Unwritten Health

ISSUE 1 · NOVEMBER 2026 · THE EXCLUSION DEBT REPORT

Lung cancer

The deadliest cancer in Europe is fought with evidence built around the patients it is least likely to kill.

A monthly report on exclusion debt: the cost of building medical evidence without the people it is about.

Unwritten Health

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THIS REPORT IN FIVE NUMBERS

370,000
lung cancer deaths a year in Europe, more than any other cancer

53%
of older adults with metastatic lung cancer would be excluded from a typical clinical trial

2×
the incidence in England's most deprived areas against the least

15%
of real-world lung cancer patients enrolled in a clinical trial; 54% of the rest had none nearby

89%
of Phase III lung cancer clinical trials treat patient-reported outcomes as secondary or exploratory

Prologue: three people, one disease

Brigid

Brigid is 81 and lives on a farm in the west of Ireland, sixty miles from the nearest hospital with a cancer centre. She has had COPD for a decade and heart failure for three years, and she gave up smoking in 1990. Last spring she was diagnosed with lung cancer that had already spread. The treatment her oncologist recommends was approved on the strength of a clinical trial whose participants were, on average, in their early sixties, and which excluded people with heart failure, so nobody can tell her with any confidence how a woman of her age, with her conditions, is likely to fare on it. What she most wants to know is whether the treatment will let her stay at home for the summer, and the clinical trial did not really measure that either.

Eva

Eva is 49, teaches at a primary school in Rotterdam and has never smoked. For most of last year she had a cough that was put down first to a chest infection and then to asthma, and by the time a scan was done her cancer was advanced. It turned out that Eva carried an EGFR mutation, the kind that responds well to targeted therapy and the kind that is most common in women like her who have never smoked. She was never eligible for screening, because screening across Europe is designed for long-term smokers, and the landmark clinical trial on which that design rests enrolled five men for every woman.

Rahim

Rahim is 58, of Bangladeshi heritage, and drives a bus in one of the most deprived boroughs of east London. Men of Bangladeshi heritage have the highest lung cancer rates of any group in England, and people in the most deprived areas are twice as likely to develop the disease and more likely to develop an aggressive form of it. Rahim's cancer is one of the aggressive forms, and there is no clinical trial at his local hospital. The clinical trials at the large academic centre across the city record his age and his tumour type but not his ethnicity, so nobody could say whether men like him are in them at all, and the AI tool that helped assess his scan was trained mostly on patients of European ancestry.

None of these three is a real person. Each is a composite built from the evidence set out in this report, and each stands for tens of thousands of people across the seven European markets we have researched. Between them they meet every one of the seven exclusions this report describes: age and multimorbidity, sex and smoking history, deprivation, ethnicity, geography, lived experience, and the datasets that now train the tools used in their care. They are also, it is worth saying plainly, exactly the kind of people lung cancer most often affects.

This report is about why the evidence that guides their care was built without people like them, what that means for them, and how it could be built differently. We return to each of them as the analysis unfolds, and in the discussion we ask what would have been different had the evidence included them from the start.

About this report, and what we mean by exclusion debt

This is the first in a series examining exclusion debt disease by disease: the cost of building medical evidence without the people it is about. Each edition takes one disease area and asks a single question across the whole evidence chain, from clinical trial design to screening to the datasets that train AI: who is missing, and what does it cost? This edition examines lung cancer across Europe, focusing on seven markets: France, Germany, Ireland, the Netherlands, Portugal, Spain and the UK.

Exclusion debt

Exclusion debt is what accumulates when evidence about a disease is built without the people who have it.

The gap is created at specific moments: the design of a clinical trial, the setting of a screening threshold, the assembly of a dataset. It grows, because every decision built on the partial evidence carries the gap forward and widens it. And it is the people left out who live with the result, through treatments never tested on anyone like them and research they never had the chance to benefit from.

Its defining property is that it cannot be undone after the fact. Once the evidence is built, the people who were missing cannot be added back by analysis. That is what gives the idea its force, and it is why this report ends with a standard for how evidence is collected rather than a plea for better statistics.

We use the term precisely, and we attach it to a protocol, a programme or a dataset, never to a group of people. Each gap in the report is read the same way: what evidence exists, what is missing, what it means for patients, and how severe the gap is. Severity is a judgement rather than a measurement, reflecting the size of the affected population, the strength of the evidence that it is excluded, and the clinical consequence. With that frame in place, the account begins with the disease itself, which is both the deadliest cancer in Europe and one that is changing in ways the evidence has not kept pace with.

A thousand people a day, and a patient who no longer fits the textbook

Lung cancer kills roughly a thousand people in Europe every day. The GLOBOCAN estimates for 2024 put it at about 478,700 new cases a year, the third most common cancer on the continent, and about 369,700 deaths, which makes it the number one cause of cancer death by a wide margin. It kills more people than colorectal cancer, half as many again as breast and prostate cancer combined, and it is the leading cause of cancer death in every one of the seven markets we researched. It has done all this for years with remarkably little of the public attention the second, third and fourth biggest cancer killers attract.[1]

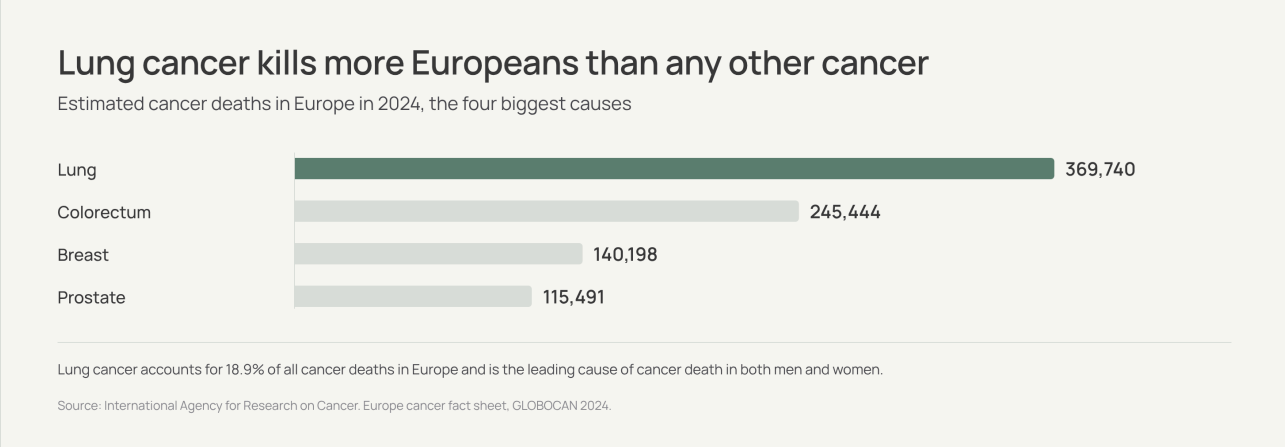


Figure 1. Lung cancer kills more people in Europe than any other cancer. Deaths in 2024, GLOBOCAN estimates.

Survival is poor, and it is uneven in a way that gives away the theme of this report. In the most recent comparable European data, for patients diagnosed between 2010 and 2014, five-year survival in the UK sat at around 13%, which placed the UK 21st of 28 countries on a range that ran from 8% in Bulgaria to 20% in Switzerland, and the same source shows the UK had the lowest uptake of lung cancer medicines among the five largest EU economies.[2] A gap of more than double between the best and worst performing countries is not explained by biology. It is explained by when the disease is found, who gets access to which treatment, and how far the evidence behind those treatments applies to the patients actually sitting in front of clinicians. Most lung cancers are still diagnosed late, when curative treatment is no longer an option, which is why screening and early detection have become the central policy response across Europe.

The reason the evidence question matters so much right now is that the disease itself is moving. The lung cancer of the textbooks, an older man who smoked, is no longer the whole picture, and each of the three shifts under way widens the gap between the disease as it is and the evidence as it was built.

The clearest shift is in people who have never smoked, who now account for somewhere between one in ten and one in four lung cancer cases worldwide, depending on the population. If lung cancer in never-smokers were counted as its own disease it would be among the more common cancers in the world, and it would be the one with the least research, the least screening and the least public understanding. In July 2025 the Sherlock-Lung project, published in *Nature*, sequenced tumours from

871 never-smokers across 28 locations and found that people living with higher levels of fine-particulate air pollution carried more mutations, more TP53 mutations, shorter telomeres and more of a mutational signature previously associated with tobacco smoke, with the effect rising in step with the level of pollution.[3] These are associations rather than proof of cause for any one patient, but they move air pollution from a suspected contributor to a strongly supported one. That reframes who is at risk, which is anyone who breathes polluted air, and in European cities that is most people, not only those with a smoking history. It reframes the stigma too, because a disease long treated as self-inflicted is, for a growing share of patients, environmental.

The second shift follows from the first, because never-smokers skew female. Lung cancer in women is diagnosed younger and more often in people who have never smoked. In a dataset of 129,309 women with lung cancer across seven US states, 11.8% had no history of tobacco use. The biology differs too. EGFR mutations, a targetable driver, account for 74.5% of never-smoking lung adenocarcinomas and are more common in women than in men who never smoked. Adenocarcinoma is rising in perimenopausal women, including smokers, and the most recent review of the subject reports insufficient data on female-specific risk factors such as reproductive and hormonal history.[4] The practical consequence is stark. The group of patients whose cancers are most likely to carry a targetable mutation, and therefore most likely to respond to modern therapy if caught early, is the group least likely to be caught early, because screening is built around the smoking history they do not have.

The third shift is in subtype, and it connects the first two to ethnicity. Adenocarcinoma, the subtype most common in never-smokers, is now the dominant form, and the largest population analysis of lung cancer in England found that women, and people of Indian, Caribbean, Black African, Chinese and other Asian backgrounds, were between one and a half and two and a half times as likely as White patients to be diagnosed with adenocarcinoma rather than squamous cell cancer.[5] Subtype is far from a detail, because it determines which targeted therapies are relevant, which biomarker tests are worth running, and which clinical trials a patient could enter. When subtype shifts across populations, what good care looks like for those populations shifts with it, and the evidence has to shift too.

No shortage of evidence, just the wrong people in it

It would be easy, and wrong, to write about lung cancer as if the evidence were thin. It is not, and any honest account of its gaps has to start by acknowledging what is there. The abundance is also exactly what makes the exclusions so instructive. A disease with little data has an obvious problem, whereas a disease with enormous amounts of data that nonetheless fails the people it affects most has a subtler and more important one.

Start with the clinical trials. Taken across the seven markets, lung cancer is the most frequently researched single disease in the Phase 2, 3 and 4 clinical trials that were recruiting on 3 November 2026, and the UK figures show the scale: 92 of the 1,154 clinical trials recruiting there were in lung cancer, more than for any other single disease, and 80 of the 92 were industry-sponsored.[6] The pipeline runs across targeted therapy, immunotherapy, early-stage treatment and screening, and the last fifteen years have produced real transformation: targeted therapies for EGFR, ALK, ROS1, KRAS G12C and other drivers, and immune checkpoint inhibitors that have changed outcomes for a substantial minority of patients. These advances came from clinical trials, so the question this report asks is not whether clinical trials work but whom they were run on.

Behind the clinical trials sit the molecular and imaging resources that made targeted therapy possible: large genomic atlases, imaging archives and molecular-profiling registries. If you want to understand the biology of a tumour, there is an enormous amount to work with. Those same resources are increasingly the raw material for artificial intelligence, for models that interpret CT scans, predict mutations from histology, or sort patients by risk, which means the composition of these datasets now decides not only what we know but what the next generation of tools will assume.

Then there is screening, where Europe is in the middle of the largest expansion in its history. The EU-funded SOLACE project brought together 37 partners from 15 countries, the Netherlands among them, and ran from April 2023 to March 2026, supporting three national programmes and five implementation pilots across eight countries including France, Germany and Ireland.[7] The UK's Targeted Lung Health Check programme was renamed the NHS Lung Cancer Screening Programme in early 2025 and is being rolled out nationally, with the government's own assessment projecting that at full rollout, expected by 2030, it could find around 9,000 cancers a year earlier.[8,9] Germany's nationwide low-dose CT programme for current and former smokers aged 50 to 75 went live on 1 April 2026.[10] France is at pilot stage. Ireland has no national programme yet, but its first Lung Health Check pilot, run by Beaumont Hospital and RCSI with Irish Cancer Society funding and SOLACE support, began inviting people in May 2025 to generate the evidence its National Screening Advisory Committee will weigh.[11] The Netherlands, whose NELSON clinical trial with Belgium is one of the two landmark pieces of evidence on which European screening policy rests, is taking part in the European 4-IN-THE-LUNG-RUN project on personalised screening intervals, led from Erasmus MC, but has not introduced a national programme.[12,13] Spain and Portugal are earlier still.

What is telling is that SOLACE's own design conceded the problem this report is about. Its three flagship pilots were built around the groups the original screening research left out: women, in France, because they had been under-represented in screening clinical trials; underserved and marginalised

communities, in Hungary, reached with mobile CT units and community outreach; and people with chronic lung disease, in Czechia.[7] Europe's flagship screening project had to create dedicated workstreams for the populations the evidence base was not built on. That is both an encouraging sign and an admission.

Two further pieces of the landscape raise the stakes. Since 12 January 2025, new cancer medicines seeking EU approval have been subject to Joint Clinical Assessment under the EU Health Technology Assessment Regulation, with member states assessing clinical evidence together rather than separately, and lung cancer therapies are among the first through the process. Joint Clinical Assessment scopes its questions in terms of populations, interventions, comparators and outcomes, and member states can ask whether the clinical trial population reflects their own, which turns representativeness from an ethical concern into a question with direct consequences for access and reimbursement across the seven markets.[14] And the European Health Data Space Regulation, in force since March 2025 and applying in stages from 2027, is intended to widen secondary use of health records, registries and linked datasets across borders.[15] The largest of those datasets, the 17.5-million-person English cohort behind the inequality figures in this report, is what makes those figures possible at all, which raises the question of whether the equity variables this report calls for are recorded anywhere else.

Patient-reported outcomes complete the picture. They are routinely collected in lung cancer clinical trials, using standard instruments such as the EORTC QLQ-C30 and the lung-specific QLQ-LC13. The question is not whether they are collected but how they are used, and the next section answers it.

In short, the problem in lung cancer is almost never the volume of data but who is, and is not, inside all that data. Abundance and exclusion are not opposites, and in lung cancer they sit side by side.

The exclusion debt: seven ways the evidence leaves lung cancer patients out

The patient who is too old for the evidence

The typical lung cancer patient is in their seventies and carries other conditions. Median age at diagnosis in England and Wales is 74, and in a real-world Italian cohort 72% of patients had at least one major comorbidity.[16,17] Yet clinical trial populations skew far younger and far fitter, because eligibility rules screen out exactly these patients. A population-based analysis of 677 older adults with metastatic lung cancer, median age 84, found that 53.4% would have been excluded from a typical clinical trial on the basis of other illnesses, poor performance status or both, and 23% met at least two exclusion criteria. The share who looked eligible fell from 66.7% at ages 65 to 74 to 41.8% at 85 and over.[18] The real-world enrolment data tell the same story from the other side: in the Italian cohort, patients who got into clinical trials had a median age of 62, against 70 for the cohort as a whole.[17]

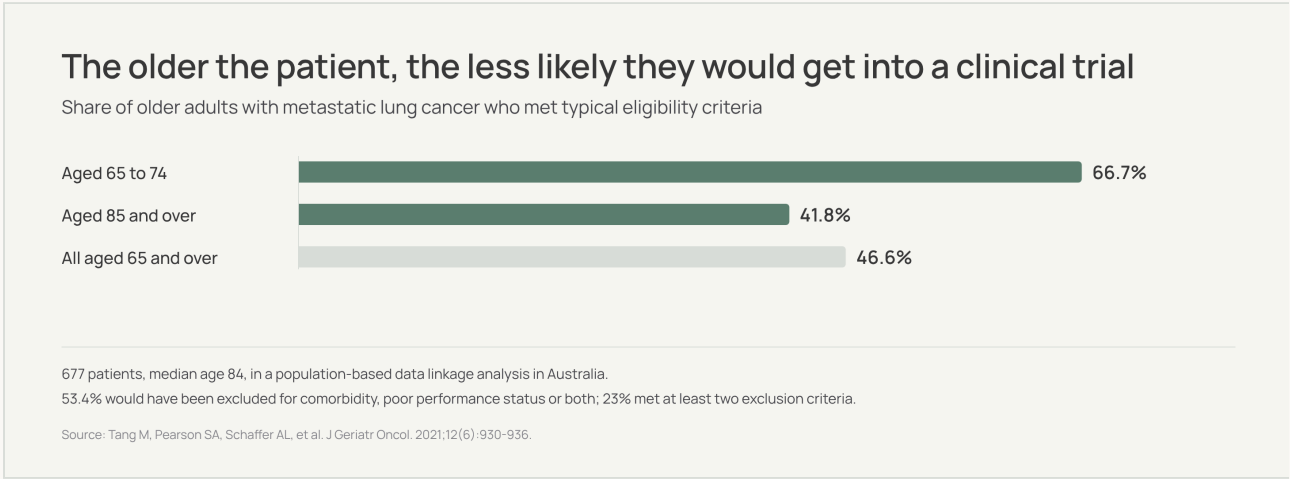


Figure 2. The older the patient, the less likely they would get into a typical clinical trial. Older adults with metastatic lung cancer, Australia.

The mechanism is worth dwelling on because it looks so reasonable. Eligibility criteria exist to protect participants and to produce a clean signal. Excluding patients with poor performance status reduces the risk of harm and the noise of competing causes of death. Excluding kidney or liver impairment avoids confounded pharmacokinetics. Each exclusion is defensible on its own, but their cumulative effect is a clinical trial population that resembles the real one in diagnosis and almost nothing else. The result is that the largest group of lung cancer patients, the old and the multimorbid, receive therapies whose dosing, toxicity and benefit were established in people unlike them, and the clinicians treating them extrapolate because they have no alternative. The consequences run in both directions. Some older patients are over-treated with regimens they cannot tolerate, on the strength of benefit shown in fitter people, while others are under-treated, denied therapies that might help them, because the evidence to support their use in frail patients was never generated. Either way, the decision is being made on evidence about someone else. The older you are, and so the more likely you are to have lung cancer, the

less likely the evidence was built on anyone like you. That is Brigid's position: the evidence behind her treatment describes someone twenty years younger without her heart or lung disease, and her oncologist has no choice but to extrapolate.

> The older you are, and so the more likely you are to have lung cancer, the less likely the evidence was built on anyone like you.

The woman who never smoked, and was never screened

Up to a quarter of lung cancers now occur in people who never smoked, and this group skews female and carries a different, more targetable biology, with EGFR mutations in roughly three quarters of never-smoking adenocarcinomas.[3,4] Air pollution is now strongly linked to the disease. Despite this, lung cancer screening eligibility across Europe is built almost entirely on smoking history and age. The thresholds vary by programme, with Czechia's SOLACE pilot, for example, inviting people aged 55 to 74, but the logic is the same everywhere: those criteria were derived from screening clinical trials dominated by people who smoked, most of them men.[4,7] A never-smoking woman with a high-risk exposure profile is, by design, outside the net, and the research literature still reports insufficient data on the female-specific risk factors that might define her risk.

This is exclusion debt in its purest form, because it was created at the moment the first screening clinical trials chose their populations. Those clinical trials, reasonably, enrolled the highest-risk group they could define, which at the time meant heavy smokers, mostly men. NELSON, the Dutch-Belgian clinical trial that is one of the two pillars of European screening policy, included 13,195 men in its primary analysis and 2,594 women in a subgroup analysis. In women the result pointed towards benefit, with a lung cancer mortality rate ratio of 0.67 at ten years, but the confidence interval ran from 0.38 to 1.14, so the finding was not statistically significant, because too few women were enrolled to tell.[12] The evidence these clinical trials produced justified screening for the group they enrolled and for no other, and everything built since, including the national programmes now scaling across Europe, inherits that boundary. A never-smoking woman told by a national health service that she is not eligible for screening has, in effect, been told she is not at risk, and the scale of the programme makes that more consequential, not less. She is not excluded because anyone decided she should be. She is excluded because nobody ran the clinical trial that would have included her, and no amount of analysis of the existing ones can produce evidence about a population they did not contain. A disease that increasingly affects women who never smoked is still being screened for, and researched, as if it were a male smoker's disease. Eva never had the chance to be screened, so her cancer was found by the persistence of her cough rather than by a scan, and by then it had spread.

> She is excluded because nobody ran the clinical trial that would have included her.

The community with the most disease and the least access

Deprivation produces the steepest gradient of all. The largest analysis of its kind, drawing on the health records of over 17.5 million people in England including more than 84,000 lung cancer cases, found the disease occurred twice as often in the most deprived areas as in the least: 215 cases per 100,000 people a year against 94 in men, and 147 against 62 in women. People from deprived areas who developed lung cancer also had a 35% higher risk of being diagnosed with the more aggressive, poorly differentiated forms of adenocarcinoma.[5]

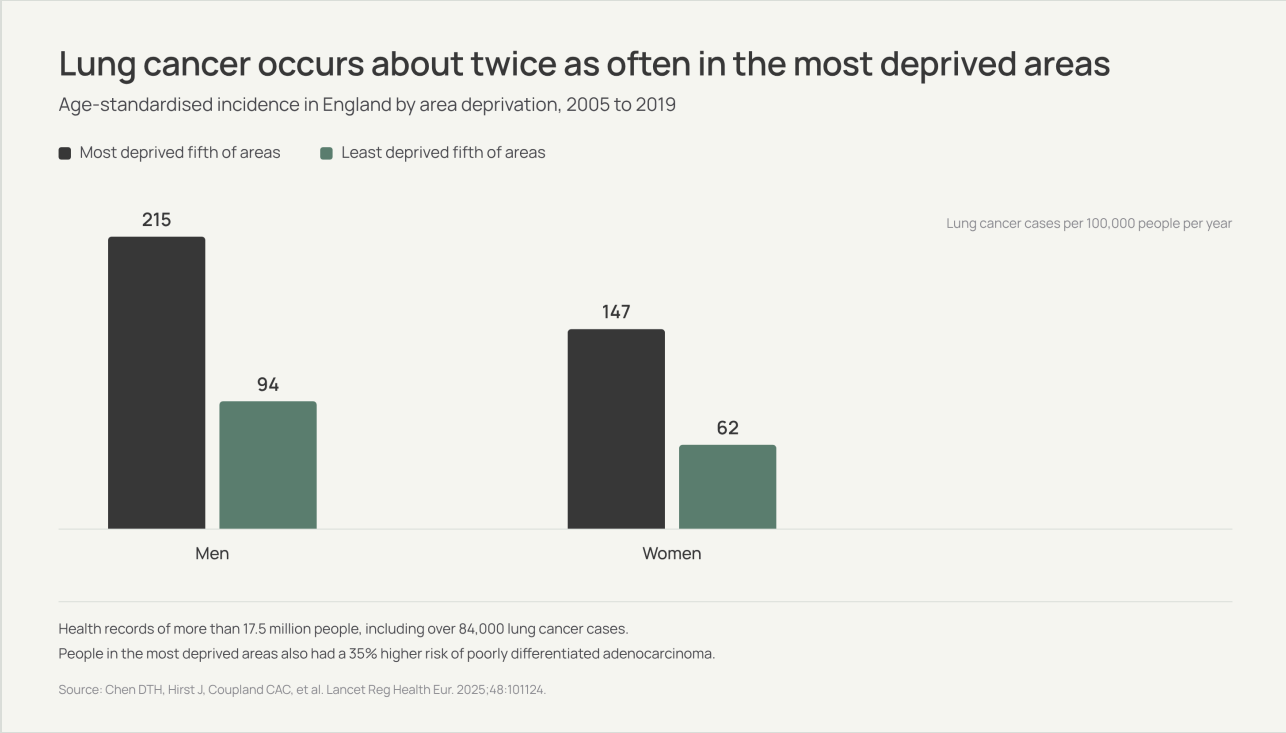


Figure 3. Lung cancer occurs about twice as often in England's most deprived areas. Cases per 100,000 people per year.

The deprivation gradient deepens every other exclusion in this report. Deprived communities smoke more, so they carry more of the traditional disease. They are more exposed to air pollution and occupational hazards, so they carry more of the emerging disease too. They present later, so their cancers are more advanced. And they live furthest from the academic centres where clinical trials run and the clinics where screening is offered. The 35% excess of aggressive disease is not a biological property of poverty. It is the signature of late diagnosis and reduced access, which is to say it is the measurable consequence of the geography gap described below. SOLACE's decision to build a dedicated pilot for underserved communities is an acknowledgement that conventional recruitment, whether to a clinical trial or a screening programme, structurally misses them. The people most likely to get lung cancer, and to get its deadliest forms, are the people least likely to be in the evidence about it. Rahim lives at the sharp end of that gradient, in a borough where the disease is most common and research is least present.

The patient whose ethnicity nobody wrote down

The same English analysis found marked ethnic patterning. Bangladeshi men had the highest lung cancer rates of any group, and women and people of Indian, Caribbean, Black African, Chinese and other Asian backgrounds were considerably more likely than White patients to be diagnosed with

adenocarcinoma.[5] These are the groups most consistently under-represented in oncology clinical trial enrolment, and, tellingly, the real-world enrolment analysis we reviewed did not report ethnicity at all.[17]

That silence is itself the finding, because across much of the European evidence base ethnicity is either not recorded or not reported, which means the degree of under-representation cannot be quantified, which means it cannot be managed, targeted or audited. The adenocarcinoma pattern matters clinically because it points to a higher likelihood of targetable mutations in these groups and therefore a higher cost of missing them. It also points to a deeper problem: if minority patients are under-represented in the genomic atlases, the biomarker thresholds and risk models derived from those atlases may be systematically miscalibrated for them, and nobody will be able to tell. The shift in who gets lung cancer is running ahead of who the evidence describes, and in many datasets we cannot even measure the gap. Whether men like Rahim are in the clinical trials at the academic centre across his city is a question that nobody running those clinical trials can currently answer.

The patient who lives in the wrong place

This dimension is rarely discussed and, in the seven markets, may be the most practically important. In a real-world analysis across four Italian cancer centres, 397 lung cancer patients started a new line of treatment over six months in 2021. Of the 374 who were candidates for active treatment, 58, or 15%, were enrolled in a clinical trial. Among the 309 who went on to standard treatment instead, the single biggest obstacle, recorded for 54%, was simply that no clinical trial was open at their own or a nearby hospital. Not meeting the eligibility criteria accounted for 12%, poor performance status for 7%, comorbidities for 3% and patient refusal for 2%.[17] Clinical trial access is concentrated in large urban academic centres, smaller markets and rural regions see fewer clinical trials, and Ireland, the Netherlands and Portugal have not yet introduced national screening programmes.

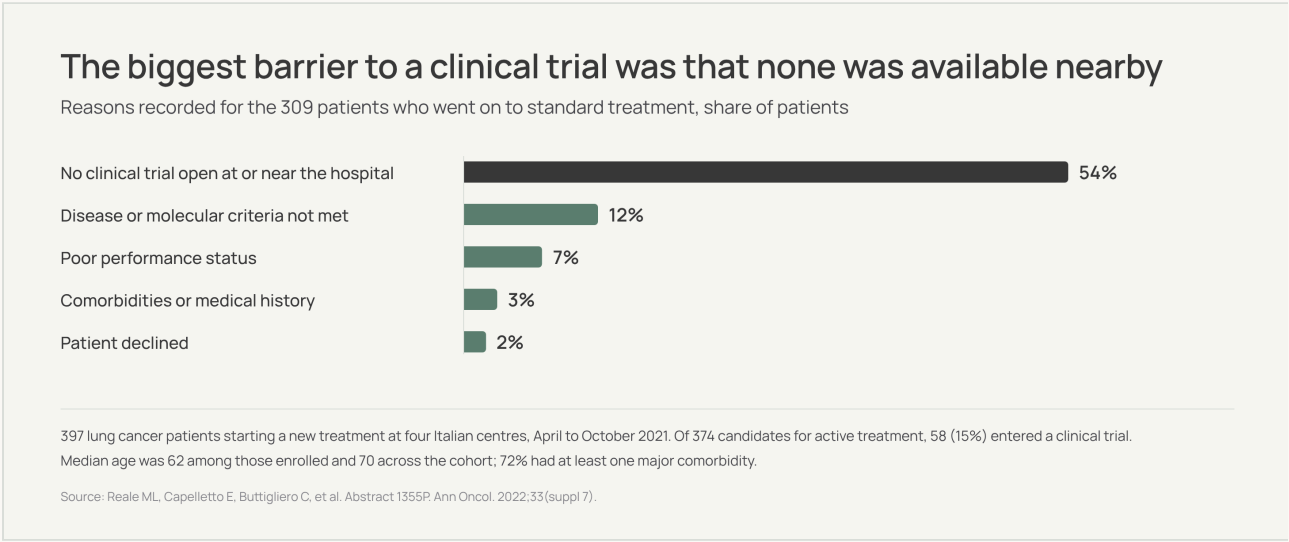


Figure 4. Why lung cancer patients on standard treatment were not in a clinical trial. Four Italian centres, 2021.

The implication is uncomfortable for a report about representativeness. Even if every eligibility criterion were rewritten tomorrow, most lung cancer patients would still not be in the evidence, because the evidence is physically somewhere else. Geography also feeds every other exclusion: rural populations are older, smaller markets have fewer academic centres and fewer sponsors competing to open sites, and patients without the means to travel are disproportionately deprived. For the seven markets this

creates a two-tier picture. Germany, France, the Netherlands, Spain and the UK host dense clinical trial activity in their major cities, while Ireland and Portugal, and the rural regions of all seven, are structurally under-represented in evidence that will nonetheless be used to decide their care. Where you live determines whether you can be in the evidence at all. For Brigid, sixty miles from the nearest cancer centre in a country with no national screening programme, geography had decided the matter long before any eligibility criterion was applied.

> Where you live determines whether you can be in the evidence at all.

The experience that was collected and then set aside

Lung cancer clinical trials collect patient-reported outcomes, but they treat them as an afterthought. A review of 61 Phase III clinical trials of first-line treatment for advanced non-small cell lung cancer, covering 20,597 patients and published between 2014 and 2023, found patient-reported outcomes were a primary endpoint in just seven of them, 11%, and a secondary or exploratory endpoint in the other 89%.[19] A 2026 survey of 252 randomised clinical trials in non-small cell lung cancer found that 70% reported missing patient-reported outcome data, but only 45% said how they had handled it statistically, and among the clinical trials where these outcomes were a secondary endpoint the figure fell to 35%.[20]

This matters more in lung cancer than in almost any other disease, for two reasons. The first is that for most patients, treatment is not curative. It is about time and the quality of that time, and the trade-off between a few months of survival and the burden of toxicity is exactly the question patient-reported outcomes exist to answer. A clinical trial that measures survival rigorously and quality of life carelessly has measured the thing patients care about least carefully. The second is that lived experience is the only part of the evidence that can capture the barriers this report documents: why an older patient declines a clinical trial, why a woman never considered herself at risk, why a patient in a rural area was not offered screening, and none of those answers is in the genomic data. Because the patients in current clinical trials are already younger, fitter and more urban than the real population, even the lived experience that is captured belongs to the included, not the excluded. The evidence about what treatment actually feels like, what trade-offs patients would accept, and what matters to them is captured, then largely set aside, and captured from the wrong people. Brigid's question, whether the treatment will let her spend the summer at home, is exactly the kind of question the evidence was never designed to answer.

The dataset that teaches the machine one ancestry

The genomic and imaging atlases that power modern lung cancer research, and increasingly the AI built on top of them, carry their own skew. A 2026 review mapping the data available for AI in women's health, including cancer registries and biobanks, reported that cancer datasets remain heavily weighted towards populations of European ancestry, limiting what can be learned about other groups and rarer tumour subtypes.[21] These datasets are the training material for diagnostic and biomarker AI, and a model learns the population it is shown.

The danger is not only that AI tools will be less accurate for under-represented groups. It is that the inaccuracy will be invisible and will carry the authority of a machine. A clinician who extrapolates from a clinical trial knows they are extrapolating, whereas a model trained on one ancestry and deployed on another does not announce the gap but simply produces a confident output. Because ethnicity is so often unrecorded in the underlying data, the audit that would reveal the bias cannot be run. This harm also looks forward rather than back: the clinical trial exclusions of the last twenty years affected the patients of those years, but the dataset exclusions of today will affect every patient assessed by a model trained on them, for as long as that model is in use. Show a model one ancestry and it gets quietly, confidently worse for everyone else, and you often cannot measure how much worse, because the data to check against is not there either. The tool that assessed Rahim's scan may be perfectly accurate for him, or it may not be, and with the data as it stands nobody can say which.

Seen one at a time, each of these exclusions has a reasonable explanation. Seen together, as the next section sets out, they form a pattern in which the same people are left out at every stage, and in which Brigid, Eva and Rahim are each excluded several times over.

One gap, not seven: why the exclusions pile up

The table reads each dimension of the exclusion debt: what evidence exists, what is missing, what it means for patients, and how severe the gap is.

Dimension	What exists	What's missing	What it means for patients	Severity
Age and comorbidity	Deep clinical trial pipeline; geriatric oncology literature; real-world cohorts	Clinical trial populations around eight years younger than patients; more than half of older metastatic patients ineligible; comorbid patients screened out	Treatments prescribed to patients the evidence never covered; clinicians guessing at the highest-risk moment	High
Women and never-smokers	Growing biology literature on EGFR and hormones; air-pollution evidence; SOLACE women's pilot	Screening evidence built on male smokers, with NELSON 84% men; female-specific risk factors under-researched; never-smokers outside eligibility	Later diagnosis for a rising group; targetable cancers found too late	High
Deprivation	Strong population evidence of the gradient from a 17.5-million-person cohort	Deprived communities far from clinical trials and screening; no routine linkage of deprivation to clinical trial populations	Highest incidence and most aggressive disease, slowest access to new options	High
Ethnicity	Population evidence of distinct patterns: Bangladeshi men; adenocarcinoma in several groups	Persistent under-enrolment; ethnicity often not recorded in clinical trial or enrolment data	Gap can't be measured, so can't be managed; biology of minority patients under-researched	High
Geography and access	Dense clinical trial activity in urban academic centres; EU screening expansion	54% of patients on standard treatment had no clinical trial nearby; about 15% enrol; no national screening yet in Ireland, the Netherlands or Portugal	Postcode determines inclusion in the evidence; smaller markets systematically under-represented	High
Lived experience	Patient-reported outcomes collected with standard instruments in most clinical trials	Secondary or exploratory in 89%; missing data often unreported; captured from already-included patients	Decisions made without knowing what matters to patients or what trade-offs they'd accept	Medium to high
Datasets and AI	Large genomic and imaging atlases	Heavy skew to European ancestry; rarer subtypes under-represented; equity variables often absent	Bias trained into diagnostic and biomarker tools, unmeasurable because audit variables are missing	High, rising

It is tempting to read the seven rows as seven separate problems with seven separate fixes, but they are closely connected. The older patient is also more likely to be multimorbid, less likely to live near an academic centre, and more likely to be in a deprived area. The never-smoking woman from a minority ethnic background is excluded three times over: by screening criteria built on smokers, by clinical trial enrolment that under-represents her ethnicity, and by datasets that under-represent her ancestry.

Exclusion accumulates across dimensions, not just over time, and that has a practical consequence for anyone trying to fix it. A programme that improves age representation but recruits only from major urban centres will enrol older patients who are wealthier and whiter than the older patients who actually have the disease. Representativeness has to be pursued across all the dimensions at once, or it produces a new, subtler skew.

The correlation leads to a second pattern: the most consequential gaps are the least measured. Ethnicity is frequently unrecorded, deprivation is rarely linked to clinical trial populations, and patient-reported outcome data goes missing without anyone saying how it was handled, which matters because nobody can audit a fairness that was never recorded. In several dimensions the first problem is not that the evidence is biased but that nobody can tell how biased it is, and that is not an accident of the data but a consequence of what was chosen to be collected. The variables that would reveal exclusion are precisely the ones that looked unnecessary to designers who were not asking the question. Measurement is not the fix, but nothing can be fixed without it, which is why the standard in the discussion places so much weight on what must be captured.

> Nobody can audit a fairness that was never recorded.

The third pattern is that the system is already conceding the point. SOLACE's pilots for women and underserved groups, the growing geriatric oncology literature, and the population questions written into Joint Clinical Assessment scoping are all acknowledgements, from inside the system, that the original evidence left people out. The question is no longer whether the gaps exist. It is whether the response arrives as a patch at the end, a dedicated pilot here and a subgroup analysis there, or as a standard at the start, built into how lung cancer evidence is designed before it is collected. The first approach is how the system has always responded, and it is why the gaps persist. The second is what this report argues for.

Which raises the obvious question of why, in the most actively researched disease in the clinical trials we track, these exclusions have survived decades of good intentions. Four forces explain most of it, and they feed each other. Speed comes first: clinical trials are expensive and sponsors are rewarded for enrolling quickly, and the fastest way to enrol is to recruit from the sites, populations and criteria that worked last time, which reproduces the existing skew. Speed is helped along by statistical cleanliness, because homogeneous populations produce clearer signals with fewer participants, so there is a permanent methodological incentive to leave out the complicated patient. Both are reinforced by site economics, since clinical trials run where the infrastructure already is, which concentrates them in large urban centres in large markets and leaves smaller markets and rural regions structurally under-served. And underneath all three sits stigma: because lung cancer has been framed as self-inflicted, the advocacy, philanthropy and political pressure that drove representativeness improvements in other cancers have been weaker here.

The funding pattern shows the effect of that stigma. In Australia, lung cancer was found to receive about 4.8% of cancer research funding while accounting for about 20% of the cancer burden, where breast cancer received around 30% of funding for about 5% of the burden.[22] The pattern repeats wherever it has been measured: an analysis of UK funding found lung cancer caused 26% of cancer deaths and received 6% of National Cancer Research Institute funding, and the equivalent US analysis found 32% of deaths and 10% of funding.[23,24] One Australian advocate, asked why lung cancer patients

do not organise the way breast cancer patients have, put it in three words: "We die fast."[25] As the never-smoker share rises and the air-pollution evidence hardens, the stigma becomes not just unfair but factually wrong, because a growing proportion of the people dying of lung cancer did nothing the stigma blames them for. None of these four forces is malicious, and all of them are rational for the actor in question, which is exactly why they will not correct themselves and why a standard, applied from outside the individual decision, is needed.

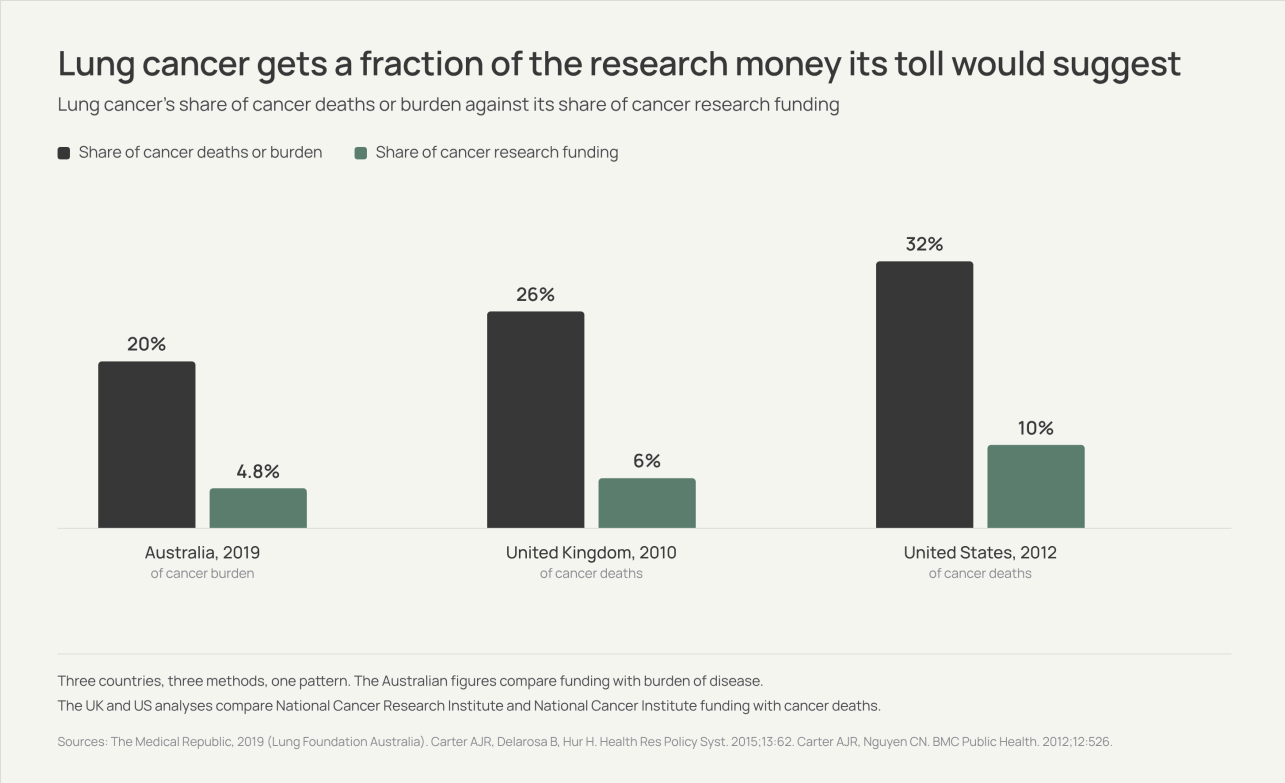


Figure 5. Lung cancer's share of cancer deaths or burden against its share of cancer research funding, three countries.

If the forces behind the gaps are rational and self-reinforcing, the question is what that standard has to contain, and the next section sets it out.

The standard: who must be in the evidence, what must be captured, and how

The one thing that cannot be done about exclusion debt is to put it right after the fact. No algorithm, however clever, can include an 84-year-old who was never eligible for the clinical trial, and no analysis, however sharp, can recover the never-smoking woman who was never screened, the deprived patient who was never near a site, or the ethnicity that was never recorded. A population not included when the evidence is built can only be included, or not, at the start. So the fix is not a better analysis but a standard for how lung cancer evidence is collected in the first place: who must be in it, what must be captured about them, and how.

Who must be in it

The principle is simple to state and demanding to apply: the evidence base should be built to reflect the people who actually get lung cancer, deliberately and in proportion, not whoever is easiest to enrol. In lung cancer that means six populations in particular, each of which the evidence shows to be systematically missing.

Older patients, including those aged 75 and over, and patients with major comorbidities or reduced performance status are the largest of these groups and the most routinely excluded. Including them is not a matter of relaxing standards but of recognising that they are the standard patient, which means eligibility broad enough to admit the multimorbid majority, with safety monitoring proportionate to the added risk rather than exclusion as the default response to it. Women, and never-smokers of both sexes including people with high air-pollution or occupational exposure, are the fastest-growing share of the disease and the share with the most targetable biology, and reaching them takes more than open eligibility: it takes actively finding people who do not consider themselves at risk because everything they have been told about lung cancer concerns smoking, and screening cohorts that define risk by exposure as well as by pack-years.

People living in the most deprived areas carry twice the incidence and the most aggressive disease, so reaching them means recruiting where they are, through the community settings, primary care and outreach approaches that SOLACE piloted, rather than expecting them to find their way to an academic centre. Minority ethnic communities need representation at the granularity the disease demands, because the English data show that the Bangladeshi, Indian, Caribbean, Black African and Chinese patterns are not interchangeable and a single "non-white" category would obscure exactly the differences in incidence and subtype that matter. People in rural areas and in smaller markets without major academic centres, which in the seven markets means much of Ireland and Portugal and the rural regions of all seven, need the evidence to come to them through decentralised designs, regional sites and remote participation. And people with language, literacy or digital-access barriers, who are missed by conventional recruitment almost by definition, need materials, consent processes and participation routes designed for them rather than adapted afterwards.

Underlying all of this is a distinction the evidence system often gets wrong. Representativeness is a property of the whole evidence base, not of any individual participant. No single older patient represents older patients; no single never-smoking woman represents never-smoking women. The aim

is reach and scale across these groups, so that the dataset as a whole carries the weight of representing the population rather than a handful of participants asked to stand in for everyone like them. That is a question of recruitment strategy and infrastructure, and it is the reason a standing, continuously maintained community of people affected by the disease matters so much more than any single enrolment effort.

What must be captured

Representativeness is unmeasurable, and therefore unmanageable, unless the right variables are recorded. Every lung cancer evidence programme, whether a clinical trial, a screening cohort, a registry or a dataset intended for AI, should capture a defined set of variables as standard.

- Age, recorded alongside performance status and a measure of frailty rather than reduced to a cut-off, because two patients aged 78 can differ more from each other than either does from a patient of 60, and treatment decisions depend on function, not years.
- Sex, together with the sex-specific history current research identifies as missing: reproductive history, hormonal exposure and menopausal status.
- Smoking status with never-smoker as a distinct category rather than a residual, accompanied by exposure history covering air pollution, secondhand smoke and occupational exposure. A dataset that records only pack-years is recording the disease of thirty years ago.
- Ethnicity, self-identified and at a useful level of detail, as a standard field in every programme. Its absence is the single most common reason the gaps in this report cannot be measured.
- Deprivation, through a recognised area-level index linked to the participant, so that the steepest gradient in the disease can be seen in the evidence rather than only in the population statistics.
- Geography, including rurality and the travel time to the nearest clinical trial site and screening centre, the variables that reveal the access gap and are almost never recorded.
- Comorbidities and concurrent medications in full, because they determine how a therapy behaves in the multimorbid majority and because their absence is the main mechanism by which older patients are excluded.
- Language, literacy and digital access, because they predict who is missed by conventional recruitment and whether patient-reported instruments are usable by the people completing them.
- Lived experience as a first-class component of the evidence: what matters to the patient, which trade-offs they find acceptable, and patient-reported outcomes collected with a pre-specified hypothesis, a plan for missing data and a commitment to subgroup analysis.
- Consent status, including its scope and whether it is revocable, because consented, re-contactable participants are the foundation of the standing community described next.

Several of these, notably ethnicity, deprivation, exposure and sex-specific history, are precisely the variables most often absent today, and their absence is not a technical oversight but a reflection of the questions the evidence was designed to answer.

How it must be collected

Knowing who should be in the evidence and what should be recorded about them is necessary but not sufficient, because the gaps persist through how evidence is collected. Representativeness has to be designed in before the protocol is finalised, not patched in after. Eligibility criteria, endpoints, site selection and recruitment strategy are set at the design stage while they are still fluid, and that is the only point at which patient input and a representativeness check can change them. A sponsor who convenes patients to review a finished protocol has done something valuable and something largely cosmetic. The same input three months earlier could have changed who the clinical trial enrols.

Designing it in early is only possible if collection is continuous rather than organised afresh for each clinical trial. Rebuilding a patient community from scratch every time is slow, expensive and structurally biased towards the already-connected, because the patients who are easy to find quickly are, by definition, the ones already in contact with the system. A standing, engaged community of people affected by lung cancer, maintained between clinical trials, consented in advance and told what happened to their contributions, is what makes representative recruitment feasible at the speed clinical trials actually run. It converts the hardest part of representativeness, reaching the people who are never reached, from a per-protocol emergency into infrastructure that is already in place when the work begins. This is the single most important structural change the evidence system could make, and it is the one that almost no sponsor or programme has made.

> A standing community of people affected by lung cancer is the single most important structural change the evidence system could make.

A standing community only works if participation rests on explicit, revocable consent and clear ownership, with people taking part on terms they can see and withdraw from, and the community retaining a stake in how its evidence is used. That is an ethical requirement, but it is also the practical basis of trust, and the communities most missing from lung cancer evidence, deprived, minority ethnic and rural, are often the communities with the best historical reasons to distrust research. Trust cannot be bought at the point of recruitment. It has to be built over time, through transparency about use and control over participation, which is one more reason continuity matters.

Two further principles follow. The evidence must be built from real people, not synthetic proxies, for the reasons set out above: for every gap in this report the synthetic route produces a confident imitation of inclusion rather than inclusion itself, and hides the gap it claims to fill. And the evidence should be linked across the lifecycle and auditable for equity, so that what is learned about the excluded in clinical trial design is not rediscovered, slowly, in screening policy and then again in Joint Clinical Assessment. Every dataset should be able to answer, with its own variables, the question "who is missing?", and should publish the answer, including the groups too small to report on. Naming the gap is the first step to closing it, and a programme that cannot say who is absent from its own data cannot credibly claim to represent anyone.

What good looks like

A lung cancer evidence programme meeting this standard would be able to publish a comparison of its clinical trial or cohort population against the real patient population on age, sex, deprivation and ethnicity, and would have tested its eligibility criteria against that real population before finalising them, justifying each exclusion that remained.

It would show never-smokers and women represented in proportion to their rising share of the disease, not as an afterthought subgroup. It would record ethnicity and deprivation for every participant as a matter of course. Its sites and recruitment would reach rural areas and smaller markets, and it would be able to show that they had. Its patient-reported outcomes would be pre-specified with a hypothesis, analysed by subgroup and visibly used in decisions. People affected by the disease would have shaped its protocol, endpoints and, where relevant, screening criteria before they were finalised, and the community that provided the evidence would be standing, consented and told what happened to it.

Most lung cancer evidence today could not demonstrate most of this. That is not a criticism of any individual sponsor, programme or investigator. It is a description of a system that was never asked to. And every element of it is achievable with current methods. What is missing is not the means but the standard.

What would have been different

Applied to the three people this report has followed, the standard becomes concrete. Had the clinical trials behind Brigid's treatment benchmarked their eligibility against the real population of lung cancer patients, they would have included people in their eighties with heart and lung disease, and her oncologist would have evidence about someone like her rather than an extrapolation. Had a standing community of patients shaped how those clinical trials were designed, the question she most wants answered, about time at home rather than months of survival alone, would have been a pre-specified outcome rather than an afterthought. Had decentralised sites and remote participation been designed in, sixty miles would not have been the end of the conversation.

Had the screening evidence that shaped Europe's programmes included never-smokers and enrolled women in proportion, Eva might have been offered a scan on the basis of her risk rather than her smoking history, and her EGFR-driven cancer, the kind that responds best when found early, might have been found early. Had the variables that define her risk, her exposure history and her reproductive and hormonal history, been captured as standard, the research that could tell women like her whether they are at risk would already be under way.

Had the clinical trials at the academic centre recorded ethnicity and deprivation as standard, and recruited through the community settings where Rahim actually lives, anyone could see whether men like him are represented and act if they are not. Had the dataset behind the AI tool that assessed his scan reported its composition and been validated in people of his ancestry, his clinicians would know how far to trust it.

None of this requires new science, since every element uses methods that already exist. What it requires is a decision, made before the evidence is built, that people like Brigid, Eva and Rahim belong in it.

What each part of the system should do

The standard translates into specific actions for each part of the evidence system, and the actions reinforce one another. A sponsor's incentive to recruit representatively is strongest when HTA bodies ask about representativeness, and a screening programme's ability to reach underserved groups is greatest when a standing community already exists.

For clinical trial sponsors, the first step is to benchmark every lung cancer protocol's planned population against the real-world age, comorbidity, sex, deprivation and ethnic profile before finalising eligibility, and to justify each exclusion in writing. This is a modest discipline with large consequences, because it forces the gap to be seen at the one moment it can still be closed. From there the rest follows: ethnicity and deprivation recorded as standard variables, patient-reported outcome hypotheses and subgroup analyses pre-specified rather than left exploratory, recruitment built on a standing, representative community rather than a per-protocol scramble, and site selection that extends beyond major academic centres with specific attention to smaller markets and rural regions. Sponsors who do this will find that Joint Clinical Assessment, which now asks population questions formally, becomes an asset rather than an obstacle.

For screening programmes, the priority as Europe's programmes scale is to revisit eligibility criteria that rest on smoking history alone. The never-smoker share of the disease and the air-pollution evidence together make a smoking-only threshold an increasingly poor proxy for risk. Programmes should build explicit pathways for never-smokers with high exposure and for women, should adopt the community outreach, mobile units and family-doctor referral that SOLACE piloted as core design rather than add-ons, and should record and publish uptake by deprivation and ethnicity, so that an equitable programme can be told apart from one that merely has equitable intentions.

For HTA bodies and regulators, the most powerful lever is disclosure. Sponsors should be required to report the representativeness of clinical trial populations against the real-world population, including who is missing, and unexplained gaps should be treated as an evidence limitation in the same way that a missing comparator or inadequate follow-up would be. Pre-specified patient-reported outcomes and evidence of patient input at the design stage should carry weight in assessment, and the inclusion of equity variables in submitted datasets should be encouraged and, over time, required, because without them the question of representativeness cannot be answered at all.

For data custodians and AI developers, the obligation is one of transparency and restraint, starting with training datasets that report their ancestry and demographic composition. The equity variables that allow fairness audits should be captured and retained. Diagnostic and biomarker models should not be deployed in populations absent from the training data without validation in those populations, and the temptation to fill gaps with synthetic substitutes for under-represented groups should be resisted in favour of real, consented, representative data, however much slower that is to assemble.

For patient organisations and advocates, the opportunity is to press for this standard to be adopted as a condition of funding and approval, and to replace the smoker's stigma with the never-smoker reality in how lung cancer is talked about and funded. The disease's advocacy gap is itself a form of exclusion, and closing it is part of closing the evidence gap.

The limits of this analysis

This report is a synthesis of published evidence, not primary research, and it carries the limits of its sources. The inequality figures come from England and may not transfer in detail to the other six markets, although the direction of the deprivation gradient is consistent across European data. The clinical trial enrolment figures come from four Italian centres over six months and indicate a pattern rather than a European rate. The eligibility figures come from Australia. The research-funding comparison draws on an Australian analysis from 2019 and UK and US analyses that are a decade old; the pattern is consistent across all three, but the current figures will differ. The UK survival and medicine-uptake comparisons are from the most recent comparable European datasets and are dated as such. The count of active clinical trials is our own weekly export of clinicaltrials.gov, filtered to Phase 2 to 4 and recruiting; group leukaemia, lymphoma and myeloma together and haematological cancers would come out ahead in the UK, which is why "most frequently researched" is a statement about single diseases. The dataset-skew finding comes from a review of women's health data rather than lung cancer specifically. Ethnicity data is thin across European sources, which is itself one of the report's findings but also a constraint on it. The severity ratings are the authors' judgement. And the report does not attempt to quantify the aggregate harm in lives or years of life lost, because that would require modelling assumptions this edition has chosen not to make. Where the evidence is thin, the honest conclusion is not that the gap is small but that it has not been measured, which is the point.

Conclusion: a gap that can only be closed at the start

Lung cancer is the deadliest cancer in Europe and the most frequently researched single disease in the clinical trials we track, and these two facts sit alongside a third: the evidence that guides its treatment was built, overwhelmingly, around patients unlike the ones who have it. The old, the multimorbid, the deprived, the minority ethnic, the rural, the never-smoking woman whose cancer may have been caused by the air she breathed: each is more likely to get the disease and less likely to be in the evidence about it, and the gaps reinforce one another so that the patient who is excluded on one dimension is usually excluded on several.

This is exclusion debt, and its defining feature is that it cannot be undone after the fact, because no analysis recovers a population that was never captured. The response has to be a standard for how lung cancer evidence is collected in the first place: who must be in it, what must be recorded about them, and how, with representativeness designed in before protocols are finalised, built on a standing and consented community rather than a per-protocol scramble, made from real people rather than synthetic substitutes, and auditable with its own variables for who is missing.

The moment for change is unusually favourable, because Europe is scaling national screening programmes whose eligibility criteria will define risk for a generation, Joint Clinical Assessment has made the representativeness of clinical trial populations a formal question with consequences for access in every member state, and the European Health Data Space will multiply the scale of the datasets that train the next generation of tools. Each of these will carry forward whatever evidence base it inherits. Built on the current one, they will entrench the exclusions this report describes at national and continental scale, whereas built on the standard above, they could do more to close the gap than any single clinical trial.

Brigid, Eva and Rahim are composites, but the people they stand for are not. Every year, across the seven markets we have researched, tens of thousands of people like them are diagnosed with lung cancer and treated on the strength of evidence that was built without them. Some will be treated well regardless, while many will be treated on extrapolation, found too late, or never offered the chance to take part in research that might have helped them. They are the reason the standard matters, and they are the measure by which any change should be judged.

The deadliest cancer in Europe deserves an evidence base built around the people it actually kills, and at present it does not have one. That gap is the exclusion debt, and it can be closed, but only at the start.

The exclusion debt at a glance

Who carries the burden	What the evidence shows	Why it matters
Older and multimorbid patients	About 53% of older metastatic patients excluded from typical clinical trials; enrolled median age 62 against 70	Treatments used on patients the evidence never covered

Who carries the burden	What the evidence shows	Why it matters
Women and never-smokers	Up to 25% of cases are never-smokers; EGFR drives about 75% of never-smoker adenocarcinoma; screening evidence built on male smokers, with NELSON 84% men	A rising, targetable group found too late
Deprived communities	Twice the incidence; 35% higher risk of aggressive adenocarcinoma	Highest burden, furthest from clinical trials and screening
Ethnic minorities	Distinct incidence and subtype patterns; persistent under-enrolment; often unrecorded	Gap can't be measured, so can't be managed
Geography and access	54% of those on standard treatment had no clinical trial nearby; about 15% enrol; no national screening yet in Ireland, the Netherlands or Portugal	Postcode decides inclusion in the evidence
Lived experience	Patient-reported outcomes secondary or exploratory in 89% of Phase III clinical trials; missing data handling stated in 45%	Decisions made without knowing what matters to patients
The datasets and AI	Cancer datasets heavily weighted to European ancestry	Bias trained into the next generation of tools

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About Unwritten Health

Unwritten Health is a UK patient experience data and patient recruitment company. More than 13,000 people living with long-term conditions have joined us, and we go back to the same people over time, as often as they choose to answer. We pay them for their time, never for joining a clinical trial, and every member hears what changed because of them. That lets us do two things for the people who build medical evidence: ask the people who live with a condition before a protocol, consent form or product is final, and follow every referral through, so a sponsor knows which ones went nowhere and why.

The Exclusion Debt Report is published monthly by Unwritten Health. Figures are drawn from the published sources listed in the references; where a figure comes from a single country or analysis, the text says so.

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