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Benefits and Side Effects of Modern (Allopathic) Medicines by Drug Category, with Paracetamol as a Worked Example: A Narrative Review

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ABSTRACT

Every effective medicine has both benefits and harms, and sound use depends on understanding the balance between them. This narrative review discusses the benefits and adverse effects of widely used modern (allopathic) medicines by category, using paracetamol (acetaminophen), an analgesic and antipyretic, as a detailed example, and considers NSAIDs, statins, proton pump inhibitors, oral corticosteroids, opioids and antibiotics. Paracetamol is generally safe at therapeutic doses, but acetaminophen accounted for 42% of 662 cases of acute liver failure in a US multicentre study, the toxic metabolite NAPQI being formed by CYP2E1 and detoxified by glutathione until stores are depleted. Its efficacy is also more limited than often assumed: A meta-analysis of randomised trials found it ineffective for low back pain and minimally effective for osteoarthritis, with raised liver enzyme levels more likely (odds ratio 3.8). Policy can reduce harm: Smaller pack sizes in the United Kingdom were followed by a 43% fall in poisoning deaths. Use in pregnancy remains debated; a Swedish sibling-controlled study found no increased risk of autism, ADHD or intellectual disability, while a systematic review concluded that evidence supports an association, and regulators have not agreed. Across other classes, risks differ widely: NSAIDs increase upper gastrointestinal complications (rate ratios 1.8–4.2) and, for coxibs and diclofenac, major vascular events (about 1.4); statins cut major vascular events by 22% per 1 mmol/L LDL-cholesterol reduction with only about 1 in 15 reports of muscle symptoms attributable to the drug; short courses of oral corticosteroids raise the risk of sepsis, thromboembolism and fracture; and antibiotic overuse drives antimicrobial resistance, linked to 4.71 million deaths in 2021. Adverse drug reactions caused 6.5% of emergency admissions in two UK hospitals. We outline how pharmacovigilance, including India's PvPI, can keep the benefit-risk balance favourable. This article is for scholarly purposes and is not medical advice.

Key words: Adverse drug reactions; paracetamol; acetaminophen; NSAIDs; statins; antibiotic resistance; pharmacovigilance; benefit-risk assessment.

1. Introduction

Medicines are among the most effective tools of modern healthcare, but none is free of risk. An adverse drug reaction (ADR) is a harmful effect of a medicine given at normal doses, and understanding how ADRs arise, how they are classified and how they can be minimised is central to rational prescribing [1]. The burden is substantial: in a prospective analysis of 18,820 patients in two UK hospitals, 6.5% of admissions were related to an ADR, in most cases the reaction was the direct cause of admission, and 0.15% of all admitted patients died as a result [2].

This review presents, category by category, what the evidence says about the benefits and adverse effects of commonly used modern medicines. The term "allopathic" is used informally to mean conventional, evidence-based pharmacological medicine. Paracetamol, an analgesic and antipyretic found in almost every household, serves as the worked example, including its chemistry, mechanism of action, evidence for benefit, hepatotoxicity and the controversy over use in pregnancy. We then summarise key evidence for six other categories, discuss challenges and describe future directions. It is a narrative review: evidence was selected by the author from major meta-analyses, trials, regulatory statements and official sources, without a systematic search.

2. Concepts: Benefit-Risk and Classification of Adverse Reactions

Adverse reactions are conventionally classified by mechanism and time course (Table 1). Type A reactions are augmented, dose-related and predictable from the drug's pharmacology; type B reactions

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are bizarre, unpredictable and not dose-related, such as allergy; further categories describe chronic effects, delayed effects, withdrawal reactions and unexpected failure of therapy [1]. The distinction matters in practice. Type A reactions can often be avoided by dose control, monitoring and attention to interactions, whereas type B reactions require rapid recognition and stopping the drug.

Table 1. Classification of adverse drug reactions [1], with illustrative examples chosen by the author.

Type	Meaning	Illustration from this review
A (augmented)	Dose-related, predictable from pharmacology	Paracetamol liver injury after overdose; NSAID gastrointestinal bleeding
B (bizarre)	Unpredictable, not dose-related, often immunological	Drug allergy
C (chronic)	Related to cumulative dose or long-term use	Repeated corticosteroid courses
D (delayed)	Appears after a delay or in later generations	Long-term or in utero effects (debated for paracetamol in pregnancy)
E (end of use)	Withdrawal reactions	Opioid withdrawal
F (failure)	Unexpected failure of therapy	Antibiotic failure through resistance

3. Paracetamol (Acetaminophen): A Worked Example

3.1 Chemistry, metabolism and mechanism

Paracetamol is N-(4-hydroxyphenyl)acetamide, a simple para-substituted phenol bearing an acetamido group (Figure 1). Its analgesic mechanism has long been debated. A recent review concluded that the older view of weak cyclooxygenase inhibition is incomplete, and that a metabolite, AM404, formed in the brain acts on TRPV1 and cannabinoid CB1 receptors, and that serotonergic descending pathways are also involved [3].

The chemistry of its toxicity is well understood. At therapeutic doses most paracetamol is conjugated and excreted, but a small fraction is oxidised by cytochrome P450 enzymes, mainly CYP2E1, to the reactive electrophile N-acetyl-p-benzoquinone imine (NAPQI). NAPQI is normally trapped by glutathione (GSH) and excreted as the GSH adduct. In overdose, GSH is depleted, NAPQI binds to proteins, particularly in mitochondria, and the resulting oxidative stress and activation of c-Jun N-terminal kinase lead to mitochondrial dysfunction and necrotic cell death [4]. N-acetylcysteine acts as the antidote by replenishing GSH [4].

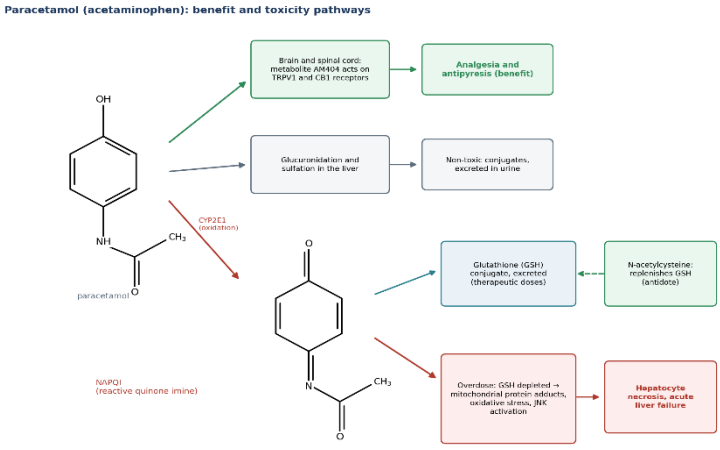


Figure 1. Benefit and toxicity pathways of paracetamol: analgesia through the brain metabolite AM404 [3], detoxification by conjugation, and formation of the reactive metabolite NAPQI by CYP2E1 with glutathione detoxification or, in overdose, glutathione depletion and liver injury [4].

3.2 Evidence for benefit

Paracetamol is a first-line choice for mild pain and fever in many guidelines because of its tolerability, but the evidence for chronic pain is weaker than commonly assumed. A meta-analysis of 13 randomised

placebo-controlled trials in spinal pain and osteoarthritis found that paracetamol was ineffective for low back pain and had a statistically significant but not clinically important effect on pain and disability in osteoarthritis of the hip or knee; patients taking paracetamol were nearly four times as likely to have abnormal liver function test results (odds ratio 3.8, 95% CI 1.9–7.4), although the clinical importance of this was uncertain [5]. The authors concluded that these results support a re-evaluation of the use of paracetamol in these conditions [5]. This illustrates a general point: a drug that is safe at ordinary doses may still have modest benefits for particular indications.

3.3 Hepatotoxicity and policy

Paracetamol is the leading single cause of acute liver failure in many Western centres. In a prospective study of 662 consecutive patients with acute liver failure at 22 US centres, acetaminophen was the cause in 42%; nearly half of these cases (48%) were unintentional, and 38% of patients had taken two or more acetaminophen-containing preparations. The proportion of acute liver failure due to acetaminophen rose from 28% in 1998 to 51% in 2003 [6]. Unintentional overdose, often through combination products, is therefore an important route to harm (Figure 2).

Regulation can reduce this harm. In the United Kingdom, legislation in 1998 limited pack sizes of paracetamol sold over the counter. Over the following 11 years, deaths from paracetamol poisoning (suicide and undetermined intent) fell by 43% and liver transplant registrations for paracetamol-induced liver injury fell by 61% compared with expected trends [7]. The measure addressed a type A reaction through supply control rather than through treatment.

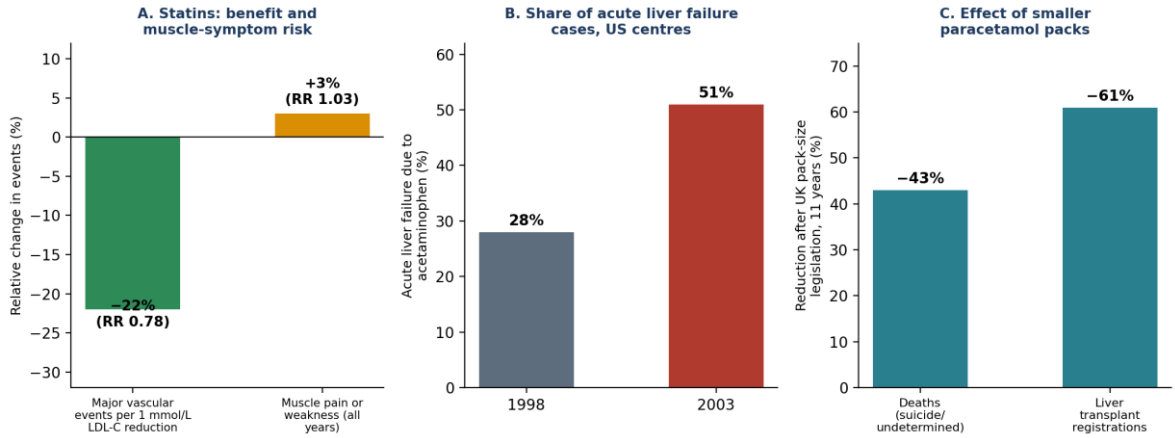


Figure 2. (A). Relative effect of statins on major vascular events per 1 mmol/L LDL-cholesterol reduction and on muscle symptoms [8,9]. (B). Share of acute liver failure cases due to acetaminophen in US centres, 1998 and 2003 [6]. (C). Reductions in paracetamol poisoning deaths and liver transplant registrations after UK pack-size limits [7]. Graphs prepared from the published values.

3.4 Use in pregnancy: An unresolved question

Whether paracetamol use in pregnancy affects neurodevelopment is contested. A Swedish population study of more than two million children, using sibling comparison to control for familial confounding, found that the small associations between prenatal acetaminophen exposure and autism, ADHD or intellectual disability seen in the full cohort were not seen when siblings were compared, suggesting confounding rather than a causal effect [10]. A systematic review using the Navigation Guide method evaluated the epidemiological and experimental literature and judged the evidence supportive of an association, while observational data cannot prove causation [11].

Regulators and professional bodies have reached different positions. On 22 September 2025 the US Food and Drug Administration announced that it was initiating a process to change the acetaminophen label to reflect a possible association with autism, while noting that a causal relationship had not been established [12]. The American College of Obstetricians and Gynecologists stated that there is no clear evidence of a causal link and that acetaminophen remains appropriate for pain and fever in pregnancy when used as needed at the lowest effective dose for the shortest time [13], and the UK regulator advised that paracetamol remains safe in pregnancy [14]. Press reports in 2026 indicated that the label wording had not changed nearly a year after the announcement; readers should check the current status [15]. The

lesson for this review is that untreated fever and pain also carry risks, and decisions should be individual and clinician-guided.

Table 2. Paracetamol: summary of benefits, risks and open questions.

Aspect	Evidence	Ref.
Mechanism	AM404 acts on TRPV1 and CB1; serotonergic pathways; older COX explanation incomplete	[3]
Benefit	Ineffective for low back pain; clinically unimportant effect in osteoarthritis (13 trials)	[5]
Liver safety at therapeutic dose	Abnormal liver tests more likely (OR 3.8); clinical importance uncertain	[5]
Overdose	42% of acute liver failure cases in a US study; 48% of those unintentional	[6]
Toxic mechanism	CYP2E1 → NAPQI; glutathione depletion; mitochondrial injury	[4]
Policy	UK pack-size limits: deaths −43%, transplants −61%	[7]
Pregnancy	Sibling-controlled study shows no increased risk; systematic review supports association; regulators differ	[10–14]

4. Other Drug Categories

4.1 Non-steroidal anti-inflammatory drugs (NSAIDs)

NSAIDs relieve pain, fever and inflammation by inhibiting cyclooxygenase (COX) enzymes and thus prostaglandin synthesis (Figure 3). The same mechanism explains their harms, because COX products protect the stomach lining and regulate platelet function. In a meta-analysis of individual participant data from randomised trials, coxibs and diclofenac increased major vascular events by about a third (rate ratio 1.37, 95% CI 1.14–1.66 for coxibs; 1.41, 1.12–1.78 for diclofenac), whereas naproxen did not (0.93, 0.69–1.27). Upper gastrointestinal complications were increased by all of these drugs: 1.81 (1.17–2.81) for coxibs, 1.89 (1.16–3.09) for diclofenac, 3.97 (2.22–7.10) for ibuprofen and 4.22 (2.71–6.56) for naproxen [16]. The balance therefore differs between drugs and patients.

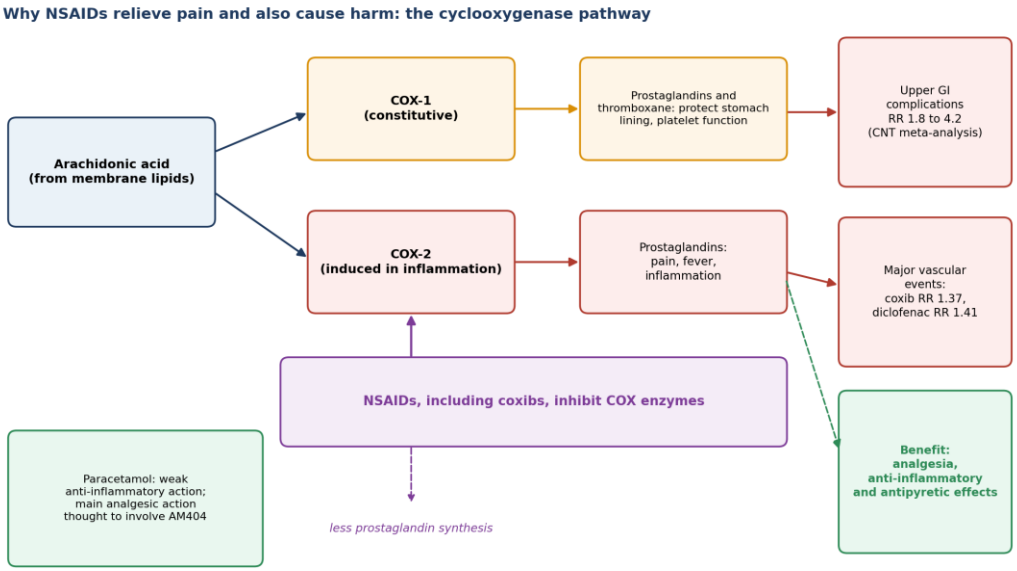


Figure 3. Cyclooxygenase pathway and the origin of benefit and harm with NSAIDs, with CNT meta-analysis rate ratios [16]. The role of paracetamol is discussed in Section 3 [3].

4.2 Statins

Statins provide among the clearest benefit–risk profiles in medicine. A meta-analysis of 26 randomised trials with about 170,000 participants found that each 1.0 mmol/L reduction in LDL cholesterol cut major vascular

events by 22% (rate ratio 0.78, 95% CI 0.76–0.80) [8]. Muscle symptoms are the commonest complaint, but in an individual participant data meta-analysis of large, double-blind trials, statin therapy increased muscle symptoms by only 3% overall (rate ratio 1.03, 1.01–1.06; 7% in the first year), meaning that only about 1 in 15 reports of muscle symptoms in people taking statins was attributable to the drug [9]. Misattribution of symptoms to statins can lead people to stop treatment that would benefit them.

4.3 Proton pump inhibitors (PPIs)

PPIs are highly effective for acid-related disease, but observational studies have linked long-term use to many adverse outcomes. A randomised placebo-controlled trial of pantoprazole in 17,598 participants (53,152 patient-years) found no significant difference in most outcomes, with only enteric infections increased (odds ratio 1.33, 95% CI 1.01–1.75) [17]. This shows how randomised data can temper associations seen in observational studies, although the trial was not designed to test all long-term harms.

4.4 Oral corticosteroids

Corticosteroids are powerful anti-inflammatory and immunosuppressive drugs, and risks accumulate with long courses, but even short courses carry risk. In a US cohort of adults given short-term oral steroids, the incidence rate ratios in the first 30 days compared with no use were 5.30 (3.80–7.41) for sepsis, 3.33 (2.78–3.99) for venous thromboembolism and 1.87 (1.69–2.07) for fracture [18]. These findings argue for using the shortest effective course.

4.5 Opioids

Opioids are effective for severe acute pain and in palliative care, but carry risks of dependence, overdose and death. The 2022 CDC clinical practice guideline for prescribing opioids for pain emphasises weighing benefits against risks of opioid use disorder, overdose and death, and recommends non-opioid therapies where appropriate for many types of pain, while not applying to cancer pain, palliative and end-of-life care [19].

4.6 Antibiotics

Antibiotics have saved countless lives, but misuse drives antimicrobial resistance (AMR), a population-level adverse effect (Figure 4). In the Global Burden of Disease analysis for 2021, bacterial AMR was directly responsible for 1.14 million deaths and associated with 4.71 million deaths globally; by 2050 these figures are forecast to rise to 1.91 million attributable and 8.22 million associated deaths per year [20]. Antibiotics illustrate how an individual benefit can carry a collective cost, and why stewardship is a duty shared by prescribers and patients.

Table 3. Benefits and adverse effects of selected drug categories.

Category	Main benefit	Main adverse effects and evidence	Ref.
Paracetamol	Analgesia and antipyresis; well tolerated at standard doses	Liver failure in overdose (42% of ALF cases in one study); modest efficacy in chronic pain; pregnancy debated	[5,6,10]
NSAIDs	Analgesic, anti-inflammatory, antipyretic	Upper GI complications (RR 1.8–4.2); major vascular events with coxibs and diclofenac (RR about 1.4)	[16]
Statins	22% fewer major vascular events per 1 mmol/L LDL-C reduction	Muscle symptoms RR 1.03 overall; about 1 in 15 reports attributable	[8,9]
PPIs	Treatment of acid-related disease	Enteric infections (OR 1.33) in a large randomised trial	[17]
Oral corticosteroids	Potent anti-inflammatory and immunosuppressant	30-day risks: sepsis (5.30), thromboembolism (3.33), fracture (1.87)	[18]
Opioids	Relief of severe acute and palliative pain	Opioid use disorder, overdose and death	[19]
Antibiotics	Cure of bacterial infection	Antimicrobial resistance: 1.14 million attributable deaths in 2021	[20]

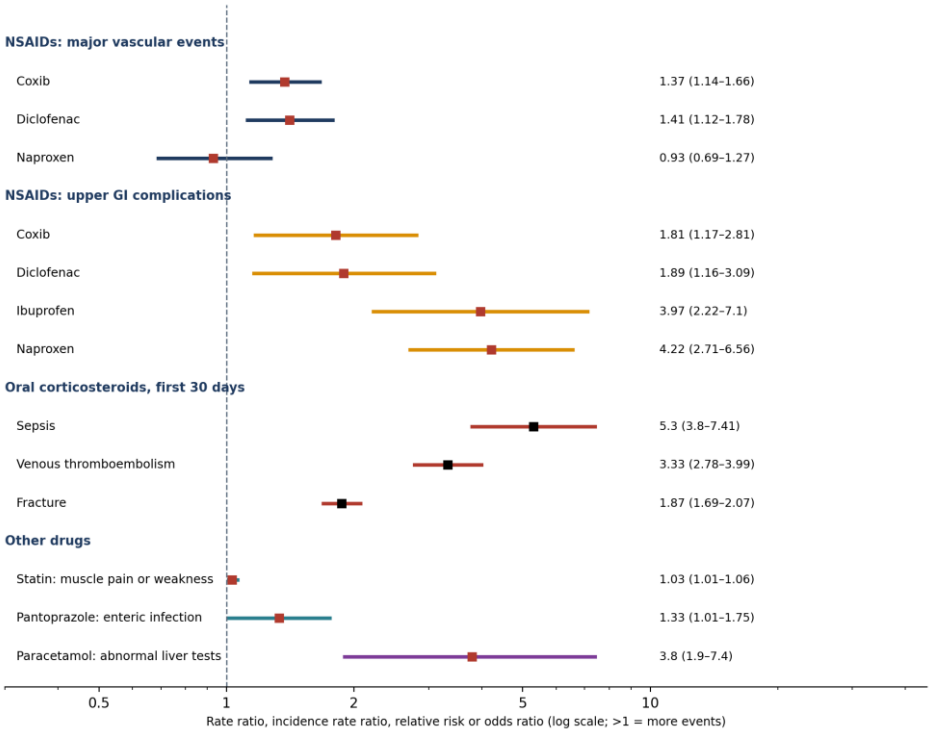


Figure 4. Selected adverse outcome measures for several drug categories with 95% confidence intervals. Metrics differ between studies (rate ratio, incidence rate ratio, relative risk or odds ratio) and are not directly comparable [5,9,16–18]. Graph prepared from the published values.

5. Population-Level Burden

Individual adverse effects add up. Beyond the 6.5% of hospital admissions related to ADRs in the UK study [2], antimicrobial resistance adds a global toll [20] (Figure 5). Because most ADRs are type A and dose-related, many are in principle preventable through appropriate prescribing, patient counselling and monitoring [1,2].

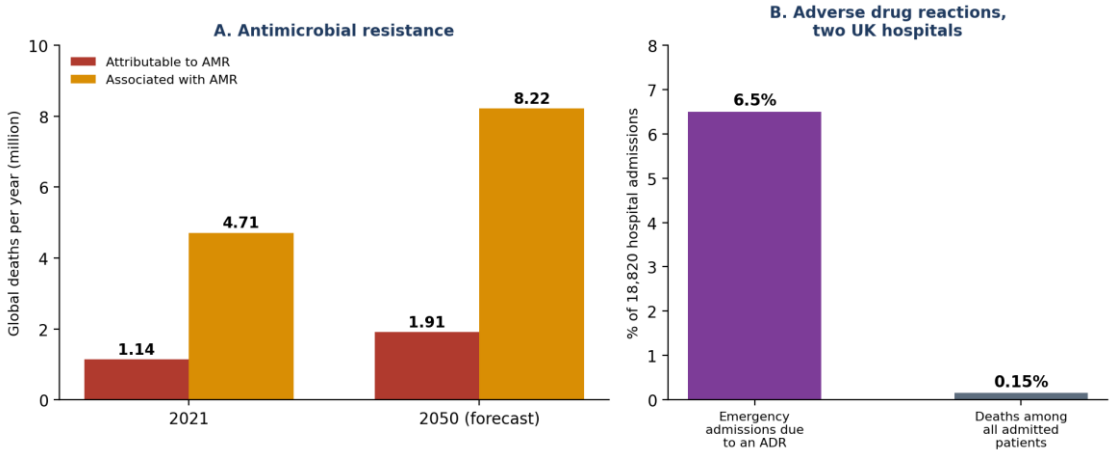


Figure 5. (A). Global deaths attributable to and associated with bacterial antimicrobial resistance, 2021 and forecast for 2050 [20]. (B). Adverse drug reactions as a cause of admission and death in two UK hospitals [2]. Graphs prepared from the published values.

6. Challenges

Several challenges recur. First, benefits and harms are unevenly documented: randomised trials are best for benefits, while rare or delayed harms often depend on observational data that may be confounded, as the pregnancy debate shows [10,11]. Second, perceived and real risks can diverge; many reports of statin muscle symptoms are not caused by the drug [9], while over-the-counter products such as paracetamol are widely assumed to be harmless although overdose is a major cause of liver failure [6].

Third, communication is difficult: regulators and professional bodies have issued conflicting messages on paracetamol in pregnancy [12–14]. Fourth, supply-side measures such as pack-size limits can work [7] but need political will. Fifth, benefit-risk balance depends on the patient and the indication, as seen with NSAIDs, where drugs differ in vascular and gastrointestinal risk [16].

7. Future Directions

Pharmacovigilance is the main safeguard (Figure 6). India's Pharmacovigilance Programme (PvPI), launched in 2010 under the Indian Pharmacopoeia Commission, collects ADR reports from across the country and feeds them into the international system [21]. Strengthening reporting by clinicians, pharmacists and patients, linking it to regulatory decisions, and communicating findings clearly would help to detect problems early. Other priorities include better randomised and sibling-controlled evidence on long-term harms, such as prenatal exposures [10]; systematic evaluation of packaging and supply controls for over-the-counter medicines, building on the UK experience [7]; wider antimicrobial stewardship to slow resistance [20]; and patient education to prevent unintentional overdose from duplicate products [6].

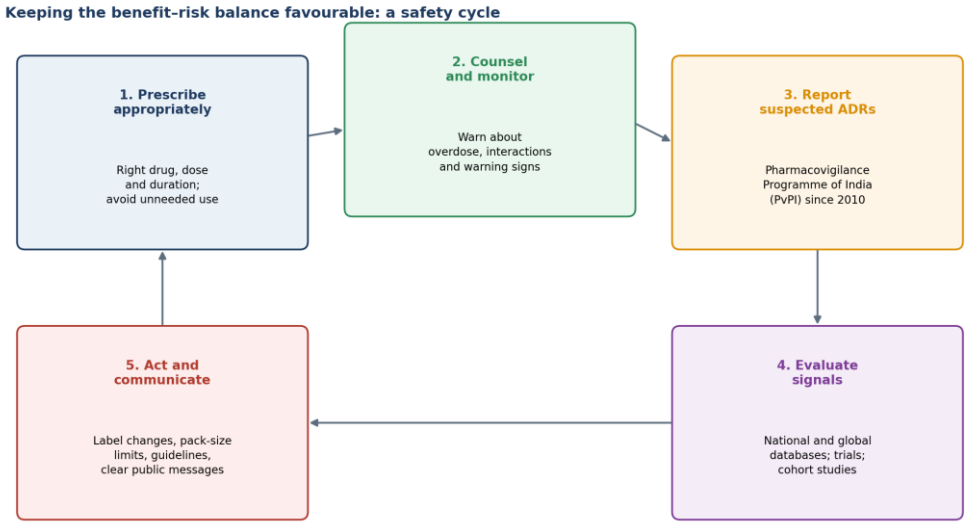


Figure 6. A safety cycle for keeping the benefit-risk balance of medicines favourable, based on refs [1,7,21].

Table 4. Practical messages for prescribers and patients.

Message	Basis	Ref.
Use the lowest effective dose for the shortest time	Short steroid courses carry sepsis, thromboembolism and fracture risk; NSAID risks vary by drug	[16,18]
Count all products containing the same drug	Unintentional paracetamol overdose, often from multiple products, is common in acute liver failure	[6]
Do not stop essential medicines because of unproven side effects	Only about 1 in 15 muscle symptoms in statin users are due to the drug; benefit is large	[8,9]
Use antibiotics only when needed	Resistance is a growing global cause of death	[20]
Discuss medicines in pregnancy with a clinician	Evidence on paracetamol is debated; regulators advise individual, clinician-guided use	[13,14]
Report suspected adverse reactions	Pharmacovigilance depends on reports	[21]

8. Conclusions

Every medicine carries a balance of benefit and harm, and that balance depends on the drug, the dose, the patient and the question being asked. Paracetamol shows the pattern clearly: It is useful and generally safe at therapeutic doses, yet its effects in chronic pain are modest, overdose is a leading cause of acute liver failure, supply controls can reduce deaths, and the pregnancy question remains unresolved. Across other classes, risks range from small and often over-attributed (statin muscle symptoms) to substantial and avoidable (NSAID bleeding, steroid-related sepsis, opioid dependence, antibiotic resistance). Safe use depends on appropriate prescribing, clear communication and strong pharmacovigilance, including in India. This article is for scholarly purposes and should not be used as a basis for treatment decisions without consulting a qualified clinician.

Declaration, Conflict of Interest, Funding and Other Statements

Use of Artificial Intelligence (AI): Artificial intelligence (AI) tools were used to assist in organizing, developing, and refining the presentation of information in this narrative review concerning the benefits, potential adverse effects, and safety considerations of modern (allopathic) medicines across different drug categories, with paracetamol as a worked example. The author is responsible for critically reviewing and verifying the content, checking the accuracy and relevance of the cited literature, ensuring appropriate interpretation of the available evidence, and maintaining the scientific integrity of the manuscript. The final manuscript has been reviewed and approved by the author.

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Data Availability: No new datasets were generated or analyzed during the preparation of this review. The information discussed is derived from previously published literature and other sources cited in the manuscript.
Author Responsibility: The author takes responsibility for the integrity of the manuscript, the appropriate interpretation of the available evidence.

Author Note

This manuscript is a narrative review intended for academic, educational, and scientific information purposes. It discusses the potential benefits, therapeutic uses, adverse effects and safety considerations of modern (allopathic) medicines across major drug categories, using paracetamol as a worked example to illustrate the importance of appropriate use, dosage, contraindications, drug interactions and awareness of potential toxicity.

The information presented is based on published literature and is intended to promote a balanced understanding of the benefits and risks associated with medicines. The discussion does not imply that all medicines within a particular drug category have identical effects or safety profiles. Individual responses to medicines may vary depending on factors such as age, dosage, medical history, liver and kidney function, concurrent medications and other relevant clinical circumstances.

This review does not constitute medical advice, a clinical practice guideline or an individualized treatment recommendation. It should not replace consultation with a qualified healthcare professional or established clinical guidance. Decisions concerning the selection, dosage, modification or discontinuation of medicines should be made in consultation with an appropriately qualified healthcare professional. Readers should not alter prescribed treatment solely on the basis of this review.

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