Antifoaming Action of Oils

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Abstract

Antifoams are widely used for control of the foam stability in various products (detergents, paints, pharmaceuticals, and many others). A significant progress in the understanding of the foam destruction mechanisms by oil-containing antifoams has been achieved recently. Experiments with antifoams comprising silicone oil and hydrophobic silica showed that the antifoam entities (emulsified globules or lenses floating on the solution surface) easily form unstable oil bridges between the two surfaces of the foam film. These bridges rapidly stretch in radial direction, due to uncompensated capillary pressures at the oil-water and oil-air interfaces, and eventually rupture the foam films. As a result, the foam is destroyed within several seconds by the mixed solid-liquid antifoams. In contrast, drops of silicone oil deprived of silica are unable to enter the foam film surface due to significant entry barriers. In these systems, the oil drops are expelled into the neighbouring Plateau borders (PBs), and the foam collapse is observed at a much later stage of the foam evolution, when the drops are compressed by the walls of the narrowing PBs (defoaming time on the order of minutes and hours). The magnitude of the entry barriers can be quantified by the so called Film Trapping Technique (FTT).

1 Introduction

Antifoams are introduced in surfactant solutions to prevent the formation of excessive foam [1]. A typical antifoam consists of an oil (polydimethylsiloxane or hydrocarbon), dispersed hydrophobic solid particles, or a mixture of both. A strong synergistic effect between the oil and the solid particles is observed in the mixed antifoams - usually they are much more efficient than either of the individual components, taken separately [1].

Many problems, related to the foam destruction by oil-containing antifoams, still remain unresolved. The main difficulties arise from the deficiency of univocal information about the main steps in the foam destruction process, and this precludes its adequate modelling. Hence, the antifoam optimisation has been based exclusively on the method of "trials and errors".

Several mechanisms of foam film destruction by oil-containing antifoams are suggested in the literature, and among them the so called "bridging-dewetting" and "spreading-fluid entrainment" are most widely discussed [1-5]. An analytical review of these mechanisms and the respective entry, E, spreading, S, and bridging, B, coefficients is presented by Garrett [1]. The coefficients E, S and B are calculated from the interfacial tensions of the air-water, σ_{AW}, oil-water, σ_{OW}, and oil-air, σ_{OA}, interfaces [1]:

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$$E = \sigma_{AW} + \sigma_{OW} - \sigma_{OA},$$

$$S = \sigma_{AW} - \sigma_{OW} - \sigma_{OA},$$

$$B = (\sigma_{AW})^2 + (\sigma_{OW})^2 - (\sigma_{OA})^2,$$

and are frequently used to quantify the antifoam activity of an oil. However, direct proofs that one or another mechanism is operative in a given particular system were missing till recently.

A related question of great practical importance is about the structural element (foam film or Plateau border) which is actually destroyed by the antifoam globules. The globule diameter should fit to the typical size of the element - to the film thickness or to the cross-section of the Plateau border (PB), respectively. Most of the researchers consider that the antifoam ruptures the foam films [1-5], while Koczo et al. [6] suggested that the antifoam globules first escape from the foam films into the neighbouring Plateau borders (PB) and get trapped there; afterwards the globules destroy the PB and the neighbouring foam films.

Another important problem concerns the unexpectedly high selectivity of the antifoams observed in some systems – antifoams that are very active in a given surfactant solution, might be rather inefficient for another surfactant of similar properties (molecular mass, *E*, *S*, and *B* coefficients, etc.). The reasons for this selectivity are still poorly understood.

To gain deeper insight into the mechanisms of antifoaming and to obtain answers to the above questions, we performed a set of experiments with several antifoams and surfactants. Mixed solid-liquid antifoams were mostly studied. For comparison, oil drops deprived of silica were also tested. This article presents a short review of the main new results and a very brief discussion - more details can be found in the cited articles.

2 Materials

Two mixed antifoams, which closely mimic the commercial ones, are studied: Compound A (CA), which is a silicone oil of viscosity 1000 cP with 4.2 % hydrophobic silica particles (0.1 to 5 μm in size) dispersed in it; Emulsion A (EA), which is an emulsion of CA stabilised by two surfactants, Span 60 and Mirj 52. Silicone oil without silica is used in some experiments for comparison.

Two surfactant solutions are used of concentration about 3 × CMC: 10 mM of sodium dioctyl-sulfosuccinate (AOT) and 0.45 mM of alkyl-$C_{12/14}$ $(glucopiranoside)_{1.2}$ (APG).

3 Mechanisms of foam destruction by oil-containing antifoams

3.1 Structural element ruptured by the antifoam - "fast" and "slow" antifoams

Direct microscopic observations by a high-speed video camera, showed that the foam destruction by typical mixed antifoams (comprising oil and silica) occurred through rupture of the foam lamellae, Fig. 1. Experiments with small (millimetre sized) and large (centimetre sized) foam films showed that the antifoam induced the formation of a hole in the foam films at the early stages of the film thinning process [7]. As a result, the films ruptured within several seconds after their formation. Accordingly, the foam produced from such solutions disappeared completely for less than 10 seconds in the standard shake tests. Therefore, we call the antifoams that are able to enter the surfaces of the foam films, the "fast antifoams". Experiments with several non-ionic surfactants (Triton X-100, Brij 58, APG) showed that in all these systems, the mixed antifoams broke the foam films. Some peculiar and non-trivial observations with APG solutions are discussed in section 3.4 below.

Interestingly, similar experiments show that the foam destruction occurs in a different manner when only silicone oil (deprived of silica) is used as an antifoam. The antifoam globules were seen to leave the foam films (without rupture) during the film thinning process. The antifoam drops were accumulated in the PBs and stayed trapped there for a certain period of time, as suggested by Koczo et al. [6]. The water drainage from the foam lead to a gradual narrowing of the PBs with time, and the oil drops became strongly compressed. When the compressing capillary pressure exceeded some critical value, the oil drops entered the walls of the PB inducing its destruction and the rupture of the neighbouring foam films (for estimates of the capillary pressure in the foam column and its relation to the mechanism of antifoaming, see section 4.1 in ref 10). Notably, much longer time was needed for foam destruction in this case - typically, tens of minutes. That is why, we call these substances the "slow antifoams". Furthermore, after an initial period of foam decay, we usually observed in these systems a residual foam of well defined height, which might remain stable for many hours.

In conclusion, the foam destruction may occur through rupture of either the foam films or the PBs depending on the particular system. Further experiments showed that the main factor determining the position of foam destruction, and whether a given antifoam behaves as "fast" or "slow", is the magnitude of the entry barrier (see section 4 below).

(A) (B)

Fig. 1: Images of small horizontal (A) and large vertical (B) foam films of 10 mM AOT solutions containing 0.01 wt % of Emulsion A (fast antifoam) [7]. (A) A characteristic pattern called the "fish-eye" (the arrow) is observed under high magnification in the small films just before their rupture. (B) In the large films, one can observe the formation of a hole in the film (the arrow) and its subsequent expansion leading to the film rupture.

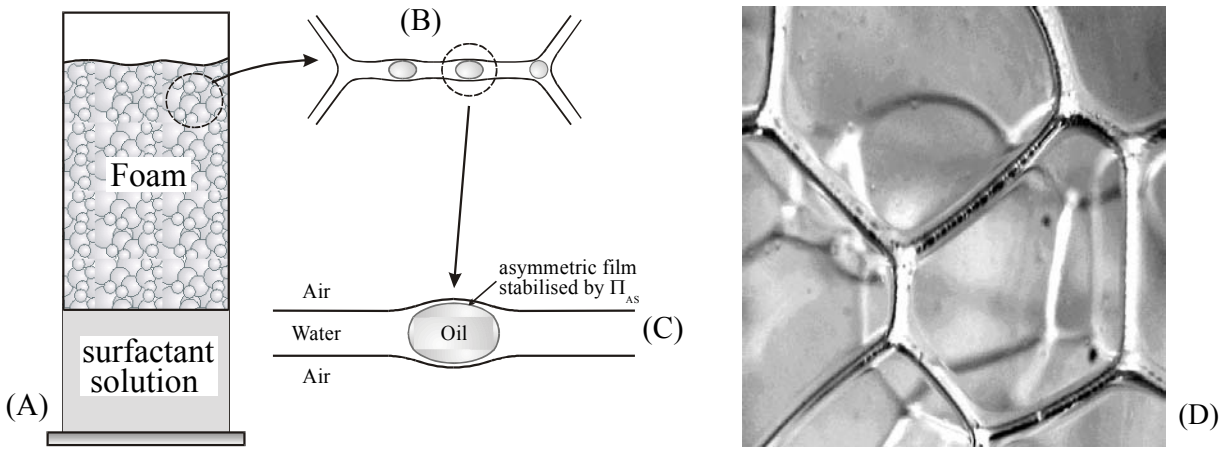

Fig. 2: Schematic illustration (A-C) of the mechanism of foam destruction by oil drops deprived of silica (slow antifoam). In (D) trains of many oil drops are seen trapped in the Plateau borders of foam, formed from surfactant solution containing 0.1 wt % of silicone oil [10].

3.2 Bridging-stretching mechanism of film rupture and stability of oil bridges

To clarify the detailed mechanism of foam film rupture by mixed antifoams, we used optical observations by a high-speed video camera [7]. When an antifoam globule connected the surfaces of the foam film, a characteristic interference pattern, called the "fish-eye" (Fig. 1A), was observed. A careful examination of this pattern showed that it indicated the formation of an unstable oil bridge, which stretched with time due to uncompensated capillary pressures at the oil-air and oil-water interfaces [7], and eventually ruptured the entire foam film (Fig. 3A-E). Similar events - bridge formation, stretching and rupture, were observed with large drops of CA in another experimental cell, Fig. 3A'-E'. The term "bridging-stretching" was suggested [7] to describe this mechanism.

The stability of the oil bridges in foam films was theoretically studied by further developing the model suggested by Garrett [11]. The theoretical analysis confirmed the conclusion of Garrett that the bridging coefficient, B, must be positive to have an unstable oil bridge. However, the calculations showed that the oil bridges, formed from oil drops of diameter comparable to, or smaller than the film thickness, might be metastable even at strongly positive values of B. This result was used to explain the reduced stability of the foam films in the presence of a spread oil layer (see section 3.3).

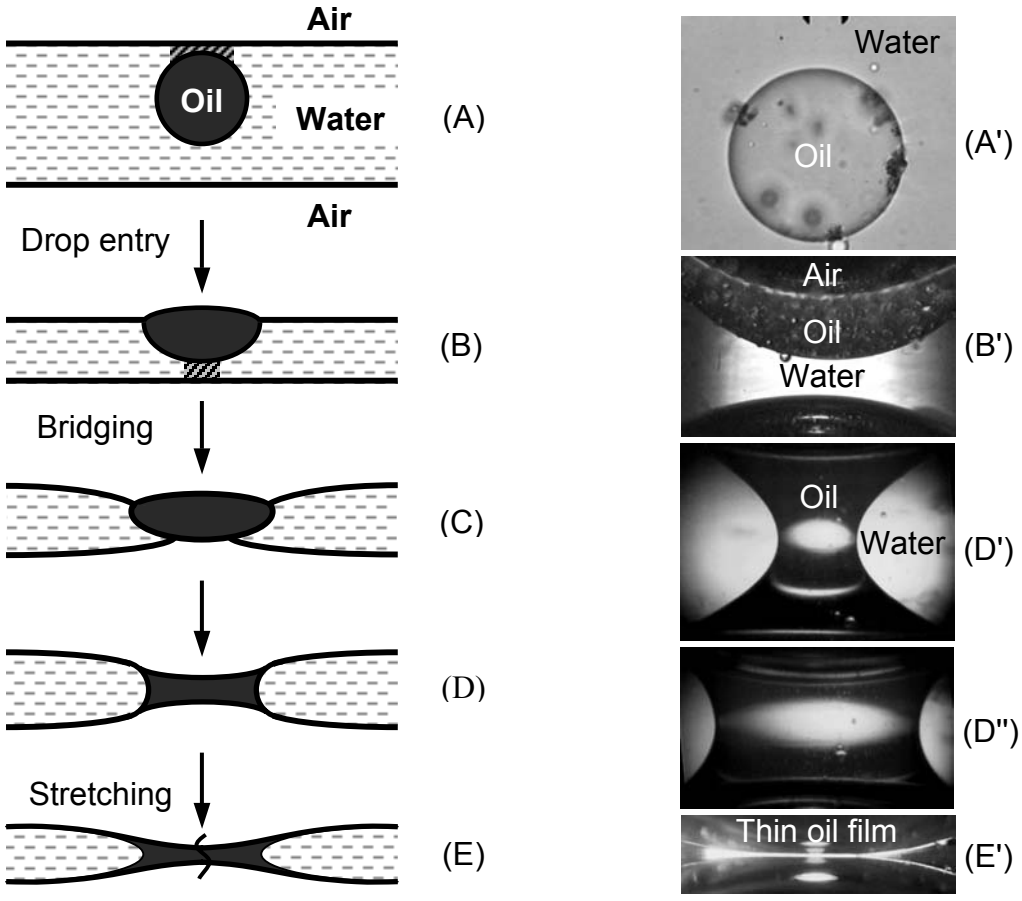

Fig. 3: Bridging-stretching mechanism of foam film rupture: (A-C) The entry of an antifoam globule leads to the formation of capillary unstable oil bridge. (C-E) The bridge stretches with time, due to the uncompensated capillary pressures at the oil-air and oil-water interfaces, and eventually ruptures, destroying the entire foam film. The asymmetric oil-water-air film is shaded in (A) and (B). (A'-E') Similar events were observed with large drops of Compound A.

3.3 Role of the oil spreading

It is well known [1,3,5] that some correlation exists between the spreading behaviour of the oils and their antifoam activity. The "spreading-fluid entrainment" mechanism was widely discussed in the literature. However, as shown by Garrett et al. [1,12], the oil spreading is not a necessary condition for having an antifoam activity and the correlation is not straightforward.

We used a simple technique (called the "two-tips procedure" [7]) to create a solution surface free of spread oil - thus the effect of the spread layer on the foam film stability could be evaluated. The experiments demonstrated an important effect of the spread oil [7]. In the presence of a pre-spread oil layer (though being very thin), the globules of the mixed antifoams readily made unstable bridges which ruptured the foam films. On the contrary, in absence of oil, the globules did not form unstable bridges and the films remained intact [7].

Two possible explanations could be given to this strong effect: (1) Measurements of the entry barrier of antifoam globules show that it is significantly reduced in the presence of spread oil, see section 4; (2) The spread oil is able to "feed" the oil bridges by a mechanism explained in ref 8, which leads to an increase of the bridge size. As discussed in section 3.2, the oil bridge should be above a certain critical size to be unstable. Therefore, an initially stable bridge could become unstable in the presence of a spread oil.

3.4 APG solutions - dynamic effects and role of the electrostatic interactions

The mechanism of foam destruction by mixed antifoams in AOT and APG solutions was essentially the same (bridging-stretching), but several important differences were observed [13]. Although the *E*, *S* and *B* coefficients were practically the same, the antifoams were significantly more active in AOT solutions. Also, the antifoams were active in APG solutions only under dynamic conditions (during foaming) and a residual foam, remaining stable for many hours, was observed after stopping the agitation. On the contrary, the antifoams were very active in AOT solutions under both static and dynamic conditions, and the foam was entirely destroyed within seconds after ceasing the shaking.

Model experiments showed that the above differences could be explained by several simple effects. The entry barrier is much higher for APG solutions in comparison with AOT (section 4). Therefore, the antifoam globules are able to enter the surfaces of the foam films stabilized by APG only if the adsorption monolayers are depleted of surfactant. In other words, the mixed antifoams have some reasonable activity in APG solutions only because the rate of surfactant adsorption is rather slow - more than 10 s are needed for saturation of the adsorption layer in the working solutions [13]. On the contrary, the entry of the antifoam globules and the subsequent film rupture are possible in AOT solutions, even when the surfactant adsorption layers are saturated.

The high entry barrier in APG solutions is related to the presence of a strong electrostatic repulsion between the surfaces of the asymmetric oil-water-air film (due to the low ionic strength of these solutions) and to the small penetration depth of the solid silica particles into the aqueous phase. Hence, the silica particles are unable to break the asymmetric film and to induce a globule entry. The asymmetric films in AOT solutions are much thinner (higher ionic strength), and the silica particles protrude deep enough across the film, as to break it.

4 Role of the entry barrier - Film Trapping Technique (FTT)

The FTT, developed by Hadjiiski et al. [14,15], allows one to measure directly the critical capillary pressure, ΔP_{CR}, which leads to entry of small oil drops trapped in aqueous films.

Therefore, this is the first experimental technique, which is able to quantify the entry barriers of real antifoam globules of micrometer size.

In Table 1 we show some of the obtained results [9,13,16], from which the following conclusions can be drawn: (1) The introduction of solid particles into the silicone oil reduces the entry barriers by about an order of magnitude. (2) The entry barriers in the presence of a pre-spread oil layer are systematically lower than those obtained in the absence of a spread oil. This important result provides a new explanation of the role of oil spreading in antifoaming. A larger set of data, along with the respective mechanistic explanation of this effect will be given in a separate article [16]. (3) The entry barriers for all antifoams in 0.45 mM APG solutions are rather high, which explains why the APG foams are stable under static conditions [13]. The entry barrier is below 10 Pa for APG solutions below CMC, when the surfactant adsorption layers are not saturated. (4) The magnitude of ΔP_{CR} which separates the fast (foam film breaking) from slow (PB breaking) antifoams is around 20 Pa - the globules having an entry barrier above 20 Pa are unable to enter the foam film surface and are expelled into the PBs during the process of foam film thinning.

In general, a very good correlation was always observed between the antifoam activity and the height of the entry barrier, while no such correlation was observed with the values of the *E*, *S*, and *B* coefficients [10,13,16].

Table 1. Critical capillary pressures, ΔP_{CR}, measured for different antifoams and surfactants.

Surfactant solution	Antifoam	Spread oil layer	ΔP_{CR}, Pa
10 mM AOT	Silicone oil	Yes	30 ± 5
	Emulsion A	Yes	13 ± 4
		No	33 ± 4
0.45 mM APG	Silicone oil	Yes	> 1250
	Emulsion A	Yes	> 120
0.045 mM APG	Emulsion A	Yes	$1 \div 10$

5 Exhaustion (deactivation) and reactivation of the antifoam

The process of antifoam "exhaustion" is illustrated in Fig. 4 - the time for foam destruction in a standard shake test is shown as a function of the number of the shaking cycle [9]. Shorter defoaming time means more active antifoam and vice versa. As seen from Fig. 4, the initial high activity of the antifoam deteriorates with the foaming cycles and the defoaming time becomes longer than 60 s after 45 cycles – the antifoam has been *exhausted*.

The addition of a new portion of oil, deprived of silica particles, leads to a complete restoration of the antifoam activity (Fig. 4). Note, that the oil itself has no activity in the absence of silica. Therefore, the *reactivation* certainly involves the solid particles that have been introduced with the first portion of mixed antifoam. The subsequent foaming cycles lead to a second exhaustion series and such consecutive periods of exhaustion/reactivation can be repeated several times (Fig. 4). Similar phenomena are observed with solutions of other surfactants, including APG. It is worth noting that the reactivation phenomenon does not solve the practical problem of antifoam exhaustion in industry, because the silicone oil is relatively expensive and the cost of each new portion of oil is comparable to the cost of the initial antifoam.

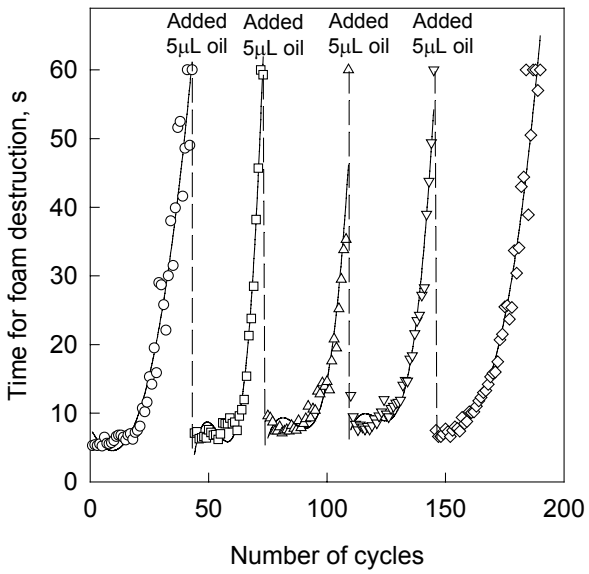

Fig. 4: Consecutive cycles of exhaustion and reactivation of CA, 0.005 wt % in 11.3 mM AOT solution. An initially active antifoam (defoaming time ≈ 5 s) gradually looses its activity. The introduction of silicone oil results in a restoration of the antifoam activity.

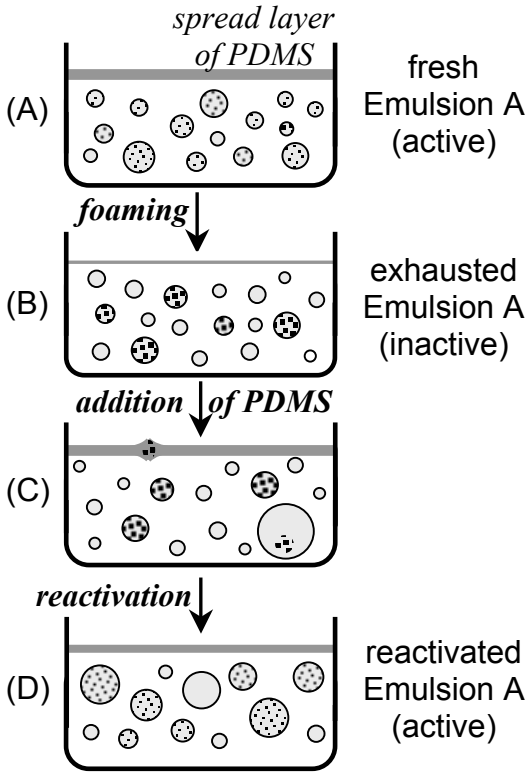

Fig. 5: Schematic presentation of the processes of antifoam exhaustion and reactivation of emulsified silica-silicone antifoam.

The systematic experiments with AOT solutions [9] showed that the exhaustion of mixed silica-silicone oil antifoams is due to two closely interrelated processes, Fig. 5: (1) partial segregation of the oil and silica into two distinct, inactive populations of antifoam globules (silica-free and silica-enriched); (2) disappearance of the spread oil layer from the solution surface. The oil drops deprived of silica, which appear in process 1, are unable to enter the air-water interface and to destroy the foam lamellae, because the entry barrier is to high for them (section 4). On the other side, the antifoam globules enriched in silica trap some oil, which is not available for spreading on the solution surface. As a result, the spread oil layer 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 exhausted state. Accordingly, the reactivation process is due to [9]: (1) restoration of the spread oil layer, and (2) rearrangement of the solid particles from the exhausted antifoam with the fresh oil into new antifoam globules having optimal silica concentration. No correlation between the size of the antifoam globules and their activity was established in these experiments, which showed that the reduction of the globule size was a second-order effect in the studied systems. Similar conclusions were drawn from the experiments with APG solutions as well [13].

6 Conclusions

- The foam destruction occurs through rupture of the foam films if mixed silica-silicone oil antifoams are used. This process is typically very fast and the foam is destroyed within

seconds - that is why these substances are termed the fast antifoams. In contrast, the foam destruction occurs through rupture of the Plateau borders (PBs) for a much longer period of time, minutes or hours, if oil drops deprived of silica (slow antifoams) are used.

- The main factor, which determines whether the antifoam breaks the foam films or the PBs is the entry barrier. If the barrier is above ca. 20 Pa, the antifoam globules are unable to enter the surfaces of the foam films - instead, the globules are expelled into the adjacent PBs when the film thickness becomes smaller than the globule diameter.
- The foam film destruction by mixed silica-silicone oil antifoams occurs through the so called "bridging-stretching" mechanism, which implies that capillary unstable oil bridges are formed when an antifoam globule connects the two opposing surfaces of the foam film.
- New explanations for the role of oil spreading in the antifoam activity have emerged from the experiments - the spread oil layer reduces the entry barriers and helps in the formation of unstable oil bridges.
- The exhaustion and reactivation of mixed antifoams, as well as the poor antifoam activity in APG solutions, are explained in the framework of the above ideas.

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