$$

where *R* is the bubble cap radius, M_v and M_a the molar mass of water and air, respectively, ρ_a the air density, p_a the atmospheric pressure, p_v^{sat} the saturation vapor pressure, D_v the diffusivity of water in air, and *H* the relative humidity. Mass conservation leads to $\rho_w S \dot{h} + \rho_w P u h + SJ = 0$, with *S* and *P* the bubble surface area and foot perimeter, respectively. Defining $a = Pu/(Sh^{3/2})$ we consider bubbles of size $R \gtrsim \ell_c$, with capillary length $\ell_c = \sqrt{\sigma/\rho_w g}$. Using $P/S \sim \ell_c/R^2$ for this geometry [29], $a \sim \sigma \ell_c/(\mu R^{7/2})$. Nondimensionalizing the cap thickness *h* and time *t* by $h_c = (J/\rho_w a)^{2/5} \sim R[\frac{R}{\ell_c} \frac{J/\rho_w}{\sigma/\mu}]^{2/5}$ and $t_c = \rho_w h_c/J$, respectively, the governing equation reads as follows:

$$\frac{d\tilde{h}}{d\tilde{t}} + \tilde{h}^{5/2} + 1 = 0, (2)$$

with $\tilde{h} = h/h_c$ and $\tilde{t} = t/t_c$. The critical thickness h_c is the thickness below which evaporation is the dominant mechanism of mass removal and cap thinning, while above h_c cap thinning remains driven by drainage. The lifetime at which this shift occurs is $t(h = h_c) \simeq t_c/2$: at early time ($t < t_c/2$, $\dot{\tilde{h}} \sim -\tilde{h}^{5/2}$) the thickness evolution is governed by drainage and follows $h(t) \sim t^{-2/3}$, while at later time $(t > t_c/2, t_c/2)$ $\tilde{h} \sim -1$) evaporation leads to a faster thinning than what drainage alone would induce for such thin bubbles, consistent with our observations [Figs. 3(a) and 3(b)]. Typically, $h_c \sim 1 \ \mu m$ and $t_c \gtrsim 20 \ s$ for the bubble size considered herein: this shows that evaporative mass loss is negligible for young bubbles typical of clean water [30]. On the other hand, we show in Fig. 3 that bacterial secretions can make bubbles live long enough, more than a minute, for evaporative thinning to become dominant. Next, using analog experiments, we determine if bacterial secretions could be considered as acting similarly to exogenous surfactants that can increase bubble surface lifetimes.

We conduct experiments using solutions of water and exogenous soluble surfactants [43] of various types: anionic (sodium dodecyl sulfate, SDS) and cationic (tetradecyltrimethylammonium bromide, $C_{14}TAB$). For robustness and validation, we vary their concentrations from 0.01 to 10 critical micelle concentration (cmc) and vary the air ambient relative humidity from unsaturated to saturated. Figure 4 shows that the thickness of bubbles in solution of both surfactants are in very good agreement with Eq. (2). In particular, the thickness evolution follows $h(t) \sim t^{-2/3}$ very closely at early time. This finding is robust even at concentrations above the cmc: at early time ($t < t_c/2$, $h > h_c$), their thinning remains distinct from the rigid soap film limit [44]. Despite the similarity between the drainage of contaminated bubbles and that of clean ones at early time, surfactants do introduce an important change: they enhance the lifetime of bubbles (Fig. 4), enabling their cap to reach the critical thicknesses h_c below which a shift to a cap thinning dominated by evaporative mass loss occurs. Similarly to what we showed for the effect of bacterial secretions [Fig. 3(b)], this shift appears in the form of a kink clearly seen in both thinning laws of the anionic and cationic surfactant-laden bubbles, which is well captured by our model [Figs. 4(a) and 4(c)].

We now turn to the thickness at burst of contaminated bubbles. Without evaporation, a lower bound for their thickness at burst h_b would be that at which the film spontaneously rupture via Van der Waals forces [45,46]: $h_b \approx 10{-}100$ nm, with corresponding maximum lifetime $t_b^{\text{drain}} = 2/(3ah_b^{3/2}) = \mathcal{O}(1 \text{ h})$. With evaporation, the maximum lifetime of contaminated bubbles is instead given from Eq. (2) by

$$t_b^{\text{evap}} = t_c \int_{h_b}^{h_0} \frac{d\tilde{h}}{1 + \tilde{h}^{5/2}} \simeq t_c \int_0^\infty \frac{d\tilde{h}}{1 + \tilde{h}^{5/2}} \simeq 1.3 t_c, \quad (3)$$

with $t_b^{\text{evap}} = \mathcal{O}(10\text{s}) \ll t_b^{\text{drain}}$. In other words, it is evaporation, not drainage, that limits the maximum lifetime accessible to very stable bubbles, here the contaminated ones. It is important to note that t_b^{evap} and t_b^{drain} are only upper bounds of lifetime. Indeed, similarly to clean water bubbles [29,30], the thickness at burst of bubbles in unfiltered solutions of bacteria is broad, with a much longer tail in the lifetime distribution of contaminated

bubbles (up to ~ 70 s) compared to clean ones (up to ~10 s) [Figs. 3(c) and 3(d)]. Note that, by contrast, the lifetime distributions of bubbles from both filtered bacteria and concentrated exogenous surfactant solutions are peaked towards long lifetimes [Figs. 4, 3(c) and 3(d)], closer to the upper bound t_b^{evap} . For bubbles contaminated with bacteria, to fully elucidate the contrast between distributions peaked toward long lifetimes for bubbles from filtered solutions versus long-tailed distributions peaked at short lifetimes for bubbles from nonfiltered bacteria solutions, further research is needed to answer the following question: what is the regime of competition between the stabilizing effect of bacteria secretions, shown in this study to act as exogenous surfactants, with the destabilizing effect of local bacterial inclusions in the film [30]?

In sum, we showed that bacterial secretions act as surfactants that increase the lifetime of surface bubbles. yet maintaining a drainage similar to that of clean water bubbles at an early time. However, with their increased lifetimes, a sharp kink in bubble cap thickness evolution arises. This kink is also observed for analog experiments with exogenous artificial soluble surfactants of various types. We elucidated it by deriving a theoretical model combining evaporation and drainage and showed that, for a given relative humidity and bubble size, a critical lifetime t_c exists at which the thickness evolution of long-lived bubbles becomes dominated by evaporative thinning rather than classical drainage, making old contaminated bubbles actually thin dramatically faster than young ones. We showed how this critical transition lifetime depends on the water and bubble properties and validated our model with experiments involving both bacteria-contaminated water and synthetic surfactants.

Surfactants and bacterial secretions lead to older bubbles that are ultimately much thinner at burst than what current bubble drainage models, derived for clean bubbles, would



FIG. 4. (a) and (b) Thickness evolution of bubbles in solutions of SDS with $R = 4.8 \pm 0.1$ mm in (a) ambient air $(H = 47, 32, 40, 53 \pm 2\%, 32, 40, 53 \pm 2\%$ for increasing SDS concentration) and (b) in saturated air (H > 95%). Solid and dotted lines are solutions of Eq. (2) for (a) $H = 50 \pm 30\%$ and (b) $H = 98 \pm 1\%$. The dashed lines corresponds to fully saturated air, H = 100%, when the thickness evolution is solely governed by drainage $(h \sim t^{-2/3})$. (c) Thickness evolution of bubbles with $R = 5.2 \pm 0.4$ mm in solutions of C₁₄TAB at 5%, 50%, and 10 cmc in ambient air $(H = 26 \pm 2\%)$ and at 50% of the cmc in saturated air. The plain and dotted line are the prediction for H = 26 and 96% from Eq. (2).

otherwise predict. It is estimated [29] that the mean diameter $\langle d \rangle$ and number N of film drops emitted by a water bubble scale as $\langle d \rangle \sim R^{3/8} h_b^{5/8}$ and $N \sim (R/\ell_c)^2 (R/h_b)^{7/8}$ with h_b the cap thickness at burst. Hence, bacterial secretions, including biosurfactants, increase the number of droplets from long-lived contaminated bubbles and can select for smaller droplets compared to those from clean bubbles (Fig. 1). These smaller and more numerous droplets can remain suspended in the air longer and have a higher probability of dispersal such as that required for airborne disease transmission [22]. In conclusion, bacterial secretions are ubiquitous and have many functions [41,47]. Here, we discovered and elucidated a previously unreported one: hv secreting them, microorganisms can manipulate the physics of thinning of a surface bubble to enhance their own water-to-air dispersal.

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- L. Bourouiba and J. W. M. Bush, Drops and bubbles in the environment, in *Handbook of Environmental Fluid Dynamics* (Taylor & Francis, Boca raton, 2013), Vol. 1, Chap. 32, pp. 427–439.
- [2] P. L. Walls, J. C. Bird, and L. Bourouiba, Moving with bubbles: A review of the interactions between bubbles and the microorganisms that surround them, Integr. Comp. Biol. 54, 1014 (2014).
- [3] L. T. Angenent, S. T. Kelley, A. St. Amand, N. R. Pace, and M. T. Hernandez, Molecular identification of potential pathogens in water and air of a hospital therapy pool, Proc. Natl. Acad. Sci. U.S.A. **102**, 4860 (2005).
- [4] P. A. Baron and K. Willeke, Respirable droplets from whirlpools: Measurements of size distribution and estimation of disease potential, Environ. Res. 39, 8 (1986).
- [5] J. Embil, P. Warren, M. Yakrus, R. Stark, S. Corne, D. Forrest, and E. Hershfield, Pulmonary illness associated with exposure to mycobacterium-avium complex in hot tub water: Hypersensitivity pneumonitis or infection?, Chest 111, 813–816 (1997).
- [6] E. L. Best, J. A. T. Sandoe, and M. H. Wilcox, Potential for aerosolization of Clostridium difficile after flushing toilets: The role of toilet lids in reducing environmental contamination risk, J. Hosp. Infect. 80, 1 (2012).
- [7] D. Johnson, R. Lynch, C. Marshall, K. Mead, and D. Hirst, Aerosol generation by modern flush toilets, Aerosol Sci. Technol. 47, 1047–1057 (2013).

- [8] G. Traverso, S. Laken, C.-C. Lu, R. Maa, R. Langer, and L. Bourouiba, Fluid fragmentation from hospital toilets, arXiv:1310.5511.
- [9] H. Bauer, M. Fuerhacker, F. Zibuschka, H. Schmid, and H. Puxbaum, Bacteria and fungi in aerosols generated by two different types of wastewater treatment plants, Water Resour. Res. 36, 3965 (2002).
- [10] K. T. Kiger and J. H. Duncan, Air-entrainment mechanisms in plunging jets and breaking waves, Annu. Rev. Fluid Mech. 44, 563 (2012).
- [11] D. C. Blanchard and A. H. Woodcock, Bubble formation and modification in the sea and its meteorological significance, Tellus 9, 145 (1957).
- [12] E. R. Lewis and S. E. Schwartz, Sea Salt Aerosol Production: Mechanisms, Methods, Measurements, and Models—A Critical Review (American geophysical union, Washington, D.C., 2004).
- [13] G. de Leeuw, E. L. Andreas, M. D. Anguelova, C. W. Fairall, E. R. Lewis, C. O'Dowd, M. Schulz, and S. E. Schwartz, Production flux of sea spray aerosol, Rev. Geophys. 49, 2010RG000349 (2011).
- [14] F. Veron, Ocean spray, Annu. Rev. Fluid Mech. 47, 507 (2015).
- [15] P. Renard, I. Canet, M. Sancelme, N. Wirgot, L. Deguillaume, and A.-M. Delort, Screening of cloud microorganisms isolated at the Puy de Dôme (France) station for the production of biosurfactants, Atmos. Chem. Phys. 16, 12347 (2016).
- [16] W. H. Horrocks, Experiments made to determine the conditions under which "specific" bacteria derived from sewage may be present in the air of ventilating pipes, drains, inspection chambers, and sewers, Proc. R. Soc. B 28, 176 (1907).
- [17] D. C. Blanchard, The ejection of drops from the sea and their enrichment with bacteria and other materials: A review, Estuaries 12, 127 (1989).
- [18] A. H. Woodcock, Note concerning human respiratory irritation associated with high concentrations of plankton and mass mortality of marine organisms, J. Mar. Res. 7, 56 (1948).
- [19] A. H. Woodcock, Bursting bubbles and air pollution, Sewage Ind. Wastes 27, 1189 (1955).
- [20] Y. S. Cheng, Y. Zhou, C. M. Irvin, R. H. Pierce, J. Naar, L. C. Backer, L. E. Fleming, B. Kirkpatrick, and D. G. Baden, Characterization of marine aerosol for assessment of human exposure to brevetoxins, Environ. Health Perspect. **113**, 638 (2005).
- [21] L. E. Fleming *et al.*, Review of Florida red tide and human health effects, Harmful algae **10**, 224 (2011).
- [22] W. F. Wells, Airborne Contagion and Air Hygiene. An Ecological Study of Droplet Infections (Harvard University Press, Cambridge, 1955).
- [23] F. C. Lessa *et al.*, Burden of Clostridium difficile infection in the United States, N. Engl. J. Med. **372**, 825 (2015).
- [24] A. J. Prussin, D. O Schwake, and L. C. Marr, Ten questions concerning the aerosolization and transmission of Legionella in the built environment, Building Services and Environmental Engineer 123, 684 (2017).
- [25] B. C. Parker, M. A. Ford, H. Gruft, and J. O. Falkinham III, Epidemiology of infection by nontuberculous mycobacteria

IV. preferential aerosolization of Mycobacterium intracellulare from natural waters, Am. Rev. Respir. Dis. **128**, 652 (1983).

- [26] J. O. Falkinham III, Mycobacterial aerosols and respiratory disease, Emerging Infect. Dis. 9, 763 (2003).
- [27] E. R. Baylor, M. B. Baylor, D. C. Blanchard, L. D. Syzdek, and C. Appel, Virus transfer from surf to wind, Science 198, 575–580 (1977).
- [28] S. Sharoni, M. Trainic, D. Schatz, Y. Lehahn, M. J. Flores, K. D. Bidle, S. Ben-Dor, Y. Rudich, I. Koren, and A. Vardi, Infection of phytoplankton by aerosolized marine viruses, Proc. Natl. Acad. Sci. U.S.A. **112**, 6643–6647 (2015).
- [29] H. Lhuissier and E. Villermaux, Bursting bubble aerosols, J. Fluid Mech. 696, 5–44 (2012).
- [30] S. Poulain, E. Villermaux, and L. Bourouiba, Ageing and burst of surface bubbles, J. Fluid Mech. 851, 636 (2018).
- [31] L. Champougny, M. Roché, W. Drenckhan, and E. Rio, Life and death of not so "bare" bubbles, Soft Matter 12, 5276 (2016).
- [32] R. L. Modini, L. M. Russell, G. B. Deane, and M. D. Stokes, Effect of soluble surfactant on bubble persistence and bubble-produced aerosol particles, J. Geophys. Res.: Atmos. 118, 1388 (2013).
- [33] W.-R. Ke, Y.-M. Kuo, C.-W. Lin, S.-H. Huang, and C.-C. Chen, Characterization of aerosol emissions from single bubble bursting, J. Aerosol Sci. 109, 1 (2017).
- [34] G. Taylor, The dynamics of thin sheets of fluid. III. Disintegration of fluid sheets, Proc. R. Soc. A 253, 313 (1959).
- [35] F. E. C. Culick, Comments on a ruptured soap film, J. Appl. Phys. **31**, 1128 (1960).
- [36] T. Maniatis, E. F. Fritsch, and J. Sambrook, *Molecular cloning: a laboratory manual* (Cold spring harbor laboratory, Cold Spring Harbor, NY, 1982), Vol. 545.
- [37] We grow *E. coli* aerobically for 14 hours in a shaking incubator at 37 °C in M9 minimal medium supplemented

with 20% glucose. To obtain the concentrations $c_1 = 3 \times 10^7$ and $c_2 = 6 \times 10^6$ cells mL⁻¹, we dilute their culture 20 and 100 times, respectively, in deionized water.

- [38] J. M. Rangel, P. H. Sparling, C. Crowe, P. M. Griffin, and D. L. Swerdlow, Epidemiology of Escherichia coli O157: H7 outbreaks, United States, 1982–2002, Emerging Infect. Dis. 11, 603 (2005).
- [39] J. K. Varma *et al.*, An outbreak of Escherichia coli O157 infection following exposure to a contaminated building, J. Am. Med. Assoc. **290**, 2709 (2003).
- [40] K. E. Eboigbodin and C. A. Biggs, Characterization of the extracellular polymeric substances produced by Escherichia coli using infrared spectroscopic, proteomic, and aggregation studies, Biomacromolecules 9, 686 (2008).
- [41] T. R. Neu and J. R. Lawrence, The extracellular matrix an intractable part of biolm systems, in *The Perfect Slime: Microbial Extracellular Polymeric Substances* (*EPS*), edited by H.-S. Flemming, T. R. Neu, and J. Wingender (IWA Publishing, London, 2017) Chap. 3, pp. 25–60.
- [42] N. A. Fuchs, Evaporation and Droplet Growth in Gaseous Media (Pergamon, New York, N.Y., 1959).
- [43] D. Talmud and S. Suchowolskaju, Stabilität des elementaren schaumes, Z. Phys. Chem. 154A, 277 (1931).
- [44] K. J. Mysels, K. Shinoda, and S. Frankel, Soap Films: Studies of Their Thinning and a Bibliography (Pergamon, New York, 1959).
- [45] J. N. Israelachvili, *Intermolecular and Surface Forces* (Academic press, Waltham, MA, 2011) 3rd ed..
- [46] G. Debrégeas, P.-G. de Gennes, and F. Brochard-Wyart, The life and death of "bare" viscous bubbles, Science 279, 1704–1707 (1998).
- [47] A. W. Decho and T. Gutierrez, Microbial extracellular polymeric substances (EPSs) in ocean systems, Front. Microbiol. 8, 922 (2017).