Studies on the bioleaching of refractory concentrates

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Synopsis

The bioleaching of arsenopyrite, the oxidation of arsenate and the precipitation of ferric arsenate are competing reactions, hence their rates depend on the relative concentrations of the respective species, and the activity of the bacterial culture. Bacterial arsenic resistance may be attributed to the Pst+ Pit- mutation and an energy dependent efflux pump. Pst+ Pit- mutations result in a reduced uptake of arsenate. This enables the bacteria to survive in solutions in which the arsenate concentration is significantly higher than the arsenite concentration. However, the excretion of arsenate requires energy. Therefore, in the absence of an energy source or during periods of reduced bacterial activity, the inhibitory effect of arsenate may manifest at concentrations to which the culture has been adapted.

The chemical ferric leaching rate of pyrite and arsenopyrite decrease with a decrease in the solution redox and may be described using a modified Butler-Volmer equation. The bacterial ferrous-iron oxidation kinetics of *L. ferrooxidans* also depend on the redox potential, and can be described using a Michaelis-Menten type model modified to account for both temperature and pH.

A model developed using the independently determined ferric leaching, and ferrous-iron oxidizing kinetics, has been shown to compare well with the experimental data for the continuous bioleaching of pyrite.

Keywords: *Leptospirillum ferrooxidans*, arsenopyrite, bioleaching, sulphide minerals ferrous-iron oxidation kinetics, ferric leaching kinetics, redox potential

Introduction

Bioleaching is now an established technology for the pre-treatment of refractory gold ores and concentrates and the leaching of whole-ore copper heaps. In many cases, it offers economic, environmental and technical advantages over pressure oxidation and roasting[1,2]. However, in order for bioleaching to compete successfully with other pre-treatment processes it needs to be optimized with regard to the parameters that affect the process. Furthermore, there is a need for mechanistically based kinetic models that can be used to derive performance equations for use in the design, optimization and control of bioleaching processes.

The modelling of bioleach reactors is complicated by the nature of microbial interactions and difficulties encountered when attempting to measure the biomass concentration and growth rate in the presence of solids, and the ferric and ferrous-iron concentrations. This has resulted in the logistic Equation [3] being the rate expression that has found the most widespread application to date. Although not mechanistically based the logistic Equation has proved useful in modelling batch, continuous pilot and full-scale plant data for several pyrite and pyrite-arsenopyrite flotation concentrates[4–9].

Recent research has provided strong evidence that the bioleaching of sulphide minerals occurs via a multiple sub-process mechanism[10]. By staged additions of pyrite to a batch bioleach, Boon *et al.*[10] were able to measure the bacterial specific oxygen utilization rate as a function of the ferric/ferrous-iron ratio or redox potential. The oxygen utilization rate was also related to the pyrite concentration via the pyrite specific oxygen utilization rate. The results showed that the bacterial specific rate of oxygen utilization, decreased with increasing ferric/ferrous-iron ratio while the pyrite specific oxygen utilization rate increased with increasing ferric/ferrous-iron ratio.

In addition, samples were taken from the batch, the pyrite removed by centrifugation, and the specific oxygen utilization rate of the bacteria measured in an off-line respirometer, using ferrous-iron medium. This enabled the specific oxygen utilization rate to be measured over a wider range of ferric/ferrous-iron ratios than in the pyrite batch. However, in the region where the ranges overlapped, the data for the pyrite and ferrous-iron grown bacteria coincided. From this it was concluded that in both cases ferrous-iron was the primary

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 SA ISSN 0038–223X/3.00 + 0.00. First presented at the SAIMM conference Metallurgy Africa*, Nov. 1999.*

substrate, and that the bioleaching of pyrite occurs via a multiple sub-process mechanism. Therefore, for the case of pyrite bioleaching the primary role of the bacteria is the oxidation of the ferrous-iron to the ferric form, thereby maintaining a high redox potential within the system and ensuring the continued leaching of the mineral.

A multiple sub-process mechanism for the bioleaching of sulphide minerals suggests that the overall process can be reduced to a number of independent sequential and/or parallel sub-processes. The kinetics of these sub-processes may be studied separately, and the results used to predict the performance of bioleaching reactors for a variety of operating conditions.

The existence of a multiple sub-process mechanism for the bioleaching of sulphide minerals is supported by:

- the identification of *Leptospirillum ferrooxidans* as the dominant ferrous-iron oxidizing species in a continuous bioleaching mini-plant oxidizing an arsenopyrite/pyrite flotation concentrate[11], and
- the identification of *L. ferrooxidans* and *Thiobacillus caldus* in continuous flow bioleaching oxidation tanks used to treat a variety of metal containing ores and concentrates[12].

According to the multiple sub-process mechanism the sulphide mineral is chemically oxidized by ferric-iron:

$$FeS_2 + 14Fe^{3+} + 8H_2O \rightarrow 15Fe^{2+} + 2SO_4^{2-} + 16H^+ \quad [13] \quad [1]$$

$$FeAsS + 5Fe^{3+} \rightarrow As^{3+} + 6Fe^{2+} + S^0 \quad [14\pm16] \quad [2]$$

The ferrous-iron produced by these reactions is subsequently oxidized to ferric-iron by ferrous-iron oxidizing micro-organisms:

$$4Fe^{2+} + O_2 + 4H^+ \rightarrow 4Fe^{3+} + 2H_2O \quad [17] \quad [3]$$

Polysulphides and sulphur or thiosulphate produced by the ferric leaching reactions are oxidized to sulphate by sulphur oxidizing micro-organisms[18]. Diagrammatic representations of the bioleaching of both pyrite and arsenopyrite are shown in Figure 1.

The aim of this paper is to present the results of recent studies on the bioleaching of arsenopyrite and pyrite in an attempt to identify areas which require further research in order to develop a model which can be used to accurately describe the kinetics of the process.

The bioleaching of arsenopyrite

From Equation [2] it is clear that the bioleaching of arsenopyrite solubilizes the arsenic and iron as arsenite, As^{3+}, and ferrous-iron, Fe^{2+}, respectively. Depending on the conditions employed, the dissolution of arsenopyrite may be followed by the oxidation of arsenite to arsenate, As^{5+}, according to:

$$H_3AsO_3 + 2Fe^{3+} + H_2O \rightarrow H_3AsO_4 + 2Fe^{2+} + 2H^+ \quad [20] \quad [4]$$

The oxidation of arsenite to arsenate may in turn be followed by the precipitation of ferric arsenate, according to:

$$2H_3AsO_4 + Fe_2(SO_4)_3 \rightarrow 2FeAsO_4 + 3H_2SO_4 \quad [9] \quad [5]$$

High concentrations of dissolved arsenic inhibit bacterial growth[20–23]. Therefore, the relative rates at which the reactions shown in Equations [2] [4] and [5] occur may influence the activity of the bacterial culture, and hence the rate of bioleaching. Recent work has shown that the reactions shown in Equations [2] [4] and [5] are competing reactions, and are therefore influenced by the concentrations of arsenite, arsenate, ferric-iron and arsenopyrite. Furthermore, the oxidation of arsenite to arsenate requires the presence of a mineral less noble than arsenopyrite[24]. At low ferric-iron concentrations (viz. prior to rapid bacterial activity) and low arsenite concentrations, the chemical leaching of arsenopyrite (Equation [2]) is the dominant reaction. At low ferric-iron concentrations and high arsenite concentrations, the abundance of arsenite in solution causes the oxidation of arsenite (Equation [5]) to dominate. However, at high ferric-iron concentrations (i.e. during periods of rapid bacterial growth), there is sufficient ferric-iron in solution to oxidize both the arsenopyrite and the dissolved arsenite. This results in the low arsenite and high arsenate concentrations observed during continuous bioleaching operations[25].

Arsenite has been reported to inhibit a wide range of microorganisms[23,26–29], including *Thiobacillus ferrooxidans*[21,30,31], *T. thiooxidans*[30], and the mixed culture used in BIOX® operations to a greater degree than arsenate[32]. Arsenite deactivates enzymes with thiol (HS) groups at the active centre[33]. Arsenate toxicity is caused by its similarity to phosphate[29,33]; arsenate replaces the phosphate in ATP to form an unstable ADP-arsenate complex[33].

The two main forms of arsenic resistance in bacteria are chromosomal arsenate resistance and plasmid determined arsenic resistance[33–36]. Chromosomal arsenate resistance reduces the amount of arsenate entering the cell via the phosphate transport system[33, 35–37]. The Pst phosphate transport system is specific to phosphate, while the Pit system will transport either phosphate or arsenate[33,37,38], hence chromosomal arsenate resistance occurs with the Pst^+ Pit^- mutation[26,33,35,38].

Plasmid-encoded resistance protects bacteria by pumping arsenic from the cells via an energy dependent membrane pump[29,33,35,36,39,40]. In bacteria which exhibit this type of resistance, plasmids encode proteins which form a membrane-bound complex, and interact to form an arsenite pump[41–43]. In some species, the membrane-bound complex is able to reduce arsenate to arsenite, which results in resistance to both arsenate and arsenite. A diagrammatic representation of the phosphate (arsenate) transport system and the arsenate efflux system of *Escherichia coli* is shown in Figure 2.

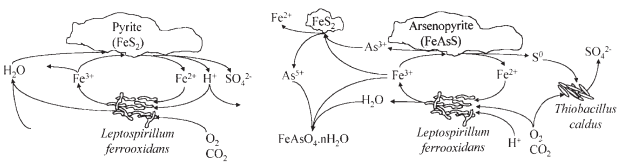

Figure 1—Diagrammatic representation of the bioleaching of pyrite and arsenopyrite via the two-step mechanism.The bacteria shown are planktonic (i.e., unattached) for reasons of clarity; however, the mechanism is the same for both attached and unattached micro-organisms (after Breed *et al*.[19])

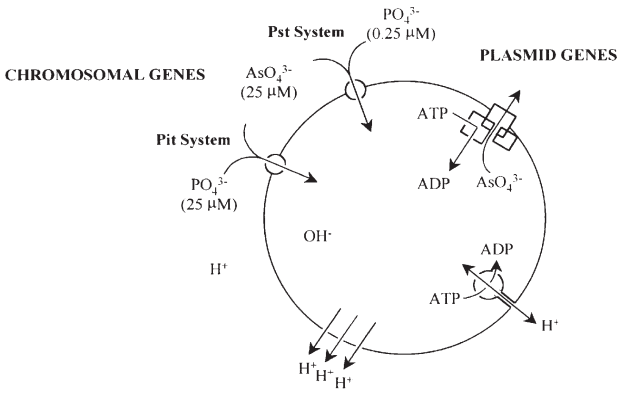

Figure 2—Model for phosphate (arsenate) transport systems and the arsenate efflux system of *Escherichia coli.* 0.25 and 25 μM, are the K_m for PO_4^{3-} and AsO_4^{3-} as a competitor for the Pit and Pst chromosomal transport systems. The three boxes represent the proteins which constitute the arsenite pump (modified from Silver and Nakahara[35] and Silver and Misra[41]) (after Breed[24])

Most researchers have reported that elevated arsenite concentrations result in an extended lag phase after which normal growth occurs[21,23,30,31] whereas elevated arsenate levels cause a reduction in the maximum growth capacity[28]. However, during the batch bioleaching of an arsenopyrite/pyrite concentrate, exposure to elevated arsenite concentrations did not result in an increase in the lag phase, whereas exposure to elevated arsenate concentrations resulted in a significant increase in the lag phase[44]. The results obtained by Breed *et al.*[44] are shown in Figure 3.

From Figure 3 it is apparent that exposure to an initial concentration of 20 mmol $As^{3+}.l^{-1}$ retarded the initial rate of bacterial metabolism. This effect was more pronounced when 40 mmol $As^{3+}.l^{-1}$ was added. The culture exposed to an initial concentration of 20 mmol $As^{3+}.l^{-1}$ appeared to recover from the effect of the added arsenic whereas the culture exposed to an initial concentration of 40 mmol $As^{3+}.l^{-1}$ did not achieve an equivalent maximum oxygen utilization rate (data not shown) and showed a lower cumulative oxygen usage.

Exposure to an initial concentration of 107 mmol $As^{5+}.l^{-1}$ increased the bacterial lag phase by 15 days and retarded the initial rate of bacterial metabolism. Furthermore, the culture did not appear to recover from the effect of the added arsenate over the 31-day period studied. The addition of 220 mmol $As^{5+}.l^{-1}$ to the nutrient solution prior to inoculation resulted in a lag phase which lasted in excess of 31 days; the bacteria exposed to an initial concentration of 220 mmol $As^{5+}.l^{-1}$ did not consume any oxygen for the duration of the experiment.

The rate at which oxygen is utilized by the bacteria, $-r_{O_2}$, is a function of both the bacterial concentration, c_X, and their activity, q_{O_2}:

$$-r_{o_2} = q_{o_2} c_x \qquad [6]$$

The specific oxygen utilization rate, q_{O_2}, can be described using an equation of the form:

$$q_{o_2} = \frac{q_{o_2}^{\max}}{1 + K\left[\frac{Fe^{3+}}{Fe^{2+}}\right]} \qquad [10] \qquad [7]$$

Hence, it was not possible to ascertain whether the arsenic species inhibit bacterial oxidation (i.e. they affect $q_{o_2}^{max}$) or are toxic to the bacteria (i.e. they affect c_X).

Although concentrations of up to 145 mmol $As^{3+}.l^{-1}$ have been reported in actively growing cultures accustomed to high arsenite concentrations[45], several researchers have found arsenite to be toxic at concentrations similar to those shown in Figure 3[21,23]. Furthermore, although some researchers have suggested that arsenite is in the region of 2 to 3 times more toxic than arsenate[21,23], Figure 3 shows that exposure to 107 mmol $As^{5+}.l^{-1}$ had a far more pronounced effect than 40 mmol $As^{3+}.l^{-1}$. It should however, be noted that most researchers have performed toxicity experiments in ferrous-iron media, or in slurries supplemented with ferrous-iron. Because ferrous-iron is the most easily used source of energy for iron-oxidizing bacteria, and because the extent of arsenic inhibition is less pronounced than if the substrate is a sulphide mineral[46,47], the conditions used may have influenced their results.

It is also important to note that the levels of arsenite shown in Figure 3 were in the region of 10 times those observed in the mini-plant[25] from which the inoculii were obtained*, yet where an inhibitory effect was observed, the bacterial culture exhibited the ability to recover. However, the levels of arsenate added to the bioreactors were similar to those observed in the mini-plant, yet the effect of the added arsenate was severe. This result, together with the hypothesis that the ferric leaching of arsenopyrite, the ferric oxidation of arsenite and the precipitation are competing reactions, suggests that the inhibitory effect of arsenate may

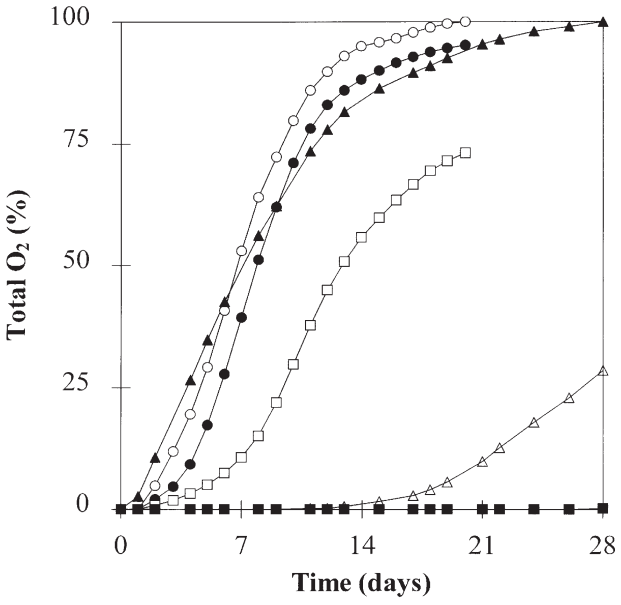

Figure 3— Variation in the cumulative amount of oxygen consumed by the bacterial culture with time at different initial arsenite and arsenate concentrations. [○] 0 mmol $As^{3+}.l^{-1}$; [●] 20 mmol $As^{3+}.l^{-1}$; [❑] 40 mmol $As^{3+}.l^{-1}$; [▲] 0 mmol $As^{5+}.l^{-1}$; [Δ] 107 mmol $As^{5+}.l^{-1}$; [■] 220 mmol $As^{5+}.l^{-1}$; (after Breed *et al.*[44])

*** The same concentrate as used in the mini-plant was used in the batch bioleaching experiments shown in Figure 3**

be linked to the availability of an energy source. At elevated arsenate concentrations the precipitation of ferric arsenate results in no ferric-iron being available for the leaching of the mineral. This results in a reduced availability of ferrous-iron, which in turn results in the inhibitory effect of arsenate becoming apparent at arsenate concentrations to which the culture has been adapted.

The above suggestion is consistent with the results of perturbation studies performed using the continuous bioleaching mini-plant from which the culture used in the experiments shown in Figure 3, were obtained. Two disruption tests were carried out. The first consisted of a 15-minute interruption in the agitation and aeration of the bacterial culture in the secondary bioreactor. This did not have an effect on the short term (or long term) activity and viability of the culture (data not shown).

However, a 17-hour interruption in the agitation and aeration of the culture in the primary bioreactor resulted in a reduced level of bacterial activity that persisted once aeration and agitation had been restored. The variation in the measured oxygen utilization rate for the duration of the perturbation study performed using the primary bioreactor is shown in Figure 4. It is apparent from Figure 4 that the oxygen utilization rate decreased during the period in which aeration and agitation of the culture was stopped and increased on resumption of aeration and agitation. However, the oxygen utilization rate of the culture did not reach the same level of bacterial activity after the restoration of aeration and agitation.

During both the short and long interruptions in the aeration and agitation of the bioreactor, the ferrous-iron and arsenite concentrations increased in the absence of bacterial activity, and decreased once aeration and agitation had been restored. However, the concentrations of these species did not reach greater concentrations than observed during the lag phase of the batch culture to which no arsenic was added (data not shown). Furthermore, the arsenate and ferric-iron concentrations did not increase, relative to their steady-state concentrations, during the course of the experiment[25]. Therefore, the reduced level of bacterial activity after the restoration of aeration and agitation could not be attributed to 'elevated' arsenic concentrations. This in turn suggests that the inhibitory effect must be attributable to the arsenic concentration and speciation present during steady-state operation†.

Therefore, the results shown in Figures 3 and 4 suggest that the mechanism of arsenic resistance in the (mesophilic) bacteria used in the bioleaching of sulphide minerals may be attributed to Pst+ Pit- mutations and an energy dependent efflux pump. The Pst+ Pit- mutations result in a reduced uptake of arsenate which enables the bacteria to survive in solutions in which the dissolved arsenate concentration is significantly higher than the dissolved arsenite concentration. However, the excretion of arsenate which enters the cell, presumably via the phosphate uptake system, requires energy. Therefore, in the absence of an energy source, e.g. ferrous-iron and/or O_2, or during periods of reduced bacterial activity, the inhibitory effect of arsenate may manifest at arsenate concentrations to which the culture has been adapted.

The above results are important if one considers that during commercial bioleaching operations disruptions in the aeration and agitation of the bioreactors may occur as a result of power failures or breakdowns. These perturbations will result in a reduced level of bacterial activity during the interruption, and may have a long-term effect on the viability of the bacterial culture, which in turn may result in a significant loss in production.

The ferric leaching of arsenopyrite

To date a number of models have been used to describe the ferric leaching kinetics of sulphide minerals[48–51]. The simplest model assumes a linear relationship between the redox potential of the solution and the rest potential of the mineral[49]. Other postulated models include those based on electrochemical theory[48,51] and on the Monod equation[50].

Although considerable work on the leaching of pyrite using ferric-iron has been reported in the literature[13,51–53], to date very little work on the leaching of arsenopyrite, using ferric-iron at concentrations and conditions similar to those used in bioleaching, has been reported. However, recent research has shown that the ferric leaching kinetics of pyrite[50,51] and arsenopyrite[16] may be dependent on the ferric/ferrous-iron ratio (i.e. redox potential), and not a function of the total or ferric-iron concentrations.

Boon[50] suggested that the ferric leaching of pyrite could be described by means of a Monod-type equation:

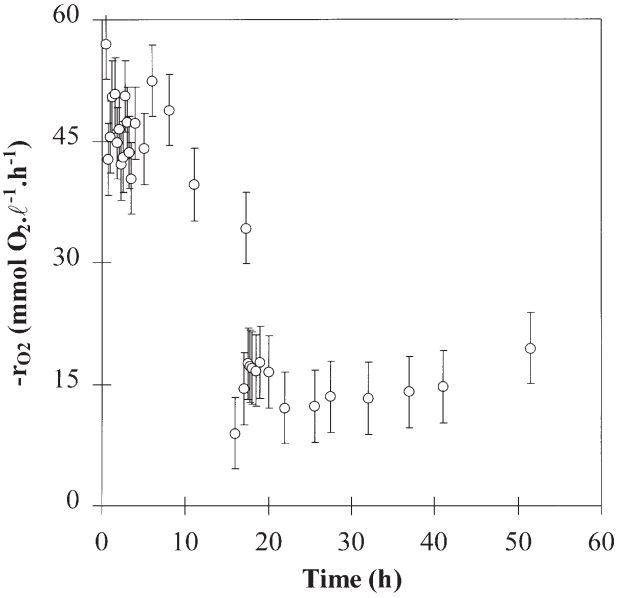

Figure 4—Variation in the oxygen utilization rate during the disruption test carried out using the primary bioreactor. The error bars represent the measured $-r_{O_2}$ ± the average variation in $-r_{O_2}$ measured during steady-state operation, at τ=4 days (after Breed *et al.*[25])

† As the feed to the bioreactor was diverted during, and immediately, after the perturbation, the reduced level of bacterial activity could not be attributed to washout. In addition, the residence time chosen once continuous operation was begun, i.e. after the perturbation, was varied to ensure that the bacteria were neither substrate limited, nor 'washed out' of the bioreactor

$$\upsilon_{Fe^{2+}} = \frac{\upsilon^{\max}_{Fe^{2+}}}{1 + B\left[\frac{Fe^{2+}}{Fe^{3+}}\right]} \quad [8]$$

However, May *et al.*[51] suggested that the ferric leaching of pyrite was an electrochemical (corrosion) phenomena, and hence chose to use an equation similar in form to the Butler-Volmer equation to describe the leaching rate:

$$-r^{chem}_{Fe^{2+}} = r_0\left(e^{\alpha\beta(E\pm E')} - e^{(1-\alpha)\beta(E-E')}\right) \quad [9]$$

The leach rates of the pyrite were calculated using the measured variation in the redox potential and the initial total iron concentration. According to the Butler-Volmer equation the dependence of the ferric leaching kinetics on the overpotential is linear at low overpotentials (0-20 mV). However, most bioleaching plants operate at higher overpotentials, which result in non-linear kinetics. In addition, May *et al.*[51] showed it was possible to achieve leaching rates in sterile media which were comparable with those achieved in bioleaching, provided that the redox potential was comparable with those observed in bioleaching.

The variation in the ferric leaching rate of arsenopyrite with changes in the solution redox potential has been shown to display the same trends, irrespective of the experimental conditions employed[54]. In the experiments performed by Ruitenberg *et al.*[54] the ferric leaching rate of arsenopyrite initially increased with a decrease in the redox potential, reached a maximum, and then decreased with a further decrease in the redox potential. An increase in the initial ferric leaching rate with decreasing redox potential was also observed during the ferric leaching of pyrite[51].

It is therefore suggested that this is a transient phenomenon and can be attributed to the rearrangement of the ions on the surface of the mineral and in the electrical double layer surrounding the mineral. It is not a result of the leaching of the mineral itself. This postulate is supported by the fact that the process was very rapid† and by observations made during studies on the effect of the ferric-iron concentration on the electrophoretic mobility of arsenopyrite[55]. Furthermore, no surface products responsible for passivation of the mineral surface were observed, nor was a decrease in reactivity observed when mineral that had been leached previously was leached for a second time. In addition, although previous workers have detected a sulfur layer on the mineral surface after both the acid, and the ferric leaching of arsenopyrite, it has not been found to hinder the dissolution reaction[14,56–58].

The decrease in the rate of leaching with a decrease in the redox potential of the solution observed for most of the experiment is in agreement with previously reported trends for the ferric leaching of sulphide minerals[51,59–61]. This suggests a dependency of the ferric leaching rate on the redox potential of the leaching solution, which in turn suggests that an electrochemical model can be used to describe the ferric leaching kinetics of arsenopyrite.

An electrochemically driven reaction should exhibit a half order dependence on the ferric-iron concentration[62]. However, the ferric leaching rate of pyrite was found independent of the total iron concentration[51], for iron concentrations ranging from 50 to 500 mmol Fe.l^{-1}. However, the ferric leaching rate of arsenopyrite did not exhibit a half order dependence on the ferric-iron concentration, nor was it found independent thereof. In addition, it was not possible to fit the electrochemically-based model proposed by Verbaan and Crundwell[48], or the Monod-type model proposed by Boon[50]. However, it was possible to model the ferric leach kinetics using the Butler-Volmer based model suggested by May *et al.*[51] (Equation [7]). Comparison of the Butler-Volmer based model prediction with a typical set of experimental results is shown in Figure 5. From Figure 5 it is apparent that the agreement between the Butler-Volmer based model and the experimental results is good.

Although it was possible to model the ferric leach kinetics of arsenopyrite across a wide range of conditions using this model, a limitation of the model appears to be its dependence on the rest potential of the mineral, E´§. The rest potential of arsenopyrite was found to increase when the initial (starting) redox potential or total iron concentration was increased, and decreased when the solids concentration or pH was increased. The results therefore suggest that an increase in the concentration of either ferric-iron or protons (based on the arsenopyrite surface area) results in a reduction in the reactivity of the mineral. This is regarded as highly unusual as most reaction mechanisms are favoured by an increase in reactant concentration.

Although the underlying mechanism responsible for the observed influence of the different parameters on the

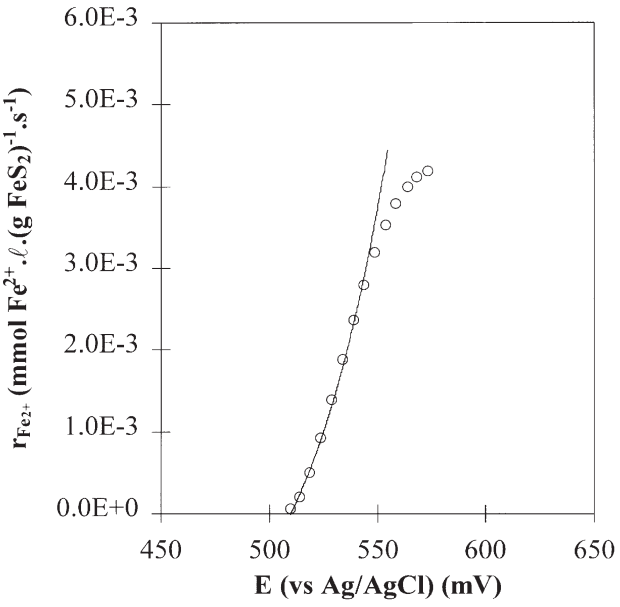

Figure 5—Comparison between the [—] Butler-Volmer based model prediction (r_O = 5x10^4, α = 0.498, β = 0.0272, E´ = 510) and the [○] experimental leaching rate data (after Ruitenberg *et al*[54].)

‡ The maximum calculated leaching rate was attained within ±20s; in comparison the overall duration of the experiments was in the region of 2 hours

§ The rest potential of the mineral is defined as redox potential of the solution at which the mineral dissolution stops

leaching rate is not clear at present, the results obtained thus far suggest that the Butler-Volmer based model has potential for predicting the ferric leaching rate of sulphide minerals over a wide range of solution conditions. It is however, necessary to modify the model to include the effect of parameters such as the pH and the ferric-iron concentration on the ferric leaching rate.

Ferrous-iron oxidation kinetics

To date a number of kinetic models for bacterial ferrous-iron oxidation have been proposed[63]. These models can be broadly classified as either empirical or Michaelis-Menten/Monod based. Empirical models use tools such as the logistic equation to model the kinetics whereas Michaelis-Menten based models assume that the rate limiting reactions can be described using traditional enzyme kinetics. A number of workers have Michaelis-Menten/Monod based models modified to account for ferric-iron inhibition[64,65] and a threshold ferrous-iron concentration[66]. However, Boon[50] showed that the threshold ferrous-iron concentration and ferrous-iron saturation terms could be ignored. Instead the ferrous-iron oxidation kinetics are assumed to be proportional to the ferric/ferrous-iron ratio (i.e. the redox potential):

$$q_{Fe^{2+}} = \frac{-r_{Fe^{2+}}}{c_x} = \frac{q^{\max}_{Fe^{2+}}}{1 + K\dfrac{Fe3+}{Fe^{2+}}} \quad [50] \qquad [10]$$

Equation [10] is consistent with the chemiosmotic theory proposed by Ingledew[67] and has been used to describe the kinetics of *T. ferrooxidans* and *L. ferrooxidans* over the range of ferric/ferrous-iron ratios found in bioleaching systems[10,11,68–69].

Most commercial bioleaching operations using mesophilic bacteria are continuous processes and are carried out using mixed cultures. They usually operate at temperatures in the region of 40°C and pH values ranging from pH 1.2–2.0. However, most research performed to date has been carried out using pure cultures in batch reactors, at temperatures in the region of 30°C and pH values in the region of pH 1.8–2.0. These conditions are similar to those that have been reported to be the optimum conditions for *T. ferrooxidans*. This has resulted in most workers having reported *T. ferrooxidans* to be the organism responsible for the bioleaching of sulphide minerals. However, recent work has suggested that *T. ferrooxidans* is unlikely to predominate in many systems[10–12,70–74]. An explanation for this has been offered by Rawlings *et al.*[75]. Furthermore, apart from the work of Nemati and Webb[76], the effect of temperature on bioleaching micro-organisms has not been studied extensively.

During a recent investigation the ferrous-iron oxidation kinetics of a culture of a predominantly *L. ferrooxidans* were studied in continuous-flow bioreactors at dilution rates ranging from 0.01 to 0.10 h^{-1}, temperatures ranging from 30 to 40°C and pH values ranging from pH 1.10 to pH 1.70[24]. Although the bacterial culture maintained at 40°C and pH 1.30 'washed out' at the highest dilution rate, the highest calculated maximum specific growth rate, $\mu^{max} = 0.1238$ h^{-1}, occurred at 40°C and pH 1.50[69].

The maximum bacterial specific ferrous-iron and oxygen utilization rates, $q^{max}_{Fe^{2+}}$ and $q^{max}_{O_2}$, respectively, and the kinetic constant increased with increasing temperature, across the range from 30 to 40°C. The temperature dependence of the maximum bacterial specific ferrous-iron and oxygen utilization rates could be accurately described using the Arrhenius Equation. However, the relationship between temperature and the kinetic constant appeared to be linear.

The kinetic constant also appeared to increase linearly with increasing pH. Although the maximum bacterial specific ferrous-iron and oxygen utilization rates appeared to achieve maximum values at pH 1.50, no simple relationship between these parameters and the solution pH was apparent. Furthermore, the variation in the maximum specific ferrous-iron and oxygen utilization rates with changes in pH was considerably less pronounced than the variation observed with changes in temperature. It is therefore possible to depict the primary differences in the effect of temperature and pH on the ferrous-iron oxidation kinetics of *L. ferrooxidans* as indicated in Figure 6.

The above trends and assumptions led to a model that predicts the bacterial specific ferrous-iron and oxygen utilization rates as a function of the ferric/ferrous-iron ratio, for temperatures ranging from 30 to 40°C and pH values ranging from pH 1.10 to pH 1.70[24]:

$$q_{Fe^{2+}} = \frac{1.204 \times 10^{7} e^{-\frac{35.33}{RT}}}{1 + \left(7.530 \times 10^{-7} T + 0.0043\text{pH} - 0.0042\right)\left[\dfrac{Fe^{3+}}{Fe^{2+}}\right]} \quad [11]$$

$$q_{O_2} = \frac{0.301 \times 10^{7} e^{-\frac{35.33}{RT}}}{1 + \left(7.530 \times 10^{-7} T + 0.0043\text{pH} - 0.0042\right)\left[\dfrac{Fe^{3+}}{Fe^{2+}}\right]} \quad [12]$$

Comparison between the experimentally measured variation in the specific ferrous-iron and oxygen utilization rates with changes in the ferric/ferrous-iron ratio and the predictions of Equations [11] and [12] showed good agreement (data not shown)[24].

Modelling continuous bioleach reactors

Steady-state ferrous-iron oxidation

If it is assumed that the maximum growth yield and the maintenance coefficient are constant for a particular bacterial species/substrate combination, and can be related via the Pirt equation[77], then, for the case where the substrate is ferrous-iron,

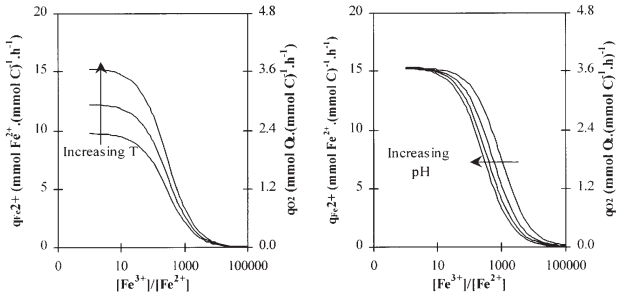

Figure 6—Effect of temperature and pH on the bacterial specific ferrous-iron and oxygen utilization rates (after Breed *et al.*[69])

$$-r^{bact}_{Fe^{2+}} = \frac{r_X}{Y^{max}_{Fe^{2+}X}} + m_{Fe^{2+}} c_X \quad [13]$$

Division of Equation [13] by c_X, and substituting assuming chemostat operation with sterile feed yields:

$$q_{Fe^{2+}} = \frac{1}{Y^{max}_{Fe^{2+}X}\tau} + m_{Fe^{2+}} \quad [14]$$

If $q_{Fe}2+$ can be related to the ferric/ferrous-iron ratio using an equation of the form of Equation [10], then substituting Equation [10] into Equation [14] and rearranging yields:

$$\left[\frac{Fe^{3+}}{Fe^{2+}}\right] = \frac{\left(\frac{\tau q^{max}_{Fe^{2+}} Y^{max}_{Fe^{2+}X}}{1 + m_{Fe^{2+}}\tau Y^{max}_{Fe^{2+}X}}\right) - 1}{K} \quad [15]$$

Equation [15] shows that the ferric/ferrous-iron ratio is a function only of the residence time and the characteristics of the bacterial species used; i.e. it is not dependent on the mineral concentration, nor the total or ferrous-iron concentrations:

$$\left[\frac{Fe^{3+}}{Fe^{2+}}\right] = f(\tau) \neq g\left(\left[FeS_2\right]_{in}, \left[Fe_T\right], \left[Fe^{2+}\right]\right) \quad [16]$$

Although this is not an intuitive result, it is expected from the assumption of Monod or Michaelis-Menten type kinetics.

Steady-state chemical (ferric) pyrite leaching

During the steady-state chemical ferric leaching of pyrite in a continuous reactor performing a pyrite mass balance yields:

$$Q_{in}\left[FeS_2\right]_{in} + Vr_{FeS_2} = Q_{out}\left[FeS_2\right]_{out} \quad [17]$$

Equation [17] can be simplified to give:

$$\left[FeS_2\right]_{in} - \left[FeS_2\right]_{out} = -\tau r_{FeS_2} \quad [18]$$

The rate of ferrous-iron production during the ferric leaching of pyrite is related to the rate of pyrite leaching via Equation [1]:

$$r_{FeS_2} = -\frac{r^{chem}_{Fe^{2+}}}{15} \quad [19]$$

Substituting Equation [19] into Equation [18] yields:

$$\left[FeS_2\right]_{in} - \left[FeS_2\right]_{out} = \frac{\tau r^{chem}_{Fe^{2+}}}{15} \quad [20]$$

By definition:

$$r^{chem}_{Fe^{2+}} = \upsilon_{Fe^{2+}}\left[FeS_2\right]_{out} \quad [21]$$

If it is assumed that the ferric leaching of pyrite can be described by an equation of the form suggested by Boon[50], viz. Equation [10], substitution of this Equation [10] into Equation [21] yields:

$$r^{chem}_{Fe^{2+}} = \frac{\upsilon^{max}_{Fe^{2+}}\left[FeS_2\right]_{out}}{1 + B\left[\frac{Fe^{2+}}{Fe^{3+}}\right]} \quad [22]$$

Substituting Equation [22] into Equation [20] and rearranging yields:

$$\frac{\left[FeS_2\right]_{in}}{\left[FeS_2\right]_{out}} = 1 + \frac{\frac{\tau}{15}\upsilon^{max}_{Fe^{2+}}}{1 + B\left[\frac{Fe^{2+}}{Fe^{3+}}\right]} \quad [23]$$

The fraction of mineral leached, X, is defined as:

$$X = \frac{\left[FeS_2\right]_{in} - \left[FeS_2\right]_{out}}{\left[FeS_2\right]_{in}} = 1 - \frac{\left[FeS_2\right]_{out}}{\left[FeS_2\right]_{in}} \quad [24]$$

Hence combining Equations [23] and [24] yields:

$$X = \frac{\frac{\tau}{15}\upsilon^{max}_{Fe^{2+}}}{1 + B\left[\frac{Fe^{2+}}{Fe^{3+}}\right] + \frac{\tau}{15}\upsilon^{max}_{Fe^{2+}}} \quad [25]$$

From Equation [25] it is clear that the mineral conversion is a function of the ferric/ferrous-iron ratio, the residence time and the characteristics of the pyrite itself, not of the inlet mineral concentration:

$$X = h\left(\left[\frac{Fe^{3+}}{Fe^{2+}}\right]\right) = j(\tau) \neq k\left(\left[FeS_2\right]_{in}\right) \quad [26]$$

However, the ferric/ferrous-iron ratio is determined by the characteristics of the bacterial species used and the prevailing residence time, hence substitution of Equation [15] into Equation [25] and rearranging yields:

$$X = \frac{\frac{\tau}{15}\upsilon^{max}_{Fe^{2+}}}{1 + B\left(\frac{K}{\left(\frac{\tau q^{max}_{Fe^{+2}} Y^{max}_{Fe^{2+}X}}{1 + m_{Fe^{2+}}\tau Y^{max}_{Fe^{2+}X}}\right) - 1}\right) + \frac{\tau}{15}\upsilon^{max}_{Fe^{2+}}} \quad [27]$$

From Equation [27], it is clear that

$$X = 1(\tau) \quad [28]$$

Figure 7 shows the predicted variation in the pyrite conversion, using *T. ferrooxidans* and a *Leptospirillum*–like bacterium in a continuous-flow bioreactor, at residence times ranging from 0 to 20 days. These predictions are compared with the results of Hansford and Chapman[6] for the continuous bioleaching of a similar sized euhedral pyrite by an unidentified microbial species.

From Figure 7 it is apparent that the data of Hansford and Chapman[6] follows the trend predicted by the model if the kinetic parameters for the *Leptospirillum*–like bacterium are assumed. Furthermore, the values of the predicted and experimental values are similar. Although the microbial species used by Hansford and Chapman[6] was not identified, the results of previous research[10–12,70–74] suggest that the micro-organism would have been *L. ferrooxidans*.

At this stage, it is important to note that the kinetic parameters of the bacteria were determined at residence times of between 10 and 100 hours, while the kinetic parameters of the pyrite were determined in batch culture. Furthermore, neither the bacterial species, nor the pyrite used to determine the kinetic parameters were the same as those

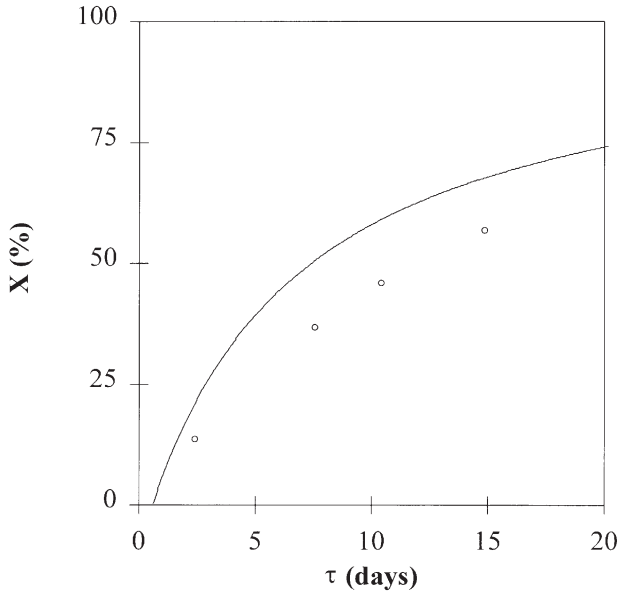

Figure 7—Comparison between the predicted variation in the pyrite conversion with changing residence time for a [—] *Leptospirillum*-like bacterium and the [○] experimental results obtained by Hansford and Chapman[6]. Data were calculated from the results of Van Scherpenzeel *et al.*[68] (after Breed[19])

used by Hansford and Chapman[6]. In spite of this it appears as though the model developed is able to predict the performance of a continuous bioleach reactor.

To date a number of researchers have reported that both the chemical ferric leaching[51] and the bioleaching[4,6] of sulfide minerals is dependent on the surface area of the mineral present. Although the results presented above are described in terms of the pyrite concentration in mmol $FeS_2.l^{-1}$ it is possible to relate the pyrite specific ferrous-iron production rate based on the pyrite concentration to the pyrite specific ferrous-iron production rate based on the pyrite surface area using:

$$r_{Fe^{2+}}^{chem} = \xi_{Fe^{2+}} \alpha \left[FeS_2 \right] \qquad [29]$$

where the pyrite specific ferrous-iron production rate based on the pyrite surface area can be described using equations analogous to Equations [8] or [9].

Summary

The multiple sub-process mechanism suggests that the kinetics of the chemical and bacterial sub-processes may be studied independently, and then combined in order to predict the steady-state and dynamic performance of bioleach reactors.

The chemical ferric leaching rate of arsenopyrite decreases with a decrease in the solution redox and may be described using a modified Butler-Volmer equation. The bacterial ferrous-iron oxidation kinetics of *L. ferrooxidans* also depend on the redox potential, and can be described using a Michaelis-Menten type model modified to account for both temperature and pH.

Although the effect of arsenic on the kinetics has yet to be determined, it is suggested that the mechanism of arsenic resistance may be attributed to the Pst+ Pit- mutation and an energy dependent efflux pump. The Pst+ Pit- mutation enables the bacteria to survive in solutions in which the arsenate concentration is significantly higher than the arsenite concentration. However, because the excretion of arsenate requires energy, in the absence of an energy source or during periods of reduced bacterial activity, the inhibitory effect of arsenate may manifest at concentrations to which the culture has previously been adapted.

The proposed model suggests that the residence time and microbial species present determine the bacterial growth rate, which in turn determines the redox potential of the bioleaching solution. The residence time and redox potential in turn determine the mineral conversion. Refinement of the model is necessary to include the effect of changes in the surface area, the formation of precipitates and the fate of the sulphur moiety. However, it appears to have potential for predicting the performance of continuous bioleach reactors.

Acknowledgements

The authors would like to acknowledge the financial support received from The Foundation for Research Development (South Africa), Billiton Process Research and the Gold Fields Foundation.

Nomenclature

B	kinetic constant in chemical ferric leaching	dimensionless
c_X	concentration of bacteria	mmol C.l^{-1}
E	solution redox potential	mV
E´	mineral rest potential	mV
F	Faraday constant	C.mol^{-1}
$[Fe^{2+}]$	concentration of ferrous-iron	mmol Fe^{2+}.l^{-1}
$[Fe^{3+}]$	concentration of ferric-iron	mmol Fe^{3+}.l^{-1}
$[Fe^{2+}]_{in}$	inlet ferrous-iron concentration	mmol Fe^{2+}.l^{-1}
$[FeS_2]$	FeS_2 concentration	mmol FeS_2.l^{-1}
$[FeS_2]_{in}$	concentration of FeS_2 in the feed	mmol FeS_2.l^{-1}
$[FeS_2]_{out}$	concentration of FeS_2 in the outlet	mmol FeS_2.l^{-1}
$[Fe_T]$	concentration of total iron	mmol Fe.l^{-1}
K	kinetic constant in bacterial ferrous-iron oxidation	dimensionless
$m_{Fe^{2+}}$	maintenance coefficient on ferrous-iron	mmol Fe^{2+}. (mmol C)$^{-1}$.h^{-1}
Q_{in}	volumetric flow rate into the bioreactor	l.h^{-1}
Q_{out}	volumetric flow rate out of the bioreactor	l.h^{-1}
$q_{Fe^{2+}}$	bacterial specific ferrous-iron oxidation rate	mmol Fe^{2+}. (mmol C)$^{-1}$.h^{-1}
$q_{Fe^{2+}}^{max}$	maximum bacterial specific ferrous-iron oxidation rate	mmol Fe^{2+}. (mmol C)$^{-1}$.h^{-1}
q_{O_2}	bacterial specific oxygen utilization rate	mmol O_2. (mmol C)$^{-1}$.h^{-1}
$q_{O_2}^{max}$	maximum bacterial specific oxygen utilization rate	mmol O_2. (mmol C)$^{-1}$.h^{-1}
R	universal gas constant	kJ.mol^{-1}.K^{-1}
r_0	kinetic constant in chemical ferric leaching	mmol Fe^{2+}.l. (g FeS_2)$^{-1}$.s^{-1}
$r_{Fe^{+2}}^{bact}$	bacterial ferrous-iron production rate	mmol Fe^{2+}. l^{-1}.h^{-1}
$r_{Fe^{+2}}^{chem}$	chemical ferrous-iron production rate	mmol Fe^{2+}. l^{-1}.h^{-1}

r_{FeS_2}	pyrite production rate	mmol FeS_2. $l^{-1}.h^{-1}$
r_{O_2}	oxygen production rate	mmol C.$l^{-1}.h^{-1}$
T	absolute temperature	K
$Y^{max}_{Fe^{+2}X}$	maximum bacterial yield on ferrous-iron	mmol C.(mmol Fe^{2+})$^{-1}$
V	bioreactor volume	l
X	fraction of mineral leached	dimensionless
z	number of electrons involved in the reaction	dimensionless
α	specific surface area	m^2 FeS_2. mmol FeS_2^{-1}
β	$\frac{zF}{RT}$	mV^{-1}
μ^{max}	maximum bacterial specific growth rate	h^{-1}
τ	residence time	h
$\upsilon_{Fe^{2+}}$	pyrite specific ferrous-iron production rate	mmol Fe^{2+}. (mmol FeS_2.)$^{-1}.h^{-1}$
$\upsilon^{max}_{Fe^{+2}}$	maximum pyrite specific ferrous-iron production rate	mmol Fe^{2+}. (mmol FeS_2)$^{-1}.h^{-1}$
$\xi_{Fe^{2+}}$	surface area specific ferrous-iron production rate	mmol Fe^{2+}. (m^2 FeS_2.)$^{-1}.h^{-1}$

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