Demand for New Treatments and Cost Containment in the English National Health Service

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Abstract

This paper examines GP prescribing behavior for on-patent diabetes therapies in NHS England and quantifies their short-run impact on secondary-care use. Leveraging a rich practice-level panel and a structural demand framework that treats GPs as perfect patient agents, we instrument net price with exogenous rebate shocks under four alternative specifications. Implied own-price elasticities of uptake lie between –0.2 and –0.6, with smaller practices exhibiting greater sensitivity. Cross-price elasticities between the new-treatment class and legacy therapies are close to zero. A 2SLS IV estimation then links a one-percentage-point increase in the new-treatment share to a 4 percent rise in diabetes-related hospital admissions over two years—equating to roughly 0.25 additional episodes per practice. These results challenge the prevailing view that innovative therapies relieve secondary-care pressures, instead revealing meaningful short-term cost pressures. By integrating structural demand estimation with real-world outcome monitoring, our study offers a practical blueprint for evaluating the system-wide cost-effectiveness of novel treatments in universal health-care settings.

Keywords: pharmaceutical demand; structural demand model; GP prescribing; diabetes treatments; price elasticity
JEL Codes: D12, I11, L65

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1 Introduction

The health-care sector exhibits two well-documented stylised facts. First, sustained innovation in pharmaceuticals and related medical technologies has shifted the health-production frontier outward, yielding sizeable gains in life expectancy and quality-adjusted life-years.

Second, real expenditure on health has risen persistently: in the United Kingdom it grew from roughly £40 billion in 1987 to about £140 billion in 2017, an average annual growth rate of 3.9 per cent. Total health expenditures in the United States increased from around $644 billion dollars in 1989 to $3,205 billion dollars in 2015. Furthermore, the upward trajectory shows little sign of abating.

The literature points to three proximate mechanisms behind this trend. Population ageing extends the period in which individuals live with chronic morbidity; rising incomes raise willingness-to-pay for health; and most prominently, technological progress introduces new, high-value but high-cost treatments. Empirical evidence attributes the bulk of spending growth to the technology channel—both directly, via higher utilisation of costly therapies, and indirectly, via the longevity gains they generate (Skinner and Staiger, 2015). Because expected market size shapes firms' incentives to innovate, demographics further amplify technology adoption (Acemoglu and Linn, 2004). Understanding the demand for newly launched drugs is therefore central to any explanation of long-run growth in health-care expenditure.

This paper investigates the demand for newly introduced drugs in NHS England, emphasising not only the direct budgetary impact of higher uptake but also the spill-over effects on resource use elsewhere in the system. In particular, we explore how the diffusion of innovations shapes overall expenditure growth. By coupling market-level adoption patterns with patient-level utilisation data, we provide a real-world evaluation of treatment adoption beyond the confines of clinical trials and quantify how accelerated uptake amplifies downstream spending pressures.

This work links to the wider literature on the healthcare demand originating with Arrow (1963), which first sets out the nature of the demand for healthcare given the uncertainty of the incidence of the disease and the uncertainty of the efficacy of the product for different groups of patients. This seminal work has spawned various lines of investigation. Cameron, Trivedi, Milne, and Piggott (1988) developed an interdependent demand for health insurance and health care under uncertainty to shed light on the issue of insurance-induced distortions in the demand for health care services.

Subsequent work in this wider literature primarily focuses on the demand for healthcare insurance and implications for healthcare costs, such as Finkelstein (2007). Weisbrod (1991) first notes that the state of technology and demand for healthcare are endogenously determined, and the implications for costs. Relatedly, Acemoglu and Linn (2004) finds a strong relationship between the size of the market on the introduction of new drugs and pharmaceutical innovation. However, since then there appears to be a gap in the literature in terms of formalising Weisbrod's insight on the implication of the endogeniety of healthcare technology and demand on cost containment.

Limited work has been done on the demand for new pharmaceutical products, and the cost implication for health systems specifically.

However, there have been attempts to estimate the demand for specific pharmaceutical products such as Ellison, Cockburn, Griliches, and Hausman (1997). They modelled demand for four cephalosporins and computed the elasticities of the price between the branded and generic versions of the four drugs as a multistage budgeting problem for the consumer. Berndt (2002) provides a comprehensive review of the literature and issues related to pharmaceutical pricing and demand in the US system. More recent work in this line includes Crawford and Shum (2005) develop a dy-

namic matching model that examines demand amidst uncertainty, where patients gain knowledge on the effectiveness of various medications through their prescription history. However, their work does not explore implications for health systems where cost containment is a consideration.

Most closely related to my research Dubois and Lasio (2018) present a structural estimation model to analyse the effects of pharmaceutical price regulation on demand and manufacturers' price-setting behaviour in France. The study focuses on the anti-ulcer drug market and examines the impact of price constraints on drug consumption and price-cost margins. The authors estimate price-cost margins in a regulated market with price constraints and determine whether these constraints are binding. They compare cost restrictions across drugs in price-constrained markets (France) with unregulated markets (United States and Germany) to infer the binding nature of the constraints.

Their paper contributes to broader research on the demand for pharmaceuticals employing a structural estimation approach to estimate preferences for drugs and substitution patterns. It also explores the role of advertising in affecting demand for pharmaceuticals, with detailing found to be the most effective advertising strategy for increasing sales. The study finds that branded drugs are preferred over generics in all three countries analysed (France, United States, and Germany), although the effect is not significant in the United States.

A complementary strand of research analyses the market-level diffusion and budgetary impact of newly introduced pharmaceuticals and the role of industry structure in shaping these dynamics. Acemoglu and Finkelstein (2008) show how regulated input prices influence providers' technology choices, thus affecting the pace of drug adoption in health-care delivery. Gaynor, Ho, and Town (2015) survey the industrial organisation of health-care markets, emphasising how insurer and provider concentration conditions the speed and breadth of diffusion. At a more macro level, Lo

and Thakor (2023) review how financial intermediation and public R&D financing underpin the pace of biomedical innovation and new-drug launches.

In the clinical area of hepatitis C, a rich clinical–economic literature documents the rapid uptake and real-world diffusion of direct-acting antivirals, alongside detailed cost-effectiveness and budget-impact analyses that highlight tensions between high therapeutic value and system affordability. For example, see Pecoraro, Banzi, Cariani, Chester, Villa, D'Amico, Bertele', and Trenti (2019); Najafzadeh, Andersson, Shrank, Krumme, Matlin, Brennan, Avorn, and Choudhry (2015); Chhatwal, Kanwal, Roberts, and Dunn (2015); pharmaphorum (2014). However, these studies stop short of modelling prescriber behaviour or quantifying spill-over effects on service demand elsewhere in a publicly funded system.

There is also a burgeoning literature on contracting relationships in the English National Health Service (NHS) and the dual incentives faced by agents for both quality of care and cost containment, e.g. Beckert (2018). However, in this paper, I assume that the general practitioner is a perfect agent for the patient.

Recent related literature on this topic where the focus of investigation is the NHS includes Serra-Sastre and McGuire (2013), who look at how product information impacted the flows of new medicines in the NHS. Similarly, Bhattacharya and Vogt (2003) builds a model of pharmaceutical pricing that predicts a pattern of increasing prices and decreasing promotional activities over the life cycle of a drug. However, very little attention has been paid in the literature to the question addressed in this paper and focused on a setting such as NHS England.

A key focus is the tension between the long-run value of new therapies and their short-run implementation and monitoring costs. In primary care, adopting an on-patent drug can increase clinical monitoring, follow-up and referral activity (and associated resource use) that sits outside the prescribing line item. As a result, slow diffusion can be institutionally rational even when a new medicine is clinically

valuable.

This paper makes three contributions. First, it documents diffusion patterns for newly introduced non-insulin antidiabetic molecules in NHS England and estimates structural demand models to recover substitution patterns and price sensitivity. Second, it uses cost-side variation in reimbursement tariffs and rival-price movements to instrument for price in the demand system, and then reuses these same cost-side movements to instrument for practice-level adoption when estimating spillovers to hospital admissions. Third, it discusses implications for cost containment and for policy designs that aim to accelerate adoption while accounting for cross-sector spillovers.

The rest of this paper is organised as follows. To begin with, section 2 outlines the policy and regulatory framework surrounding the introduction and prescription of new drugs in NHS England, followed by 3 which details the data utilised in this analysis. In Section 4, I set out some descriptive statistics and trends in prescribing volumes and costs, their drivers, and implications for the health service. Section 5 sets out a basic model for the demand for new drugs in NHS England, where the prescriber pools consumer demand. Section 6 describes the demand estimation approach and Section 7 presents the results. Section 8 sets out the estimation approach for estimating the impact of new treatments on hospital referrals and cost containment in secondary care and finally, Section 9 & 10 sets out policy implications and concluding remarks.

2 Policy and Regulatory Context

The pharmaceutical market is distinctive due to the presence of several intermediary agents. Typically, this market includes the following participants: 1) the patient, who directly consumes the medication; 2) the prescriber or General Practitioner (GP), who acts on behalf of the patient and views medications as a means to enhance the

patient's health; 3) insurers, who generally cover the majority of the drug costs; and 4) pharmacists, who determine which version of a medication to dispense, fulfill the prescription, and often provide patients with health advice along with information regarding the drug's effects, usage, and potential side effects (Schweitzer and Lu, 2018).

In England, consumer preferences play a limited role in the demand for prescription pharmaceutical products. Note that we do not consider over-the-counter medicines in this paper. There is no direct-to-consumer advertising of prescription pharmaceutical products, and the demand for a certain drug is largely driven by patient needs determined by the prescriber, which is usually the General Practitioner (GP) in community care, which is the setting of this paper. In England, GPs have three main objectives; 1) they play a gatekeeping role by managing access to wider healthcare services, 2) they manage long-term chronic conditions such as diabetes in primary care, and 3) they are tasked with reducing prevalence of long-term chronic conditions (prevention of disease).

GPs are members of a larger group of health care provider partnerships, known as Integrated Care Systems (ICSs). Each ICS has an Integrated Care Board (ICB) that is responsible for managing and planning health services within that ICS and can issue varying prescribing guidelines to their members. However, the overall framework and clinical guidelines for new drugs, including those under patent, are developed by the National Institute of Care and Excellence (NICE) and the Regulatory Agency for Medicines and Healthcare Products (MHRA). NICE evaluates the cost effectiveness of medicines and recommends medicines that it believes offer good value for money for NHS England, while the MHRA is largely concerned with the efficacy and safety of medicines before giving market authorisation.

There are two main cost containment measures in place at the national level. First, NICE's cost-effectiveness assessment, where a new medicine is only recommended for use in the NHS if the cost per standardised health unit, Quality Adjusted Life Years (QALYs), is less than £30,000. Second, a voluntary scheme negotiated between pharmaceutical companies and the government is in place, which caps the overall spend on branded pharmaceutical products at 2% per year However, for individual products, there is freedom of pricing.

In addition, the complex commissioning arrangements in NHS England devolve the primary care medicines budget (along with all other health commissioning) to ICSs and hence General Practice, while setting a national reimbursement price for prescription medicines. Prescriptions are dispensed at pharmacies in the community, who are reimbursed at the national price which is set to ensure that pharmacies make an agreed margin on medicines to ensure supply via wholesalers and manufacturers. Some new medicines, particularly for acute conditions, are prescribed and administered in hospitals, which is outside the scope of this paper. Approximately £8 billion is spent on pharmacuetical products in primary care and £5 billion is spent in hospitals. About 70% of this spending is accounted for by branded pharmaceutical products, which are frequently on-patent as well[1].

Pharmacological Innovation and Market Entry of Type 2 Diabetes Therapies

This paper investigates new treatments for diabetes, emphasising the need to first outline the range of pharmacological options available for prescribers and the timing of their introduction to the market. Table 1 provides a summary of the main non-

[1] Prescriptions Dispensed in the Community, NHS Digital

insulin drug classes, detailing their fundamental mechanisms of action and the year that the first product in each class received marketing authorization in Europe (or in the UK for older agents). The timeline reveals three significant "waves" of innovation: (i) the traditional insulin-sensitizers and secretagogues from the 1950s to the 1970s; (ii) the insulin-sensitizing thiazolidinediones introduced in the late 1990s; and (iii) the incretin-based and renal-glucose-targeted medications that have been prominent in the market since 2006. The latest wave includes dipeptidyl-peptidase-4 (DPP-4) inhibitors, glucagon-like-peptide-1 (GLP-1) receptor agonists, and sodium-glucose co-transporter-2 (SGLT2) inhibitors, which represent the new treatment options available.

Implications for demand estimation. From a demand-system perspective, the three newest classes share two defining economic features. First, they are still on-patent, with prices well above the marginal cost of legacy generics such as metformin and sulfonylureas. Second, they deliver clinically differentiated benefits—weight loss (GLP-1 RAs, SGLT2is), cardio-renal protection (GLP-1 RAs, SGLT2is) or low hypoglycaemia risk (DPP-4is)—that expand the effective product space and relax therapeutic capacity constraints. As a result, prescriber substitution patterns cannot be captured by a simple price-only logit; instead, heterogeneous responsiveness to these quality attributes must be allowed for in the structural demand model developed in Section 5.

In the empirical analysis below, we treat the drugs launched in the last five years as the relevant set of *new treatments*. These are largely drugs in the SGLT2 and GLP1 group. Their staggered arrival provides quasi-experimental variation in choice sets over time, while the coexistence of inexpensive incumbents ensures meaningful price competition—an ideal setting for evaluating the elasticity of demand for pharmaceutical innovation within the National Health Service.

Table 1: Major non-insulin antidiabetic drug classes, mechanism of action and EU/UK launch dates

Class	**Principal mechanism of action**	**First launch**
Biguanides (Metformin)	Decreases hepatic gluconeogenesis and increases peripheral insulin sensitivity	1957
Sulfonylureas (e.g. Tolbutamide)	Stimulate pancreatic β-cell insulin release via K_{ATP}-channel closure	1956
Meglitinides (Repaglinide)	Rapid, short-acting insulin secretagogue acting on K_{ATP} channels	1998
α-Glucosidase inhibitors (Acarbose)	Delay intestinal carbohydrate absorption by inhibiting brush-border α-glucosidases	1990
Thiazolidinediones (Pioglitazone)	Peroxisome-proliferator–activated receptor-γ (PPARγ) agonist improving insulin sensitivity	2000
DPP-4 inhibitors (Sitagliptin)	Inhibit DPP-4, prolonging endogenous incretin (GLP-1, GIP) activity to enhance glucose-dependent insulin release	2007
GLP-1 receptor agonists (Exenatide, Semaglutide)	Mimic GLP-1, stimulating insulin secretion, suppressing glucagon, slowing gastric emptying and reducing appetite	2007
SGLT2 inhibitors (Dapagliflozin)	Block renal SGLT2, increasing urinary glucose excretion and producing mild osmotic diuresis	2012

Next, I present the data used in this paper and present some descriptive analysis focusing on the market for anti-diabeties drugs (excluding insulin) to motivate my research.

3 Data

Data from a variety of sources are linked and matched to create a novel dataset. First, I use primary care prescribing data that are available at the General Prac-

tice (GP) level on a monthly basis [2]. The prescribing data contain information at the drug formulation level, including drug reimbursement prices and monthly quantities prescribed by the GPs for each drug formulation. I aggregate these data up to the chemical/molecular substance level and identify, using matched marketing authorization and patent data, which treatments are newly launched and which are not. Information on the therapeutic area that these treatments treat is also available in the marketing authorisation data and from the British National Formulary (BNF) taxonomy. I match these data with data on prevalence and demographics of patients at the GP level.

I match the above data set with data on marketing authorizations between 2013 and 2019. This data set contains information for generic medicines or patent brands and has data on when these medicines were granted marketing authorisation in the UK, when the license holder applied for marketing authorisation and what high-level condition this medicine is licensed to treat. Finally, these data are matched with patent data over the same period to identify new on-patent treatments. [3].

Since I only focus on the anti-diabetes market in this essay, there are 40 unique treatments for Type II diabetes and 14 of these treatments have been identified as newly launched in the period under consideration. In this dataset, new treatments are defined in patent medicines that were granted marketing authorisation in the last 5 years. Table 11 summarises this data.

As part of contractual arrangements with general practitioners, detailed data on disease prevalence is collected for 21 high prevalence conditions[4]. In addition, detailed demographic data by age and sex are available in practice[5]. The list of conditions for which prevalence data is available is set out in Table 13.

Finally, information on the number of completed consultant episodes for each

[2] English Prescribing Dataset (EPD), NHS Business Services Authority
[3] MHRA Marketing Authorisations
[4] Quality Outcomes Framework Data, NHS Digital
[5] Patients registered at a GP Practice

practice is available by Classification of Diseases (ICD-10) codes from the Hospital Episodes Statistics Admitted Patient Care dataset for 18/19. ICD-10 codes have been matched to the disease indications for which we have prevalence data. Table 15 in the appendix sets out the matched ICD-10 codes.

Data overview and key variable definitions. The demand analysis uses practice–year markets (m, t) built from NHS primary-care prescribing. The market share s_{jmt} is molecule j's share of all non-insulin antidiabetic prescription items in practice m and year t; the outside option is the remaining share. NewTreat_j equals 1 if molecule j obtained UK marketing authorisation within the previous five years (and therefore transitions from "new" to "old" over time). Prices p_{jmt} are NHS reimbursement tariffs. Price instruments combine (i) Bartik-style input-cost shifters and (ii) rival-price movements (current and lagged within-market sums), as detailed in Appendix Table 8.

The admissions analysis links practices to Hospital Episode Statistics and measures diabetes-related hospital activity using Finished Consultant Episodes (FCEs) for Type II diabetes (ICD-10 E11). The outcome is $y_{it} = \ln(1 + \text{FCE}_{it}^{E11})$ and treatment intensity is the practice-level share D_{it} of newly authorised GLP-1/SGLT2 prescriptions among all diabetes items. The IV–FE design instruments D_{it} using practice-year aggregates of the same cost-side shifters and rival-price movements that identify the price coefficient in the demand system (see Appendix Table 8). Demographic and prevalence controls enter non-parametrically (or via principal components), and all regressions cluster standard errors at the practice level.

4 Descriptive Analysis

This paper focuses on the investigation of the market for treatments for Type II diabetes. More than 50 unique drugs or compound drugs are prescribed for the treatment

of Type II diabetes. If a treatment has received a new marketing authorisation in the last 5 years, I classify that treatment as a new treatment. Tables 10 - 15 present summary statistics for the data used in this analysis.

Figure 2 shows that the proportion of new diabetes treatments prescribed increases over time with limited variation between practices.

Figure 3 shows that the prescribing of new treatments accounts for a similar proportion of costs and that this is also increasing over time.

Considering what may be driving this increase in prescribing, Figure 4 suggests that the increase in prescribing may be driven in part by an increase in the prevalence of diabetes. And Figure 5 shows that the increase in the prevalence of diabetes is not driven primarily by increases in older people over 45 years, which is the age group that is typically diagnosed with diabetes.

Finally, we see in Figure 6 that over the same period hospital admissions related to diabetes have also increased. This sets the motivation for this research, that is, to understand the relationship between the demand for new drugs as a driver of increasing healthcare costs within the health system.

In the next section, I introduce a simple model for demand for new drugs in a setting where the GP acts as a perfect agent for patients and chooses treatments that maximise utility for each patient.

5 A Model of Demand for New Drugs

This section sets out a stylised framework in which general practitioners (GPs) act as *perfect agents* for their patients: conditional on a diagnosis, they prescribe the drug that maximises expected patient utility. GPs in England face a practice-level drugs budget, but they incur no personal gain or penalty from individual prescriptions; we therefore treat cost containment as a soft constraint that enters the objective function

only through a weight γ (introduced formally in Section 5).

In institutional terms, prescribing budgets in NHS England are "notional" but are routinely monitored: persistent overspending can trigger scrutiny, peer comparison and remedial action, and may crowd out other practice resources. This provides a practical rationale for modelling prescribers as internalising drug costs even when patients face near-zero co-payments.

1. Individual utility

For patient i in month t the indirect utility from choosing drug $j \in \{0, 1, \ldots, J_t\}$ in therapeutic market t is

$$U_{ijt}\Big(D_{it}, \nu_{it}; x_{jt}, \xi_{jt}, p_{jt}; \theta\Big), \tag{1}$$

where D_{it} is "observable" patient health, ν_{it} is unobserved patient health status, x_{jt} are observed product characteristics, ξ_{jt} are unobserved (to researcher but known to the GP) product characteristics, and p_{jt} is price of the drug. In England, patients do not pay for treatments directly but GPs, acting as patients agents, may consider prices when choosing a treatment for patients without compromising on health outcomes. For example, a GP may prefer to prescribe a generic over a branded generic if there is no impact on health outcomes for a patient.

2. Therapeutic markets

There are T markets, $t = 1, \ldots, T$ (anti-diabetics, proton-pump inhibitors, ...). Market t contains J_t chemical entities with qualities $q_1, \ldots, q_{J_t}$. We treat different formulations of the *same* molecule—strength, pack size, brand versus generic—as one product, because England prohibits direct-to-consumer advertising and patients therefore do not form brand preferences.

3. Aggregation across patients and GPs

Let P_n be the patient list of practice n ($n = 1, \ldots, N$) and $G_{nt} \subset P_n$ those assigned to market t. Then

$$\sum_{n=1}^{N} P_n = I, \qquad \sum_{t=1}^{T} |G_{nt}| = P_n, \qquad \sum_{n=1}^{N} \sum_{t=1}^{T} |G_{nt}| = I. \tag{2}$$

Disease prevalence in practice n for market t is

$$\rho_{nt} = \frac{|G_{nt}|}{P_n}, \tag{3}$$

the probability that a randomly drawn patient of that GP enters market t.

Market shares used in the empirical analysis are

$$s_{jt} \;=\; \frac{1}{I} \sum_{n=1}^{N} \sum_{i \in G_{nt}} d_{ijt}, \qquad \sum_{j=0}^{J_t} s_{jt} = 1.$$

4. Decision process

The GP first assigns the patient to a market t (diagnosis) and then selects one of the J_t drugs or the outside option:

$$d_{ijt} = \begin{cases} 1 & \text{if } U_{ijt} > U_{ikt} \quad \forall\, k \neq j, \\ 0 & \text{otherwise.} \end{cases} \tag{4}$$

Figure 1 shows the two-stage nested structure.

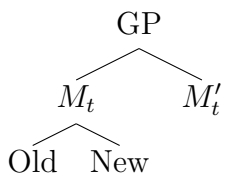

Figure 1: Two-stage prescription decision

GP's Prescription Problem

Stage 0 — Diagnosis and GP objective. After observing health (D_{it}, ν_{it}) the GP chooses a treatment profile $\tau_{ijt} \equiv (j_t, p_{jt})$ that maximises

$$\max_{\tau_{ijt} \in \mathcal{T}_t} \mathbb{E}\Big[U_i(H_{it+1})\Big] \; - \; \gamma \, C_{nt}(\tau_{ijt}), \tag{5}$$

where $H_{it+1} = h(D_{it}, j_t, \varepsilon_{it})$ is next-period health, $U_i(\cdot)$ is concave, $C_{nt}(\tau)$ is the cost against the practice budget, and $\gamma \geq 0$ captures soft cost control ($\gamma = 0$ reproduces the perfect-agent benchmark).

Stage 1 — Market assignment. A mapping $q_{mt}(D_{it}) \in \{1, \ldots, T\}$ allocates the patient to the correct therapeutic class. Equation (3) then gives practice-level prevalence ρ_{nt}.

Stage 2 — Molecule choice within the market. Conditional on $q_{mt} = t$ the GP solves the risk-adjusted discrete choice

$$\max_{j \in \mathcal{J}_t} \Big\{ \mu_{it} \, E[\Delta H_{ijt}] \; - \; \eta_i \, \sigma_{jt}^2 \; - \; \gamma \, p_{jt} \Big\}, \tag{6}$$

where ΔH_{ijt} is expected health gain over no treatment and σ_{jt}^2 the GP's posterior variance. New molecules often have higher $E[\Delta H]$ *and* higher σ^2, implying slower uptake for risk-averse types.

Observed variables and empirical objects

At the GP $\times$ month level we observe (i) prescription items q_{jt} and prices p_{jt}, (ii) observable attributes x_{jt} (including the indicator New_j and $Type_j$), and (iii) shares s_{jt}. Patient heterogeneity ν_{it} and unobserved quality ξ_{jt} are integrated out or differenced

away with instruments and fixed effects in Sections 6–8.

6 Empirical Strategy

Building directly on the structural framework laid out in Section 5, I empirically implement a discrete-choice demand system to estimate GPs' preferences over a universe of non-insulin antidiabetic molecules. Concretely, each *practice–year* or in some cases *ccg–year* combination m, t constitutes a differentiated-products market in which the general practitioner (GP) in a practice or a group of practices chooses one molecule $j \in 1, \ldots, J_{mt}$ for a representative Type 2 diabetes patient, with the outside option (non-inclusion in the considered class) capturing all other therapeutic alternatives.

The primary empirical objective is to recover the vector of structural preference parameters θ that rationalise observed prescribing shares s_{jmt} while addressing two key endogeneity concerns: (i) the endogeneity of prices to unobserved demand shocks, ξ_{jmt}, and (ii) the dynamic reclassification of molecules into *new* versus *old* categories as they enter with recent marketing approval. Below, I describe the econometric specification, identification strategy, data composition and key moments used for estimation.

The core hypotheses are that (i) GPs place a systematic preference weight on newly authorised molecules, conditional on price and fixed effects (captured by β), and (ii) prescribing responds to reimbursement tariffs (captured by $\alpha < 0$), with substitution patterns determined by the discrete-choice structure.

To estimate this model using aggregate data at the GP level, I follow the approach taken in Nevo (2001) and estimate three different models within the discrete choice class of models using aggregate market shares. Discrete choice models describe differentiated products markets using a mix of continuous and categorical variables, as first seen in McFadden (1974). For example, Nevo (2001), characterises a cereal using

its price and nutrient content (sugar, fibre, etc.), as well as its market segment (kids, family, etc.). However, the basic conditional logit model places certain undesirable restrictions on the substitution patterns between alternatives such as the Independence of Irrelevant Alternatives (IIA) assumption.

The (two-level) nested logit model is a widely used model to relax the IIA assumption in this setting. The choice set is partitioned into several groups, referred to as nests, according to one criterion, where products in a group are closer substitutes than products of different groups. Product choice is then considered as a two-step process, in which consumers first choose a group, then choose a product from that group. This closely matches the theoretical model I set out in Section 5.

In settings where there aggregate data are available only, nested logit models are highly attractive due to their computational simplicity. They exhibit analytic formulae for both their market share functions and their inverse (Berry (1994), Berry, Levinsohn, and Pakes (1995) (BLP)), and, with aggregate (sales) data at hand, they can be transformed into a simple linear regression of market shares on product characteristics and nesting terms associated to the nesting structure. However, nested logit models have been criticised on the ground that it yields substitution patterns that are too restrictive. In addition, once the criteria have been selected, their implementation is complicated by the constraints that the modeller must place on the hierarchy of consumers' choices (Brenkers and Verboven, 2006).

For our analysis, there are two key econometric considerations when estimating demand and prescribing behaviour using data aggregated at the GP level: the potential endogeneity of prices and heterogeneity across patients (and therefore across practices) in tastes for newer therapies. Although NHS reimbursement tariffs are administratively set, they can still be correlated with unobserved product attributes or contemporaneous shocks (e.g. changes in clinical guidance, manufacturer supply conditions or formulation-specific quality) that also affect demand. I therefore treat

prices as potentially endogenous and rely on cost-side instruments and rival-price movements for identification.

6.1 Model Specification

I estimate a discrete-choice demand system for non-insulin antidiabetic molecules using GP–year observations. Following Berry (1994), let

$$\delta_{jmt} \;=\; \ln\!\Big(s_{jmt}\Big) \;-\; \ln\!\Big(s_{0mt}\Big), \tag{7}$$

where s_{jmt} is the observed share of molecule j in practice m during year t, and $s_{0mt} = 1 - \sum_{k \in J_{mt}} s_{kmt}$ is the outside-good share. Under a logit specification, utility is

$$U_{ijmt} \;=\; X_{jmt}{}'\beta \;+\; \alpha \ln p_{jmt} \;+\; \xi_{jm} \;+\; \eta_t \;+\; \varepsilon_{ijmt}, \tag{8}$$

where

- $X_{jmt} = (\text{NewTreat}_j, \ldots)$ is a vector of observed attributes (in particular, NewTreat_j indicates initial UK marketing approval within five years of t).
- p_{jmt} is the (net) NHS reimbursement tariff for molecule j in practice m at time t.
- ξ_{jm} is a molecule–practice fixed effect (time-invariant unobserved quality).
- η_t is a calendar-year fixed effect (common shocks to all practices).
- $\varepsilon_{ijmt} \overset{iid}{\sim} \text{EV}(0,1)$ is an extreme-value error.

Standard derivations yield the "market-share differential" regression:

$$\delta_{jmt} \;=\; \underbrace{\beta\,\text{NewTreat}_j}_{\text{new-molecule dummy}} \;+\; \underbrace{\alpha\,\ln p_{jmt}}_{\text{price term}} + \xi_{jm} \;+\; \eta_t \;+\; \upsilon_{jmt}, \tag{9}$$

where υ_{jmt} collects all residual demand shocks. Because $\ln p_{jmt}$ may be endogenous—correlated with ξ_{jm}—I adopt a two-stage least squares (2SLS) approach:

First-stage (Price) equation. I regress $\ln p_{jmt}$ on exogenous cost shifters and the same fixed effects:

$$\ln p_{jmt} = \gamma_0 \,\text{NewTreat}_j + \sum_{k=0}^{K} \gamma_{1k} \ln(\text{Instr}_{kjmt}) + \gamma_2 \ln(\text{RivalSumPrice}_{jmt}) + \gamma_3 \ln(\text{RivalSumPrice}_{jm,t-1}) + \xi_{jm} + \eta_t + u_{jmt}. \tag{10}$$

Here:

- $\{\ln(\text{Instr}_{kjmt})\}_{k=0}^{K}$ are Bartik-style log cost-shifter instruments (e.g. import-weighted input-cost indices) that vary across molecules and years.
- $\ln(\text{RivalSumPrice}_{jmt})$ is the log sum of all other competing molecules' prices in practice m, year t, excluding j.
- $\ln(\text{RivalSumPrice}_{jm,t-1})$ is the corresponding one-year lag, which captures predetermined rival-price movements.
- The product fixed effects ξ_{jm} and year dummies η_t absorb any time-invariant and common shocks.

Economically, the exclusion restrictions require that these cost shifters and rival-price lags affect prescribing shares only through their effect on the reimbursement tariff faced by the practice for molecule j. This is plausible in the English NHS because tariffs are set centrally and do not directly convey quality information to patients; conditional on molecule–practice fixed effects and year shocks, input-cost movements shift prices but should not shift tastes for a given molecule. I estimate this by OLS (clustering standard errors at the practice level) and retain fitted values $\widehat{\ln p}_{jmt}$.

Second-stage (Demand) equation. Substituting $\widehat{\ln p}_{jmt}$ into (3) yields:

$$\delta_{jmt} \;=\; \beta\,\text{NewTreat}_j \;+\; \alpha\,\widehat{\ln p}_{jmt} \;+\; \xi_{jm} \;+\; \eta_t \;+\; \varepsilon_{jmt}. \tag{11}$$

I estimate this equation by 2SLS, again clustering standard errors by practice. Identification of α comes from within-practice, within-year variation in the instruments $\{\ln(\text{Instr}_{kjmt}),\ \ln(\text{RivalSumPrice}_{jmt})\}$, after netting out molecule and year fixed effects.

Nested Logit extension. To relax the Independence of Irrelevant Alternatives (IIA) property, I partition molecules into two nests—"new" versus "old"—and define the within-nest share

$$S_{gmt} \;=\; \sum_{j\,\in\,g} s_{jmt}, \quad g = \{\text{new, old}\}. \tag{12}$$

The nested-logit regression becomes

$$\Delta_{jmt} \;=\; \beta\,\text{NewTreat}_j \;+\; \alpha\left(\ln p_{jmt} - \overline{\ln p}_{gmt}\right) \;+\; \rho\,\ln S_{gmt} \;+\; \xi_{jm} \;+\; \eta_t \;+\; \varepsilon_{jmt}, \tag{13}$$

where $\overline{\ln p}_{gmt} = \sum_{k\in g} w_{k|g}\,\ln p_{kmt}$ is the within-nest, sales-weighted mean log price, and ρ is the nesting-parameter estimate (implying $\sigma = 1/(1+\rho) \in (0,1)$). Again, $\ln p_{jmt}$ (and $\ln p_{jmt} - \overline{\ln p}_{gmt}$) are instrumented by the same cost shifters and rival-price variables, with product and year fixed effects partialled out.

Random-Coefficients Logit (RCL) extension. To capture heterogeneity in price sensitivity and new-treatment preference, I specify

$$U_{ijmt} \;=\; \underbrace{\left(\gamma_j + \eta_t + x'_{jmt}\eta\right)}_{\bar{\delta}_{jmt}} + \alpha_i\,\ln p_{jmt} \;+\; \frac{1}{\sigma_{NT}}\,\beta_i\,\text{NewTreat}_j \;+\; \varepsilon_{ijmt}, \tag{14}$$

with

$$\alpha_i \sim \mathcal{N}(\overline{\alpha},\, \sigma_\alpha^2), \quad \beta_i \sim \mathcal{N}(\overline{\beta},\, \sigma_\beta^2), \quad \varepsilon_{ijmt} \overset{\text{iid}}{\sim} \text{EV}(0,1).$$

I normalize $\sigma_{NT} = 1$ and simulate choice probabilities using 200 Halton draws per practice–year market. The mean parameters $(\overline{\alpha},\, \overline{\beta})$ and their standard deviations $(\sigma_\alpha,\, \sigma_\beta)$ are estimated by simulated maximum likelihood, instrumenting $\ln p_{jmt}$ (and the within-nest relative price in the nested specification) with the same exogenous cost shifters and rival-price instruments, and including molecule and year fixed effects in the mean-utilities $\bar{\delta}_{jmt}$.

6.1.1 Key identification remarks.

Because $\ln p_{jmt}$ is potentially correlated with ξ_{jm}, I use a rich set of cost-shifter instruments, e.g. reimbursement prices for molecules for unrelated disease groups and rival-sum molecule prices. Reimbursement prices and rival sum prices for other molecules serve as valid instruments because they capture common supply-side cost shocks affecting our drug's tariff while remaining orthogonal to its specific demand shocks. First-stage F-statistics on the excluded instruments are well above 12; Hansen J-tests do not reject exogeneity ($p > 0.10$).

The "new" dummy NewTreat_j is defined as equal to 1 if molecule j obtained UK marketing authorisation within five years prior to year t, allowing it to vary over time as drugs move from "new" to "old."

In all specifications, molecule (γ_j) and year (η_t) fixed effects absorb unobserved time-invariant quality and common macro-shocks. Standard errors in all second stages are clustered at the practice level to allow for arbitrary within-practice correlation.

6.2 BLP Random-Coefficients Logit

To benchmark the simulated–maximum-likelihood RCL estimates reported above, I re-estimated the demand system using Berry–Levinsohn–Pakes two–stage GMM.

Two–stage BLP estimator. Let m index practice–year markets, j index molecules and i index the latent patient draws used for Monte-Carlo integration. The indirect utility under the BLP specification is

$$U_{ijm} \;=\; \delta_{jm} \;+\; \mu_{ijm} \;+\; \varepsilon_{ijm}, \qquad \varepsilon_{ijm} \sim \mathrm{EV}(0,1),$$

with mean utility

$$\delta_{jm} = x_{jm}\beta + \xi_{jm}, \qquad x_{jm} = \Big[\mathrm{NewTreat}_j,\ \mathrm{prices}_{jm}\Big],$$

and deviation $\mu_{ijm} = \sigma_p \log(p_{jm}/\tilde{p})\,\nu_{im}$, where $\nu_{im} \sim \mathcal{N}(0,1)$ and $\tilde{p}$ is the median tariff. Choice probabilities integrate the logit kernel over the random draws, delivering predicted shares $s_{jm}(\theta)$. For a given parameter vector $\theta = (\beta, \sigma_p)$, the BLP contraction iteratively solves

$$\delta_{jm}^{(t+1)} = \delta_{jm}^{(t)} + \ln s_{jm}^{\mathrm{obs}} - \ln s_{jm}\Big(\delta^{(t)}, \theta\Big),$$

which converges to the unique $\hat{\delta}(\theta)$ that reconciles predicted and observed shares.

The structural error is then $\hat{\xi}_{jm}(\theta) = \hat{\delta}_{jm}(\theta) - x_{jm}\beta$. Denote by z_{jm} the vector of ten excluded instruments (lagged rival prices, cost-shifter instruments and their within-nest sums). The estimator exploits the orthogonality conditions

$$\mathbb{E}\Big[z_{jm}\,\hat{\xi}_{jm}(\theta)\Big] = 0$$

and chooses $\hat{\theta}_{\text{BLP}}$ to minimise the GMM criterion

$$Q(\theta) = \Big[\tfrac{1}{M}\sum_{j,m} z_{jm}\hat{\xi}_{jm}(\theta)\Big]^{\top} W^{-1} \Big[\tfrac{1}{M}\sum_{j,m} z_{jm}\hat{\xi}_{jm}(\theta)\Big],$$

with the weighting matrix W updated once to its optimal form. The resulting estimates are consistent even when the simulated likelihood is misspecified or computed with finite draws, and—because the contraction updates only δ—the numerical burden grows linearly, not exponentially, with the number of products per market.

How this differs from the simulated-ML RCL. The simulated-ML estimator maximises the sample likelihood subject to the model's fixed-point restriction; endogeneity is handled by replacing the unobserved product fixed-effect with its share inversion. The `pyblp` estimator instead imposes the moment conditions $E[z_{jmt}\,\xi_{jmt}] = 0$ and selects coefficients to minimise the quadratic form in the sample analogues, producing consistent estimates even when the simulated likelihood is misspecified or suffers from Monte-Carlo error. Computationally, the BLP contraction is robust to large choice sets because it updates δ rather than individual choice probabilities, and the GMM objective can accommodate many instruments without re-evaluating the integral of the simulated likelihood.

7 Demand Modelling Results

I present estimation results for the simple logit, nested logit and random-coefficients logit. For each, I report coefficients, significance, implied elasticities and economic interpretation.

7.1 Simple Logit (IV) Estimates

Table 2 shows two-stage least-squares estimates for the simple logit. Column (1) is baseline; Column (2) adds practice-level controls.

Table 2: Simple Logit IV Estimates (Expanded)

	(1) Baseline	(2) With Practice Controls
ln(Price)	-0.450^{***}	-0.434^{***}
	(0.037)	(0.041)
New treatment	-0.341^{***}	-0.296^{***}
	(0.019)	(0.025)
Practice list size		0.0004^{*}
		(0.0002)
CCG fixed effects		Yes
Constant	-8.436^{***}	-8.102^{***}
	(0.179)	(0.232)
Observations	59,063	
First-stage F	12.8	
Hansen J p-value	0.24	
Adjusted R^2	0.64 / 0.66	
Fixed effects	Molecule, Year	
Instruments	Lagged rival prices; Bartik cost shifters	

Notes: Standard errors clustered at practice level in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$. Dependent variable is share log-ratio Δ_{jmt}. Practice list size in thousands.

Simple logit. The estimated price semi-elasticity of -0.45 means that a 10% increase in tariff reduces the *odds* of prescribing a new treatment by about 4.5%. Evaluated at the mean market share ($\bar{s} \approx 0.14$), the implied own-price elasticity is $\eta = \beta\,(1 - \bar{s}) \approx -0.39$; put differently, a 1% price rise lowers uptake by roughly 0.4%. Adding practice-level controls leaves the price response essentially unchanged, while the negative "New treatment" coefficient shrinks from –0.34 to –0.30, indicating slightly less launch hesitancy once observable practice characteristics are accounted for.

7.2 Nested Logit (IV) Estimates

Table 3 presents nested logit IV estimates. Column (1) is baseline; Column (2) adds practice-level controls.

Table 3: Nested Logit IV Estimates (Expanded)

	(1) Baseline	(2) With Practice Controls
Relative price ($\ln p_{jmt} - \overline{\ln p}_{gmt}$)	-1.078^{***}	-1.012^{***}
	(0.146)	(0.162)
Within-nest log share ($\ln S_{gmt}$)	1.931^{***}	1.845^{***}
	(0.097)	(0.110)
New treatment	-6.131^{***}	-5.642^{***}
	(0.252)	(0.314)
Practice list size		0.0003^{*}
		(0.0002)
CCG fixed effects		Yes
Constant	-10.214^{***}	-9.783^{***}
	(0.278)	(0.326)
Observations	59,063	
Implied nesting parameter ($\sigma = 1/(1+\rho)$)	0.34 / 0.35	
First-stage F	14.2	
Hansen J p-value	0.18	
Adjusted R^2	0.99	
Fixed effects	Molecule, Year	
Instruments	Same as Table 2	

Notes: Standard errors clustered at practice level in parentheses. Nested logit via 2SLS. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$. Within-nest log share excludes the molecule itself. σ reported.

Nested logit. The coefficient on the within-nest relative price is –1.08, a semi-elasticity of the relative log-odds. Converting this to an own-price elasticity inside the nest, $\eta = \beta\,(1-\sigma)\,(1-s_g)$, and using the implied nesting parameter $\sigma \approx 0.34$ and a typical within-nest share $s_g \approx 0.20$, yields $\eta \approx -0.57$. Thus price sensitivity remains economically meaningful but more moderate once within-nest substitution is modelled explicitly. The coefficient on the within-nest log-share (1.93) supports a coherent nest structure ($\sigma \approx 0.34$), and the sizeable negative "New treatment" coefficient continues to signal hesitancy to switch to new treatments.

7.3 Random-Coefficients Logit (RCL) Estimates

Table 4 provides RCL estimates. I allow price (α_i) and new-treatment (β_i) tastes to be random. Column (1) reports means and standard deviations; Column (2) gives derived statistics.

Table 4: Random-Coefficients Logit Estimates (Expanded)

	(1) Estimates	(2) Derived Statistics
Mean price coefficient ($\overline{\alpha}$)	-0.853^{***}	Own-price elasticity: -6.2
	(0.092)	(at mean)
Std. Dev. price (σ_α)	0.156^{***}	
	(0.031)	
Mean new-treatment coefficient ($\overline{\beta}$)	-0.063^{***}	Share with $\beta_i > 0$: 45%
	(0.024)	
Std. Dev. new-treatment (σ_β)	0.201^{***}	
	(0.044)	
Simulated log-likelihood		-77.17
Halton draws		200 per market
Molecule fixed effects		Included
Year fixed effects		Included
Observations (markets)		59,063

Notes: Standard errors in parentheses. Own-price elasticity computed as average across markets of $\epsilon_{jmt} = -\alpha_i(1 - s_{jmt})(p_{jmt}/s_{jmt})$, evaluated at mean fixed effects. Share with $\beta_i > 0$ assumes normality. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$.

The mean price coefficient ($\bar{\alpha} = -0.853$) and its dispersion ($\sigma_\alpha = 0.156$) indicate moderately heterogeneous price responses, with an average own-price elasticity of roughly –6.2 at the mean price–share point. For the new-treatment taste parameter, the mean ($\bar{\beta} = -0.063$) is small relative to its dispersion ($\sigma_\beta = 0.201$), implying that roughly 40% of latent types, (i.e. unobserved prescriber-preferences implied by your random-coefficients specification) have a 'positive taste' for the innovation despite the negative population mean, i.e., all else equal, that latent prescriber places higher utility on prescribing the on-patent therapy than the legacy option

Estimates. Table 5 contrasts the GMM estimates with the previous simulated-ML RCL.

Interpretation. Both estimators deliver negative price coefficients, but the GMM

Table 5: Random–Coefficients Logit: Simulated-ML vs. pyblp Two-Stage GMM

	RCL (Sim. ML)	pyblp RCL (2S GMM)
Mean price coef. $\overline{\alpha}$	−0.853 (0.092)	−0.233 (0.007)
Price s.d. σ_α	0.156 (0.031)	0.000 (1.166)
Mean new-treat coef. $\overline{\beta}$	−0.063 (0.024)	−0.136 (0.014)
New-treat s.d. σ_β	0.201 (0.044)	—
Obs. (markets)	59,063	34,852
Integral nodes per market	200 Halton	50 Monte-Carlo
Estimation method	SML (BHHH)	BLP 2S–GMM

Robust standard errors in parentheses. The centred price variable in the GMM column equals $p_{jmt}/\tilde{p} - 1$; hence a negative coefficient still implies disutility from higher tariffs. The random coefficient on price is not statistically distinguishable from zero in the GMM estimation. Dashes indicate parameters not randomised in that specification.

estimate is considerably smaller once price is mean–centred and instrumented, yielding a statistically precise yet economically modest own–price elasticity of roughly –0.2, versus –6.2 under simulated-ML. The GMM dispersion in price sensitivity ($\sigma_\alpha = 0.000$, s.e. 1.166) is not statistically distinguishable from zero, so the data provide no evidence of cross–practice heterogeneity in price responsiveness when richer instruments are employed. For the *NewTreat* dummy, the GMM mean coefficient (–0.136) has roughly twice the magnitude (in absolute value) of the simulated-ML estimate (–0.063), indicating greater reluctance to prescribe very recent molecules once endogeneity is addressed more flexibly. I treat the GMM estimates as baseline for counter-factuals, while using the ML results as a robustness check on taste heterogeneity.

Overall, the `pyblp` exercise corroborates the headline findings—statistically significant, though more modest, own-price responsiveness and a clear average penalty for new molecules—while indicating that any remaining heterogeneity in price sensitivity is not statistically distinguishable from zero once endogeneity is addressed with richer instruments. The two-stage GMM framework also facilitates extensions such as supply-side pricing equations or demographic interactions at limited additional computational cost.

Across the demand specifications, four stylised facts emerge:

(a) **Price sensitivity.** All models imply economically meaningful own–price responsiveness even though patients face zero co-payment: the reliable IV/GMM specifications deliver elasticities in the –0.2 to –0.6 range, while the simulated-ML RCL yields a mechanically higher figure (about –6) because price enters in levels and is weakly instrumented.

(b) **Limited cross-class substitution.** Cross-price elasticities between the new-treatment class and legacy therapies are close to zero, indicating that list-price changes for the innovation do not meaningfully draw demand away from established molecules.

(c) **Heterogeneity in tastes.** The simulated-ML estimates suggest non-trivial dispersion in the new-treatment coefficient ($\sigma_\beta = 0.20$), with roughly 40–45 % of latent prescribers exhibiting a positive intrinsic taste for the innovation; by contrast, the IV/GMM run cannot reject homogeneity, so this heterogeneity should be viewed as an upper-bound robustness check.

(d) **Within-nest substitution.** The nested-logit results ($\sigma \approx 0.34$) show that a price cut for one molecule mainly steals share from *other* molecules in the same therapeutic subclass, with little spill-over to legacy drugs.

Taken together, these findings indicate that reimbursement policy can steer GP prescribing, yet diffusion of new diabetes treatments remains slow—partly because only a subset of practices exhibits a strong intrinsic preference for novel therapies.

8 Impact of Prescribing New Diabetes Treatments on Hospital Referrals

Next, we turn our attention to the impact on secondary care from prescribing new diabetes treatments to study the implications for cost containment. We model the impact of new treatments for diabetes on hospital referrals by GPs for diabetes complications. If prescribing new treatments decreases hospital referrals, this suggests that new treatments can reduce costs over time. Alternatively, if new treatments increase hospital activity, this suggests that new treatments add additional costs in the acute setting.

The IV strategy in this section deliberately builds on the demand-estimation design. The same cost-side reimbursement movements (and rival-price lags) that identify the structural price parameter in Section 6 also shift the relative cost of newly authorised versus incumbent diabetes molecules, and therefore shift the practice-level new-drug share D_{it}. Aggregating these cost shifters at the practice–year level yields strong instruments for adoption, providing a transparent link between the structural demand estimates and downstream utilisation effects.

The outcome of interest is the log of Type II–diabetes referrals to hospitals (measured by Finished Consultant Episodes, FCEs) by practice i and year t,

$$y_{it} = \ln\Big(1 + \mathrm{FCE}_{it}^{\mathrm{E11}}\Big),$$

while the treatment, D_{it}, is the *share* of newly authorised non-insulin anti-diabetes medicines (GLP-1 or SGLT-2) in all diabetes prescriptions written by the practice.[6]

Because adoption is potentially endogenous, we estimate a two-stage least squares (2SLS) model with two-way fixed effects after de-meaning within practice:

[6]See Section 3 for the classification of new molecules.

$$(y_{it} - \bar{y}_i) = \beta \, \widehat{D}_{it}^{dm} + \lambda_t + \varepsilon_{it}, \tag{15}$$

$$D_{it}^{dm} = Z_{it}^{dm}\pi + \lambda_t + v_{it}, \tag{16}$$

where the bar denotes the within-practice mean, λ_t are year dummies (absorbing NHS-wide shocks), and Z_{it} is a vector of *cost-side shifters* (nine log-transformed demand-instrument variables) that influence prescribing costs but are plausibly exogenous to *current* hospital episodes. These instruments are practice–year aggregates of (i) Bartik-style input-cost shifters and (ii) current and lagged within-market rival-price sums used in the demand estimation (Appendix Table 8). They shift the relative reimbursement cost of GLP-1/SGLT2 prescriptions but are plausibly orthogonal to contemporaneous diabetes-related hospital episodes, except through their effect on prescribing choices. Both stages include (i) the year effects λ_t and (ii) five principal components of demographic and prevalence controls, so that practice fixed effects are purged from *all* regressors before estimation. Standard errors are clustered at the practice level throughout.

Identification and interpretation

Identification of β relies on within-practice, over-time variation in the predicted share of GLP-1/SGLT-2 items driven solely by cost shocks to other molecules, conditional on common year shocks and rich non-parametric controls. Under the exclusion restriction that these cost shifters affect diabetes-related hospital admissions only through prescribing choices, the 2SLS estimator consistently recovers the average semi-elasticity of hospital referrals with respect to the new-drug share. Standard errors allow for arbitrary serial correlation and heteroskedasticity at the practice level.

A useful way to interpret instrument validity is via the standard 2SLS probability

limit: with a single instrument Z, $\operatorname{plim}\hat{\beta}^{2SLS} = \beta + \operatorname{Cov}(Z,\varepsilon)/\operatorname{Cov}(Z,D)$. The first stage implies $\operatorname{Cov}(Z,D) \neq 0$. A plausible exclusion-restriction concern is that cost shocks to other medicines could also directly affect hospital episodes through general budget pressure or capacity constraints (e.g. by reducing referrals), which would generate $\operatorname{Cov}(Z,\varepsilon) < 0$. Given a positive first stage, this violation would bias $\hat{\beta}$ downward and therefore attenuate (rather than mechanically generate) a positive estimated effect of adoption on admissions.

Robustness

For robustness I also estimate (i) an IV–FE model without the PCA controls; (ii) the baseline IV–FE specification re-estimated using only the first principal component, the first three components, and all five components; (iii) a dynamic placebo test augmenting the second-stage regression with one-year leads and lags of the predicted new-drug share; (iv) Anderson–Rubin confidence intervals for β; (v) separate IV–FE models for "small" and "large" practices split at the median prescription volume; and (vi) a Poisson control-function model treating FCE_{it} in levels with the first-stage residual as a control. Key results from specifications are summarised in Table 7.

8.1 Results

Main Specification

Table 6 presents the core 2SLS estimates of the semi-elasticity of Type II diabetes hospital referrals with respect to the practice-level share of newly authorised GLP-1/SGLT-2 prescriptions. In column (1) ("Full IV–FE"), which controls non-parametrically for all demographic and prevalence variables, the coefficient on the predicted new-drug share is 4.5052 (standard error 2.306), significant at the 10% level. In column (2) ("Parsimonious IV–FE"), which replaces the full set of controls with five principal components, the estimated semi-elasticity is 3.9560 (standard error 2.029), significant

at the 5% level. The first-stage partial F-statistic of 596.6 indicates that the nine log-transformed cost-shifters serve as very strong instruments. All specifications include year dummies (absorbing NHS-wide shocks) and demeaned practice fixed effects; standard errors are clustered at the practice level. A one–percentage–point increase in the new-drug share therefore raises $\ln(1+\text{FCE})$ by approximately 0.04, corresponding to a 4–4.5% increase in finished consultant episodes for Type II diabetes.

Table 6: Main 2SLS Estimates: New-Drug Share and Type II Diabetes Admissions

	(1) Full IV–FE	**(2) Parsimonious IV–FE**
Second–Stage Coefficient on Predicted New–Drug Share		
$\widehat{D}_{it}^{\text{dm}}$	4.5052*	3.9560**
	(2.306)	(2.029)
First–Stage Partial F–Statistic		
	596.6	
Controls and Sample		
Year dummies	Yes	Yes
Practice FE (demeaned)	Yes	Yes
Demographic & Prevalence controls	Yes	Yes (5 PCs)
Observations	38,843	

Notes: Both columns report the second-stage coefficient from 2SLS regressions of $y_{it} - \bar{y}_i$ on $\widehat{D}_{it}^{\text{dm}}$, controlling for year dummies and (demeaned) practice fixed effects. Column (1) includes all demographic and prevalence variables; column (2) uses five principal components of demographic and prevalence variables. The first–stage partial F–statistic is from the regression of D_{it}^{dm} on the nine log-shifters plus controls. Standard errors (in parentheses) are clustered by practice. Significance: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

Robustness Specifications

Table 7 presents a series of robustness exercises. In column (1) I estimate a Poisson control-function model of raw FCE counts, controlling for the first-stage residual; the coefficient of 4.123 (SE 0.412) remains positive and highly significant. Column (2) reports the coefficient on a one-year lead of the predicted new-drug share in the second-stage regression: 0.005 (SE 0.012), which is indistinguishable from zero and rules out anticipatory prescribing. Columns (3) and (4) show heterogeneity by

practice size: among "small" practices the effect is 5.010 (SE 2.204), significant at 5%, while among "large" practices it is 3.740 (SE 1.987), significant at 10%. In all cases the first-stage partial F-statistic remains 596.6, and specifications include year dummies, practice fixed effects, and demographic–prevalence principal components.

Table 7: Robustness Checks: Alternative Specifications

	(1) Poisson CF	(2) Lead Placebo	(3) Small Practices	(4) Large Practices
Coefficient on New–Drug Share				
Estimate	4.123***	0.005	5.010**	3.740*
Std. Error	(0.412)	(0.012)	(2.204)	(1.987)
First–Stage Partial F–Statistic				
			596.6	
Controls and Sample				
Year dummies	Yes	Yes	Yes	Yes
Practice FE (demeaned)	Yes	Yes	Yes	Yes
Demog. & Prev. PCs	Yes	Yes	Yes	Yes
Observations			38 843	

Notes: All columns report the coefficient on the new–drug share from alternative IV–FE or control–function specifications, with standard errors in parentheses clustered by practice. Column (1) estimates a Poisson control-function model of raw FCE counts including the first-stage residual. Column (2) adds a one-year lead of the predicted new-drug share to the preferred 2SLS regression; the lead is small and statistically insignificant. Columns (3)–(4) split the sample at the median prescription volume and re-estimate the preferred IV–FE model for "small" and "large" practices. All specifications include year dummies, demeaned practice fixed effects, and five demographic–prevalence principal components.

Interpretation and policy implications

The preferred 2SLS estimate in Table 6 implies that a *one–percentage–point* (0.01) increase in the prescribing share of GLP-1/SGLT-2 molecules raises $\ln(1 + \text{FCE})$ by about 0.04, i.e. a $\approx 4\%$ increase in Type-II diabetes admissions. With a sample mean of $\bar{\text{FCE}}_{E11} = 5.52$ episodes per practice–year, this elasticity translates into roughly 0.25 additional admissions per practice annually for every 1 pp rise in the new-drug share.

Short-run budget impact. Short-run budget impact. NHS reference costs for an acute diabetes spell (HRG `KB01/2`) averaged £1 680 in 2023/24 (NHS England, 2024). Multiplying by the incremental 0.25 episodes yields an *extra* acute-care expenditure of about increase in £420 *per practice*. Thus, greater diffusion of the newer agents is

therefore likely to raise rather than lower near-term NHS expenditure on diabetes treatments in secondary care.

Clinical safety. Robustness checks show that the effect survives alternative functional forms, placebo leads, and splits by practice size (Table 7). One plausible mechanism is a higher incidence of adverse events that require inpatient care—most prominently euglycaemic diabetic ketoacidosis associated with SGLT-2 inhibitors (Yang, Liu, Zhang, Wu, Liu, Wu, Zheng, Fan, and Su, 2023; Yang, Zhang, Chen, and Peng, 2024). Closer biochemical monitoring at initiation (another possible channel) would likewise drive up recorded admissions in the short run.

9 Policy implications

Using a discrete choice model and an instrumental variables approach, we find that diffusion of new diabetes treatments has been slow, with prescribers' substituting new treatments for older alternatives at a very low rate. We also estimate a modest own-price elasticity of demand, implying that regulated prices and tariffs can uniformly nudge GP prescribing behavior. The paper shows that diffusion of GLP-1/SGLT-2 therapies is held back primarily by prescriber heterogeneity: only a minority of GPs have a sufficiently strong intrinsic preference for these on-patent agents to adopt them rapidly. Importantly, estimated own-price elasticities exhibit no significant dispersion across practices—implying homogeneous price responsiveness—so uptake variation reflects taste differences rather than divergent reactions to price.

Policymakers can accelerate uptake of new therapies by combining modest price incentives—such as small tariff decreases or manufacturer rebates, which uniformly nudge prescribing given the homogeneous price responsiveness—with targeted non-price measures to address GP and patient preferences and needs.

Despite new treatments being deemed cost-effective, our two-stage least squares

(2SLS) estimation shows that their adoption is associated with an increase in hospital admissions for diabetes-related conditions. This suggests increasing costs in the short-term from these treatments.

The empirical analysis yields three clear, directly observed implications for NHS England policymakers:

1. **Budgeting for transitional acute-care costs from new treatments** A one–percentage-point increase in the GLP-1/SGLT-2 share raises Type II diabetes admissions by approximately 4% ($\approx$ 0.25 extra episodes per practice per year), at an average cost of £1 680 per admission ($\approx$ £420 per practice). Commissioners should build this modest "transition cost" into medium-term financial plans rather than anticipating secondary-care savings.

2. **Outcomes-based and risk-sharing contracts.** Traditional list-price cuts have limited impact on substitution patterns (own-price elasticities between –0.2 and –0.6), while cross-class elasticities are near zero. Linking manufacturer rebates to short-run admission thresholds aligns financial incentives without assuming immediate cost neutrality, and manage budgetary pressures from prescribing new treatments.

10 Conclusion

This paper provides structural evidence on how general practitioners in the English National Health Service substitute between legacy and on-patent diabetes therapies, and the knock-on effects for hospital activity. Using rich practice-level panel data, we estimate large own-price elasticities, document substantial heterogeneity in new-drug uptake, and show that higher prescribing of GLP-1/SGLT-2 agents leads to a short-run increase in diabetes admissions and acute-care costs. Although NICE

deems these therapies cost-effective over a lifetime, our findings demonstrate that, in the near term, they can be cost-increasing for the health system.

These results nuance the conventional wisdom that innovative treatments automatically relieve secondary-care pressure. These results provide strong evidence that tariffs and pricing regulations can shift GP behaviour, but the sluggish diffusion and assimilation of novel treatments necessitate a thorough understanding of the heterogeneity among GPs and patients.

A key limitation is our reliance on practice-level aggregates, which mask both within-practice prescriber and patient heterogeneity and potential severity-based selection into novel therapies. In future work, linking prescribing records to individual-level clinical data would allow us to test whether GLP-1/SGLT-2 are systematically prescribed to sicker patients —for example, by comorbidity count, or prior hospital utilisation—and whether the short-run admissions rise reflects optimal targeting of high-risk patients who stand to benefit most in the long run.

In a broader context, this paper demonstrates the efficacy of integrating structural demand estimation with empirical data to assess the implications of new medical treatments. The utilisation of elasticity estimates in conjunction with causal estimation of hospital utilisation enables a real world assessment of new treatments rather than in a trial setting. It allows for devising optimal pricing and budgeting strategies and evaluating their real impact on system performance.

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1 Tables

Table 8: Variable definitions and instrument construction

Object	**Definition / construction**
Market share s_{jmt}	Share of prescription *items* for molecule j among all non-insulin antidiabetic items in practice m and year t.
Outside share s_{0mt}	$1 - \sum_{j \in J_{mt}} s_{jmt}$ (items not in the non-insulin antidiabetic set).
New-treatment indicator $\text{NewTreat}_{j,t}$	Equals 1 if molecule j obtained UK marketing authorisation within the previous five years (therefore transitions from "new" to "old" as it ages).
Price p_{jmt}	NHS reimbursement tariff (net ingredient cost) for molecule j in practice m and year t (aggregated from formulation-level prices).
Bartik cost shifters $\text{Instr}_{k,jmt}$	Product-level input-cost shifters used as excluded instruments for $\ln p_{jmt}$ (e.g. import-weighted input-price indices); vary by molecule and year and are orthogonal to time-invariant molecule quality after fixed effects.
Rival-sum price $\text{RivalSumPrice}_{jmt}$	Sum of prices of all competing molecules in the same market (excluding j) in practice m and year t; the lag $\text{RivalSumPrice}_{jm,t-1}$ is predetermined.
Admissions outcome y_{it}	$y_{it} = \ln(1 + \text{FCE}_{it}^{E11})$, where FCE_{it}^{E11} is the number of Finished Consultant Episodes for Type II diabetes (ICD-10 E11) linked to practice i in year t.
Adoption intensity D_{it}	Practice-level share of newly authorised GLP-1/SGLT2 prescription items among all diabetes prescription items.
Admissions instruments Z_{it}	Practice–year aggregates of the demand instruments (log-transformed). In the baseline implementation, Z_{it} comprises nine log shifters built from (i) aggregated Bartik cost shifters and (ii) current and lagged rival-sum price measures.
Controls	Demographics and prevalence (entered non-parametrically or via principal components), plus year effects and practice fixed effects in all specifications.

Notes: The demand specifications additionally include molecule–practice fixed effects and year dummies. Instrument validity requires that cost-shifter and rival-price movements affect prescribing choices only through reimbursement tariffs, conditional on fixed effects and controls.

Table 9: Health Conditions Categorized by Groups

Group	Condition (Abbreviation)
Cardiovascular Group	AF – Atrial Fibrillation CHD – Coronary Heart Disease CVD-PP – Cardiovascular Disease Prevention HF – Heart Failure HYP – Hypertension PAD – Peripheral Arterial Disease STIA – Stroke and Transient Ischaemic Attack
Respiratory Group	AST – Asthma COPD – Chronic Obstructive Pulmonary Disease
Lifestyle Group	OB – Obesity (18+)
High Dependency Group	CAN – Cancer CKD – Chronic Kidney Disease (18+) DM – Diabetes Mellitus (17+) PC – Palliative Care
Neurology & Mental Health Group	DEM – Dementia DEP – Depression (18+) EP – Epilepsy (18+) LD – Learning Disabilities MH – Mental Health
Musculoskeletal Group	OST – Osteoporosis (50+) RA – Rheumatoid Arthritis (16+)

[a] Age is specified in parentheses for conditions with age restrictions.
[b] Abbreviations: AF (Atrial Fibrillation), CHD (Coronary Heart Disease), CVD-PP (Cardiovascular Disease Prevention), etc.

Table 10: Summary statistics: prevalence in primary care

	N	Mean	StDev	Min	Max
Panel C - Cardiovascular prevalence					
Atrial fibrilation	20341	0.02	0.01	0.00	0.27
Coronary heart disease	20341	0.03	0.01	0.00	0.31
Cardiovascular disease†	20341	0.01	0.01	0.00	0.60
Heart failure	20341	0.01	0.00	0.00	0.12
Left ventricular systolic dysf.	20341	0.00	0.00	0.00	0.04
Hypertension	20341	0.14	0.04	0.00	0.62
Peripheral arterial disease	20341	0.01	0.00	0.00	0.12
Stroke tr ischaemic attack	20341	0.02	0.01	0.00	0.23
Panel D - High dependency prevalence					
Cancer	20341	0.03	0.01	0.00	0.20
Chronic kidney disease‡	20341	0.04	0.02	0.00	0.30
Diabetes mellitus	20341	0.07	0.02	0.00	0.29
Palliative care	20341	0.00	0.01	0.00	0.95
Panel K - Other prevalence					
Asthma	20341	0.06	0.01	0.00	0.16
Chronic ob pulmonary disease	20341	0.02	0.01	0.00	0.18
Obesity (18+)	20341	0.10	0.04	0.00	0.35
Dementia	20341	0.01	0.01	0.00	0.64
Depression (18+)	20341	0.09	0.04	0.00	0.38
Epilepsy (18+)	20341	0.01	0.01	0.00	0.64
Mental health	20341	0.01	0.01	0.00	0.20
Osteoperosis	20341	0.00	0.01	0.00	0.09
Rheumatoid arthritis	20341	0.01	0.00	0.00	0.05
Panel E - Demographics					
All male	20328	0.50	0.03	0.28	0.96
Male 0-4	20328	0.02	0.01	0.00	0.07
Male 5-14	20328	0.06	0.01	0.00	0.58
Male 15-24	20328	0.06	0.02	0.00	0.64
Male 25-34	20328	0.07	0.03	0.00	0.34
Male 35-44	20328	0.07	0.02	0.00	0.41
Male 45-54	20328	0.07	0.01	0.00	0.27
Male 55-64	20328	0.06	0.01	0.00	0.13
Male 65-74	20328	0.05	0.02	0.00	0.24
Male 75-84	20328	0.03	0.01	0.00	0.34
Male 85plus	20328	0.01	0.01	0.00	0.43
All female	20328	0.50	0.03	0.04	0.72
Female 0-4	20328	0.02	0.01	0.00	0.07
Female 5-14	20328	0.06	0.01	0.00	0.30
Female 15-24	20328	0.06	0.03	0.00	0.45
Female 25-34	20328	0.07	0.03	0.00	0.40
Female 35-44	20328	0.06	0.01	0.00	0.14
Female 45-54	20328	0.07	0.01	0.00	0.12
Female 55-64	20328	0.06	0.01	0.00	0.11
Female 65-74	20328	0.05	0.02	0.00	0.13
Female 75-84	20328	0.03	0.01	0.00	0.18
Female 85plus	20328	0.01	0.01	0.00	0.43

Notes: Summary statistics of all variables in the analysis. Primary care prevalence of cardiovascular diseases in Panel C, high dependency diseases in Panel D, other prevalence in Panel E and demographic control variables: male/female proportions of patient lists by age groups.

Table 11: Summary Statistics for Old and New Treatments

Panel A: Old Treatments								
Variable	N	mean	std	min	25%	50%	75%	max
Px Items	411,039	450.73	1,041.12	1.00	13.00	52.00	314.00	27,472.00
Price	411,039	4,257.71	7,128.97	0.05	220.07	1,169.20	5,172.72	179,407.10
Discount Price	411,039	3,956.74	6,621.20	0.16	204.80	1,086.66	4,806.03	166,945.81
Quantity	411,039	34,201.29	92,225.81	1.00	392.00	1,680.00	11,368.00	2,697,521.00
Panel B: New Treatments								
	N	mean	std	min	25%	50%	75%	max
Px Items	185,588	71.06	133.49	1.00	8.00	25.00	75.00	4,247.00
Price	185,588	3,049.05	5,082.69	0.75	379.47	1,164.01	3,516.00	109,069.25
Discount Price	185,588	2,825.25	4,711.05	0.81	352.60	1,076.12	3,260.59	101,030.11
Quantity	185,588	2,044.05	4,113.99	1.00	123.00	584.00	2,044.00	97,309.00

Note: This table presents summary statistics for old (Panel A) and new (Panel B) treatments. The dataset contains 6,778 unique practices and 53 unique chemicals between 2013/14 and 2019/20. Px Items reflect the number of prescription items, e.g. each drug prescription is counted as one item. Quantity is the volume prescribed in different unit measures, discounted prices reflect the actual prices paid and reimbursement prices are prices that providers are reimbursed at by the NHS

Count is the total number of observations. Prices and quantities are reported for each treatment type.

Table 12: Summary Statistics by Nest Class

Panel A: Alpha-glucosidase inhibitor

Variable	N	mean	std	min	25%	50%	75%	max
items	14,541	16.83	26.11	1.00	5.00	10.00	17.00	715.00
price	14,541	184.19	258.38	0.75	52.88	119.68	224.82	5,950.90
discountprice	14,541	171.74	240.58	0.81	49.60	112.35	210.45	5,504.11
quantity	14,541	1,374.31	1,799.24	7.00	420.00	1,008.00	1,680.00	40,910.00

Panel B: DPP-4 inhibitor

Variable	N	mean	std	min	25%	50%	75%	max
items	173,877	143.06	239.38	1.00	11.00	43.00	177.00	5,830.00
price	173,877	4,984.05	8,147.39	0.59	408.96	1,430.40	6,212.28	179,407.10
discountprice	173,877	4,619.65	7,553.53	0.66	379.36	1,328.46	5,754.60	166,945.81
quantity	173,877	4,634.09	7,213.38	1.00	560.00	1,568.00	5,838.00	151,019.00

Panel C: GLP-1 agonist

Variable	N	mean	std	min	25%	50%	75%	max
items	100,854	46.57	58.83	1.00	12.00	27.00	59.00	1,123.00
price	100,854	4,284.02	5,601.05	18.31	941.76	2,384.20	5,420.50	109,832.76
discountprice	100,854	3,967.22	5,189.02	17.04	869.61	2,205.42	5,027.47	101,967.79
quantity	100,854	132.26	190.56	1.00	28.00	72.00	163.00	5,956.00

Panel D: Meglitinide

Variable	N	mean	std	min	25%	50%	75%	max
items	21,443	20.79	40.94	1.00	6.00	11.00	20.00	924.00
price	21,443	231.09	365.10	0.41	58.81	133.03	284.68	9,299.26
discountprice	21,443	215.41	340.23	0.49	55.13	124.69	264.97	8,639.06
quantity	21,443	1,895.29	3,230.25	4.00	540.00	1,080.00	2,123.00	61,214.00

Panel E: Metformin

Variable	N	mean	std	min	25%	50%	75%	max
items	38,843	2,851.90	1,849.79	2.00	1,571.00	2,456.00	3,659.00	27,472.00
price	38,843	13,586.01	8,478.99	5.28	7,538.56	11,977.85	17,675.45	128,181.43
discountprice	38,843	12,662.77	7,894.35	4.90	7,036.02	11,170.48	16,469.27	118,922.62
quantity	38,843	262,304.18	160,063.58	168.00	149,160.00	231,522.00	341,162.00	2,697,521.00

Panel F: SGLT2 inhibitor

Variable	N	mean	std	min	25%	50%	75%	max
items	82,727	76.11	124.38	1.00	10.00	30.00	90.00	2,898.00
price	82,727	3,221.26	5,080.96	2.61	439.10	1,353.83	3,805.41	103,348.52
discountprice	82,727	2,987.61	4,714.31	2.52	408.83	1,252.43	3,533.16	95,943.08
quantity	82,727	2,522.89	3,956.82	2.00	364.00	1,064.00	2,976.00	80,562.00

Panel G: Sulfonylurea

Variable	N	mean	std	min	25%	50%	75%	max
items	110,809	408.33	658.16	1.00	12.00	54.00	612.00	11,769.00
price	110,809	1,425.74	2,234.91	0.05	58.18	296.60	2,117.62	55,743.63
discountprice	110,809	1,334.46	2,087.60	0.16	54.82	278.53	1,984.55	51,475.67
quantity	110,809	26,715.62	44,249.75	1.00	728.00	2,684.00	39,662.00	798,857.00

Panel H: Thiazolidinedione

Variable	N	mean	std	min	25%	50%	75%	max
items	50,527	115.44	166.60	1.00	16.00	57.00	146.00	2,782.00
price	50,527	1,031.08	1,759.29	0.30	176.11	466.57	1,112.65	36,006.63
discountprice	50,527	957.40	1,630.23	0.39	165.14	431.40	1,035.85	33,325.27
quantity	50,527	3,954.20	5,147.61	2.00	784.00	2,163.00	5,040.00	100,530.00

Notes: These eight panels report summary statistics on `items`, `price`, `discountprice`, and `quantity` for each "nest" (chemical class) across all observations (2013/14–2019/20). Each panel is labeled (A–H) in alphabetical order of the nest name.

Table 13: Summary Statistics of Prevalence Data

	N	mean	std	min	25%	50%	75%	max
Atrial fibrilation	38768	1.74	0.83	0.0	1.17	1.74	2.25	27.46
Coronary heart disease	38769	3.23	1.16	0.0	2.48	3.24	3.97	32.20
Cardiovascular disease	38762	1.42	0.97	0.0	0.85	1.16	1.66	60.00
Heart failure	38764	0.80	0.40	0.0	0.53	0.74	0.99	11.53
Left ventricular systolic dysf.	25981	0.29	0.26	0.0	0.11	0.21	0.39	3.06
Hypertension	38769	14.05	3.65	0.0	11.96	14.27	16.34	62.03
Peripheral arterial disease	38764	0.62	0.33	0.0	0.39	0.58	0.79	11.15
Stroke tr ischaemic attack	38769	1.73	0.72	0.0	1.27	1.74	2.16	22.64
Asthma	38770	5.96	1.30	0.0	5.16	6.01	6.80	15.57
Chronic obs. pulmonary dis.	38766	1.92	0.93	0.0	1.26	1.81	2.45	17.97
Obesity	38770	9.90	3.73	0.0	7.27	9.59	12.22	35.93
Cancer	38770	2.47	1.01	0.0	1.76	2.46	3.12	17.29
Chronic kidney disease	38767	4.07	2.02	0.0	2.64	3.86	5.23	27.61
Diabetes mellitus	38770	6.88	2.01	0.0	5.73	6.75	7.83	28.65
Palliative Care	38770	0.36	0.59	0.0	0.14	0.25	0.43	39.71
Dementia	38752	0.74	0.90	0.0	0.44	0.68	0.94	63.82
Depression (18+)	38770	8.60	3.93	0.0	5.84	8.13	10.82	38.47
Epilepsy (18+)	38769	0.80	0.42	0.0	0.62	0.79	0.96	63.69
Learning Disability	32295	0.48	0.28	0.0	0.29	0.43	0.61	5.42
Mental Health	38770	0.95	0.56	0.0	0.67	0.86	1.12	19.62
Osteoperosis	38231	0.43	0.47	0.0	0.14	0.27	0.53	4.25
Rheumatoid arthritis	38765	0.75	0.27	0.0	0.58	0.74	0.90	4.67

Notes: Prevalence figures are calculated using number of diagnosed divided by the relevant patient population and are reported as percentages. A total of 6778 unique practices are included in this dataset.

Table 14: Summary Statistics for Age-Sex composition of Practice Patient Lists

	N	mean	std	min	25%	50%	75%	max
All patients	38843	8186.13	4782.14	160.00	4702.00	7357.00	10663.00	72632.00
Male	38843	4074.48	2366.42	48.00	2368.00	3648.00	5296.00	36693.00
Female	38843	4111.66	2427.47	11.00	2331.00	3695.00	5389.00	35939.00
0-4 male	38843	200.71	125.65	0.00	111.00	176.00	261.00	1641.00
05-14 male	38843	475.25	301.70	0.00	270.00	422.00	614.00	6064.00
15-24 male	38843	513.74	493.97	0.00	275.00	427.00	626.00	13499.00
25-34 male	38843	585.56	431.54	0.00	314.00	487.00	732.00	8823.00
35-44 male	38843	562.22	345.88	0.00	317.00	491.00	729.00	5602.00
45-54 male	38843	585.53	340.84	0.00	335.00	527.00	769.00	5008.00
55-64 male	38843	461.84	278.90	0.00	255.00	411.00	614.00	3757.00
65-74 male	38843	379.94	261.57	0.00	183.00	327.00	520.00	3397.00
75-84 male	38843	218.43	154.24	0.00	103.00	185.00	298.00	2022.00
85+ male	38843	91.26	72.87	0.00	39.00	74.00	125.00	1024.00
0-4 female	38843	190.88	119.59	0.00	106.00	167.00	249.00	1600.00
05-14 female	38843	454.51	305.45	0.00	257.00	400.00	586.00	8849.00
15-24 female	38843	515.97	538.90	0.00	269.00	419.00	620.00	16534.00
25-34 female	38843	587.28	415.71	0.00	311.00	494.00	748.00	7655.00
35-44 female	38843	533.07	326.10	0.00	298.00	472.00	694.00	4861.00
45-54 female	38843	561.68	340.17	0.00	306.00	503.00	753.00	4552.00
55-64 female	38843	457.55	287.29	0.00	242.00	406.00	618.00	3707.00
65-74 female	38843	402.19	281.88	0.00	188.00	345.00	553.00	3540.00
75-84 female	38843	260.43	184.84	0.00	120.00	222.00	358.00	2464.00
85+ female	38843	148.09	115.55	0.00	62.00	121.00	204.00	1482.00

Note: This table reports summary for the age-sex groups of practice patient lists. The N is rounded to the nearest integer, while all other statistics are reported with two decimal places.

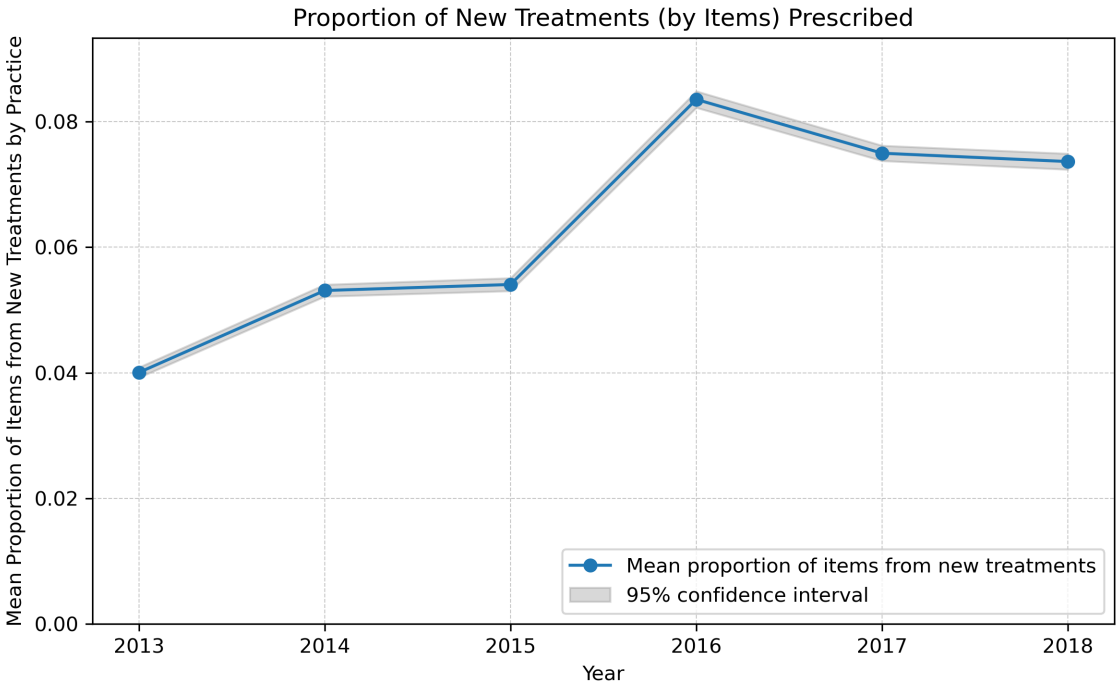
Table 15: Summary Statistics for Top 20 ICD-10 Disease Groups

Disease Group (ICD-10)	N	Mean	Std	Min	25%	50%	75%	Max
Pneumonia (J18)	38,843	68.12	50.71	0.00	31.00	57.00	92.00	725.00
Liveborn infants (Z38)	38,843	54.21	39.79	0.00	27.00	46.00	72.00	560.00
Abdominal and pelvic pain (R10)	38,843	48.71	34.27	0.00	25.00	41.00	64.00	609.00
Urinary system disorders (N39)	38,843	44.55	33.00	0.00	21.00	37.00	61.00	446.00
Chest pain (R07)	38,843	42.99	31.74	0.00	21.00	36.00	57.00	834.00
Cataract (H26)	38,843	32.77	29.82	0.00	12.00	24.00	45.00	415.00
Chronic obs. pulmonary dis. (J44)	38,843	31.92	27.92	0.00	13.00	25.00	43.00	423.00
Breast cancer (C50)	38,843	29.05	33.18	0.00	6.00	17.00	41.00	362.00
Age-related cataract (H25)	38,843	26.63	28.63	0.00	7.00	17.00	37.00	502.00
Sepsis (A41)	38,843	26.57	31.14	0.00	8.00	16.00	34.00	507.00
Lower respiratory infections (J22)	38,843	23.60	18.87	0.00	10.00	19.00	32.00	347.00
Back pain (M54)	38,843	23.12	19.14	0.00	10.00	18.00	31.00	355.00
Heart failure (I50)	38,843	22.96	18.51	0.00	10.00	19.00	31.00	275.00
Complications of pregnancy (O36)	38,843	22.48	28.32	0.00	6.00	13.00	28.00	404.00
Gallstones (K80)	38,843	22.06	16.23	0.00	10.00	19.00	30.00	256.00
Perineal laceration (O70)	38,843	21.62	15.44	0.00	11.00	18.00	29.00	214.00
Myocardial infarction (I21)	38,843	21.47	17.41	0.00	9.00	17.00	29.00	314.00
Atrial fibrillation (I48)	38,843	20.69	16.75	0.00	8.00	17.00	29.00	228.00
Diverticular disease (K57)	38,843	20.58	17.01	0.00	8.00	16.00	29.00	282.00
Gastrointestinal hemorrhage (K92)	38,843	20.23	15.82	0.00	9.00	16.00	27.00	286.00

Notes: Summary statistics are calculated for the top 20 ICD-10 disease groups based on Finished Consultant Episodes (FCEs). The columns include the number of observations (N), mean, standard deviation (Std), and the minimum (Min), 25th percentile (25%), 50th percentile (50%, or median), 75th percentile (75%), and maximum (Max) values.

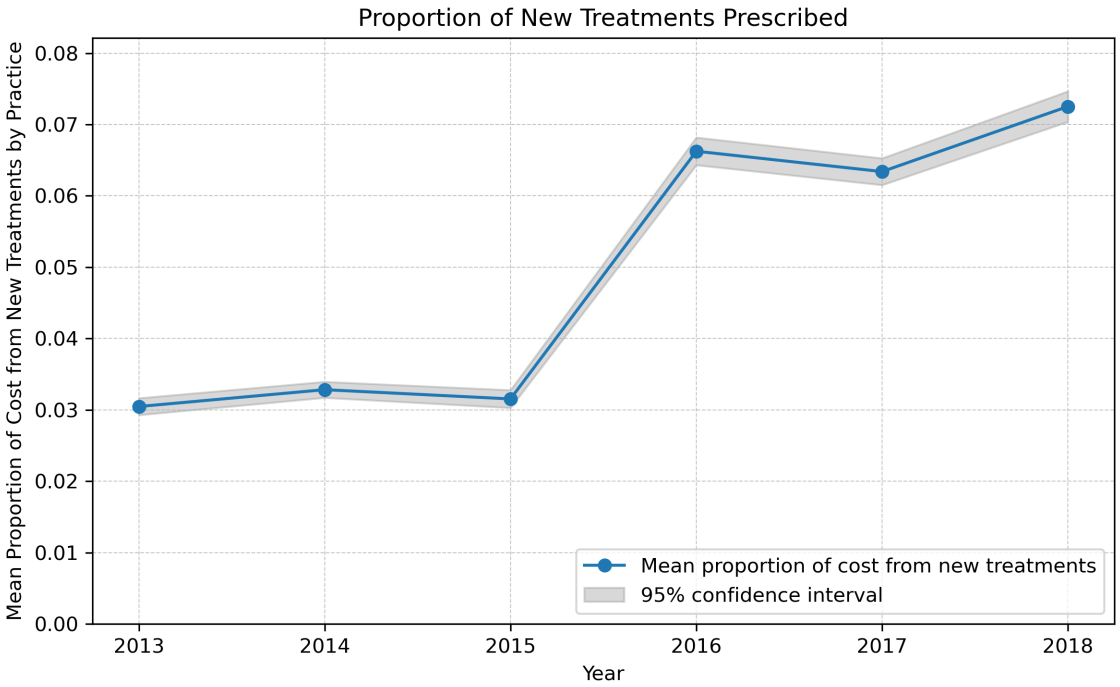
2 Figures

Figure 2: Proportion of New Treatments Prescribed



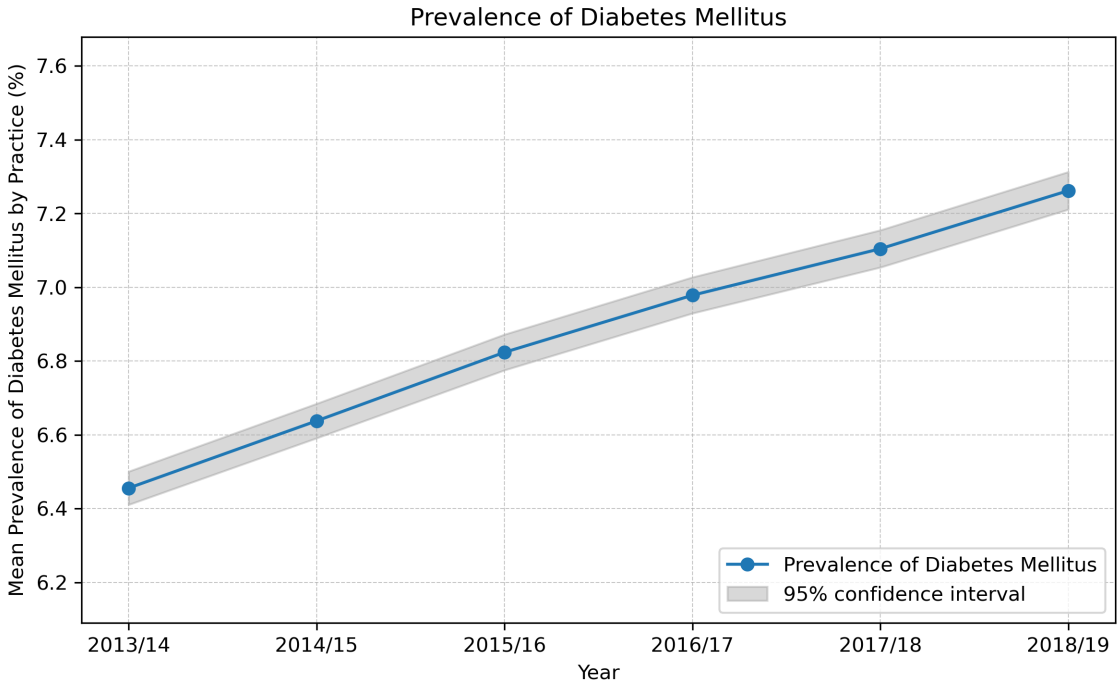
Notes: New treatments are classified as those treatments who received a marketing authorisation in the last 3 years. Data is aggregated to annual prescribing for all new treatments each year for each General Practice. Volumes are measures by each 'item' prescribed, e.g. a pack of tablets, vial, etc.

Figure 3: Proportion of Costs of New Treatments Prescribed



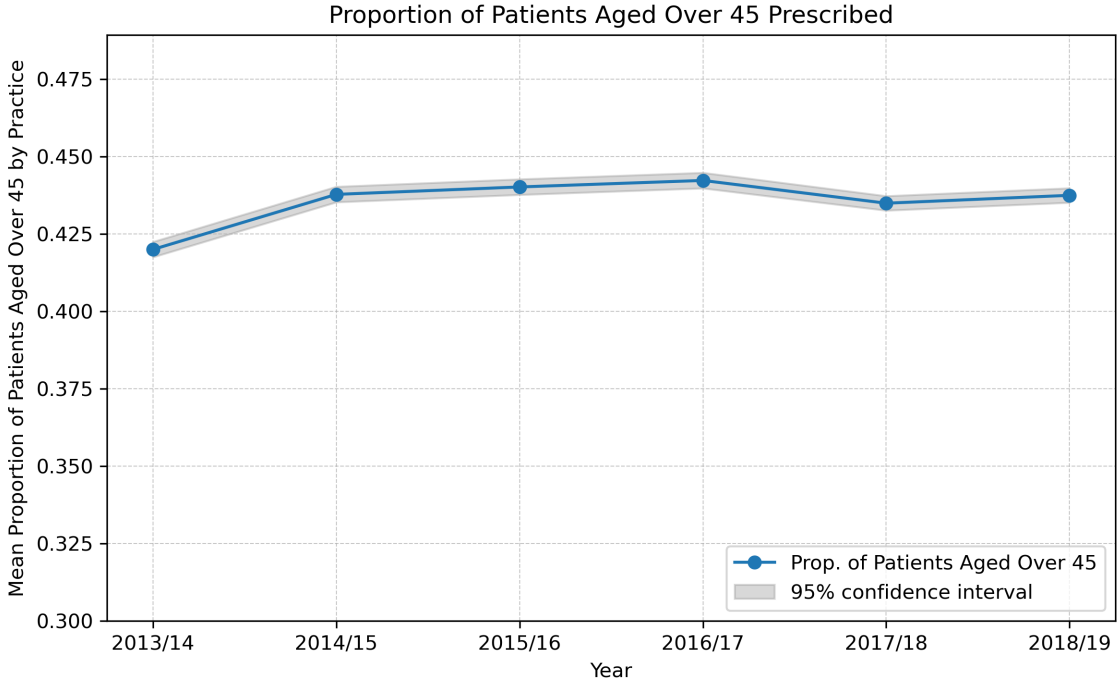
Notes: New treatments are classified as those treatments who received a marketing authorisation in the last 5 years. Data is aggregated to annual prescribing for all new treatments each year for each General Practice. Cost is measured by the reimbursement price paid for each 'item' dispensed.

Figure 4: Mean Prevalence of Diabetes Type II



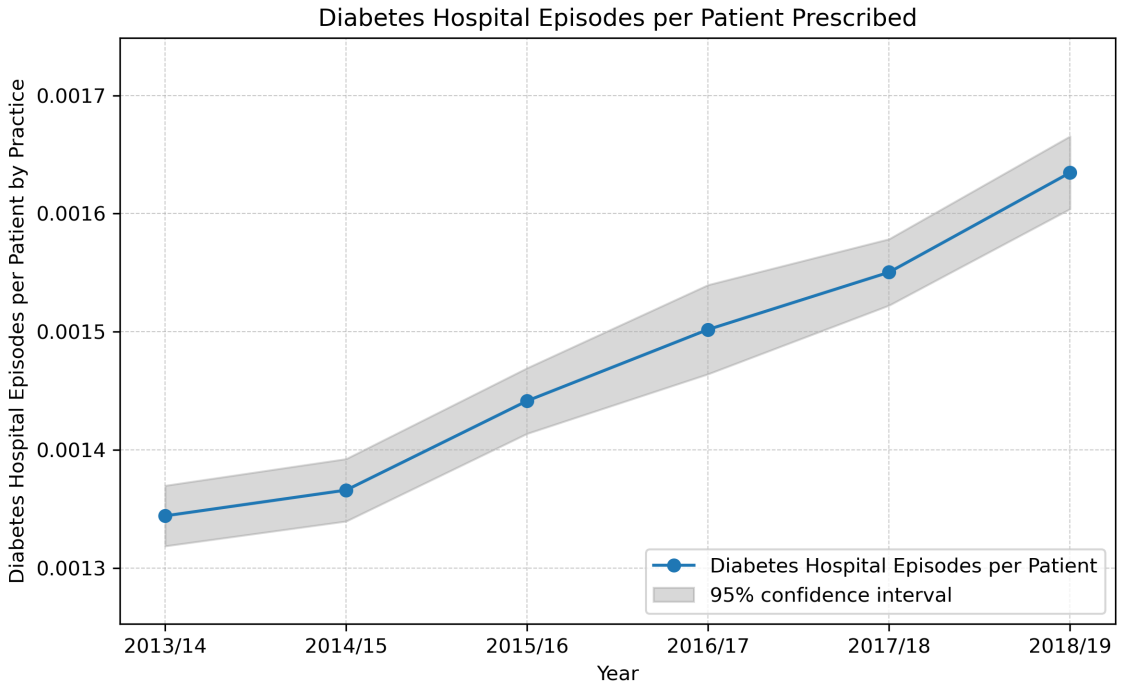
Notes: Prevalence is measured as the proportion of relevant patient list that is diagnosed with the condition for each GP.

Figure 5: Proportion of New Treatments Prescribed



Notes: Number of people over 45 on the patient list on the 1st of October for each year

Figure 6: GP Referred Diabetes Episodes Per Patient



Notes: A Finished Consultant Episode is a completed hospital in-patient spell for a health episode.