Achieving dynamic efficiency through pharmaceutical policy: the balance of push and pull incentives

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Abstract

Achieving dynamic efficiency in pharmaceutical policy requires considering pull (expected reward) and push (publicly funded R&D) incentives simultaneously. We develop a general framework with a structural model founded on how causal estimates of the effect of pull and push incentives on the quantity of innovation have been estimated and where the dynamics are captured in the reduced form of causal estimates of the pull and push elasticity of innovation. We illustrate the welfare response to the combination of both policy levers based on a parameterisation that reflects the UK context, but is generalisable to other health care systems and a range of welfare arguments. We find that a rebalancing is required, with more investment in push and less in pull. This rebalancing to achieve dynamic efficiency can be revenue neutral for manufacturers and neutral for public expenditure on new medicines, while increasing innovation and net population health. Relying solely on reducing the reward to manufacturers requires the health system to accept a reduction in innovation and manufacturers to accept reduced revenues. However, policy which combines less pull with more push investment delivers greater net health gains, increased innovation, and sustainable revenues, with other possible spillover benefits. We demonstrate that contemporary policy debates that frame the public research contribution as 'paying-twice' for innovation are conceptually unhelpful and do not guide policy to dynamically efficient solutions. Rather than regarding public research as a subsidised investment that must be recovered from manufacturers' rewards, health systems should recognise that signalling expected rewards and investment in public research are not antagonistic but complementary policy levers. Although, for ease of exposition, welfare is initially defined as a public health objective of a social planner, we examine the implications of considering other welfare arguments including producer surplus.

Keywords: Pharmaceutical Innovation; Push and Pull Incentives; Dynamic Efficiency; Population Health
JEL: O31, I11, I13, I18

1. Introduction

Determining the optimal payment for new drugs poses a challenge for health policy, public finances, and industrial strategy. To balance the affordability of healthcare with the need to encourage pharmaceutical innovation, drug-pricing mechanisms need to reward therapeutic advances. Governments grant patent protection and market exclusivity to protect new treatments from market competition, which enables manufacturers to charge prices above the costs of manufacture and supply. While price-setting mechanisms vary across countries, the overarching economic design is to incentivise investment in private research and development (R&D) by guaranteeing returns on successful innovations. The rationale for using this policy lever is that *pull incentives* (e.g., market rewards, patents, sales, payment policies (Kyle, 2020)) are the main driver of pharmaceutical innovation, as they align private R&D investments with potential market demand (Acemoglu & Linn, 2004; Dubois et al., 2015).

Although the majority of the empirical literature has sort to quantify how innovation responds to these pull incentives (Acemoglu & Linn, 2004; Cerda, 2007; Blume-Kohout & Sood, 2013; Dubois et al., 2015; Dubois, 2025; Maini et al., 2026; Filson et al., 2025) fewer studies have evaluated the joint effect of incorporating both pull and *push incentives* (e.g., R&D-related public investments, tax credits, and subsidies). Unlike pull mechanisms, push incentives aim to stimulate innovation by reducing upfront development costs (Kyle, 2020; Oliver et al., 2024). Public R&D funding has also been found to expand the scientific knowledge frontier (Sampat, 2009; Sampat & Lichtenberg, 2011; Blume-Kohout & Sood, 2013), crowd in private investment (Sussex et al., 2016; Toole, 2007; Ward & Dranove, 1995), and yield long-run societal returns (Azoulay et al., 2019). Yet, causal evidence on how public R&D interacts with pull incentives to generate pharmaceutical innovation remains limited.

Previous economic models and frameworks have concluded that as manufacturers face the full cost of R&D, they should also be rewarded with the full benefits to achieve dynamic efficiency (Nordhaus, 1969; Jena & Philipson, 2008; Vernon et al., 2009; Danzon et al., 2015; Lakdawalla, 2018; Basu, 2026). However, these models assume that private manufacturers bear the entire financial and scientific burden of drug discovery, so do not fully reflect the

role of public R&D funding. They often implicitly assume that the level of Intellectual Property Protection (IPP) is already optimal (Basu, 2026) and generally do not recognise the role of health and other opportunity costs. They often fail to account for the social costs of bringing an innovation to market when considering how much producer surplus might be retained and how it should be valued (Jena & Philipson, 2008; Vernon et al., 2009; Danzon et al., 2015; Lakdawalla, 2018; Basu, 2026). Similarly, although the Congressional Budget Office (CBO) of the United States (US) has considered the impact of publicly funded R&D on innovation (Swagel, 2025), they have not incorporated this within their structural model of pharmaceutical innovation (Adams, 2021).

The omission of public R&D is particularly problematic given how the public and private sectors interact. Early models of innovation described a strictly linear process, assuming the public sector generated basic research, which the private sector subsequently applied to develop and commercialise new drugs (Cockburn & Henderson, 2000; Rejon-Parrilla et al., 2025). Recent frameworks, however, describe innovation as an interactive and highly complex process. Under this contemporaneous model, there is no one-way path: knowledge, materials, and human capital flow bidirectionally between sectors (Rejon-Parrilla et al., 2025). To formally capture this complexity, pharmaceutical innovation in this paper is depicted by a joint production function driven by both private and public efforts. In this structural framework (more on this in Section B), market reward (pull incentive) affects innovation only *indirectly* by stimulating private R&D (Acemoglu & Linn, 2004; Finkelstein, 2004; Blume-Kohout & Sood, 2013; Dubois et al., 2015; Garthwaite et al., 2021). In contrast, public R&D (push incentive) operates through two distinct mechanisms. First, it can *directly* create pharmaceutical innovation (Cohen et al., 2002; Sampat, 2009; Galkina Cleary et al., 2018, 2023). Second, it operates through an *indirect* crowd-in channel (Sussex et al., 2016; Toole, 2007). By subsidising private drug discovery costs and de-risking the developmental pipeline, so public efforts make subsequent private investment inherently more attractive (Sampat, 2021). Therefore, evaluating pull incentives in isolation neglects the effects of concurrent public investments and fails to capture the true structural mechanisms of drug development.

These dynamics have given rise to a concern that social planners may be *paying twice* for innovation, first through taxpayer-funded R&D (e.g., via research grants) and again through

higher temporary monopoly prices. This concern has driven legislative changes and policy proposals in the US and Europe. Examples include measures aimed at increasing transparency regarding R&D contributions (such as Article 57 of the proposed European Union pharmaceutical legislation (Wild et al., 2025)), as well as policies designed to adjust or reduce drug prices based on public support (such as the US Bayh-Dole Act (Hyman & Silver, 2020), the US Inflation Reduction Act, and the fair pricing calculator developed by the Association of Mutual Benefit Societies in the Netherlands (Stokx et al., 2026)).

Despite these policy initiatives, a coherent economic framework to guide these decisions and ensure optimal pricing is lacking. Current HTA practices often focus on de facto price regulation within the period of IPP and neglect the long-term value post-IPP. Conversely, while a recent study by Woods et al., (2024) estimated the optimal share of long-term innovation value offered to manufacturers that maximises long-term population health, their model evaluates pull incentives in isolation. By omitting public R&D, this framework reinforces a structurally imbalanced policy environment as well, which relies on pull incentives while leaving the role of push incentives unexamined.

Therefore, building upon the model of Woods et al., (2024), the purpose of this paper is to present a coherent and evidence-based approach to pharmaceutical pricing policies that formally integrates both push and pull mechanisms. By bringing public R&D directly into the innovation equation, we show that current policy suffers from a fundamental imbalance: over-relying on pull incentives while under-investing in public R&D push. Ultimately, the research question that guides this analysis is: to achieve dynamic efficiency in pharmaceutical innovation, what is the optimal balance between pull (payment) and push (public R&D investment) incentives to maximise welfare. For the purposes of exposition, we initially define welfare as the long-term population health effects, as it is an often-stated objective of pharmaceutical and health policy. However, the framework can be generalised to other welfare arguments. We consider those, including how the welfare effects of evidence of any retained producer surplus might be included, in the discussion.

The remainder of this paper is structured as follows. Section 2 introduces the analytical framework, detailing a model of optimal pull incentives and extending it to formally

incorporate public R&D (push incentives). Section 3 outlines the model parameterisation. Section 4 presents the main results, while Section 5 discusses them and the policy implications for dynamic efficiency and health system sustainability.

2. An extended framework for dynamic efficiency: integrating push and pull incentives

2.1 Baseline model: Share of value paid to manufacturers (pull Incentive)

To establish the trade-off between current population health and future pharmaceutical innovation, we build upon the analytical framework developed by Woods et al., (2024). This model adopts a health system perspective to maximise Net Health Effects (NHE). It balances static efficiency (health gains from existing drugs) against dynamic efficiency (health gains from future innovation).

Total health benefits (T) are composed of static (Tb) and dynamic (Td) benefits, both measured using any generic health metric that combines morbidity and mortality effects, here operationalised as Quality-Adjusted Life Years (QALYs):

$$T = Tb + Td \tag{1}$$

These benefits represent the health gains offered by a new pharmaceutical relative to existing comparators, accounting for additional health opportunity costs associated with producing and delivering the product and downstream impacts on patient management costs. To incentivise future R&D, the health system foregoes a share ($s \in [0,1]$) of this value to reward manufacturers. This occurs via a payment that is either described by its health impact ($s \cdot Tb$) or by the health care resources required to deliver it ($p = s \cdot Tb \cdot k$, where k is the marginal cost of producing one QALY). Thus, the share represents a health opportunity cost, as the resources to fund manufacturers' rewards could have otherwise generated health gains elsewhere in the system. Dynamic benefits are generated through this pull mechanism, where the payment to manufacturers to deliver a particular share acts as a signal of expected reward and a credible long-term policy commitment for future drugs. Assuming a constant value-of-innovation elasticity (ε), Woods et al., (2024) formulate dynamic benefits as a power function of this long-term payment signal:

$$Td = \alpha \cdot p^{\varepsilon} = \alpha(s \cdot Tb \cdot k)^{\varepsilon} \tag{2}$$

NHE are the total health value retained by the health system after rewarding the manufacturer.

$$NHE = (Tb + Td) - s(Tb + Td) = (1 - s)(Tb + Td) \quad (3)$$

This model characterises the effect of payment on dynamic benefits assuming that any other determinants of innovation are reflected in α. A limitation is that it lacks a formal mechanism to explicitly account for the role of public investment in R&D in stimulating innovation.

2.2 Extending the model: Incorporating public R&D (push incentives)

To address this limitation and establish a unified framework of pull and push incentives, we extend the baseline model by explicitly integrating public sector R&D (pRD). Public R&D has a direct effect on innovation (Galkina Cleary et al., 2018; Sampat, 2009; Sampat & Lichtenberg, 2011), and an indirect effect by crowding in private R&D, primarily via increased productivity of private R&D and de-risking of early discovery (Ward & Dranove, 1995; Toole, 2007; Sussex et al., 2016). Therefore, the effects of public R&D need to be accounted for in the dynamic benefits. We specify the new dynamic health benefit function (Td')[1] as a Cobb-Douglas production function of both private and public efforts:

$$Td' = \alpha' \cdot p'^{\varepsilon_1} \cdot pRD^{\varepsilon_2} \quad (4)$$

where ε_1 is now the reward elasticity of innovation, ε_2 is the public R&D elasticity of innovation, and α' is a calibrated scale parameter. These reduced-form parameters reflect distinct direct and indirect structural mechanisms (see Appendix, section Structural mechanisms of Td' for full derivations). While reward/payment to manufacturers (p') acts entirely indirectly $(\varepsilon_1 = \beta_1 \cdot \eta_1)$ by inducing private R&D investment, with β_1 capturing the private investment response to reward, public R&D operates via a dual mechanism $(\varepsilon_2 = \beta_2\eta_1 + \eta_2)$. This combines a direct contribution to innovation (η_2) with an indirect crowding-in effect on private R&D $(\beta_2\eta_1)$, where β_2 represents the returns of public R&D to private investments.

[1] To maintain a formal analytical clarity, we adopt the convention of denoting all parameters associated with the extended model using a prime ('). Thus, dynamic benefits, payment, the share of value paid to the manufacturers, total net health effects, and the calibrated scale parameter are now denoted as $Td', p', s', NHE', \alpha'$, respectively.

Under this extension, total benefits are expressed as before ($T = Tb + Td'$); however, health opportunity costs must be redefined to reflect public resources committed to both the pull and push policy levers. In addition to the manufacturers' payments forgone by the health system $s'(Tb + Td')$, costs must also include the health opportunity cost of public R&D investment pRD_{cost} / k [2]. Subtracting these comprehensive costs from the total health gains, the new objective function becomes:

$$NHE(s', pRD) = \left(1 - s'\right)(Tb + Td') - \left(\frac{pRD_{\text{cost}}}{k}\right) \quad (5)$$

Equation (5) can also be expressed in monetary terms, i.e. the money that the health care system would need to generate those net health benefits $(NHE_{£} = k \cdot NHE_{QALYs})$. This formulation partitions total effects into the total payment to manufacturers (PtM), $PtM_{£} = s' \cdot k(Tb + Td')$, and the net monetary value retained by the health system, $NHE_{£} = (1 - s') \cdot k(Tb + Td') - pRD_{\text{cost}}$.

As established by the literature (Acemoglu & Linn, 2004; Dubois et al., 2015; Woods et al., 2024), payment to the manufacturer must be viewed as a commitment in perpetuity to shape market expectations. As rewards serve as a long-term signal to the market, any policy choice regarding the share (s') must be sustained for the pull incentive to work. In contrast, the policy choice for the level of pRD may or may not require the same long-term commitment. Public investment can be a one-off shock, a temporary increase, or a sustained investment.

[2] Using *k* assumes funds for public R&D could instead have supported the healthcare budget, offering health gains based on the marginal cost of producing one QALY. If funded via an increase in public expenditure the opportunity costs would fall on consumption once account has been taken of the marginal cost of public funds (*MCPF*). However, once raised the social planner has a choice of how to allocate the additional public finance, so the opportunity costs of spending on public R&D is the value of the benefit forgone of not spending it on the 'best' other claim on public funds. Here we imagine the only other claim is health spending. However, this framework can be generalised to multiple claims if we either assume that the social planner allocates expenditure to equalise the marginal value of public funds (*MVPF*) across competing claims (*i*), where *MVPF* = $v_i/k_i > MCPF$, and *v* reflects some societal willingness-to-pay for health (see Longo et al 2024). This approach respects the social democratic process with all its checks and balances in deciding levels of public finance while providing evidence about $MVPF_i$>MCPF to inform this social choice and how it is 'best' allocated across competing claims (Longo et al., 2024).

Optimisation of Equation (5) with respect to the share offered to manufacturers[3] and the level of public R&D[4] investment yields two First-Order Conditions (FOCs) that dictate the optimal policy mix. The FOCs with respect to s' formalise the dynamic trade-off regarding pull incentives; the marginal gain in future health generated by higher manufacturer rewards is balanced against the immediate health opportunity cost of diverting healthcare resources away from healthcare. Crucially, because the marginal effect of the value share on dynamic benefits is a function of pRD, public R&D investment directly raises the marginal productivity of payment. To maximise NHE, the health system increases s' to capture this higher return, up until the point where the marginal cost offsets the marginal benefit.

The second FOC balances health lost today against health gained tomorrow. Diverting £1 from the healthcare budget into public research creates an immediate health opportunity cost $(1/k)$. In return, that investment generates future scientific discoveries, of which the health system retains a fraction $(1-s')$ after rewarding manufacturers.

This condition demonstrates how the optimal level of public research is structurally dependent on the payment (s'). If the health system concedes too high a share to manufacturers, the retained fraction of health gains $(1-s')$ shrinks. Consequently, excessive pull rewards fundamentally disincentivise push investments, as the health system cannot retain enough surplus to justify the opportunity cost. Optimal pRD occurs where the marginal health retained from future innovation exactly offsets the health forgone from current care.

These FOCs can be recast as the marginal cost per QALY $(MCpQ)$ for each policy lever by dividing the partial derivative of the total cost by the marginal dynamic health benefit for

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[3] $\frac{\partial NHE}{\partial s'} = 0 \Rightarrow (1-s')\left(\frac{\partial Td'}{\partial s'}\right) = Tb + Td'$ where $\frac{\partial Td'}{\partial s'} = \varepsilon_1 \frac{Td'}{s'}$. Thus, $\varepsilon_1 \frac{1-s'}{s'} = \frac{Tb}{Td'} + 1$

[4] $\frac{\partial NHE}{\partial pRD} = 0 \Rightarrow (1-s')\left(\frac{\partial Td'}{\partial pRD}\right) = \frac{1}{k}$ where $\frac{\partial Td'}{\partial pRD} = \varepsilon_2 \frac{Td'}{pRD}$

each respective policy lever[5] (details found in the Appendix section H. Derivation of Marginal Costs per QALY (MCpQ).

Because the dynamic benefits function is highly non-linear and additively combined with static benefits within the objective function, the system of FOCs does not yield a tractable closed-form analytical solution for the optimal policy mix. Consequently, we solve this system numerically to identify the precise combination of the optimal share $\left(s'^{*}\right)$ and public R&D investment $\left(pRD^{*}\right)$ that jointly maximises overall health benefits[6].

3. Model parametrisation

Though our model is intended to be generalisable across jurisdictions, we present model outcomes for the United Kingdom (UK) National Health Service (NHS). Because pharmaceutical-related R&D is a global public good, individual countries rarely incentivise innovation in isolation. Thus, we assume that a multilateral global policy for rewarding innovation is in place or that UK domestic policies fully internalise the dynamic health spillover benefits that accrue to other jurisdictions.

In the absence of robust causal evidence of the effects of global public R&D on innovation, we use empirical evidence from the US (which accounts for ~70% of global new medicines sales (EFPIA, 2024)) as a proxy for global pull and push incentives.

To align the model's parameterisation with our analytical framework, parameter inputs and results are conceptualised and presented at the level of an individual representative drug. Following Woods et al., (2021), baseline Tb is set at 31,121 QALYs, derived as the average total potential net health effect across a sample of 12 National Institute for Health and Care Excellence (NICE) technology appraisals to reflect expected lifetime net health gains over a

[5] $MCpQ_{s'} = k \cdot s' \left(1 + \frac{Tb + Td'}{\varepsilon_1 \cdot Td'}\right)$ and $MCpQ_{pRD} = k \cdot s' + \frac{pRD_{\text{cost}}}{\varepsilon_2 \left[Td' - \alpha\left(p'\right)^{\varepsilon_1}\left(1-\lambda\right)\right]}$

[6] Following the approach of Woods et al., (2024), we also analytically estimate a theoretical upper bound of the optimal share $\left(s'^{*U}\right)$ under the extreme assumption that dynamic benefits are unbounded relative to static benefits, meaning Tb becomes negligible; further details about this are provided in Appendix G. Derivation of maximum optimal share ($\boldsymbol{s'^{*U}}$).

100-year lifecycle. Likewise, the baseline value share offered to manufacturers (s'^{0}) is set at 0.51, representing the empirical share retained by manufacturers for this sample. Future health benefits and costs are discounted at a rate of $r = 3.5\%$ per annum, in line with HM Treasury Green Book guidelines and NICE reference cases (HM treasury, 2026; NICE, 2022). Health opportunity costs use £15,000 per QALY adopted by UK Department of Health and Social Care when undertaking Impact Assessment of policy (Department of Health and Social Care, 2025). This reflects earlier empirical estimates of the marginal productivity of changes in UK NHS expenditure (Lomas et al., 2019). More recent empirical estimates of NHS marginal productivity suggest the cost per QALY to reflect opportunity cost is likely to be lower (Martin et al., 2021, 2023).

Within our framework, public R&D is parameterised through incremental expansions (ΔpRD) relative to a baseline research expenditure (details in Appendix section Parameterisation of the dynamic benefits function (Td')). Public R&D plays a dual role: first as an input that generates future innovation (within the dynamic benefits function), and second as a financial expenditure that incurs an immediate health opportunity cost (pRD_{cost} / k in the NHE objective function). To parameterise ΔpRD as an innovation input, we distinguish between a temporary single-year funding shock and a sustained perpetual investment commitment. While a shift in the manufacturer value share impacts innovation relatively quickly, public R&D is subject to significant translation lags, averaging 12 years from patent discovery to clinical readiness (Beall et al., 2019; Lexchin, 2020), and yields subsequent innovations over a 20-year horizon (Azoulay et al., 2019). Using these assumptions, we annualise both private revenues and public R&D expenditures to align timing horizons and derive a common numeraire (λ) that is used in the Td' function (see Appendix E. Derivation and integration of a common numeraire (λ) for further details).

To determine the scale of annual average pRD expenditure per drug, we convert the manufacturer's total lifecycle reward into an annualised revenue flow and then apply the five-year average ratio of public R&D to private drug revenue reported by the (approximately 14%) (CBO, 2021). This reflects the empirical balance between public research allocations and net pharmaceutical market revenues; see Appendix F. Public R&D

cost for the step-by-step derivation. This gives annual $pRD^{baseline}$ = £3.5 million and represents the proportional public R&D contribution attributable to developing a drug, not the aggregate national research budget [7]. Public R&D expenditure entering the objective function (Equation 5) is calculated as $pRD_{\text{cost}} = pRD^{baseline} \cdot (1 + \Delta pRD)$, scaling this baseline outlay by the proportional push expansion.

Elasticity parameters are calibrated to isolate push and pull effects. The base case reward elasticity of innovation $\left(\hat{\varepsilon}_1 = 0.45\right)$ is based on CBO's structural model (Adams, 2021). It also reflects a plausible but optimistic view of the balance of the other recent evidence (Filson et al., 2025) and is the central estimate used in Woods et al 2025. This elasticity and others within the literature do not simultaneously identify the causal effects of reward and public R&D (Dubois et al., 2015). Because public R&D allocations and healthcare payment decisions concurrently target the same therapeutic areas, estimates of ε_1 that do not simultaneously identify ε_2 may suffer from omitted variable bias (OVB). As detailed in Appendix section Literature gap on ε_1 and structural adjustment, we apply a structural OVB correction using the correlation between public R&D and payment. After controlling for these public R&D spillover effects, the direct payment elasticity $\left(\varepsilon_1\right)$ reduces to 0.23 (CBO, 2021; Sussex et al., 2016). The total innovation elasticity with respect to public R&D $\left(\varepsilon_2\right)$ is set at 0.381, drawing on causal empirical estimates from Azoulay et al., (2019)[8].

To comprehensively evaluate the optimal policy mix, we estimate the model across four distinct scenarios. These scenarios vary by two key parameters: the elasticity parameter applied to the pull incentive, and the temporal nature of the public R&D investment (a temporary 1-year versus a sustained perpetual commitment).

[7] We rely on recent US CBO data for this baseline calibration due to the availability of contemporary, high-quality public R&D reporting, whereas comparable UK-specific estimates (e.g., Sussex et al., 2016) rely on data extending only up to 2012.

[8] This study provides a causally identified estimate of the effect of public R&D on innovation by analysing US National Institutes of Health (NIH) grant funding and subsequent private-sector pharmaceutical patenting. To address potential endogeneity, the study exploits quasi-experimental variation from NIH peer-review scoring rules and funding cutoffs, using bibliometric links to track citations from grants to private patents across all therapeutic areas. We select their baseline elasticity estimate of 0.381 because it captures long-run, spillover-inclusive private innovation responses across the broader life-sciences sector without imposing restrictive disease-specific boundaries.

For narrative clarity and to avoid redundant exposition, the next section focuses exclusively on the results of Scenario 1 (evaluating a one-year public R&D shock alongside the unadjusted elasticity). We prioritise this scenario because it aligns both with the foundational parameters used in the original model and with the empirical evidence on elasticities established in the underlying literature (e.g., (Azoulay et al., 2019)). Thus, this scenario provides easy-to-compare results to show the core structural mechanics of the model before introducing OVB corrections or perpetual funding dynamics. It also represents an optimistic assessment of the marginal benefits of pull incentives. The full results and sensitivity analyses for the other scenarios are detailed in Appendix Section Sensitivity results.

4. Results

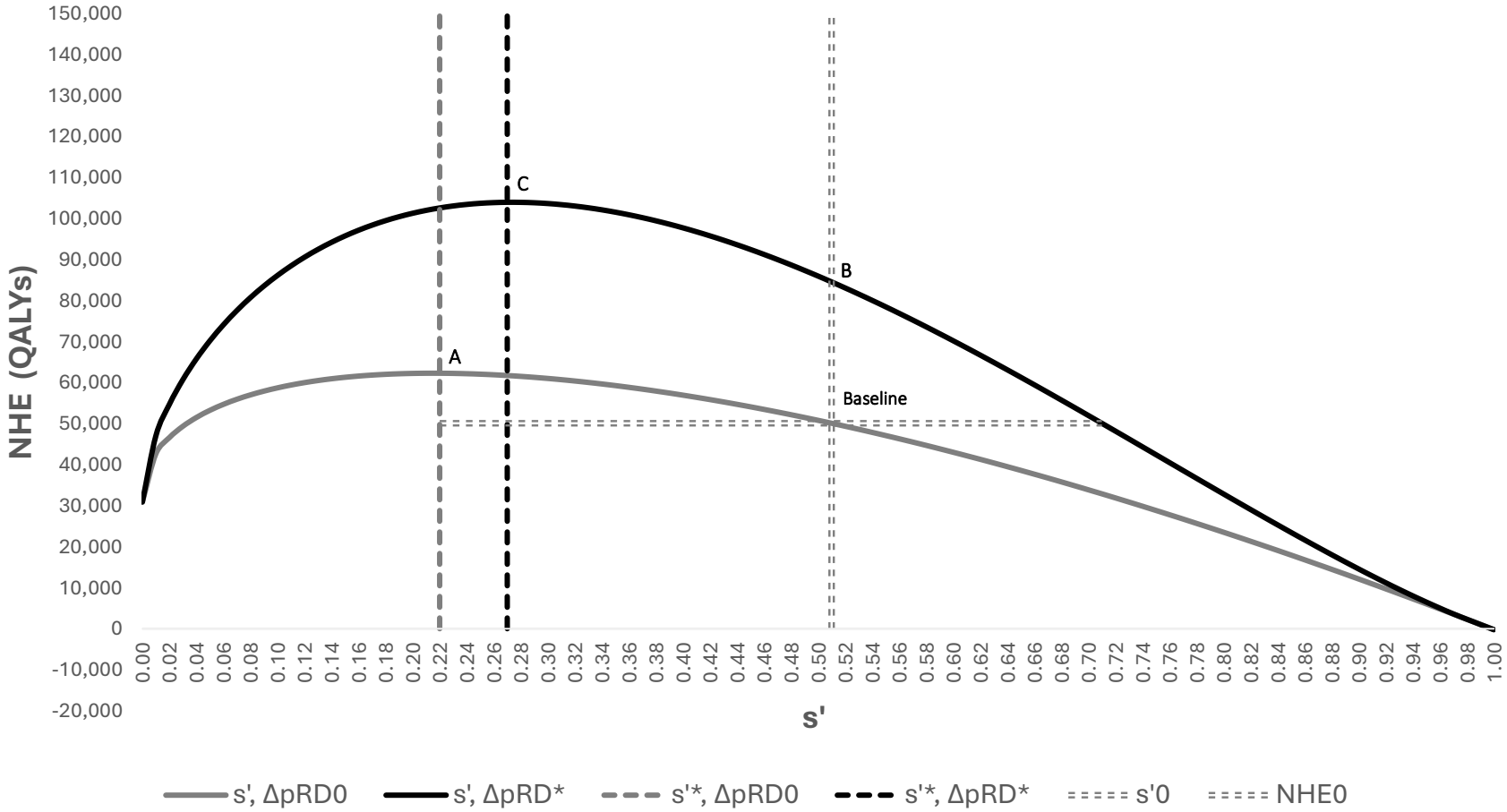
Trade-offs between push and pull incentives are illustrated in Figure 1. This shows the NHE trajectories measured in QALYs across the full range of manufacturer value shares $\left(s' \in [0-1]\right)$. Two concave objective function curves are depicted: the grey solid curve $(s', \Delta pRD^0)$ indicates the health benefits at current public R&D, which is $\Delta pRD = 0$, whereas the black solid curve $(s', \Delta pRD^*)$ represents the benefits under optimal public R&D investment for each level of value shared to manufacturers. There are also three vertical references: the light grey double-dashed vertical line at $s'^0 = 0.51$, which indicates the current value share retained by manufacturers; the dark grey dashed line at $s'^*, \Delta pRD^0 = 0.22$ [9]; and the heavy black dashed line at $s'^*, \Delta pRD^* = 0.27$ that identifies the joint optimal value share when ΔpRD is expanded to ΔpRD^*. Comparing optimal shares across both curves shows that expanding public R&D increases the optimal manufacturer share. Because public research enhances private R&D productivity, a stronger public scientific foundation increases the marginal return on pull incentives, justifying a higher payment share. Finally, the horizontal dashed line references the baseline net health benefits

[9] This optimal value coincides exactly with that of Woods et al., (2024), providing an empirical validation of our extended model. When public R&D remains at baseline, the push expansion term collapses to zero, ensuring the dynamic benefits function converges mathematically with their original framework.

achieved under the current policy (NHE^0) . This provides a reference point (Baseline) against which the net health gains of moving to points A, B, and C, are measured.
The movement to point A provides a comparison of the health effects under current institutional arrangements with those when only the share of value paid to manufacturers is optimised, at the current level of public R&D. This pull-only policy shows that the current share over-rewards innovation, being inefficiently generous and creating excessive health opportunity costs that outweigh the marginal health gains generated by additional innovation. However, by decreasing the share, even when push incentives remain unchanged, net health gains increase. Similarly, pull incentives can be kept unchanged while increasing investment in push to its corresponding point on the black curve (point B). The benefits are higher than at A, showing that even under an unfavourable reward policy, expanding public R&D increases net benefits above the baseline. It suggests there is underinvestment in public R&D even when accounting for the overinvestment in pull incentives. The gains of moving to a global optimum by using both policy levers is the vertical distance from baseline to point C, the peak of the black curve $(s'^*, \Delta pRD^*)$. This is achieved through less pull and more push. The magnitude of NHE at point C demonstrates that the gains of joint optimisation exceed what either policy lever can accomplish in isolation. At this point, the health system achieves full dynamic efficiency in terms of both pull and push incentives.

To complement the characterisation of the net health response, Figure 2 presents NHE across varying expansions of public sector R&D $(\Delta pRD \in [0, 200])$ for different levels of pull incentives. Two concave curves are displayed: the solid grey curve $(s'^0, \Delta pRD)$ illustrates health gains as public R&D expands at the current manufacturer share, whereas the solid black curve $(s'^*, \Delta pRD)$ traces the health benefits for each local optimal share for a given ΔpRD. The same vertical reference lines as in Figure 1 are depicted: the light grey dotted line (which coincides with the y-axis) represents baseline public investment, the dark grey dashed line is $\Delta pRD^*, s'^0 = 122$, and the heavy black dashed line represents $\Delta pRD^*, s'^* = 148$ (representing an expansion of public R&D to 122 and 148 times, respectively, its baseline). The horizontal dashed line is the reference for NHE^0.

Figure 1. Population health gains under pull-only optimisation, push-only expansion, and joint pull-push optimisation.



Notes: The horizontal axis plots the current level of Net Health Effects. Key benchmarks include current value share (light grey double-dashed), optimal value share under baseline public R&D (dark grey dashed), and joint optimal value share (heavy black dashed).

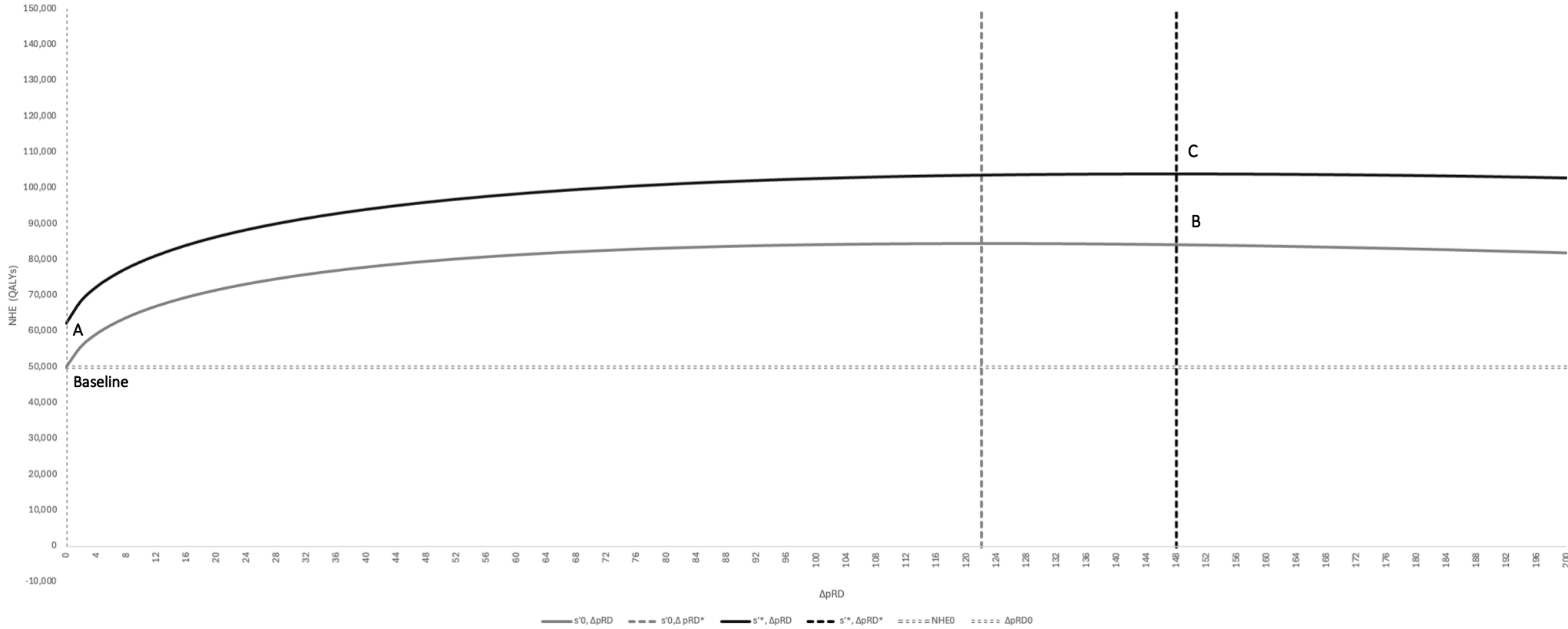
Points A, B, and C in Figure 2 directly mirror those in Figure 1, but Figure 2 provides key added value by revealing the optimal levels of private R&D under each policy, which is not visible in Figure 1. At point B, where pull is unchanged but push increases, health benefits increase from baseline levels. The curve reaches its peak, where the marginal health gain from public R&D exactly offsets the marginal health opportunity cost of expenditure on public R&D. Point C indicates the same global maximum as in Figure 1. This illustrates that reducing the manufacturer share increases the optimal public R&D. When the health system reduces pull incentives, it retains a greater fraction of the resulting dynamic health gains. This higher retention rate increases the health system's net return on public R&D, making it welfare-maximising to absorb a higher level of public R&D expenditure.

To provide a complete picture, Figure 3 illustrates how these policy configurations translate into total payments to manufacturers and the revenue they receive (PtM). Under pull-only optimisation, manufacturer revenues decrease compared to the baseline (the move to point A), due to a lower share of value for each innovation and lower innovation levels, so making use of only the pull-policy lever is highly unfavourable to manufacturers. Conversely, under push-only optimisation, payment to manufacturers increases significantly (the move to point B). This is due to the share of value remaining stable alongside greater public R&D investment and therefore future levels of innovation. While representing substantial financial gains for manufacturers, this outcome is unfavourable for public expenditure as the health system must fund expanded public research while continuing to pay a high share across both current products and the additional future innovations generated by public funding.
Finally, point C, identifies the payment to manufacturers under the joint pull-push optimisation policy: manufacturers receive a total payment comparable to baseline levels, while society achieves significantly higher net health gains (QALYs) without increasing public expenditure on reward. However, public expenditure on R&D increases and overall public expenditure devoted to both push and pull incentives would increase by 62% in this scenario[10].

[10] Where over investment in pull incentives is even more pronounced than in our sample of UK innovations it is clearly possible for joint optimisation to be neutral for overall public expenditure or even reduce it. For example, in the UK a more recent sample of innovations or a more evidence-based assessment of the marginal cost of

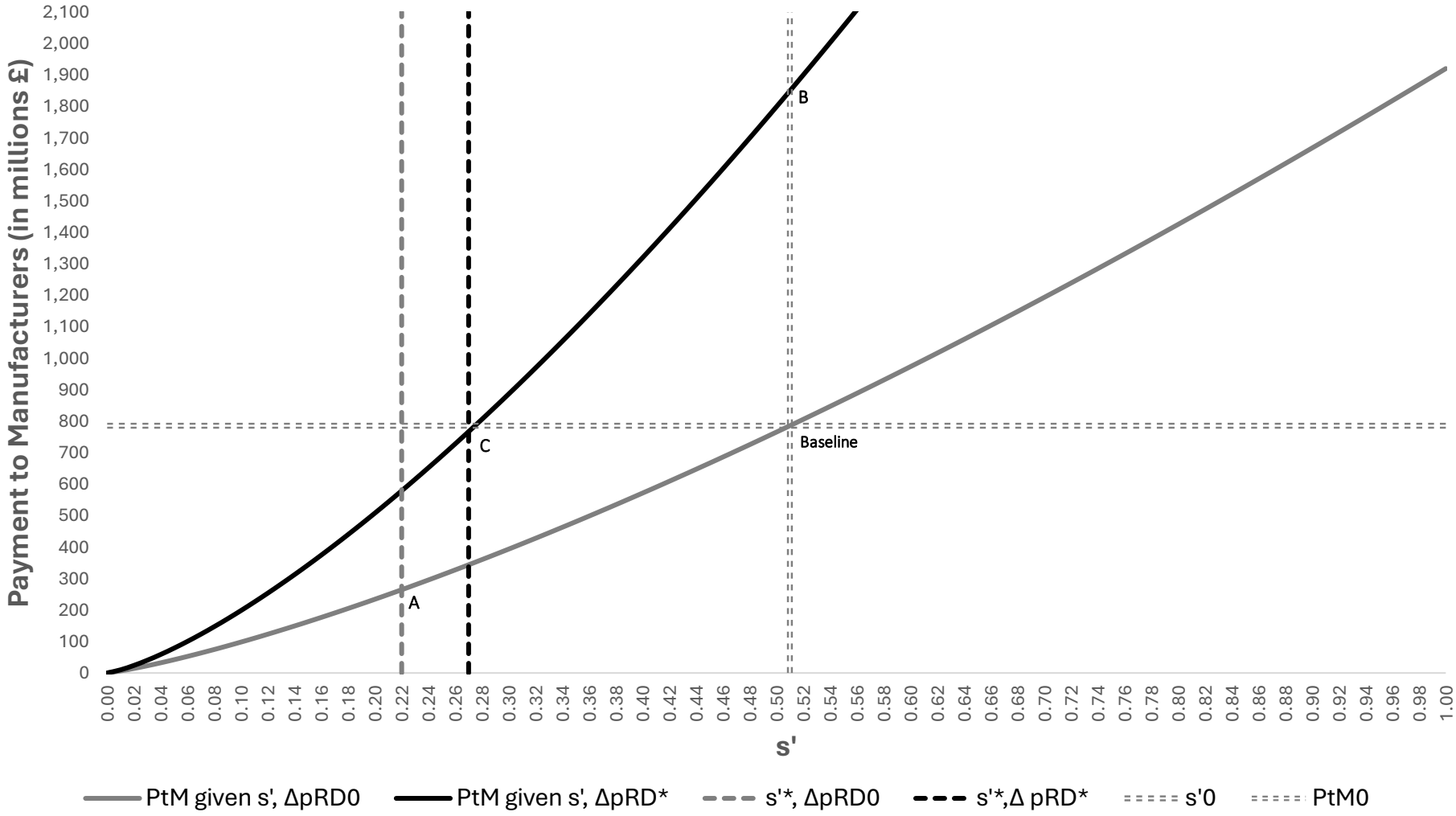
producing a QALY would reduce the overall public expenditure required. The same would be true for other health care systems which have overinvested in pull to an even greater extent or where the reward elasticity of innovation is lower (see Scenario 3 in Table A1) . However, in scenario 1 with this UK sample it would require more than a 60% increase in public expenditure devoted to pharmaceutical innovation. Although a significant commitment it is substantially less that the 100% increase in expenditure on new medicines that has been agreed as part of the UK-US Pharmaceutical Trade Deal.

Figure 2. Population health gains across changes in public R&D under current and optimised manufacturers' value shares.



Notes: the horizontal axis plots percentage changes in public R&D investment. Key benchmarks include current ΔpRD (light grey double-dashed), optimal ΔpRD^* under current manufacturers' share (dark grey dashed), and optimal ΔpRD^* under joint pull and push optimisation (heavy black dashed).

Figure 3. Payment to manufacturers under pull-only optimisation, push-only expansion, and joint pull-push optimisation.



Notes: The horizontal axis plots the current level of payment to manufacturers. Key benchmarks include current value share (light grey double-dashed), optimal value share under baseline public R&D (dark grey dashed), and joint optimal value share (heavy black dashed)

The need to rebalance investment from push towards pull incentives is evident in the marginal cost per QALY generated by each policy lever. Table 1 shows that at baseline, expanding innovation via pull incentives incurs a marginal cost of approximately £32,000 per QALY, compared to just £8,000 via public R&D. This cost-effectiveness advantage for push incentives persists across pull-only (A) and push-only (B) policies. Under the joint pull-push optimisation policy (C), both policy levers reach marginal equilibrium, with a marginal cost per QALY that converges near the health system's marginal cost of producing one QALY of £15,000.

Table 1. Marginal Cost per QALY of both policy levers.

	s'	ΔpRD	pRD cost	Dynamic benefits	Marginal Cost per QALY (£)	
			(£)	(QALYs)	s'	ΔpRD
Baseline	0.51	0	3,449,181	71,578	£32,041	£8,023
Optimal policy A: Pull only	0.22	0	3,449,181	49,030	£15,288	£3,844
Optimal policy B: Push only	0.51	122	424,249,286	199,149	£27,307	£14,983
Optimal policy C: Pull and push	0.27	148	513,927,997	158,229	£14,820	£15,044

Notes: These results are only for Scenario 1. At the welfare-maximising equilibrium, the optimality condition dictates that the marginal cost of dynamic innovation across both policy instruments converges to the cost-effectiveness threshold $(MCpQ_{s'} = MCpQ_{pRD} = k)$ .Evaluating our model at the optimal policy mix yields an empirical marginal cost of £14,820 and £15,044. Both values align closely with the benchmark threshold of k=£15,000 per QALY, with small residual differences attributable purely to point decimal precision in the underlying numerical estimation procedures.

4.1 Results from sensitivity analyses

To evaluate the robustness of these results, Table A1 extends the findings across scenarios considering a lower reward-innovation elasticity and a different push funding horizon (perpetual public research commitments). Across all scenarios, joint pull-push optimisation (Policy C) consistently delivers the highest NHE, reinforcing the fundamental limitation of pull-only mechanisms.

However, there are some nuances worth mentioning. Relative to Scenario 1, sustaining public R&D in perpetuity given the same pull-related elasticity (Scenario 2) leaves optimal policy levers relatively stable. However, when innovation is less responsive to pull incentives (Scenarios 3 and 4), the social planner relies more on push incentives. Under a single-year funding shock (Scenario 3), the optimal manufacturer value share falls while push expansion

increases. A similar situation occurs under perpetual push investment (Scenario 4), where the optimal value share drops and push expansion increases. Thus, lower reward-innovation elasticity shifts reliance away from pull toward push incentives. Regardless of elasticity assumptions or time horizons, joint optimisation remains unequivocally superior to single-instrument policies.

Regarding the payment to manufacturers, pull-only optimisation (Policy A) consistently decreases manufacturer revenues relative to baseline. Conversely, push-only expansion (Policy B) substantially increases payments across all scenarios. Under joint optimisation (Policy C), however, payments to manufacturers vary meaningfully across scenarios relative to Scenario 1. While Scenario 1 achieves near cost-and-payment neutrality, sustaining public R&D in perpetuity under baseline elasticity (Scenario 2) increases total manufacturer payments significantly. This occurs because sustaining public R&D over time generates a large expansion in overall dynamic health gains. In contrast, when the elasticity responsiveness is lower under a single-year shock (Scenario 3), manufacturers receive less than baseline; here, the sharp reduction in value share cannot be offset by the dynamic health gains generated by a one-time public R&D investment. Finally, when lower elasticity is paired with perpetual public R&D investment (Scenario 4), payment to manufacturers increases because sustained public R&D generates substantial dynamic health gains that outweigh the lower value share.

These results aid the evaluation of a range of policy responses, including the potential for pharmaceutical 'clawback' mechanisms. These are regulatory policy instruments that require manufacturers to rebate a portion of their revenues to ensure prices reflect public contributions and mitigate the concern that the public pays twice for innovation, first through taxpayer-funded R&D and later when facing monopoly prices. These are reflected in policy responses including the US Inflation Reduction Act, the Bayh-Dole Act, proposed EU transparency rules (Article 57), and the Dutch fair pricing calculator. In our framework, we assume that public R&D funding expansions have an opportunity cost evaluated at k, reflecting the health gains foregone had those funds been allocated directly to the healthcare system. Within this context, we evaluate two potential 'clawback' mechanisms: recoupment, where public funds are recovered (reclaimed from manufacturer revenues and returned to the healthcare budget), and reinvestment, where those recouped funds are reinvested into

public research. The former operates purely as an implicit reduction in the manufacturer value share; the latter combines a pull reduction with a push expansion. However, relying on either mechanism yields sub-optimal welfare outcomes. In Figure 1, if the health care system retains clawed-back revenues for healthcare spending without expanding public research, the policy simply shifts the system leftward along the baseline pull-only trajectory $(s', \Delta pRD^0)$ in Figure 1. The maximum revenue collectable via the clawback equals the baseline public R&D investment, which equals $pRD^{baseline}$ = £3.5 million (see Section 3). While this reduces the value share and increases NHE above baseline, welfare improvements are bounded by the insufficient pull-only response. If these recouped resources are reinvested into public R&D such that $pRD^{baseline}$ doubles, the policy still generates a lower manufacturer value share but achieves higher NHE than recoupment alone due to the push expansion. However, this policy response remains constrained by the size of the public R&D expansion, which is equal to the recouped funds. Thus, this maximum revenue collectable via clawback is substantially lower than the public R&D expansion required to reach the global optimum (C) (around £514 million). Consequently, 'clawback' does not sufficiently reduce the current overinvestment in pull incentives, and its reinvestment can capture only a fraction of the potential welfare gains of the push expansion required. This demonstrates that these mechanisms alone, whether invested in health care or public R&D, cannot substitute for direct push investments combined with a rebalancing of pull incentives.

5. Discussion

The results from this study show that achieving dynamic efficiency in pharmaceutical policy requires considering pull and push incentives simultaneously. Based on a parameterisation that reflects the UK context, a rebalancing is required with more investment in push and less in pull. This rebalancing to achieve dynamic efficiency can be revenue neutral for manufacturers and neutral for public expenditure on new medicines, while increasing innovation and net health benefits. Relying solely on reducing the share of value and payments to manufacturers to deliver improved net health benefits requires the health system to accept a reduction in innovation ($Tb + Td'$, falling from 102,299 QALYs at baseline to 80,151 QALYs at Point A), and manufacturers to accept reduced revenues (decreasing from £786 million to £264 million see Table A1). However, by reshaping the policy to

combine less pull with more push (point C), the health system can maximise net health benefits without reducing innovation; in fact, overall innovation expands substantially, delivering 189,350 QALYs while manufacturer revenues are sustained at £767 million. This combined policy delivers greater net health gains, increased innovation, and sustainable revenues, with other possible spillover benefits from improving the life sciences sector and the general research environment. However, expanding push incentives without adjusting the current share paid to manufacturers fails to maximise net population health gains and requires substantially greater public finance to cover increased total payments to manufacturers and the public R&D investment. It suggests that expansion of public investment in R&D should be conditional on reducing the rewards per innovation received by manufacturers to move towards an optimal value share.

Contemporary policy debates have framed public research contributions through the *paying-twice* narrative. We have shown that this is conceptually unhelpful and does not guide policy to dynamically efficient solutions. Rather than viewing public research as a subsidised investment that must be 'clawed' back from manufacturers, health systems should recognise manufacturer rewards and public research spending not as antagonistic, but as complementary policy levers designed to shape the innovation landscape, both of which must be balanced against the health (and other) opportunity cost falling on the health care system. Regulatory mechanisms that rely only on 'clawback' to mitigate the impact of health care costs suffer from fundamental limitations. By failing to reinvest recouped surpluses into public research, it operates merely as limited containment of pull incentives, foregoing the substantial dynamic health gains made possible by an expanded scientific infrastructure. However, 'clawback' may remain an important, pragmatic policy tool in contexts where implementing more appropriate price regulation to move towards optimal value shares is politically challenging.

When evaluating these policy shifts, it is important to consider the temporal dynamics and revenue implications for manufacturers. Because our model evaluates long-run equilibria, transitioning to a higher-push environment involves an intertemporal misalignment: manufacturers operate with a higher cost of capital (discount rates of approximately 11%) compared to the societal discount rate (3.5%) used to estimate the long-run net health

effects. This misalignment means that while rebalancing toward push is mutually beneficial in the long run, the transition period may appear more unfavourable to manufacturers.

Rebalancing incentives away from pull (reward) towards greater investment in push (public R&D) has qualitative advantages that extend beyond our quantitative modelling. Although additional reward increases the quantity of innovation, it also incentivises the preservation of monopoly rights through strategies to delay generic entry and uptake. It also encourages competition for market shares through marginal innovations, marketing and other activities of limited social value. Push incentives, however, can be strategically directed toward foundational scientific discovery, unaddressed epidemiological burdens, and high-risk translational platforms that private capital tend to underfund (Galkina Cleary et al., 2018; Moses et al., 2015). This highlights the need to better understand the effect of combined reward and public R&D policy levers on the overall quality of innovation, an important direction for future empirical research. Policymakers should therefore view our findings as an indicator that, at the margin, public R&D currently operates in a state of under-investment relative to the public resources committed to pull rewards.

Our findings must also be interpreted within the context of specific analytical limitations. First, the substantial magnitude of optimal public R&D expansion warrants careful interpretation. We adopted a log-linear Cobb-Douglas functional form to be consistent with how the established econometric literature has estimated the innovation effects of reward (generally using a log-log constant elasticity function form). Since our central estimate is less than one, it suggests diminishing marginal returns to increasing reward. Causal estimates of the effect of public R&D on innovation, such as Azoulay et al., which report an elasticity evaluating 1-year investment exposures, are also less than one. This indicates diminishing returns to public R&D, but it was not estimated using a log-log functional form, so returns may diminish more quickly. Therefore, while our functional form for pull incentives is firmly grounded in the empirical literature, the precise optimal public R&D expenditure figures should be viewed as a directional indicator at the margin rather than exact expenditure prescriptions.

Second, our model implicitly evaluates public R&D expansion within a global framework rather than an isolated national effort. If a single country, like the UK, were to unilaterally expand public R&D at the scale suggested here, it would not capture the full extent of the predicted health effects, as the benefits of the stimulated innovation will accrue to others. Consequently, a unilateral domestic expansion would fall well short of the welfare gains projected by our global optimum. Achieving the full welfare improvements demonstrated in our analysis would ultimately require multilateral coordination, ensuring that local health systems collectively contribute to publicly funded R&D and pay rewards based on how much their health care system can afford to pay for the optimal share of long-term benefit they receive. However, from a domestic economic perspective, it remains politically and economically important to recognise that a pound invested directly into national public R&D infrastructure is likely to yield substantially higher direct national economic returns than a pound spent on pull payments to the global pharmaceutical sector.

For the purposes of exposition, we initially defined welfare as the long-term population health effects, as this is a commonly stated goal of pharmaceutical and health policy. The measure of health we have used (QALY) was for convenience. It is a measure commonly used outside the USA and estimates are available to illustrate the impact of the policy levers. However, the findings would not change if other measures of health were substituted to measure the benefits and opportunity costs. Similarly, valuing health effects in terms of their equivalent consumption, using whichever of the wide range of estimates of willingness to pay for health is preferred (Phelps, 2019), would not change the findings either, including the reward to manufacturers which are paid predominantly by third party payers rather than consumers. Our exposition and illustration appears most closely related to a health care system like the UK NHS. However, the framework and our findings do not rest on whether there is a central politically determined fixed budget. The question of what health benefits could have been gained if additional reward to manufacturers was instead made available for health care remains even if total health care expenditure was increased to accommodate it. Of course, not all health care is collectively funded through social insurance or centralised tax-funded systems. Although the US has a well-developed private insurance market, almost half of health care expenditure is state- or federal-tax funded. However, even in private insurance systems, the additional costs of offering more pull incentives also have health

consequences and the payment of reward to manufacturers is predominantly made by third-party payers (Vanness et al., 2021). The health consequences of increased health care costs might be set aside, if one was willing to assuming that patients at point for care or citizens making insurance plan choices are fully informed by perfect agents. This would, however, conflict with the considerable body of theoretical and empirical evidence of market failure in health care even when distributional concerns are set aside (Arrow, 1963; Rice, 1997). Therefore, we believe that the framework and our qualitative findings are also relevant to private health insurance systems.

Although it seems self-evident that health will be an argument in any welfare function used to evaluate health policy, it is unlikely to be the only one (Neumann et al., 2022). However, our framework can be generalised to other welfare arguments by simply redefining static and dynamic benefits, while reflecting other arguments in the opportunity costs as well (Longo et al., 2024; Sculpher et al., 2026). The findings will be unchanged if any other non-health arguments are related to health in the same way, whether health is gained or foregone. Of course, an innovation may offer 'other' benefits that are greater (less) than the 'other' opportunity costs. In which case optimal payments would be higher (lower), but this would not change our findings.

Which particular welfare function ought to be adopted to identify and value all the arguments to reflect the social good is neither self-evident nor undisputed (Diamond, 1967; Sen, 1977), especially when many attributes of social value are not marketed and are related to goods in markets which are often distorted (Lipsey & Lancaster, 1956). However, in other literature, much is made of including producer surplus as an important welfare argument (Jena & Philipson, 2008; Vernon et al., 2009; Danzon et al., 2015; Lakdawalla, 2018; Basu, 2026). The point at which the social planner must decide what value share to offer as a sustained long-term signal of reward is when the costs of bringing an innovation to market have not yet been sunk (if they had, there would be no pull incentive from greater reward). Therefore, one cannot regard payment to manufacturers above the marginal cost of manufacture and supply as producer surplus. To do so would be either to count the considerable costs of bringing an innovation to market as also a benefit, or to imagine that innovations can be realised at no cost. In a first-best world, the market value of all the

resources required to bring an innovation to market is an estimate of its social costs, which, at the margin, also indicates the social benefits forgone of not being able to use these resources in other ways. Indeed, it is plausible that very little or no producer surplus will be retained after the costs of research and development, regulatory hurdles, marketisation, and the competition for temporary monopoly rights and for market share among temporary monopolists producing (imperfect) substitutes have taken place. This, combined with the effects of competitive capital flows, means that any residual producer surplus is likely to be quickly eroded until there is a normal risk-adjusted return to capital across research programs and manufacturers.

Should convincing theoretical and empirical evidence emerge which supports sustained producer surplus in this sector, increasing rewards is not a simple transfer between consumers and producers for three reasons. Firstly, the loss of consumer surplus will exceed any gains in producer surplus due to the cost of bringing an innovation to market. Second, the common assumption that any transfers simply cancel because the welfare losses to consumers are equal to the welfare gains from those individuals that ultimately benefit from producer surplus can only be sustained in an abstract, pure exchange closed economy with no distributional concerns. The reality is much more complex, because the welfare effects depend on who ultimately receives it, where it is received and how the surplus is used (Mishan, 1968). Finally, in collectively funded health care it is public expenditure, raised from distortionary but socially acceptable forms of taxation, that pays the reward. Since estimates of the marginal cost and the marginal value of public funds exceed one (Ruggeri, 1999; Finkelstein & Hendren, 2020; Bastani, 2025), the $ value of any retained producer surplus is worth less than the same $ amount of public expenditure.

For all these reasons, but especially the lack of convincing evidence of any sustained producer surplus, we have set it aside in this analysis. However, our framework can be extended to include it with evidence of whether and how much is retained. Nevertheless, it is unlikely to change our core findings. For example, in a sensitivity analysis, Woods et al., (2024) examined the impact of imagining that 20% of future payments are retained as producer surplus and that the payment above marginal costs for the current product is producer surplus, as most (although not all) of the costs of bringing an innovation to market

and gaining market share have been sunk. This had limited impact on their findings (Woods et al., 2024, p. 10)

Therefore, despite inevitable limitations, this framework integrates the best available causal and econometric evidence into a unified model to support policy design to achieve dynamic efficiency. Future progress will depend on developing richer empirical datasets that simultaneously estimate how public R&D and market payment structures jointly influence the quantity, quality, and long-term value of pharmaceutical innovation.

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Conflict of Interest

All authors declare no conflict of interest.

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Appendix

A. Sensitivity results

Table A1. Summary of results across all scenarios

Scenarios	s'^*, ΔpRD^0	ΔpRD^*, s'^0	s'^*, ΔpRD^*	ΔpRD^*, s'^*	Net gains in health (QALYs) under Policies:							Net gains in payment (in millions £) under Policies:						
					BL	A	BL to A	B	BL to B	C	BL to C	BL	A	BL to A	B	BL to B	C	BL to C
Scenario 1 Reward-innovation elasticity=0.45 pRD 1-year cost	0.22	122	0.27	148	50,093	62,288	**12,195**	84,549	**34,456**	103,964	**53,871**	786	264	**-521**	1,762	**976**	767	**-19**
Scenario 2 Reward-innovation elasticity=0.45 pRD cost in perpetuity	0.22	124	0.31	150	43,753	55,948	**12,195**	1,036,608	**992,855**	1,283,887	**1,240,134**	786	264	**-521**	29,005	**28,219**	15,338	**14,552**
Scenario 3 Reward-innovation adjusted elasticity=0.23 pRD 1-year cost	0.13	122	0.16	190	50,093	72,320	**22,227**	84,549	**34,456**	128,191	**78,098**	786	163	**-623**	1,762	**976**	492	**-294**
Scenario 4 Reward-innovation adjusted elasticity=0.23 pRD cost in perpetuity	0.13	124	0.19	194	43,753	65,980	**22,227**	1,036,608	**992,855**	1,693,806	**1,650,053**	786	163	**-623**	29,005	**28,219**	10,467	**9,682**

Notes: Policies (A): Pull-only optimisation, (B) push-only expansion, and (C) joint pull and push optimisation. BL means Baseline.

Figure A1-Scenario 2 (ε_1=0.45, Perpetual pRD_{cost}). Population health gains under pull-only optimisation, push-only expansion, and joint pull-push optimisation.

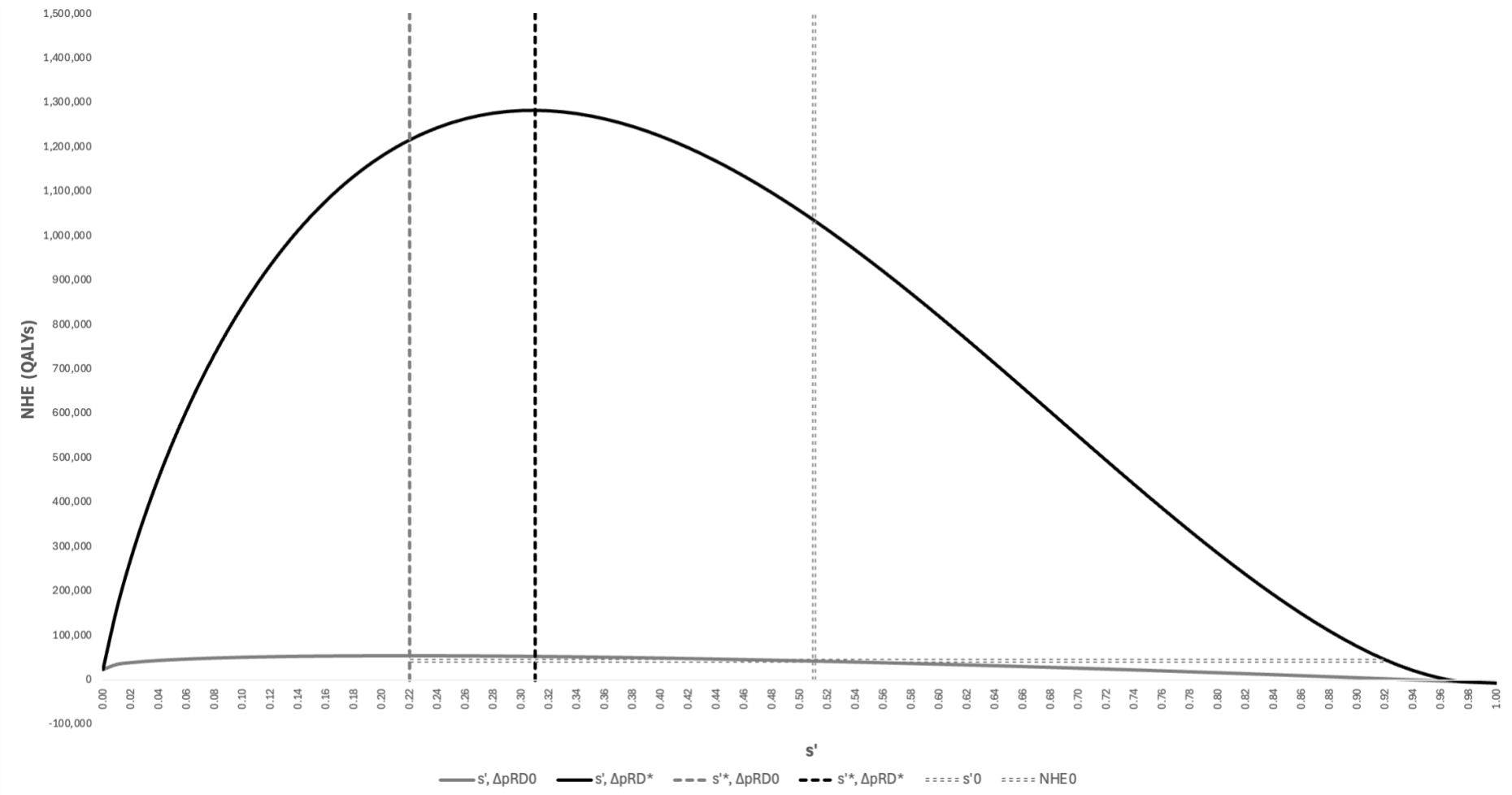


Figure A2-Scenario 2. Population health gains across changes in public R&D under current and optimised manufacturers' value shares.

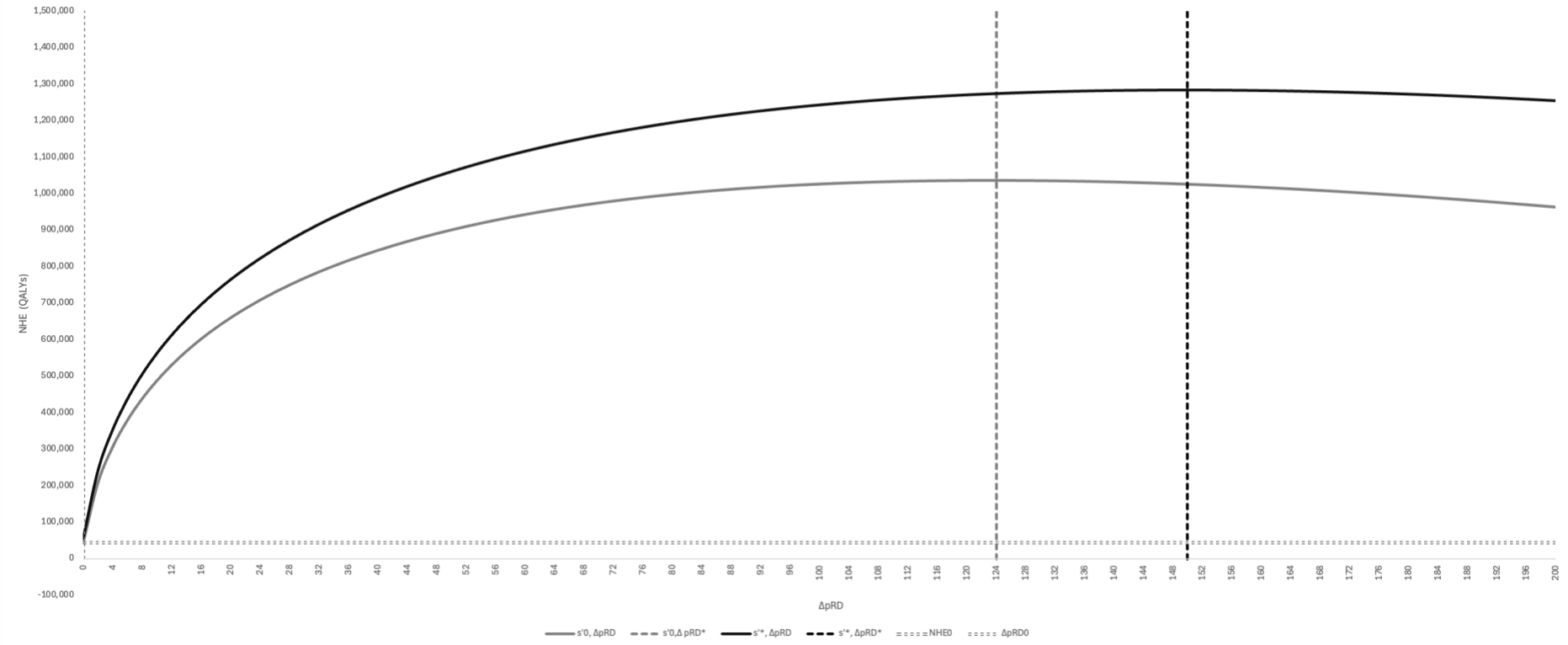


Figure A3-Scenario 2. Payment to manufacturers under pull-only optimisation, push-only expansion, and joint pull-push optimisation.

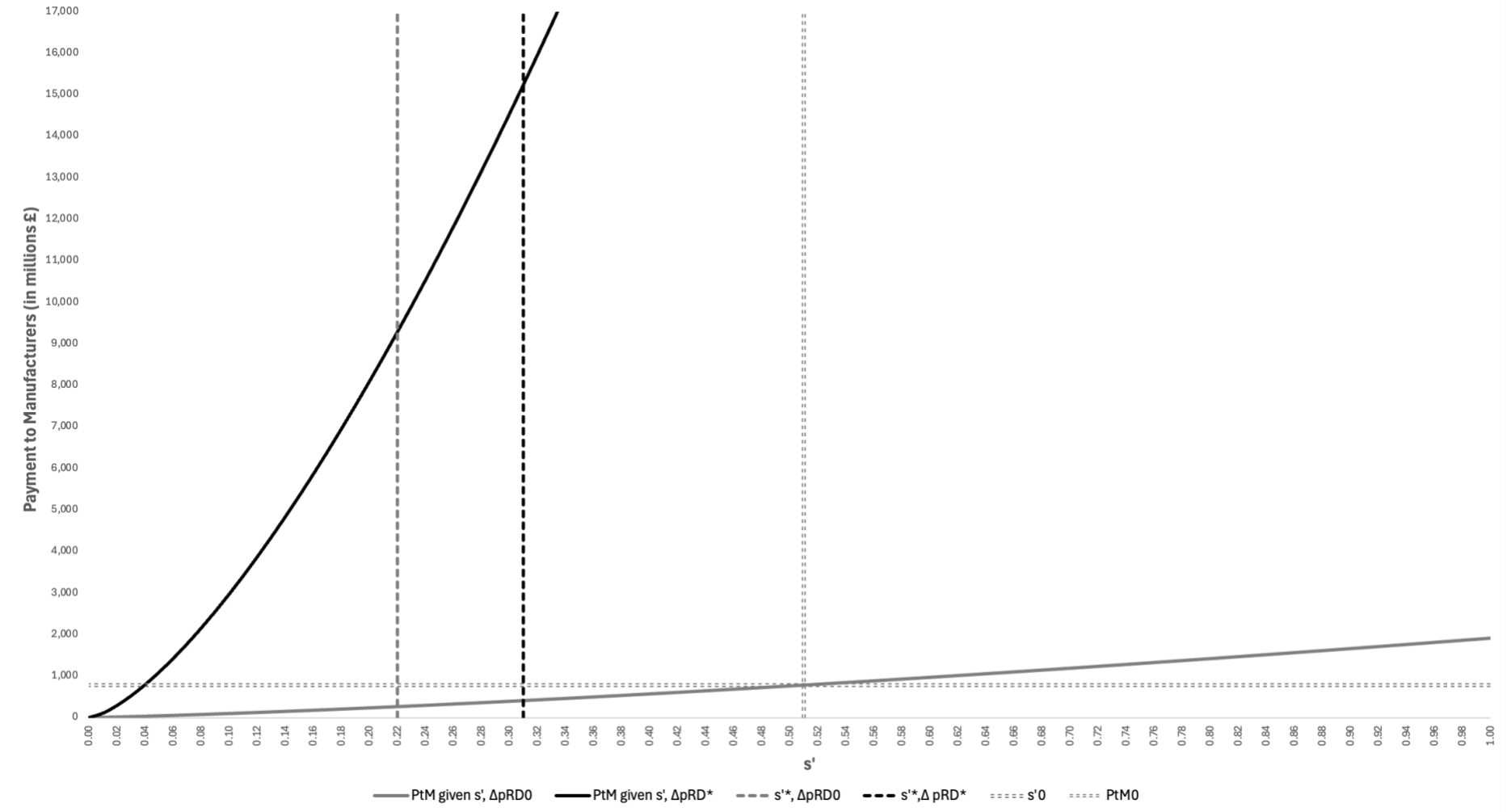


Figure A1-Scenario 3 (ε_1=0.23, 1-year pRD_{cost}). Population health gains under pull-only optimisation, push-only expansion, and joint pull-push optimisation.

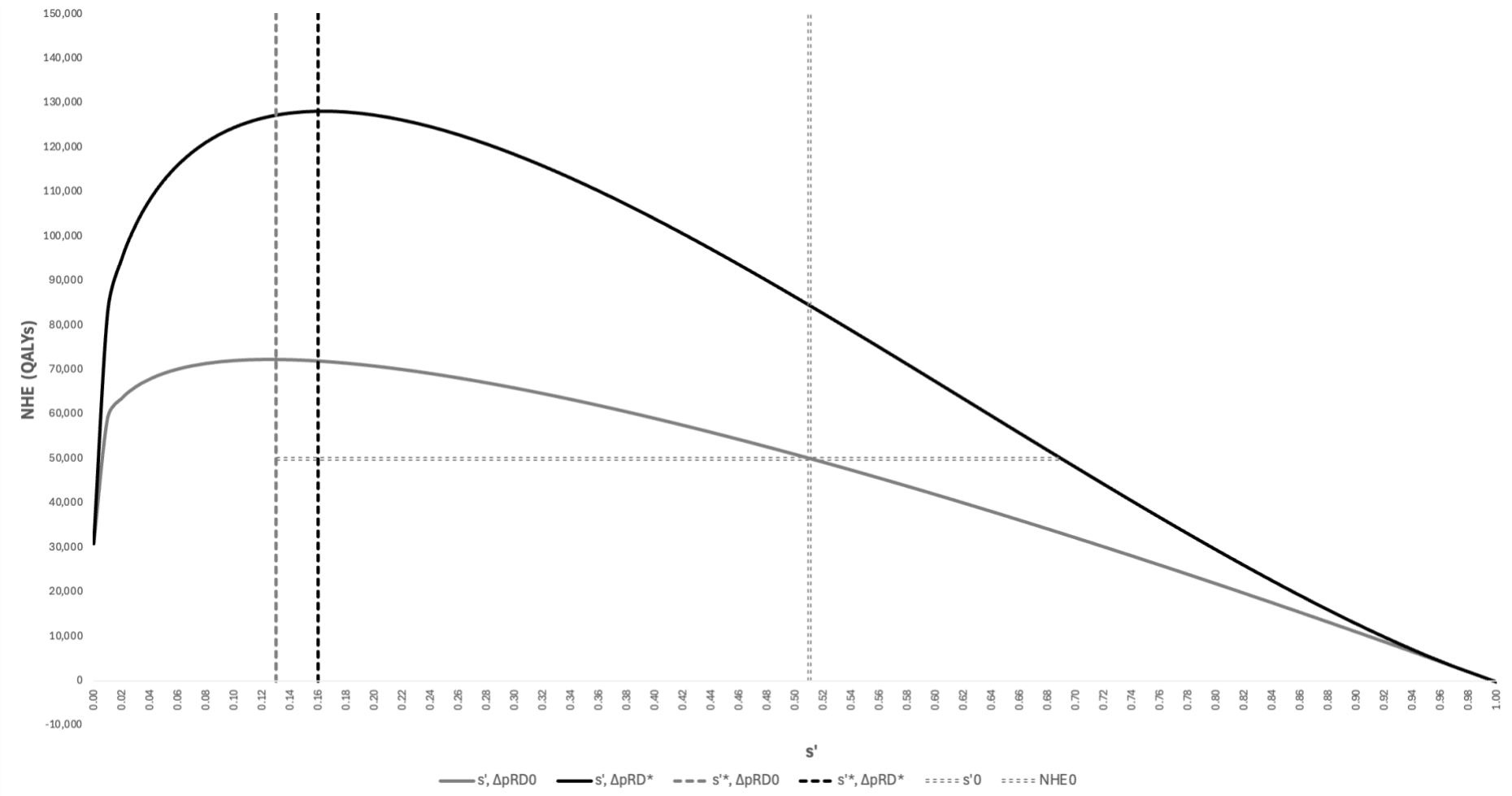


Figure A2-Scenario 3. Population health gains across changes in public R&D under current and optimised manufacturers' value shares.

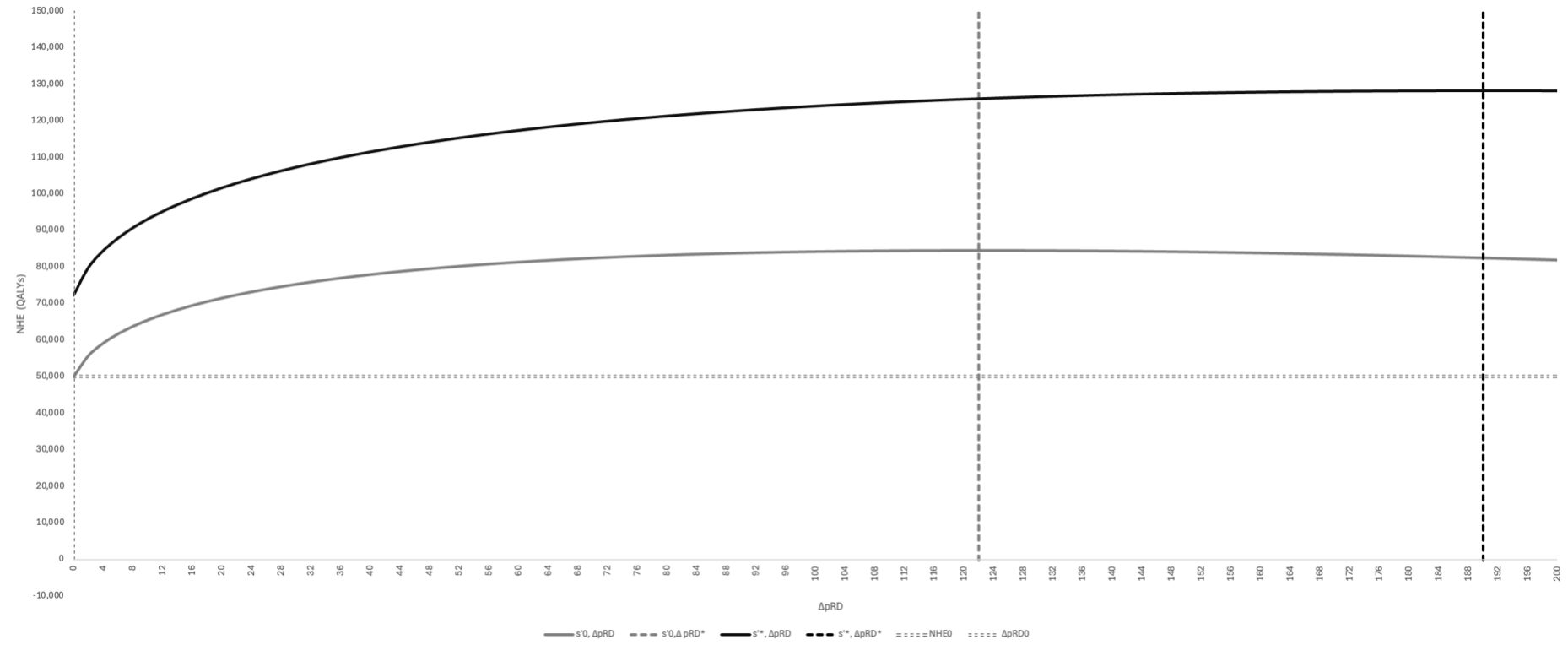


Figure A3-Scenario 3. Payment to manufacturers under pull-only optimisation, push-only expansion, and joint pull-push optimisation.

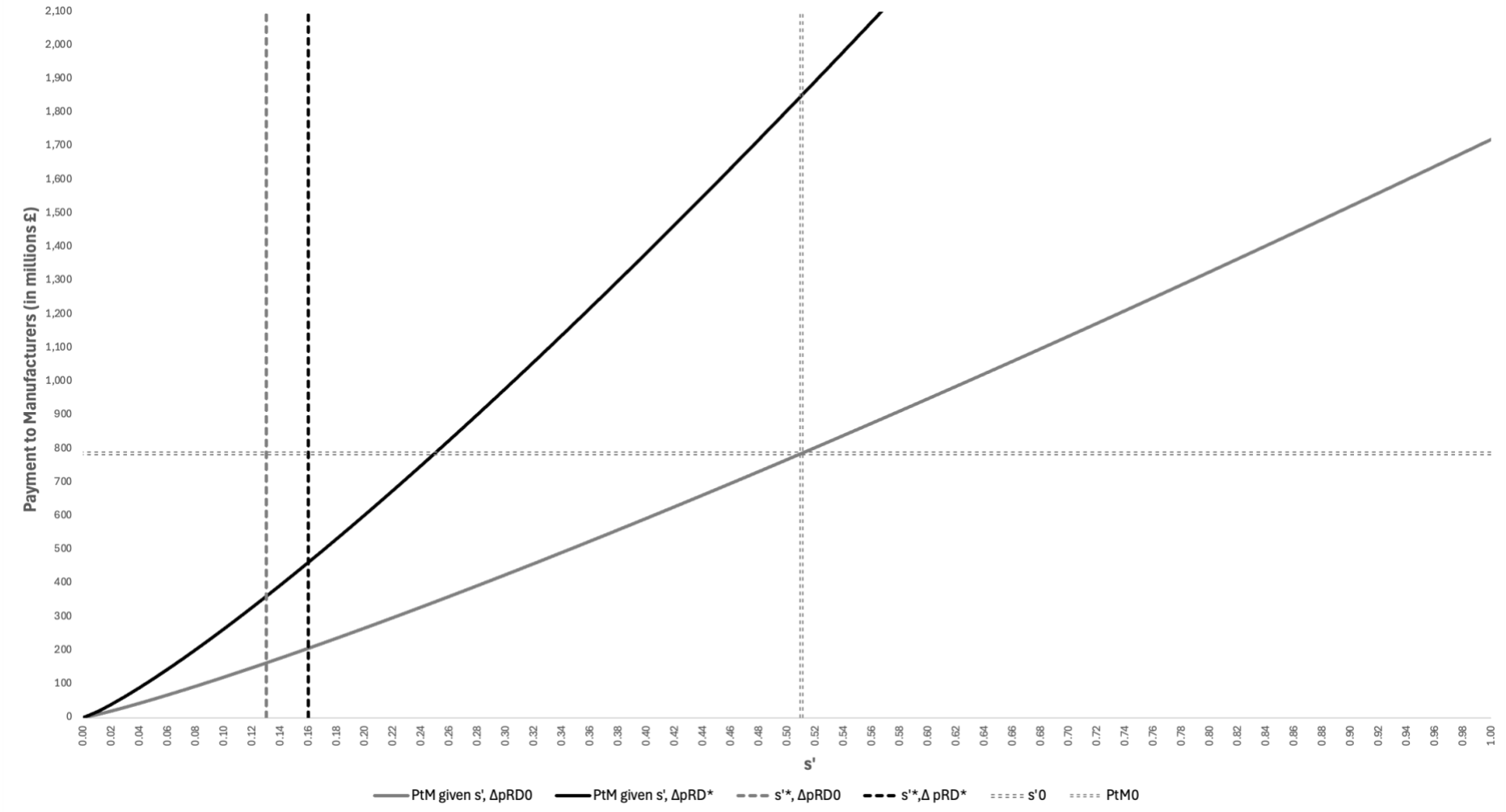


Figure A1-Scenario 4 (ε_1=0.23, Perpetual pRD_{cost}). Population health gains under pull-only optimisation, push-only expansion, and joint pull-push optimisation.

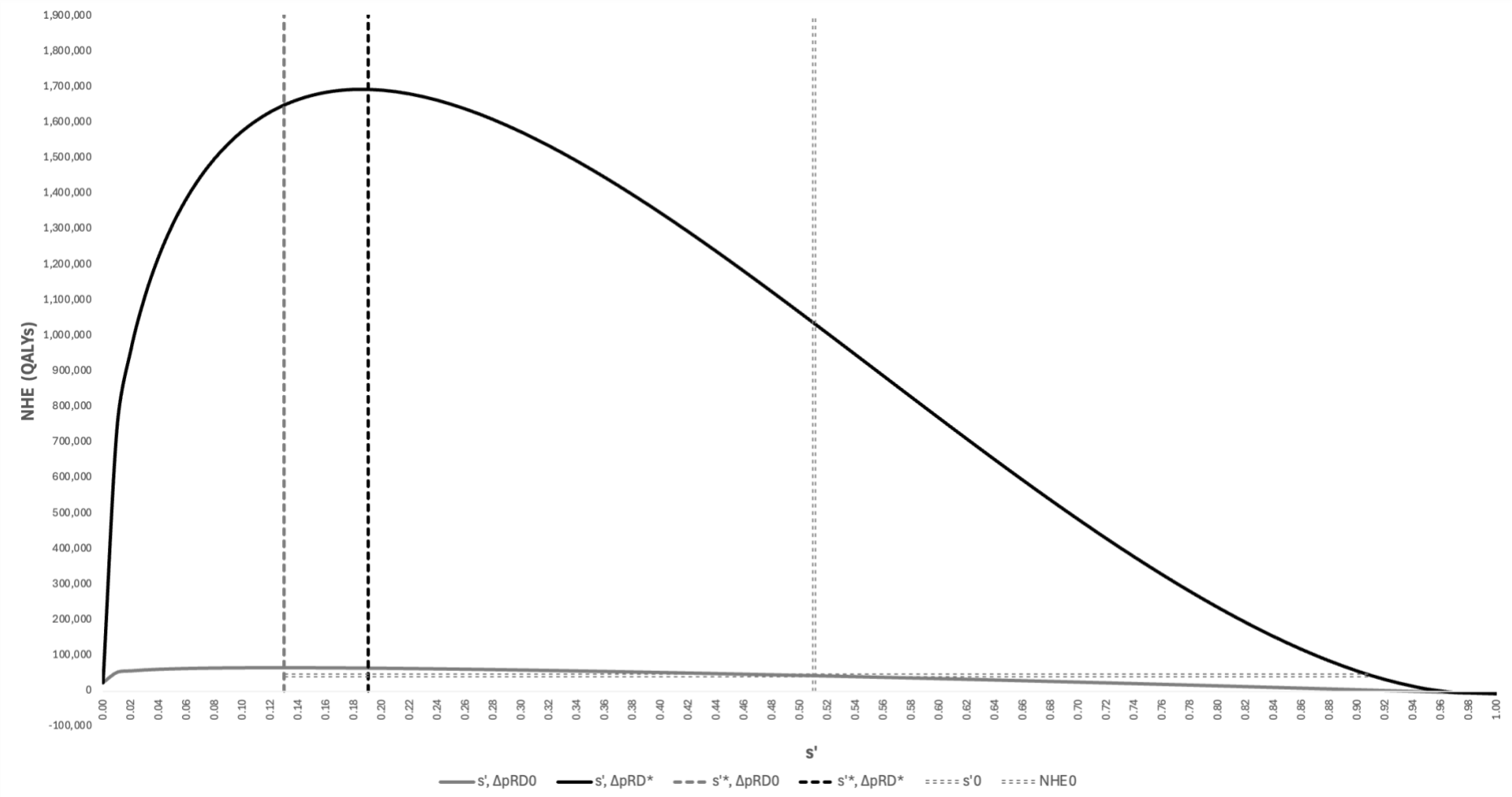


Figure A2-Scenario 4. Population health gains across changes in public R&D under current and optimised manufacturers' value shares.

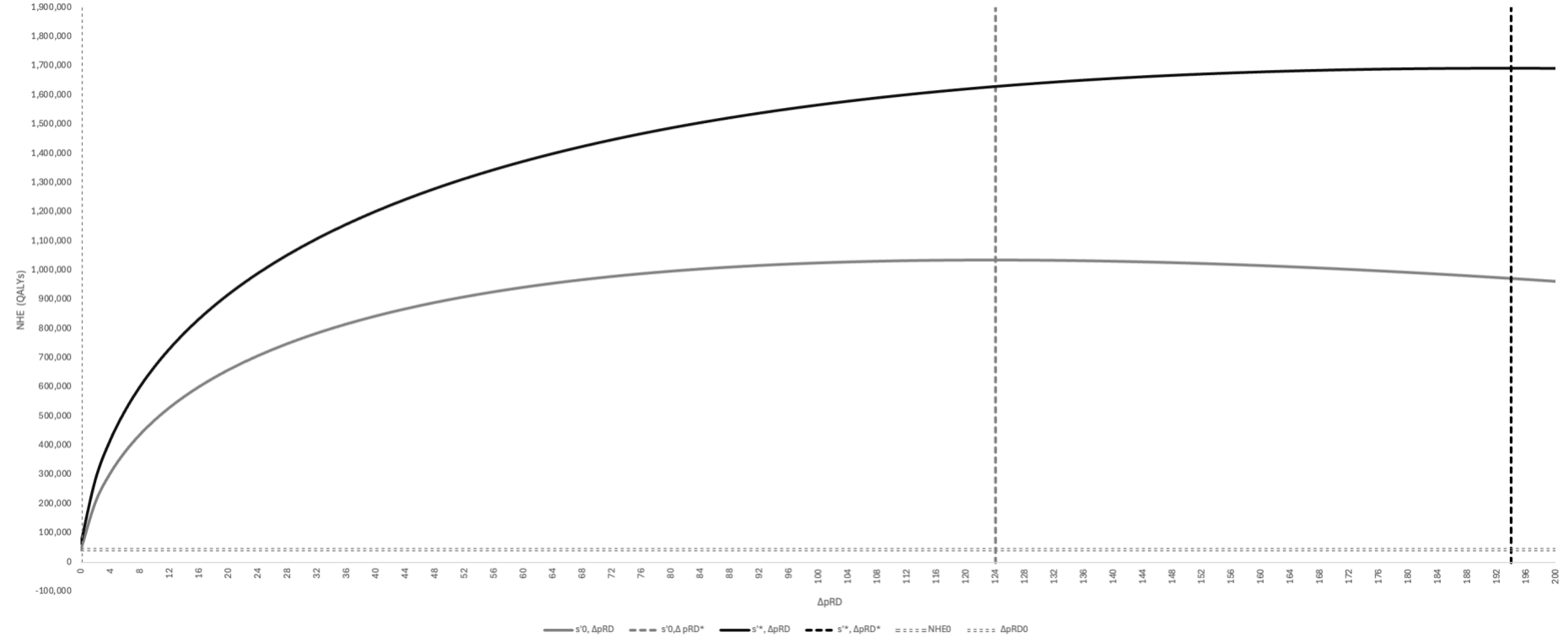
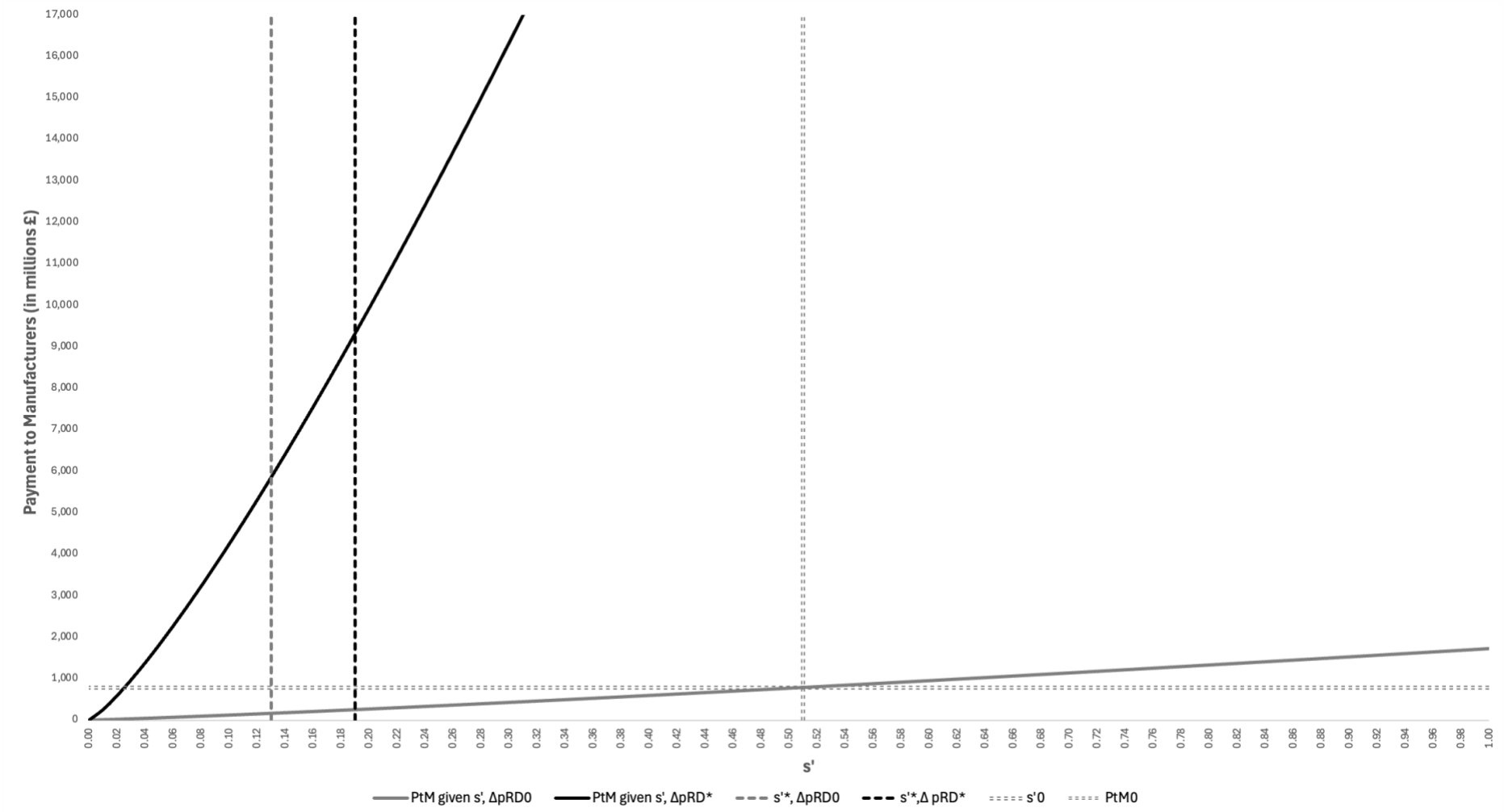


Figure A3-Scenario 4. Payment to manufacturers under pull-only optimisation, push-only expansion, and joint pull-push optimisation.



B. Structural mechanisms of $Td^{'}$

To understand how dynamic benefits $Td^{'}$ are generated, we move from a simple relationship to a system of structural equations that characterise the innovation production function. We assume that innovation is produced by both private (R) and public (pRD) efforts:

$$Td^{'} = \eta_0 \cdot R^{\eta_1} \cdot pRD^{\eta_2} \quad (6)$$

where η_1 and η_2 represent the marginal productivities of private and public R&D, respectively. We adopt a Cobb-Douglas specification for the innovation production function for theoretical and empirical reasons. First, it captures essential multiplicative complementarities between public and private R&D, reflecting the economic findings that private investment relies on public scientific knowledge. Second, its log-linear property allows elasticities to be mapped to established empirical estimates in the literature. The standard power form does not impose prior restrictions on returns to scale, providing a flexible structural framework that naturally accommodates empirically calibrated parameters

We also introduce an investment function, where private investment responds to manufacturers' revenue (payment received from the health system) and public investment:

$$R = \beta_0 \cdot p^{\beta_1} \cdot pRD^{\beta_2} \quad (7)$$

Here β_1 is the investment response to the pull incentive (payment), while β_2 represents the crowd-in effect, where public efforts make private investment more attractive. This private investment function follows established empirical models in the economics literature (Ward & Dranove, 1995; Toole, 2007; Sussex et al., 2016).

By substituting the investment function (7) into the production function (6), we derive the reduced form equation for $Td^{'}$:

$$
\begin{aligned}
Td' &= \eta_0 \cdot R^{\eta_1} \cdot pRD^{\eta_2} \\
&= \eta_0 \cdot (\beta_0 \cdot p^{\beta_1} \cdot pRD^{\beta_2})^{\eta_1} \cdot pRD^{\eta_2} \\
&= \eta_0 \cdot \beta_0^{\eta_1} \cdot p^{\beta_1\eta_1} \cdot pRD^{\beta_2\eta_1} \cdot pRD^{\eta_2} \\
&= (\eta_0 \cdot \beta_0^{\eta_1}) \cdot p^{(\beta_1\eta_1)} \cdot pRD^{(\beta_2\eta_1+\eta_2)} \\
&= \alpha' \cdot p^{\varepsilon_1} \cdot pRD^{\varepsilon_2}
\end{aligned}
\tag{8}
$$

This derivation of a reduced form allows us to observe the two types of incentives incorporated and the direct and indirect mechanisms behind. Payment only indirectly affects innovation via private investment ($\varepsilon_1 = \beta_1\eta_1$).

pRD can operate through two mechanisms: an indirect ($\beta_2\eta_1$), which is the crowd-in channel, and a direct (η_2), in which pRD can directly create pharmaceutical innovation.

C. Literature gap on ε_1 and structural adjustment

While Equation (8) establishes the theoretical boundaries separating the structural impacts of payment (ε_1) and public R&D (ε_2), a joint causal estimation of this two-lever system does not exist in the current empirical literature. Foundational work by Acemoglu & Linn, (2004) , quantified the relationship between potential market size, which acts as a proxy for the market payment incentive (p) and pharmaceutical innovation (Td'). However, their empirical framework focuses exclusively on pull incentives and does not instrument for public R&D, leaving the system susceptible to omitted variable bias (OVB) and simultaneity.

These two policy levers suffer from severe confounding due to their systematic correlation across therapeutic domains. This co-movement is driven by institutional and political design: public R&D allocations (e.g., NIH grants) and healthcare payment decisions target the same therapeutic areas. Empirically, we capture this alignment via the correlation parameter (δ) reflecting the degree to which public R&D co-moves with payment.

Prominent empirical estimations of the reward-innovation elasticity (Dubois, 2025; Dubois et al., 2015) employ instrumental variable (IV) strategies to address endogeneity. However, these IVs (e.g., demographic changes, disease mortality, or GDP per capita) do not resolve the structural omission of public R&D. Because public funding agencies (e.g., NIH grants) explicitly allocate R&D budgets based on disease burden and demographic priorities, these IVs fail the exclusion restriction; they correlate directly with omitted public research funding. Consequently, the IV estimator does not remove public R&D; it passes the combined variation of both reward and public research funding into the estimated coefficient, yielding a composite total elasticity rather than the isolated direct elasticity of reward. To recover the true direct elasticity of payment net of public research spillovers, we apply a classical econometric OVB correction (Greene, 1993), decomposing the total effect into its direct and indirect components:

$$\varepsilon_1 = \hat{\varepsilon}_1 - \left(\varepsilon_2 \cdot \delta\right) \tag{9}$$

Where $\varepsilon_2 = \beta_2 \eta_1 + \eta_2$. Drawing from the available empirical literature, we leverage the total effect of public R&D estimated by Azoulay et al., (2019) ε_2 = 0.381, and the correlation between UK-public R&D and global payment of δ = 0.5796, capitalising on the available data from Sussex et al., (2016). In their study, they compiled a uniquely standardised disease-level

dataset matching public and private research spending in the UK with global sales across therapeutic categories over the 1982-2012 period.

By operationalising this structural deduction using the payment elasticity of the CBO parameter (as used in the past (Woods et al., 2024)), our estimation yields:

$$\begin{aligned} \varepsilon_1 &= 0.45 - \left(0.381 \cdot (0.5796)\right) \\ &= 0.45 - 0.22 \\ &= 0.23 \end{aligned} \tag{10}$$

While this adjustment uses distinct data sources and should be interpreted as an indicative order-of-magnitude correction rather than a precise point estimate, it underscores that failing to account for public R&D efforts leads to estimations that overestimate the effect of reward.

D. Parameterisation of the dynamic benefits function (Td')

A desirable property of the model is that under a given payment policy environment (s'), there is a constant supply of new drugs over time. This results in a dynamic-to-static benefits ratio of γ =2.3, discounted at 3.5% per annum.

As Azoulay et al., (2019) focused on an exogenous, one-off shock of pRD with a complex profile of effects, it is difficult to parameterise how these shocks might deliver a constant stream of innovations or to adjust the scaling parameter (α') in Equation (4) accordingly. Thus, we propose to subsume the effects of baseline public R&D (pRD^0) within α', as in the original model. This leaves the original γ and α' unchanged and allows us to explicitly model only the effects of changing pRD:

$$Td' = \alpha' \cdot p'^{\varepsilon_1} \cdot (1 + \Delta pRD_{t=1})^{\varepsilon_2} \tag{11}$$

Where $\Delta pRD_{t=1} = \dfrac{pRD_{t=1} - pRD^0}{pRD^0}$ is the proportional change in pRD relative to the baseline level, where $\Delta pRD = 1$ means doubling the baseline budget. Note that we use the relative change, rather than the absolute one, to preserve the multiplicative nature of the Cobb-Douglas functional form. This ensures that the model remains structurally consistent. Consequently, whether ε_2 is measured against the total level of pRD or the relative change ($\Delta pRD_{t=1}$), the result is mathematically equivalent, as both describe the same proportional response of innovation to a marginal shift in public R&D. However, a potential limitation arises if we were to multiply the base function directly by the change itself; in a scenario where the relative change is zero, the entire dynamic benefits function would collapse to zero, which is theoretically inconsistent. To resolve this, we formulate the multiplier as $(1 + \Delta pRD_{t-1})^{\varepsilon_2}$. This guarantees model stability by ensuring that in the case of no change in public R&D ($\Delta pRD = 0$), the dynamic benefit function simplifies to $Td' = \alpha' \cdot p^{\varepsilon_1}$.

E. Derivation and integration of a common numeraire (λ)

Unlike the payment signal, pRD does not generate health value immediately, as it is subject to a translation lag and a temporal profile of innovation. To account for this, we incorporate three key empirical assumptions:

1. A single-year exogenous shock to pRD impacts patent yields over a 20-year window (Azoulay et al., 2019).
2. On average, a 12-year translational lag elapses between initial patent filing and the development of a New Molecular Entity (NME) ready for clinical use (Beall et al., 2019; Lexchin, 2020). Consequently, while the translation lag spans years t = 1 to t = 12, the resulting health benefits materialise starting in year t = 13 and extend through year t=32
3. The downstream NMEs scale proportionally with the upstream patent generation. While we acknowledge that the patent-to-NME ratio varies for several reasons (e.g., clinical success rates across therapeutic classes), this proportional relationship has been found in the empirical literature (Li et al., 2017).

To bring the pRD onto the same common numeraire scale as the payment one, we define a modified ratio of dynamic to static benefits of a one-year pRD shock. To calculate this scalar, we sum the present value of health benefits over the active 20-year patent yield window (t=13 to t=32), applying an annual discount rate of r=3.5% $(1/(1+r)^t)$, and divide this cumulative sum by the 12-year duration over which commercial market rewards are evaluated.

Formally, let $C_t \in \{0,1\}$ represent a binary benefit indicator taking a value of 0 during the translation lag $(t \in [1,12])$ and 1 when dynamic health benefits are realised $(t \in [13,32])$:

$$\gamma_{pRD}^{1year} = \frac{\sum_{t=13}^{t=32} \frac{C}{(1+r)^t}}{12} \tag{12}$$

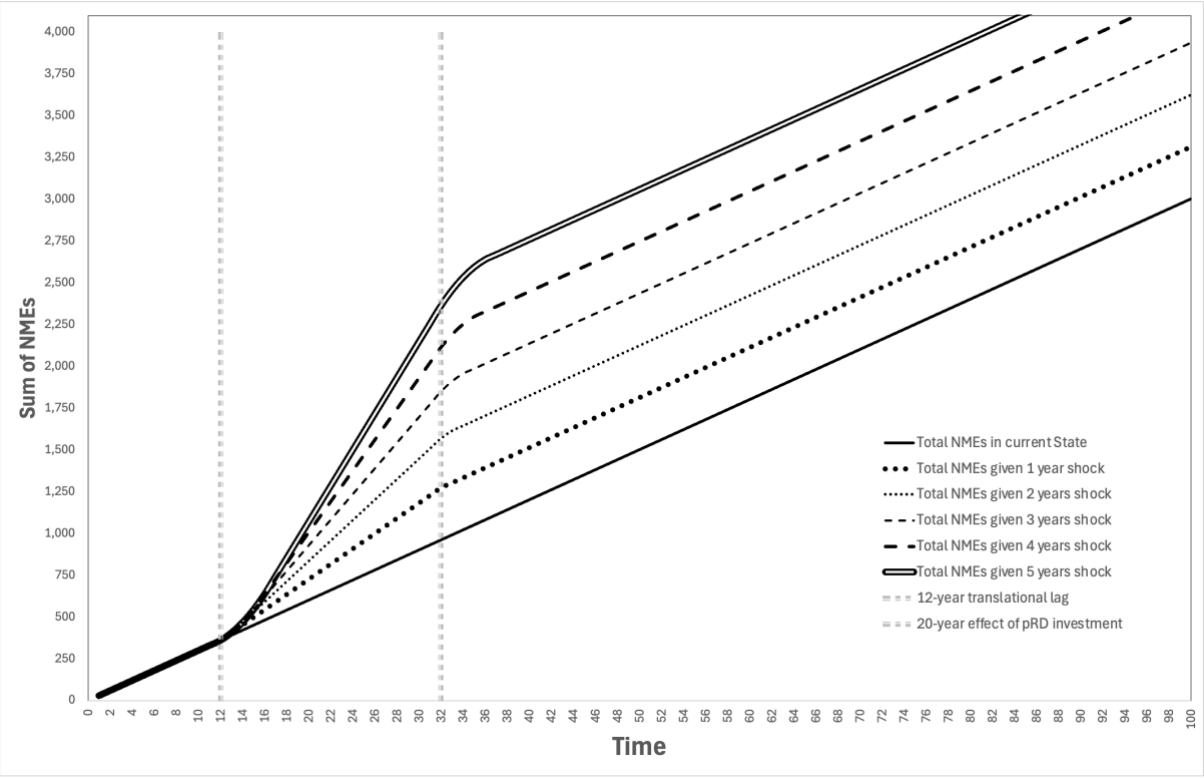
This equation computes the expected Net Present Value (NPV) of future drugs affected by a one-year public R&D shock relative to currently developed drugs over the baseline payment horizon. Evaluating this given the discount rate results in a $\gamma_{pRD}^{1year} \approx 0.78$.

Beyond a temporary funding shock, another relevant policy question relates to the broader evaluation of a long-term shift in public R&D. Thus, we assess an alternative scenario assuming a permanent commitment in pRD, by taking the value of a one-year shock divided by the discount rate,

$$\gamma_{pRD}^{\infty} = \frac{\gamma_{pRD}^{1year}}{r} \tag{13}$$

Based on the latter discount rate, this yields $\gamma_{pRD}^{\infty} \approx 22.39$. By formulating this as a perpetuity, we model a permanent policy shift as a continuous, consecutive sequence of single-year funding additive effects. As illustrated in Figure A1, because each annual investment yields a finite 20-year window increase in new drugs following a 12-year translation lag, sustained consecutive funding creates overlapping, cumulative cohorts of innovation. The perpetuity calculation assumes that these overlapping cohorts stack additively, meaning each year's public investment produces its own independent 20-year stream of health gains (e.g., independent constant returns to scale per cohort). We acknowledge that this additive behaviour is a simplifying assumption, made in the absence of direct empirical evidence regarding the dynamic effects of long-term funding shocks. Consequently, a multi-year commitment to public R&D generates a steadily compounding pipeline of dynamic benefits.

Figure A1. Cumulative New Molecular Entities (NMEs) over time under five consecutive multi-year public R&D funding shocks



Note: Simulated cumulative New Molecular Entities (NMEs) over a 100-year horizon across baseline and 1- to 5-year public R&D funding shocks of $\Delta pRD = 2$. During the initial 12-year translation lag, all shock trajectories remain identical to the baseline control. Beginning at t = 13, a single-year funding shock elevates the annual discovery rate across a finite 20-year innovation window (t = 13 to 32), steepening the cumulative trajectory. Once all 20-year cohort windows expire, the annual discovery rate reverts to the baseline slope, leaving the cumulative stock of NMEs permanently elevated above the counterfactual scenario.

To directly compare this cumulative, long-term impact of public science against the immediate effects of reward, we define a common numeraire (λ). Importantly, this numeraire can also be derived specifically for a one-year funding shock, and either the perpetual or single-year derivation can be fed into the dynamic benefit equation. This parameter is calculated as the ratio of the total perpetual value generated by public R&D (γ_{pRD}^{∞}) to the baseline value generated by the reward signal (γ =2.3, as defined in Woods et al., (2024))

$$\lambda = \frac{\gamma_{pRD}^{\infty}}{\gamma} \tag{14}$$

Deriving this common numeraire allows us to harmonise the intrinsically different timing horizons of both policy levers onto a single scale for Td'. While a permanent shift in payment policy impacts a continuous pipeline of baseline innovation, public R&D influences

future innovation through the stacking effects of consecutive scientific discoveries. Thus, we specifically modelled a one-year shock to public R&D in reflection of the Azoulay et al., (2019) evidence, whereas the evidence on payment is long-term.
Based on our empirical parameters, $\lambda \approx 9.74$ (22.39 / 2.3). Note that the effects of a single year shock are shorter in duration and offer a lower lambda ($\gamma_{pRD}^{1year} \approx 0.78$). This larger magnitude shows that, on a perpetual basis, the cumulative dynamic health returns of expanding public R&D are significantly larger than the dynamic effects of adjusting pull incentives alone, precisely because of the compounding pipeline illustrated in Figure A1.

Integration of the common numeraire (λ) to the Td' function

We use this common numeraire to adjust the dynamic benefits function, which, as currently stands, implicitly assumes that the value created due to pRD is on the same timing horizon as p'. We make this adjustment through a two-step structural transformation. First, we isolate the net additional effect of innovation driven specifically by increases in public R&D by subtracting 1 from the gross multiplier, yielding $\left[(1+\Delta pRD_{t=1})^{\varepsilon_2} - 1\right]$. The subtraction takes away baseline innovation so that the common numeraire (λ) rescales strictly the incremental dynamic gains generated by public R&D, preventing the numeraire from improperly scaling the effects of payment. Second, to reconstruct total dynamic health benefits, this rescaled net growth is added back onto the baseline level of innovation (represented by the leading $+1$). This allows an evaluation of the push and pull trade-off on a unified scale while ensuring that if no change in public funding occurs $(\Delta pRD = 0)$, the equation collapses cleanly back to the original dynamic benefits function $Td' = \alpha' \cdot p'^{\varepsilon_1}$,

$$Td' = \alpha' \cdot p'^{\varepsilon_1} \cdot \left\{1 + \lambda\left[\left(1+\Delta pRD_{t=1}\right)^{\varepsilon_2} - 1\right]\right\} \quad (15)$$

F. Public R&D cost

In our model, public R&D costs represent the health opportunity cost of its investment in pRD. We explore two timing-related scenarios for how public R&D is funded: a one-time upfront investment and a continuous investment sustained in perpetuity.

To parameterise these scenarios within an annual framework, we derive the annualised per-drug financial cost of public R&D in three sequential steps:

1) Deriving the annualised payment per drug $C^{1\text{year}}$

We first calculate the annual payment made to the manufacturers per drug. To do this, we equalise the total monetary health value captured by the manufacturer ($s^0 \cdot Tb \cdot k$) with the discounted stream of annualised payments.

$$s^0 \cdot Tb \cdot k = \sum_{t=1}^{t=12} \frac{C}{(1+r)^t} \tag{16}$$

Factoring out the 12-year annuity term isolates the annual payment $(C^{1\text{year}})$:

$$C^{1\text{year}} = \frac{s^0 \cdot Tb \cdot k}{\frac{1-(1+r)^{-12}}{r}} \tag{17}$$

2) Scaling to public R&D via empirical proportions (ω):

Next, using the most recent empirical data (CBO, 2021), we obtain the baseline ratio of public sector biomedical research investment relative to pharmaceutical revenues, denoted as ω. Multiplying the annual payment by this proportion yields the baseline annual per-drug public R&D investment/expenditure.

3) Parameterising policy scenarios (1-year funding vs perpetuity)

Finally, we scale this baseline expenditure by the public R&D policy shock (ΔpRD). For the single-year funding scenario, the annualised financial cost of public R&D is expressed as:

$$pRD_{\text{cost}}^{1year} = \left[(\omega \cdot C^{1year}) \cdot (1 + \Delta pRD_{t=1}) \right] \tag{18}$$

For the second scenario, the investment is modelled directly as an ongoing, continuous annual financial flow. Thus, the financial cost of public R&D for each respective scenario is defined as:

$$pRD_{\text{cost}}^{\infty} = \frac{pRD_{\text{cost}}^{1\,year}}{r} \tag{19}$$

Finally, the health opportunity cost of pRD is obtained by dividing these financial costs by the cost-effectiveness threshold (k).

In the current state, where investments are at the baseline level (there is no change) in public R&D, $pRD_{t=1} = pRD^{0}$, which means that $\Delta pRD_{t=1} = 0$.

G. Derivation of maximum optimal share (s'^{*U})

To determine the theoretical upper bound of the share of value offered to manufacturers $\left(s'^{*U}\right)$, we evaluate the model under the extreme assumption that dynamic benefits (Td') dominate static benefits (Tb), rendering current static gains negligible $\left(Tb \to 0\right)$.

Recall the extended Net Health Effect objective function:

$$NHE(s', pRD) = \left(1 - s'\right)(Tb + Td') - \left(\frac{pRD_{\text{cost}}}{k}\right) \tag{20}$$

Differentiating this equation with respect to the manufacturer value share (s') yields the unconstrained First-Order Condition (FOC):

$$\frac{\partial NHE}{\partial s'} = (1 - s')\left(\varepsilon_1 \frac{Td'}{s'}\right) - (Tb + Td') = 0 \tag{21}$$

Under the boundary condition where dynamic benefits are unbounded relative to static benefits $\left(Tb \to 0\right)$, the equation simplifies to the condition governing the maximum optimal share:

$$\left(1 - s'^{*U}\right)\left(\varepsilon_1 \frac{Td'}{s'^{*U}}\right) - Td' = 0 \tag{22}$$

Dividing Equation (22) through by Td', since $Td' > 0$:

$$\left(1 - s'^{*U}\right)\left(\frac{\varepsilon_1}{s'^{*U}}\right) - 1 = 0 \tag{23}$$

Rearranging Equation (23) algebraically:

$$\begin{aligned} &\left(1 - s'^{*U}\right)\varepsilon_1 = s'^{*U} \\ &\varepsilon_1 - s'^{*U}\,\varepsilon_1 = s'^{*U} \\ &\varepsilon_1 = s'^{*U}(1 + \varepsilon_1) \end{aligned} \tag{24}$$

Solving explicitly for s'^{*U},

$$s'^{*U} = \frac{\varepsilon_1}{1 + \varepsilon_1} \tag{25}$$

Equation (25) is mathematically identical in functional form to the maximum share derived in Woods et al., (2024). This confirms that formally incorporating public R&D push incentives into a joint production framework does not alter the theoretical maximum value share that can be offered to manufacturers under boundary conditions.

H. Derivation of Marginal Costs per QALY (MCpQ)

Given total monetary cost $C = s' k(Tb + Td') + pRD$ and the dynamic benefit function $Td' = \alpha' \cdot p'^{\varepsilon_1} \cdot \left\{1 + \lambda\left[\left(1 + \Delta pRD_{t=1}\right)^{\varepsilon_2} - 1\right]\right\}$, the marginal cost per unit of dynamic innovation $(MCpQ_X)$ for any policy lever $X \in \{s', pRD\}$ is:

$$MCpQ = \frac{\frac{\partial C}{\partial X}}{\frac{\partial Td'}{\partial X}} \tag{26}$$

- MCpQ for manufacturers' share:

$$MCpQ_{s'} = \frac{k(Tb + Td') + s' \cdot k(\varepsilon_1 \frac{Td'}{s'})}{\varepsilon_1 \frac{Td'}{s'}} = k \cdot s' \left[\frac{1}{\varepsilon_1}\left(\frac{Tb}{Td'} + 1\right) + 1\right] \tag{27}$$

- MCpQ for public R&D:

$$MCpQ_{pRD} = \frac{s' \cdot k \cdot \frac{\partial Td'}{\partial pRD} + 1}{\frac{\partial Td'}{\partial pRD}} = k \cdot s' + \frac{pRD_{\text{cost}}}{\varepsilon_2\left[Td' - \alpha\left(p'\right)^{\varepsilon_1}\left(1 - \lambda\right)\right]} \tag{28}$$