Mechanisms of Action of Mixed Solid−Liquid Antifoams: 3. Exhaustion and Reactivation

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A major problem in the practical application of antifoams (substances used to avoid undesirable foam) is the gradual loss of their activity in the course of foam destruction. Several experimental methods are combined in the present study to reveal the origin of this phenomenon, usually termed as the antifoam "exhaustion" or "deactivation". A typical mixed antifoam, comprising silicone oil and hydrophobized silica aggregates of fractal shape and micrometer size, has been studied in solutions of the anionic surfactant sodium dioctylsulfosuccinate (AOT). The results unambiguously show that the exhaustion in this system is caused by two interrelated processes: (1) segregation of oil and silica into two distinct populations of antifoam globules (silica-free and silica-enriched), both of them being rather inactive; (2) disappearance of the spread oil layer from the solution surface. The oil droplets deprived of silica, which appear in process 1, are unable to enter the air−water interface and to destroy the foam lamellae. On the other side, the antifoam globules enriched in silica trap some oil, which is not readily available for spreading on the solution surface. As a result, the spread layer of silicone oil gradually disappears from the solution surface (process 2) due to oil emulsification in the moment of foam film rupture. Ultimately, both types of globules, silica-enriched and silica-free, become unable to destroy the foam films, and the antifoam transforms into an inactive (exhausted) state. The introduction of a new portion of oil (without any silica) on the surface of an exhausted solution results in a perfect restoration of the antifoam activity−reactivation of the antifoam. The experiments show that the reactivation process is due to restoration of the spread oil layer and to rearrangement of the solid particles from the exhausted antifoam with freshly added oil into new antifoam globules having optimal silica concentration. The results provide deeper insight into the mechanisms of antifoam action and suggest ways for improving the antifoam efficiency and durability.

Introduction

Antifoams are important components of many commercial products, like detergents, paints, pharmaceuticals, and others.[1] A typical antifoam for detergency consists of an oil (hydrocarbon or poly(dimethylsiloxane)), dispersed hydrophobic solid particles, or a mixture of both.[2,3] It has been demonstrated[2] that mixed solid−liquid antifoams are usually much more efficient than antifoams containing the individual components (oil or solid particles) taken separately. Two mechanisms of foam film destruction by oil-containing antifoams are usually discussed in the literature: spreading-fluid entrainment[4−7] and bridging−dewetting.[2,5−11] A comprehensive analytical review on these mechanisms has been presented by Garrett.[2]

Recently, microscopic observations revealed that another mechanism, called "bridging-stretching",[12] is responsible for the foam film destruction by typical antifoams

comprising silicone oil and hydrophobized silica particles. Briefly, unstable oil bridges are formed in the foam film, which stretch with time due to uncompensated capillary pressures at the oil−air and oil−water interfaces, and eventually rupture the film (see Figure 1). These oil bridges are formed from either initially emulsified antifoam globules, or oil lenses floating on the air−water interface. It was shown[12] that the presence of an oil layer, prespread over the foam film surfaces and having a thickness of several nanometers, is very important, because this layer substantially facilitates the formation of unstable oil bridges and the film rupture. The theoretical analyzis[13] showed that the oil bridges are unstable only if their volume is above a certain critical value, which depends on the thickness of the foam film and on the three-phase contact angles. The effect of the spread oil was explained[13] by an accumulation of oil into the bridge (from the spread layer), which leads to an actual increase of the bridge size.

The main role of the solid particles in mixed antifoams is to destabilize the oil−water−air film, thus facilitating the drop entry (pin-effect).[2,7−9,14−16] Another role of the

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(1) *Defoaming: Theory and Industrial Applications*; Garrett, P. R., Ed.; Marcel Dekker: New York, 1993; Chapters 2−8.

(2) Garrett, P. R. In *Defoaming: Theory and Industrial Applications*; Garrett, P. R., Ed.; Marcel Dekker: New York, 1993; Chapter 1.

(3) Exerova, D.; Kruglyakov, P. M. *Foams and Foam Films*; Elsevier: Amsterdam, 1998; Chapter 9.

(4) Ross, S. *J. Phys. Colloid Chem.* **1950**, *54*, 429.

(5) Aveyard, R.; Binks, B. P.; Fletcher, P. D. I.; Peck, T.-G.; Garrett, P. R. *J. Chem. Soc., Faraday Trans.* **1993**, *89*, 4313.

(6) Aveyard, R.; Binks, B. P.; Fletcher, P. D. I.; Peck, T. G.; Rutherford, C. E. *Adv. Colloid Interface Sci.* **1994**, *48*, 93.

(7) Bergeron, V.; Cooper, P.; Fischer, C.; Giermanska-Kahn, J.; Langevin, D.; Pouchelon, A. *Colloids Surf. A: Physicochem. Eng. Asp.* **1997**, *122*, 103.

(8) Garrett, P. R.; Moor, P. R. *J. Colloid Interface Sci.* **1993**, *159*, 214.

(9) Garrett, P. R.; Davis, J.; Rendall, H. M. *Colloids Surf. A: Physicochem. Eng. Asp.* **1994**, *85*, 159.

(10) Koczo, K.; Koczone, J. K.; Wasan, D. T. *J. Colloid Interface Sci.* **1994**, *166*, 225.
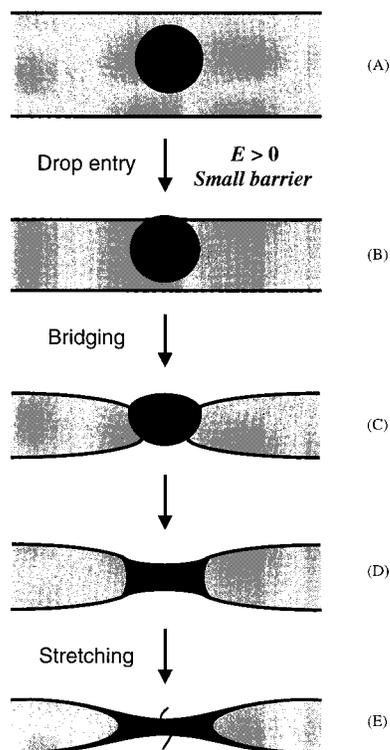
(11) Aveyard, R.; Cooper, P.; Fletcher, P. D. I.; Rutherford, C. E. *Langmuir* **1993**, *9*, 604.

(12) Denkov, N. D.; Cooper, P.; Martin, J.-Y. *Langmuir* **1999**, *15*, 8514.

(13) Denkov, N. D. *Langmuir* **1999**, *15*, 8530.

(14) Frye, G. C.; Berg, J. C. *J. Colloid Interface Sci.* **1989**, *130*, 54.

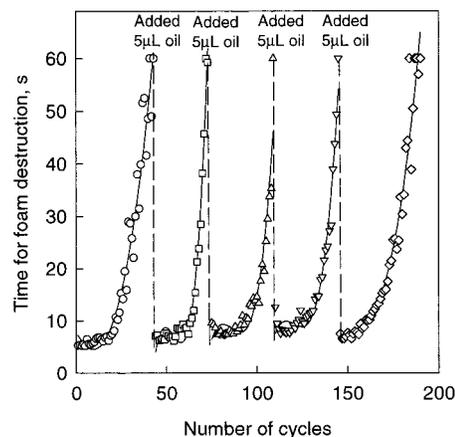(15) Aronson, M. *Langmuir* **1986**, *2*, 653.

Figure 1. Bridging—stretching mechanism of foam film rupture by antifoam globules.[12,13] After an oil bridge is formed (A—C), it stretches due to uncompensated capillary pressures at the oil—water and oil—air interfaces (C—E). Finally, the oil bridge ruptures in its thinnest central region (the vertical wavy line in E).

Figure 2. Consecutive phases of exhaustion/reactivation of compound A, 0.005 wt %, in 11.3 mM AOT solution (see Experimental section for details). An initially active antifoam (defoaming time $\tau_D \approx 5$ s) gradually loses its activity with the number of shaking cycles; $\tau_D \to 60$ s after 43 shaking cycles and the antifoam is considered as being exhausted. The introduction of a silicone oil (5 μL) results in a complete restoration of the antifoam activity—τ_D falls down again to 5 s. Five consecutive exhaustion profiles, along with four reactivation events are shown.

solid particles is to increase the penetration depth of the oil lenses, floating on the film surfaces, which in turn facilitates the oil bridge formation.[13,14] On the other side, the presence of solid particles in an already formed oil bridge could lead to its relative stabilization, because an excess of oil is required for the formation of an unstable bridge.[13]

In the present study, we continue the examination of the mechanisms of antifoam action in the experimental system explored in ref 12: silica—silicone oil antifoams in solutions of the anionic surfactant sodium dioctylsulfosuccinate (hereafter, denoted as AOT). More specifically, we study the so-called process of "exhaustion"[7,9,10,12,17–19] (deactivation) of the antifoams in the course of their action. This process is illustrated in Figure 2, which shows the time for destruction of a foam column produced in a standard shake test (see the section Methods for details), as a function of the number of shaking cycles. The initial high activity of the antifoam (foam destruction within 6—7 s) is preserved almost constant within the first 15—20 cycles, followed by a rapid increase of the foam destruction time (after 45 cycles it becomes longer than 60 s, which is considered as an upper limit of the acceptable antifoam activity in this test)—the antifoam has been exhausted.

The addition of a new portion of oil, deprived of silica particles, into an already exhausted foaming solution leads

to complete restoration of the antifoam activity—a "reactivation" is observed (Figure 2). The oil itself has no activity at these conditions in the absence of silica particles, which means that the process of foam destruction after reactivation certainly involves the solid particles which have been introduced with the first portion of mixed antifoam. The subsequent foam generation/destruction cycles lead to a second exhaustion series and the shape of the respective curve is almost the same as that of the first one. These consecutive periods of exhaustion—reactivation can be repeated several times (Figure 2).

Several hypotheses have been proposed in the literature to explain the antifoam exhaustion. Kulkarni et al.[17] suggested that the exhaustion is due to gradual extraction of silica particles from the antifoam globules into the aqueous phase. This mechanism implies that initially hydrophobic solid particles become hydrophilic (and hence inactive) due to adsorption of surfactant on their surface in the moment of globule entry, when the oil spreads on the air—water surface and presumably the solid particles are deprived of their oil coat. This explanation was criticized by Garrett et al.,[9] and Koczo et al.,[10] who showed that the addition of fresh oil to suspensions of hydrophobic particles (which are rather inactive as antifoam without oil) lead to significant increase of the antifoam efficiency— the latter became comparable to the efficiency of premixed oil and silica particles. These results indicated that the explanation of Kulkarni et al.[17] could not be applied to the systems studied in refs 9 and 10. Instead, Koczo et al.[10] suggested that the main reason for the antifoam exhaustion was the observed reduction of the globule size from 2 to 30 μm down to 1—3 μm. On the basis of the mechanism of foam destruction suggested by these authors (bridging at the Plateau borders), the antifoam deactivation was explained[10] as a result of the reduced probability to trap smaller droplets in the Plateau borders.

Bergeron et al.[7] also observed a reduction of the antifoam globules size down to ca. 8 μm during the foaming process. The authors explained the effect of the reduced globule size by an increase of the drainage time of the foam films before bridging and film destruction to occur and by the reduced probability for rupture of the smaller oil—water—

(16) Aveyard, R.; Clint, J. H. *J. Chem. Soc., Faraday Trans.* **1995**, *91*, 2681.

(17) Kulkarni, R. D.; Goddard, E. D.; Kanner, B. *Ind. Eng. Chem., Fundam.* **1977**, *16*, 472.

(18) Pouchelon, A.; Araud, A. *J. Dispersion Sci. Technol.* **1993**, *14*, 447.

(19) Racz, G.; Koczo, K.; Wasan, D. T. *J. Colloid Interface Sci.* **1996**, *181*, 124.

air films formed between the antifoam globules and the foam film surfaces.

A different explanation was given by Racz et al.,[19] who suggested that the foam films are destroyed mainly by spread oil layer, possibly containing solid particles. These authors considered the emulsification of the spread oil in the moment of film rupture as the main reason for the loss of antifoam efficiency. By surface tension measurements Racz et al.[19] found out that the loss of antifoam activity correlated well with the moment of monolayer coverage (0.7 mg/m²) of the solution surface by silicone oil. Many commercial antifoams, however, are produced as emulsions; i.e., the antifoam is in the form of droplets even in the initial active period. Therefore, the hypothesis of Racz et al.,[19] cannot be directly applied for explaining the exhaustion of such pre-emulsified antifoams.

Pouchelon and Araud[18] observed macroscopic white agglomerates in exhausted, highly concentrated solutions containing 4.5 wt % AOT and 0.1 wt % silicone-based antifoam. Infrared analysis of these agglomerates revealed that the silica concentration had raised from 2.5 wt % in the initial antifoam up to 17 wt % in the white agglomerates. On the basis of this observation, the authors suggested that the accumulation of silica into dense oil−silica agglomerates, which are rather inactive, may be the cause for the antifoam deactivation.

The main goal of the present study is to obtain a detailed, unambiguous information about the actual mechanism of antifoam exhaustion in the particular system under investigation. To achieve this aim, we formulated in advance all hypotheses we could envisage and designed a set of complementary experiments to verify them. The following possible explanations have been examined: The exhaustion is due to (1) reduction of the average size of the antifoam globules, (2) emulsification of the spread oil layer, (3) changes in the properties of the oil during its contact with the surfactant solution (4) changes in the properties of the solid particles (e.g., the three-phase contact angles), or (5) segregation of oil and silica.

We could say in advance that our results have revealed the antifoam exhaustion as a combination of two closely interrelated processes: (1) segregation of the silicone oil and silica into two distinct populations of antifoam globules (silica-free and silica-enriched); (2) disappearance of the spread oil layer from the solution surface.

The article is organized as follows: the Experimental Section describes the used materials and methods; the main observations are presented in the Results and analyzed from the viewpoint of different hypotheses in the Discussion; a summary of the Conclusions is outlined at the end.

Experimental Details

Materials. Sodium dioctylsulfosuccinate, $C_{20}H_{37}O_7SNa$ (AOT), is used as a surfactant (Sigma Catalog No D-0885). Its concentration in the working solutions is 10 mM (unless another value is specified), which is about 3.5 times the critical micellar concentration, cmc = 2.8 mM. All solutions are prepared with doubly distilled water.

Three antifoam substances are studied:

(1) Poly(dimethylsiloxane) (PDMS) Oil. This has a dynamic viscosity of 1000 mPa·s, which is produced by Rhodia Silicones Europe (Saint Fons, France) under a commercial name 47V1000. In some of the experiments the oil is "pre-equilibrated" with the surfactant solution−1 mL of silicone oil is placed in glass cylinder containing 100 mL of the surfactant solution. The cylinder is shaken vigorously by hand for several minutes to emulsify the oil. The obtained emulsion is shaken mildly for 3 days to achieve complete equilibrium between the oil and the surfactant solution.

Finally, the emulsion is centrifuged to separate the oil as a macrophase, which is used in some of the experiments.

(2) PDMS Oil Containing 4.2 wt % of Hydrophobized Silica Particles of Pyrogenic Origin (Fumed Silica). The silica particles are produced by flame hydrolysis of silicone tertachloride (Degussa AG). In this process primary silica spheres (12 nm in diameter) are produced, which partially fuse with one another forming submicrometer-sized, branched aggregates.[20] The dispersion of silica aggregates in silicone oil leads to formation of larger entities (usually called agglomerates), which have a fractal structure and rather broad size distribution−from 0.1 to 5 μm. Hereafter this composition is labeled as compound A.

Several other compounds (mixtures of silicone oil and hydrophobized silica) containing 7, 10, 13, and 16 wt % silica, respectively, are used to study the rate of oil spreading on the surface of the surfactant solution, as a function of the silica concentration.

(3) Stable 10 wt % Stock Emulsion of Compound A. This is further diluted to the desired final concentration in the surfactant solution. The stock emulsion is stabilized by two nonionic surfactants (sorbitan monostearate−Span 60, and ethoxylate of stearic acid with 40 ethoxy groups−stearyl-EO₄₀). Microscope observations show that this emulsion is relatively polydisperse with drop diameters ranging from ca. 1 to 10 μm. This emulsion is denoted hereafter as emulsion A.

Compound A and emulsion A closely mimic the typical silicone-based commercial antifoams. The antifoam concentration in the working solutions is 0.005, 0.01, or 0.02 wt % (defined as the mass of silicone oil over the mass of the working solution), which is a typical concentration range for mixed silica−silicone antifoams.

Methods. *Foam Stability Evaluation.* The foam stability is evaluated by using an automated shake test (AST): 100 mL of the surfactant solution is poured in a standard 250 mL glass jar; the antifoam is then added by commercial micropipet M800 (Nichiryo Co., Tokyo, Japan), which is specially designed for small volumes of viscous fluids. The jar is shaken in a series of cycles, and the time for foam destruction (defoaming time, τ_D) is measured as a function of the cycle number. In a given cycle, the jar is first shaken for 10 s by an automated Prolabo Oscill 8 shaker (frequency 250 min⁻¹, amplitude 5 cm), followed by a 60 s period, during which the solution remains quiescent and the defoaming time is recorded. The moment of foam destruction is detected automatically by fiber optic detectors, which register the light reflection by clean (devoid of bubbles) solution−air interface. These cycles are repeated until τ_D exceeds 60 s. A small sample (ca. 2−3 mL) of the surfactant solution is taken after some of the shaking cycles for light scattering determination of the antifoam globule size.

Dynamic Light Scattering (DLS) and Optical Microscopy. The variation of the globule size with the antifoam exhaustion and after reactivation is studied in these experiments. A Malvern 4700C light scattering system (Malvern Instruments), equipped with an argon laser operating at 488 nm light wavelength and with a 7032 CE 8-bit correlator, is used. This equipment measures the diffusion coefficient of the dispersed antifoam globules. The size distribution of the globules is calculated from the diffusion coefficients by using the Stokes−Einstein relation

$$d = \frac{kT}{3\pi\eta D} \tag{1}$$

where d is the particle diameter, kT is the thermal energy, η is the dynamic viscosity of the disperse medium, and D is the measured diffusion coefficient. The method is applicable to particles of size varying between ca. 5 nm and 5 μm.

Since the antifoam globules in the studied samples exhibit a very broad size distribution, the value of the mean size for a given sample depends very much on the employed procedure of averaging: by particle volume, by particle number, or by scattered light intensity. In all figures below the volume average diameter, $\langle d \rangle_v$, is presented, because this value is determined relatively

(20) Degussa Technical Bulletin No. 42 (Synthetic Silicas for Defoamers), 3rd ed.; Degussa AG: Frankfurt, Germany, 1988.

reliably by DLS and because it represents the typical size of the globules containing most of the dispersed oil.

DLS does not provide a complete description of the globule size distribution, because the largest globules (of size around and above ca. 5 μm) sediment rapidly in the experimental cuvette and are not detected by DLS equipment. That is why additional microscope observations, which cover the size range above ca. 1 μm, are performed with the same samples. For the optical observations we use a microscope Axioplan (Zeiss, Jena, Germany) in transmitted light and a long distance objective $\times$32. The size of more than 800 globules is measured in each sample. Two characteristics of the dispersed globules are determined: (1) the fraction of visible globules whose diameter is above 2 μm and (2) the mean size of the globules, which are larger than 2 μm. Therefore, DLS and optical microscopy complement each other by covering the whole range of particle sizes, which is of interest in the present study.

Electron Cryomicroscopy. Electron cryomicroscopy of frozen (vitrified) specimens[21–24] is used to compare the structure of the antifoam globules in fresh and exhausted emulsion A. In particular, the cryomicroscopy allows one to check how the silica particles are distributed among the antifoam globules.

The following procedure for preparation of vitrified samples is employed.[23] About 5 μL of the studied emulsion is placed on a copper specimen grid covered by a holey carbon supporting layer, which is prehydrophilized by glow discharge in air. The excess of liquid is removed from the grid by filter paper, to reduce the thickness of the liquid film and to obtain sufficient electron transparency of the final frozen specimen. The sample is vitrified by plunging into a supercooled liquid ethane of temperature -185 °C. An ultrarapid freezing (vitrification) of water in the sample results in a very good preservation of the structure of the dispersed antifoam globules. The vitrified sample is transferred by a cryogenic specimen holder into a transmission electron microscope (JEM-1200 EX JEOL equipped with anticontaminator; acceleration voltage 120 kV) for observation at low temperature. The micrographs are taken at -190 °C by using the low-dose system of the microscope to reduce the exposure time and the ensuing sample damage by the electron beam.

Optical Observation of Foam Films. Millimeter-sized foam films are observed in reflected monochromatic light by using the method of Scheludko and Exerova.[25,26] A foam film is formed from a biconcave drop placed in a short capillary (internal diameter 2.5 mm, height 4 mm in our experiments) by sucking out liquid through a side orifice. Fiber optic illumination of the film and a long-focus lens (CTL-6, Tokyo Electronic Industry Co., Ltd.; magnification $\times$6, working distance 39 mm) attached to a CCD camera (Panasonic WV-CD20) are used for these observations—see refs 12 and 24 for details. The interference of light reflected from the upper and lower surfaces of the foam film leads to the appearance of dark and bright interference fringes, each of them corresponding to a given film thickness. The difference Δh in the film thickness between two neighboring dark (or two neighboring bright) fringes is equal to

$$\Delta h = \lambda/2n \approx 203 \text{ nm} \quad (2)$$

where $\lambda \approx 540$ nm is the wavelength of the illuminating light and $n = 1.33$ is the refractive index of the surfactant solution. One can easily distinguish changes in the film thickness on the order of $\Delta h/4 \approx 50$ nm (bright to gray, gray to dark, and so on). A major advantage of the Scheludko cell is that experiments can be performed with actual antifoams, dispersed into micrometer-sized globules or lenses; that is, the films in the Scheludko cell closely mimic the behavior of small films in real foams.

Surface Tension Measurements and Oil-Spreading Experiments. The surface tension of surfactant solutions (with and without antifoam) is measured by Wilhelmy plate method using a Kruss K12 tensiometer and a platinum plate. Before each measurement the plate is cleaned by immersion in hydrofluoric acid and by heating it in a flame.

The spreading rate of oil originating from a compound (mixture of oil and silica) is measured as a function of silica concentration. Glass Petri dish of diameter 20 cm and depth 2 cm is filled with surfactant solution, and trace particles (hydrophobized silica) are sprinkled over the solution surface. No change of the surface tension is detected after the trace particles have been dispersed, which indicates that no surface active contamination is generated by them. A thin glass rod, whose tip has been soaked by tested oil or compound, is gently placed in contact with the solution surface by using a micrometer drive device. The radial motion of the trace particles, indicating the front of the spreading precursor oil film, is observed and recorded by means of a CCD camera connected to a video recorder and monitor. The video records are afterward processed and the time period needed for oil spreading up to 5 cm radial distance from the oil source is measured. At least four independent experiments are performed with each of the tested oils or compounds. All experiments are carried out at ambient temperature (23 ± 1 °C).

Ellipsometry. The thickness of the precursor oil layer which spreads upon addition of silicone oil or compound on the surface of AOT solution is measured by ellipsometry. The setup is based on a commercial null-type ellipsometer (LEF 3M, Novosibirsk, Russia), which has been modified by introducing an additional rotating analyzer in the optical system.[27] The light source is a He−Ne laser ($\lambda = 632.8$ nm) with diameter of the beam about 2 mm. The angle of incidence of the laser beam is 50 degrees. The experimental system is driven by computer, and the raw data are instantaneously recomputed to provide the values of the ellipsometric angles, ψ and Δ, which are stored in 0.1 s intervals. The variations of ψ and Δ are analyzed, and the characteristics of the spread layer (its thickness and refractive index) are calculated as a function of time.
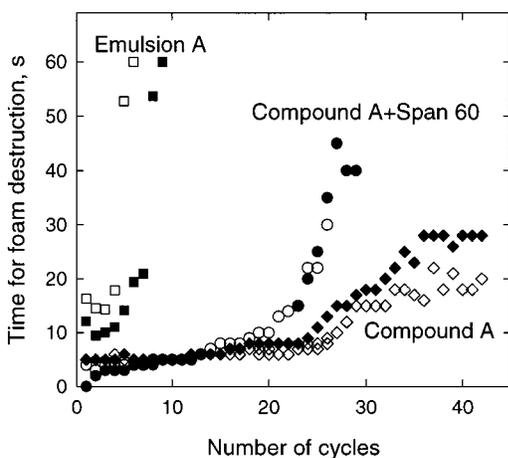
The studied surfactant solution is placed in a cylindrical glass container of both diameter and height equal to 9 cm, with the cylinder axis being perpendicular to the plane of incidence of the laser light (only half of the container is filled with solution). First, the solution surface is carefully cleaned by sucking with a glass pipet connected to a vacuum pump and the ellipsometric signal is registered for 15 min—the purity of the surface is controlled in this way, because the appearance of any contamination would be registered. Then, a small drop of silicone oil or compound is gently deposited on the solution surface, 4 cm away from the laser spot, by using a glass rod. Almost immediately the spreading of an oil precursor layer of nanometer thickness is detected; the spreading of a much thicker layer is registered several seconds later.

Surfactant solutions containing pre-emulsified antifoam (fresh or exhausted emulsion A) were studied in another set of experiments. The experimental cell is first filled with surfactant solution (no antifoam). The surface is cleaned by suction and the ellipsometric signal is recorded for 15 min. Afterward, a concentrated stock emulsion is injected beneath the solution surface, so that the antifoam concentration is adjusted to 0.03 wt %.

Results

Shake Test Experiments for Studying the Antifoam Exhaustion and Reactivation. *Comparison of Compound A and Emulsion A.* The antifoam activity (initial defoaming time) and durability (number of cycles leading to exhaustion) of compound A and emulsion A are compared in Figure 3. Note that the silicone oil devoid of silica has no antifoam activity in the studied AOT solution at the time scale of interest (60 s). One sees from Figure 3 that compound A is much more effective than emulsion A—the initial defoaming time for compound A is 3−4 s

(21) Dubochet, J.; Adrian, M.; Chang, J.-J.; Homo, J. C.; Lepault, J.; McDowall, A. W.; Schultz, P. *Q. Rev. Biophys.* **1988**, *21*, 129.

(22) Vinson, P. K.; Bellare, J. R.; Davis, H. T.; Miller, W. G.; Scriven, L. E. *J. Colloid Interface Sci.* **1991**, *142*, 74.

(23) Aguerre-Chariol, O.; Deruelle, M.; Boukhnikachvili, T.; In, M.; Shahidzadeh, N. *Proceedings of the Second World Congress on Emulsion*; 23−26 Sept 1997, Bordeaux, France; EDS Editeur: Paris, 1997; Vol. 4, p 165.

(24) Denkov, N. D.; Yoshimura, H.; Nagayama, K. *Ultramicroscopy* **1996**, *65*, 147.

(25) Scheludko, A.; Exerova, D. *Kolloid Z.* **1957**, *155*, 39.

(26) Scheludko, A. *Adv. Colloid Interface Sci.* **1967**, *1*, 391.

(27) Russev, S. C.; Argirov, T. V. *Rev. Sci. Instrum.* **1999**, *70*, 3077.

Figure 3. Comparison of the exhaustion profiles of different antifoams (0.01 wt %) in the shake test: (■, □) emulsion A; (◇) compound A; (◆) compound A in the presence of stearyl-EO$_{40}$; (○) compound A in the presence of Span 60; (●) compound A in the presence of stearyl-EO$_{40}$ and Span 60. Results from two independent tests of emulsion A are plotted to show the reproducibility of the test.

and it is very active even after 40 shaking cycles, while the initial defoaming time for emulsion A is about 9 s and the exhaustion occurs for less than 10 cycles. Additional experiments have shown that the concentration of emulsion A should be around 0.03 wt % to achieve an initial activity and durability comparable to those of 0.01 wt % compound A. Despite the observed quantitative differences in the antifoam efficiency, the basic mechanism of foam destruction by compound A and emulsion A is the same (bridging−stretching).[12] The general behavior of these two antifoams with respect to their exhaustion and reactivation is also very similar, which allows us to discuss their properties on a common basis.

To check whether the different activity of compound A and emulsion A is due to the presence of the nonionic surfactants Span 60 and stearyl-EO$_{40}$ (both used to fabricate emulsion A), we performed additional experiments with compound A in the presence of these two emulsifiers—see Figure 3. For this purpose 5 wt % of Span 60 (which is a water insoluble, solid substance at room temperature) was introduced into compound A under continuous stirring for 2 h at 70 °C (conditions similar to those during the emulsion preparation). Stearyl-EO$_{40}$ is a water-soluble surfactant, and it was directly added into the working surfactant solutions at a concentration corresponding to 0.01 wt % of emulsion A.

The shake test experiments show that the presence of stearyl-EO$_{40}$ in the foaming solution affects neither the initial activity nor the durability of compound A (Figure 3). Most probably, this surfactant is solubilized in the micelles of AOT, because the latter is in a big excess. On the other side, the introduction of Span 60 leads to notably faster exhaustion of compound A. Still, the initial activity and the durability of the compound containing Span 60 is much better compared to those of emulsion A. Therefore, one can conclude that the presence of the two nonionic surfactants in emulsion A is not the main reason for its reduced antifoam efficiency compared to compound A.

As discussed in ref 12, the higher initial activity of the compound at the same total concentration is probably due to the higher concentration of antifoam material deposited on the film surfaces (in the form of lenses), which leads to increased probability for bridge formation and film rupture. On the contrary, only those globules in emulsion A that enter the foam film surfaces may lead to film
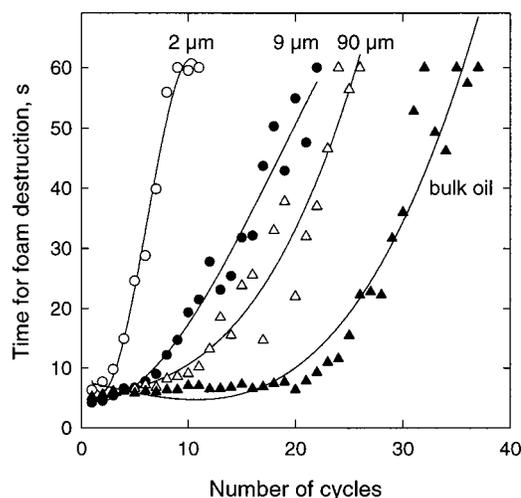

Figure 4. Reactivation of compound A (0.005 wt %) by addition of silicone oil (5 μL) in the form of an emulsion with different mean drop size.

rupture. The observations[12] showed that many of the antifoam globules in the experiments with emulsion A left the foam films without rupturing them. A similar explanation could be given to the better durability of compound A as compared to emulsion A. Since the compound is deposited on the solution surface before starting the foam generating cycles, practically the whole antifoam is involved in the foam destruction process. As discussed below, some of our observations indicate that part of the globules in fresh emulsion A might be inactive due to inappropriate silica/oil ratio or to the very small (submicrometer) size of the globules. In other words, part of the antifoam material might be deactivated during the fabrication process, in which the emulsion was produced from the compound.

Exhaustion after a Long Contact of the Antifoam with the Surfactant Solution. Some of the hypotheses outlined in the Introduction imply that the exhaustion might be caused by a gradual change of the properties of the oil and/or solid particles when the antifoam globules are placed in contact with the surfactant solution. To check this possibility we put 0.01 wt % of emulsion A in AOT solution and stirred mildly this emulsion without foam formation for 12 h. Then shake tests were carried out with this "pre-equilibrated" antifoam emulsion. No difference in the antifoam activity and durability of this sample was observed in comparison with "freshly" prepared working solution of emulsion A. Similar results were obtained with compound A as well. Therefore, the antifoam exhaustion in the studied systems is intrinsically related to the process of foam destruction and cannot be explained as a result of changes caused by the contact of the antifoam globules with the surfactant solution.

Reactivation. To reveal further details in the process of reactivation several modifications of the used procedure were explored. First, several emulsions of silicone oil of *different mean drop size* (2, 9, and 90 μm, respectively) were prepared. The reactivation of an already exhausted solution of compound A by these emulsions was studied and compared to the reactivation by bulk silicone oil—see Figure 4. The results demonstrated that the reactivation efficiency of the oil strongly depended on the mean size of the emulsion drops. The antifoam activity was completely recovered (defoaming time around 5 s) in all of the experiments; however, the rates of exhaustion of the reactivated samples were very different. The compound regenerated by addition of bulk oil was active for another
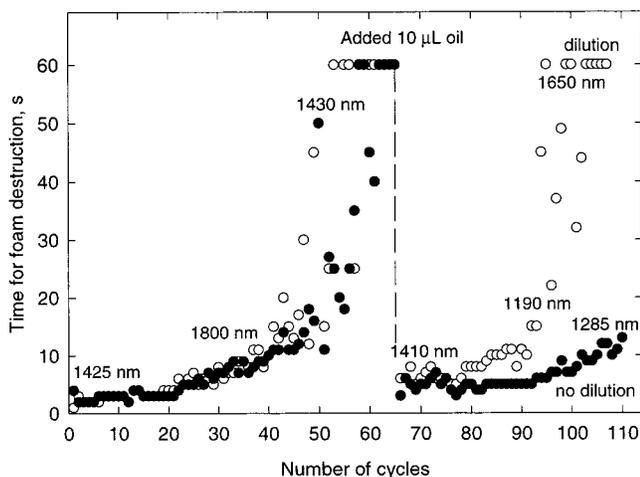
Figure 5. Exhaustion and reactivation of emulsion A (0.02 wt %). The exhaustion profiles after reactivation with (○) and without (●) dilution of the reactivated sample are compared—see the text. The numbers indicate the volume averaged diameter (measured by DLS) of the antifoam globules at different stages of the exhaustion/reactivation cycle. The reproducibility of the measured diameters within a given sample is relatively low (±20%), due to the wide size distribution of the antifoam globules; therefore, the observed variation in the mean globule size among the different samples is not statistically significant (see also Figure 6).

35 cycles, while the compound reactivated by an emulsion containing mainly small droplets (2 μm in diameter) lost its activity much more rapidly (9 cycles). The implications of these results, in connection with the other results from the present study, are discussed in the next section.

Another modification of the procedure was designed to check whether a "fresh" oil (that has not been in contact with surfactant solution) is needed for an efficient reactivation. For this purpose, a silicone oil was pre-equilibrated with AOT solution as explained in the Materials section. Two samples were prepared: (1) bulk oil, and (2) emulsion of mean drop size around 1 μm. The experiments showed that the reactivation by pre-equilibrated bulk oil (sample 1) was very efficient and similar to that with "fresh" oil. On the contrary, the reactivation by emulsified oil was very poor. One can conclude from these experiments that the pre-equilibration of the bulk silicone oil with the surfactant solution does not alter its reactivation capability. This is also another indication that the exhaustion process is not caused by changes in the silicone oil, as a result of the antifoam contact with the surfactant solution.

One might suggest that the reactivation is simply due to an increase of the total antifoam concentration in the surfactant solution. To check this possibility, we performed reactivation experiments, in which the addition of fresh oil was followed by dilution of the working solution by AOT solution devoid of antifoam. In this way the total antifoam concentration was recovered after the reactivation to its initial value, while keeping constant the surfactant concentration (see Figure 5). The results show that the exhaustion of the reactivated/diluted sample is faster compared to the exhaustion of the reactivated/nondiluted sample. Nevertheless, the results clearly demonstrate that a significant reactivation exists even after dilution of the sample; i.e., the reactivation is not a trivial effect caused by simple increase of the total antifoam concentration.

Size of the Antifoam Globules. In several studies,[7,10] a significant reduction of the antifoam globule size was

detected, which was correlated to the process of exhaustion. That is why we performed several sets of experiments aimed to clarify the relation between the globule size and the antifoam exhaustion and reactivation.

The volume average diameter of the antifoam globules was measured by DLS in the course of the exhaustion/reactivation experiment shown in Figure 5. Remarkably, we did not detect any significant change of the measured mean size either in the processes of exhaustion or in the process of reactivation for this system—see Figure 5. All values fell in the range 1.2−1.8 μm, and no detectable trend was observed upon exhaustion or reactivation. The other types of average diameters that could be extracted from the same DLS data (the number-average diameter and the so-called "z-average" diameter) remained also constant in the framework of the experimental reproducibility (±20%).

The microscope observations of antifoam globules in fresh and exhausted emulsion A also did not demonstrate any detectable reduction of the globule size upon exhaustion (Figure 6A,B)—many globules of sizes between 1 and 10 μm were seen in both samples (globules of diameter below 1 μm are not seen in the optical microscope). The fraction of visible globules, whose diameter was larger than 2 μm was about 70% in both cases; the mean diameter of these globules was 4.3 ± 2.8 μm in the fresh emulsion and 4.3 ± 2.3 μm in the exhausted emulsion. Notably, many of the large globules in fresh emulsion A appeared ribbed, while the largest globules in exhausted emulsion A were round in shape. The latter observation suggests that the largest globules in the exhausted emulsion probably contain no solid particles (as explained below, some fraction of the solid silica and Span particles is gathered upon the exhaustion process within white spongy macroagglomerates containing up to 15% solid).

Since compound A is introduced into the working solution in the form of macroscopic lenses, such an investigation of the drop size evolution during the exhaustion/reactivation cycle is not sensible. That is why we measured the globule size in exhausted solutions of compound A (0.02 wt %) when the whole compound had been already emulsified. The volume average globule diameter in these samples was measured by DLS to be about 1.7 ± 0.8 μm, which is similar to the globule size in fresh emulsion A (1.5 ± 0.5 μm). Again numerous globules of size between 1 and 10 μm were observed by optical microscopy in the sample of exhausted compound A (Figure 6C). The optical observations showed that about 60% of the visible globules were larger than 2 μm and the mean diameter of these globules was 3.7 ± 1.9 μm. In conclusion, no correlation between the globule size and the activity of the antifoam has been registered in the studied systems.

Additional experiments with AOT solutions containing emulsion A of much higher concentration (0.1 and 1 wt %) showed that the exhaustion of these samples, in terms of the used shake test and defoaming time, was indeed accompanied by some measurable reduction of the average globule size. As discussed in the following section, we do not exclude the possibility that the globule size reduction plays some role at higher antifoam concentration or in other systems (other antifoams and/or surfactants).[10,28]

Structure of the Antifoam Globules. The strong synergism of silica and silicone oil in their antifoam activity suggests that the presence of silica and its concentration in the mixed globules should be very important for the
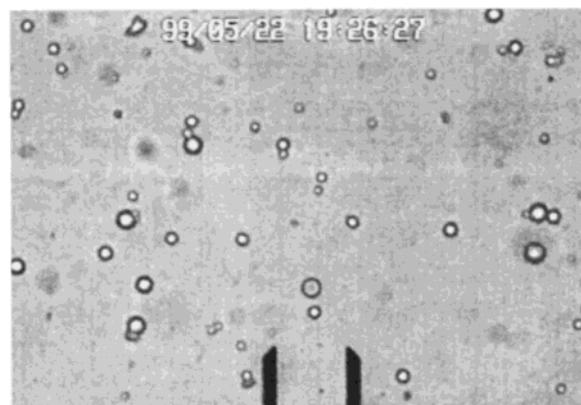
(28) Basheva, E.; Ganchev, D.; Denkov, N. D.; Kasuga, K.; Satoh, N.; Tsujii, K. *Langmuir* **2000**, *16*, 1000.
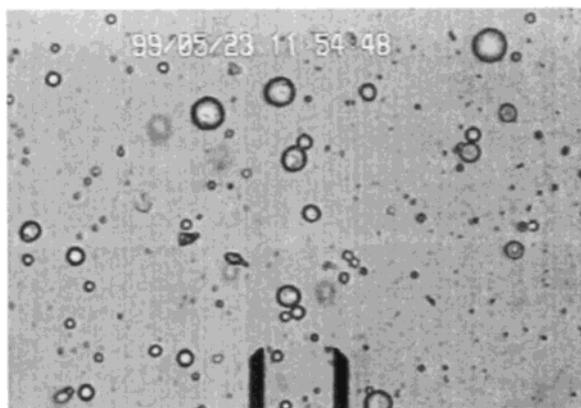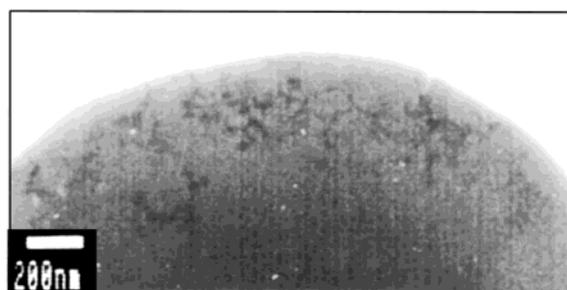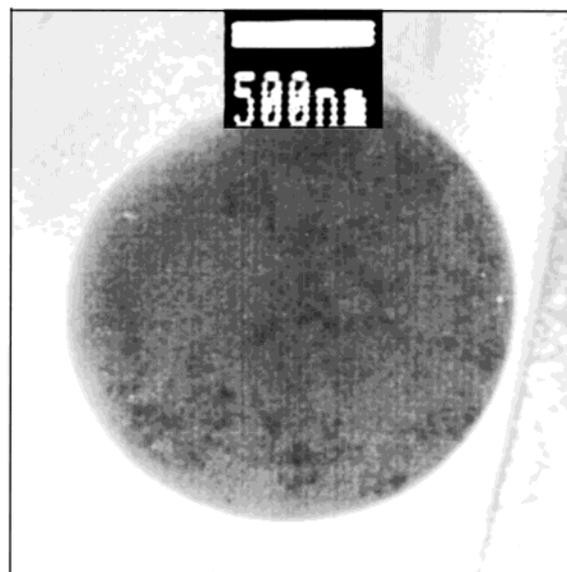
(A)
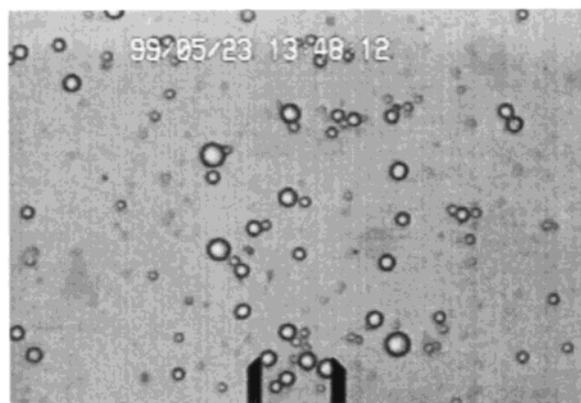
(B)

(C)

Figure 6. Antifoam globules as seen by optical microscopy: (a) fresh emulsion A; (B) exhausted emulsion A; (C) exhausted compound A. The bar is 31 μm.


Figure 7. Antifoam globules in fresh emulsion A observed by electron cryomicroscopy. Fractal silica aggregates are seen as dark dots in the oil phase.

overall process of foam destruction. Transmission electron cryomicroscopy is used to see what is the distribution of silica among the different globules in fresh and exhausted emulsion A. It is important to note that one can study only relatively small globules by this method, due to the low electron transparency of the vitrified aqueous layer, in which the globules are embedded. The transfer of these observations to the general population of globules should be made with care.

Two types of globules are observed in fresh emulsion A. The bigger globules (of diameter above ca. 300 nm) often contain some silica, which is located preferably at the drop periphery—see Figure 7. Similar structure of mixed globules was observed by Garrett et al.[9] in a mixed silica-hydrocarbon antifoam. One might expect that these globules are very effective as foam film breakers, because the silica facilitates drop entry, while the excess of oil could lead to formation of unstable bridges in the foam films. Smaller drops, which do not contain any silica are also observed in this sample. These small, silica-free droplets are produced during the fabrication of the batch sample of emulsion A from compound A—they should be completely inactive in the studied AOT solutions.

Not surprisingly, we observe notably larger number of silica-free droplets in the specimen of exhausted emulsion A—see Figure 8A,B. On the other side, some silica-rich antifoam globules are also found in this sample (Figure 8C). Such globules are probably also poor foam breakers in the absence of a prespread oil layer, because the fraction of oil within such a globule might be insufficient for the formation of unstable bridge in the foam film.[13]

Structure and Reactivation of the Large Agglomerates Observed in Exhausted Solutions. The exhaustion of an antifoam is often accompanied by the formation of millimeter-sized, white spongy agglomerates containing silica of relatively high concentration.[18] Although these macroagglomerates contain a significant fraction of silicone oil (above 80 wt %), they are inactive and do not destroy the foam. Several types of experiments are performed with these agglomerates to clarify their role in the exhaustion process.

Composition of the Macroagglomerates. Agglomerates formed in an exhausted solution of 1 wt % emulsion A were collected and analyzed by IR spectroscopy following the procedure by Pouchelon and Araud.[18] The silica
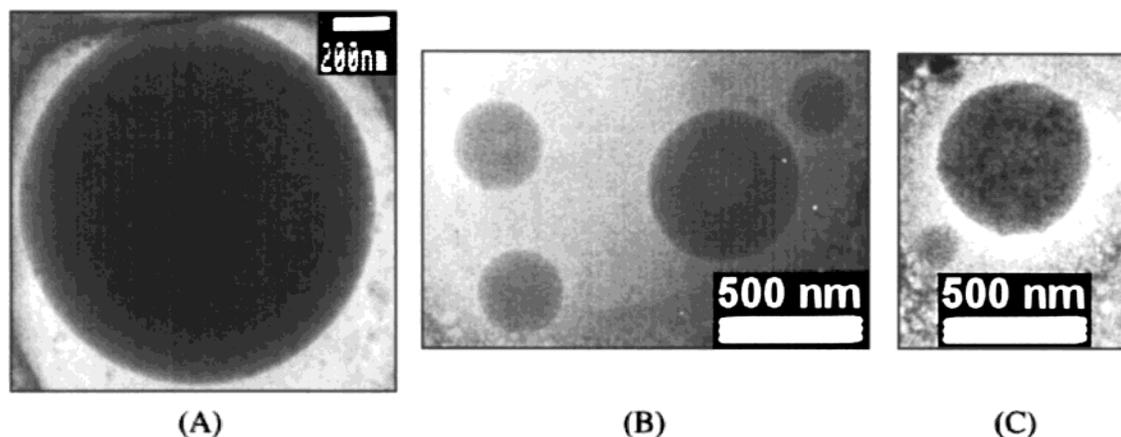
Figure 8. Droplets of silicone oil deprived of silica (A, B) and a silica-enriched globule (C) in exhausted emulsion A (electron cryomicroscopy).

concentration in these agglomerates was 14 ± 2 wt %, which is close to the result obtained in ref 18 (17 wt %). These agglomerates contained also Span 60 of concentration equivalent to the initial one in the stock emulsion A.

Reactivation Experiments. In a second series of experiments, we added silicone oil into an exhausted surfactant solution from which the macroscopic agglomerates were completely removed. The addition of oil led to a very efficient reactivation of the antifoam, which shows that enough number of silica particles had remained suspended in the exhausted solution. Therefore, one cannot explain the exhaustion process just by accumulation of silica into macroscopic agglomerates.

We found that the macroscopic agglomerates could be also reactivated. After exhaustion of a sample containing 0.1% emulsion A, we gathered the formed macroagglomerates and introduced 10 μg of them into 100 mL of AOT solution (this corresponds to 0.01 wt % of the total antifoam concentration). The macroagglomerates showed no antifoam activity. However, a consecutive addition of 5 μL fresh silicone oil resulted in a very efficient restoration of the antifoam activity. Most probably, the new silicone oil "diluted" the agglomerates, which lead to redistribution of silica and to formation of active antifoam globules of lower silica concentration.

Rate of Oil Spreading. The rate of oil spreading from a compound vs silica concentration is measured to evaluate how rapidly the silicone oil is released from the macro-agglomerates in the foam. The results for the time period needed for oil spreading at 5 cm radial distance from the oil source (a glass rod, whose tip is coated by a compound) are shown in Figure 9 for samples containing 4.2, 7, 10, 13, and 16 wt % of silica. The results show that silica of concentration below ca. 8 wt % in the compound does not influence significantly the rate of oil spreading. However, the oil originating from concentrated compounds (more than 10 wt % silica) spreads much slower.

This result means that the oil in concentrated compounds is not readily available for spreading. Indeed, the amount of oil needed to cover the solution surface in these experiments is only $\approx 1.5 \times 10^{-2}$ mm^3, while the compound deposited on the glass rod contains more than 1 mm^3 of oil. Therefore, the reduced rate of oil spreading is not caused by deficiency of oil. The most probable explanation of the reduced spreading rate at high silica concentrations is the strong interaction between silicone oil and silica.[29] When the silica concentration is below ca. 10 wt %, there
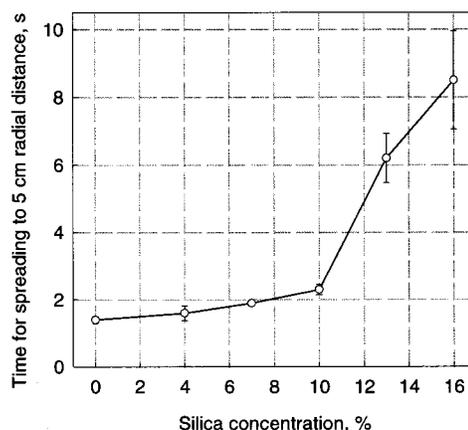
Figure 9. Time for oil spreading at 5 cm radial distance from the oil source (a compound containing oil and silica) as a function of silica concentration.

is a free silicone oil, which spreads without feeling the presence of silica. However, when the silica concentration is above 10 wt %, the silica—oil interaction affects the whole amount of oil present in the compound—the spreading oil should "overcome" the silica—oil binding force, which causes a significant deceleration of the spreading process.

Changes in the spread oil layer during antifoam exhaustion. *Surface Tension Measurements and Optical Observations.* As described earlier,[12] the surface tension of fresh solutions containing emulsion A or compound A is typically reduced by 2.5 to 3 mN/m compared to the surface tension of AOT solutions in the absence of antifoam. This reduction indicates the presence of a spread oil layer on the solution surface,[30] which appears as a result of two different processes. First, the compounds are usually deposited onto the solution surface where they form macroscopic lenses coexisting with a thin layer of spread oil. Similarly, when a pre-emulsified antifoam is used, some oil is transported from the surface of the concentrated batch emulsion by the used transferring device (e.g., the pipet).[12] This way of direct oil transfer from the surface of the "mother" emulsion onto the surface of the working solution can be eliminated by using the so-called "two-tips procedure" (TTP)[12]—see Table 1. Second, the coalescence of antifoam globules with the solution surface, which takes place during shelf storage of the antifoam emulsion and during foaming of the working solutions, also generates spread oil.

(29) Aranguren, M. I.; Mora, E.; Macosko, C. W. *J. Colloid Interface Sci.* **1997**, *195*, 329.

(30) Bergeron, V.; Langevin, D. *Macromolecules* **1996**, *29*, 306.

Table 1. Surface Tension of 10 mM AOT Solutions and Approximate Thickness of the Spread PDMS Layer in the Presence of Emulsion A

system	surface tension (mN/m)	$\Delta\sigma$ (mN/m)	layer thickness (nm)[a]
no antifoam	27.85 ± 0.05	0	0
0.01% fresh emulsion A (active)	$25.0-25.45$	$2.4-2.85$	>2
0.01% fresh emulsion A (active) loaded by TTP[b]	27.8 ± 0.05	≈ 0.05	<0.8
0.01% fresh emulsion A (active) loaded by TTP (12 h later)	$24.95-25.0$	≈ 2.9	>2
0.01% exhausted emulsion A (inactive)	27.7 ± 0.10	$0-0.3$	<0.8

[a] The layer thickness is estimated from the measured surface tensions and the data of Bergeron and Langevin.[30]   [b] TTP: Two-Tips Procedure,[12] which ensures solution surface free of oil (see the text).

To understand better the role of the spread oil layer in the exhaustion process we compared the surface tension of fresh and exhausted solutions and performed optical observations of their surfaces. The surface of solutions containing fresh compound A is covered by many lenses. The amount of antifoam on the surface and the size of the lenses gradually diminish with the number of foam production/destruction cycles—the antifoam is emulsified in the process of foam destruction. At a given stage of the exhaustion process (when the antifoam is still active) the macroscopic lenses, seen by naked eye, completely disappear from the solution surface. After several shake cycles, a rapid loss of antifoam activity is observed. Notably, the surface tension of the working solution remains almost constant while the antifoam is still active. The moment of antifoam exhaustion correlates very well with the moment when the surface tension of the solution starts to increase up to the value corresponding to pure AOT solution.

The surface of solutions containing 0.01 wt % fresh emulsion A is free from macroscopic lenses visible by the naked eye. Under a microscope one can occasionally see small oil lenses, possibly containing silica. The absence of macroscopic lenses in this system is not surprising, because the prevailing fraction of the antifoam is emulsified in advance. The surface tension of the working solutions remains low (Table 1) while emulsion A is active in the shake tests, indicating in this way the presence of a spread oil layer of nanometer thickness. The moment of antifoam exhaustion again correlates very well with the moment when the surface tension of the solutions starts to increase. The surface tension of solutions containing exhausted emulsion A is equal to that of pure AOT solution (see Table 1).

In conclusion, the above observations clearly show that the exhaustion of compound A and emulsion A correlates with the disappearance of the spread oil layer from the surface of the working solutions.

Ellipsometry. The ellipsometric experiments show that a precursor oil layer of thickness 2 ± 0.5 nm spreads almost immediately after the deposition of compound A on the solution surface. This value is slightly lower compared to the thickness that can be calculated from the equilibrium experiments described by Bergeron and Langevin[30] (2.6 mg/m^2, which corresponds to a thickness of 2.6 nm). The measured layer thickness in the experiments with pure PDMS is the same, in the framework of the experimental accuracy. Therefore, the silica in compound A does not affect the thickness of the spreading precursor film. A thick oil layer spreads on the surface 3 to 5 s after the precursor film.

Another series of experiments is performed with emulsion A (0.03 wt %). When the emulsion is introduced in the surfactant solution directly by a pipet (without a two-tips procedure), a thin oil layer immediately spreads over the surface of the studied solution. This layer originates from the oil spread over the surface of the concentrated "mother" emulsion (the latter statement is evidenced by

the absence of such a layer when the TTP is used). The thickness of this "inherited" layer is typically around 1 nm immediately after the transfer. Afterward, a continuous increase of the layer thickness is observed as a result of the coalescence of antifoam globules with the solution surface. The average thickness of the spread layer is ≈ 5 nm after 15 min, while it reaches 15 nm for 1 h. Since the measured layer thickness is averaged over a spot of diameter 2 mm on the solution surface, one may expect that microlenses (invisible by the naked eye) coexist with a thin oil layer, whose equilibrium thickness is around 2.6 nm.[30]

Remarkably, no formation of a spread oil layer was detected for more than 2 h when samples containing 0.03 wt % of *exhausted* emulsion A were studied. The latter result clearly shows that the entry of oil droplets in exhausted emulsions is strongly suppressed, which leads to a solution surface deprived of spread oil.

Thin Foam Films in the Scheludko Cell. The mechanism of foam film destruction by compound A and emulsion A was studied in detail in ref 12. The film lifetime in these experiments was very short—typically between 1 and 10 s, because the experiments were performed with fresh, very active antifoam. The aim of the experiments described below is to observe how the film thinning process changes in the presence of exhausted emulsion A. In addition, we "simulated" the process of antifoam reactivation by addition of oil on the surface of a foam film produced from an exhausted solution.

Not surprisingly, the foam films obtained from exhausted solutions are very stable. Although antifoam globules are seen within the foam film just after its formation, these globules leave the film in the process of its thinning without film rupture—see Figure 10A. At the end of the film thinning process a very thin film (appearing black in reflected light) is formed and remains stable, similar to the experiments in the absence of antifoam.[12] It is worth noting that at least several antifoam globules are seen in each foam film. The size of these globules is similar to the globule size in fresh solutions. Therefore, the different stability is not due to absence of antifoam globules or to some significant reduction of the globule size. The main difference is that the antifoam globules in exhausted solutions do not form unstable oil bridges, which is the critical event leading to foam film rupture in fresh solutions.

To mimic the process of antifoam reactivation we touched the upper surface of a foam film, produced from an exhausted solution, by an oil-soaked glass rod—many oil lenses appeared on the film surface, indicating an oil transfer from the glass rod (Figure 10B). Notably, the foam films from exhausted solutions remained relatively stable in the initial period after the oil was deposited on their surface—they lived for about 10−30 s. With the subsequent formation of foam films in the Scheludko cell (without changing the surfactant solution) we observed two phenomena: (i) entrapment of many silica particles in oil lenses (Figure 10C) and (ii) a rapid deterioration of
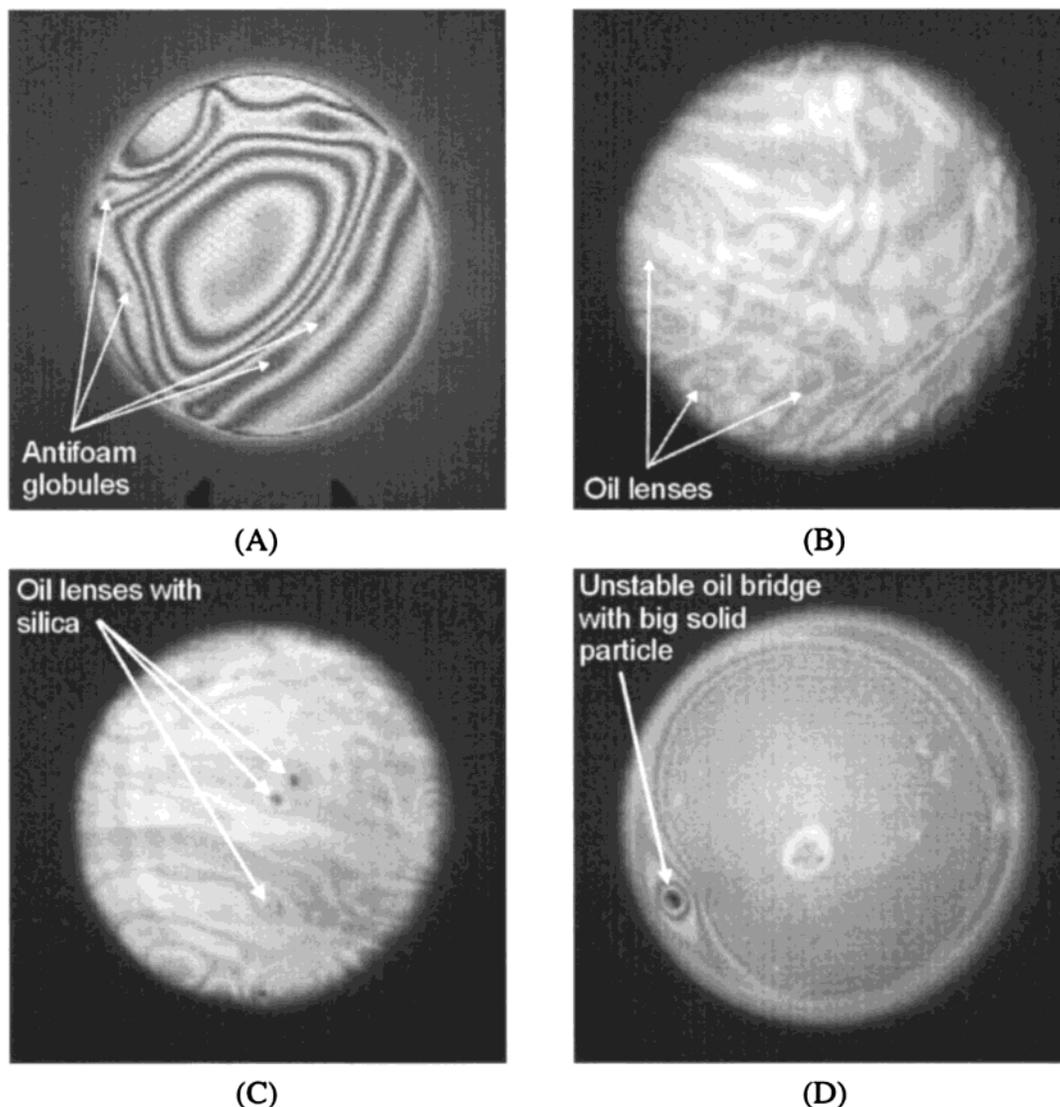
Figure 10. Foam films obtained from an exhausted solution of emulsion A, and the process of antifoam reactivation as observed in Scheludko cell: (A) several antifoam globules trapped in a foam film from an exhausted solution are indicated by arrows, the antifoam globules leave the foam films without film rupture; (B) the deposition of oil on the surface of the foam film leads to the appearance of numerous oil lenses devoid of silica particles, and the lifetime of these films is typically 10−30 s; (C) with the further formation of foam films in the Scheludko cell (without changing the surfactant solution) a transfer of silica particles from the aqueous phase into the oil lenses is observed—the films are very unstable and rupture within several seconds; (D) the characteristic interference pattern ("fish-eye") indicating the formation of unstable oil bridges[12,13] is often observed in these films.

the film stability—the film lifetime shortened to 1−3 s, which is a typical value in the presence of a very active antifoam. Note that the reactivation is made by oil, which does not contain any solid particles, which means that the silica observed in the oil lenses at this stage is "extracted" from the aqueous phase. Not surprisingly, this configuration (oil lenses with trapped silica particles) leads to a very rapid film rupture—it resembles the configuration in the experiments with fresh, very active compound A. The typical interference pattern indicating the formation of unstable bridges (the so-called "fish-eye",[12] Figure 10D) is observed in many foam films just before their rupture.

Discussion—Mechanisms of Exhaustion and Reactivation

In this section we analyze the results from the viewpoint of the different hypotheses for the mechanism of exhaustion, which are listed in the Introduction.

Exhaustion Caused by Changes in the Properties of the Solid Particles. This idea can be traced back to

Kulkarni et al.,[17] who suggested that the contact of silica with the surfactant solution leads to particle hydrophilization. Indeed, if the procedure of particle treatment results in an unstable attachment of the silicone (alkyl) chains onto the particle surface, then a gradual deterioration of the particle hydrophobicity might be expected with time. Arguments that such a process took place when silica particles were used as an antifoam (without oil) were presented in section 4.2.3 of ref 9. The fact, that in our experiments the exhausted solutions can be efficiently reactivated by addition of oil devoid of silica, proves that this hypothesis is not relevant to the present system.

Exhaustion Caused by Changes in the Properties of the Oil. If the antifoam contains mixture of oils (which is sometimes done in practice), one of these oils could be gradually extracted as a result of its solubilization in surfactant micelles. The accompanying change in the composition of the oil phase (presumably pre-optimized to give an active antifoam) could lead to reduction of the antifoam activity. Such a mechanism of exhaustion might

be particularly important for organic antifoams, because many hydrocarbons are soluble in surfactant micelles.

Silicone oils are typically nonsoluble in surfactant solutions, especially if their molecular mass is high. However, the commercial silicone oils contain oligomers of low molecular mass, which can be solubilized in AOT micelles.[31] If these oligomers play some role in the antifoam activity (a possibility that cannot be rejected a priori), then the process of oligomer extraction by surfactant solubilization would lead to reduced antifoam activity. The fact that in our experiments exhausted solutions are perfectly reactivated by a pre-equilibrated oil proves that this mechanism is irrelevant to the present system.

Exhaustion Caused by Reduction of the Size of the Antifoam Globules. The discussion of this mechanism is more complicated for two reasons. First, it was unambiguously shown, for one particular system,[28] that the reduction of the size of silicone oil droplets (devoid of any solid particles) leads to a loss of their antifoam activity. Second, we cannot exclude entirely the possibility that some slight reduction of the antifoam globule size (undetectable by the available methods) could occur in our experiments.

Before going into the analysis of the specific system, it is instructive to emphasize that the globule size must be always considered in relation to the characteristic dimension of the structural element in the foam, which is actually destroyed by the globules.[12] If these are the liquid films, as it is the case in the present system, then the globule diameter should be compared to the foam film thickness. The experiments[12] with both small (millimeter sized) and large (centimeter sized) foam films have shown that their thickness becomes around 1 μm within several seconds after film formation. This is the main reason for the rapid foam destruction (typically within 5−15 s) by the studied fresh antifoams.

Alternatively, if the antifoam globules are less active and are unable to rupture the foam films in the process of their thinning, then the globules escape into the neighboring Gibbs−Plateau borders (GPB).[10,28] If such is the case, the antifoam globules enter the air−water interface only after being compressed by the walls of the narrowing GPBs, whose cross section is typically tens or hundreds of micrometers. The drainage of water from the foam column is a relatively slow process,[32,33] and a much longer time, on the order of minutes or dozens of minutes, is needed until the cross section of the GPBs becomes comparable to the diameter of the typical antifoam globules. In other words, the optimal sizes of the antifoam globules and the time scales of the antifoaming process are very different in the systems containing "fast" (film breaking) and "slow" (GPB breaking) antifoams.

It is worth discussing briefly the main factor determining whether a given antifoam would be "fast" or "slow". The accumulated experience with different antifoams has shown that positive values of the entry, E, and bridging, B, coefficients is a necessary, but insufficient condition for having an active antifoam;[2,3] other factors are also important. One major factor is the barrier to drop entry.[28,34−36] Recently a new method, termed "the film

trapping technique" (because the antifoam globules are trapped in wetting films on a solid substrate) was used to measure the barrier to globule entry with real antifoams.[37] The experiments showed that the barrier to entry is about 1 order of magnitude lower for silica-containing globules compared to the barrier for silica-free drops (qualitatively similar results were obtained also by Koczo et al.[10] and by Bergeron et al.[7]). If the barrier to drop entry is low, the globules are able to enter the foam film surface during the process of film thinning.[12] Alternatively, the globules are unable to enter the surfaces of the foam film if the barrier is high,[28] because the capillary pressure compressing the globules in the film is insufficient. A higher capillary pressure (realized in the narrowing GPBs of the draining foam[3,33]) is needed to induce an oil drop entry and foam destruction.[28]

After the above general remarks, we are able to discuss the role of the globule size in the specific system under consideration. The microscopic observations and the light scattering experiments show that in all of the studied samples (both fresh and exhausted) we have numerous drops of size between 1 and 10 μm, which are expected to be active. Moreover, we do not detect any significant difference in the globule size between fresh and exhausted samples. The experiments with foam films from exhausted solutions demonstrated that the main difference between the globules in a fresh and in an exhausted solution is the loss of the globule activity (see below for the reasons), but not a significant reduction of the globule size. Therefore, we can conclude from the obtained results that the effect of globule size reduction is not important for the exhaustion and reactivation processes in our system.

We refrain from discussion of results published in other studies, where the reduced size of the antifoam globules is suggested as the main reason for antifoam exhaustion— such a discussion would be unjustified without having a more precise information about the structural element (foam film or GPB) that was actually destroyed in these systems. It is worth noting, however, that a foam destruction within seconds indicates a destruction of the foam films (not of the GPBs). In this case the globule size might be a crucial factor for the antifoam exhaustion only if the average globule diameter becomes smaller than ca. 1 μm. Globules of diameter 1 μm are already large enough to be trapped in the foam films (and to rupture them if the globules are active) within several seconds after film formation. Note that a reduced globule size corresponds to an actual increase of the number concentration of the globules (at fixed weight concentration), which may compensate for the needed longer time for film thinning before capturing the globules.[38]

Exhaustion Caused by Disappearance of the Spread Oil Layer and by Redistribution of Silica between the Antifoam Globules. These two mechanisms will be considered together, because they are both important in our system. The experimental results demonstrate a clear correlation between the antifoam exhaustion and the disappearance of the spread oil from the solution surface. One might suggest that this is the only important event, which could explain the main observations. This suggestion seems very reasonable if the exhaustion of compound A is considered alone: the compound is initially deposited on the solution surface (where it is active), and the foam destruction process

(31) Binks, B. P.; Dong, J.; Rebolj, N. *Phys. Chem. Chem. Phys.* **1999**, *1*, 2335.

(32) Weaire, D.; Hutzler, S.; Verbist, G.; Peters, E. *Adv. Chem. Phys.* **1997**, *102*, 315.

(33) Narsimhan, G.; Ruckenstein, E. In *Foams: Theory and Practice*; Prud'homme, R. K.; Khan, S. A., Eds.; Surfactant Science Series Vol. 57; Marcel Dekker: New York, 1996; Chapter 2.

(34) Kulkarni, R. D.; Goddard, E. D.; Kanner, B. *J. Colloid Interface Sci.* **1977**, *59*, 468.

(35) Lobo, L.; Wasan, D. T. *Langmuir* **1993**, *9*, 1668.

(36) Bergeron, V.; Fagan, M. E.; Radke, C. J. *Langmuir* **1993**, *9*, 1704.

(37) Hadjiiski, A.; Cholakova, S.; Ivanov, I. B.; Gurkov, T. D.; Leonard, E. *Langmuir* **1999**, Submitted for publication.

(38) Garrett, P. R. *Langmuir* **1995**, *11*, 3576.

results in its gradual emulsification, disappearance from the solution surface, and ultimate exhaustion. However, a closer look on the obtained results reveals a more complex picture of the real process:

1. One can calculate the amount of prespread oil in the experiments with emulsion A. The surface area of the solution in the shake test experiments, before starting the foam formation cycles, is about 20 cm^2. If we assume that the thickness of the prespread layer is about 10 nm (which is an upper limit—the real value might be an order of magnitude lower), the amount of oil in this prespread layer can be estimated as 2×10^{-5} cm^3. This is a very tiny fraction (0.2%) of the total amount of oil, 10^{-2} cm^3, which is introduced in the form of antifoam. Next, the air−water surface created in the foam shake test can be estimated from the foam volume ($\approx$ 100 cm^3) and the mean radius of the air bubbles ($\approx$ 1 mm) to be on the order 10^3 cm^2. If only the initial prespread oil is available during the foam formation period, then the expansion of the solution surface would stretch the spread layer $\approx$10^2 times, diminishing its thickness below 0.1 nm, i.e., the spread layer would practically disappear from the stretched air−water interface.

As proven in ref 12, the foam films in the absence of a prespread layer are stable and do not rupture even in the presence of fresh antifoam globules. Therefore, the activity of fresh emulsion A in the shake test can be explained only by assuming that new portions of oil emerge on the foam film surfaces from entering antifoam globules during the foam production cycle—otherwise, emulsion A would be rather inactive or would lose its activity almost immediately.

In other words, the actual foam destruction process in the case of emulsion A certainly involves a continuous emergence/emulsification of oil onto/from the solution surface. If the number of antifoam globules able to supply oil on the solution surface decreases with time (see below for the respective explanation), then the emulsification process will start to prevail, and the spread oil will disappear after a certain number of cycles (see Figure 11B). As a result, the antifoam globules will stop breaking the foam films—the antifoam will be deactivated. The addition of a new portion of oil on the surface of an exhausted solution supplies a new "fuel" for the emergence/emulsification cycles—the antifoam is reactivated (Figure 11C,D).

2. By using the two-tips procedure (TTP) one can prepare a working solution containing emulsion A, whose surface is free of prespread oil. Shake test experiments with this sample show that its activity is practically the same as that of emulsion A in the conventional experiments (without using the TTP). Furthermore, surface tension measurements show that there is a newly generated spread oil layer on the solution surface after the first shaking cycle. The only source of spread oil in this sample could be antifoam globules, which enter the solution surface during foaming. Note that without foaming, a spread layer is also created by entering antifoam globules, but much longer time is needed (at least several hours).[12]

3. A process of oil release (spreading) from antifoam globules onto the film surface was observed[12] in the experiments with foam films formed after using the TTP (Scheludko cell). The oil spreading from these globules did not rupture the foam films, because the formed oil−silica bridges remained stable due to deficiency of oil.[12,13] In fact, the oil spreading from the globules served to saturate the film surfaces, which were "hungry for oil"— the respective explanation in terms of the chemical potential of the oil is given in ref 13.
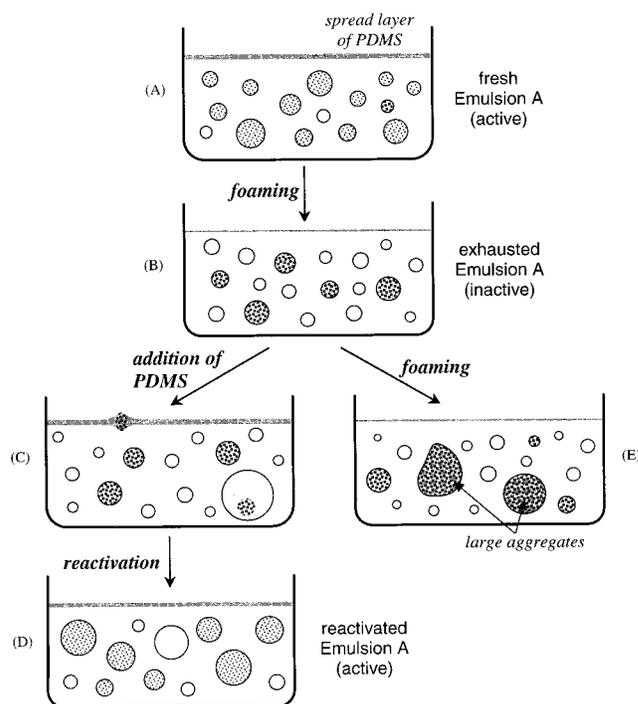
Figure 11. Schematic presentation of the processes of antifoam exhaustion and reactivation of emulsion A: (A) An initially active (fresh) emulsion A contains globules of optimal silica/oil ratio; a layer of spread oil is formed on the surface of the surfactant solution. (B) The foam destruction by the antifoam globules leads to gradual segregation of oil and silica into two inactive populations of globules (silica-free and silica-enriched); the spread oil layer disappears from the solution surface—the antifoam becomes inactive. (C) The introduction of a new portion of oil leads to restoration of the spread oil layer and to redistribution of silica, so that active silica globules are formed again—the antifoam is reactivated (D). The macroaggregates (see the text) appear as a result of aggregation of the silica-enriched globules (E).

4. Our observations show that similar emergence/emulsification circulation of oil takes place in the experiments with compound A as well. Several large, visible by naked eye antifoam globules are typically seen after the first foaming cycle in the shake test. The coalescence of these large globules with the surface of the solution is often observed—a process which transforms an emulsified globule into a lens.

All these results clearly demonstrate that the oil, which is prespread on the surface of the foaming solution at the beginning of the shake tests, is not the only oil involved in the foam destruction. It would be much more realistic to talk about a circulation of oil between the bulk of the surfactant solution and its surface. If the oil is able to perform larger number of cycles between the bulk and the surface, this would postpone the moment of spread oil disappearance, and would lead to longer durability of the antifoam.

Let us discuss here which is the process leading to deceleration of oil supply on the solution surface. The only explanation we could suggest, which does not contradict any of the obtained results, is that the oil circulation described above is accompanied by a gradual segregation of oil and silica into two distinct populations of globules: silica-free and silica-enriched. Both these populations are rather inactive and are poor source of oil, for different reasons, however: (i) The silica-free drops are unable to enter the foam film surfaces, because the barrier to entry is rather high for them. (ii) The silica-enriched globules are able to enter the foam film surface and even to make

bridges; however, these globules behave as nondeformable entities, which are unable to break the film in the absence of a spread oil layer, because neither the bridging−stretching mechanism (which requires deformability of the globules) nor the bridging−dewetting mechanism (which requires appropriate contact angles, unrealized in the studied system) are operative. These silica-enriched globules are able to release some oil at the initial stage of the exhaustion process, but the oil spreading is notably slower, which explains the reduced rate of foam destruction—a longer time is needed for accumulation of a spread layer of critical thickness (probably, around 1 nm), which ensures the formation of unstable oil bridges.

The amount of the released oil becomes so small with the further evolution of the silica−oil segregation that the spread layer on the surfaces of the generated foam films cannot reach the critical thickness leading to formation of unstable bridges—the antifoam becomes completely exhausted. Part of the silica in exhausted solutions is gathered in macroagglomerates, while another significant part is still dispersed in the form of micrometer sized silica-enriched globules. Notably, a substantial fraction of the used oil (about 25% or even more) is arrested in the silica-enriched globules, and cannot be released on the solution surface.

It is worth emphasizing the fact, that a pronounced process of silica−oil segregation should be always expected when the size of the antifoam globules approaches the size of the silica agglomerates. Such small globules can be produced in the emulsion fabrication process or during exhaustion. If we have had a monodisperse silica agglomerates (which is not the case with commercial antifoams), one may expect that the distribution of silica among the antifoam globules would acquire a "quantum" character in this size range—in each antifoam globule, one would have either one silica particle or no particle. The broad size distribution of silica in commercial antifoams prevents the realization of this extreme example, but one may expect a significant silica−oil segregation even with polydisperse particles.

As evidenced by the foam film experiments in the Scheludko cell, the deposition of a new portion of oil onto the solution surface leads to rearrangement of the silica particles—they are transferred into the newly created lenses of oil, forming in this way active entities of appropriate silica concentration. All these interrelated processes are schematically presented in Figure 11.

We can explain by similar arguments the results for the reactivation with oil drops of different size (Figure 4). When emulsions of large mean size (90 μm) are used for reactivation, probably almost all of the drops coalesce with the solution surface, thus creating a spread oil layer comparable to that after the initial deposition of compound A. The complete emulsification of this layer takes long time, and the number of foaming cycles before exhausting the reactivated sample is comparable to that for fresh compound A. When an emulsion with a small mean drop size (2 μm) is used, only a small fraction of these drops succeeds to enter the solution surface. The emulsions used for reactivation are of wide size distribution, and probably only the biggest drops in this sample coalesce with the solution surface releasing much less oil in this case. As a result, the emulsification of the oil layer and the ensuing antifoam exhaustion is much faster.

Foam Film Rupture as a Critical Step in the Antifoam Exhaustion. The results described above imply that the exhaustion is intrinsically connected to the foam destruction process. However, the experimental data obtained so far do not provide unambiguous infor-
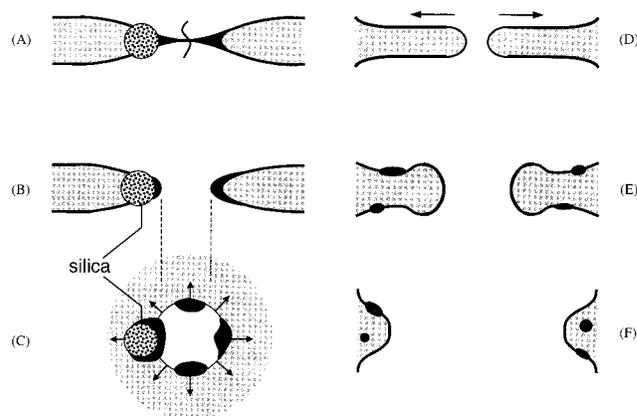
Figure 12. Possible mechanisms of antifoam fragmentation. After an oil bridge ruptures (A), the formed hole in the film rapidly expands (B). The oil rim, which has remained from the bridge, is stretched and fragments into several smaller oil droplets (C). Some of them contain silica particles, while others are deprived of silica. These droplets hit with high velocity the adjacent Gibbs−Plateau borders and are emulsified there. Part of the spread, ultrathin oil layer can be also emulsified in the moment of foam film rupture (D−F). The expansion of the hole in the film (D) leads to a rapid contraction of film surfaces (E). The excess of spread oil forms oil lenses, which are dragged toward the GPB by the perimeter of the expanding hole. The impact of these lenses with the GPB could lead to oil emulsification (F).

mation about the specific step leading to emulsification of the spread oil layer and to silica−oil segregation. We suggest below two possible ways—both of them seem reasonable and can be realized in practice.

One possible way of oil emulsification and silica−oil segregation is suggested by the bridging−stretching mechanism[12] (Figure 1). The process of bridge stretching and rupture is accompanied by a very rapid expansion of the oil rim formed at the periphery of the bridge (where the bridge is thicker)—see Figure 12A,B. This rapid expansion could lead to a Rayleigh-type of instability and to possible fragmentation of the oil rim into several oil droplets. Some of these droplets might be devoid of silica, while others might be enriched in silica—thus a process of silica−oil segregation is induced (Figure 12C). The oil fragments are dragged by the expanding hole in the film and are projected with velocity on the order 10 m/s toward the Gibbs−Plateau borders.[12,39] It is likely that some of the oil fragments will enter the GPB and will be trapped there in the form of emulsion droplets (some of them containing silica). The accumulation of oil from the spread oil layer into the oil bridges before their rupture[13] could explain the gradual disappearance of the spread layer from the solution surface. Therefore, the exhaustion process can be naturally explained in the framework of the bridging−stretching mechanism.

Another possibility, which would lead to similar results, is illustrated in Figure 12D−F. The foam film rupture leads to an ultrarapid contraction of the film surfaces with inevitable formation of an excess of spread oil. This oil will be forced to form lenses on the contracting film surface (Figure 12E). One can expect that these lenses, dragged by the expanding hole perimeter in the broken film, could enter the GPB in the form of emulsion droplets (Figure 12F). Subsequent cycles—globule entry → oil spreading → film rupture → emulsification of the spread oil—could explain the observed process of silica−oil segregation.

The above mechanisms explain the fact (well-known to the practitioners) that there is an optimum in the viscosity

(39) Culick, F. E. C. *J. Appl. Phys.* **1960**, *31*, 1128.

of oils used for antifoam formulations. If an oil of low viscosity is used, the antifoam exhibits a high initial activity but the exhaustion is typically rapid, which is probably due to easier segregation of oil and silica during the film rupture process. On the other side, too viscous oils lead to antifoams of low activity and several possible explanations can be envisaged: (i) the dispersion of the antifoam into numerous active globules becomes difficult, so that the overall number of active entities remains small; (ii) the rate of oil spreading decreases above a certain oil viscosity—see Figure 4 in ref 7; (iii) the deformation of the antifoam globules, which is a necessary condition for realization of the bridging-stretching mechanism,[12,13] becomes too slow for efficient film rupture.

Conclusions

The major aim of this study is to reveal the physico-chemical processes leading to exhaustion (deactivation) of mixed silica−silicone oil antifoams in the course of their action (Figure 2). Several possible explanations of the exhaustion phenomenon are formulated and systematically examined. The results show that the exhaustion process in the studied system is caused by two closely interrelated processes (Figure 11): (1) disappearance of the spread oil layer from the surface of the solution (Table 1); (2) segregation of oil and silica into two inactive populations of antifoam globules, silica-free and silica-enriched (Figures 7 and 8).

The reduction of the average globule size, which is sometimes considered to be the main factor in the antifoam exhaustion, is of secondary importance in the studied system (Figures 5 and 6). A mechanistic explanation of processes (1) and (2) is given in the framework of the bridging−stretching mechanism of foam film rupture (Figure 12).

The reactivation of an antifoam, by addition of oil devoid of silica, is a result of the recombination of exhausted silica-enriched globules with the newly introduced oil on the surface of the solution; this process leads to formation of active antifoam lenses and globules of optimal silica−oil ratio (Figures 10 and 11).