THERAPEUTIC POTENTIALS OF BIOACTIVE COMPOUNDS

A HOLISTIC APPROACH



Editors:
Khanage Shantaram G.
Waghamare Suresh
Sayyad Jahed

Bentham Books

Therapeutic Potentials of Bioactive Compounds: A Holistic Approach

Edited by

Khanage Shantaram G.
Department of Pharmaceutical Chemistry
Rashtriya College of Pharmacy, Hatnur
Aurangabad, Maharashtra, India

Waghamare Suresh
Department of Pharmaceutical Science
NIMS Institute of Pharmacy, NIMS University
Jaipur, Rajasthan, India

&

Sayyad Jahed
Department of Pharmaceutics
Rashtriya College of Pharmacy
Hatnur, Aurangabad
Maharashtra, India

Therapeutic Potentials of Bioactive Compounds: A Holistic Approach

Editors: Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed

ISBN (Online): 979-8-89881-804-3

ISBN (Print): 979-8-89881-805-0

ISBN (Paperback): 979-8-89881-806-7

© 2026, Bentham Books imprint.

Published by Bentham Science Publishers Pte. Ltd. Singapore, in collaboration with Eureka Conferences, USA. All Rights Reserved.

First published in 2026.

BENTHAM SCIENCE PUBLISHERS LTD.

End User License Agreement (for non-institutional, personal use)

This is an agreement between you and Bentham Science Publishers Ltd. Please read this License Agreement carefully before using the ebook/echapter/ejournal (**"Work"**). Your use of the Work constitutes your agreement to the terms and conditions set forth in this License Agreement. If you do not agree to these terms and conditions then you should not use the Work.

Bentham Science Publishers agrees to grant you a non-exclusive, non-transferable limited license to use the Work subject to and in accordance with the following terms and conditions. This License Agreement is for non-library, personal use only. For a library / institutional / multi user license in respect of the Work, please contact: permission@benthamscience.org.

Usage Rules:

1. All rights reserved: The Work is the subject of copyright and Bentham Science Publishers either owns the Work (and the copyright in it) or is licensed to distribute the Work. You shall not copy, reproduce, modify, remove, delete, augment, add to, publish, transmit, sell, resell, create derivative works from, or in any way exploit the Work or make the Work available for others to do any of the same, in any form or by any means, in whole or in part, in each case without the prior written permission of Bentham Science Publishers, unless stated otherwise in this License Agreement.
2. You may download a copy of the Work on one occasion to one personal computer (including tablet, laptop, desktop, or other such devices). You may make one back-up copy of the Work to avoid losing it.
3. The unauthorised use or distribution of copyrighted or other proprietary content is illegal and could subject you to liability for substantial money damages. You will be liable for any damage resulting from your misuse of the Work or any violation of this License Agreement, including any infringement by you of copyrights or proprietary rights.

Disclaimer:

Bentham Science Publishers does not guarantee that the information in the Work is error-free, or warrant that it will meet your requirements or that access to the Work will be uninterrupted or error-free. The Work is provided "as is" without warranty of any kind, either express or implied or statutory, including, without limitation, implied warranties of merchantability and fitness for a particular purpose. The entire risk as to the results and performance of the Work is assumed by you. No responsibility is assumed by Bentham Science Publishers, its staff, editors and/or authors for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products instruction, advertisements or ideas contained in the Work.

Limitation of Liability:

In no event will Bentham Science Publishers, its staff, editors and/or authors, be liable for any damages, including, without limitation, special, incidental and/or consequential damages and/or damages for lost data and/or profits arising out of (whether directly or indirectly) the use or inability to use the Work. The entire liability of Bentham Science Publishers shall be limited to the amount actually paid by you for the Work.

General:

1. Any dispute or claim arising out of or in connection with this License Agreement or the Work (including non-contractual disputes or claims) will be governed by and construed in accordance with the laws of Singapore. Each party agrees that the courts of the state of Singapore shall have exclusive jurisdiction to settle any dispute or claim arising out of or in connection with this License Agreement or the Work (including non-contractual disputes or claims).
2. Your rights under this License Agreement will automatically terminate without notice and without the

need for a court order if at any point you breach any terms of this License Agreement. In no event will any delay or failure by Bentham Science Publishers in enforcing your compliance with this License Agreement constitute a waiver of any of its rights.

3. You acknowledge that you have read this License Agreement, and agree to be bound by its terms and conditions. To the extent that any other terms and conditions presented on any website of Bentham Science Publishers conflict with, or are inconsistent with, the terms and conditions set out in this License Agreement, you acknowledge that the terms and conditions set out in this License Agreement shall prevail.

Bentham Science Publishers Pte. Ltd.
No. 9 Raffles Place
Office No. 26-01
Singapore 048619
Singapore
Email: subscriptions@benthamscience.net



CONTENTS

FOREWORD i

PREFACE ii

LIST OF CONTRIBUTORS iii

CHAPTER 1 ADVANCED ALKALOID DELIVERY SYSTEMS FOR EFFECTIVE MANAGEMENT OF PARKINSON'S SYMPTOMS 1

Annasaheb Kudhekar, Waghamare Suresh, Poonam Kodag, Sayyad Jahed, Darwade Amol and *Garad Manoj*

INTRODUCTION 2

Current Treatment Options 2

ROLE OF ALKALOIDS IN PARKINSON'S DISEASE 4

Alkaloids Relevant to PD Treatment 4

Challenges in Parkinson's Treatment 5

Blood-brain Barrier (BBB) and Drug Delivery 5

Side Effects and Efficacy Limitations 6

Alkaloid-based Therapeutics in Parkinson's Disease 7

Pharmacological Mechanisms of Alkaloids 8

Mechanism of Alkaloids in Parkinson's Symptoms 9

Modulation of the Dopaminergic System 9

Neuroprotection and Antioxidant Effects 9

Anti-Inflammatory Actions 10

Promotion of Autophagy and Clearance of α-Synuclein Aggregates 10

Mechanisms by Which Alkaloids Exacerbate Parkinson's Symptoms 10

Dopaminergic Modulation and Receptor Interactions 11

Enzyme Inhibition or Stimulation 12

Notable Alkaloids for PD 13

LIMITATIONS OF CURRENT ALKALOID THERAPIES 13

Short Half-life and Poor Bioavailability 14

Neurotoxicity and Side Effects 15

Challenges in Drug Delivery to the Brain 15

BLOOD-BRAIN BARRIER (BBB) 16

Structure and Function of BB 16

Barriers to Effective Drug Delivery 17

Pharmacokinetics of Alkaloids 17

Absorption, Distribution, Metabolism, and Excretion (ADME) Concerns 18

Issues with Alkaloid Stability and Solubility 19

NEED FOR ADVANCED DELIVERY SYSTEMS 19

Why Conventional Delivery Systems Fall Short 19

Overview of Advanced Delivery Strategies 19

ADVANCED ALKALOID DELIVERY SYSTEMS 20

Nanotechnology-based Delivery Systems 20

Use of Biodegradable Polymers 21

Controlled Release Mechanisms and Sustained Delivery 21

Receptor-Mediated and Carrier-Mediated Delivery 22

Transport Across the BBB using Carrier Proteins 22

Surface Modification to Enhance Targeting 22

Pro-drug Strategies 23

Development of Prodrugs for Enhanced BBB Permeability 23

INNOVATIVE DELIVERY APPROACHES FOR PARKINSON'S TREATMENT 23

Microneedle Arrays and Transdermal Systems 24
Non-invasive Delivery of Alkaloids 24
Benefits of Sustained Transdermal Drug Release 25
Gene Therapy and Alkaloid Gene Delivery 25
Genetic Modifications to Improve Alkaloid Metabolism in the Brain 25
Intranasal Delivery Systems 26
Non-invasive Route for Drug Delivery Directly to the Brain 26
RESEARCH PROGRESS AND CHALLENGES 27
PHARMACODYNAMICS AND EFFICACY OF ADVANCED ALKALOID DELIVERY SYSTEMS 27
CLINICAL OUTCOMES AND CASE STUDIES 27
Review of Clinical Trials and Preclinical Studies 28
Results from Alkaloid-based Systems in PD Treatment 28
SIDE EFFECTS AND RISK EVALUATION 29
Potential Toxicities of Advanced Systems 29
Balancing Eficacy and Safety in Drug Delivery 29
FUTURE PERSPECTIVES AND CHALLENGES 30
Emerging Technologies in Alkaloid Delivery 30
Smart Delivery Systems 30
Combination Therapies for Synergistic Effects 31
CONCLUSION 31
CONSENT FOR PUBLICATION 31
ACKNOWLEDGEMENTS 32
AI USE DISCLOSURE 32
REFERENCES 32

CHAPTER 2 FORMULATIONS OF GLYCOSIDES FOR CARDIAC DISEASES IN HEART FAILURE MANAGEMENT 39
Chandrashekhar D. Khadse and *Avinash S. Jiddewar*
INTRODUCTION 39
CARDIAC DISEASES 43
Signs and Symptoms 43
Symptoms of Heart Issues 43
Symptoms of Blockages in Vessels 43
Risk Factors 43
Types of Cardiovascular Diseases 44
Causes of Cardiovascular Disease 44
Diagnosis of Cardiovascular Diseases 44
Mechanism of Action 45
POTENTIAL SIDE EFFECTS 45
CARDIAC GLYCOSIDE POISONING 46
Specific Treatments for Cardenolide Poisoning 46
Novel Therapeutic Applications 47
CONCLUSION 47
CONSENT FOR PUBLICATION 48
ACKNOWLEDGEMENTS 48
AI USE DISCLOSURE 48
REFERENCES 48

CHAPTER 3 THERAPEUTIC APPLICATION OF GLYCOSIDE-BASED FORMULATIONS IN GASTROINTESTINAL AND METABOLIC DISORDERS 51

Ganesh Godge, Umesh Panchal, Bhagyashri Randhawan, Manish Raskar and *Varsharani Avhad*
INTRODUCTION 52
Overview of Glycosides 53
Classification of Glycosides 53
Pharmacokinetics of Glycosides 54
Mechanism of Action 55
Therapeutic Applications in Gastrointestinal Disorders 55
Therapeutic Application in Metabolic Disorders 55
THERAPEUTIC APPLICATION OF TYPES OF GLYCOSIDES ON GASTROINTESTINAL AND METABOLIC DISORDERS 56
Cardiac Glycoside 56
Cardiac Glycosides in Gastrointestinal Disorders 56
Mobility Regulation in the GI Tract 57
Cardiac Glycosides in Metabolic Disorders 58
Flavonoid Glycoside 59
Flavonoid Glycosides in Gastrointestinal Disorders 59
Flavonoid Glycoside in Metabolic Disorders 61
Antidiabetic Effects 61
Anti-Obesity Effects 61
Mechanism of Action 62
Clinical Application 62
Antioxidant and Anti-Inflammatory Effects in Metabolic Disorders 62
Saponin Glycoside 62
Gastrointestinal Benefits of Saponin Glycosides 63
Protection Against Gastric Ulcers 63
Antimicrobial Activity in the Gut 63
Anti-Inflammatory Effects on the Gut Lining 64
Role in Intestinal Permeability and Leaky Gut 64
Digestive Enzyme Modulation 64
Metabolic Benefits of Saponin Glycosides 64
Cholesterol and Lipid-Lowering Effects 65
Anti-obesity Effects 65
Hepatoprotective Effects 65
Anthraquinone Glycosides 65
Gastrointestinal Benefits of Anthraquinone Glycosides 66
Laxative and Bowel-Stimulating Effects 66
Effect on Gut Microbiota 66
Anti-Inflammatory and Anti-Ulcer Effect 66
Protection Against Colorectal Cancer 67
Metabolic Benefits of Anthraquinone Glycosides 67
Anti-Diabetic Effects 67
Lipid-Lowering and Anti-Obesity Effects 67
Hepatoprotective Effect (Liver Protection) 68
Phenolic Glycoside 68
Gastrointestinal Benefits of Anthraquinone Glycosides 68
Protection against Gastric Ulcers and Mucosal Damage 68
Anti-microbial activity in the Gut 69
Modulation of Gut Microbiota and Prebiotic Effects 69
Anti-Inflammatory Effects on Intestinal Lining 69
Role in Intestinal Permeability and Leaky Gut 70

Metabolic Benefits of Phenolic Glycosides 70
Regulation of Glucose Metabolism 70
Cholesterol and Lipid-Lowering Effects 70
Anti-Obesity Effects 71
Hepatoprotective Effects (Liver Protection) 71
Alkaloid Glycoside 71
Gastrointestinal Benefits of Alkaloid Glycosides 71
Protection against Gastric Ulcers and Acid Regulation 71
Regulation of Gut Motility and Constipation Relief 72
Metabolic Benefits of Alkaloid Glycosides 73
Anti-diabetic Effects and Glucose Regulation 73
Lipid Lowering and Cardio-protective Effects 73
Anti-Obesity Effects 74
Hepatoprotective Effects (Liver Protection) 74
Formulation for Treatment of Gastrointestinal and Metabolic Disorders 74
Laxative Effect (Treatment of Constipation) 74
Gastroprotective and Anti-Ulcer Activity 75
Flavonoid Glycosides 75
Saponin Glycosides 75
Anti-Inflammatory Action (Treatment of IBD and Colitis) 76
Iridoid Glycosides 76
Saponin Glycosides (Ginsenodies, Disogenin) 76
Hepatoprotective Effect (Treatment of Liver Disorders) 76
Flavonoid and Saponin Glycoside 76
Anti-Microbial and Antidiarrheal Effects 77
Alkaloidal Glycosides (Berberine) 77
CONCLUSION 77
CONSENT FOR PUBLICATION 78
ACKNOWLEDGEMENT 78
AI USE DISCLOSURE 78
REFERENCES 78

CHAPTER 4 UTILIZING PHENOLIC COMPOUNDS TO MITIGATE OXIDATIVE STRESS IN TYPE 2 DIABETES MANAGEMENT 84
Poonam Patil, Sunil Aher, Sarita Pawar, Seema Gosavi and *Komal Shelar*
INTRODUCTION 84
A SUMMARY OF PHENOLIC SUBSTANCES 86
PHENOLIC COMPOUNDS MECHANISMS FOR INHIBITING TYPE II DIABETES RELATED ENZYMES A-AMYLASE AND A-GLUCOSIDASE 89
How Stress Produced from Oxidation Affects the Pathogenesis of Diabetes 91
Polyphenolic Activities for the Prevention of Diabetes Mellitus: Antioxidant Activity 92
Diabesity 94
Anti-inflammatory Activity 96
THE USE OF PHYTOCHEMICALS TO PREVENT CARDIOVASCULAR DISEASE 97
POLYPHENOLS AND GUT MICROBIOTA: DECIPHERING THEIR FUNCTION IN PREVENTING DIABETES 98
Examining Natural Polyphenols' Synergistic Potential for Metabolic Conditions 99
THE NEGATIVE ASPECTS OF PHENOLICS: POTENTIAL HAZARDS AND TOXICOLOGICAL EFFECTS 100
CONCLUSION 102
ACKNOWLEDGEMENTS 103

AI USE DISCLOSURE 103
REFERENCES 103

CHAPTER 5 SAPONINS AS NATURAL CHOLESTEROL-LOWERING AGENTS FOR REDUCING THE RISK OF CARDIOVASCULAR DISEASE DEVELOPMENT 111
Anurag Mishra, Sarita Rawat, Karuna Dhaundhiyal, Mohit Agarwal and *Divya Negi*
INTRODUCTION TO CARDIOVASCULAR DISEASES (CVDS) 112
Etiology of Cardiovascular Diseases 113
Global Burden of CVDs: Epidemiology and Risk Factors 113
ATHEROSCLEROSIS IN CARDIOVASCULAR DISEASE 114
CURRENT APPROACHES TO CVD PREVENTION AND MANAGEMENT 115
UNDERSTANDING CHOLESTEROL AND LIPID METABOLISM 116
Conversion of Mevalonate to Isopentenyl Phosphate 118
Formation of Squalene 118
Synthesis of Lanosterol 118
Formation of Cholesterol 119
Energy Demands 119
Possible Biochemical Mechanism of the Saponins as Cholesterol-lowering Agents 120
Inhibition of Cholesterol Absorption in the Intestines 120
A. Activation of Hepatic Cholesterol Metabolism 121
B. Alteration of Gut Microbiota 121
C. Antioxidant and Anti-inflammatory Effects 121
D. Impact on Lipid Profile Beyond Cholesterol 122
E. Clinical Evidence and Effectiveness 122
ROLE OF LIPOPROTEINS: LDL, HDL, AND TRIGLYCERIDES IN CVD RISK 122
NATURAL PRODUCTS IN CARDIOVASCULAR HEALTH 123
Flavonoids 124
Olive Oil 124
Omega-3 Fatty Acids 124
Resveratrol 125
Coffee 125
Soy 125
SAPONINS: STRUCTURE AND BIOCHEMICAL PROPERTIES 126
Chemistry of Saponins 127
Diverse Structures and Functions of Saponins 127
Cardioprotective Activity of Saponins 128
Antioxidant Activity 128
Anti-atherosclerosis and Hypolipidemic Activity 128
Saponins as Antidepressant Agents 129
Saponins as Anti-cancer Agents 129
Angiogenic Activity 130
Other Pharmacological Activities 132
Dietary Sources and Phytochemical Profiles of Saponins 132
Applications of Saponins in Foods 133
Advances in Analytical Technique for Saponin Research 134
Conventional Extraction Method 134
Maceration Extraction 134
Soxhlet Extractions 135
Subsequent Extraction 135

Green Extraction Method 135
Ultrasonic Extraction 135
Microwave-assisted Extraction 136
Accelerated Solvent Extraction 136
Isolation of Saponin 136
SFC 137
Foam Fractionation 137
ANALYTICAL METHODS 137
GC/GC-MS 137
LC and LC/MS 138
Thin Layer Chromatography 138
Near-infrared Spectroscopy (NIRS) 138
HPLC 138
Quantification of Saponins 138
Spectrophotometric Method 139
Chromatographic Method 139
Integrative and Multidisciplinary Approaches 139
Saponin Content in Food 139
Saponins as a Food Supplement and Medication 140
Use of Saponins in the Industrial and Agricultural Sectors 140
CONCLUSION 141
CONSENT FOR PUBLICATION 141
ACKNOWLEDGEMENT 142
AI USE DISCLOSURE 142
REFERENCES 142

CHAPTER 6 TANNINS' ROLE IN ELEVATING SYMPTOMS OF IRRITABLE BOWEL SYNDROME THROUGH GUT HEALTH IMPROVEMENT 149
Kalyani Autade, R. B Pandhare, Snehal R. Kadam, Shivani S. Kale, Rajashri B. Sumbe and *S. S. Dange*
INTRODUCTION 150
INTRODUCTION OF TANNINS 151
Chemistry of Tannin 153
Hydrolysable Tannin 153
Complex Tannin 154
Phlorotannins 154
Condensed Tannin 154
PATHOPHYSIOLOGY OF IBS 155
TANNINS AND GUT HEALTH: MECHANISM OF ACTION 158
Evidence-based Studies on Tannins in IBS Management 162
Dietary Sources of Tannins for IBS Patients 163
Challenges and Future Direction 165
Limitations of Current Research on Tannins and IBS 165
Bioavailability and Dosage Optimization 165
Bioavailability of Tannins 165
Dose Optimization of Tannins 166
Pharmacokinetics of Tannins 166
DEVELOPMENT OF FUNCTIONAL FOOD FOR TARGETING IBS 166
Probiotics 166
Dietary Fiber 166
Omega-3 Fatty Acids 167

Low-FODMAP Ingredients 167
POTENTIAL SIDE EFFECTS AND CONTRAINDICATIONS OF SPECIFIC POPULATIONS 167
CONCLUSION 167
CONSENT FOR PUBLICATION 168
ACKNOWLEDGEMENT 168
AI USE DISCLOSURE 168
REFERENCES 168

CHAPTER 7 ESSENTIAL OILS IN ASTHMA MANAGEMENT: EASING SYMPTOMS THROUGH ANTI-INFLAMMATORY PROPERTIES 173
Anurag Mishra, Waghamare Suresh, Annasaheb Kudhekar, Pooja Paulbudhe and *Sayyad Jahed*
INTRODUCTION 174
HISTORY OF ESSENTIAL OILS IN TRADITIONAL MEDICINE 175
Ancient Egypt 175
Traditional Chinese Medicine (TCM) 176
Ayurveda [Indian Traditional Medicine] 177
Ancient Greece and Rome 177
The Islamic Golden Age [8th to 13th Century CE) 178
Medieval and Renaissance Europe 178
Modern Aromatherapy Revival [Early to Mid-20th Century) 178
MODERN INDUSTRIAL USES OF ESSENTIAL OILS 179
UNDERSTANDING ASTHMA PATHOPHYSIOLOGY 180
Triggers and Exacerbating Factors 181
Traditional Treatments and their Shortcomings 181
ESSENTIAL OILS COMPOSITION AND MECHANISM OF ACTION 182
Mechanisms of Anti-Inflammatory and Bronchodilatory Effects 183
Anti-Inflammatory Effects of Essential Oils 183
Bronchodilatory Actions of Essential Oils 183
Methods of Action 184
Inhalation 184
Topical Application 184
Oral Use 185
KEY ESSENTIAL OILS FOR ASTHMA MANAGEMENT 185
Eucalyptus Oil: Mucolytic and Bronchodilator Properties 185
Peppermint Oil: Smooth Muscle Relaxant Properties 186
Lavender Oil: Anti-Inflammatory and Sedative Effects 186
Tea Tree Oil: Antimicrobial Properties 187
Frankincense Oil: Immune-Modulating Properties 188
Scientific Evidence and Clinical Studies 190
Summary of Research on Essential Oils and Asthma 190
Potential Risks and Gaps in Current Studies on Essential Oils for Asthma 191
SAFE AND EFFECTIVE USE OF ESSENTIAL OILS 192
Proper Dilution and Application Methods for Essential Oils in Asthma Management 192
Dilution Guidelines 192
Diffusion [Aromatherapy] 193
Steam Inhalation 193
Topical Application [Chest Rubs, Massage] 193
Personal Inhalers 193

Precautions and Contraindications 193
INTEGRATIVE APPROACH TO ASTHMA MANAGEMENT 194
Combining Essential Oils with Conventional Asthma Treatments 194
Complementary Benefits 194
Lifestyle Modifications for Better Asthma Control 195
Physical Activity & Breathing Techniques 195
Stress Management & Sleep Hygiene 195
Diet & Hydration 195
Dietary Considerations for Asthma Management 196
Beneficial Foods 196
Foods to Avoid 197
Environmental Considerations for Asthma Control 197
Indoor Air Quality 198
Allergen & Irritant Control 198
Smoke & Pollution 198
FUTURE DIRECTIONS 199
Future Prospects of Essential Oils in Asthma Management 199
Progressing Clinical Research and Evidence-Based Utilization 199
Mechanistic Insight 199
Blending Essential Oils with Traditional Therapies 199
New Delivery Methods 199
Personalized and Preventive Asthma Care 200
Regulatory Standards and Safety Guidelines 200
Widening Awareness and Medical Integration 200
CONCLUSION 200
CONSENT FOR PUBLICATION 201
ACKNOWLEDGEMENT 201
AI USE DISCLOSURE 201
REFERENCES 201

CHAPTER 8 THE USE OF NATURAL RESINS IN TREATING FUNGAL SKIN INFECTIONS AND PROMOTING HEALING 209
Rajashri B. Sumbe, Atul A. Shirkhedkar, Shivani S. Kale, Snehal R. Kadam, Kalyani Autade and *Vishal V. Pande*
INTRODUCTION 209
Fungal Infection 210
Historical Background 212
CHEMISTRY OF NATURAL RESINS 213
Dracaena Draco L 213
Frankincense Oleogum Resin 214
Black Dammar Resin 215
Mango Gum-Resin 215
Mechanisms of Antifungal Action 216
SOURCES OF NATURAL RESINS 218
Myrrh (oleo-gum resin) 219
Harvesting Methods 219
Ecological Consideration 219
Propolis 219
Frankincense 219
Harvesting 220
Ecological Condition 220

Pine Resin 220
Fir Resin 220
ANTIFUNGAL PROPERTIES OF NATURAL RESINS 220
Propolis 220
Spectrum Activity 221
Topical Application 221
Oral Administration 221
Frankincense 221
Spectrum Activity 222
Topical Application 222
Application in the Vagina 222
Myrrh 222
Spectrum Activity 222
Topical Application 223
ROLE OF NATURAL RESINS IN PROMOTING HEALING 224
Myrrh- Natural Healing Agent 224
Wound-Healing Properties 224
Mechanism of Action 225
Combination therapy 225
ADVANCES IN RESIN-BASED NANOTECHNOLOGY FOR TARGETED DELIVERY 226
Nanotechnology 226
CASE STUDIES AND PRACTICAL APPLICATIONS 227
Dracaena Cinnabari Resin 227
Conventional Success Stories 227
Practical Application 227
GUIDELINES FOR SAFE USE IN VULNERABLE POPULATIONS 228
General Guidelines 228
For Children 229
For Pregnant Women 229
FUTURE PERSPECTIVE 229
Synthetic Alterations to Increase Effectiveness 229
Trends in Resin-based Skincare And Pharmaceutical Products 230
CONCLUDING REMARKS 230
CONSENT FOR PUBLICATION 230
ACKNOWLEDGEMENT 230
AI USE DISCLOSURE 231
REFERENCES 231

CHAPTER 9 ANTIMICROBIAL PROPERTIES OF ANTHRAQUINONES FOR TREATING BACTERIAL INFECTIONS IN GASTROINTESTINAL DISORDERS 238
Waghamare Suresh, Khanage Shantaram G. and *Sayyad Jahed*
INTRODUCTION 239
INTRODUCTION TO ANTHRAQUINONES AND THEIR BIOLOGICAL PROPERTIES 240
Importance of Antimicrobial agents in Gastrointestinal Diseases 241
Anthraquinones: Chemistry and Sources 242
Natural Sources of Anthraquinones 243
Pharmacological Properties of Anthraquinones 243
Laxative Activity 243
Antimicrobial and Antiviral Activity 244
Anticancer Properties 244
Anti-inflammatory and Antioxidant Effects 244

Hepatoprotective and Nephroprotective Effects 244
Cardiovascular and Antidiabetic Activity 244
Mechanism of Action of Anthraquinones against Bacterial Pathogens 245
Inhibition of Bacterial Cell Wall Synthesis 245
Disruption of Bacterial Membrane Integrity 246
Impacts on Bacterial Metabolic Processes 247
Interaction with Bacterial Enzymes and DNA 248
Antibacterial Activity of Anthraquinones 248
Spectrum of Antibacterial Activity of Anthraquinones 248
Activity of Anthraquinones against Common Gastrointestinal Pathogens 249
Helicobacter pylori 249
Escherichia coli 249
Salmonella spp. 249
Clostridioides difficile 250
Vibrio cholerae 250
Comparison of Anthraquinones with Conventional Antibiotics 250
Mechanism of Action 250
Effectiveness against Drug-resistant Bacteria 250
Spectrum of Activity 251
Side Effects and Toxicity 251
Potential for Clinical Application 251
Antimicrobial Resistance and Anthraquinone 251
Effects on Drug-resistant Bacteria 252
Inhibition of Efflux Pumps 252
Biofilm Disruption and Anti-Quorum Sensing Activity 252
Potential Limitations and Challenges 252
Pharmacokinetics and Bioavailability of Anthraquinones 253
Absorption in the Gastrointestinal Tract 253
Distribution and Bioavailability 253
Excretion and Gut Microbiome Interaction 253
METABOLISM OF ANTHRAQUINONES 254
Gut Microbiota-Mediated Metabolism 254
Hepatic Metabolism (Phase I and Phase II Reactions) 254
Phase I (Oxidation and Reduction) 254
Phase II (Conjugation and Detoxification) 254
Excretion and Enterohepatic Circulation 254
Implications for Therapeutic Use 255
Delivery Systems to Improve Targeting to the Gastrointestinal Tract 256
NANOTECHNOLOGY AND ANTHRAQUINONE-BASED DRUG DELIVERY 257
Nanoparticle-based Delivery Systems 257
Targeted and Smart Drug Delivery 258
Synergistic Effects of Anthraquinone with Other Antimicrobial Agents and Conventional Antibiotics 258
Potential for Reducing Antibiotic Resistance 259
TOXICOLOGICAL CONSIDERATIONS AND SAFETY PROFILE OF ANTHRAQUINONE 259
Animal Toxicity Studies 259
Human Toxicity and Clinical Observations 260
Safety Concerns in Long-Term Use 260
Future Directions and Challenges 260

Nanotechnology-Enhanced Drug Delivery 261
Structural Modifications and Synthetic Derivatives 261
Expanding Therapeutic Applications 261
CONCLUSION 262
CONSENT FOR PUBLICATION 262
ACKNOWLEDGEMENT 262
AI USE DISCLOSURE 262
REFERENCES 262

CHAPTER 10 UTILIZING COUMARINS AS ANTICOAGULANTS FOR STROKE PREVENTION IN PATIENTS WITH ATRIAL FIBRILLATION 270
Vishal V. Pande, Nandini P. Dolase, Rajashri B. Sumbe, Kalyani Autade, Sofiya Mujawar and *Diksha M. Randive*
INTRODUCTION 271
INITIATION OF ATRIAL FIBRILLATION 271
Ectopic Activation from the Pulmonary Veins 271
Maintenance of Atrial Fibrillation 272
Multiwavelet Re-entry 272
Localized Regions, Focal and Rotational Drivers 272
Focal Activation 272
Rotational Drivers 273
SILENT AF AND RISK OF STROKE IN AF 274
PATHOPHYSIOLOGY OF AF 275
COUMARINS AS ANTICOAGULANTS 275
Coumarins 275
Coumarin Anticoagulants: Indications 276
Pharmacokinetics, Metabolism, and Differences between Coumarin 276
VKORC1-pharmacodynamics 278
COMMON COUMARIN DERIVATIVES 278
Warfarin 278
Pharmacology 279
Pharmacokinetics 280
Pharmacodynamics 281
Drug Interactions 282
Acenocoumarol 283
Molecular Structure and Spectral Identification 283
Synthesis 283
Pharmacodynamic and Pharmacokinetics of Acenocoumarol 284
Metabolism, Absorption, and Elimination 284
Clopidogrel 285
Combination Anticoagulant and Antiplatelet Therapy 285
DOSING STRATEGIES 287
Genotype-guided Dosing Algorithms for Warfarin 287
Genotype-guided Dosing Algorithms for Acenocoumarol and Phenprocoumon 287
EFFICACY AND SAFETY PROFILE 288
Comparison with Direct Oral Anticoagulant 288
Study Design 288
Results of Drug Exposure 288
Statistical Analysis 289
Safety 290
Subgroup Analysis 290

Summary 290
ADVERSE EFFECTS AND MANAGEMENT 291
Bleeding Risk and Reversal Agents (Vitamin K) 291
Pharmacology of Aitamin K Antagonist Drugs 291
Mechanism of Action of Coumarin-based Oral Anticoagulant Drugs 291
Drug Interactions 291
Advances in Oral Anticoagulant Coumarin-based Therapy 292
Complications 293
PREVENTION MEASURES OF AF 293
Primary Prevention of Atrial Fibrillation 293
Secondary Atrial Fibrillation Prevention 294
MANAGEMENT OF AF 294
Stroke Risk Assessment 294
Bleeding Risk Assessment 295
Absolute Contraindications to Oral Anticoagulants 295
Active Bleeding on Anticoagulant Therapy: Management and Reversal Drugs 295
Patient-reported Outcomes 296
SPECIAL CONSIDERATION 297
Use in Elderly Patients 297
Atrial Fibrillation and Chronic Kidney Disease 297
Anticoagulation in Patients with Comorbidities 298
Atrial Fibrillation and Heart Failure 298
Atrial Fibrillation and Endocrine Disorders 298
Atrial Fibrillation and Gastrointestinal Disorders 298
Atrial Fibrillation and Hematological Disorders 299
Patients with Cognitive Impairment 299
FUTURE PERSPECTIVE 300
Role of Personalized Medicine in Stroke Prevention 300
Tools to Support Clinical Decision-making 301
Advances in Anticoagulation Therapy 301
EMERGING ALTERNATIVES TO ANTI-COAGULATION 302
Endovascular Treatment and Catheter-based Thrombectomy 302
Carotid Stenting 302
Patent Foramen Ovale Closure 303
Pulmonary Veins Isolation 303
Left Atrial Appendage Occlusion or Removal 303
CONCLUSION 304
CONSENT FOR PUBLICATION 304
ACKNOWLEDGEMENT 304
AI USE DISCLOSURE 305
REFERENCES 305

SUBJECT INDEX 309

FOREWORD

The rapid developments in modern medicine, driven by discoveries and technological advances, have revolutionized how we diagnose, treat, and prevent diseases. Yet, chronic and complex illnesses like cancer, diabetes, neurodegenerative diseases, and cardiovascular conditions remain significant global health challenges. This underscores the pressing need for safer, more effective therapies. Recently, there has been a renewed interest in bioactive compounds derived from natural sources, many of which have been used in traditional medicine for centuries. These compounds, with their broad pharmacological effects, have attracted scientific attention because of their potential to fill unmet medical needs. Progress in molecular biology and biotechnology has enhanced our understanding of how these compounds interact with biological systems, paving the way for new drug discovery and treatment options.

The book 'Therapeutic Potentials of Bioactive Compounds: A Holistic Approach' arrives at a pivotal moment as awareness of nature's pharmacological potential increases. It gathers leading experts from diverse fields to present the latest research on bioactive compounds, highlighting their therapeutic roles in current disease treatments. Topics covered include identifying bioactive molecules, understanding their mechanisms, clinical uses, and health benefits. This volume's interdisciplinary approach blends traditional knowledge with modern scientific standards, deepening our understanding of bioactive compounds and stressing the need to incorporate natural products into contemporary healthcare. As we encounter complex modern diseases, exploring innovative, safe, and sustainable therapies remains crucial. The book embodies a collective effort to advance research and develop new treatments, offering hope for better health outcomes globally.

Balaji Panchal
Hebei University of Engineering
Handan, Hebei, China 056038

PREFACE

In recent decades, the prevalence of chronic and lifestyle-related diseases has surged, posing significant challenges to healthcare systems worldwide. Traditional treatments, while effective in many cases, often come with limitations, including adverse side effects, high costs, and the development of drug resistance. As modern medicine seeks new and more efficient ways to combat these challenges, the focus on natural bioactive compounds has gained tremendous momentum. Bioactive compounds, derived from natural sources such as plants, have been used for centuries in Indian Ayurveda for their medicinal properties. With advancements in scientific research and technology, it is now possible to unravel the complex mechanisms through which these compounds exert their therapeutic effects. This emerging knowledge has opened new avenues for the treatment of a wide range of diseases, including cancer, cardiovascular disorders, diabetes, neurodegenerative conditions, and inflammatory diseases.

The purpose of this book is to provide a comprehensive overview of the therapeutic potentials of bioactive compounds in the context of modern disease treatment. By bringing together the latest research findings, we aim to highlight the role of bioactive compounds in drug discovery and development, offering alternative or complementary therapeutic options to existing treatments. The chapters cover various bioactive compounds, exploring their sources, mechanisms of action, pharmacological properties, and potential clinical applications. This book is intended for researchers, healthcare professionals, pharmaceutical experts, and students who are interested in the interface between natural products and modern medicine. It also seeks to inspire future research and development in this exciting field, promoting interdisciplinary collaboration to explore the untapped potential of nature's pharmacy.

We extend our gratitude to the contributors who have shared their invaluable knowledge and insights in this volume. Their dedication to exploring the therapeutic possibilities of bioactive compounds has enriched this field of study. We hope this book will serve as a valuable resource for advancing scientific research and improving healthcare outcomes for patients suffering from diseases.

Khanage Shantaram G.
Department of Pharmaceutical Chemistry
Rashtriya College of Pharmacy, Hatnur
Aurangabad, Maharashtra, India

Waghamare Suresh
Department of Pharmaceutical Science
NIMS Institute of Pharmacy, NIMS University
Jaipur, Rajasthan, India

&

Sayyad Jahed
Department of Pharmaceutics
Rashtriya College of Pharmacy
Hatnur, Aurangabad
Maharashtra, India

List of Contributors

Annasaheb Kudhekar	Shri Sai College of Pharmacy, Khandala, Maharashtra, India
Avinash S. Jiddewar	NSM College of Pharmacy, Darwha, Yavatmal, Maharashtra, India
Anurag Mishra	Department of Pharmaceutical Science, NIMS Institute of Pharmacy, NIMS University, Jaipur, Rajasthan, India
Atul A. Shirkhedkar	R.C Patel Institute of Pharmaceutical Education and Research, Karwand Naka, Shirpur, Dhule, Maharashtra, India
Bhagyashri Randhawan	Arihant College of Pharmacy, Ahilyanagar, Maharashtra, India
Chandrashekhar D. Khadse	NSM College of Pharmacy, Darwha, Yavatmal, Maharashtra, India
Darwade Amol	SND College of Pharmacy, Babhulgaon, Dist- Nashik, Maharashtra, India
Divya Negi	Faculty of Pharmaceutical Sciences, Amrapali University, Haldwani, Uttarakhand, India
Diksha M. Randive	Amrutvahini Institute of Pharmacy, Sangamner, Maharashtra, India
Garad Manoj	SND College of Pharmacy, Babhulgaon, Dist- Nashik, Maharashtra, India
Ganesh Godge	Department of Pharmaceutics, Dr Vithalrao Vikhe Patil Foundation's College of Pharmacy, Ahilyanagar, Maharashtra, India
Komal Shelar	Department of Pharmaceutical Chemistry, Sanjivani College of Pharmaceutical Education and Research, Savitribai Phule Pune University, Kopargaon, Ahmednagar, Maharashtra, India
Karuna Dhaundhiyal	Faculty of Pharmaceutical Sciences, Amrapali University, Haldwani, Uttarakhand, India
Kalyani Autade	Department of Pharmacology, RSM's N. N. Sattha College of Pharmacy, Ahmednagar, Maharashtra, India
Manish Raskar	Department of Pharmaceutical Chemistry, Dr Vithalrao Vikhe Patil Foundation's College of Pharmacy, Ahilyanagar, Maharashtra, India
Mohit Agarwal	Department of Pharmaceutical Science, NIMS Institute of Pharmacy, NIMS University, Jaipur, Rajasthan, India
Nandini P. Dolase	Government College of Pharmacy, Karad, Maharashtra, India
Poonam Kodag	Department of Pharmaceutics, Rashtriya College of Pharmacy, Hatnur, Aurangabad, Maharashtra, India
Poonam Patil	Department of Pharmaceutical Chemistry, Sanjivani College of Pharmaceutical Education and Research, Savitribai Phule Pune University, Kopargaon, Ahmednagar, Maharashtra, India
Pooja Paulbudhe	Oyster Institute of Pharmacy, Chh. Sambhaji Nagar, Maharashtra, India
R. B. Pandhare	Department of Pharmacology, MES's College of Pharmacy, Newasa, Maharashtra, India

Rajashri B. Sumbe	Department of Pharmacology, RSM's N. N. Sattha College of Pharmacy, Ahmednagar, Maharashtra, India
Sayyad Jahed	Department of Pharmaceutics, Rashtriya College of Pharmacy, Hatnur, Aurangabad, Maharashtra, India
Sunil Aher	Department of Pharmaceutical Chemistry, Sanjivani College of Pharmaceutical Education and Research, Savitribai Phule Pune University, Kopargaon, Ahmednagar, Maharashtra, India
Sarita Pawar	Department of Pharmaceutical Chemistry, Sanjivani College of Pharmaceutical Education and Research, Savitribai Phule Pune University, Kopargaon, Ahmednagar, Maharashtra, India
Seema Gosavi	Department of Pharmaceutical Chemistry, Sanjivani College of Pharmaceutical Education and Research, Savitribai Phule Pune University, Kopargaon, Ahmednagar, Maharashtra, India
Sarita Rawat	Faculty of Pharmaceutical Sciences, Amrapali University, Haldwani, Uttarakhand, India
Shivani S. Kale	SNJB's Shriman Sureshdada Jain College of Pharmacy, Neminagar, Chandwad, Nashik, Maharashtra, India
S. S. Dange	Ashvin College of Pharmacy, Sangamner, Maharashtra, India
Snehal R. Kadam	SNJB's Shriman Sureshdada Jain College of Pharmacy, Neminagar, Chandwad, Nashik, Maharashtra, India
Suresh Waghamare	Department of Pharmaceutical Science, NIMS Institute of Pharmacy, NIMS University, Jaipur, Rajasthan, India
Khanage Shantaram G.	Department of Pharmaceutics, Rashtriya College of Pharmacy, Hatnur, Aurangabad, Maharashtra, India
Sofiya Mujawar	Government College of Pharmacy, Karad, Maharashtra, India
Umesh Panchal	Department of Pharmaceutics, Dr Vithalrao Vikhe Patil Foundation's College of Pharmacy, Ahilyanagar, Maharashtra, India
Varsharani Avhad	Arihant College of Pharmacy, Ahilyanagar, Maharashtra, India
Vishal V. Pande	Department of Pharmacology, RSM's N. N. Sattha College of Pharmacy, Ahmednagar, Maharashtra, India
Waghamare Suresh	Department of Pharmaceutical Science, NIMS Institute of Pharmacy, NIMS University, Jaipur, Rajasthan, India

CHAPTER 1

Advanced Alkaloid Delivery Systems for Effective Management of Parkinson's Symptoms

Annasaheb Kudhekar[1,*], Waghamare Suresh[2], Poonam Kodag[3], Sayyad Jahed[3], Darwade Amol[4] and **Garad Manoj[4]**

[1] *Shri Sai College of Pharmacy, Khandala, Maharashtra, India*

[2] *Department of Pharmaceutical Science, NIMS Institute of Pharmacy, NIMS University, Jaipur, Rajasthan, India*

[3] *Department of Pharmaceutics, Rashtriya College of Pharmacy, Hatnur, Aurangabad, Maharashtra, India*

[4] *SND College of Pharmacy, Babhulgaon, Dist- Nashik, Maharashtra, India*

Abstract: Parkinson's Disease (PD) poses a major global health challenge, particularly among the elderly population. According to the World Health Organization, approximately 8.5 million people worldwide were living with PD in 2019. In the United States alone, nearly one million individuals are affected, with about 90,000 new cases diagnosed each year. PD is a progressive neurodegenerative movement disorder characterized by the gradual loss of neurons in specific regions of the brain, leading to symptoms such as tremors, rigidity, impaired movement, and postural instability. This chapter focuses on the development of advanced alkaloid-based drug delivery systems for the management of Parkinson's disease. These approaches utilize modern technologies to enhance the therapeutic effectiveness, bioavailability, and targeted delivery of alkaloids employed in PD treatment. Alkaloids, including apomorphine and other natural or synthetic compounds, are essential in compensating for the dopaminergic deficiency associated with the disease. However, conventional treatment strategies suffer from several drawbacks. Emphasis is therefore placed on the physicochemical properties, mechanisms of action, and drug release profiles of these agents, as well as innovative delivery methods designed to bypass the blood–brain barrier. The chapter highlights emerging strategies such as nanoparticle-mediated delivery, microneedle-based transdermal systems, intranasal administration, hydrogel platforms, brain-targeted carriers, prodrug approaches, liposomal formulations, and implantable drug delivery devices.

Keywords: Alkaloids, Brain, Drug delivery, Nanoparticle, Parkinson's disease.

[*] **Corresponding author Annasaheb Kudhekar:** Shri Sai College of Pharmacy, Khandala, Maharashtra, India; E-mail: annasahebkudhekar125@gmail.com

Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed (Eds.)
All rights reserved-© 2026 Bentham Science Publishers

INTRODUCTION

Parkinson's Disease (PD) is a progressive disorder of the nervous system that mainly affects the ability to control movement. It is characterized by symptoms such as muscle stiffness, slowed movements (bradykinesia), and impaired balance. In addition to these motor difficulties, individuals with PD often experience non-motor symptoms, including depression, cognitive impairment, and sleep-related problems. Although the exact cause of Parkinson's Diseaseremains unknown, it is thought to develop due to a combination of genetic and environmental influences. While no cure currently exists, various treatments—such as medications like levodopa, physical therapy, and surgical procedures including deep brain stimulation—can help reduce symptoms and improve overall quality of life [1]. Parkinson's Disease (PD) is a long-term, progressive neurological disorder marked by the gradual degeneration of dopamine-producing neurons, particularly within the substantia nigra, a brain area responsible for movement regulation. Dopamine is crucial for coordinated and smooth muscle activity, and its decline leads to characteristic motor symptoms such as tremors, bradykinesia (slowed movement), muscle rigidity, and postural instability. In addition to dopamine loss, PD is associated with the buildup of abnormal protein deposits called Lewy bodies, which contribute to neuronal dysfunction. Damage to dopaminergic neurons disrupts the basal ganglia circuitry involved in movement control, resulting in both motor impairments and non-motor symptoms, including cognitive impairment, sleep disorders, and mood disturbances [2].

Current Treatment Options

Parkinson's Diseasepresents with both motor and non-motor symptoms. Motor symptoms include involuntary shaking, slowed movement (bradykinesia), muscle stiffness, and difficulties with balance and posture. These impairments can interfere with everyday tasks such as walking, getting dressed, and speaking. Non-motor symptoms, which can be just as disruptive, involve cognitive changes like memory problems or dementia, emotional conditions such as depression and anxiety, sleep disorders, and autonomic system dysfunction. Autonomic issues may cause symptoms including constipation, low blood pressure, and urinary problems. Notably, non-motor symptoms can have a major impact on quality of life and may appear before motor symptoms develop. Although Parkinson's Diseasehas no cure, various treatments aim to reduce symptoms and enhance daily functioning. The primary medication is levodopa, which is converted into dopamine in the brain to compensate for reduced dopamine levels. It is commonly administered with carbidopa to prevent its premature breakdown before reaching the brain. Additional medications, including dopamine agonists, MAO-B

inhibitors, and COMT inhibitors, help manage symptoms by imitating dopamine's effects or slowing its degradation. In later stages of the disease, or when medications lose effectiveness, surgical interventions such as Deep-Brain Stimulation (DBS) may be recommended. DBS involves placing electrodes in targeted brain regions to help control abnormal neural activity. Alongside DBS, supportive approaches such as physical, speech, and occupational therapy play an important role in addressing both motor and non-motor symptoms, with multiple treatment strategies used to comprehensively manage Parkinson's disease (Table **1**) [3].

Table 1. Comprehensive treatment approaches for parkinson's disease [4 - 7].

Treatment Type	Description	Examples/Medications
Medication (Dopaminergic Agents)	Aim to increase dopamine levels or mimic dopamine function.	Levodopa (Sinemet, Parcopa)
		Dopamine agonists (Pramipexole, Ropinirole)
		MAO-B inhibitors (Selegiline, Rasagiline)
		COMT inhibitors (Entacapone, Tolcapone)
		Amantadine
Deep Brain Stimulation (DBS)	Surgical intervention involves implanting electrodes in the brain to stimulate areas affected by Parkinson's disease.	No specific medication (surgical intervention)
Physical Therapy	Focuses on improving mobility and balance, and reducing falls.	Exercise, gait training, stretching
Occupational Therapy	Helps patients maintain independence with daily activities.	Adaptive equipment, strategies for dressing and eating
Speech Therapy	Aims to improve speech, swallowing, and communication.	Voice exercises, swallowing techniques
Surgical Interventions	For advanced cases or when medications are not effective.	Pallidotomy, Thalamotomy, DBS
Gene Therapy	An experimental approach to deliver genes that may produce dopamine.	Ongoing clinical trials
Stem Cell Therapy	An experimental therapy that focuses on regenerating dopamine-producing cells.	Ongoing clinical trials
Dietary Management	Focuses on managing symptoms and improving overall health.	High-fiber diet, low-protein meals, hydration

CHAPTER 2

Formulations of Glycosides for Cardiac Diseases in Heart Failure Management

Chandrashekhar D. Khadse[1,*] and **Avinash S. Jiddewar**[1]

[1] *NSM College of Pharmacy, Darwha, Yavatmal, Maharashtra, India*

Abstract: Cardiac glycosides are medications used to help people with certain heart conditions. They are a class of organic compounds that increase the heart's output force and speed up contractions by acting on the cellular sodium-potassium ATPase pump. These selective steroidal glycosides are important drugs for treating heart failure and rhythm disorders. Digitalis and its derivatives, such as digoxin, are commonly used in the treatment of congestive heart failure and atrial fibrillation. Other cardiac glycosides include oleandrin, bufalin, and ouabain. Digoxin is the most frequently prescribed cardiac glycoside. The basic structure of cardiac glycosides consists of a sterol nucleus and an unsaturated conjugated lactone ring attached at the C17 position, formed by condensing a hydroxyl group at C3 of the nucleus with a sugar. Recently, significant advances have been made in understanding the effectiveness and safety of herbal medicines for patients with cardiovascular diseases (CVDs). Systematic reviews provide strong evidence for their clinical use. This special issue presents updated knowledge on partial herbal medicines for CVDs, involving complex, gene-modifiable mechanisms, and emphasizes an evidence-based approach. The role of herbal drugs in treating CVDs still requires more scientific and clinical data to confirm their efficacy and safety. This issue aims to summarize current progress on promising herbal medicines and their extracts for formulations addressing various CVDs.

Keywords: Angina, Arrhythmia, Atrial fibrillation, Bufadienolides, Bufalin, Cardiovascular diseases, Cardenolides, Cardiac disease, Congestive heart failure, Digitalis, Digitoxin, Heart failure, Oleandrin, Ouabain, Sodium-Potassium ATPase.

INTRODUCTION

Cardiac glycosides are the natural compounds that boost the output force of the heart and help to increase the rate of contractions of the heart by acting on a cellular sodium-potassium ATPase pump. These glycosides are steroidal and are used significantly for the management of heart failure and cardiac rhythm

[*] **Corresponding author Chandrashekhar D. Khadse:** NSM College of Pharmacy, Darwha, Yavatmal, Maharashtra, India; E-mail: khadsecd@gmail.com

Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed (Eds.)
All rights reserved-© 2026 Bentham Science Publishers

disorders. Plants containing cardiac glycosides have a long history in the treatment of cardiac diseases. A group of chemical compounds obtained from foxglove plants is generally known as digitalis. The use of cardiac glycosides from Digitalis purpurea in the management of heart conditions was first reported in the English-speaking medical literature in 1785 [1], marking the beginning of modern therapeutics. Cardiac glycosides have also been isolated from the skin and toads' carotid gland [2, 3]. Structurally, cardiac glycosides are composed of a steroidal ring and a conjugated unsaturated lactone ring attached to the C17 position and a sugar moiety attached at the C3 position, which is shown in Fig. (**1**). Mostly, the common monosaccharides like glucose, 6-deoxy monosaccharide glucose, fructose, glucuronide, rutinose, and rhamnose are found in cardiac glycosides. These are shown in Fig. (**2**). Cardiac glycosides are patient medications received for heart failure, atrial fibrillation, and atrial flutter. It shows toxicity if taken as an overdose; therefore, cardiac glycosides are not a first-line treatment for these conditions. Most preferably, these medications are given if other heart medicines do not work, but at low doses to ensure patient safety. Recent research has also shown that cardiac glycosides exhibit promising anticancer activity. Some cardiac glycosides, such as digitoxin, digoxin, and bufalin (Fig. **3**), inhibit cancer cell proliferation and tumor growth by inducing immunogenic cell death [4]. Cardiovascular disease is the leading cause of death worldwide. Almost half of adults in the U.S. have some form of cardiovascular disease. The number of plant glycoside constituents has shown activity to treat cardiac diseases in the management of heart failure, which are shown in Table **1**. However, due to their toxicity in higher doses, these glycosides have been used in second-line treatment [5].

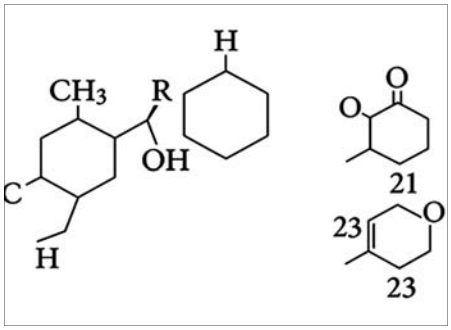


Fig. (1). Structure of cardiac Glycosides.

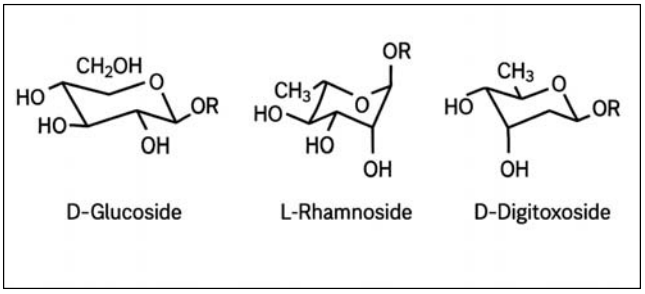


Fig. (2). Example of some residue attached to Cardiac Glycoside.

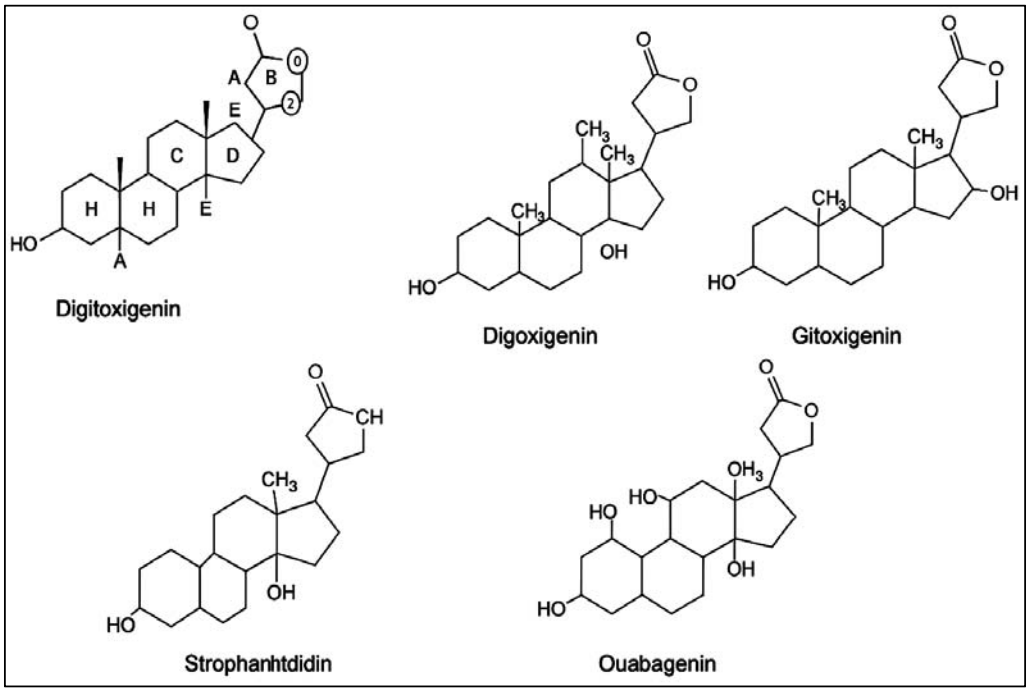


Fig. (3). Structure of some common cardiac Glycosides.

Table 1. Some common herbs contain cardiac glycosides.

Sr. No.	Common Name	Scientific Name	Family	Chemical Constituents	Part Use	Ref.
01	Foxglove	*Digitalis purpurea*	Scrophulariaceae	Purpurea glycoside A & B, glycogitaloxin, digitoxin, digoxin	Leaves	[6, 7]
02	Adonis fulgens	*Adonis aleppica*	Ranunculaceae	Alepposide A, B, C, D	Aerial Part	[8]

CHAPTER 3

Therapeutic Application of Glycoside-Based Formulations in Gastrointestinal and Metabolic Disorders

Ganesh Godge[1,*], Umesh Panchal[1], Bhagyashri Randhawan[2], Manish Raskar[3] and **Varsharani Avhad[2]**

[1] *Department of Pharmaceutics, Dr Vithalrao Vikhe Patil Foundation's College of Pharmacy, Ahilyanagar, Maharashtra, India*

[2] *Arihant College of Pharmacy, Ahilyanagar, Maharashtra, India*

[3] *Department of Pharmaceutical Chemistry, Dr Vithalrao Vikhe Patil Foundation's College of Pharmacy, Ahilyanagar, Maharashtra, India*

Abstract: Glycoside-based formulations are becoming more popular due to their wide range of therapeutic uses, especially in gastrointestinal and metabolic diseases. Glycosides are naturally occurring substances with special pharmacological characteristics, such as immunomodulatory, antioxidant, and anti-inflammatory actions. They are composed of a sugar moiety linked to a non-sugar aglycone. Interestingly, glycosides such as iridoid glycosides, flavonoid glycosides, and saponins have demonstrated potential in the treatment of gastrointestinal disorders such as peptic ulcers, irritable bowel syndrome (IBS), and inflammatory bowel disease (IBD). These substances address microbial dysbiosis while maintaining beneficial flora by modulating gut microbiota, reducing intestinal inflammation, promoting mucosal healing, and strengthening the gut barrier. Glycosides help control obesity, diabetes, and dyslipidemia in metabolic disorders. For instance, triterpenoid glycosides reduce oxidative stress and fat buildup, whereas anthocyanin and cardiac glycosides improve insulin sensitivity and control important metabolic pathways like AMP-activated protein kinase (AMPK). Furthermore, glycosides—like steviol glycosides—act as organic, low-calorie sweeteners that support weight management and blood sugar regulation. Despite their potential, their practical applicability is limited by issues such as low absorption, metabolic instability, and toxicity at larger doses. To improve their pharmacokinetic and pharmacodynamic properties, advanced formulation techniques such as encapsulation, chemical modification, and nanotechnology are being investigated. Standardized formulations, optimal doses, and long-term safety evaluations are necessary to fully realize the therapeutic promise of glycoside-based therapies, despite preclinical and clinical research showing encouraging results.

[*] **Corresponding author Ganesh Godge:** Department of Pharmaceutics, Dr Vithalrao Vikhe Patil Foundation's College of Pharmacy, Ahilyanagar, Maharashtra, India; E-mail: grgodge@yahoo.com

Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed (Eds.)
All rights reserved-© 2026 Bentham Science Publishers

Keywords: Bioavailability, Diabetes, Dyslipidaemia, Flavonoids, Gut microbiota, Glycosides, Gastrointestinal, Inflammation, Metabolic disorders, Obesity, Saponins.

INTRODUCTION

Because of the complex interplay between the glycone and the aglycone, glycosides exhibit unique physicochemical properties that enable them to interact with biological systems. These compounds release one or more sugar moieties along with the glycone upon enzymatic or acid hydrolysis, offering a variety of medicinal opportunities. The complicated interaction between the glycone and aglycone is further highlighted by the glycosidic linkages, such as S-, N-, C-, and O-glycosidic bonds, which affect the molecules' stability and biological activity [1, 2]. The therapeutic potential of glycosides derived from medicinal plants has been the focus of recent studies. These substances have shown impressive health benefits across a range of illnesses, including anti-inflammatory, cardiovascular, and neurological effects, as well as antibacterial and anticancer properties. They work by interacting with particular biomolecules, altering vital cellular signaling pathways, and controlling a range of physiological functions. Moreover, the complexity of glycosides' therapeutic applications is increased by their structural diversity and potential synergistic interactions with other bioactive components in medicinal plants [3]. Innovative treatment approaches are made possible by the complex interaction between glycosides and cellular processes, which emphasizes their significance in contemporary pharmacological research. A deeper understanding of the chemical and biological aspects of glycoside-based treatments could lead to the development of long-lasting, effective treatments for a range of medical conditions. Glycosides are structurally diverse substances made up of an aglycone and a sugar moiety [4]. They are useful for treating a range of illnesses, including metabolic and gastrointestinal issues, due to their pharmacological characteristics. Global health is greatly impacted by diseases like diabetes, obesity, irritable bowel syndrome (IBS), and stomach ulcers. By modifying gut flora, altering metabolic pathways, and providing protection against oxidative stress, glycoside-based formulations offer intriguing alternatives. Plants, fungi, and microbes produce glycosides, which are secondary metabolites that are vital to defense systems and physiological functions. Their biological activity is determined by their aglycone component, which is used to classify them. While the aglycone specifies specific interactions with biological targets, the sugar moiety improves solubility and transport inside the organism [5].

Overview of Glycosides

A glucose molecule joined to an aglycone forms a glycoside. Glycosides are referred to as glycone, aglycone, and the particular kind of glycosidic bond. One or more sugar moieties and non-sugar components are released when the glycosidic link in glycosides is hydrolyzed by enzyme action or acidic circumstances. The sugar component is called glycone, and the non-sugar component is called aglycone or genin. The structure of the glycoside, which gives these molecules a wide range of properties and biological roles, is determined by the interaction of an aglycone with the anomeric carbon (C-1) of a glycone. The primary structural component of glycosides, the glycosidic link, can be broken down by enzymes, usually through acid hydrolysis or β-glucosidases. Glycosyl transferases, which attach sugar moieties to particular places on the aglycone to change secondary metabolites, control the formation of glycosides in plants. Sometimes, additional metabolic activities like oxidation, acylation, or degradation are required during the production of glycosides to guarantee the proper chemical structure and pharmacological characteristics [4]. The glycone moiety of glycosides is responsible for their water solubility, which increases their bioavailability and makes it easier for them to interact with biological systems. The glycone is usually made up of monosaccharide units, mostly glucose, it can also contain di-, tri-, or tetrasaccharides. Because glucose is a common component of glycone, those glycosides made with glucose are referred to as glycosides. Additionally, glycosides can be classified according to the particular glycone unit they contain, such as glucuronic acid (which yields glucuronides), fructose (which yields fructosides), arabinose (which yields arabinosides), and rhamnose (which yields rhamnosides). The structural diversity of glycosides is highlighted by this classification, which is based on the number of saccharide units in the glycone and results in names like monodesmosides or monosides (one sugar unit), bidesmosides or biosides (two sugar units), and tridesmosides or triosides (three sugar units) [5, 6].

Classification of Glycosides

The specific sugar moiety and the properties of the aglycone are the main factors that determine glycoside classification. O-glycosides, N-glycosides, and S-glycosides are the three main subtypes of glycosides according to a commonly accepted taxonomy. O-glycosides have a glycosidic connection between the aglycone and an oxygen atom and are frequently present in plant secondary metabolites such as flavonoids, anthraquinones, and cardiac glycosides. On the other hand, N-glycosides, which form a glycosidic bond with the aglycone through a nitrogen atom, are typically found in alkaloids like morphine and quinine. Even though they are less common, S-glycosides participate in

CHAPTER 4

Utilizing Phenolic Compounds to Mitigate Oxidative Stress in Type 2 Diabetes Management

Poonam Patil[1,*], Sunil Aher[1], Sarita Pawar[1], Seema Gosavi[1] and **Komal Shelar[1]**

[1] Department of Pharmaceutical Chemistry, Sanjivani College of Pharmaceutical Education and Research, Savitribai Phule Pune University, Kopargaon, Ahmednagar, Maharashtra, India

Abstract: Precision medicine and lifestyle interventions are becoming increasingly important in the evolving landscape of diabetes care. Dietary polyphenols, bioactive compounds found in plants that help defend against oxidative stress, are gaining popularity as natural alternatives for the treatment of diabetes mellitus and its complications. Unlike conventional drugs that often come with drawbacks such as adverse effects and drug resistance, polyphenols offer a safer and more versatile solution. Some of the ways phenolic acids (such as chlorogenic acid), flavonoids (such as quercetin), proanthocyanidins (such as procyanidin B2), and gallotannins help regulate blood sugar include improved insulin sensitivity, accelerated secretion, and enzyme inhibition. These compounds also play a role in modifying the gut microbiome, scavenging free radicals, and reducing chronic inflammation. Due to their anti-inflammatory properties, polyphenols may help treat cardiovascular disorders, in addition to diabetes. This review summarizes recent advances in elucidating the multifunctional roles of phenolic compounds, including their anti-inflammatory effects and the molecular pathways through which they modulate metabolic disorders. It also discusses the chemical and biological contributions to managing cardiovascular disease, current challenges, and prospects for therapeutic applications, providing a natural approach to enhance patient outcomes.

Keywords: Anti-inflammatory, Blood sugar levels, Cardiovascular disorder, Chlorogenic acid, Diabetes mellitus, Dietary polyphenols, Enzymes, Flavonoids, Gallotannins, Gut microbiome.

INTRODUCTION

The worldwide incidence of diabetes mellitus (DM) has risen significantly over recent decades, making it a significant global public health concern [1]. Hyper-

[*] **Corresponding author Patil Poonam:** Department of Pharmaceutical Chemistry, Sanjivani College of Pharmaceutical Education and Research, Savitribai Phule Pune University, Kopargaon, Ahmednagar, Maharashtra, India; E-mail: poonamprakashpatil4174@gmail.com

Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed (Eds.)
All rights reserved-© 2026 Bentham Science Publishers

glycemia, a physiologically abnormal condition characterized by prolonged high blood sugar levels, is a hallmark of DM, a complex metabolic disease [2]. Insulin is an essential anabolic hormone because it stimulates the abnormal metabolism of triglycerides and carbohydrates. Several metabolic problems arise from issues with insulin receptors, the signaling system, or effector enzymes. Target tissues primarily skeletal muscles and adipose tissue, and to a lesser extent, the liver develop resistance to insulin and/or fail to respond adequately, resulting in an inefficient response [3]. Diabetes is classified into four categories: gestational, type I, type II, and other distinct forms [4]. The most common autoimmune disorder in young people is type 1 diabetes, caused by the destruction of pancreatic endocrine β-cells, which produce insulin in some areas of the pancreas called the islets of Langerhans. This destruction leads to hyperglycemia and insulin deficiency [5]. Over 90% of diabetes cases are type II, making it the most prevalent form of the disease. Its key feature is insulin shortage due to pancreatic β-cell dysfunction and decreased responsiveness in target organs [6]. Pregnant women, even those with pre-existing diabetes, may develop new high blood sugar levels, a primary feature of gestational diabetes mellitus (GDM), a metabolic condition that generally resolves shortly after delivery [7]. The pathophysiology of both type I and type II diabetes involves impaired pancreatic β-cells, though through different mechanisms. In type II, the progressive loss of β-cell mass and function is mainly due to metabolic stress, including inflammation, endoplasmic reticulum stress, mitochondrial damage, and excessive production of reactive oxygen species (ROS). In contrast, in type I, β-cell failure is primarily immune-mediated [8].

Diabetes brings about enormous stress on both individuals and medical facilities throughout the globe, as the number of people diagnosed continues to climb without pause. Diabetes may adversely affect almost every organ of the body, including the kidneys, the nervous system, and the eyes [9]. Diabetes affects a considerable portion of the human population worldwide, causing a significant threat to global wellness due to its widespread mortality and morbidity [10]. Research conducted by Saeedi *et al*. (2019) to assess the prevalence of diabetes worldwide found that 463 million people aged 20-79 were diagnosed with the disease. As per their investigation and knowledge, this number may increase to 578 million by 2030 and 700 million by 2045, especially in Southeast and Western regions. This report highlights the need to take preventive measures to reduce the prevalence of diabetes [11]. Novel solutions to diabetes management and treatment are necessary due to its global prevalence. The mainstays of the current diabetes care strategy include pharmaceutical treatments, dietary adjustments, and lifestyle modifications. Despite their varied degrees of benefit, these strategies usually fail to prevent or lessen the problems linked to long-term diabetes. Despite the ease of access to synthetic antidiabetic drugs, the

development of novel and practical antidiabetic drugs is imperative because of the rise in drug resistance and the adverse effects of long-term use of these medications. Due to their demonstrated ability to boost insulin secretion and control blood sugar, dietary phenolic compounds have generated considerable interest of recent decades regarding their potential applications in the treatment of diabetes [12]. In this way, natural products, particularly phenolic compounds, can be a valuable source of bioactive compounds with anti-diabetic properties, especially for the prevention and treatment of type II diabetes [13]. This review covers current developments in understanding the multipurpose activities of phenolic substances, especially their anti-inflammatory properties, and the molecular mechanisms by which they can exert anti-inflammatory effects in metabolic illnesses. The chemical and biological contributions to managing cardiovascular disease have been described, presenting hurdles and prospective therapeutic opportunities, offering a natural route to improve patient outcomes.

A SUMMARY OF PHENOLIC SUBSTANCES

Medicinal herbs have been used for centuries to treat a variety of disorders, notably diabetes. Recent studies have shown that natural products exhibit anti-diabetic properties due to their bioactive components [13]. Phenols are a family of phytochemicals that include phenol rings. They are further divided into groups according to their chemical structures, biological roles, and places of origin. A class of secondary metabolites, or organic substances produced by plants, known as dietary phenols (DPs), is crucial to the plant's defensive systems, but not necessary for its regular growth. DPs are frequently present in wine, tea, chocolate, other cocoa products, fruits, and vegetables. Flavones, isoflavones, flavonols, catechins, and other phenolic acids like caffeic, chromogenic, and ferulic acid are certain main constituents of polyphenols [14]. Crucially, the structure of polyphenols and the metabolic changes they undergo during digestion and absorption dictate their bioavailability and interactions with cells and biomolecules in vivo, which, in turn, determine their bio-efficacy [15].

The most prevalent antioxidants in food, polyphenols, have attracted much attention due to their potential pharmacological uses. They are thought to improve health and reduce the likelihood of non-communicable conditions, including type 2 diabetes [16]. Vázquez-Ruiz *et al*. [17] investigated the relationship between dietary phenolic compounds and the occurrence of Type II Diabetes Mellitus (TIIDM) in the 'Seguimiento Universidad de Navarra' (SUN) cohort. Following 17,821 participants for 10 years, their study found that reduced stilbene intake was strongly associated with a higher prevalence of TIIDM, especially among those over 50. These results demonstrate the critical function of bioactive food ingredients in controlling metabolism and preventing diabetes. Getting sufficient

CHAPTER 5

Saponins as Natural Cholesterol-Lowering Agents for Reducing the Risk of Cardiovascular Disease Development

Anurag Mishra[1,*], Sarita Rawat[2], Karuna Dhaundhiyal[2], Mohit Agarwal[1] and **Divya Negi[2]**

[1] *Department of Pharmaceutical Science, NIMS Institute of Pharmacy, NIMS University, Jaipur, Rajasthan, India*

[2] *Faculty of Pharmaceutical Sciences, Amrapali University, Haldwani, Uttarakhand, India*

Abstract: Saponins are a varied class of naturally occurring plant-based glycosides that have attracted a lot of scientific attention due to their cholesterol-lowering properties and the ability to minimize the risk of heart disease. These bioactive compounds form insoluble complexes with bile acids, increasing their excretion and reducing cholesterol reabsorption in the gastrointestinal tract. Saponins also possess anti-inflammatory and antioxidant properties, which regulate lipid metabolism, combat atherosclerosis, improve cardiovascular health, and lower low-density lipoprotein (LDL) cholesterol levels. This chapter provides a detailed discussion of the molecular mechanisms, natural sources, and structural diversity of saponins that contribute to their cholesterol-lowering effects. Saponins are highlighted, with a focus on food sources such as legumes, oats, and medicinal plants, as well as the potential of synthetic and semisynthetic derivatives for improved therapeutic applications. The chapter also discusses the pharmacokinetics and bioavailability of saponins, emphasizing challenges with their absorption and metabolism that may limit their therapeutic use. The use of saponins in combination therapies, functional foods, and nutraceuticals has been thoroughly studied, and data from human and animal studies confirm their hypocholesterolemic benefits. Despite current challenges, advances in formulation technology and ongoing research are enabling saponins to play a greater role in treating dyslipidemia and preventing CVD. This chapter provides a comprehensive overview of saponins, natural cholesterol-lowering agents, and their therapeutic potential for promoting cardiovascular health. It also outlines potential directions for future research and application.

[*] **Corresponding author Anurag Mishra:** Department of Pharmaceutical Science, NIMS Institute of Pharmacy, NIMS University, Jaipur, Rajasthan, India; E-mail: anurag.mishra@nimsuniversity.org

Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed (Eds.)
All rights reserved-© 2026 Bentham Science Publishers

Keywords: Atherosclerosis, Bioavailability, Cardiovascular disease (CVD), Cardioprotective, Cholesterol, Hypertension, Hypercholesterolemia, High-density lipoprotein, Resveratrol, Saponins, Therapeutic potential.

INTRODUCTION TO CARDIOVASCULAR DISEASES (CVDS)

According to estimates from the World Health Organization, 17.9 million people die from cardiovascular disease (CVD) every year. Heart failure (HF), myocardial infarction (MI), cardiac ischemia (CI), and cardiac hypertrophy (CH) are all linked to cardiovascular disease (CVD). Since the incidence of CVD-related challenges and death is increasing, more effective treatments are required [1]. Patients with coronary artery disease experience narrowing of their arteries due to thrombus production or atheromatous plaques. This causes morbidity by blocking the myocardium's blood supply. The most prevalent kind of coronary artery disease is called atherosclerosis. A common sign of ischemia, or insufficient oxygen and blood supply to the heart's tissues, is angina. Myocardial infarction is caused by chronic ischemia, which causes irreversible damage to the myocardium (necrosis) [2]. Lifestyle decisions have a significant impact on the onset or prevention of ischemic heart disease. By lowering certain variables, including nutrition, alcohol use, smoking, sedentary lifestyle, obesity, and stress, the risk of this illness can be reduced. Other variables include diabetes, sex, and siblings with a history of the disease. Once myocardial damage is severe, the heart's ability to contract can become compromised. Depending on where and how large the affected region is, the impact will vary. The term “heart failure,” which may have developed from this, may apply to many cardiac dysfunctions that lead to insufficient tissue perfusion. Damage to the heart's conduction system can result in arrhythmias, or irregular heartbeats [3].

The heart and blood vessels comprise the cardiovascular system. Abnormalities in the conduction system, rheumatic heart disease, endocarditis, and circulatory disease (CVD) are only a few of the numerous problems that can arise in the circulatory system. Angina, myocardial infarction (MI), and/or heart failure may all arise from coronary artery disease (CAD), occasionally referred to as coronary heart disease (CHD), which can be triggered by reduced myocardial perfusion (4). Approximately one-third to half of CVD cases are driven by it. Transient ischemic episodes (TIA) and stroke are both considered to be types of cerebrovascular disease (CVD). Aortic atherosclerosis, thoracic and abdominal aneurysms, and other limb-specific vascular diseases that can cause claudication are examples of peripheral artery disease (PAD), with various risk factors and underlying pathophysiological mechanisms summarized in Table **1** [4].

Table 1. CVD susceptibility and the pathophysiological processes behind its onset and progression with risk factors.

CVD	Risk Factor	Pathological Mechanism	
		Endothelial Dysfunction	**VSMC Alterations (Vascular Smooth Muscle Cell)**
Hypertension Endocrinopathies Stroke Heart failure Coronary Artery Disease Cardiomyopathies Myocardial Infraction Peripheral Vascular Disease	Smoking Unhealthy diet Diabetes mellitus Aging Hyperlipidaemia Obesity Hypertension	Narrowing of the arterial lumen Impaired vasomotion Platelet activation Thrombosis Inflammation Metabolic abnormalities Decreased vascular permeability Vascular Fibrosis Impaired vascular tone	Mitochondrial dysfunction Metabolic abnormalities EMC remodelling Inflammation VSMC proliferation VSMC stiffness VSMC migration

Etiology of Cardiovascular Diseases

As atherosclerosis is a prevalent component of the pathophysiology of CVD, it is imperative to address the risk variables related to its development. Many different etiologies may directly lead to CVD, such as emboli among individuals with atrial fibrillation, resulting in ischemic stroke, or rheumatic fever that leads to valvular heart disease. The inter heart investigation, which had participants from 52 nations, including those with rich, middle, and poor incomes, discovered nine modifiable risk factors, including smoking, dyslipidemia, hypertension, obese abdomens, psychological variables, consuming fruits and vegetables, regular intake of alcohol, and lack of physical activity, together accounted for 90% of the risk of experiencing a first MI [4]. To lower the risk of CVD, the American Heart Association has incorporated these outcomes into health awareness initiatives, emphasizing seven recommendations: abstaining from smoking, maintaining a balanced diet, exercising, and keeping blood pressure, body weight, glucose, and cholesterol levels within normal ranges. Additionally, microalbuminuria, a history of mediastinal or chest wall radiation, HIV (human immunodeficiency virus), and higher inflammatory indicators have been linked to a higher incidence and risk of cardiovascular disease [5].

Global Burden of CVDs: Epidemiology and Risk Factors

Between 2000 and 2010, CAD claimed 56 million lives worldwide. Inpatient cardiovascular treatments increased by 28%, rising from about 6 million to 7.5 million. The World Bank and WHO launched the Global Burden of Disease

CHAPTER 6

Tannins' Role in Elevating Symptoms of Irritable Bowel Syndrome through Gut Health Improvement

Kalyani Autade[1,*], R. B Pandhare[2], Snehal R. Kadam[3], Shivani S. Kale[3], Rajashri B. Sumbe[1] and **S. S. Dange[4]**

[1] *Department of Pharmacology, RSM's N. N. Sattha College of Pharmacy, Ahmednagar, Maharashtra, India*

[2] *Department of Pharmacology, MES's College of Pharmacy, Newasa, Maharashtra, India*

[3] *SNJB's Shriman Sureshdada Jain College of Pharmacy, Neminagar, Chandwad, Nashik, Maharashtra, India*

[4] *Ashvin College of Pharmacy, Sangamner, Maharashtra, India*

Abstract: Tannins are a class of polyphenol compounds that have been studied to modulate symptoms of irritable bowel syndrome and gut health regulation. This chapter defines the molecular processes underlying tannins' influence on the pathophysiology of IBS and on gut health. Tannins modulate gut microbiota composition by selectively promoting the proliferation of pathobionts, including *Escherichia* and *Clostridium* species, while concomitantly suppressing the growth of beneficial microorganisms, such as bifidobacteria and *Lactobacillus*. This tannin-induced dysbiosis disrupts the homeostatic production of short-chain fatty acids (SCFAs), including acetate, propionate, and butyrate, which are essential for maintaining the integrity and function of the intestinal epithelial barrier. The resulting impairment of gut epithelial barrier function triggers a pro-inflammatory response, characterized by the upregulation of pro-inflammatory cytokines and activation of immune cells, ultimately leading to gut inflammation and potentially contributing to the development of gastrointestinal disorders. Tannins modulate the expression of genes governing inflammation, secretion, and gut motility, inducing pro-inflammatory cytokines (TNF-α, IL-1β) and downregulating the serotonin transporter (SERT). Activation of NF-κB, MAPK, and PI3K/Akt signalling pathways exacerbates IBS symptoms and gut inflammation. Elucidating these molecular mechanisms may inform novel therapeutic strategies for IBS management, targeting specific pathways, modulating gut microbiota, and tailoring personalized medications.

Keywords: Gut microbiota, Gut health, Irritable bowel syndrome, Molecular mechanisms, Tannins.

[*] **Corresponding author Kalyani Autade:** Department of Pharmacology, RSM's N. N. Sattha College of Pharmacy, Ahmednagar, Maharashtra, India; E-mail: kalyanirajdev@gmail.com

Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed (Eds.)
All rights reserved-© 2026 Bentham Science Publishers

INTRODUCTION

Some of the intestinal conditions that are generally diagnosed include irritable bowel syndrome (IBS). When no other diseases are identified, IBS can be defined through altered bowel habits and abdominal pain or discomfort. Indicators of this condition include indigestion, loss of stool, constipation, or a combination of these, rectal mucus, and changes in the structure or appearance of stools. Since multiple factors contribute to its development, the leading cause of the illness is often unclear. IBS cannot be proven by a single test; however, the Rome criteria, which include specific symptoms, are used to diagnose IBS [1]. In the overall population, the risk of IBS ranges from 9% to 22% in the USA and the European Union. Southeast Asia's health and ecological conditions are evolving due to the continent's rapid socioeconomic growth over the past twenty years. It appears that IBS is becoming more common in Asian communities. Based on the Rome II criteria, the incidence rates in Singapore (8.6%) and Japan (9.8%) are comparable to those in Australia (6.9%) and the continent (9.6%), although lower than in Canada and the UK (12%). According to other criteria, such as the Manning or Rome criteria, the prevalence of IBS varies across communities and even within outpatient clinics [2]. It has often been found to be more severe in IBS patients compared to organic diseases, as IBS significantly impacts patients' lives, similar to chronic illnesses like type 2 diabetes and heart failure [3]. Socioeconomic factors are also linked to major determinants of quality of life; older patients tend to report better social functioning, while women generally experience lower quality of life [4]. Although the severity of gastrointestinal symptoms affects daily functioning in IBS, it appears to explain only a portion of the other contributing factors. Some of the least noticed factors are systemic symptoms beyond gut symptoms, which seem to be the primary cause of mental and physical quality of life issues in IBS. Non-GI symptoms, often called non-GI physical manifestations in the DGBI literature, are frequently associated with mental health issues. These signs can manifest as fatigue, pain, or insomnia [5]. Drug therapy remains the primary treatment for IBS at this time, supplemented by other approaches. The five main classes of drugs used to treat IBS include aminosalicylates, corticosteroids, immunosuppressants, biologics, and microbes [6]. Immunosuppressive agents such as cyclosporine, methotrexate, and thiopurines are currently in use. These medications can offer relief for patients with severe IBS, especially those experiencing acute, serious ulcerative colitis (UC), and can benefit patients dependent on corticosteroids. The current treatment options and their limitations (highlighted in red) are detailed in Fig. (**1**) [7].

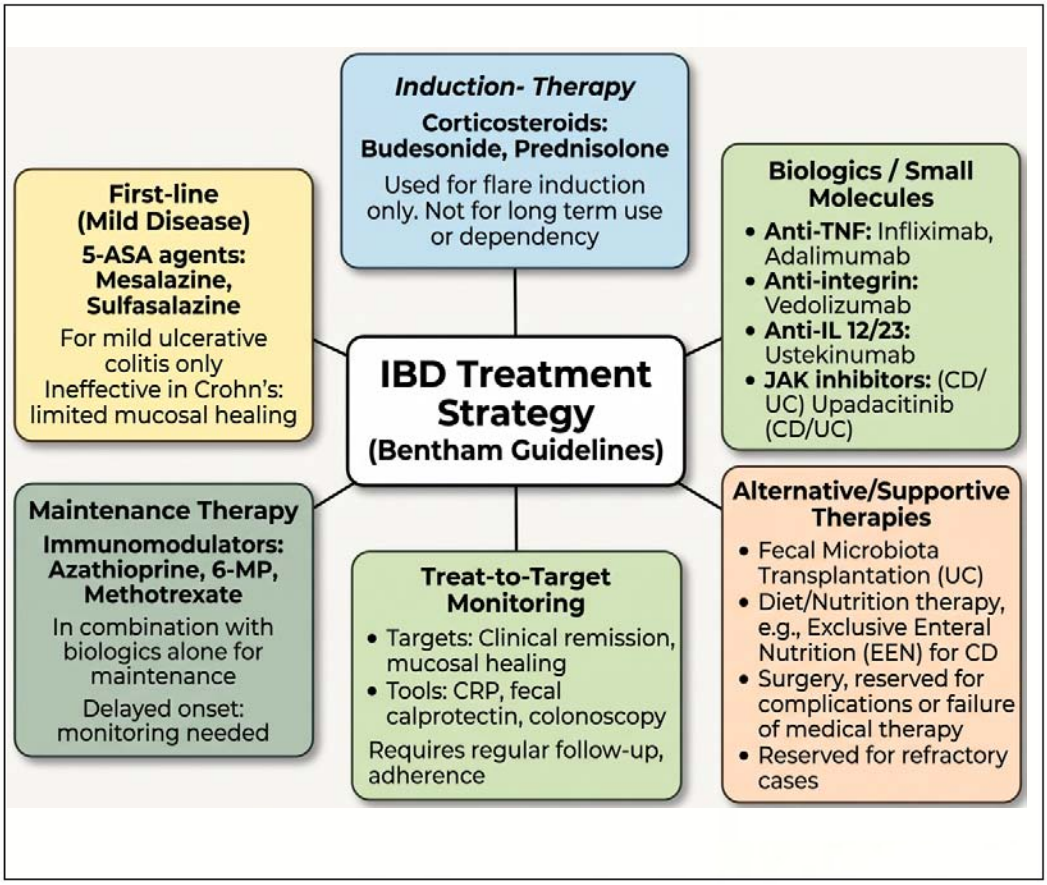


Fig. (1). Current therapeutic drugs and methods of IBD. Content highlighted in red font indicates a limitation of the method.

INTRODUCTION OF TANNINS

Tannins may form proteins and alkaloids, which are water-soluble phenolic compounds with molecular masses between 0.5 and 3 kDa. Tannin protein-precipitating limitations are offset by their unique ability to form complex interactions with proteins, leveraging their distinct characteristics [8]. Tannins are classified into four main classes: hydrolysable, condensed, complex, and phlorotannins. Hydrolysable tannins are further subdivided into gallic acid and ellagic acid types, as shown in the classification summarized in Table **1**.

CHAPTER 7

Essential Oils in Asthma Management: Easing Symptoms through Anti-Inflammatory Properties

Anurag Mishra[1], Waghamare Suresh[1,*], Annasaheb Kudhekar[2], Pooja Paulbudhe[3] and **Sayyad Jahed[4]**

[1] *Department of Pharmaceutical Science, NIMS Institute of Pharmacy, NIMS University, Jaipur, Rajasthan, India*

[2] *Shri Sai College of Pharmacy, Khandala, Maharashtra, India*

[3] *Oyster Institute of Pharmacy, Chh. Sambhaji Nagar, Maharashtra, India*

[4] *Department of Pharmaceutics, Rashtriya College of Pharmacy, Hatnur, Aurangabad, Maharashtra, India*

Abstract: Asthma is a long-term respiratory condition marked by bronchoconstriction, inflammation of the airways, and excessive mucus production. Even though common treatments like bronchodilators and corticosteroids work well, prolonged use of them can have negative effects. Because of their anti-inflammatory, bronchodilatory, and antioxidant qualities, essential oils made from medicinal herbs have been taken into consideration for the treatment of asthma. Bioactive substances like eucalyptol, menthol, and linalool found in oils like eucalyptus, peppermint, lavender, and tea tree oil have been shown to have anti-inflammatory and smooth muscle-relaxant properties. These substances' qualities lessen bronchospasms and airway inflammation, and enhance respiratory health. Additionally, essential oils can affect immunological responses by inhibiting the release of histamines and pro-inflammatory cytokines. However, possible concerns including allergic responses and airway irritation must be taken into account despite encouraging results from preclinical and modest clinical trials. Benefits can be maximized while hazards are reduced with appropriate dilution and delivery techniques, such as diffusion or steam inhalation. To provide standardized procedures for their safe and efficient usage, more clinical research is required. An all-encompassing and natural approach to symptom relief and better respiratory health may be provided by incorporating essential oils as an auxiliary method in addition to conventional asthma therapies.

Keywords: Asthma control, Anti-inflammatory, Airway inflammation, Bronchodilation, Complementary medicine, Essential oils, Eucalyptus oil, Natural therapy, Peppermint oil, Respiratory health.

[*] **Corresponding author Waghamare Suresh:** Department of Pharmaceutical Science, NIMS Institute of Pharmacy, NIMS University, Jaipur, Rajasthan, India; E-mail: waghasuresh@gmail.com

Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed (Eds.)
All rights reserved-© 2026 Bentham Science Publishers

INTRODUCTION

Asthma is an ongoing respiratory condition that involves the airways and causes inflammation and constriction, which makes breathing challenging. Asthma is triggered by a mixture of genetic and environmental factors. The most typical triggers are allergens like pollen, dust mites, animal dander, and mold, as well as irritants like smoke, air pollution, and strong odors. Respiratory infections, physical exertion, and cold air may also trigger asthma symptoms in predisposed patients [1]. Asthma symptoms can vary in intensity and include chest tightness, wheezing, shortness of breath, and persistent coughing, particularly at night or in the early morning. These symptoms can be chronic or sporadic, and they frequently get worse after an asthma attack. If asthma episodes are not adequately controlled, they can be fatal, necessitating prompt treatment with bronchodilators and, in extreme situations, emergency medical care [2].

Both the individual and society are greatly impacted by asthma. Asthma can cause several hospital stays, interfere with everyday activities, and reduce productivity at work or school. Asthma in children can affect their quality of life and physical development. Due to treatment and hospitalization expenses, asthma places a higher financial strain on healthcare systems. For a healthy quality of life and to reduce the overall effects of the condition, asthma must be well managed with medication, lifestyle changes, and avoidance of triggers. One of the main causes of asthma etiology and start is inflammation. Because their airways are constantly irritated, asthmatics are more sensitive to a variety of stimuli, such as allergens, irritants, and respiratory viruses. The thickening of the airway wall, increased mucus production, and tightening of the surrounding muscles brought on by this persistent inflammation all contribute to airway blockage and dyspnea [2, 3]. The immune system overproduces in response to an asthma trigger, releasing inflammatory mediators such as cytokines, leukotrienes, and histamines. These substances cause increased airway edema, excessive mucus production, and bronchial muscle spasm, all of which manifest as wheezing, coughing, and dyspnea. Over time, this heightened immune response also makes the airways more sensitive, increasing the frequency and intensity of asthma episodes [4]. Asthma control involves the suppression of inflammation. Anti-inflammatory drugs, including inhaled corticosteroids, reduce swelling and protect against permanent airway injury. In more serious cases, biologic treatments targeting specific inflammatory pathways are used. With the reduction of airway inflammation, asthma symptoms are under control, decreasing the threat of severe attacks and enhancing general lung function [5]. Essential oils are highly concentrated plant extracts derived from processes such as steam distillation or cold pressing. They preserve the natural aromatic chemicals of plants, which are thought to possess therapeutic qualities. Essential oils have been used for

centuries in traditional medicine and aromatherapy and are renowned for their ability to promote physical and emotional health.Widely utilized essential oils with a variety of advantages include lavender, tea tree, eucalyptus, and peppermint. Crucial oils' bioactive compounds, which have antibacterial, anti-inflammatory, and stress-relieving properties, are what give them their therapeutic potential. Essential oils can affect mood, lower anxiety, promote relaxation, and even relieve pain if inhaled or applied topically [in conjunction with carrier oils]. While some oils, like lavender and chamomile, are peaceful and sedative, others, like eucalyptus and peppermint, are also frequently used for their advantages on respiratory health [6]. Despite their many health advantages, essential oils must be used properly and carefully. Certain oils can induce allergic responses or interfere with medicine when applied straight to the skin without being properly diluted. Furthermore, not all essential oils should be consumed, and improper use might result in toxicity. To maximize their advantages while ensuring safety, it is essential to understand their appropriate use and related risks [7].

HISTORY OF ESSENTIAL OILS IN TRADITIONAL MEDICINE

For thousands of years, traditional medicine has relied on essential oils. They are extracted from various plant parts, including trees and herbs, and are valued for their therapeutic, aromatic, and medicinal properties. Their history demonstrates an unbroken legacy of holistic healing, spanning from ancient cultures to contemporary aromatherapy. An extensive summary of the ways in which essential oils were integrated into different ancient medical systems is provided below:

Ancient Egypt

One of the first societies to harness the therapeutic and spiritual benefits of essential oils was the ancient Egyptians. Their creative approaches to oil extraction served as the model for modern distillation procedures. In Egyptian society, essential oils were used for a variety of purposes, including as cosmetics, medicine, and religion [8]. Due to their potent antibacterial and anti-inflammatory qualities, essential oils like frankincense and myrrh were frequently utilized in medicine. They demonstrated an early understanding of its medical potential by their use to treat infections, ease pain, and disinfect wounds. They were an essential component of traditional medicine because of their capacity to improve physical health [9].

Essential oils were used extensively for spiritual and beauty purposes in addition to medicine. In beauty treatments, oils like rose and cypress were utilized to support personal grooming and skin health. On the other hand, frankincense and myrrh demonstrated their hallowed status in ancient civilization by playing crucial

CHAPTER 8

The Use of Natural Resins in Treating Fungal Skin Infections and Promoting Healing

Rajashri B. Sumbe[1,*], Atul A. Shirkhedkar[2], Shivani S. Kale[3], Snehal R. Kadam[3], Kalyani Autade[1] and **Vishal V. Pande[1]**

[1] *Department of Pharmacology, RSM's N. N. Sattha College of Pharmacy, Ahmednagar, Maharashtra, India*

[2] *R.C Patel Institute of Pharmaceutical Education and Research, Karwand Naka, Shirpur, Dhule, Maharashtra, India*

[3] *SNJB's Shriman Sureshdada Jain College of Pharmacy, Neminagar, Chandwad, Nashik, Maharashtra, India*

Abstract: Natural resins have long been used for their medicinal properties, particularly in the treatment of skin infections and wound healing. This study investigates the antifungal properties of natural resins, including frankincense, myrrh, copal, and dragon's blood, against common fungal pathogens that cause skin infections. The bioactive compounds in these resins have potent antimicrobial, anti-inflammatory, and wound-healing properties, making them viable alternatives to synthetic antifungal agents. The study focuses on the mechanisms by which these resins prevent fungal growth, reduce inflammation, and promote tissue regeneration. Their potential incorporation into modern pharmaceutical formulations is also discussed. This study emphasizes the potential of natural resins for dermatological applications, offering a sustainable, biocompatible solution for fungal skin infections.

Keywords: Antifungal properties, Bioactive compounds, Dermatological applications, Frankincense, Natural resins, Skin infections, Wound healing.

INTRODUCTION

Worldwide, fungal diseases are a leading source of mortality and morbidity, especially for individuals with weak immune systems or individuals admitted to hospitals with severe underlying conditions [1]. Due to a mix of early mortality, health care costs, and reduced productivity, the economic burden of fungal disease in the United States was recently estimated at over $11 billion per year, in

* **Corresponding author Rajashri B. Sumbe:** Department of Pharmacology, RSM's N. N. Sattha College of Pharmacy, Ahmednagar, Maharashtra, India; E-mail: sumberajashri450@gmail.com

Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed (Eds.)
All rights reserved-© 2026 Bentham Science Publishers

addition to the human cost of invasive fungal infections. *Candida*, *Aspergillus,* and *Cryptococcus* are the most frequent fungal pathogens that cause severe human diseases. In certain areas, endemic mycoses, including Histoplasma, Blastomyces, Coccidioides, and Paracoccidioides, are the most common causes [2].

Fungal Infection

Fungal infection, also known as mycosis, is a condition caused by fungi. Fungi consist of millions of different species. They reside in your skin, the soil, plants, and surfaces in your home. Skin-related problems, such as rashes or bumps, can occasionally result from them Table **1** [3].

Table 1. Types of natural resins with their medicinal properties.

Sr. No.	Fungal Skin Infection	Causes	Area of infection	Prevalences
1.	**Cutaneous candidiasis**	*Candida albicans*	Although yeast infections can occur on almost any part of the body, they are most prone to happen in warm, moist, wrinkled areas like the groin and armpits.	Skin candidiasis is thought to affect 1.4% to 2.6% of people worldwide [4, 5].
2.	**Athlete's foot (Tinea pedis)**	*Epidermophyton, or Trichophyton Microsporum.*	It infects the foot.	Global prevalence is estimated to be between 10% and 20%. Approximately 1 billion people worldwide are affected [6].
3.	**Ringworm of the body (Tinea corporis)**	*Microsporum, or Trichophyton*	Affects the torso and limbs.	It is estimated that between 20 and 25% of people worldwide have fungal infections, including Tinea corporis [7, 8].
4.	**Jock itch (tinea cruris)**	*Trichophyton rubrum, Microsporum canis*	A fungal skin infection called jock itch may appear on your groin and thigh area.	Global Prevalence: 4-7% [9].
5.	**Ringworm of scalp (tinea capitis)**	*Dermatophyte fungi.*	Infected scalp.	Its prevalence varies worldwide; in Europe, it has been estimated at around 1.5% of the population [10].
6.	**Tinea versicolor**	*Fungal or yeast*	Tiny, oval, discoloured patches on the skin.	Prevalence estimates worldwide: 2-8%. Approximately 40–50 million people worldwide are affected [11].

Millions of people worldwide are affected by fungal infections such as ringworm, foot infections, and candidiasis, which can be challenging, painful, and psychologically upsetting. While standard antifungal medicines may be helpful, they frequently trigger unintended side effects and may lead to drug-resistant fungal strains. On one hand, natural remedies offer a safer, longer-lasting, and more effective method of managing fungal infections.

- **Effective against fungal infections:** Many of the treatments exhibit antifungal activity, making them effective against a variety of fungal infections [12].
- **Promote general health:** Natural remedies often offer additional medicinal benefits, such as anti-inflammatory and antioxidant properties, which may improve overall health and well-being [13].
- **Low toxicity:** Additionally, natural remedies are less likely to cause side effects, as they are typically less toxic than traditional antifungal drugs [14].
- **Decrease antibiotic resistance:** The overuse of traditional antifungal medicines has contributed to the emergence of fungal strains resistant to antibiotics. Reliance on these drugs can be diminished with the use of natural remedies.
- **Cost-effective:** Because many alternative therapies are widely available and low-cost, they are an affordable option for treating fungal infections.
- **Complementary therapy:** A complementary approach to treating fungal infections is the use of natural remedies alongside traditional treatments [15].

Resins are complex, amorphous substances. Resins are typically extracted from various plant parts, especially rhizomes, bark, seeds, trunks, roots, herbs, and twigs. Resins sometimes combine with acids, while at other times they combine with alcohols or esters. It's possible to obtain a variety of resins, such as oleoresins (resins mixed with oils), glycoresins (resins mixed with sugar), and oleo-gum-resins (resins mixed with gum and volatile oils). Resins can be found in a variety of plants, including Asafoetida, ginger, capsicum, myrrh, jalap, guggul, and others [16].

The resins protect plants from various wounds, such as cuts, incisions, and other injuries. The plant is also shielded from diseases, worms, insects, and other pests by the resins. Resins are used in a variety of medical conditions, including the treatment of eczema, diabetes, ulcers, cancer, fever, constipation, arthritis, and motion sickness. Likewise, some resins are used as condiments and spices. Additionally, they are used as sedatives, flavouring agents, carminatives, cathartics, purgatives, appetizers, stomachics, diuretics, expectorants, *etc.* (Fig. **1**) [17 - 19]

CHAPTER 9

Antimicrobial Properties of Anthraquinones for Treating Bacterial Infections in Gastrointestinal Disorders

Waghamare Suresh[1], Khanage Shantaram G.[2,*] and **Sayyad Jahed[3]**

[1] *Department of Pharmaceutical Science, NIMS Institute of Pharmacy, NIMS University, Jaipur, Rajasthan, India*

[2] *Department of Pharmaceutical Chemistry, Rashtriya College of Pharmacy, Hatnur, Aurangabad, Maharashtra, India*

[3] *Department of Pharmaceutics, Rashtriya College of Pharmacy, Hatnur, Aurangabad, Maharashtra, India*

Abstract: Anthraquinones are a group of natural phenolic compounds found in various plants. They have drawn interest for their ability to fight bacteria. Their potential against bacterial infections, especially those related to gastrointestinal (GI) disorders, offers a chance for new treatment strategies as antimicrobial resistance increases. A systematic review looked at the effectiveness of anthraquinones as antimicrobials. The review gathered studies from databases like PubMed, Scopus, and Web of Science. It focused on their antibacterial effects against GI pathogens, such as *Helicobacter pylori*, *Escherichia coli*, and *Clostridium* difficile. Researchers examined how these compounds work, including how they disrupt membranes, inhibit enzymes, and interfere with bacterial biofilms. Anthraquinones showed strong antibacterial activity against both Gram-positive and Gram-negative bacteria. Compounds like aloe-emodin, chrysophanol, and rhein were particularly effective against important GI bacteria. Studies into their mechanisms revealed that these compounds can disrupt bacterial membranes, block quorum sensing, and inhibit biofilm formation. Their low toxicity to host cells highlights their potential for therapy. The antimicrobial properties of anthraquinones suggest a promising role in treating bacterial infections associated with GI disorders. Their multi-target action and low toxicity distinguish them from standard antibiotics. However, more clinical trials and pharmacokinetic research are needed to confirm their effectiveness and safety in medical use. Anthraquinones could be a good option for tackling bacterial infections associated with GI disorders and addressing the growing problem of antimicrobial resistance.

Keywords: Anthraquinones, Antimicrobial properties, Gastrointestinal disorders, Bacterial infections, Natural products, Antimicrobial resistance, Biofilm inhibition.

[*] **Corresponding author Khanage Shantaram G.:** Department of Pharmaceutical Chemistry, Rashtriya College of Pharmacy, Hatnur, Aurangabad, Maharashtra, India; E-mail: shantaram1982@gmail.com

Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed (Eds.)
All rights reserved-© 2026 Bentham Science Publishers

INTRODUCTION

Conditions affecting the digestive tract, which includes the esophagus, stomach, intestines, liver, pancreas, and gallbladder, are together referred to as gastrointestinal (GI) disorders. These conditions can be either acute or chronic, and they can be brought on by inflammation, infections, structural anomalies, genetics, or lifestyle decisions. Common GI conditions include gastroesophageal reflux disease (GERD), peptic ulcers, irritable bowel syndrome (IBS), inflammatory bowel diseases (IBD) such as Crohn's disease and ulcerative colitis, celiac disease, and liver diseases like hepatitis and cirrhosis. Abdominal pain, bloating, nausea, vomiting, diarrhea, constipation, and weight loss are common symptoms, though they might vary depending on the particular illness [1]. GI diseases are diagnosed and treated using a combination of laboratory testing, endoscopic procedures, imaging, and clinical assessment. Proton pump inhibitors, antispasmodics, anti-inflammatory medicines, and antibiotics are among the pharmaceuticals used in treatment, along with dietary and lifestyle changes. Surgical procedures like liver transplantation or colon resection may be required in extreme circumstances. Maintaining intestinal health requires preventative actions, such as eating a balanced diet, exercising frequently, and abstaining from smoking and excessive alcohol use [2, 3]. With early detection and appropriate management, many GI disorders can be effectively controlled, improving patients' quality of life.

Bacteria play a significant role in gastrointestinal (GI) infections, which are a major cause of illness worldwide. Pathogenic bacteria can enter the digestive system through contaminated food or water, or through contact with infected individuals [4]. Once ingested, these bacteria can disrupt the normal gut flora, invade the intestinal lining, and produce toxins that cause inflammation and damage. Common bacterial pathogens responsible for GI infections include *Escherichia coli* (E. coli), *Salmonella*, *Shigella*, *Clostridioides difficile* (*C. difficile*), and *Campylobacter*. These bacteria can cause a range of symptoms, including diarrhea, abdominal cramps, nausea, vomiting, and fever. Some bacterial strains, such as *Shiga-toxin-producing E. coli (STEC), can cause severe complications, including* hemolytic uremic syndrome (HUS) [4, 5].

Depending on the organism, bacterial GI illnesses have different pathophysiologies. Certain bacteria, such as Campylobacter and Salmonella, infiltrate intestinal cells and cause tissue damage and inflammation. Some bacteria, such as *Vibrio cholerae*, produce toxins that interfere with fluid absorption, leading to copious watery diarrhea. Antibiotic use is frequently associated with Clostridium difficile overgrowth and toxin release, which can result in severe colitis. The intensity of infection depends on factors such as

bacterial virulence, host immunity, and environmental circumstances. Additionally, bacterial infections can disturb the delicate balance of the gut microbiome, further worsening symptoms and delaying recovery [6 - 8].

Management of bacterial GI infections often entails supportive care, including hydration and electrolyte replacement, to prevent dehydration. In rare circumstances, antibiotics are important, particularly for serious illnesses or immunocompromised patients. However, incorrect antibiotic usage can promote antibiotic resistance and aggravate illnesses like *C. difficile* infections. Reducing the spread of microorganisms requires preventive measures, including safe food handling, a clean water supply, and excellent hygiene. Vaccination, if available, also plays a key role in avoiding bacterial GI illnesses, such as those caused by Salmonella typhi (typhoid fever) and *Vibrio cholerae* (cholera). In order to enhance preventative and treatment results, ongoing research focuses on comprehending bacterial mechanisms and creating innovative treatments [8].

INTRODUCTION TO ANTHRAQUINONES AND THEIR BIOLOGICAL PROPERTIES

A class of manufactured and naturally occurring chemicals known as anthraquinones is distinguished by an anthracene backbone with two ketone groups at positions 9 and 10. These substances are frequently found in plants, fungi, lichens, and some insects. Aloe vera, Rheum (rhubarb), Cassia, and Senna are among the medicinal herbs that contain them. Anthraquinones exhibit a broad spectrum of biological actions due to their varied chemical structures and functional groups, which makes them important in both conventional and contemporary medicine. They can exist in free form or as glycosides, which alter their solubility, absorption, and bioavailability [9]

Because they can increase intestinal motility and fluid output, anthraquinones are well known for having strong laxative effects. Herbal medicines for constipation make extensive use of this characteristic. Additionally, these chemicals exhibit strong antibacterial, antiviral, and antifungal activity, making them effective against a range of diseases. Many anthraquinones also display anti-inflammatory and antioxidant effects, helping to counteract oxidative stress and inflammatory disorders. Moreover, certain anthraquinones, such as emodin and aloe-emodin, have been examined for their anticancer potential since they can reduce cancer cell proliferation, induce apoptosis, and regulate signaling pathways involved in tumor growth [10].

Anthraquinones are utilized in industrial and cosmetic compositions, such as dyes, food colorants, and skincare products, in addition to their medical uses. However, despite their favorable effects, several anthraquinones, particularly synthetic

CHAPTER 10

Utilizing Coumarins as Anticoagulants for Stroke Prevention in Patients with Atrial Fibrillation

Vishal V. Pande[1,*], Nandini P. Dolase[2], Rajashri B. Sumbe[1], Kalyani Autade[1], Sofiya Mujawar[2] and **Diksha M. Randive[3]**

[1] *Department of Pharmacology, RSM's N. N. Sattha College of Pharmacy, Ahmednagar, Maharashtra, India*

[2] *Government College of Pharmacy, Karad, Maharashtra, India*

[3] *Amrutvahini Institute of Pharmacy, Sangamner, Maharashtra, India*

Abstract: Atrial fibrillation (AF) is a major cause of ischemic stroke because of its inherent thromboembolic risk, making it a major global health burden. For AF patients, effective anticoagulation continues to be the key to preventing stroke. Among the many anticoagulants on the market, coumarin derivatives, including Warfarin, Acenocoumarol, and Phenprocoumon, have been widely used for many years. The clinical usefulness of coumarins in the treatment of AF is thoroughly covered in this chapter. In order to highlight the importance of prompt anticoagulation, it first clarifies the pathophysiological connection between AF and stroke. A thorough examination of the pharmacological profile of coumarins is conducted, emphasizing their chemical structure, mode of action via vitamin K antagonism, and metabolic factors. Clinical use in AF is examined, with particular attention to therapeutic INR (International Normalized Ratio) monitoring, customized dosing, and strategies to maximize benefits and minimize side effects. Clinical trials provide comparative insights into the safety and efficacy of coumarins in comparison to direct oral anticoagulants (DOACs), providing a nuanced understanding of their ongoing relevance. Alongside elements impacting patient adherence, adverse event management is discussed, specifically bleeding risks and the function of reversal agents. Special considerations are looked at for vulnerable groups, such as the elderly and people with comorbid or renal conditions. Lastly, the chapter looks at how anticoagulant therapy may develop in the future, including new alternatives and the use of personalized medicine to improve stroke prevention techniques for AF patients. The chapter emphasizes the crucial role coumarins play in modern and developing anticoagulation techniques through this synthesis.

Keywords: Atrial fibrillation, Anticoagulation therapy, Coumarin derivatives, Direct oral anticoagulants (DOACs), International Normalized Ratio, Vitamin K antagonists.

[*] **Corresponding author Vishal V. Pande:** Department of Pharmacology, RSM's N. N. Sattha College of Pharmacy, Ahmednagar, Maharashtra, India; E-mail: drvishalpande@gmail.com

Khanage Shantaram G., Waghamare Suresh & Sayyad Jahed (Eds.)
All rights reserved-© 2026 Bentham Science Publishers

INTRODUCTION

The most prevalent heart rhythm disorder, atrial fibrillation (AF), affects over 30 million people globally and is linked to a higher risk of stroke and death [1]. The debate over the mechanisms underlying AF is more complicated because different mapping techniques propose different mechanisms, which distinguishes AF from organized rhythms like atrial flutter, where most techniques concur. AF is dependent on various mechanisms for both initiation and maintenance, as we currently understand. Although there is widespread agreement about the mechanisms causing AF, there is still disagreement among scientists regarding the mechanisms maintaining AF [1 - 5].

INITIATION OF ATRIAL FIBRILLATION

Ectopic Activation from the Pulmonary Veins

Ectopic activation from the pulmonary vein region failed to cause AF in in vitro tests. Haissaguerre and colleagues initially showed the clinical significance of the pulmonary veins in AF in vivo in the late 1990s. They showed that pulmonary vein isolation (PVI) could lower the incidence of AF at follow-up. PVI has remained the most widely used ablation technique for AF treatment ever since. Atrial repolarisation and conduction are necessary for the onset of AF. The action potential duration (APD) and the diastolic interval (DI), the interval between heartbeats, can be used to demonstrate this. APD and DI oscillations are brought on by ectopic beats [3]. APD is first significantly shortened by an early heartbeat (short DI), which lengthens the subsequent DI and so on. In regions with favourable electroanatomical characteristics, aberrant atrial architecture, surface curvature, or fibrosis, this APD alternans alters atrial conduction velocity, promoting wave break and A-Fonset. Patients who report atrial flutter and AF are more likely to require treatment for atrial flutter because, in addition to ectopic beats from the pulmonary veins, bursts of atrial flutter can combine with areas of high heterogeneity [3] to cause fibrillation. The fact that many triggers from various locations may initiate AF at the same spatially conserved spot in a particular patient may be explained by interactions with atrial architecture or fibrosis [4]. Autonomic innervation, which can be regulated by renal denervation or ablation of the epicardial ganglionated plexi, may be one of the factors that facilitate the onset of AF. Nowadays, most people agree that ectopic activation plays a significant role in the development of AF. It is therefore unclear how pulmonary vein triggers contribute to the maintenance of AF. Numerous studies and clinical experience have demonstrated that PVI frequently does not stop AF. Additionally, evidence suggests that isolated pulmonary veins do not always

prevent AF recurrence. These findings imply that different mechanisms maintain AF after it is initiated by ectopic stimulation from the pulmonary veins.

Maintenance of Atrial Fibrillation

Various theories have been developed on the processes that maintain AF following its beginning by pulmonary vein triggers. One of the two main ideas involves localised driving zones in AF, while the other is based on self-perpetuating waves. The duration of AF is determined by the ratio of wavelet generation to extinction, a factor that is crucial for both models.

Multiwavelet Re-entry

Multiwavelet reentry states that the basis of atrial fibrillation (AF) is wavelets of activation that propagate randomly. A wavelet disappears when it reaches the refractory endocardium or scar tissue. Wavelet propagation becomes chaotic as a result of a shift in wavelet direction brought on by an area of electrophysiologic heterogeneity. A minimal quantity of atrial tissue with electrophysiologic heterogeneity is required to maintain AF [4]. Multiwavelet reentry states that the basis of atrial fibrillation (AF) is wavelets of activation that propagate randomly. A wavelet disappears when it reaches the refractory endocardium or scar tissue. Wavelet propagation becomes chaotic as a result of a shift in wavelet direction brought on by an area of electrophysiologic heterogeneity. A minimal quantity of atrial tissue with electrophysiologic heterogeneity is required to maintain AF [4].

Localized Regions, Focal and Rotational Drivers

In addition to having disordered waves, AF also has regular frequency gradients, activation vectors, and electrogram shapes. In this chapter, the second idea is based on organised focal or rotational sources that sustain AF by causing surrounding disorganised wavelets to interact with tissue anisotropy or fibrosis zones [5].

Focal Activation

Centrifugal activation that originates from a single central point is known as focal activation. A breakthrough or focused pattern on the endocardium indicates emerging activation from intramural reentry, according to optical mapping studies using voltage-sensitive dyes to measure endocardial, epicardial, and intramural activation independently [6].

SUBJECT INDEX

A

Airway inflammation 173, 174, 180, 181, 182, 185, 190, 194, 195, 196, 197, 200
  lower 195, 196
Alkaloid-based treatments 13, 14, 16, 19, 20, 21, 24, 25, 26, 27, 30
  efficacy of 21
  improved 27
Aloe-emodin 238, 240, 241, 242, 244, 245, 248, 249, 250, 251, 252, 255, 256, 258, 259
Anthraquinone glycosides 54, 55, 65, 66, 67, 68, 74, 243, 254
  gut bacteria cleave 254
Anti-inflammatory effects 62, 64, 69, 84, 86, 121, 125, 160, 167, 182, 183, 186, 187
  strong 186
Antibacterial activity 69, 77, 221, 238, 245, 248, 262
  broad-spectrum 248
  selective 77
  strong 69, 238, 245, 262
Antioxidant activity 59, 92, 100, 128, 154, 164, 227
  strong 59
  synergistic 100
  vigorous 154
Antioxidant properties 15, 54, 56, 59, 93, 95, 97, 100, 101, 102, 124, 125, 211, 213
Antioxidant qualities 9, 55, 69, 91, 93, 97, 121, 124, 173, 223, 229
  strong 55, 223
Asthma attacks 174, 181, 183, 184, 187, 190, 193, 195, 196, 197, 200
  acute 195
  diet-induced 195
  life-threatening 195
  severe 181, 193, 197
Asthma symptoms 174, 190, 191, 195, 196, 197, 198, 201
help ease 190
  managing 190, 195
Asthma treatments 173, 185, 191, 194, 199, 200
  effective use of essential oils in 199
  prescribed 194
  standard 191, 194
  traditional 199
Atherosclerosis 58, 62, 97, 98, 101, 111, 112, 113, 114, 115, 117, 119, 122, 123, 125
  combat 111
  induced 101

B

Bacterial infections 159, 238, 240, 247, 248, 249, 251, 257, 261
  drug-resistant 248
Bioactive Compounds 90, 134, 136, 213
  extract 134, 136
  extraction of 136
  key 213
  obtaining 134
Biological activities 4, 52, 54, 139, 226, 241, 253
  associated 241
  strong 4
Bleeding risks 270, 277, 290, 291, 295, 297, 299, 303
  high 303
  measured 277
  total 290
Blood pressure 2, 43, 45, 97, 98, 113, 116, 124, 194, 275, 294
  high 43, 97, 275, 294
    high diastolic 116
  low 2
  lower arterial 98
Blood sugar levels 44, 73, 84, 85, 89
  high 85
  lower fasting 73
  prolonged high 85
  reducing postprandial 89

All rights reserved-© 2026 Bentham Science Publishers

Blood vessels 5, 43, 44, 45, 67, 98, 112, 130, 224
brain's 5
heart's 44
Blood-brain barrier 13, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 30
improved 27
improving 23
increasing 21
restricted 19, 20
Bowel movements 55, 66, 75, 243
improved 243
irregular 75
promoting 55

C

Cardiac glycosides 39, 40, 41, 45, 46, 47, 51, 53, 54, 55, 56, 57, 58, 59
common 41
effective 47
Cardiovascular diseases 39, 40, 43, 44, 84, 86, 97, 112, 113, 114, 115, 116, 117, 121, 123, 124, 125
managing 84, 86
risk of 113, 115
severe 43
Cardiovascular health 56, 58, 65, 68, 71, 111, 121, 122, 123, 141
promoting 111
Carrier oil 175, 177, 184, 185, 188, 192, 193, 222, 223
tsp 192
Cell death 40, 67, 88, 244, 246, 247, 248
bacterial 246
cationic peptides cause 247
inducing bacterial 247
programmed 67, 244
Cell membranes 29, 56, 116, 117, 188, 217, 223, 242, 244, 245, 262
breaking bacterial 262
cardiac 56
integrity of 29, 217
microbial 188, 244, 245
Chemical structure 53, 55, 86, 126, 154, 165, 240, 249, 253, 270
varied 240
Cholesterol levels 58, 67, 74, 111, 113, 116, 117, 119, 121, 122, 126
decreasing LDL 116
elevated 117
higher high-density lipoprotein 74
lower blood 122, 126
Chronic inflammation 62, 71, 84, 94, 96, 115, 121, 157, 181, 183, 188
reducing 84
systemic 157
Clinical research 4, 27, 28, 51, 65, 73, 74, 173, 199
early 27
Clinical trials 3, 27, 28, 29, 31, 68, 102, 122, 162, 163, 173, 191, 192, 199, 201, 278, 288, 289
high-quality 199
human 122, 162
important randomized 278
ongoing 28, 29
Constipation 2, 55, 57, 66, 72, 74, 75, 150, 155, 211, 239, 240, 260
chronic 57, 260
opioid-induced 57
persistent 57, 75
relieve 55, 72
Coumarin derivatives 270, 275, 276, 280, 281, 284, 301, 304
single-ring 284
CVD 39, 111, 112, 113, 114, 122, 123, 124, 125, 126, 128
atherosclerotic 114, 123
preventing 111
risk of 113, 122, 124, 126
treating 39
Cytokines 60, 87, 96, 101, 121, 122, 157, 160, 174, 180, 183, 223
anti-inflammatory 160, 223
inflammatory 87

D

Delivery methods 1, 16, 18, 19, 20, 24, 31, 100
cutting-edge 31
cutting-edge alkaloid 31
cutting-edge drug 16
innovative 1
Delivery techniques 12, 17, 19, 24, 26, 27, 28, 173
advanced drug 12
cutting-edge 24
cutting-edge drug 17, 19

non-invasive 27
Diabetes mellitus (DM) 43, 58, 61, 77, 84, 85, 90, 92, 113, 276, 298
Dopamine 2, 3, 4, 5, 6, 9, 11, 12, 23, 25, 26, 27
down 9
endogenous 9
replenish 4
steady 27
Dopamine levels 2, 4, 12, 15, 24
depleted 4
low 4
maintaining 12
reduced 2
Drug absorption 22, 24, 26, 261
increasing 24
quick 26
selective 22
Drug delivery 1, 5, 15, 16, 19, 22, 23, 26, 29, 256
effective brain-targeted 23
enabling direct 26
long-lasting 26
targeted 22, 256
Drug release 20, 21, 25, 30, 258, 261
controlled 25, 261
prolonged 21, 30
regulated 20, 21
sustained 20, 21, 258
Dysbiosis 10, 51, 63, 74, 77, 149, 157, 242
enteric microbiome 157
intestinal 63
microbial 51, 77
tannin-induced 149

E

Efflux pumps 16, 223, 249, 250, 251, 252, 258
bacterial 250, 252
blocking 223, 258
Emodin 66, 67, 68, 240, 241, 242, 244, 245, 248, 249, 250, 252, 255, 256, 258, 259
high-dose 259
Epithelial cells 21, 59, 64, 67, 69, 76, 162, 249, 250, 253
intestinal 64, 67, 69, 76, 162, 250, 253
Essential oils 173, 175, 176, 177, 178, 179, 180, 182, 183, 184, 185, 189, 190, 191, 192, 193, 194, 195, 199, 200, 222, 226
aerosolized 199
incorporating 173
mild 194
use of 177, 200

F

Fatty acids 66, 69, 95, 116, 121, 123, 124, 149, 159, 161, 195, 196
polyunsaturated 116
releasing free 123
saturated 116
short-chain 66, 69, 95, 121, 149, 159
Fibrosis 47, 65, 74, 93, 271, 275, 293
cystic 47
hepatic 74
islet 93
stretch-induced 275
Free radicals 62, 67, 84, 91, 92, 93, 94, 96, 101, 102, 154, 161, 164
Fungal infections 180, 210, 211, 218, 220, 221, 227
common 221
invasive 210
life-threatening 220
managing 211

G

Gastrointestinal tract 54, 64, 69, 90, 111, 141, 162, 253, 256, 257, 261, 276
upper 90
Glucose levels 61, 64, 67, 70, 87, 90, 91, 96, 99, 115
controlling postprandial 91
lower fasting blood 64, 67
lower plasma 99
lowering blood 61, 96
Gut barrier 51, 64, 66, 69, 70, 73, 98, 159, 162
weakened 64
Gut flora 63, 65, 66, 69, 73, 76, 99, 121, 239, 242
balanced 63
healthy 121
human 99
maintaining good 242
Gut health 55, 60, 66, 68, 72, 76, 149, 157, 158, 159, 161, 162, 163, 166, 167, 242
modulate 166
preserving 66, 242
preventing 161

promoting 167
Gut microbiota 52, 68, 72, 74, 77, 95, 96, 98, 99, 121, 155, 156, 157, 158, 159, 160, 161, 162, 167, 251, 253
altering 68
balanced 72, 158
healthy 159, 160
modifying 77

H

Harmful bacteria 63, 66, 77, 159, 188, 216
blocking 159
growth of 66, 77, 188
Heart disease 111, 112, 113, 114, 115, 116, 125, 275
coronary 112
hypertensive 114
ischemic 112, 114, 125
rheumatic 112, 114
Heart failure 39, 40, 44, 45, 46, 47, 54, 56, 112, 276, 293, 294, 295, 298
chronic 45
congestive 39, 45, 54, 276, 294
Heart rate 44, 74, 191, 274, 275, 298
elevated 74
increased 191

I

IBD (inflammatory bowel disease) 51, 55, 60, 64, 66, 67, 72, 73, 76, 77, 166, 167, 239, 256, 261
IL-6 10, 64, 67, 69, 72, 73, 157, 160, 223, 224, 244
Immune responses 97, 156, 157, 158, 174, 180, 181, 185, 188, 189, 190, 199, 200, 223
excessive 188, 223
healthy 189
heightened 174
low-level 157
Immune system 157, 180, 183, 184, 188, 189, 194, 195, 196, 197, 200
Insulin resistance 58, 61, 62, 87, 88, 92, 93, 95, 96, 102
decreasing 61
diabetes-associated 92
multi-organ 95
reducing 58
Insulin sensitivity 51, 55, 58, 61, 64, 67, 68, 70, 73, 84, 87, 88, 96, 100, 102
diminished 96
improved 84, 102
increasing 73
Ischemic stroke 113, 130, 270, 285, 289, 295, 299, 301, 304
reducing 295
risks of 285, 301

J

Juniper 176, 178

K

Kidney disease 167, 297
end-stage 297

L

LDL 65, 70, 74, 98, 117, 119
lower 70
lowering 65
modified 117, 119
reduced 74
LDL (low-density lipoprotein) 67, 70, 73, 74, 97, 111, 115, 117, 121, 122, 123, 124, 141
Lipid metabolism 55, 58, 61, 62, 68, 70, 96, 98, 101, 102, 111, 116
controlling 70
promoting 58
Liver damage 15, 65, 76, 259, 299
preventing drug-induced 76
toxin-induced 76
undiagnosed severe 299
Low-density lipoproteins 70, 73, 97, 111, 121, 123, 124, 128, 141
lower 70, 111
oxidized 128
removing 121

M

Medicinal plants 7, 52, 54, 65, 97, 125, 126, 137, 138, 243, 253
Meta-analyses 70, 74, 123, 277, 278, 285, 286, 294, 297

recent 278
Metabolic pathways 51, 98, 122, 247, 250, 255
important 51
targeting 247
Mucus production 56, 59, 173, 174, 180, 181
encouraging 56
excessive 173, 174
increased 174, 181

N

Natural compounds 4, 39, 123, 124, 220, 245, 247, 248, 262
potential 245
Nervous system 2, 4, 16, 18, 20, 22, 23, 25, 26, 45, 57, 85, 183, 187
central 16, 18, 20, 22, 23, 25
quick Central 26
restricted Central 23
Neurons 1, 2, 4, 7, 9, 13, 66, 72
cholinergic 13
dopamine-producing 2, 4, 7
enteric 66, 72
surviving 9

O

Oral anticoagulants 275, 281, 283, 292, 295, 301
novel 292
synthetic 283
Oxidative damage 56, 60, 61, 70, 71, 87, 88, 91, 92, 93, 94, 95, 97, 98, 100, 101, 102, 128
decreasing 95
intracellular 128
preventing 97
reducing 56, 93, 98
Oxidative stress 9, 10, 13, 51, 52, 59, 62, 69, 88, 91, 121, 124, 129, 161, 244

P

Pancreatic β-cells 58, 61, 64, 70, 85, 87, 88, 93, 244
impaired 85
Parkinson's Disease 1, 4, 5, 25, 26, 28, 31
alkaloid-based 26
controlling 25
management of 1, 28
treatment of 4, 5, 31
Peppermint oil 173, 183, 184, 186, 189, 190, 194
diluted 186
undiluted 186
Pharmacological activities 7, 125, 132, 221, 243, 253, 255, 280
diverse 7
prolonged 255
well-reported 243
Polyphenols 69, 84, 86, 89, 90, 93, 95, 96, 97, 98, 99, 100, 101, 102, 103, 161, 162
dietary 84, 99
down 95
green tea 69
nutritional 95, 98
Pro-inflammatory cytokines 64, 67, 69, 72, 76, 97, 102, 149, 157, 160, 173, 185, 186, 190, 223, 224
increased 157
inducing 149
lower 97, 190
synthesis of 67, 223, 224

Q

Quercetin 59, 60, 61, 62, 75, 84, 87, 102, 215

R

Respiratory health 173, 175, 182, 184, 186, 188, 195, 196, 200
improved 200
Risk factors 43, 112, 113, 114, 115, 117, 275, 276, 279, 286, 288, 294, 295, 299, 300
addressing modifiable bleeding 299
clinical 286
common 294
main 117
ROS (reactive oxygen species) 55, 85, 91, 92, 93, 95, 97, 98, 101, 122, 128, 161, 213, 223, 247

S

Signaling pathways 52, 70, 73, 93, 96, 240, 247
blocking particular 73
cellular 52

Solvent extraction 134, 136, 137
accelerated 134, 136
enhanced 136
pressurised 136
Staphylococcus aureus 188, 216, 221, 222, 246, 248, 251, 252, 259
methicillin-resistant 246, 252, 259
Stroke risk 274, 294, 295, 300, 303
residual 294
total 300
Sugar moieties 40, 51, 52, 53, 54, 59, 126, 127
particular 54
Symptom relief 5, 25, 26, 28, 57, 173, 190, 200
inconsistent 5
moderate 190
quicker 28

T

Therapeutic effects 14, 21, 25, 26, 27, 59, 77, 125, 281
delayed 26
long-lasting 21, 27
significant 59
stable 25
Therapeutic efficacy 17, 18, 19, 22, 23, 31, 226, 255, 260
increasing 22, 23
Traditional medicine 123, 140, 175, 213, 218, 224, 225, 227, 243, 251, 256
Triglycerides 58, 59, 67, 71, 73, 74, 85, 102, 115, 122, 123
altering 71
reducing 115
transports 123

U

Ulcers 51, 60, 72, 75, 77, 130, 211, 239, 242, 249
developing 60
peptic 51, 60, 75, 77, 239, 242, 249
Ultrasonic bath 135
Upholstered furniture 198
UTIs (Urinary tract infections) 221, 249

V

Venous insufficiency 132
VKAs (vitamin K antagonists) 270, 276, 288, 292, 296, 297, 299, 301

W

Water solubility 19, 53, 139, 226
limited 139
poor 226
reduced 19

X

Xylose 126, 128

Y

Yeast infections 210



Khanage Shantaram G.

Dr. Khanage Shantaram G. (M.Pharm, Ph.D) Prof. and Principal Rashtriya Charitable Trust's Rashtriya College of Pharmacy, Hatnoor, Kannada, Dt. Aurangabad. He has more than 17 years of teaching, research and administrative experience. He has published 110 research papers in various National and International peer reviewed Journals, and presented more than 45 research papers in various National and International conferences, workshops and seminars. He has published 2 international Patents. He is a recognized PhD guide currently 3 students are perusing their PhD under his guidance. He has guided 40 M. Pharm students. He has delivered more than 50 guests, plenary lectures and invited lecture in various seminars and conferences. He has received research grant from reputed government funding agencies like BCUD, SPPU. He is a member of UG and PG syllabus committee of SPPU, Pune. He is an Invited editor and expert reviewer for some International Journals to his credit. He is a Life Member of APTI, and registered with Maharashtra State Pharmacy Council.



Waghamare Suresh

Waghamare Suresh is a pharmaceutical researcher, academician, and author specializing in pharmaceutics, drug delivery systems, herbal therapeutics, natural products, and emerging pharmaceutical technologies. He holds an M. Pharm and Ph.D. from NIMS University, Rajasthan. His research interests include controlled-release formulations, microspheres, transdermal and microneedle-based delivery, nanotechnology, hydrogels, medicinal plants, bioactive compounds, and cancer therapeutics. He has contributed to 49 research and review publications, 6 international book chapters, and 9 books in pharmaceutical and allied sciences. He also holds 3 patent registrations related to herbal microemulsions, phototherapy, and neurological disorder detection. His research focuses on innovative therapeutic approaches integrating natural bioactive compounds and advanced drug delivery technologies.



Sayyad Jahed

Sayyad Jahed has completed his B.Pharmacy And M.Pharmacy Y. B. Chavan College of Pharmacy Aurangabad (Dr. BAMU University Aurangabad) PhD pursuing from (NIMS University Jaipur) and currently working as an Associate professor in Pharmaceutical chemistry Department (HOD) at Rashtriya College of Pharmacy Hatnoor Tq. Kannad Dist Aurangabad Maharashtra. It is his passion to design, synthesize & characterize synthetic derivatives as well as design molecules by computer-aided drug design Methods. He has authored 05 research papers and 2 review articles published in national and international journals. He has attended several national and international conferences, workshops, and faculty development programs. He has completed one Indian Patent on neurological disorders.

.

ISBN 979-8-89881-805-0