Cambridge Biomedical

Q Fever in Serum

Specimen Type	Serum		
Specimen Volume	1 mL		
Collection	Red top tube with no additives or serum gel tube. Collect blood. Allow to clot. Centrifuge sample at 3000 rpm for 10 minutes. Separate serum immediately into transfer tube and freeze immediately.		
Minimum Volume	0.5 mL		
Handling	Ship frozen on dry Ice.		
Rejection Criteria	• Contaminated specimens. • Grossly hemolyzed specimens. • Grossly lipemic specimens. • Specimens outside of listed stability.		
Stability	Ambient for 48 hours. Refrigerated for 30 days. Frozen for 30 days. 4 Freeze-Thaw cycles.		
Methodology	IFA		
Reference Ranges	**Q Fever IgG**	Screen	<1:16 No Antibody Detected Phase I and II
	Q Fever IgM	Screen	<1:16 No Antibody Detected Phase I and II
	Q Fever IgG and IgM Antibody Screens are run on each sample. Samples reported as Antibody Detected will reflex automatically to IgG or IgM antibody titers. Titers are reported for Phase I and Phase II of the IgG or IgM.		
Turnaround Time	Up to 7 business days.		
CPT Code	Q Fever IgG: 86638 Q Fever IgM: 86638 Each Titer: 86638		

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Clinical Significance	Coxiella burnetii, the causative agent of Q fever, is an obligate intracellular parasite from the family Rickettsiae, with worldwide distribution. The infection is spread by the inhalation of infected material, mainly from sheep and goats. Infection in these animals is enzootic and nearly always unapparent. They shed the organism in feces, milk, nasal discharge, placental tissue, and amniotic fluid. The clinical spectrum of disease ranges from unapparent to fatal. Respiratory manifestations usually predominate. Endocarditis and hepatitis can be complications. During the course of the infection, the outer membrane of the organism undergoes changes in its lipopolysaccharide structure called phase variation. Differences in phase I and phase II antigen presentation can help determine if the infection is acute or chronic. In acute Q fever, the phase II antibody is usually higher than the phase I titer, often by 4-fold, even in early specimens. Although a rise in phase I as well as phase II titers may occur in later specimens, the phase II titer remains higher. In chronic Q fever, the reverse situation is generally seen. Serum specimens drawn late in the illness from chronic Q fever patients demonstrate significantly higher phase I titers, sometimes much greater than 4-fold. In the case of chronic granulomatous hepatitis, IgG and IgM titers to phase I and phase II antigens are quite elevated, with phase II titers generally equal to or greater than phase I titers.
Principle	The Q Fever Immunofluorescent Antibody (IFA) assay is a 2-stage “sandwich” test. In the first stage, the patient sera is diluted and incubated in appropriate slide wells. Following incubation, the slide is washed to remove unbound serum antibodies. In the second stage, each antigen well is overlaid with fluorescein-labeled antibody to IgG or IgM, depending on the type of assay. The slide is incubated allowing antigen-antibody complexes to react with the fluorescein-labeled anti-IgG or anti-IgM. After the incubation the slide is washed, dried, mounted, and examined using fluorescence microscopy. Positive reactions appear as bright apple-green fluorescent “rickettsia-like” particles dispersed within a background matrix of yolk sac. Semi-quantitative endpoint titers are obtained by testing serial dilutions of positive specimens.