

3.3.7 Reduction in growth of *Coxiella burnetii* exposed to Virkon® S

Results from experiments assessing the efficacy of disinfection with Virkon S® are shown in Table 3.12. Results from the “kill” controls used in each experiment are shown in Table 3.13.

Table 3-12. Reduction in growth of *Coxiella burnetii* (% red) after exposure to Virkon® S at varying concentrations in HP water and in wastewater using a qPCR

| Exp. | Conc. | Treated in | Exposure | % red. | SEM (% red.) |
|-------------|--------------|-------------------|-----------------|---------------|---------------------|
| 6.1 | 2.00% | HP Water | 30 min | 99.93 | 0.02 |
| | 1.00% | HP Water | 30 min | 99.38 | 0.16 |
| | 0.50% | HP Water | 30 min | 99.44 | 0.10 |
| | 0.05% | HP Water | 31 min | 97.27 | 0.52 |
| 6.2 | 2.00% | Wastewater | 30 min | 99.8 | 0.01 |
| | 1.00% | Wastewater | 30 min | 99.75 | 0.02 |
| | 0.50% | Wastewater | 30 min | 99.33 | 0.04 |
| | 0.05% | Wastewater | 30 min | 96.00 | 0.23 |

Table 3-13. The reduction in growth of *Coxiella burnetii* (% red) in “kill” controls used with each Virkon[®] S disinfection experiment

| Exp. | Kill control | Kill Ctrl % red. | SEM (Kill Ctrl) |
|-------------|---------------------|-------------------------|------------------------|
| 6.1 | 3 M NaOH | 97.77 | 0.96 |
| 6.2 | 30min @95°C | 99.88 | 0.02 |

There was significantly greater reduction in amplifiable DNA in samples all treatment and “kill” control groups compared to the NT controls suspended in HP water and waster water ($p < 0.001$). There was significantly greater reduction in amplifiable DNA in cells suspended in HP water and exposed to 2% w/v Virkon S[®] compared to cells suspended in wastewater and exposed to 2% w/v Virkon S[®] ($p < 0.005$).

3.4.6 Virkon® S

Treatment of *C. burnetii* cells with Virkon® S produced significant inactivation at all concentrations used in both pure water and wastewater. This suggests that Virkon® S may be appropriate for small-scale use in an agricultural or laboratory setting. Virkon® S is a commercially available oxidative disinfectant that is purported to contain a balanced, stabilised blend of peroxygen compounds, surfactant, organic acids and an inorganic buffer system, which in solution is activated to form hypochlorous acid (DuPont 2008). The mode of action of Virkon® S will thus be similar to the other oxidants described previously. However, the combination of these oxidants with a surfactant in a stable composition may produce greater bactericidal action in a variety of conditions. Virkon® S appeared to maintain its activity in the presence of organic matter and suspended solids. However, the efficacy of Virkon® S has been shown to be variable in conditions of high organic load (McComick and Maheshwari 2004). The low pH that resulted from adding Virkon® S to both of the water types used in these experiments is likely to have played a significant role in the disinfection efficacy observed, particularly in relation to hypochlorous acid. It appears that the resistance of *C. burnetii* to the normally peroxide-rich host cell phagolysosome (Clark 1990) is attributed to inhibition of the respiratory burst by acid phosphatase activity (Baca, Roman et al. 1993). In light of the metabolic effort *C. burnetii* assigns to inhibit host cells production of peroxide, peroxygens may be ideal candidate disinfectants. However, Virkon® S is becoming more difficult to obtain in Australia and its cost is prohibitive for large-scale use. Therefore, its use may only be applicable to laboratories and small volumes of animal-associated effluent.