Aspects of the Interaction of Silicic Acid with Aluminium in Dilute Solution and Its Biological Significance

J. S. CHAPPELL* and J. D. BIRCHALL

Imperial Chemical Industries p.l.c., P.O. Box 8, The Heath, Runcorn WA7 4QE, Cheshire, U.K.

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Aluminium is implicated in the toxicity of acidified waters to fish [1] and the exposure of humans to it in renal dialysis is known to promote osteodystrophy and a fatal encephalopathy [2]. In Alzheimer's disease, aluminium has been found within diseased neurons [3] and amorphous aluminosilicates have been reported at the core of the senile plaques characteristic of this disease [4]. Much work has been carried out on the speciation of aluminium in aqueous solution in response to pH and on its complexation with ligands which may be important in natural waters and in biology. However, little consideration has been given to the reaction of aqueous aluminium species with silicic acid, $Si(OH)_4$†, the dissolved form of the abundant element silicon.

The affinity of silicic acid for aluminium is a unique one in chemistry [5], owing to ionic size, charge, and coordination geometry of the species involved [6]. The chemistry of aluminosilicates generally has been concerned with the solid state (minerals such as clays, feldspars and zeolites) [7], and with highly concentrated and alkaline solutions (as in zeolite synthesis) [8], but not with dilute solutions at near neutral pH. Aluminosilicates are highly insoluble near neutral pH, but as regards their precipitation under dilute conditions, the kinetics of colloid formation can be quite slow and the solution precursors to the solid phase may be surprisingly metastable. When equilibrium is approached, the solubility levels vary with the aluminosilicate phase but fall in the range of 0.05–0.28 μmol/l Al and 18–210 μmol/l Si [9, 10]. These soluble species are usually regarded as simple hydroxyaluminium ions and silicic acid. The soluble concentrations of these two elements in natural waters (mean value for world rivers at 15 μmol/l Al, 218 μmol/l Si) [11] and in biological fluids (human plasma levels at 0.06–0.54 μmol/l Al, 14–39 μmol/l Si) [12, 13] fall at or above the saturation values, so aluminosilicate formation could occur in these environments. The interaction of silicic acid with aluminium may therefore be of biological importance in the transport, binding, availability and toxicity of aluminium.

Experimental

The solutions studied were composed of aluminium chloride (0.10 mmol/l) with and without silicic acid (0.50 mmol/l) present in a background concentration (10 mmol/l) of either NaCl or $NaHCO_3$. These solutions were prepared fresh and allowed to age for 20 h at a set pH to allow a metastable condition to be established. The reagents, except for one, were all of spectroscopic grade quality with impurity levels <3 ppm. The silicic acid was prepared by passing a solution (2.0 mmol/l) of reagent grade sodium orthosilicate over a sulphonate functional ion exchange resin in the H^+ form. This treatment converted the silicate to silicic acid as well as removing any metal impurities.

Colloidal solids were measured by filtering solutions through 0.22 μm-pore membrane filters. The membrane is composed of hydrophilised poly(vinylidene difluoride) and was chosen for its reasonably selective removal of particles >0.2 μm. Many filter membranes will remove species much smaller than the quoted pore size‡. The composition of the filtered solids was determined by digesting the solids in 3 mol/l HCl and analysing this extract solution.

An ion exchange technique was used to examine sub-colloidal solution species by employing ion exchange resins with either sulphonate or iminodiacetate functional groups. These ligating groups can bind aluminium-based species of a small polymeric nature, but the nature of the resin is such that species of ~0.1 μm or greater can not access the groups. Solutions under study were passed through a 'mini-column' containing ~0.3 ml of resin (as a cylindrical bed of 4 mm diameter and 20 mm height). The functional groups were neutralized to the desired pH with a rinse solution (10 mmol/l) of NaCl or $NaHCO_3$ prior to passing the test solution at a rate of 3 ml/min. The species retained on the resin were then extracted by passing 3 mol/l HCl at a rate of 1 ml/min. The small bed volume enabled a minimal amount of HCl solution to be used in order to maximize the concentrations in the extract solution whilst still providing a 10-fold excess in binding groups.

Analysis of the extract solutions was conducted by atomic absorption spectroscopy with an instrument employing an inductively coupled plasma to heighten sensitivity.

*Author to whom correspondence should be addressed.

†The principal species at neutral pH is the acid form (since pK_a is 9.8), and it is predominantly monomeric at concentrations <2 mmol/l.

‡Some membranes, such as cellulose acetate, remove species as small as 0.01 μm.

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Results and Discussion

The formation of colloidal solids as a function of solution pH is shown in Fig. 1 for solutions in a background of 10 mmol/l NaCl. Both curves display a minimum near pH ~6 as is expected for aluminium hydroxide and some aluminosilicate precipitates. However, the curves are well removed from equilibrium where the soluble aluminium would fall below 1 μmol/l for $5.5 < pH < 8.0$. These results illustrate that the growth to micron size of both hydroxyaluminium and aluminosilicate species in dilute solution is exceedingly slow. Curiously, the more stable aluminosilicate phase is actually slower to condense than the aluminium hydroxide phase. The colloid which does form is found to possess a Si:Al ratio of 0.3–0.6. This composition is consistent with the aluminosilicate phase of imogolite or its amorphous precursor protoimogolite studied in soil science [14].

The solution species giving rise to these solids are confirmed to be aluminosilicate by the ion exchange technique using a resin with sulphonate groups. Silicate is found to be associated with the aluminium retained on the resin for $pH > 5.5$. The Si:Al ratio of these retained species is in the range of 0.25–0.35, which agrees with that of amorphous protoimogolite. These species are metastable and do eventually aggregate into an insoluble phase.

The ion exchange results thereby complement the filtration results since the resin can bind some of the solution species which pass through the membrane filter. This is evident in Table I, where the aluminium retained by iminodiacetate groups is listed for solutions at pH 6.0. The fraction of aluminium retained from solutions with a NaCl background is qualitatively consistent with the filtration results in Fig. 1. The amount of aluminium as solution species is indicated to be greater in the presence of silicic acid. Interestingly, a background of $NaHCO_3$ dramatically increases the retainable aluminium for both hydroxyaluminium and aluminosilicate species. The effect is transient, and colloidal solids do start to form in both solutions after a few days. This result does indicate that bicarbonate increases the metastability of the solution precursors of both aluminium-based insoluble phases, and suggests that a significant interaction occurs between aluminium and bicarbonate in aqueous solution. A solubilizing effect by CO_2 has been indicated in geochemistry [15], although generally any direct interaction between aluminium and bicarbonate species is regarded as negligible. This effect is potentially important in biological fluids where bicarbonate concentrations can be high (human plasma at 2–53 mmol/l) [16] and the hydroxyaluminium or aluminosilicate species are dilute.

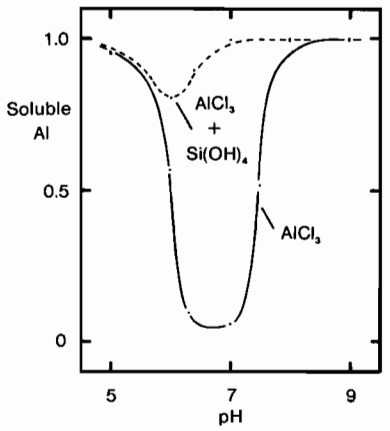

Fig. 1. Fraction of aluminium remaining in solutions (age 20 h) after filtration (0.2 μm pore) as a function of solution pH. Solutions contain aluminium (0.1 mmol/l $AlCl_3$) with and without silicic acid (0.5 mmol/l $Si(OH)_4$) in a background concentration of chloride (10 mmol/l NaCl).

The metastable solution species present in a bicarbonate background were studied as a function of pH by the ion exchange technique. Their retention by iminodiacetate groups is illustrated in Fig. 2. The retention of aluminium is plotted as Fig. 2a, where the two curves display a similar drop with increasing pH. The presence of silicic acid shifts the curve to lower pH as the interactions with silicic acid begin to interfere with the binding of the hydroxyaluminium species to the resin at $pH > 6$. This is attributed to the formation of stable aluminosilicate species as evident in Fig. 2b where the composition of the retained species reveals that silicate is coordinated with the retained aluminium at $pH > 7$. The Si:Al ratio attains a maximum level (0.25–0.35) which is consistent with the composition of the colloidal phase. This behaviour appears to reflect a relative stability for the aluminosilicate species in the presence of iminodiacetate groups.

The transition in stability observed in Fig. 2 is consistent with the stability known for the coordination between aluminium and carboxylate ligands. At physiological concentrations, the coordination of aluminium to citrate becomes unstable relative to hydroxy species at $pH > 7.5$ [17]. In the presence of silicic acid, aluminosilicate formation is favoured over the hydroxy species near neutral pH and this

TABLE I. Fraction of Aluminium Retained by Iminodiacetate Functional Groups from Solutions of Age 20 Hours at pH 6.0

Solution composition	Al retained
0.1 mmol/l $AlCl_3$ in 10 mmol/l NaCl	0.14
0.1 mmol/l $AlCl_3$, 0.5 mmol/l $Si(OH)_4$ in 10 mmol/l NaCl	0.29
0.1 mmol/l $AlCl_3$ in 10 mmol/l $NaHCO_3$	0.99
0.1 mmol/l $AlCl_3$, 0.5 mmol/l $Si(OH)_4$ in 10 mmol/l $NaHCO_3$	0.98

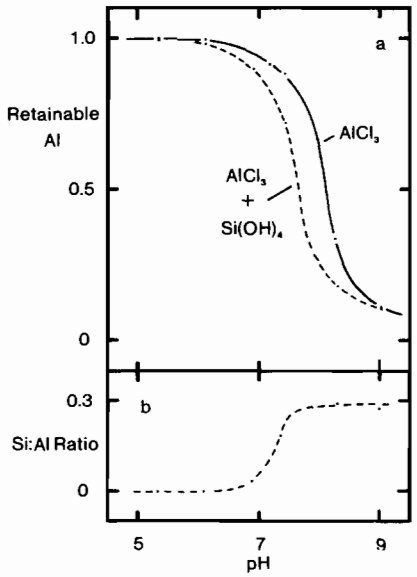

Fig. 2. (a) Fraction of aluminium retained from solutions (age 20 h) by an ion exchange resin (iminodiacetate functional group) as a function of solution pH. (b) Si:Al ratio of the retained species *vs.* pH. Solutions contain aluminium (0.1 mmol/l $AlCl_3$) with and without silicic acid (0.5 mmol/l $Si(OH)_4$) in a background concentration of bicarbonate (10 mmol/l $NaHCO_3$). Data points were reproducible among several determinations.

increase in stability would dictate carboxylate coordination becoming unstable relative to silicate coordination at a lower pH. This is what is observed in Fig. 2a, where the drop in aluminium retention is shifted to lower pH with the addition of silicic acid. The shift is particularly significant since it falls in the range of physiological pH (6.6–7.4). According to Fig. 2b, the transition from carboxylate to silicate coordination is at pH ~7. Consequently, the formation of aluminosilicate species is not necessarily prevented by organic complexing agents at pH 7.4 as is generally believed [18]. Citrate is observed to prevent the formation of colloidal aluminosilicate solids although solution species still exist at physiological pH. Solutions of aluminium chloride (0.10 mmol/l) with equimolar concentrations (0.50 mmol/l) of citrate and silicic acid at pH 7.4 yield no colloidal solids after 12 weeks ageing, yet 20-hour old solutions reveal species (by the ion exchange technique) with a Si:Al ratio >0.5.

The present work shows that metastable solution species of aluminosilicate form in dilute solutions of aluminium and silicic acid from pH 5.5 upwards, and their polymerization and precipitation are further retarded by bicarbonate. The transition in aluminium coordination with carboxylate ligands to silicate at pH ~7 is an important feature of the chemistry, as stable species of aluminosilicate can exist at pH 7.4 whilst in the presence of citrate. The stability of these sub-colloidal aluminosilicate species suggests that silicic acid which is present in natural waters is a ligand that cannot be ignored in considering the transport, bio-availability, and toxicity of aluminium in the environment [19].

Aluminium is known to be neurotoxic [20] and is transported in plasma bound to transferrin [17, 21]. The binding of Fe^{3+} in transferrin is known to require the concomitant binding of bicarbonate ion [22], and it is interesting that a significant interaction between bicarbonate and hydroxyaluminium species is observed here. In plasma, aluminium will be bound to transferrin with silicic acid free in solution. Aluminosilicates will form and deposit in focal areas within which aluminium is free as $Al(OH)_4^-$ or weakly bound and silicic acid is present, a condition that may apply at the nucleus of senile plaques in Alzheimer's disease. The damage wrought by aluminium is possibly brought about by aluminium within the neuron and bound to phosphate [23]. Aluminosilicates at the core of plaques appear then to be the result of the export of aluminium from diseased cells to the extracellular space in which its combination with silicic acid is possible.

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