CLINICAL UPDATE

Analgesia for non-specific low back pain

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What you need to know

- Analgesics have limited effect on low back pain and some, such as opioids and benzodiazepines, have substantial risks
- Oral and, less certainly, topical non-steroidal anti-inflammatory drugs have small benefits that may not be outweighed by risks (particularly gastrointestinal) for short term use for low back pain
- Acute low back pain typically improves within a few weeks without treatment; for chronic low back pain, the focus of management should be on non-pharmacological treatments to improve function and address the broader determinates of pain

Low back pain is the world's leading cause of disability.[1] At any time, half a billion (9%) adults are affected.[1] Many are prescribed, or use, analgesics for pain relief.[2] In this article, we review what is known about common analgesics for treating non-specific low back pain (defined as pain without an identifiable structural or disease cause). We focus on adults aged 18-60 years. A previous *BMJ* education paper describes the management of low back pain in people aged 60 and over, in whom the likelihood of there being a specific cause is greater and the risk-benefit balance of analgesics differs.[3] The treatment of radicular low back pain (such as sciatica) has been detailed elsewhere[4] and is not addressed here.

What is low back pain?

Low back pain (synonymous with "primary" low back pain) is pain felt between the lower ribs and the buttocks.[5] This differs from radicular low back pain, which is when a spinal nerve root is affected, resulting in pain that extends down the legs.[6] Approximately 90% of cases are non-specific, meaning a specific cause has not been identified. The causes of the remaining 10%, affecting around <1 in 100 in primary care[7] and about 5 in 100 in emergency departments,[8] include fractures, infections, malignancies, inflammatory disorders such as spondylarthritis, spinal stenosis, or non-spinal problems such as kidney problems or menstrual related pain.[9]

The prognosis for a new episode of non-specific low back pain is good. Most people recover from acute low back pain within a few weeks irrespective of any treatment.[5] Some people, however, develop chronic low back pain (typically defined as symptoms lasting for over three months), either as a fluctuating/recurring or continuous problem.[10] Of those who present to primary care with acute low back pain, a quarter will have some ongoing pain or functional impairment at three months (chronic pain), although the estimates from individual studies range widely from 2% to 48%.[11]

Who is affected?

Low back pain affects all people of all ages, genders, and ethnic or racial groups, although the point prevalence is slightly higher in women than men (~10% absolute difference consistently across age groups).[12] Prevalence peaks in the 40-69 years age group.[12] Disability resulting from low back pain is similar in high, middle, and lower income countries[1 13] and in urban and rural areas.[12] A 2018 umbrella review identified 54 risk factors associated with low back pain.[14] Examples of potentially modifiable ones include current smoking (odds ratio (OR) 1.8 (95% confidence interval 1.3 to 2.7)), sleep problems (OR 3.2 (1.9 to 5.5)), >2 hours daily spent driving (OR 4.9 (1.4 to 16.4)), prolonged standing or walking (OR 2.9 (1.5 to 5.5)), and mental distress (OR 2.2 (1.3 to 3.7)).[14]

How is low back pain managed?

Most international guidelines advise non-drug treatment and limited, careful use of some analgesic treatments, including those endorsed by the World Health Organisation, National Institute for Health and Care Excellence (NICE), and the American College of Physicians.[15-18] People with non-specific low back pain should be advised to keep active (continue usual physical activities as much as possible), avoid bed rest (as it does not aid recovery),[19] and use self management strategies such as heat packs.[15] About one in five people with chronic low back pain will experience major life or work limitations and may benefit from further treatment.[20 21] For people with chronic non-specific low back pain, optimal approaches use physical and psychological therapies that improve function and address psychosocial contributors to low back pain (see infographic and table 1).[15]

Table 1 | Summary of current guideline recommendations for non-specific low back pain

Treatment	Recommendation (certainty of evidence)
Acute	
Certain analgesics (non-steroidal ani-inflammatory drugs (NSAIDs) and muscle relaxants)	Effective (moderate)
Superficial heat	Effective (moderate)
Acupuncture and needling therapies	Effective (low)
Massage	Effective (low)
Spinal manipulation therapy	Effective (low)
Exercise	Not effective (low)
Orthotics	Not effective (low)
Other analgesics	Not effective (low)
Chronic	
Multicomponent biopsychosocial care	Effective (moderate)
Structured exercise programmes	Effective (moderate)
Certain analgesics (NSAIDs and topical cayenne pepper)	Effective (moderate to low)
Acupuncture and needling therapies	Effective (low)
Structured exercise advice	Effective (low)
Cognitive behavioural therapy	Effective (very low)
Massage	Effective (very low)
Operant therapy	Effective (very low)
Spinal manipulation therapy	Effective (very low)
Mobility assistive products	No evidence, good practice recommendation only
Other analgesics	Not effective (moderate to low)
Therapeutic ultrasound	Not effective (low)
Orthotics (braces, supports)	Not effective (very low)
Pharmacological weight loss	Not effective (very low)
Traction	Not effective (very low)
Transcutaneous electrical stimulation	Not effective (very low)

Certainty of evidence as measured by GRADE approach (box 1)

Acute treatment as recommended by 2017 American College of Physicians guideline[18]

Chronic treatment as recommended by 2023 WHO guidelines[16]

NICE guidance[22] broadly agrees with the above except it recommends against acupuncture and makes recommendations on invasive/surgical procedures (consider radiofrequency denervation or spinal decompression for chronic low back pain in limited circumstances).

Both prescription and over-the-counter analgesics are easily accessible and widely used as an alternative or addition to non-drug treatments. Across primary care services in high income countries Australia,[2] Portugal,[23] US,[24] and Switzerland,[25] 66-89% of consultations for low back pain result in a prescription for analgesia. There are limited prescribing data from low and middle income countries. Herbal medicines (such as topical cayenne pepper) and homoeopathy are prescribed for low back pain in some settings, but the type of agents used varies between regions and is influenced by cultural practices.[26 27]

Efficacy of analgesia

Most commonly used analgesics used to treat low back pain offer no to small benefit versus placebo, and all have a risk of harm (to varying degrees). In this article, we focus primarily on reporting pain intensity, as this is the outcome most consistently reported in literature and a recommended core outcome across acute and chronic pain studies.[28] We also briefly consider other important outcomes such as disability and sleep, which have been shown to correlate with pain. We have included analgesics delivered by any route in this evidence summary.

In table 2, we summarise the efficacy (compared with placebo) of analgesics to reduce low back pain. We summarise data from placebo controlled evidence only because placebo controls in clinical trials are the best method to control for confounding and potential biases.[42] Head-to-head studies comparing one analgesic with another are not included in this summary because determining efficacy is the necessary first step before comparative effectiveness can be explored. Where available, we present the between-group difference between medicine and placebo groups using a 0-10 scale. We report the certainty of the evidence as reported by authors of the cited systematic reviews as per the Grading of Recommendations Assessment, Development and Evaluation (GRADE) (box 1).[43]

Table 2 | Summary of evidence from systematic reviews of efficacy of analgesics versus placebo for low back pain

Study	Analgesic	Chronicity of population*	No of studies	Benefits for pain: mean difference (95% CI) on 10-point scale	Harms: risk ratio (95% CI). Absolute data provided if available	Certainty of evidence	Other outcomes reported (for further reading)
Machado 2015[29]	Paracetamol	Acute and chronic	2 (efficacy), 9 (harms)	Immediate term (<2 weeks): MD 0.1 (−0.1 to 0.4). Short term (2 weeks to <3 months): MD 0.0 (−0.3 to 0.2)	RR 1.0 (0.9 to 1.1)	High (efficacy) and moderate (harms)	Disability (no benefits)
Machado 2017[30]	NSAIDs	Acute and chronic	35 (efficacy), 21 (harms)	Immediate term (<2 weeks): MD −0.9 (−1.1 to−0.7). Short term (2 weeks to <3 months): MD −0.8 (−1.1 to −0.4)	Overall: RR 1.1 (1.0 to 1.2). Gastro related: RR 2.5 (1.2 to 5.2) (28/702 taking NSAIDs *v* 9/465 taking placebo)	Moderate (efficacy) and high (harms)	Disability (small benefits)
Enthoven 2016[31]	NSAIDs	Chronic	13 (efficacy and harms)	Medium term: MD −0.7 (−1.0 to −0.3)	RR 1.0 (0.9 to 1.2) (410 per 1000 report ≥1 adverse event in placebo groups *v* 427 per 1000 in NSAID groups)	Low (both efficacy and harms)	Disability (small benefits)
Ferreira 2021[32]	Antidepressants	Chronic	17 (efficacy), 21 (harms)	**SNRIs.** Short term (<2 weeks): MD −0.4 (−0.6 to −0.1). Medium term (3-13 weeks): −0.5 (−0.7 to 0.3). **TCAs.** Short term (<2 weeks): MD −0.1 (−0.5 to 0.4). Medium term (3-13 weeks): −1.0 (−2.1 to 0.2). Long term (3-12 months): MD −0.8 (−1.6 to 0.0)	**SNRIs.** RR 1.23 (1.16 to 1.30) (63% in antidepressant groups *v* 50% in placebo groups). **TCAs.** RR 1.49 (0.95 to 2.34) (22% in antidepressant groups *v* 13% in placebo groups)	Moderate (SNRI efficacy). Low/very low (TCA efficacy). Low (SNRI and TCA harms)	Disability (small benefits)
Abdel Shaheed 2016[33]	Opioids	Chronic	13 (efficacy), 8 (harms)	Short term (<3 months): MD −1.0 (−1.3 to −0.7). Intermediate term (3-12 months): −0.8 (−1.0 to −0.6)	RR 1.3 (no CI reported) (49% in placebo groups and 69% in opioid groups reported adverse events). In 8 of the 13 trials, more than 50% of participants dropped out due to adverse events including lack of efficacy	Moderate (efficacy)	Disability (no benefits)
Enke 2018[34]	Anticonvulsants	Chronic	4 (efficacy), 6 (harms)	Short term (>2 weeks but <3 months): MD 0.0 (−0.8 to 0.7). Intermediate term (>3 but <12 months): MD −0.1 (−1.4 to 1.2)	RR 1.4 (1.2 to 1.7)	High (efficacy short term and harms). Low (efficacy intermediate term)	Disability (no benefits)
Van Tulder 2003[35]	Benzodiazepines	Acute and chronic	4 (efficacy)	No pooled results available. Trials show various results, are of varying quality, and all conducted in 1992 or earlier	Not reported	Not assessed	Global improvement (no pooled results)
Cashin 2021[36]	Other muscle relaxants	Acute and chronic	17 (efficacy), 22 (harms)	Immediate term (<2 weeks): MD −0.8 (−1.2 to −0.3). Short term (3-13 weeks): MD 0.0 (−0.5 to 0.6)	RR 1.6 (1.2 to 2.0)	Low and very low (efficacy and harms)	Disability (no benefits)

Table 2 | Summary of evidence from systematic reviews of efficacy of analgesics versus placebo for low back pain *(Continued)*

Study	Analgesic	Chronicity of population*	No of studies	Benefits for pain: mean difference (95% CI) on 10-point scale	Harms: risk ratio (95% CI). Absolute data provided if available	Certainty of evidence	Other outcomes reported (for further reading)
Chou 2022[37]	Oral corticosteroids	Acute and chronic	1 (efficacy and harms)	Short term (2 weeks to 3 months): MD 0.6 (CI −1.0 to 2.2)	RR 0.5 (0.2 to 0.9)	Low (efficacy and function)	Function (may be small benefits)
Chou 2022[38]	Cannabinoids	Chronic	0	No evidence	No evidence	NA	NA
Mathieson 2018[2]	Combination oral medicines	Acute and chronic		No benefits for all combinations except a single study of buprenorphine plus pregabalin *v* buprenorphine for chronic back pain at immediate (MD 2.3 (2.8 to 1.9)	No difference in any combination	Low (efficacy and harms)	Disability (no benefits)
Derry 2017[39]	Topical preparations	NA	0	No evidence for topical analgesics for low back pain directly. Non-direct evidence (sprains and strains, other musculoskeletal pain) shows some benefit of rubefacients and topical NSAIDs over placebo (effect sizes not reported)	NA	NA	NA
Mathie 2017[40]	Homoeopathy	NA	0	No direct or indirect evidence of acceptable quality	NA	NA	NA
Oltean 2014[41]	Herbal remedies	Acute and chronic	14	Various effect sizes of different herbal remedies ranging from small to moderate sized effects. Herbal remedies with moderate certainty evidence of effectiveness over placebo are white willow bark for acute pain and *Capsicum frutescens* cream for chronic pain	Unclear/unknown	Varying from moderate to low	Disability (some benefits)

SNRIs = serotonin-norepinephrine reuptake inhibitors. TCAs = tricyclic antidepressants.

* Where separate effect sizes are available for acute and chronic presentations, we have provided both. Where reviews have compiled the evidence and report only the overall effect size including both acute and chronic presentations, we have reported that.

Box 1: Certainty of evidence (GRADE)[43] and size of treatment effects[18]

- *High certainty*—We are confident that the true effect lies close to that of the estimate
- *Moderate certainty*—We are moderately confident in the effect estimate: the true effect is likely to be close to the estimate of the effect, but there is a possibility that new research may change the estimate
- *Low certainty*—Our confidence in the effect estimate is limited: the true effect may be substantially different from the estimate of the effect
- *Very low certainty*—We have very little confidence in the effect estimate: the true effect is uncertain
- *No effect*—The point estimate is very close to zero, and the confidence interval is precise around either side of zero
- *Small effect*—The point estimate of the between-group difference is less than 1 point out of 10 above that of placebo, with confidence intervals that do not cross zero or cross into what would be considered a moderate effect
- *Moderate effect*—The point estimate of the between-group difference is less than 2 points out of 10 above that of placebo, with confidence intervals that do not cross zero or cross into what would be considered a small or large effect
- *Large effect*—The point estimate of the between-group difference is ≥2 points out of 10 above that of placebo, with confidence intervals that do not cross zero or cross into what would be considered a small or moderate effect
- If confidence intervals span multiple effect descriptor categories, we use both descriptors and highlight that the evidence is imprecise or unclear

Paracetamol (acetaminophen)

There is moderate to high certainty evidence of no effect over placebo for both acute and chronic low back pain. A systematic review of 13 randomised controlled trials (RCTs) in Australian and Austrian cohorts of patients with low back pain found a mean difference in pain score of −0.5 (95% confidence interval −2.9 to 1.9).[29] In trials of patients with low back pain, there was no increased risk of harms. However, in RCTs assessing people with osteoarthritis or low back pain, using paracetamol was more likely to cause abnormal liver function tests (though the clinical importance of the changes remains unknown).[29]

Observational data and trials in broader populations using paracetamol for any reason (such as other pain, fever) at standard therapeutic doses show a dose-dependent increase in risk of harms (cardiovascular, gastrointestinal, and renal).[44] Data informing these risk estimates are potentially confounded by not adjusting for concomitant use of non-steroidal ani-inflammatory drugs (NSAIDs) and by including participants who were unsuitable for NSAIDs (with cardiac, gastrointestinal, and renal risk factors), who are prescribed paracetamol as an alternative. We observe in practice that paracetamol is typically safe when taken as directed in people without contraindications and is generally safer than anti-inflammatories.[45 46]

Non-steroidal anti-inflammatories (NSAIDs)

NSAIDs include ibuprofen, diclofenac, naproxen, and celecoxib. For acute low back pain, there is moderate certainty evidence of small benefit above placebo (mean difference in pain intensity −0.9 (95% CI −1.1 to −0.7) from six RCTs conducted in Norway, Belgium, France, Australia, and Germany)[30] and a risk ratio of 2.5 (1.2 to 5.2) of gastrointestinal harms (28/702 participants taking NSAIDs compared with 9/465 in the placebo groups).[30] For chronic low back pain, there is low certainty evidence of small average benefits (mean difference −0.7 (−1.1 to −0.3, from six RCTs conducted in Italy, UK, and US).[31]

The available data from RCTs of chronic low back pain do not report increased risk of harm compared with placebo.[31] However, RCTs and observational studies of populations taking NSAIDs for any reason (such as osteoarthritis or rheumatoid arthritis) showed increased NSAID related adverse events (such as gastrointestinal, cardiovascular, and renal) which escalate with increasing doses and long term use.[47]

Antidepressants

There are no data on antidepressants for treating acute low back pain. For chronic low back pain, there is moderate certainty evidence that serotonin-norepinephrine reuptake inhibitors (such as duloxetine) may have a small effect (mean difference −3.67 (−5.91 to −1.42) from three RCTs conducted in the US and Japan) and low certainty evidence that tricyclic antidepressants are ineffective (mean difference −0.9 (−5.4 to 3.7) from three RCTs conducted in the US and Switzerland).[32] Harms when used specifically for low back pain are unclear, although in broader populations (such as mood disorders) antidepressants are associated with nausea, weight gain, sexual dysfunction, and sleep problems.[48]

Opioids

A 2023 trial conducted in Australia in 347 patients with acute low back pain found strong evidence of no effect of oxycodone (mean difference 0.5 (0.0 to 1.1)), and worse long term outcomes (such as worse pain and a higher risk of opioid misuse) compared with placebo.[49] For chronic low back pain, there is moderate certainty evidence that opioids (numerous single or combination opioid formulations including morphine, tramadol alone or with paracetamol, tapentadol, oxycodone, and fentanyl) probably have a small average effect (mean difference −1.0 (−1.3 to −0.7) from 13 RCTs conducted in Germany, US, Canada, and Australia).[33] Despite this, opioids are not recommended to treat chronic pain because of risks of harms with long term use, including dependence, misuse, overdose, and tolerance resulting in dose escalations that may further contribute to increased risk of harms.[33 50]

Anticonvulsants

There are no data on efficacy for acute low back pain. However, there is high certainty evidence of no effect above placebo of gabapentin or pregabalin for chronic low back pain (mean difference 0.0 (−0.8 to 0.7) from 14 RCTs conducted in Australia, US, and Ireland).[34] The use of anticonvulsants causes increased risk of harms including drowsiness, somnolence, dizziness, and nausea.[34 51]

Benzodiazepines

Small trials conducted in the 1970s to 1990s report some effect on acute or chronic low back pain (effect size and level of certainty not reported).[35] Other studies indicate that benzodiazepines do not possess meaningful analgesic properties separate from their sedative properties.[52] A 2017 RCT of 114 patients conducted in the US did not find that that adding diazepam to diclofenac in people attending an emergency department improved functional outcomes or pain at one week.[53] Benzodiazepines are associated with increased falls, cognitive impairment, and risk of addiction.[54]

Non-benzodiazepine muscle relaxants

This category of medicine is broad and includes a variety of pharmacologically unrelated medications such as cyclobenzaprine, tolperisone, baclofen, and orphenadrine with similar indications. In both acute and chronic low back pain, there is low and very low certainty evidence that non-benzodiazepine muscle relaxants might offer small benefits (mean difference −0.8 (−1.2 to −0.3) from 14 RCTs from the US, Finland, UK, Turkey, and India) but increase the risk of harms, primarily sedation.[36]

Oral corticosteroids

Limited evidence suggests they may be not effective for acute or chronic low back pain (mean difference 0.6 (−2.2 to 1.0) from one RCT conducted in the US), and may not cause harm in short courses.[37]

Cannabinoids

A single RCT of oral cannabidiol in 100 patients conducted in Australia found no effect on low back pain compared with placebo (mean difference −0.3 (−1.3 to 0.6)) and no increase in short term harms in a hyperacute, emergency department setting.[55] There are no data for chronic low back pain, and no data on harms associated with long term use in other chronic conditions.[38]

Oral combination medicines

There is low certainty evidence that combining medicines does not produce superior effect sizes and may increase the risk of harms.[56] An example of combination medicines sometimes used to treat low back pain is an opioid plus an NSAID or paracetamol.

Topical preparations

There is some indirect evidence (level of certainty not assessed) that some formulations of NSAIDS and rubefacients may reduce back pain attributed to muscle strains or sprains more than placebo (effect size not reported),[39] which may apply to some cases of acute

low back pain, with no increased risk of harms.[39] There are no data on efficacy for chronic low back pain, but indirect evidence from other chronic pain conditions such as knee osteoarthritis has shown limited effect.[39]

Homoeopathy

There is no reliable evidence of efficacy of homoeopathy for acute or chronic low back pain.[57] While many may consider homoeopathy harmless, some indirect risks may apply, including the risk of harm to patient's trust and delaying use of treatments that may be more effective such as those recommended in table 1.[57]

Herbal medicines

Some herbal remedies have reported efficacy compared with placebo (with low to moderate certainty) for both acute and chronic low back pain, such as cayenne or capsaicin plasters, devil's claw, willow bark, and topical lavender oil.[41] However, the size of the effects is unclear due to critical heterogeneity across trials (such as use of a variety of non-standard outcome measures for pain or recovery and widely varying formulations and quality of the herbal products used). Most available trials are limited by authors' conflicts of interest.[41] Harms are uncertain, with suboptimal reporting in trials.[41]

Dissonance between evidence and practice

Despite limited supportive evidence, analgesics are still commonly used for low back pain.[2 12] In our experience, reasons for this include a real or perceived lack of alternatives, pressure from patients, a strong desire to help, and the lower cost and better accessibility of medicines compared with physical and psychological therapies. We recommend clinicians discuss with patients the evidence and explore more effective alternatives such as those listed in table 1.

If an analgesic is to be recommended for management of low back pain, oral NSAIDs (or topical NSAIDs if there are contraindications to oral formulations) probably have the most favourable benefit-harm balance (see infographic and appendix on bmj.com). There is high to moderate certainty evidence that paracetamol, opioids, antidepressants, and anticonvulsants are not effective. These medicines have associated harms, many of which are serious. Box 2 outlines special considerations when managing patients with low back pain.

Box 2: Special considerations when managing patients with low back pain

- The evidence presented here should be used to guide clinical practice around starting new medicines for patients who are not already long term users
- Despite the lack of evidence for efficacy, many people with chronic low back pain are already using long term analgesics
- Rapid or forced tapering of analgesics with risk of withdrawal (such as opioids, anticonvulsants, and benzodiazepines) can cause serious harm
- Long term users of medicines should be assessed individually to determine the benefit-harm balance of reducing their use of pain medicines
- Some patients may require supportive treatments while undergoing tapering, and appropriate treatment for substance use disorders if they are identified[58]
- Some of the medicines used for low back pain carry the risk of drug interactions. For example, combining opioids with other sedatives or respiratory depressants, such as benzodiazepines, increases the risk of mortality and serious adverse events[59]

If analgesics are used, international guidelines agree they should be at the lowest effective dose for the shortest duration, and as an adjunct to other non-medicine strategies which address the broader biopsychosocial aspects of pain.[16 -18]

How patients were involved in the creation of this article

Two authors of this article are patients. MS had severe chronic disabling back pain and has now recovered. He is a consumer representative at Cochrane Back and Neck since 2000. JC has ongoing chronic disabling back pain that came on after a workplace injury that prevented him working. He has been prescribed various pharmaceutical and non-pharmaceutical strategies. Both authors reviewed multiple drafts of the article. They optimised the use of language throughout, recommended using examples of medicines that patients might be more familiar with than the overarching class, and explained that patient readers may want more explanation about how to interpret effect sizes. They also acknowledged the practical reasons some patients may elect to use medicines that are not supported by empirical evidence.

Other authors CMPJ, MU, and CWCL have all had episodes of acute low back pain.

Education into practice

- Think about the last time you reviewed a patient who presented with low back pain. What factors led to your decision whether to prescribe an analgesic or not?
- How might you explain effectiveness and risks of oxycodone to a patient who requests it for low back pain?

Questions for future research

- How can we reduce prescribing of analgesia when benefits do not outweigh harms?
- How can we best support people using ineffective drugs for low back pain on a regular basis to stop using these analgesics?
- How effective are analgesics for low back pain in the immediate term (within 2 hours)?

How this article was created

We initially searched the *Cochrane Database of Systematic Reviews* using terms including various names of the medicines of interest and "back pain" to find sources for the evidence summary. Where there was no relevant Cochrane review or it was outdated by a more recent, high quality, systematic review (high quality defined as following PRISMA guidelines, and assessing certainty of evidence), we used the non-Cochrane systematic review. If no relevant systematic reviews were available, we referred to single randomised controlled trials for evidence of efficacy.

We also consulted our networks and other experts in the field to identify if any relevant evidence was missing. We did not use observational or non-randomised studies as evidence for efficacy but occasionally referred to them when reporting risk of harm (as these study designs are often more suitable for detecting rare events). When reported certainty of evidence, we report what the systematic review authors report (we did not undertake an independent assessment).

Contributors: CMPJ, CWCL, RC, MU, and MS were involved in the conception of the project. SS and JC were invited to join the group after the idea was conceived. CMPJ wrote the first draft. All authors provided critical review and agreed the final version. MS and JC provided a patient perspective. SS provided a perspective from a lower-middle income country.

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