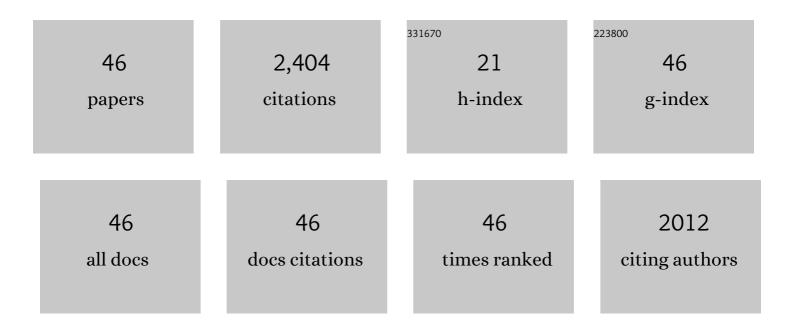
## Milena M Awad

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Towards an understanding of the role of <i>Clostridium perfringens</i> toxins in human and animal disease. Future Microbiology, 2014, 9, 361-377.	2.0	328
2	Skewed genomic variability in strains of the toxigenic bacterial pathogen, Clostridium perfringens. Genome Research, 2006, 16, 1031-1040.	5.5	281
3	Alpha-Toxin of Clostridium perfringens Is Not an Essential Virulence Factor in Necrotic Enteritis in Chickens. Infection and Immunity, 2006, 74, 6496-6500.	2.2	226
4	Identification and molecular analysis of a locus that regulates extracellular toxin production in Clostridium perfringens. Molecular Microbiology, 1994, 12, 761-777.	2.5	187
5	Synergistic Effects of Alpha-Toxin and Perfringolysin O in Clostridium perfringens -Mediated Gas Gangrene. Infection and Immunity, 2001, 69, 7904-7910.	2.2	173
6	The α-toxin ofClostridium septicumis essential for virulence. Molecular Microbiology, 2005, 57, 1357-1366.	2.5	120
7	<i>Clostridium difficile</i> virulence factors: Insights into an anaerobic spore-forming pathogen. Gut Microbes, 2014, 5, 579-593.	9.8	110
8	Disruption of the Gut Microbiome: Clostridium difficile Infection and the Threat of Antibiotic Resistance. Genes, 2015, 6, 1347-1360.	2.4	82
9	Molecular and Cellular Basis of Microvascular Perfusion Deficits Induced by Clostridium perfringens and Clostridium septicum. PLoS Pathogens, 2008, 4, e1000045.	4.7	78
10	Use of Genetically Manipulated Strains of <i>Clostridium perfringens</i> Reveals that Both Alpha-Toxin and Theta-Toxin Are Required for Vascular Leukostasis To Occur in Experimental Gas Gangrene. Infection and Immunity, 1999, 67, 4902-4907.	2.2	78
11	TcsL Is an Essential Virulence Factor in Clostridium sordellii ATCC 9714. Infection and Immunity, 2011, 79, 1025-1032.	2.2	51
12	The NanI and NanJ Sialidases of <i>Clostridium perfringens</i> Are Not Essential for Virulence. Infection and Immunity, 2009, 77, 4421-4428.	2.2	45
13	Clostridium sordellii genome analysis reveals plasmid localized toxin genes encoded within pathogenicity loci. BMC Genomics, 2015, 16, 392.	2.8	39
14	Construction and virulence testing of a collagenase mutant of Clostridium perfringens. Microbial Pathogenesis, 2000, 28, 107-117.	2.9	38
15	Antibiotic resistance, virulence factors and genetics of Clostridium sordellii. Research in Microbiology, 2015, 166, 368-374.	2.1	36
16	Comparing the identification of Clostridium spp. by two Matrix-Assisted Laser Desorption lonization-Time of Flight (MALDI-TOF) mass spectrometry platforms to 16S rRNA PCR sequencing as a reference standard: A detailed analysis of age of culture and sample preparation. Anaerobe, 2014, 30, 85-89.	2.1	34
17	Functional Analysis of the VirSR Phosphorelay from Clostridium perfringens. PLoS ONE, 2009, 4, e5849.	2.5	31
18	Expression of the large clostridial toxins is controlled by conserved regulatory mechanisms. International Journal of Medical Microbiology, 2014, 304, 1147-1159.	3.6	31

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19	Isolation of α-toxin, Î,-toxin and κ-toxin mutants ofClostridium perfringensby Tn916mutagenesis. Microbial Pathogenesis, 1997, 22, 275-284.	2.9	30
20	Functional analysis of an feoB mutant in Clostridium perfringens strain 13. Anaerobe, 2016, 41, 10-17.	2.1	27
21	Cephamycins inhibit pathogen sporulation and effectively treat recurrent Clostridioides difficile infection. Nature Microbiology, 2019, 4, 2237-2245.	13.3	27
22	Paeniclostridium (Clostridium) sordellii–associated enterocolitis in 7 horses. Journal of Veterinary Diagnostic Investigation, 2020, 32, 239-245.	1.1	26
23	The Level of Expression of $\hat{I}_{\pm}$ -toxin by Different Strains ofClostridium perfringensis Dependent on Differences in Promoter Structure and Genetic Background. Anaerobe, 1996, 2, 365-371.	2.1	24
24	The FxRxHrS Motif: A Conserved Region Essential for DNA Binding of the VirR Response Regulator from Clostridium perfringens. Journal of Molecular Biology, 2002, 322, 997-1011.	4.2	24
25	The Pore-Forming α-Toxin from Clostridium septicum Activates the MAPK Pathway in a Ras-c-Raf-Dependent and Independent Manner. Toxins, 2015, 7, 516-534.	3.4	22
26	Necrotic Enteritis in Chickens Associated withClostridium sordellii. Avian Diseases, 2015, 59, 447-451.	1.0	20
27	Lectin Activity of the TcdA and TcdB Toxins of Clostridium difficile. Infection and Immunity, 2019, 87, .	2.2	20
28	Tranexamic Acid Influences the Immune Response, but not Bacterial Clearance in a Model of Post-Traumatic Brain Injury Pneumonia. Journal of Neurotrauma, 2019, 36, 3297-3308.	3.4	20
29	pCP13, a representative of a new family of conjugative toxin plasmids in Clostridium perfringens. Plasmid, 2019, 102, 37-45.	1.4	17
30	Novel Use of Tryptose Sulfite Cycloserine Egg Yolk Agar for Isolation of <i>Clostridium perfringens</i> during an Outbreak of Necrotizing Enterocolitis in a Neonatal Unit. Journal of Clinical Microbiology, 2010, 48, 4263-4265.	3.9	16
31	Structural Characterization of Clostridium sordellii Spores of Diverse Human, Animal, and Environmental Origin and Comparison to Clostridium difficile Spores. MSphere, 2017, 2, .	2.9	16
32	Clostridium sordellii Pathogenicity Locus Plasmid pCS1-1 Encodes a Novel Clostridial Conjugation Locus. MBio, 2018, 9, .	4.1	16
33	TcdB or not TcdB: a tale of twoClostridium difficiletoxins. Future Microbiology, 2011, 6, 121-123.	2.0	15
34	The Cysteine Protease α-Clostripain is Not Essential for the Pathogenesis of Clostridium perfringens-Mediated Myonecrosis. PLoS ONE, 2011, 6, e22762.	2.5	15
35	Chromosome Segregation and Peptidoglycan Remodeling Are Coordinated at a Highly Stabilized Septal Pore to Maintain Bacterial Spore Development. Developmental Cell, 2021, 56, 36-51.e5.	7.0	13
36	The Sialidase NanS Enhances Non-TcsL Mediated Cytotoxicity of Clostridium sordellii. Toxins, 2016, 8, 189.	3.4	12

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#	Article	IF	CITATIONS
37	<i>Clostridium septicum</i> α-toxin activates the NLRP3 inflammasome by engaging GPI-anchored proteins. Science Immunology, 2022, 7, .	11.9	12
38	Clostridium sordellii outer spore proteins maintain spore structural integrity and promote bacterial clearance from the gastrointestinal tract. PLoS Pathogens, 2018, 14, e1007004.	4.7	11
39	Perfringolysin O Expression in Clostridium perfringens Is Independent of the Upstream pfoR Gene. Journal of Bacteriology, 2002, 184, 2034-2038.	2.2	8
40	Utility of the Clostridial Site-Specific Recombinase TnpX To Clone Toxic-Product-Encoding Genes and Selectively Remove Genomic DNA Fragments. Applied and Environmental Microbiology, 2014, 80, 3597-3603.	3.1	8
41	The NEAT Domain-Containing Proteins of Clostridium perfringens Bind Heme. PLoS ONE, 2016, 11, e0162981.	2.5	8
42	Opioid Analgesics Stop the Development of Clostridial Gas Gangrene. Journal of Infectious Diseases, 2014, 210, 483-492.	4.0	7
43	Human Plasminogen Exacerbates Clostridioides difficile Enteric Disease and Alters the Spore Surface. Gastroenterology, 2020, 159, 1431-1443.e6.	1.3	7
44	Paeniclostridium sordellii and Clostridioides difficile encode similar and clinically relevant tetracycline resistance loci in diverse genomic locations. BMC Microbiology, 2019, 19, 53.	3.3	5
45	A dynamic, ring-forming MucB / RseB-like protein influences spore shape in Bacillus subtilis. PLoS Genetics, 2020, 16, e1009246.	3.5	5
46	A Highly Specific Holin-Mediated Mechanism Facilitates the Secretion of Lethal Toxin TcsL in Paeniclostridium sordellii. Toxins, 2022, 14, 124.	3.4	5