Dimaria, Michele J. "St. John Baptist de La Salle: A Retrospective Medical Differential Diagnosis." *AXIS: Journal of Lasallian Higher Education 17*, no. 1 (Christian Brothers Conference: 2026).

Saint John Baptist de La Salle: A Retrospective Medical Differential Diagnosis

Michele J. Dimaira, D.M.D., M.S., M.H.A., MATHEM, M.Ed.[1]

Introduction

Saint John Baptist de La Salle (1651-1719), founder of the Institute of the Brothers of the Christian Schools and patron saint of teachers, is widely revered for his transformative contributions to education and his profound spiritual leadership. Amidst his tireless work establishing schools for the poor and reforming the educational system of France, historical records also describe a persistent pattern of physical ailments that significantly impacted his later years. Biographers recount his experiences with debilitating fatigue, joint pain, skin lesions, kidney problems, and a general decline in health that forced him to withdraw from active leadership multiple times.[2] While these symptoms have often been attributed to the physical and emotional strain of La Salle's ascetic lifestyle, a closer examination of historical records raises the possibility of an underlying autoimmune disorder. The range of symptoms documented in his biographies and personal correspondence aligns with several recognized autoimmune conditions, including systemic lupus erythematosus (SLE), rheumatoid arthritis, and dermatomyositis among others. These diseases are all capable of producing the kind of multisystem involvement described in historical accounts.

This paper explores the possibility that Saint John Baptist de La Salle may have suffered from an autoimmune disorder, using a retrospective diagnostic approach to interpret the symptoms described in historical accounts. While acknowledging the inherent limitations of applying modern medical frameworks to premodern narratives, this method allows for a critical synthesis of historical biography with contemporary clinical insight. The aim is not to assign a definitive diagnosis, but to offer a medically plausible interpretation of his physical decline one that may enrich our understanding of how chronic illness intersected with his spiritual life and vocational resilience.

To better understand the progression and nature of De La Salle's symptoms, a chronological overview of his documented illnesses is essential. While the records are not clinical in nature, the biographical accounts offer valuable insight into the frequency, severity, and recurrence of his physical ailments. The following timeline highlights key episodes in his life that may point to an underlying systemic condition and serves as a foundation for the subsequent differential analysis.

Timeline of Illnesses and Physical Decline

1690: First Major Health Crisis and Near-Death Experience

In late 1690, at the age of thirty-nine, De La Salle experienced a serious illness that nearly proved fatal following a physically demanding journey on foot from Paris to Rheims. Preexisting

physical fragility, likely due to a combination of constitutional delicacy, chronic stress, ascetic practices, and prolonged exertion may have contributed to the severity of his condition. Upon arrival in Rheims, he collapsed and became bedridden. Contemporary accounts describe significant emaciation and profound fatigue, with some temporary improvement following a period of rest and nutritional support. During this period, he received a visit from his maternal grandmother, Perrette Lespagnol, whom he met in accordance with strict community protocols, despite his debilitated state.[3]

Shortly thereafter, upon his return to Paris, ignoring medical advice, he experienced a rapid relapse marked by extreme exhaustion and acute urinary retention, which was considered life-threatening. Adrien Helvétius, a prominent physician of the time, was consulted and administered an aggressive treatment, warning that it carried a high risk. Prior to the intervention, De La Salle, recognizing the gravity of his condition, requested and received the Viaticum. Remarkably, the treatment proved successful, and he gradually regained strength over the following days. Eyewitness accounts also highlighted his humility and spiritual composure during convalescence, including his request to be treated as one of the poor by being sent to the General Hospice.[4]

1692-1693: Rheumatism

Around 1692, Saint John Baptist de La Salle suffered a debilitating episode of what was then described as rheumatism, likely worsened by his intense ascetic practices. At the time, he was living in a cold, drafty house, sleeping on a stone floor with minimal bedding, and depriving himself of adequate nourishment. These mortifications, intended as acts of penance, severely weakened his body.

Eventually, the pain became so intense and widespread that he was rendered immobile. His joints had grown so stiff and inflamed that the Brothers were compelled to summon Doctor Helvétius once again. The prescribed treatment was both archaic and excruciating: De La Salle was placed on a slatted wooded grill beneath which pots of burning coal were set. Juniper and other herbs were thrown onto the coals to create pungent, therapeutic smoke believed to open the pores and draw out inflammation.[5]

One side of his body was exposed to the smoke and heat while the other side was covered with blankets to trap and intensify the warmth. The room quickly filled with dense, suffocating smoke and the wooden slats grew so hot that the assisting Brother could not touch them. Despite the agony of the treatment, De La Salle endured it silently and without complaint. Although the treatment provided temporary relief, it did not last. Similar painful interventions would be required again in the years to come.[6]

1706-1707: Tumor on Knee, Fall, and Multiple Surgeries

Around 1706, De La Salle accepted an invitation to open a school in the parish of Saint Roch in Paris. During this period, he began experiencing a painful medical condition, reportedly resulting from extended periods of kneeling in prayer on bare ground. According to contemporary accounts, this practice led to the development of a wen, likely a cyst or soft tissue tumor, on his

knee. Rather than seek immediate medical attention, De La Salle interpreted the condition as an opportunity for penance and continued to neglect it until the swelling progressed to the point that he could no longer bend the affected leg.[7]

While returning from Saint Roch one evening and crossing the Tuileries, he stumbled and fell directly onto an iron spike embedded in the ground. The blow impacted the tumor on his knee, causing intense pain and rendering him unconscious. Two passing laborers initially assumed he was intoxicated but, upon discovering his true condition, assisted him. After regaining partial mobility, De La Salle slowly made his way back to the Brothers' residence on rue Princesse. Too weak to ring the doorbell upon arrival, he requested help from a passerby and collapsed into the arms of the Brother Porter. He remained bedridden for over six weeks following the incident.[8] In a letter dated April 1, 1707, he wrote to Brother Gabriel Drolin, "I too have been very unwell, not able to walk for six weeks; I am feeling much better now."[9]

Later that year, while in Rouen for the establishment of a new school in Darnétal, De La Salle sought help from Brother Cosmas, a Capuchin friar known for his surgical ability. The friar performed a rudimentary excision of the tumor, making an incision in the form of a cross using a razor. The procedure was reportedly endured in silence; it failed to resolve the condition. Upon returning to Paris, De La Salle underwent further treatment from local surgeons, who applied lunar caustic (silver nitrate) and excised additional infected tissue. According to Blain, he endured the procedure without visible signs of distress, maintaining the same calm and reverent demeanor with which he typically celebrated Mass – an expression of his deep spiritual discipline and tolerance for physical suffering.[10]

1714: Severe Rheumatic Attack in Grenoble

In February 1714, while residing in modest and harsh conditions at the top of a drafty tower in Grenoble, referred to by one biographer as a "hole in the wall,"[11] De La Salle experienced a severe recurrence of rheumatism, consistent with previous episodes. Prolonged exposure to cold, inadequate living conditions and physical overexertion from extended prayer and ministry likely contributed to this acute and debilitating flare of joint pain, which was so intense that many feared for his life.

News of his critical condition spread quickly throughout Grenoble. De La Salle's reputation for sanctity and self-sacrifice had earned the admiration of the local population, prompting an outpouring of prayer on his behalf. Leading the intercessions were Fathers Yve de Saléon and Claude Canel, canons of the Church of Saint André and early supporters of the Brothers' mission in the city.[12]

In response to the severity of his illness, De La Salle once again submitted to the same grueling treatment he had endured earlier in Paris. As before, he was laid upon a grill over burning coals infused with steaming medicinal herbs, a traditional remedy meant to induce perspiration and draw out inflammation. Though the procedure remained physically punishing, it was again effective in alleviating his symptoms. His gradual recovery was marked, as always, by his unwavering endurance and spiritual composure amid intense physical suffering.[13]

1716: Letter Written by De La Salle Demonstrates Chronic Illness

In another letter written to Brother Gabriel on December 5, 1716, from Saint Yon, Suburb of Rouen, De La Salle states, "For nearly ten months now, I have been ill in this community in which I have been living for a year."[14]

Lent 1719: Final Illness

As the Lenten season of 1719 approached, Saint John Baptist de La Salle's health declined markedly. His rheumatism, long a source of suffering, had progressed to a chronic, treatment-resistant stage. Simultaneously, he began to experience frequent episodes of respiratory distress consistent with asthma, further complicating his already deteriorating condition. Despite the worsening symptoms, De La Salle continued to observe the Lenten fast with extreme rigor, adhering to practices of self-denial and penitence. When the Brothers urged him to ease his austerities, he resisted, stating that "the victim was ready to be immolated and needed to be purified."[15] Intervention by his confessor was ultimately required to impose limits on his fasting for medical reasons.

A series of accidents further exacerbated his fragile state. In one incident, a Brother inadvertently removed a chair just as De La Salle was about to sit, resulting in a fall that caused a head injury described as a deep laceration accompanied by severe cranial pain. Shortly thereafter, a door fell on him, followed by the onset of acute and persistent pain in his side. These events led to a sharp deterioration in his overall condition, rendering him bedridden. His attending physician, upon assessment, informed the Brothers that his condition was terminal. De La Salle reportedly responded with composure, declining further medical treatment and entrusting himself to divine care, referring to God as the "divine Physician."[16]

For a brief period, he continued to celebrate Mass and hear confessions, but progressive weakness soon rendered even these activities impossible. On March 19, the feast of Saint Joseph, patron of the Institute, he experienced a short-lived improvement that enabled him to celebrate Mass once more.

In his final days, De La Salle revised his will and offered spiritual and communal guidance to his followers, urging fidelity to ecclesial authority, devotion to the Eucharist, and perseverance in community life and religious discipline.[17] On Holy Wednesday, he received Holy Communion for the last time; on Holy Thursday, he was administered the sacrament of the sick. At approximately midnight, when asked whether he accepted his suffering, he gave his final response: *"Oui, j'adore en toutes choses la conduite de Dieu à mon égard"* ("Yes, I adore all the ways God has acted in my life").[18] He died peacefully at approximately 4:00 a.m. on Good Friday, April 7, 1719.

Other Pertinent Information

According to Blain, De La Salle was "a little above average in height, with a frame well proportioned and solidly built. A wide forehead, prominent nose, and two large beautiful eyes,

nearly blue, made up his arresting countenance. His features were gentle and agreeable, his voice, strong and distinct. Exteriorly he appeared cheerful, serene, modest, and pious. His skin, somewhat tanned by his long travels, usually appeared slightly flushed."[19]

Summary of De La Salle's Medical History

There are several clues to De La Salle's medical diagnosis that can be obtained from the various illnesses encountered during his life. Over the course of three decades, he experienced a constellation of recurring and chronic symptoms that hint at a systemic and progressive condition. These include repeated episodes of severe joint pain diagnosed as rheumatism, a vague but common historical term encompassing inflammatory joint disease. He also suffered from prolonged periods of fatigue and debilitation, respiratory distress such as asthma, susceptibility to infection, delayed wound healing, and unusual skin features. His persistent physical weakness, recurring inflammatory flares, and episodes of dramatic systemic decline – some of which were life-threatening – suggest a pattern inconsistent with simple overexertion or aging. The development of a tumor on the knee, extreme photosensitivity indicated by a "flushed" complexion, and signs of neurological involvement and collapse following minor trauma further complicate the clinical picture. Taken together, these diverse symptoms strongly suggest an underlying autoimmune disorder. The remainder of this paper will examine a differential diagnosis of possible conditions as potential explanations for the lifelong suffering of this saintly Founder.

Introduction to Autoimmune Disease and Differential Diagnosis

Autoimmune diseases arise when the immune system malfunctions and begins attacking the body's own tissues, leading to inflammation, tissue damage, and a variety of systemic symptoms.[20] These diseases can affect almost any organ system and often present with overlapping clinical features such as fatigue, joint pain, skin rashes, respiratory issues, and neurological symptoms.[21] Because of this complexity, the process of differential diagnosis – the method by which physicians distinguish between diseases with similar presentations – is especially important in evaluating autoimmune conditions. In the case of Saint John Baptist de La Salle, who lived in the seventeenth and early eighteenth centuries, there is no medical record in the modern sense. However, biographical accounts describe numerous recurring and unexplained physical ailments that may be consistent with autoimmune pathology. In evaluating his condition retrospectively, several possibilities warrant consideration, including rheumatoid arthritis, systemic lupus erythematosus (SLE), dermatomyositis, sarcoidosis, granulomatosis with polyangiitis and eosinophilic granulomatosis with polyangiitis. The following analysis will explore the features of each of these conditions in the context of De La Salle's documented symptoms.

Rheumatoid Arthritis

Rheumatoid arthritis (RA) is defined as a systemic autoimmune pathology associated with a chronic inflammatory process, which can damage both joints and extra-articular organs, including the heart, kidney, lung, digestive system, eye, skin, and nervous system.[22] It affects approximately 0.5% to 2% of the general population, with women, smokers, and individuals with

a family history at higher risk. Though the exact cause remains unclear, genetic, autoimmune, and environmental factors contribute to its development. The disease is marked by the overproduction of pro-inflammatory cytokines such as TNF and IL-6, which drive joint inflammation, synovial cell proliferation, cartilage damage, and bone erosion. Clinically, RA presents as a chronic, symmetrical arthritis, typically beginning in small joints like the fingers and progressing to larger joints. Common symptoms include joint pain, stiffness, fatigue, fever, and weight loss, often accompanied by elevated inflammatory markers like C-reactive protein (CRP) and Erythrocyte Sedimentation Rate (ESR). Autoantibodies such as rheumatoid factor (RF) and anti-citrullinated protein antibody (ACPA) are also characteristic; ACPA is particularly specific to RA and may be present years before symptoms arise. First-line treatment typically includes methotrexate (MTX), a disease-modifying anti-rheumatic drug (DMARD), often combined with low-dose glucocorticoids.[23]

Extra articular manifestations usually identify a subset of patients with a more severe disease associated with a higher mortality and morbidity. The prevalence of extra-articular manifestations is estimated to be from 17.8 to 40.9% of all RA patients. Usually these features are associated with the course of RA, although they are not always associated with the severity of joint involvement and it may be dissociated from the activity of the disease.[24]

The skin is one of the most commonly affected extra-articular organs in rheumatoid arthritis (RA), especially in patients with more severe disease. Cutaneous manifestations are broadly classified as specific or non-specific. Non-specific lesions, which are more prevalent, include diffuse skin atrophy (particularly over interphalangeal joints), palmar erythema, and bluish discoloration of the fingertips that may mimic Raynaud's phenomenon. Nail changes such as onycholysis, longitudinal ridging, clubbing, and splinter hemorrhages are also observed, particularly in cases with RA-associated vasculitis. Specific skin lesions include rheumatoid nodules and neutrophilic dermatoses, which are strongly associated with rheumatoid factor (RF) positivity and certain genetic markers like HLA-DRB1*0401. Rheumatoid nodules are the most common specific manifestation, seen in about 25% of patients – more often in males and those with seropositive RA. A variant known as rheumatoid nodulosis features subcutaneous nodules and cystic bone lesions with a relatively benign clinical course. These dermatological findings, while variable, can be significant markers of disease severity and systemic involvement in RA.[25] Rheumatoid vasculitis (RV) is a severe, extra-articular manifestation of longstanding rheumatoid arthritis, marked by inflammation of small- to medium-sized blood vessels. Although clinically rare – affecting about 1% of RA patients annually – it has been observed in up to 25-30% of postmortem and biopsy studies, even in asymptomatic tissue. RV typically develops after a decade or more of active disease and is associated with high rheumatoid factor titers, joint erosions, and the presence of rheumatoid nodules, particularly in male patients. Cutaneous manifestations are most common, including nail fold infarctions (Bywaters lesions), palpable purpura, livedo reticularis, and chronic leg ulcers-especially near the lateral malleolus or pretibial area. These vascular lesions result from occlusive arteritis and intimal proliferation. Neurological complications, such as mononeuritis multiplex or sensory neuropathy, occur in about half of affected individuals, while systemic organ involvement is rare but potentially life-threatening. Recognizing RV is critical due to its potential to mimic other systemic autoimmune diseases, including systemic lupus erythematosus.[26]

Ocular involvement is a relatively common extra-articular manifestation of rheumatoid arthritis (RA), occurring in approximately 25-39% of patients and sometimes presenting as the initial symptom of disease. The risk of eye complications increases with disease duration and may signal a more severe form of RA, particularly in patients with anti-citrullinated protein antibodies (ACPA). Because the eye is considered an immune-privileged site with immunosuppressive properties, inflammation in this region may reflect systemic immune dysregulation. The most frequent ocular manifestation is keratoconjunctivitis sicca (KCS), or dry eye syndrome, reported in 15-28% of RA patients. Less common but more serious conditions include episcleritis (0.17-3%), scleritis (0.67-6%), and the rare peripheral ulcerative keratitis (PUK) seen in 0.1-2% of cases. While KCS is generally independent of RA disease activity, scleritis and PUK are often associated with active, severe disease and require prompt recognition due to their potential to cause significant visual impairment or signal systemic vasculitis.[27]

Cardiac and pulmonary involvement are serious extra-articular manifestations of rheumatoid arthritis (RA) that significantly contribute to patient morbidity and mortality. Pericarditis is the most common cardiac complication, though RA may affect all layers of the heart, including the myocardium and conduction system, leading to pericardial effusion, myocarditis, and arrhythmias. These cardiac manifestations are associated with an increased risk of heart failure and may occur independently of joint symptoms. Similarly, pulmonary involvement is common and can precede or accompany joint disease. The lungs may be affected at multiple levels, including the pleura, airways, interstitium, and vasculature. Interstitial lung disease (ILD), pleuritis, rheumatoid pulmonary nodules, and airway disorders such as cricoarytenoiditis are among the most frequently reported complications. Notably, RA-associated ILD often develops insidiously and may remain asymptomatic for a long time. When symptoms occur, they typically include exertional dyspnea, a dry cough, fatigue, and weight loss. These manifestations may be under-recognized due to physical activity limitations imposed by joint disease or the nonspecific nature of systemic symptoms like fatigue and malaise. Both cardiac and pulmonary complications underscore the systemic nature of RA and its potential to cause significant organ damage beyond the joints.[28]

Renal involvement in rheumatoid arthritis (RA) is relatively common, with prevalence estimates ranging from 20% to 50%. Kidney complications may arise directly from the disease process or indirectly due to chronic inflammation and long-term medication use. The most frequently reported renal manifestations include various forms of glomerulonephritis, which may progress to chronic kidney disease if left untreated. In rare cases, prolonged systemic inflammation can lead to secondary amyloidosis or vasculitic involvement of the renal vasculature. Additionally, RA can impact bladder function, often presenting as urinary urgency, frequency, or incontinence. These symptoms may result from pelvic floor dysfunction, nerve involvement, or fluid retention, highlighting the broader systemic impact of RA beyond the musculoskeletal system.[29]

A Case for RA

Rheumatoid arthritis (RA), a chronic systemic autoimmune disorder, presents with a wide spectrum of articular and extra-articular manifestations, several of which appear to align with the historical accounts of Saint John Baptist de La Salle's illnesses. De La Salle is repeatedly described as suffering from "rheumatism," particularly affecting his knee, with pain, stiffness,

and difficulty walking-symptoms that are consistent with chronic synovitis and joint destruction, hallmark features of RA. The reported "bony growth" or persistent swelling in his knee could plausibly represent a rheumatoid nodule or cystic bone lesion, both of which are recognized complications of RA, especially in seropositive patients with long-standing disease.
In addition to chronic joint involvement, De La Salle experienced recurrent episodes of fatigue, malaise, and fevers-nonspecific systemic symptoms that are frequently associated with rheumatoid arthritis (RA), particularly during periods of increased disease activity. Documented episodes of asthma are also noteworthy, as pulmonary manifestations, including airway inflammation, can occur in RA. His experience of urinary retention, though not a classical feature of RA, may suggest autonomic nervous system involvement, which has been reported in rare extra-articular cases. If the swelling in his leg was painful or ulcerative, it may be consistent with rheumatoid vasculitic lesions, which frequently affect the lower extremities. Although no information is available regarding his renal or ocular health-both of which can be compromised in severe RA-the constellation of chronic joint inflammation, systemic symptoms, and possible extra-articular features makes RA a clinically plausible component in the retrospective differential diagnosis of his condition.

Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease affecting multiple organ systems and is linked to high morbidity and mortality. Its exact cause is unknown, but genetic, immunological, endocrine, and environmental factors contribute to its development. The disease is characterized by the loss of immune tolerance leading to the production of autoantibodies that damage tissues. SLE presents with a wide range of symptoms, from mild skin issues to severe organ involvement, and is more common in women (about 90% of cases). Environmental triggers such as UV light, smoking, certain viruses, and occupational exposures may trigger disease in genetically predisposed individuals. Despite advancements, diagnosis remains challenging, and treatment is based on which organs are affected.[30]

SLE commonly presents with mucocutaneous manifestations, affecting over 80% of patients. These may include lupus-specific lesions such as acute cutaneous lupus erythematosus (ACLE), subacute cutaneous lupus erythematosus (SCLE), and chronic cutaneous lupus erythematosus (CCLE). ACLE typically appears as a malar or "butterfly" rash on the cheeks and nasal bridge, often triggered by sun exposure and associated with disease activity. SCLE is a photosensitive, widespread, non-scarring rash, often seen in patients with anti-Ro (SSA) antibodies. CCLE includes discoid lupus erythematosus (DLE), which presents with scarring, disk-shaped plaques that can cause permanent alopecia when on the scalp. Other variants like hypertrophic DLE, lupus panniculitis, chilblains lupus, and mucosal discoid lupus may also occur. Oral and nasal ulcers, often painless, are common, and photosensitivity is a hallmark feature of SLE. Patients may also experience scarring or non-scarring alopecia and various nonspecific skin lesions such as vasculitis, Raynaud's phenomenon, livedo reticularis, and bullous eruptions.[31]

Musculoskeletal involvement occurs in 80-90% of SLE patients and can range from mild arthralgias to deforming arthritis. Lupus arthritis is typically non-erosive, symmetric, and affects small joints such as those in the hands and wrists. A distinctive deforming arthritis known as Jaccoud arthropathy may occur due to ligament laxity, resulting in reversible joint deformities.

Avascular necrosis, especially of the hips, may develop in about 10% of patients, often bilaterally. Less commonly, patients may experience inflammatory myopathy resembling polymyositis or develop fibromyalgia, which is reported in up to 20% of cases. Rheumatoid nodules have also been reported in some individuals with SLE.[32]

Hematologic abnormalities are frequent in SLE. Anemia is present in more than half of patients, most commonly due to chronic disease, but may also result from iron deficiency, autoimmune hemolysis, red blood cell aplasia, or microangiopathic hemolytic anemia, particularly in association with antiphospholipid syndrome. Leukopenia, from neutropenia or lymphopenia, is common and may be severe. Thrombocytopenia can range in severity and may be autoimmune in origin. Pancytopenia can also occur and has been associated with myelofibrosis. Patients may have soft, non-tender lymphadenopathy, and although rare, histiocytic necrotizing lymphadenitis (Kikuchi-Fujimoto disease) can occur. Splenomegaly is common, while splenic atrophy and functional asplenism have also been described.[33]

Neuropsychiatric manifestations of SLE involve both the central and peripheral nervous systems, as well as psychiatric symptoms. Central nervous system (CNS) involvement may include persistent headaches, which affect more than 50% of patients, as well as seizures, which are often associated with disease activity and generally have a favorable prognosis. Other CNS findings include aseptic meningitis, optic neuritis, myelitis, chorea, cognitive dysfunction, and an increased risk of ischemic stroke. Peripheral nervous system (PNS) involvement may manifest as cranial or peripheral neuropathies, mononeuritis multiplex, autonomic neuropathy, and syndromes mimicking Guillain-Barré syndrome or myasthenia gravis. Psychiatric symptoms such as depression, anxiety, and psychosis can also be present and may be difficult to diagnose and manage.[34]

Renal involvement, particularly lupus nephritis, is a well-known and serious complication of SLE. It can present with a spectrum of symptoms, from mild proteinuria to severe, progressive glomerulonephritis. Lupus nephritis often develops early in the disease course, with signs such as new-onset hypertension, hematuria, proteinuria, lower extremity edema, and elevated creatinine prompting evaluation. Kidney biopsy is crucial for classification and guides treatment. Prognosis varies with histologic class, with classes I and II generally having excellent outcomes, classes III and IV associated with poor outcomes, and class V often showing favorable prognosis though complicated by nephrotic syndrome and thromboembolic risk. Other renal issues may include thrombotic microangiopathy, interstitial nephritis, vasculitis, and lupus vasculopathy.[35]

Pulmonary manifestations of SLE include pleuritis, the most common lung-related issue, which may or may not be associated with pleural effusion. Additional complications can include acute lupus pneumonitis, interstitial lung disease (typically nonspecific interstitial pneumonia or usual interstitial pneumonia), diffuse alveolar hemorrhage, pulmonary arterial hypertension, pulmonary embolism (particularly in the context of antiphospholipid syndrome), and the rare shrinking lung syndrome.[36]

Cardiovascular manifestations can affect any part of the heart. Pericarditis, often accompanied by exudative pericardial effusion, is the most common cardiac issue in SLE, although cardiac tamponade is rare. Myocarditis is uncommon but can occur, particularly in those with anti-Ro

(SSA) antibodies, and should be distinguished from hydroxychloroquine-induced cardiomyopathy, which may require endomyocardial biopsy for confirmation. Valvular disease, especially Libman-Sacks endocarditis involving the mitral valve, is frequently observed and may be associated with antiphospholipid syndrome. Patients with SLE are at increased risk of coronary artery disease, either due to coronary vasculitis or, more often, accelerated atherosclerosis.[37]

Gastrointestinal manifestations of SLE can involve any part of the GI tract. Common features include esophageal dysmotility (particularly of the upper esophagus), mesenteric vasculitis, lupus enteritis, peritonitis with ascites, protein-losing enteropathy, pancreatitis, and lupoid hepatitis. Additionally, patients with antiphospholipid syndrome may develop vascular complications such as Budd-Chiari syndrome, mesenteric thrombosis, and hepatic veno-occlusive disease.[38]

A Case for SLE

Many of the documented symptoms Saint John Baptist de La Salle experienced during his life align with the diverse, multisystem nature of SLE. Although SLE was unknown in the seventeenth and eighteenth centuries, modern medical knowledge offers a framework through which his illnesses can be reexamined.

Throughout his adult life, De La Salle suffered from recurring, debilitating episodes historically described as rheumatism-a broad term once used for various inflammatory joint conditions. These episodes left him immobilized and in severe pain, requiring aggressive treatments such as prolonged heat exposure and medicinal smoke therapy. Today, such symptoms may be more precisely identified as inflammatory arthritis, a common manifestation of systemic lupus erythematosus (SLE). The recurrent, symmetrical, and severe nature of his joint pain-along with the intensity of interventions-supports the possibility of an underlying systemic autoimmune disease like lupus.

In addition to joint involvement, De La Salle also suffered from pleuritic chest pain during his final illness, described as a sudden, excruciating pain in his side that confined him to bed. This could indicate serositis, the inflammation of the linings of the lungs (pleura) or heart (pericardium), both recognized manifestations of SLE. His asthma-like symptoms and fatigue could also reflect pulmonary involvement or a general inflammatory response. Earlier in his life, he experienced urinary retention, a rare but possible complication in lupus when neurogenic bladder or neurologic involvement affects normal function.

There is also some indication of neuropsychiatric involvement, another recognized domain in SLE classification. While De La Salle remained mentally sharp for most of his life, the end of his life included a significant fall, reports of head trauma, and at least one suggestion that he may have experienced confusion or disorientation. Coupled with long-term fatigue, malaise, and systemic symptoms, these could be viewed in retrospect as part of a chronic autoimmune condition affecting multiple body systems.

One additional clue comes from descriptions of De La Salle's physical appearance. Blain describes his skin as "somewhat tanned by his long travels" and "usually slightly flushed."[39]

While easily overlooked, this observation may point to one of the most recognizable dermatologic signs of lupus: the malar rash, or "butterfly rash." This fixed erythema across the cheeks and the bridge of the nose can resemble a constant blush or mild sunburn. Notably, individuals with lupus often exhibit photosensitivity, in which sunlight exacerbates this characteristic rash. Given De La Salle's frequent exposure to the elements during his many journeys, the persistent flushed appearance described by Blain and later echoed in posthumous portraits could reflect a chronic malar rash worsened by sun exposure. Although artistic conventions of the time often portrayed subjects with rosy cheeks as a sign of health or piety, the consistency of this feature in both textual and visual representations lends weight to its possible clinical significance. Taken alongside his history of joint pain, serositis, neurological symptoms, and systemic inflammation, the presence of a persistent facial rash offers yet another compelling piece of evidence in support of systemic lupus erythematosus as a retrospective diagnosis.

Other Possibilities

Another autoimmune condition worth considering is granulomatosis with polyangiitis (GPA), a necrotizing vasculitis affecting small- to medium-sized vessels. GPA often presents with a triad of upper respiratory symptoms, lung involvement, and renal disease. While De La Salle is not known to have had hematuria or classic sinus symptoms, he did suffer from chronic respiratory symptoms described historically as "asthma," which may have reflected underlying pulmonary vasculitis. Additionally, urinary retention in the context of systemic disease raises the possibility of either neurogenic bladder or renal involvement, both seen in autoimmune vasculitides. Though not as compelling as systemic lupus erythematosus or rheumatoid arthritis, GPA remains a viable differential due to its capacity for multisystem involvement, including joints, lungs, and kidneys.[40]

Another autoimmune condition to consider is eosinophilic granulomatosis with polyangiitis (EGPA), formerly known as Churg-Strauss syndrome. EGPA is a rare systemic vasculitis characterized by adult-onset asthma, eosinophilia, and inflammation of small- to medium-sized blood vessels affecting multiple organs. De La Salle's reported history of asthma, which may have developed in adulthood, raises the possibility of EGPA, especially when viewed in the context of his systemic and musculoskeletal complaints. Additionally, while urinary retention is not a hallmark feature of EGPA, the condition can involve the nervous system, including autonomic neuropathy, which may disrupt normal bladder function. Though documentation of peripheral eosinophilia or sinus involvement is lacking in the historical record, EGPA remains a relevant differential diagnosis due to its respiratory, neurologic, and systemic manifestations, all of which align with aspects of De La Salle's described illnesses. It also shares clinical overlap with systemic lupus erythematosus and rheumatoid arthritis, further supporting its consideration.[41]

Dermatomyositis, an idiopathic inflammatory myopathy, is another condition worth considering in the differential diagnosis due to its systemic features, muscular weakness, and cutaneous findings. Classically, dermatomyositis presents with proximal muscle weakness and characteristic skin rashes, such as the heliotrope rash and Gottron's papules. Although these hallmark rashes are not mentioned in historical accounts of De La Salle, descriptions of chronic weakness, difficulty walking, and inflammatory symptoms may reflect underlying myositis.

Additionally, systemic forms of dermatomyositis can involve the lungs, gastrointestinal tract, and even cardiac muscle, potentially contributing to multi-organ decline. Dermatomyositis also shares clinical overlap with systemic lupus erythematosus, including photosensitivity, which strengthens its inclusion in the differential.[42]

Sarcoidosis is a systemic granulomatous disease of unclear etiology that can affect nearly any organ system. It commonly involves the lungs, lymph nodes, eyes, and skin, and often presents with constitutional symptoms such as fatigue, malaise, and weight loss. Although classically considered a respiratory illness, sarcoidosis can also present with neurologic, musculoskeletal, and renal manifestations. Given De La Salle's chronic illness, musculoskeletal complaints, and urinary retention, sarcoidosis with neurosarcoidosis affecting autonomic function may be a possibility. Additionally, sarcoidosis may cause uveitis or other ocular inflammation, which could correspond with De La Salle's recorded vision problems. Although rare and challenging to diagnose without biopsy, sarcoidosis remains a reasonable consideration due to its multisystem impact and clinical similarity to other autoimmune diseases.[43]

Visual Evidence: Portraiture and Artistic Interpretation

Visual representations of Saint John Baptist de La Salle have long been used to preserve and honor his legacy, though none were created during his lifetime. The most iconic image is the oil painting by Fr. Joseph Laberius, completed in 1888 and displayed at the Generalate of the Institute of the Brothers of the Christian Schools in Rome (see figure 1). This portrait, based on earlier canonical images such as the 1734 painting by Pierre Léger, shows De La Salle with a serene expression and notably flushed cheeks. While this rosy complexion may reflect artistic conventions of the time – wherein warm tones were used to convey health, sanctity, or piety – it is significant that this feature appears consistently across other depictions as well, including modern icons and mosaics (see figures 2 and 3).

One such contemporary mosaic, available from Mozaico,[44] prominently portrays De La Salle with a marked red flush across the malar region. This visual resemblance to a "butterfly rash" is striking, particularly in light of biographer Jean-Baptiste Blain's written description of De La Salle's skin as "somewhat tanned by his long travels" and "usually slightly flushed." The malar rash is one of the most recognizable signs of systemic lupus erythematosus (SLE), often worsened by sun exposure. Given De La Salle's frequent travels and outdoor exposure, the combination of textual and visual references may reflect more than stylistic choices; they may, in fact, offer subtle corroboration of a chronic dermatologic manifestation consistent with SLE. While these depictions are not diagnostic, they provide compelling artistic continuity that aligns with other suspected symptoms of autoimmune disease.

Figure 1. Portrait of Saint John Baptist de La Salle by Fr. Joseph Laberius, 1888. Displayed at the Generalate, Rome. Photograph by Rocco DiPede, June 2025. Used with permission.

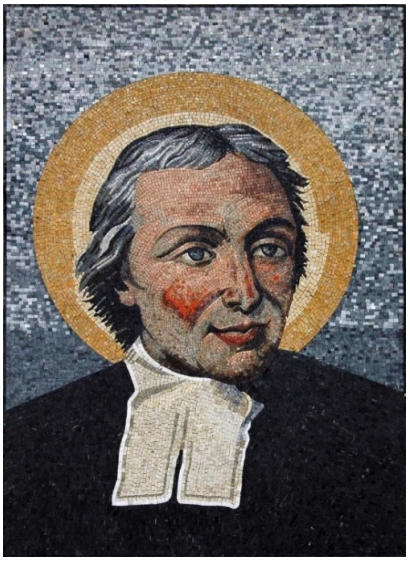

Figure 2. Mosaic icon of Saint John Baptist de La Salle. Source: Mozaico, https://www.mozaico.com/products/mosaics-designs_religious_saint-john-baptist-de-la-salle-marble-mosaic-icon_mr216.

Figure 3. Widely circulated portrait of Saint John Baptist de La Salle. Artist unknown. Public domain image; source and date unverified.

Osteological Observations: Periodontal Analysis of the Skull Relic

Further support for the hypothesis of an autoimmune condition comes from physical examination of De La Salle's remains. During a recent visit to the Generalate, the author closely examined the skull relic of Saint John Baptist de La Salle which is displayed in a glass reliquary (see figure 4). The mandible is absent, but the maxilla remains partially intact. Of particular interest are two teeth still present: tooth #6 (maxillary right canine) and tooth #13 (maxillary left premolar). Tooth #6 exhibits approximately 40% alveolar bone loss, with severe attrition to the point that it resembles a premolar in form (see figure 5). This degree of wear may indicate bruxism, which has been associated in medical literature with chronic stress and certain autoimmune diseases. Tooth #13 shows roughly 50% bone loss (see figure 6). The residual ridges of the maxilla also demonstrate generalized alveolar resorption. While such bone loss could be partially attributed to age or post-extraction remodeling, the severity and pattern raise the possibility of chronic periodontitis. Autoimmune diseases have well-documented associations with periodontal disease, including alveolar bone destruction and accelerated tooth loss.

Recent literature has highlighted a strong biological and clinical connection between systemic lupus erythematosus (SLE) and periodontal disease. In a 2021 review, Sojod et al.[45] emphasized that patients with SLE experience markedly higher rates of periodontitis, pointing to a complex interplay between immune dysregulation, chronic oral inflammation, and systemic autoimmunity. The authors cite a 2015 study by Wang et al.,[46] which found that 79% of SLE patients examined had periodontitis-over four times the prevalence typically seen in the general population. This association may be mediated by β2-glycoprotein I-dependent anti-cardiolipin antibodies that contribute to periodontal tissue destruction. A 2017 systematic review and meta-analysis by Rutter-Locher et al.[47] further confirmed a significantly increased risk and severity of periodontitis in SLE patients, reinforcing the theory of a bidirectional relationship between the two conditions. Such findings are particularly relevant in the case of Saint John Baptist de La Salle, whose skull exhibits 40-50% alveolar bone loss and near-total loss of dentition. In the absence of modern dental care, these features strongly suggest the presence of inflammatory periodontal disease, plausibly linked to an underlying autoimmune condition such as lupus.

Rheumatoid arthritis (RA), like systemic lupus erythematosus, has also been strongly associated with periodontal disease. A 2020 meta-analysis by Qiao et al.[48] analyzed data from over one million individuals and found that patients with periodontitis had a 69% higher risk of developing RA compared to those without periodontitis. The pooled odds ratio (OR) was 1.69 (95% CI: 1.31-2.17, $P<0.0001$), providing robust statistical evidence of this relationship. When stratified by disease type, the association held across both incident and mixed RA cases. Furthermore, patients with longer disease durations (>5 years) exhibited even stronger associations (OR=2.88), suggesting a potential cumulative inflammatory burden. These findings support a model in which chronic periodontal inflammation may contribute to systemic immune activation, particularly in genetically or immunologically predisposed individuals. This correlation strengthens the hypothesis that the significant bone loss observed in the maxilla of Saint John Baptist de La Salle may reflect longstanding, immune-mediated periodontal disease consistent with RA or another systemic autoimmune condition.

Although no definitive diagnosis can be made from these observations alone, the evidence of severe bone loss in conjunction with De La Salle's other systemic symptoms strengthens the plausibility of an underlying autoimmune condition. The physical remains, therefore, not only serve as relics of veneration but also as biological artifacts that offer insight into the chronic suffering endured by this saintly founder.

Figure 4. Reliquary containing the skull of Saint John Baptist de La Salle. Maxilla with remaining dentition visible. Photograph by Rocco DiPede, June 2025. Used with permission.

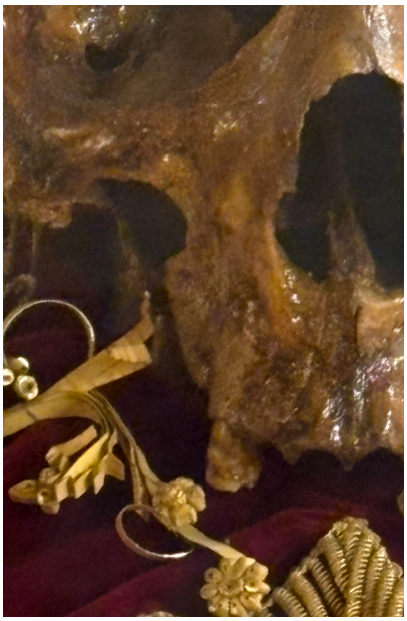

Figure 5. Close-up of tooth #6 (maxillary right canine) showing alveolar bone loss and attrition. Photograph by Rocco DiPede, June 2025. Used with permission.

Figure 6. Close-up of tooth #13 (maxillary left premolar) showing significant alveolar bone loss. Photograph by Rocco DiPede, June 2025. Used with permission.

Conclusion

Although it is impossible to provide a definitive medical diagnosis for Saint John Baptist de La Salle with the tools available in his time, a careful retrospective analysis of his symptoms, as documented in biographical sources and personal correspondence, strongly suggests the presence of a chronic systemic autoimmune disease. The recurring episodes of joint pain, severe fatigue, pleuritic chest pain, neurological symptoms, and possible photosensitive skin manifestations indicate multisystem involvement that cannot be adequately explained by aging, asceticism, or stress alone.

Among the differential diagnoses considered – rheumatoid arthritis, dermatomyositis, vasculitides such as granulomatosis with polyangiitis, and sarcoidosis – systemic lupus erythematosus (SLE) emerges as the most consistent with the broad spectrum and fluctuation of De La Salle's ailments. His probable malar rash, recurrent inflammatory arthritis, serositis, and neurologic and renal symptoms align closely with classic features of SLE. While rheumatoid arthritis is also a strong contender, the wider systemic manifestations and cutaneous clues more closely reflect the presentation of lupus.

Seen through this lens, the physical suffering endured by De La Salle may not have simply been the result of voluntary mortification or the natural rigors of his mission. Rather, it is likely that he bore the burdens of a complex and painful autoimmune disease with extraordinary patience and sanctity. Recognizing this possibility not only humanizes his life but also elevates our appreciation for his perseverance. His legacy as the patron saint of teachers is thus complemented by a deeper awareness of how chronic illness may have shaped his spirituality, empathy, and determination to serve the poor. In the mystery of divine providence, even suffering can become a means of grace – and in De La Salle's case, perhaps even a hidden path to sainthood.

[1] Michele J. Dimaira, D.M.D., M.S., M.H.A., MATHEM, M.Ed., teaches Religion and Health Science and is the Coordinator of the Health Science Concentration at La Salle College High School in Wyndmoor, Pennsylvania. She also serves as an adjunct instructor in Catholic Bioethics at Saint Joseph's University in Philadelphia. A former periodontist, her interests include the intersection of faith and medicine.
[2] Jean-Baptiste Blain, *The Life of John Baptist de La Salle: A Biography in Three Books*, ed. Luke Salm, trans. Richard Arnandez, vol. 2 (Landover, MD: Lasallian Publications, 2000, from 1733 manuscript).
[3] Luke Salm, *The Work Is Yours* (Landover, MD: Lasallian Publications, 1996), page 65.
[4] Blain, 2:279-280.
[5] Ibid., 2:314-316.
[6] Salm, *The Work Is Yours,* page 74.
[7] W. J. Battersby, *SaintJohn Baptist de La Salle* (New York: The Macmillan Company, 1957), page 184.
[8] Ibid., pages 184-185.
[9] John Baptist de La Salle, *The Letters of St. John Baptist de La Salle*, ed. and trans. Colman Molloy (Washington, DC: Christian Brothers Conference, 1988), page 96.
[10] Battersby, *St. John Baptist de La Salle,* page 185.
[11] Salm, *The Work Is Yours,* page 160.
[12] Ibid.
[13] Ibid., page 16.
[14] De La Salle, *Letters*, page 117.
[15] Salm, *The Work Is Yours*, page 182.
[16] Ibid., page 183.
[17] Ibid., page 184.
[18] Bernard Hours, *Jean-Baptiste de La Salle: A Mystic in Action*, trans. Anna Fitzgerald (Rome: Brothers of the Christian Schools, 2010), page 620.
[19] Jean-Baptiste Blain, *The Life of John Baptist de La Salle*, 3:742.
[20] Anne Davidson and Betty Diamond, "Autoimmune Diseases," *New England Journal of Medicine* 345, no. 5 (2001): 340-350, https://doi.org/10.1056/NEJM200108023450506.
[21] Noel R. Rose and Ian R. Mackay, *The Autoimmune Diseases*, 5th ed. (Amsterdam: Academic Press, 2014).
[22] Alexandru-Florin Radu and Simona Gabriela Bungau, "Management of Rheumatoid Arthritis: An Overview," *Cells* 10, no. 11 (2021): 2857, https://doi.org/10.3390/cells10112857.
[23] Alessia Conforti et al., "Beyond the Joints, the Extra-Articular Manifestations in Rheumatoid Arthritis," *Autoimmunity Reviews* 20, no. 2 (2021): 102735, https://doi.org/10.1016/j.autrev.2020.102735.
[24] Ibid.
[25] Ibid.
[26] Ibid.
[27] Ibid.
[28] Ibid.

[29] Ahmad O. Oweis et al., "Renal Dysfunction among Rheumatoid Arthritis Patients: A Retrospective Cohort Study," *Annals of Medicine and Surgery* 60 (2020): 280-284, https://doi.org/10.1016/j.amsu.2020.11.011.
[30] C. Siegel and L. R. Sammaritano, "Systemic Lupus Erythematosus," *Journal of the American Medical Association* 331, no. 17 (2024): 1480–1491, https://doi.org/10.1001/jama.2024.2315.
[31] A. A. Justiz Vaillant, A. Goyal, and M. A. Varacallo, "Systemic Lupus Erythematosus," *StatPearls* [Internet], updated August 4, 2023 (Treasure Island, FL: StatPearls Publishing, January 2025), https://www.ncbi.nlm.nih.gov/books/NBK535405/.
[32] Ibid.
[33] Ibid.
[34] Ibid.
[35] Ibid.
[36] Ibid.
[37] Ibid.
[38] Ibid.
[39] Blain, *The Life of John Baptist de La Salle*, 3:742.
[40] J. C. Jennette et al., "2012 Revised International Chapel Hill Consensus Conference Nomenclature of Vasculitides," *Arthritis & Rheumatism* 65, no. 1 (2013): 1-11, https://doi.org/10.1002/art.37715.
[41] A. Mahr et al., "Eosinophilic Granulomatosis with Polyangiitis (Churg-Strauss): Evolutions in Classification, Etiopathogenesis, Assessment and Management," *Current Opinion in Rheumatology* 26, no. 1 (2014): 16-23, https://doi.org/10.1097/BOR.0000000000000015.
[42] M. E. DeWane, R. Waldman, and J. Lu, "Dermatomyositis: Clinical Features and Pathogenesis," *Journal of the American Academy of Dermatology* 82, no. 2 (2020): 267-281, https://doi.org/10.1016/j.jaad.2019.06.1309.
[43] M. C. Iannuzzi, B. A. Rybicki, and A. S. Teirstein, "Sarcoidosis," *New England Journal of Medicine* 357, no. 21 (2007): 2153-2165, https://doi.org/10.1056/NEJMra071714.
[44] *Saint John Baptist de La Salle Marble Mosaic Icon*, Mozaico, accessed June 19, 2025, https://www.mozaico.com/products/mosaics-designs_religious_saint-john-baptist-de-la-salle-marble-mosaic-icon_mr216.
[45] B. Sojod et al., "Systemic Lupus Erythematosus and Periodontal Disease: A Complex Clinical and Biological Interplay," *Journal of Clinical Medicine* 10 (2021): 1957, https://doi.org/10.3390/jcm10091957.
[46] C.-Y. Wang et al., "β2-Glycoprotein I-Dependent Anti-Cardiolipin Antibodies Associated with Periodontitis in Patients with Systemic Lupus Erythematosus," *Journal of Periodontology* 86, no. 8 (2015): 995-1004, https://doi.org/10.1902/jop.2015.140664.
[47] Z. Rutter-Locher et al., "Association between Systemic Lupus Erythematosus and Periodontitis: A Systematic Review and Meta-Analysis," *Frontiers in Immunology* 8 (2017): 1295, https://doi.org/10.3389/fimmu.2017.01295.
[48] Yiqiang Qiao et al., "Rheumatoid Arthritis Risk in Periodontitis Patients: A Systematic Review and Meta-Analysis," *Joint Bone Spine* 87, no. 6 (2020): 556-564, https://doi.org/10.1016/j.jbspin.2020.04.024.

Bibliography

Battersby, W. J. *St. John Baptist de La Salle*. New York: Macmillan, 1957.

Blain, Jean-Baptiste. *The Life of John Baptist de La Salle: A Biography in Three Books*. Edited by Luke Salm. Translated from the 1733 manuscript by Richard Arnandez. Landover, MD: Lasallian Publications, 2000.

Conforti, Alessia, et al. "Beyond the Joints, the Extra-Articular Manifestations in Rheumatoid Arthritis." *Autoimmunity Reviews* 20, no. 2 (2021): 102735. https://doi.org/10.1016/j.autrev.2020.102735.

Davidson, Anne, and Betty Diamond. "Autoimmune Diseases." *New England Journal of Medicine* 345, no. 5 (2001): 340-350. https://doi.org/10.1056/NEJM200108023450506.

De La Salle, John Baptist. *The Letters of St. John Baptist de La Salle*. Edited and translated by Colman Molloy. Washington, DC: Christian Brothers Conference, 1988.

DeWane, M. E., R. Waldman, and J. Lu. "Dermatomyositis: Clinical Features and Pathogenesis." *Journal of the American Academy of Dermatology* 82, no. 2 (2020): 267-281. https://doi.org/10.1016/j.jaad.2019.06.1309.

Iannuzzi, M. C., B. A. Rybicki, and A. S. Teirstein. "Sarcoidosis." *New England Journal of Medicine* 357, no. 21 (2007): 2153-2165. https://doi.org/10.1056/NEJMra071714.

Jennette, J. C., et al. "2012 Revised International Chapel Hill Consensus Conference Nomenclature of Vasculitides." *Arthritis & Rheumatism* 65, no. 1 (2013): 1-11. https://doi.org/10.1002/art.37715.

Justiz Vaillant, A. A., A. Goyal, and M. A. Varacallo. "Systemic Lupus Erythematosus." *StatPearls* [Internet]. Updated August 4, 2023. Treasure Island, FL: StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK535405/.

Mahr, A., et al. "Eosinophilic Granulomatosis with Polyangiitis (Churg-Strauss): Evolutions in Classification, Etiopathogenesis, Assessment and Management." *Current Opinion in Rheumatology* 26, no. 1 (2014): 16-23. https://doi.org/10.1097/BOR.0000000000000015.

Mozaico. *Saint John Baptist de La Salle Marble Mosaic Icon*. https://www.mozaico.com/products/mosaics-designs_religious_saint-john-baptist-de-la-salle-marble-mosaic-icon_mr216.

Oweis, Ahmad O., et al. "Renal Dysfunction among Rheumatoid Arthritis Patients: A Retrospective Cohort Study." *Annals of Medicine and Surgery* 60 (2020): 280-284. https://doi.org/10.1016/j.amsu.2020.11.011.

Qiao, Yiqiang, et al. “Rheumatoid Arthritis Risk in Periodontitis Patients: A Systematic Review and Meta-Analysis.” *Joint Bone Spine* 87, no. 6 (2020): 556-564. https://doi.org/10.1016/j.jbspin.2020.04.024.

Rose, Noel R., and Ian R. Mackay. *The Autoimmune Diseases*. 5th ed. Amsterdam: Academic Press, 2014.

Rutter-Locher, Z., et al. “Association between Systemic Lupus Erythematosus and Periodontitis: A Systematic Review and Meta-Analysis.” *Frontiers in Immunology* 8 (2017): 1295. https://doi.org/10.3389/fimmu.2017.01295.

Salm, Luke. *The Work is Yours*. Landover, MD: Lasallian Publications, 1996.

Siegel, C., and L. R. Sammaritano. “Systemic Lupus Erythematosus.” *Journal of the American Medical Association* 331, no. 17 (2024): 1480-1491. https://doi.org/10.1001/jama.2024.2315.

Sojod, B., et al. “Systemic Lupus Erythematosus and Periodontal Disease: A Complex Clinical and Biological Interplay.” *Journal of Clinical Medicine* 10 (2021): 1957. https://doi.org/10.3390/jcm10091957.

Van Grieken, George. *To Touch Hearts: The Pedagogical Spirituality of St. John Baptist de La Salle*. Napa, CA: Lasallian Resource Center, 2019.

Wang, C.-Y., et al. “β2-Glycoprotein I-Dependent Anti-Cardiolipin Antibodies Associated with Periodontitis in Patients with Systemic Lupus Erythematosus.” *Journal of Periodontology* 86, no. 8 (2015): 995-1004. https://doi.org/10.1902/jop.2015.140664.