

Environmental Isolates of *Serratia marcescens*

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Received: October 31, 2022; **Published:** November 01, 2022

Serratia marcescens, a Gram-negative bacteria classified as a member of *Enterobacteriaceae*, has been recognized as a cause of hospital-acquired infection for the last two decades. It is a widely distributed saprophytic bacterium, and has been found in food, particularly in starchy variants which provide an excellent growth environment. While this organism was known formerly by a variety of names. Gaughran, *et al.* [1] used the name *S. marcescens* that had been assigned by Bizio in 1823.

S. marcescens was considered originally to be an innocuous, pathogenic saprophytic water organism and was often used as a biological marker because of its easily recognised red colonies. It has now been implicated as an aetiological agent in every conceivable kind of infection, including respiratory tract infection, urinary tract infection (UTI), septicemia, meningitis and wound infections [2-4]. It has been reported to cause infective endocarditis acquired in the community (Mills, *et al.* 1976) and in hospitals. In contrast to the Gram-negative bacteria, it usually affects the left side of the heart [5]. *S. marcescens*, endocarditis acquired in the hospital is usually an exogenous infection associated with cardiac surgery [6]. Today *S. marcescens* has attained the status of a highly fledged pathogen that causes infections particularly in two disparate groups: heroin addicts and hospitalized patients.

Environmental isolates of *S. marcescens* characteristically produce a red pigment, prodigiosin, and in early times such growth was often mistaken for fresh blood. The pigmented bacterium is found in various ecological niches, including soil, water, air, plants and animals [7]. The ability to form prodigiosin is characteristic of *S. marcescens*, but the function of this red pigment remains unclear because clinical isolates are rarely pigmented.

Enzyme therapies are becoming more prevalent in medicine today, with many manufactures targeting their advantages in disease treatment. In the last 100 years, enzyme have been increasingly used to treat various diseases. Today, enzymes are used as anti-coagulants, oncolytics, thrombolytics, anti-inflammatories, fibrinolytics, mucolytics, anti-microbials and digestive aids. Enzymes are found throughout the natural world, the number of uses for them in various fields of industry in addition to medicine is staggering. Enzymes are found in animal and in plant sources. Enzymes can be thought of protein molecules with a specific mission to initiate and regulate countless biologic reactions in living organisms.

Enzymes, like their application in medicine, exert their effects in a multitude of ways. One primary focus of enzymatic action is on the protein fibrin. Fibrin is an insoluble protein involved in blood clotting. In the many steps of the clotting cascade, fibrin is the final product. It is derived from its soluble protein precursor, fibrinogen. Fibrin is laid down inside blood vessels that have been compromised by disease or injury. Fibrin forms minuscule strands that eventually dry and harden, capturing blood vessel components effectively. Certainly, fibrin occupies a vital role in health and healing; however, fibrin may also be responsible for an overzealous propensity to form inappropriate clots in the body. In appropriate clotting, of course, is a major risk factor for myocardial infarction and stroke.

Fibrinolysis is a process that prevents blood clots from growing and becoming problematic [8]. In fibrinolysis, a fibrin clot, the product of coagulation, is broken down [9]. Its main enzyme plasmin cuts the fibrin mesh at various places, leading to the production of circulating

fragments that are cleared by other proteases or by the kidney and liver. When correctly balanced, deposition and removal of fibrin maintains avoidance of blood loss and adverse viscosity in the vascular system. A balance tipped in favor of fibrin over production leads to dangerous clotting. Various types of thrombosis are responsible for an increasing number of deaths each year. According to a report published by the World Health Organization (WHO) in 2001, 17 million people die every year of cardiovascular diseases (CVDs). The information of a blood clot in a blood vessel (intravascular thrombosis) is one of the main causes of CVDs. The major protein component of blood clots is fibrin. There are two fibrinolytic bacterial enzymes with thrombolytic activity formed from fibrinogen via proteolysis by thrombin. Meanwhile, fibrin clots can be hydrolyzed by plasmin to avoid thrombosis in blood vessels. In an unbalanced situation due to some disorders, the clots are not hydrolyzed and thus thrombosis occurs [10]. So, several investigations are being pursued to enhance the efficacy and specificity of fibrinolytic therapy and microbial fibrinolytic enzymes have attracted much more medical interest in recent decades [11].

Bibliography

1. Gaughran ERL. "Division of microbiology from superstition to science: the history of a bacterium". Transactions of the New York Academy of Sciences (1968).
2. Gouin F., *et al.* "A non-comparative study of the efficacy and tolerance of cefepime in combination with amikacin in the treatment of severe infections in patients in intensive care". *Journal of Antimicrobial Chemotherapy* (1993): 32.
3. Cox CE. "Aztreonam therapy for complicated urinary tract infections caused by multidrug resistant bacteria". *Reviews of Infectious Diseases* 7.4 (1985): S767-S770.
4. Komer RJ., *et al.* "Ciprofloxacin resistant *Serratia marcescens* endocarditis as a complication of non-Hodgkin's lymphoma". *Journal of Infection* (1994): 29.
5. Cohen PS., *et al.* "Infective endocarditis caused by-negative bacteria: a review of the literature, 1945-1977". *Multimorbid Patients with Cardiovascular Disease* 22 (1997): 205-242.
6. Sleigh JD. "Antibiotic resistance in *Serratia marcescens*". *British Medical Journal* (1983):1651-1653.
7. Grimont PADF. "Genus VIII. *Serratia*". In: Krieg NR, Holt JG (editions) *Bergey's Manual of systematic bacteriology*, vol1. Baltimore, Williams and Wilkins (1984): 477-484.
8. Dugdale and David., *et al.* "Primary or secondary fibrinolysis". Medline Plus (2011).
9. Cesarman-Maus GKA Hajjar. "Molecular mechanisms of fibrinolysis". *The British Journal of Haematology* 129.3 (2005): 307-321.
10. Lopez-Sendon J., *et al.* "Diagnosis of subacute Ventricular wall rupture after acute myocardial infarction: sensitivity and specificity of clinical, hemodynamic and echocardiographic criteria". *Journal of the American College of Cardiology* 19 (1995): 1145-1151.
11. Goldhaber SZ and H Bounameaux. "Thrombolytic therapy in pulmonary embolism". *Seminars in Vascular Medicine* 1.2 (2001): 213-220.

Volume 10 Issue 11 November 2022

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