c = fluid concentration, mol/L
c^* = fluid concentration in equilibrium with solid-phase concentrations, mol/L
D_M = mass molecular diffusivity, cm^2/min
E_D = eddy axial dispersion, cm^2/min
F = solute feed rate, mol/min
L = length of bed, cm
q = solid-phase concentration, mol/kg of dry solid
t = time, min
t_1, t_2 = times that diffuse wave intersects $z = L_F$, eq 18 and 19 and Figure 3, min
$u_A, u_B,$
u_c, u_D = solute wave velocity for solutes A, B, D or for a general case (c), cm/min
v = interstitial fluid velocity, cm/min
V_{Feed} = velocity of feed injection point, cm/min
z = axial distance, cm

Greek Letters

α = interparticle void fraction
ϵ = intraparticle void fraction
ρ_s = structural density of solid, kg/cm^3

Subscripts

A, B, D = solute species
AVG = averaged solute concentration, eq 17
c = concentration-related value
F = feed
j = conditions before shock wave
$j + 1$ = conditions after shock wave
sh = shock

Literature Cited

Baker, B., Pigford, R. L., *Ind. Eng. Chem., Fundam.,* **10**, 283)1971).
Broughton, D. B., *Chem. Eng. Prog.,* **64** (8), 60 (1968).
de Rosset, A. J., Neuzil, R. W., Korous, D. J., *Ind. Eng. Chem., Process Des. Dev.,* **15**, 261 (1976).
King, C. J., "Separation Processes", McGraw-Hill, New York, N.Y., 1971.
Sherwood, T. K., Pigford, R. L., Wilke, C. R., "Mass Transfer", McGraw-Hill, New York, N.Y., 1975.
Vermeulen, T. G., Klein, G., Hiester, N. K., in R. H. Perry and C. H. Chilton, Ed., "Chemical Engineer's Handbook", 5th ed, Sect. 16, McGraw-Hill, New York, N.Y., 1973.
Wankat, P. C., *Separat. Sci.,* **12,** (1977).

Mechanism of Antifoaming: Role of Filler Particle

R. D. Kulkarni,* E. D. Goddard, and B. Kanner

Union Carbide Corporation, Tarrytown, New York 10591

A model is proposed describing the mechanism of foam bubble rupture by silicone-based antifoams containing finely divided silica as filler. It assumes that the filler particle needs to be hydrophobic for effective antifoaming action and that these hydrophobic particles are primarily responsible for bubble rupture in aqueous systems. Silicone fluids used in the antifoam act mainly as a carrier fluid which transports and exposes the active hydrophobic silica particles to the surface for rupture of the bubble. Rupture involves the creation of a surface chemical shock by rapid adsorption and local immobilization of the foaming surfactant by the hydrophobic particles. In other words, sudden adsorption and local depletion of surface molecules cause bubble rupture. During the process of bubble rupture, the hydrophobic silica particles will, by surfactant adsorption, become hydrophilic and be extracted from the antifoam phase, via the air/water interface, into the aqueous phase. Experimental evidence is described in this paper.

Introduction

Silicone antifoams, used for foam inhibition in aqueous foaming systems, contain essentially two components: (1) polydimethylsiloxane, known as silicone oil, and (2) a small proportion of finely divided filler which is often a fumed or precipitated silica of 100–2000-Å particle size. Even though such compositions have been known and used for over three decades, the reason for their effectiveness is not known nor has the role of each of the components in the foam inhibition process been defined.

In the literature there has, however, been scattered speculation as to the role of the above two antifoam components (Bhute, 1971; Pattle, 1950; Povich, 1975; Robinson and Wood, 1948; Ross, 1950, 1967; Ross et al., 1953; Ross and McBain, 1944; Ross and Young, 1951). It was first believed that the spontaneous spreading of silicone oil over the bubble surface is primarily responsible for bubble rupture (Pattle, 1950; Ross, 1950) and that the presence of silica filler increases the spreading pressure and improves dispersibility of silicone oil simply by reducing the interfacial tension between silicone oil and the aqueous solution. This mechanism was based

purely on speculative ideas and was not confirmed experimentally. Recently, however, Povich (1975) has shown that the silica filler, if anything, slightly lowers the spreading pressure of silicone oil on aqueous solutions. With other factors constant, this would mean a lowering of the antifoaming efficiency which is contrary to experience.

Moreover, even though it is universally agreed that the spreading of the antifoam is necessary for the antifoaming action, there is no evidence in the literature that the spreading of silicone oil is the actual cause of foam bubble rupture. Thus, spreading of antifoam fluid may be a necessary but not sufficient condition for bubble rupture (Kulkarni et al., 1977; Kulkarni and Goddard, 1977). There is much evidence supporting this argument (Bhute, 1971; Pattle, 1950; Ross, 1950; Kulkarni et al., 1977; Kulkarni and Goddard, 1977) and, indeed, a positive spreading coefficient of itself does not necessarily imply favorable foam inhibition (Pattle, 1950; Ross, 1950; Bhute, 1971). This clearly suggests that an alternative mechanism is necessary to explain the phenomenon.

In recent papers (Kulkarni et al., 1977; Kulkarni and Goddard, 1977) we demonstrated that electrical forces can be

Table I

Fluids, silicone oil with additives	Spreading coefficient	Antifoam efficiency
None	7.2	1.0
Silicone derived surfactant	16.8	1.0
Hydrophobic silica	7.2	61.0

an important factor limiting the access of antifoam droplets to the air/water interface. In the present paper we are concerned with the role of silicone oil and filler particles once in the interface and present a model which satisfactorily explains the mechanism of ensuing foam bubble rupture.

Experimental Section

Silicone antifoams were prepared by first mixing silicone oil (Union Carbide product) with 3% hydrophobic silica (Tullenox-500, Tulco Corp.). The fumed silica used was supplied by Cabot Corp. (Cab-o-Sil HS-5).

Stable foams were produced by using (a) 98% sodium dodecyl sulfate, SDS, (E-M Laboratories Inc.), or (b) potassium oleate soap (Eli Lilly and Co., 70% active in ethanol), or (c) TERGITOL 15-S-9, an ethoxylate of a secondary C_{11-15} alcohol (Union Carbide Corp.).

The antifoams were tested using a standard test procedure described elsewhere (Kulkarni et al., 1977). In this test, 250 mL of foaming solution is aerated, at constant gas flow rate through a glass frit, in a 1000-mL graduated cylinder and the rate of foaming with and without the antifoam is noted. The antifoam efficiency is calculated as the ratio of foaming rate without antifoam to that with antifoam.

The surface and interfacial tension of various solutions was measured by a Wilhelmy plate method. The details of these measurements are described elsewhere (Kulkarni et al., 1977).

Results and Discussion

Our initial research on the antifoaming phenomenon revealed the following features. (1) Silicone oil alone in most cases does not act as a foam inhibitor in aqueous foaming systems. (2) The combination silicone oil/hydrophilic silica (Cab-O-Sil), at normally used ratios (~97/3), is ineffective as an antifoam unless it is heat treated at 150 °C for ca. 2 h. Under these conditions the silicone oil presumably reacts with the silica surface. (3) A mixture of silicone oil and hydrophobic fine silica is effective as an antifoam even without heat treatment. (4) Hydrophobic fine silica alone is ineffective as a foam *inhibitor*, but is capable of *defoaming* aqueous foams.

These observations clearly show that the presence of hydrophobic fine silica is necessary in the silicone oil for foam inhibition in aqueous foaming systems and that neither hydrophobic silica nor silicone oil alone can act as effective foam inhibitor but their combination does. This suggests that these components perform different functions in the foam inhibition process which are complementary to each other.

The above aspect is further illustrated by Table I. In this table the spreading coefficient of silicone oil, with and without additive, on a 0.5% sodium dodecyl sulfate solution is compared with the antifoaming action in this system. It is seen that, even though under all conditions the spreading coefficient is positive, there is no apparent correlation between it and antifoaming efficiency. Only the silicone oil containing hydrophobic silica showed significant antifoaming action, indicating that the hydrophobic silica filler plays a vital role in the antifoaming action.
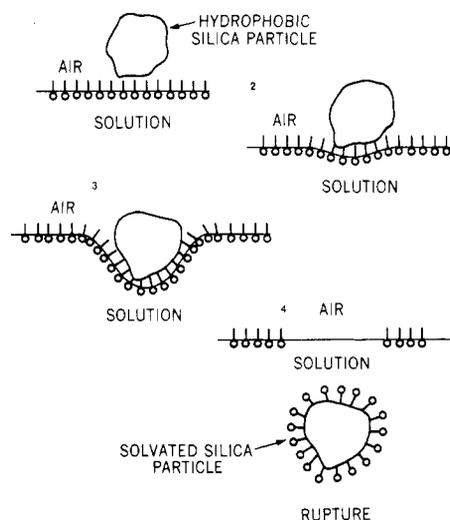
Figure 1. Bubble rupture by hydrophobic silica particle.

Role of Hydrophobic Silica in Antifoaming

If the filler particle is primarily responsible for bubble rupture, then one should be able to observe antifoaming action using the filler alone without silicone oil. The following experiments revealed that hydrophobic fine silica alone can indeed break foam.

(1) Spraying hydrophobic silica over a preformed foam column of potassium oleate soap solution rapidly destroyed the foam bubbles which were contacted by silica, while a spray of hydrophilic silica, such as Cab-O-Sil, under similar conditions did not cause foam breakage.

(2) A foam column was obtained when 100 mL of 0.1% SDS was shaken with 0.1 g of hydrophobic silica; i.e., the silica does not inhibit foam formation. However, the foam was rather unstable and it collapsed completely in less than 2 min. Under similar conditions, when hydrophobic silica was replaced by 0.1 g of silicone oil, a stable foam column was obtained which did not collapse even after 10 min. This was also the case in the presence of hydrophilic silica.

(3) In a glass tray containing aqueous SDS foam, addition of a drop of silicone oil did not cause any foam destruction. Sprinkling hydrophobic silica over the tray instantly destroyed the bubbles which came in contact with these particles. This effect was not seen when hydrophilic or partially hydrophobic silica was sprayed over the foam. Finally, the introduction of a drop of a formulated silicone antifoam was also seen to cause destruction of bubbles.

These experiments show clearly that the hydrophobic silica alone can cause bubble rupture. It can also destroy foam masses, but somewhat inefficiently. The effectiveness of the foam breaking process is limited by the inability of the solid particles to come into contact with the bubbles rapidly. Under these conditions incorporation of the hydrophobic silica particles into a fluid of positive spreading coefficient will aid the mobility of the particles and hence will improve their antifoaming action.

Mechanism of Bubble Rupture by Filler Particle

The phenomenon of bubble rupture by the fine hydrophobic filler particles can be explained on the basis of surfactant adsorption onto the particle as represented in Figure 1. The filler particle is first transported and exposed to the interface by the silicone oil which acts as a very efficient carrier and spreading vehicle. Once the filler is exposed to the bubble surface, it will tend to adsorb surfactant causing its own surface to become hydrophilic. When the particle is sufficiently

hydrophilic it will be extracted into the aqueous phase and a net transfer of silica from antifoam phase into the aqueous phase occurs. We postulate that if the local depletion of surfactant at the interface is rapid, the resulting surface stress renders the interface unstable and rupture occurs. The overall process can be represented as

$$\text{(silicone oil + silica}_{(o)}\text{)} + \text{air bubble} \xrightarrow{\text{spreading} \rightarrow \text{bubble rupture}} \text{silicone oil} + \text{silica}_{(aq)}$$

where silica$_{(o)}$ refers to silica particles in the antifoam phase and silica$_{(aq)}$ refers to silica particles in the aqueous foaming solution. This mechanism suggests that, as antifoaming proceeds, there will be a buildup of hydrophilic silica in the aqueous solution and therefore one should be able to isolate this silica. Indeed, using a centrifuge, we have been able to separate silica from solutions in which antifoaming has proceeded for a prolonged time.

The inability of filler particles alone to act as a foam inhibitor is due to the fact that these particles will adsorb surfactant and become hydrophilic as soon as they come into contact with the aqueous solution. In this way these particles become deactivated even before coming into contact with a bubble surface. Thus, an important role of the silicone oil is to protect the hydrophobic silica particles from contacting the surfactant until exposure occurs at the actual bubble surface. The following two findings give further experimental evidence of the hypothesis.

(1) No silica transfer into the aqueous phase is observed, even over a period of days, when hydrophobic silica is spread over the surface of double distilled water. The silica remains floating on top of the water. However, slow but steady extraction of hydrophobic silica from the surface into the solution occurs when such silica is sprinkled over a 0.5% SDS solution. This shows that the process is associated with surfactant adsorption; it can be significantly accelerated if one produces foam in the system. The process can be followed simply by examining the solution turbidity. The ability of hydrophobic silica to adsorb surfactant was demonstrated directly by exposing the powder to solutions of radiotagged SDS. Under the same conditions, hydrophilic silica showed no adsorption.

(2) In one test, 400 ppm of formulated silicone/silica antifoam was shaken well with 250 mL of 0.5% SDS solution and foamed by passing air at a rate of 450 mL/min at STP through a coarse glass frit. This is equivalent to the production of nearly 8×10^6 bubbles/min. As foaming and antifoaming proceeded, the solution became increasingly cloudy. The bulk of the antifoam liquid itself tended to coagulate and separate from the system as aggregated large drops just below foam level. Development of turbidity did not occur if the same procedure was repeated but without any foaming surfactant, i.e., with no antifoaming taking place. Under these conditions only antifoam aggregation and gradual clearing of the solution was observed. Lastly, it was confirmed that the change in the turbidity occurs in only those systems where effective foam inhibition occurs and in which hydrophobic silica is present.

It is likely that a similar mechanism to that proposed above operates for traditional antifoams which are based on animal fats, for example, lard. At use temperature, which is generally close to their melting point, these fats are composed of a liquid triglyceride phase and a finely dispersed solid triglyceride phase. According to the above model, the liquid component would serve the role of carrier fluid and spreading agent, and the (normally finely divided and hydrophobic) solid particles the role of the actual foam breaker. This is probably the reason why animal fats act most effectively as antifoams around their melting point where solid phase does exist and also why highly purified fats, exhibiting sharp melting points, are rather ineffective as antifoamers. It should be pointed out that triglyceride antifoams are not expected to attain the efficiency of silicone based antifoams because of their much lower spreading coefficient.

Overall Picture

The overall picture which emerges from this analysis is that the hydrophobic silica filler is the actual foam breaker. The silicone oil acts as an efficient carrier fluid which protects the potent filler particles from coming into contact with surfactant molecules until they are transported to the surface of the foam bubble where the true antifoam reaction takes place. The uniqueness of silicone oil lies in its inertness and its ability to spread spontaneously on almost any aqueous fluid, except those containing fluorocarbon surfactant, and to maintain its dispersed state in a foaming solution. The action of silica particles as a foam breaker is primarily dependent on their hydrophobic character. Higher degrees of hydrophobicity not only give more potency to the filler particle but also assist dispersion in the silicone oil. Thus the combination of silicone oil and highly hydrophobic silica particles forms a universal antifoam which can perform effectively in most foaming systems.

Conclusions

In silicone oil based antifoams the hydrophobic silica filler particles are primarily responsible for the foam bubble rupture. Silicone oil acts as an excellent carrier fluid. It transports the hydrophobic silica particles to the foam bubble surface and, by spreading, releases these potent antifoaming particles into the interface.

It is proposed that the hydrophobic silica causes bubble rupture by adsorbing surfactant molecules, leading to local depletion of surfactant at the bubble surface and causing surface shock and bubble instability. During this process the silica particle itself becomes hydrophilic and is therefore deactivated. Thus bubble rupture results in the extraction of silica particles from the antifoam phase into the aqueous phase, which, in this way, develops turbidity owing to suspended silica particles. The ability of the foam to self-heal will increase as the foaming surfactant concentration increases, so providing another explanation of why antifoam efficiency diminishes as the surfactant concentration increases, especially beyond the critical micelle concentration (Kulkarni et al., 1977; Kulkarni and Goddard, 1977).

Ross and Nishioka (1977) have independently recognized the essential role of hydrophobic silica particles in the surface during the defoaming process but make no specific mention of the importance of adsorption of foaming surfactant into the silica particles, nor of the unique role of silicone oil as a carrier fluid.

Literature Cited

Bhute, R. S., *J. Sci. Ind. Res.*, **30**, 241 (1971).
Kulkarni, R. D., Goddard, E. D., Kanner, B., *J. Colloid Interface. Sci.*, **59** (3), 468 (1977).
Kulkarni, R. D., Goddard, E. D., "Droplet/Foam Bubble Interactions As Applied to Antifoaming", to appear in "Croatica Chemica Acta", 1977.
Pattle, R. E., *J. Soc. Chem. Ind. (London)*, **69**, 363 (1950).
Povich, M. J., *AICHE J.*, **21** (5), 1016 (1975).
Robinson, J. V., Woods, W. W., *J. Soc. Chem. Ind. (London)*, **67**, 361 (1948).
Ross, S., *J. Phys. Colloid Chem.*, **59**, 429 (1950).
Ross, S., *Chem. Eng. Prog.*, **63** (9), 41 (1967).
Ross, S., Hughes, A. F., Kennedy, M. L., Mardoian, A. R., *J. Phys. Chem.*, **57**, 684 (1953).
Ross, S., McBain, J. W., *Ind. Eng. Chem.*, **36**, (6), 570 (1944).
Ross, S., Nishioka, G., Abstracts of Papers 51st Colloid and Surface Science Symposium, Grand Island, N.Y., June 1977.
Ross, S., Young, G. J., *Ind. Eng. Chem.*, **43** (11), 2520 (1951).