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The Dark Side of Oral Antibiotics: Adverse Events of Consideration in Dermatology

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ABSTRACT

Antibiotics remain a cornerstone in the management of dermatologic conditions such as acne and hidradenitis suppurativa, underscoring the need for responsible antibiotic stewardship. This article explores the "dark side" of antibiotics, highlighting their role in disrupting the gut microbiome, elevating risks for infections like *Clostridium difficile*, and increasing resistance in *Cutibacterium acnes* and other microbes. Emerging evidence also links antibiotic use to reduced vaccine efficacy and diminished responses to cancer immunotherapy. To mitigate these risks, dermatologists should prioritize narrow-spectrum antibiotics and incorporate combination topical therapies containing benzoyl peroxide (BPO), such as the triple-combination of clindamycin, adapalene, and BPO, to help curb antibiotic resistance. Prudent antibiotic use, combined with topical regimens utilizing BPO, optimizes treatment outcomes while minimizing systemic adverse effects and resistance. Ongoing education and research are essential to refine prescribing practices that balance therapeutic benefits with long-term patient and public health.

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INTRODUCTION

Antibiotics are among the most frequently prescribed medications in clinical medicine and have long been central to dermatologic practice, particularly for the management of chronic inflammatory conditions such as acne and hidradenitis suppurativa. Although their antimicrobial and anti-inflammatory properties provide clinical benefit, accumulating evidence reveals that antibiotic use, especially when prolonged or broadly targeted, can lead to unintended and sometimes serious systemic consequences. Chief among these is the disruption of the gut microbiome, a diverse ecosystem of commensal microorganisms that play a vital role in immune regulation, metabolic homeostasis, epithelial barrier maintenance, and neurologic function.[1] Microbiota dysbiosis is the primary mechanism through which antibiotics contribute to chronic disease development. Antibiotic-induced dysbiosis can persist well beyond the treatment window, setting the stage for chronic diseases ranging from obesity and diabetes to autoimmune disorders and neuropsychiatric conditions. Of particular concern is the growing recognition that antibiotics can impair the immune system's ability to respond effectively to vaccinations and immunotherapies. Both pediatric and adult studies have shown that recent antibiotic exposure is associated with reduced antibody titers following routine vaccinations and diminished efficacy of immune checkpoint inhibitors in cancer. Additionally, the widespread and often inappropriate use of antibiotics in dermatology has contributed to the global crisis of antimicrobial resistance (AMR), with *Cutibacterium acnes* strains exhibiting alarming rates of resistance to commonly used agents such as macrolides and clindamycin. Beyond microbiome disruption and resistance, antibiotics are also implicated in a range of serious adverse drug reactions, including drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome, Stevens-Johnson syndrome, and autoimmune hepatitis. In rare cases, they may even trigger autoimmune bullous diseases or cause idiopathic intracranial hypertension. This manuscript reviews the expanding landscape of systemic effects associated with antibiotic use, particularly in dermatologic settings, and underscores the need for improved antibiotic stewardship.

Oral Antibiotics and the Gut Microbiome

Antibiotic exposure disrupts the gut microbiome by eradicating key taxa and reducing overall microbial diversity.[2,3] While patients are often told that antibiotic effects on the microbiome are temporary, growing evidence shows that even brief courses can cause long-lasting alterations in gut microbial communities.[4] For example, a 5-day course of ciprofloxacin reduces microbial richness and diversity in the distal colon, with most changes reversing within 4 weeks; however, repeated courses cause more persistent shifts.[5] Similarly, a single 7-day course of oral clindamycin treatment dramatically lowers the diversity of anaerobic *Bacteroides* species, with effects lasting up to 2 years.[6] These drug-specific and dose-dependent impacts highlight individual variability in susceptibility to long-term microbiome changes. Antibiotic-induced dysbiosis triggers complex downstream effects on gut barrier integrity, gene expression, epigenetic modifications, and the establishment of niches favorable to pathogenic bacteria and biofilms, ultimately influencing local and systemic immune responses.[7]

Prenatal antibiotic exposure correlates with aberrant DNA methylation linked to metabolic and growth disturbances like low birth weight, which is associated with adult obesity, cardiovascular disease, diabetes, and cancer.[8] Postnatally, the gut microbiota plays a key role in epigenetic programming that governs barrier function and immune homeostasis.[9]

Animal studies confirm that prenatal antibiotic exposure disrupts gut gene expression and barrier function, increasing mucosal immune exposure to luminal antigens and promoting inflammation.[10] Notably, these effects vary by antibiotic class; for example, ampicillin—but not tetracycline—induces barrier disruption and bacterial translocation (ie, movement of bacteria from the gut to the bloodstream).[11] Dysbiosis may also create ecological niches that facilitate colonization by pathogenic bacteria such as *Clostridioides difficile* and *Salmonella typhimurium*, illustrating the multifaceted consequences of antibiotic therapy on host-microbiota interactions and underscoring the need for careful antibiotic stewardship.[3]

Antibiotic-induced microbiome disruption can also favor tumorigenic microbes like *Fusobacterium, Porphyromonas,* and toxin-producing *Bacteroides fragilis* and *Escherichia coli*.[12] These pathogens often form polymicrobial biofilms, especially in the proximal colon, driving chronic mucosal inflammation and promoting colorectal cancer. Epidemiologic data link antibiotic exposure to increased proximal colon cancer risk, suggesting that regional differences in microbial populations and colonocyte sensitivity contribute to tumorigenesis.[13]

Beyond cancer, microbiome dysbiosis is implicated in metabolic disorders such as obesity and type 2 diabetes by altering energy harvest, fat storage, and systemic inflammation.[14] Dysbiotic patterns often include reduced microbial diversity and increased pro-inflammatory bacteria, which contribute to insulin resistance. Immune-mediated diseases—including asthma, inflammatory bowel disease, and allergies—are also linked to dysbiosis, with early-life antibiotic exposure impairing immune development and increasing susceptibility to these conditions.[15]

Emerging evidence connects dysbiosis to neurological and psychiatric disorders via the gut-brain axis, which mediates communication between the microbiota and the central nervous system. Altered microbiomes have been observed in autism spectrum disorder, depression, and multiple sclerosis, potentially contributing to neuroinflammation and neurotransmitter imbalances that affect brain function and behavior.[16]

Clostridium Difficile

Oral antibiotic use is a significant risk factor for developing *Clostridium difficile* infection (CDI), a potentially serious condition caused by disruption of the normal gut microbiota. While *C. difficile* may exist harmlessly in the gut, antibiotics can disturb the balance of beneficial bacteria, allowing the pathogen to overgrow and produce toxins that damage the intestinal lining.[17]

Broad-spectrum antibiotics such as clindamycin, fluoroquinolones, cephalosporins, and penicillins are strongly linked to CDI.[18] These medications eliminate not only the harmful bacteria causing infection but also the protective flora that normally suppresses *C. difficile* growth. As a result, spores can germinate and lead to infection. Symptoms of CDI range from mild diarrhea to severe colitis and life-threatening complications like toxic megacolon. Higher-risk groups include the elderly, hospitalized patients, and those with weakened immunity, but cases have also been reported in healthy outpatients, including patients with acne.[19]

Importantly, systemic antibiotics commonly used to treat acne, such as doxycycline, minocycline, and clindamycin, may also contribute to CDI risk, especially with long-term use.[20] While tetracyclines are generally considered lower-risk compared to other classes, prolonged treatment—often spanning several months—can still disturb the gut ecosystem. Clindamycin is particularly associated with a higher risk of CDI, although topical clindamycin usage in acne treatment has a low rate of gastrointestinal adverse events (0.000045%).[21,22] Though topical antibiotics have

less systemic absorption, extended use may still impact the microbiome, particularly when combined with oral agents. Azithromycin has also been implicated in increased CDI risk.[23]

Prevention of *C. difficile*, therefore, involves antibiotic stewardship, prescribing the narrowest-spectrum antibiotic for the shortest necessary duration, and patient education on CDI risks.

Antibiotics and Immune Function

Antibiotics Impair Vaccine Response

Accumulating evidence suggests that antibiotic-induced disruption of the gut microbiome can impair vaccine-induced immune responses, particularly during early life when microbial colonization and immune development are tightly interconnected. A retrospective cohort study of 560 children aged 6 to 24 months investigated the association between systemic antibiotic exposure and humoral responses to multiple routine pediatric vaccines, including diphtheria-tetanus-acellular pertussis (DTaP), inactivated poliovirus (IPV), *Haemophilus influenzae* type b (Hib), and pneumococcal conjugate vaccine (PCV).[24] The study found that antibiotic use was significantly associated with lower serum antibody concentrations both before and after the booster dose. Notably, each additional course of antibiotics was linked to progressive declines in vaccine-specific antibody levels, with postbooster reductions ranging from 18% to 21% across vaccine types. Moreover, antibiotic-exposed children were more likely to have antibody titers below established protective thresholds at key time points, particularly at 9 and 12 months of age. These findings provide compelling clinical evidence that early-life antibiotic use may attenuate vaccine immunogenicity in a dose-dependent manner. Although the study was observational, the authors propose that disruption of the gut microbiota likely underlies these effects, consistent with prior animal models demonstrating microbiome-mediated regulation of vaccine responses. These results underscore the importance of antibiotic stewardship in early childhood and highlight the potential unintended consequences of diminished vaccine protection. Further prospective studies are warranted to determine causality, assess the duration of immune impairment, and explore whether microbiome-targeted interventions could restore or enhance vaccine efficacy in antibiotic-exposed individuals.

A study in healthy adults demonstrated that short-term administration of broad-spectrum antibiotics prior to influenza vaccination led to a markedly diminished antibody response, accompanied by systemic immune alterations.[25] These individuals exhibited elevated levels of pro-inflammatory cytokines and a profound depletion of key microbial-derived metabolites, particularly secondary bile acids such as lithocholic acid, which dropped nearly 1,000-fold. These metabolites are known to modulate inflammation and support immune homeostasis, suggesting that their absence may contribute to a less effective vaccine response. Supporting this, murine studies have shown that microbiota disruption can blunt vaccine-induced immunity, but reconstitution with beneficial bacteria, such as a consortium of *Bifidobacterium* species, successfully restored the immunogenicity of the 13-valent pneumococcal vaccine.[26] In addition, Oh et al also demonstrated in a mouse model using the trivalent inactivated influenza vaccine that depletion of the intestinal microbiota using broad-spectrum antibiotics led to a significantly impaired antibody response.[27] Collectively, these findings underscore the critical interplay between the gut microbiota and the immune system, with antibiotics potentially undermining vaccine efficacy through microbiome-mediated pathways.

Antibiotics Reduce Immunotherapy Efficacy in Malignancy

Antibiotic use has been increasingly recognized as a significant factor diminishing the efficacy of immune checkpoint inhibitors (ICIs) across various cancers, including urothelial carcinoma (UC). A recent systematic review and meta-analysis focusing specifically on UC patients treated with ICIs demonstrated that antibiotic exposure was associated with a 45% increased risk of mortality (hazard ratio [HR] 1.45) and a 40% increased risk of disease progression (HR 1.40), regardless of the timing of antibiotic administration or type of ICI used.[28] Complementing this, a broader meta-analysis encompassing over 41,000 cancer patients across multiple tumor types revealed that antibiotics significantly worsened overall survival (HR 1.61) and progression-free survival (HR 1.45), while also increasing rates of disease progression.[29] These findings are thought to stem from antibiotics' disruptive effects on gut microbiota, which play a crucial role in modulating the immune response necessary for effective ICI therapy. The negative impact is particularly notable when antibiotics are administered near the initiation of immunotherapy. While antibiotics remain vital for managing infections, their use in cancer patients undergoing ICI treatment should be judicious and carefully balanced to minimize compromise of therapeutic outcomes. Future research is warranted to investigate microbiome-targeted interventions to mitigate these effects and optimize immunotherapy efficacy.

Oral Antibiotics Are Associated With Systemic Adverse Reactions

DRESS Syndrome

Antibiotic-induced DRESS syndrome most commonly involves several key drug classes with distinct clinical patterns and risks.[30] Prevalence ranges from 1 in 1000 and 1 in 10,000 exposures with a female preponderance.[31] Across all antibiotics, the latency from drug initiation to symptom onset varies widely, typically between two and eight weeks but extending up to 300 days. Drug re-exposure may result in symptoms within 24 hours.[32] Clinical features are consistent, with widespread rash and fever nearly universal, eosinophilia present in approximately 94% of cases, and hepatic involvement observed in roughly 76%, highlighting the multisystem nature of antibiotic-induced DRESS.[30]

Anti-tuberculosis agents represent the largest proportion of cases (42%), with isoniazid, rifampicin, ethambutol, and pyrazinamide frequently implicated. Glycopeptides, primarily vancomycin, account for about 18% of antibiotic-related DRESS cases.[30] Vancomycin-induced DRESS commonly presents with the characteristic triad of rash, fever, and eosinophilia, alongside organ involvement affecting the kidneys and liver. The latency period ranges from two to six weeks, and patients generally respond well to drug withdrawal and corticosteroid treatment. Sulfonamides, including sulfamethoxazole-trimethoprim, are responsible for approximately 9% of cases and frequently induce rash, fever, hematologic abnormalities, and hepatic involvement.[30] Though less common, tetracyclines—especially doxycycline—have been associated with DRESS cases exhibiting prominent hepatic and pulmonary complications and comparatively higher mortality rates.[30] Other antibiotic classes, such as beta-lactams, macrolides, and fluoroquinolones, have been reported less frequently but can still provoke severe hypersensitivity reactions.[30]

Stevens-Johnson Syndrome (SJS)/Toxic Epidermal Necrolysis (TEN)

Stevens-Johnson syndrome (SJS) and Toxic epidermal necrolysis (TEN) are rare but severe and life-threatening mucocutaneous reactions characterized by widespread epidermal necrosis and detachment, most commonly triggered by medications. Average incidence in the general population is reported to range from 1 to 2 cases per million per year.[33] Antibiotics are among the most frequent culprits, particularly sulfonamides (eg, trimethoprim-sulfamethoxazole), penicillins, cephalosporins, and fluoroquinolones.[34] These reactions are immune-mediated, typically occurring within 1 to 3 weeks of drug exposure, and are associated with high morbidity and mortality, especially in TEN, where skin detachment exceeds 30% of body surface area.[35] The pathogenesis involves drug-specific cytotoxic T-cell activation and subsequent keratinocyte apoptosis via Fas-FasL interaction, granulysin release, and other pro-apoptotic pathways.[36]

Clinically, SJS/TEN begins with prodromal flu-like symptoms followed by painful erythematous macules, targetoid lesions, and rapid skin sloughing, along with severe mucosal involvement in the oral, ocular, and genital areas.[37] Prompt recognition and immediate withdrawal of the offending antibiotic are essential and remain the cornerstone of management. Supportive care in an intensive care or burn unit is often required. Some evidence supports the use of systemic immunomodulators, such as cyclosporine or intravenous immune globulin, though data remain inconclusive. Genetic predisposition—such as HLA-B*15:02 in Southeast Asians exposed to certain drugs—further underscores the importance of pharmacogenetic screening where available.[38]

Acute Generalized Exanthematous Pustulosis (AGEP)

AGEP is a rare but severe cutaneous adverse reaction with an estimated incidence rate of 3 to 5 cases per million per year.[39] It is primarily triggered by medications, with antibiotics being the most common culprits. Among these, beta-lactam antibiotics, particularly aminopenicillins (eg, amoxicillin, ampicillin) and cephalosporins, are the most frequently implicated.[40,41] Macrolides such as erythromycin, as well as quinolones like ciprofloxacin, have also been reported to induce AGEP.[42] The condition is characterized by a sudden onset of widespread, nonfollicular sterile pustules on an erythematous base, often accompanied by fever and leukocytosis, typically within 1 to 2 days of drug exposure. Histopathology reveals subcorneal or intraepidermal pustules with spongiosis and a dermal neutrophilic infiltrate. AGEP is thought to be a T-cell–mediated type IV hypersensitivity reaction, with rapid recruitment of neutrophils via interleukin-8 and granulocyte-macrophage colony-stimulating factor (GM-CSF).[43] Discontinuation of the offending antibiotic usually leads to resolution within two weeks, but supportive care and monitoring are essential, particularly in severe cases. Recognition and avoidance of the responsible drug are critical for effective management and prevention of recurrence.

Minocycline-induced Lupus, Autoimmune Hepatitis, and Hyperpigmentation

Minocycline, a semisynthetic tetracycline antibiotic commonly used for acne and other chronic infections, has been associated with several distinct autoimmune and pigmentary adverse effects, including drug-induced lupus erythematosus, autoimmune hepatitis, and

hyperpigmentation. Minocycline-induced lupus is a well-characterized syndrome that typically develops after months to years of therapy and presents with arthralgia, myalgia, fever, and positive antihistone or antinuclear antibodies, often with anti-double-stranded DNA negativity, distinguishing it from idiopathic systemic lupus erythematosus.[44] The condition generally resolves within weeks to months following drug discontinuation. Minocycline has also been implicated in autoimmune hepatitis, presenting with elevated transaminases, fatigue, and serologic markers such as anti-smooth muscle or liver-kidney microsomal (LKM) antibodies.[45] Liver biopsy may reveal interface hepatitis and plasma cell infiltrates resembling idiopathic AIH, but drug cessation typically leads to resolution without long-term immunosuppression.

In addition to autoimmune sequelae, minocycline is well known for causing cutaneous and mucosal hyperpigmentation. This pigment deposition may appear as blue-gray discoloration on the skin, nails, sclera, teeth, or oral mucosa, and is categorized into 3 types based on location and iron/melanin content. Type I pigmentation appears in acne scars, type II on normal skin (particularly shins), and type III as diffuse muddy-brown discoloration due to melanin deposition.[46] Hyperpigmentation may persist long after discontinuation and can be cosmetically distressing. Given these risks, especially with prolonged use, clinicians should monitor for early signs of autoimmunity and counsel patients on pigmentation changes during minocycline therapy.

Autoimmune Bullous Diseases

Autoimmune bullous diseases (AIBDs) are a heterogeneous group of rare, potentially life-threatening blistering disorders characterized by autoantibody-mediated disruption of epidermal or dermal-epidermal adhesion. While genetic susceptibility and environmental triggers are known contributors, increasing evidence implicates certain medications—particularly antibiotics—as causative agents.[47] Antibiotic-induced AIBDs, including bullous pemphigoid (BP), pemphigus vulgaris (PV), and linear IgA bullous dermatosis (LABD), have been documented with various drug classes, most notably penicillins, cephalosporins, and fluoroquinolones.[48-50] The most common is drug-induced BP, with a rising incidence of between 4 to 22 new cases per million individuals per year in Europe.[51] These reactions are believed to occur via several immunopathologic mechanisms, including haptenization, epitope spreading, and immune dysregulation.[52] In particular, the disruption of immune tolerance may be exacerbated by antibiotic-induced gut microbiome dysbiosis, which has emerged as a key modulator of systemic immune homeostasis.[53] Clinically, drug-induced AIBDs are often indistinguishable from idiopathic forms, although they may present more acutely and resolve upon withdrawal of the offending agent. Direct immunofluorescence remains essential for diagnosis, revealing linear or intercellular deposition of immunoglobulins and complement.[54] A detailed drug history is critical, and early identification of antibiotics as a potential trigger can inform management, including prompt cessation and targeted immunosuppression. As antibiotic usage continues to rise globally, awareness of this rare but significant adverse effect is essential for dermatologists and prescribing clinicians. Further research is needed to elucidate the precise immunological mechanisms linking antibiotic exposure to AIBD onset and to identify patient-specific risk factors that may predispose to such immune-mediated sequelae. Understanding these associations will be critical in developing preventive strategies and more personalized therapeutic approaches.

Pseudo-allergic Drug Reactions

Pseudo-allergic drug reactions are nonimmune-mediated adverse drug responses that mimic the clinical presentation of true allergic (immunoglobulin [Ig] E-mediated) hypersensitivity but occur through direct activation of immune effector pathways without prior sensitization. Unlike classical allergic reactions, pseudo-allergic responses do not involve antigen-specific immune recognition but can produce similar symptoms such as urticaria, flushing, hypotension, and bronchospasm.[55] A key mechanism involves the activation of mast cells via the Mas-related G protein-coupled receptor X2 (MRGPRX2), which has been implicated in reactions to drugs such as vancomycin, fluoroquinolones, and neuromuscular blocking agents.[55] These reactions are often dose-dependent and can occur upon first exposure, distinguishing them from delayed-type hypersensitivity. Clinically, distinguishing pseudo-allergic from true allergic reactions is important, as re-exposure may be safe with modified administration strategies, such as slower infusion or premedication with antihistamines.

Vancomycin-induced Red Man Syndrome (RMS) is the canonical drug pseudo-allergic response that is an acute, infusion-related hypersensitivity reaction characterized by pruritus, erythema, and flushing of the upper body, neck, and face.[56] It typically occurs during or shortly after rapid intravenous administration of vancomycin. Incidence varies between 4% and 50%.[57] RMS is not an IgE-mediated allergic reaction but results from direct histamine release from mast cells, likely via stimulation of the MRGPRX2 receptor.[55] Symptoms may include hypotension, chest discomfort, and, in severe cases, angioedema.[56] The incidence of RMS can be significantly reduced by slowing the vancomycin

infusion rate (eg, over ≥60 minutes) and premedicating with antihistamines.[56] Unlike true anaphylaxis, RMS does not contraindicate future vancomycin use if administered cautiously.[58] It is important to distinguish RMS from true allergic reactions to avoid unnecessary discontinuation, especially when vancomycin is clinically indicated. Appropriate infusion protocols and patient education are essential for the prevention and management of RMS.

Pseudotumor Cerebri Due to Tetracycline Class

Pseudotumor cerebri is defined by elevated intracranial pressure with normal neuroimaging and cerebrospinal fluid (CSF) composition, presenting with headache, pulsatile tinnitus, transient visual obscurations, abducens palsy, and papilledema that can progress to irreversible vision loss.[59] Among medication-associated cases, tetracycline antibiotics, doxycycline, minocycline, and tetracycline are the most consistently implicated; pharmacovigilance analyses suggest hundreds of reports worldwide, indicating under recognition of this adverse effect.[60] In 1 study, the incidence was estimated to be at least 63.9 per 100,000 person-years.[60] Although the pathophysiology is incompletely understood, experimental and clinical data indicate that tetracyclines impede CSF absorption at the arachnoid villi (where oral retinoids can be directly toxic), thereby raising intracranial pressure.[61,62] The risk becomes synergistically higher when tetracyclines are co-prescribed with isotretinoin, another retinoid that independently alters CSF dynamics; concurrent use is therefore contraindicated. Drug-induced pseudotumor cerebri often mimics the idiopathic form but may occur outside the classic demographic of young, obese women and usually resolves after withdrawal of the offending agent.[63] In contrast, spontaneous remission is uncommon in classical pseudotumor cerebri, so a rapidly improving course after drug cessation strongly supports a causal association. Prompt recognition is critical: stopping the antibiotic and, when necessary, commencing acetazolamide, performing serial lumbar punctures, or undertaking optic nerve sheath fenestration can preserve vision.[64] Because long-term tetracycline therapy is common in dermatology practice, clinicians must counsel patients on early neurologic and visual symptoms, avoid overlapping isotretinoin courses, and maintain a low threshold for ophthalmic referral. Continued mechanistic research is needed to refine dose–response data and identify patient-specific susceptibilities, ensuring safe use of these valuable antibiotics.

Acne and Antibiotics: A Study in Antibiotic Resistance

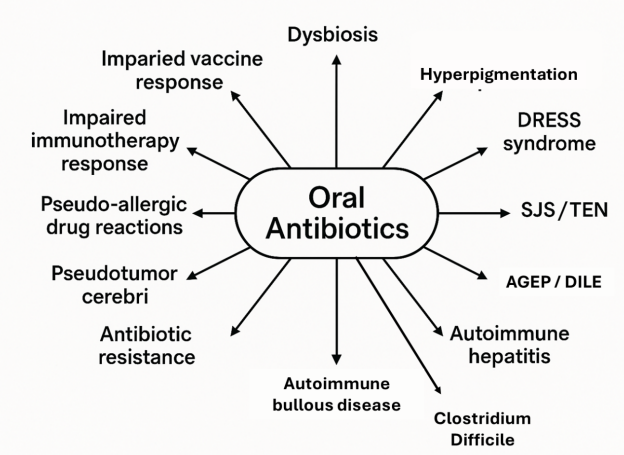
Antibiotics have long been central to acne treatment due to their anti-inflammatory and antimicrobial activity against *Cutibacterium acnes*, but rising global resistance poses a major concern.[65] Dermatologists prescribe over 8 million oral antibiotics annually in the United States, contributing to microbiome disruption and selection of resistant organisms beyond *C. acnes*. Prolonged use (≥3 months) has been associated with a 2.15-fold increased risk of upper respiratory tract infections and increased oropharyngeal colonization by *Streptococcus pyogenes*, with 85% of isolates resistant to at least one tetracycline. Topical erythromycin use has also been shown to increase nasal carriage of *Staphylococcus aureus* and resistant coagulase-negative staphylococci, with resistance persisting for weeks after discontinuation. *C. acnes* develops resistance through mutations in 23S and 16S rRNA, efflux pumps, and plasmid-mediated genes like *erm(x)* and *erm(50)*, which can transfer to other skin bacteria such as *Staphylococcus epidermidis*. Resistance rates vary globally, with macrolide resistance reaching 91% in France and Spain, clindamycin resistance at 53.5% in China, azithromycin resistance up to 100% in India, and tetracycline resistance ranging from 0 to 26.4% in Europe to over 50% in Thailand—underscoring the urgent need for antibiotic stewardship in dermatologic care.[65]

However, antibiotics are still a valuable tool for treating dermatologic disorders such as acne and rosacea. It is therefore important to exercise antibiotic stewardship through the responsible use of these treatments to maximize efficacy while mitigating resistance. The Scientific Panel on Antibiotic Use in Dermatology (SPAUD), which operates under the purview of the American Acne and Rosacea Society (AARS), makes the following recommendations for antibiotic usage: (1) concomitant use of topical antibiotic therapy with BPO to reduce anti-biotic resistance *C. acnes*, (2) use oral antibiotic therapy for acne vulgaris only when felt to be definitively needed and in combination with a topical regimen that preferably contains benzoyl peroxide and a topical retinoid, (3) utilize sub-antimicrobial dosing of oral antibiotics for the treatment of papulopustular rosacea.[66]

CONCLUSION

Dermatologists are the leading prescribers of antibiotics, particularly for conditions like acne and hidradenitis suppurativa, which makes their role crucial in antibiotic stewardship. This is particularly crucial as oral antibiotic use has been associated with numerous potential adverse events, as well as gut dysbiosis, and may potentially affect the efficacy of vaccinations and immunotherapy (Figure 1). To address rising antibiotic resistance, microbiome disruption, and systemic adverse effects, dermatologists must prescribe antibiotics judiciously—favoring narrow-spectrum agents and incorporating topical therapies like the triple-combination treatment (benzoyl peroxide, clindamycin, and adapalene) that enhance efficacy while minimizing resistance. These targeted strategies help preserve antibiotic

FIGURE 1. Adverse effects of systemic oral antibiotics.



effectiveness, reduce adverse outcomes, and protect both individual patient health and broader public health.

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