Hypertensive disorders of pregnancy and onset of chronic hypertension in France: the nationwide CONCEPTION study

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Received 20 April 2021; revised 23 July 2021; editorial decision 7 September 2021; accepted 14 September 2021

Aims
Hypertensive disorders of pregnancy (HDP) are a leading cause of maternal and foetal morbidity and mortality. We aimed to estimate the impact of HDP on the onset of chronic hypertension in primiparous women in the first years following childbirth.

Methods and results
This nationwide cohort study used data from the French National Health Data System (SNDS). All eligible primiparous women without pre-existing chronic hypertension who delivered between 2010 and 2018 were included. Women were followed up from six weeks post-partum until onset of hypertension, a cardiovascular event, death, or the study end date (31 December 2018). The main outcome was a diagnosis of chronic hypertension. We used Cox models to estimate hazard ratios (HRs) of chronic hypertension for all types of HDP. Overall, 2,663,573 women were included with a mean follow-up time of 3.0 years. Among them, 180,063 (6.73%) had an HDP. Specifically 66,260 (2.16%) had pre-eclampsia (PE) and 113,803 (4.27%) had gestational hypertension (GH). Compared with women who had no HDP, the fully adjusted HRs of chronic hypertension were 6.03 [95% confidence interval (CI) 5.89–6.17] for GH, 8.10 (95% CI 7.88–8.33) for PE (all sorts), 12.95 (95% CI 12.29–13.65) for early PE, 9.90 (95% CI 9.53–10.28) for severe PE, and 13.17 (95% CI 12.74–13.60) for PE following GH. Hypertensive disorders of pregnancy exposure duration was an additional risk factor of chronic hypertension for all PE subgroups. Women with HDP consulted a general practitioner or cardiologist more frequently and earlier.

Conclusion
Hypertensive disorders of pregnancy exposure greatly increased the risk of chronic hypertension in the first years following delivery.
Keywords
Pre-eclampsia • Gestational hypertension • Hypertension • Blood pressure • Epidemiology • Pregnancy complication

Introduction
Hypertensive disorders of pregnancy (HDP)—defined as pre-existing chronic hypertension, gestational hypertension (GH), and pre-eclampsia (PE)/eclampsia—constitute a global public health issue which is insufficiently tackled. They are the main cause of maternal morbidity during pregnancy and following childbirth in industrialized countries. Hypertensive disorders of pregnancy prevalence in pregnant women is estimated to be between 5% and 10%, and believed to impact primiparous women more often than their multiparous counterparts. A recent large-scale study of pregnant women in France, using data from the National Health Data System (SNDS), estimated an age-standardized HDP prevalence of 7.4% (9.1% and 6.3% in primiparous and multiparous women, respectively), and observed an upward trend in HDP prevalence due to older age at first childbirth and rising prevalence of obesity.

In the long term, HDP are associated with an increased risk of chronic hypertension and cardiovascular or renal events. However, little is known about the short-term impact of HDP on these outcomes. In a population-based study, Ray et al. reported that the risk of cardiovascular disease was higher after a maternal placental syndrome but did not assess the onset of hypertension. A single-centre study by Black et al. found that women with HDP were 2.36 [95% confidence interval (CI) 1.97–2.83] and 2.48 (95% CI 1.99–3.11) times more likely to develop pre-hypertension and hypertension in the year after delivery. To the best of our knowledge, the impact of the exposure duration of HDP on the early onset of chronic hypertension has not been assessed, nor have the different
configurations of PE (early PE, severe PE, PE following GH). In this context, we aimed to estimate the impact of different types of HDP on the early onset (i.e. first years after delivery) of chronic hypertension in women in France, and to assess the medical follow-up of women with a history of HDP.

Methods

Data source
The CONCEPTION (Cohort of Cardiovascular Diseases in Pregnancy) study is a prospective cohort designed to study hypertensive disorder and cardiovascular event epidemiology in French women who gave birth, using data from the French National Health Insurance Information System database (Système National des Données de Santé, or SNDS). This database contains comprehensive information on all healthcare expenditures reimbursed by the national health insurance system for the entire French population (~66 million people). It comprises two information sources: the National Hospital Discharge Database (PMSI), which records information on public and private hospital stays—including diagnoses—under ICD-10 codes, and the Interscheme Consumption Data Mart (DCIR), which records information on out-of-hospital care. More specifically, the DCIR contains reimbursements of healthcare expenditures (e.g. medicines, outpatient medical care).

Study population
All first deliveries of women included in CONCEPTION (hereafter referred as primiparous women) were eligible for the present analysis. The cohort population is described in detail elsewhere. Briefly, the national health insurance general scheme and mutual insurance companies (which provide complementary healthcare insurance cover) combined cover ~90% of the population in France. Of those covered, all primiparous women who gave birth in a hospital after 22 weeks of gestation between 1 January 2010 and 31 December 2018 were eligible for inclusion in CONCEPTION. Women with missing data in their medical history, those aged under 15 or over 49 years, those with a multiple pregnancy, those with a history of cardiovascular events, end-stage renal disease, women aged under 15 or over 49 years, those with a multiple pregnancy, and those with a history of cardiovascular events, end-stage renal disease, cardiac malformation, inflammatory disease, HIV before delivery, or chronic hypertension before pregnancy, were all excluded. Follow-up started 6 weeks after childbirth and ended either when hypertension occurred, when the woman died, when a second pregnancy occurred, or at the study end date (31 December 2018), whichever came first.

Exposure to hypertensive disorders of pregnancy
We used different algorithms to identify HDP in the SNDS, based on antihypertensive drug delivery and diagnoses of HDP during hospital stays.

Gestational hypertension was defined as having at least one delivery of antihypertensive drugs between 20 weeks of gestation and 6 weeks postpartum, or a hospital stay with a diagnosis of GH (ICD-10 code O13). To avoid potential misclassification, women who were reimbursed for antihypertensive drugs and were hospitalized with preterm labour as primary diagnosis (ICD10 codes O47, O60.0-O60.2, O60.9) were excluded from the GH group, as the antihypertensive treatment may have been prescribed for preterm labour.

Pre-eclampsia, eclampsia, and HELLP syndrome were identified from diagnoses in hospital stays (ICD-10 codes O14, O15, and O14.2, respectively). We considered PE to be severe when diagnosed during hospitalization (ICD-10 code O14.1), or when eclampsia or HELLP syndrome were diagnosed. For all other situations, it was considered simple.

Outcomes
Chronic hypertension was identified by at least three refills for antihypertensive drugs on different dates over a 12-month period, or on two dates if at least one large pack (90 pills) of antihypertensive drugs was dispensed. We considered the date of first drug delivery to be the date of chronic hypertension diagnosis.

Covariates
Sociodemographic data and medical history of the women included in the study, as well as information related to their pregnancy, post-partum period, and healthcare management, were all taken from the SNDS. Delivery mode (e.g. caesarean section), intrauterine foetal death, obesity (ICD-10 I66), and pregnancy-related haemorrhaging were identified from hospital records. Tobacco use was identified using an algorithm combining specific hospital coding and delivery of prescribed nicotine replacement therapy before or during pregnancy. A history of cardiovascular events and end-stage renal disease was identified in hospital records from 2006 onwards using the following ICD-10 codes: I20.0 and I21–I23 for acute coronary syndrome, I50 for heart failure, I26 for thrombocytopenic disease, and both O225 and O873 for cerebral thrombosis. Pre-existing diabetes was identified using an algorithm based on delivery of three antidiabetic drugs on three different dates in the year preceding pregnancy (or on two dates if at least one large package of antidiabetic drugs was delivered). Gestational diabetes was identified using an algorithm combining the delivery of insulin and glucose strips, or a diagnosis of diabetes during pregnancy with no pre-existing diabetes. Persons who benefited from Universal Medical Coverage (CMU) —a social benefit in France for those whose income is below a certain ceiling— were defined as living in social deprivation. All outpatient visits with a general practitioner, a cardiologist, a neurologist, a nephrologist, an endocrinologist, a gynaecologist, or a midwife were identified from one year before pregnancy until the end of follow-up.

Statistical analysis
We described the study population characteristics, follow-up time, and healthcare management, and computed a cumulative incidence curve for chronic hypertension stratified by type of HDP. Crude prevalence was then calculated for each type of HDP. Crude and adjusted hazard ratios (HR and aHR) with 95% CI for the development of chronic hypertension stratified by type of HDP. Crude prevalence was then calculated for each type of HDP, and from 0 to 20 weeks for GH as these periods comprised >99% of the onset of chronic hypertension according to the exposure duration to HDP. GH followed by PE was classified in the ‘PE following GH’ subgroup. Pre-eclampsia with foetal growth restriction was defined by a birth weight inferior to the tenth percentile for gestational age and sex. HDP exposure duration was calculated as the number of days between HDP (any type) diagnosis and childbirth.

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exposure durations in these populations. In order to better distinguish the specific effects of early PE and exposure duration to PE, we estimated HRs for the development of chronic hypertension in women with early PE compared to women with late PE, using Cox models (unadjusted, adjusted on covariates, and adjusted on exposure duration to PE and covariates).

### Results

The present analysis included 2,663,573 women participating in the CONCEPTION cohort for the period 1 January 2010 to 31 December 2018 (Figure 1). Table 1 presents their characteristics, according to the type of HDP diagnosed. Mean follow-up time was 3.0 years (interquartile range 1.3–4.2). Those with a history of HDP were older and had a lower gestational age at childbirth than those with no HDP history. The former were more likely to be obese, to live in social deprivation, and to have had pre-existing or gestational diabetes, a caesarean section, intravenous foetal death, and/or haemorrhaging during pregnancy. During follow-up, 47,533 (1.78%) women developed chronic hypertension. Women with HDP were more likely to develop it (8.69% and 10.84% of those with GH and PE, respectively) than women with no HDP (1.23%). In the former group, the mean duration of exposure to HDP was 35.8 days for GH and 7.8 days for PE.

Table 2 shows the crude prevalence of all types of HDP. Overall, 180,063 women (6.76%) were diagnosed with HDP during their pregnancy, specifically 113,803 women (4.27%) with GH and 66,260 women (2.49%) with PE. Among the latter, 11,666 (17.61%) had early onset PE, 26,921 (40.62%) had severe PE, and 86,665 (13.08%) had PE following GH.

The cumulative incidence curves (Figure 2) showed that the onset of chronic hypertension was faster in women diagnosed with GH or PE than in those with no HDP. Table 3 presents time-to-event analysis results according to type of HDP. Women with a history of HDP had a higher risk of chronic hypertension than those without HDP (aHR 6.77 [95% CI 6.64–6.90]; P < 0.0001). This risk was slightly higher for those with PE (aHR 8.10 [95% CI 7.88–8.33], P < 0.0001) than for those with GH (aHR 6.03 [95% CI 5.89–6.17], P < 0.0001). When compared to women with no HDP, the adjusted HRs of developing chronic hypertension were 9.90 (95% CI 9.53–10.28) for women with severe PE, 12.95 (95% CI 12.29–13.65) for women with early PE, 13.17 (95% CI 12.74–13.60) for women with PE following GH, and 12.49 (95% CI 11.76–13.27) for women with PE with foetal growth restriction. Complete outputs of the models are shown in Supplementary Material 1.

The Cox models with a natural spline on the exposure duration to HDPs showed that the adjusted HRs of developing chronic hypertension significantly and continuously increased with exposure duration to PE (Figure 3). This effect was greater for severe PE and for PE with foetal growth restriction, whereas for GH, this relation was fluctuating and of low magnitude.

When compared to late PE, early-onset PE was associated with an increased risk of chronic hypertension in both the unadjusted [HR 1.92 (95% CI 1.83–2.02), P < 0.0001] and adjusted [aHR 1.80 (95% CI 1.71–1.90), P < 0.0001] models. When the HDP exposure duration was taken into account, the association of chronic hypertension with early-onset PE persisted but was attenuated [aHR 1.69 (95% CI 1.61–1.79), P < 0.0001] (Figure 4).

Supplementary Material 2 displays consultation patterns of women according to type of HDP. During the first year following delivery, 2,198,194 (82.5%) women visited a general practitioner, 30,343 (1.1%) a cardiologist, 1,519,700 (57.1%) a gynaecologist or a midwife, and 116,510 (4.4%) a physician from another specialty (i.e., neurologist, endocrinologist, nephrologist). At the end of follow-up, these figures were, respectively, 2,457,109 (92.2%), 112,760 (4.2%), 2,010,689 (75.5%), and 233,702 (8.8%). Median time to first visit was ~2 months for a general practitioner and gynaecologist or midwife (64 and 53 days, respectively), 717 days for a cardiologist, and 343 days for a physician from another specialty. Women diagnosed with PE visited a gynaecologist or a midwife less often and later than those with no PE (P < 0.0001). However, women with a history of HDP, and especially former PE women, visited physicians with other specialties more often and earlier than those without HDP.

### Discussion

#### Main findings

This large-scale nationwide prospective cohort of 2,663,573 primiparous women in France enabled us to accurately estimate chronic hypertension onset according to various types of HDP. We found that the risk of chronic hypertension increased dramatically and rapidly in the years following delivery in women who developed GH (aHR 6.03) or PE (aHR 8.10) (Graphical abstract). This risk was even greater in women with early PE, severe PE, or PE following GH. While the duration of PE was a risk factor of chronic hypertension, this effect was unclear for GH. We also found that women with a history of HDP, and especially former PE women, visited physicians more often and earlier than those without HDP.

#### Interpretation

One interesting finding of the present analysis is that while GH is a less severe hypertensive disorder than PE, they were both associated with similarly high risks of developing chronic hypertension. Many previous studies have clearly shown that women with a history of HDP, in particular PE, have an increased risk of developing chronic hypertension and cardiovascular diseases. Some authors have more specifically studied early PE or PE with foetal growth restriction and have demonstrated a further increased risk. Fewer authors have focused their reports on GH without PE. Several risk factors for HDP, for example, family history of hypertension, obesity, and diabetes are also risk factors for chronic hypertension outside pregnancy. It remains unclear whether these shared risk factors explain both the occurrence of HDP and the subsequent chronic hypertension, or if HDP generates, with a causal relationship, chronic hypertension. Given that our models were adjusted for the main risk factors of hypertension onset, including obesity and cardio-metabolic history, the latter hypothesis appears to be the most likely. Moreover, we found a significant relationship between the duration of exposure to PE and the subsequent occurrence of chronic hypertension. To the best of our knowledge, this result has never previously been reported, unlike the effects of severe or early PE. This increased risk of chronic hypertension, depending on the duration of...
exposure to PE, would rather be in favour of a vascular toxicity of pathophysiological phenomena linked to PE. Without prejudging the pathophysiological mechanisms that were not accessed in our study, we can hypothesize that the vascular damages induced by PE directly and quantitatively contribute to the future development of chronic hypertension. Indeed, Pruthi et al.\textsuperscript{21} reported that after vessel injury, PE-exposed mice had significantly enhanced vascular remodelling with increased vascular smooth muscle cell proliferation and vessel...
fibrosis compared to control pregnancy. These data support a model in which vessels exposed to PE quantitatively retain a persistently enhanced vascular response to injury. Gestational hypertension followed by PE is rarely reported in the international literature and is often included in the PE group. The statistical power of our study allowed us to study this specific type of HDP, hypertensive disorder of pregnancy; NA, not available; SD, standard deviation.

### Table 1 Population characteristics by type of hypertensive disorder of pregnancy

<table>
<thead>
<tr>
<th>Total</th>
<th>Hypertensive disorders</th>
<th>Gestational hypertension</th>
<th>Pre-eclampsia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>% or mean (SD)</td>
<td>N</td>
</tr>
<tr>
<td>Maternal age (years)</td>
<td>2 663 573</td>
<td>28.17 (5.32)</td>
<td>2 483 510</td>
</tr>
<tr>
<td>Social deprivation*</td>
<td>367 841</td>
<td>13.81</td>
<td>341 343</td>
</tr>
<tr>
<td>Obesity</td>
<td>103 617</td>
<td>3.89</td>
<td>88 719</td>
</tr>
<tr>
<td>Pre-existing diabetes</td>
<td>11 631</td>
<td>0.44</td>
<td>9633</td>
</tr>
<tr>
<td>Tobacco</td>
<td>233 940</td>
<td>8.78</td>
<td>218 434</td>
</tr>
</tbody>
</table>

### Table 2 Crude prevalence of hypertensive disorders of pregnancy

<table>
<thead>
<tr>
<th>Hypertensive disorders of pregnancy</th>
<th>N</th>
<th>Crude prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No hypertensive disorder of pregnancy</td>
<td>2 483 510</td>
<td>93.24</td>
</tr>
<tr>
<td>Gestational hypertension</td>
<td>113 803</td>
<td>4.27</td>
</tr>
<tr>
<td>Pre-eclampsia (total)</td>
<td>66 260</td>
<td>2.49</td>
</tr>
<tr>
<td>Pre-eclampsia following gestational hypertension</td>
<td>8665</td>
<td>0.33</td>
</tr>
<tr>
<td>Early pre-eclampsia</td>
<td>11 666</td>
<td>0.44</td>
</tr>
<tr>
<td>‘Simple’ pre-eclampsia*</td>
<td>39 339</td>
<td>1.48</td>
</tr>
<tr>
<td>Severe pre-eclampsia</td>
<td>26 921</td>
<td>1.01</td>
</tr>
</tbody>
</table>

*Persons who benefitted from Universal Medical Coverage (CMUc), a social benefit in France for those whose income is below a certain ceiling, were defined as living in Social Deprivation. According to the guidelines of the European Society of Cardiology and the European Society of Hypertension, annual visits to primary care physician to check blood pressure and metabolic factors are recommended for women who developed GH or PE. In the present study, although women with HDP consulted more frequently and earlier than those with no HDP, their healthcare management was not optimal. Moreover, women with a history of PE consulted a physician more frequently and earlier than those with a history of GH. However, both groups had quite a similar risk of developing chronic hypertension. To adequately screen for hypertension and prevent associated renal events.
complications, patients with HDP and attending physicians should be made more aware of the importance of follow-up.

**Strengths**

The main strength of this study is its nationwide design. This was made possible by the use of data from the SNDS, a national medico-administrative database with near-exhaustive data for hospital records, which provides great accuracy when identifying pregnancies in France (99.6% of all births in France are reported in the SNDS). The comprehensiveness of the data on our study population and on their health expenditures (including antihypertensive drug delivery) ensured optimal statistical power to obtain precise estimates for each type of HDP, and to perform subgroup analyses. Furthermore, our decision to assume that antihypertensive drug delivery was a

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**Figure 2** Cumulative incidence of chronic hypertension following childbirth according to type of hypertensive disorder of pregnancy. ‘At risk’ means that the subject did not have an event before time t, and was not censored before or at time t. HDP, hypertensive disorder of pregnancy.

**Table 3** Hazard ratios of hypertensive disorders of pregnancy in unadjusted and adjusted Cox regression models explaining the onset of persistent hypertension

<table>
<thead>
<tr>
<th></th>
<th>Unadjusted Cox models</th>
<th>Fully adjusted Cox models</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>HR</td>
</tr>
<tr>
<td>Any type of HDP</td>
<td>No</td>
<td>2,483,510</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>180,063</td>
</tr>
<tr>
<td>GH alone</td>
<td>No</td>
<td>2,483,510</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>113,803</td>
</tr>
<tr>
<td>Any type of pre-eclampsia</td>
<td>No</td>
<td>2,483,510</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>66,260</td>
</tr>
<tr>
<td>GH with superimposed pre-eclampsia</td>
<td>No</td>
<td>2,483,510</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>8665</td>
</tr>
<tr>
<td>Severe pre-eclampsia</td>
<td>No</td>
<td>1,571,891</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>17,026</td>
</tr>
<tr>
<td>Early pre-eclampsia</td>
<td>No</td>
<td>1,571,891</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>7455</td>
</tr>
<tr>
<td>Pre-eclampsia with foetal growth restriction</td>
<td>No</td>
<td>1,571,891</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>14,271</td>
</tr>
</tbody>
</table>

CI, confidence interval; GH, gestational hypertension; HDP, hypertensive disorder of pregnancy; HR, hazard ratio; Ref, reference.

*Adjusted on year of delivery, maternal age, social deprivation, gestational diabetes, obesity, tobacco and history of diabetes.

*Based on deliveries during the period 2013–2018.
proxy for the diagnosis of chronic hypertension probably underestimates the true prevalence of this condition in post-partum women in France. Finally, by studying the impact of the exposure duration for each type of HDP, we were able to acquire a greater understanding of the mechanisms behind the increased risk of chronic hypertension in this population.

Limitations

As we identified hypertension only by refunds of antihypertensive drugs, the proportion of women who develop hypertension is probably underestimated. However, this ranking bias is probably not differential between women who developed HDP and those who did not. A previous evaluation of algorithms used to identify GH and PE showed that physicians may decide not to treat women with non-severe GH (i.e. <160/110 mmHg). Consequently, untreated moderate GH not reported during hospitalization could have been missed, and therefore GH underestimated. Furthermore, misclassifying women with moderate GH in the group of women without HDP (reference) may have led to an underestimation of the risk of chronic hypertension in all HDP groups. In addition, we cannot exclude residual confounding, especially concerning obesity, a major risk factor of chronic hypertension. Indeed, we may have underestimated the prevalence of obesity as the condition was identified through hospital records for hospitalization for childbirth, leading to a non-differential bias of the estimates towards the null. Similarly, we probably underestimated tobacco use, as it was only assessed by the delivery of nicotine replacement therapy before or during pregnancy. Moreover, we had no data on alcohol consumption, family history of hypertension, and weight gain during pregnancy.

Figure 3 Hazard ratios of developing chronic hypertension according to exposure duration of hypertensive disorders of pregnancy (A–E). Hazards ratios modelled with Cox regression models with a natural spline on the exposure duration, adjusted on year of delivery, maternal age, social deprivation, gestational diabetes, obesity, tobacco, and history of diabetes (the reference modality for hazard ratios is an exposure duration of 0 week). Gray stripes represent the 95% confidence interval of the hazard ratios. PE, pre-eclampsia.
Finally, since our study focused on primiparous women, our results cannot be extrapolated to all pregnancies. Further studies should be conducted to study the impact of repeated HDP on chronic hypertension.

**Conclusion**

Hypertensive disorders of pregnancy in primiparous women were associated with a substantially increased risk of developing chronic hypertension in the first years following delivery. Active screening strategies for hypertension should be implemented for women with HDP, especially those with early or severe PE, or PE following GH. Further research is needed to better understand the reasons underlying the early onset of chronic hypertension following delivery, and guidelines should be developed to improve hypertension screening and management as well as cardiovascular prevention for these women.

**Supplementary material**

Supplementary material is available at European Heart Journal online.

**Declaration of Helsinki and ethics approval**

The authors state that this study complies with the Declaration of Helsinki. In line with French national regulations and ethics committee, participant consent was not required for this study. Santé Publique France—the French public health agency—has full and chronic access to the SNDS (governmental deliberation no. 2016-316, 13 October 2016).

**Acknowledgements**

We thank Ms Alice Martin for revising the manuscript and Mr Edouard Chatignoux for his methodological support with Cox models with natural splines.

**Funding**

This work was supported by the French Hypertension Society (SFHTA), the Hypertension Research Foundation (FRHTA), and the French Cardiology Federation through the call for scientific projects ‘Thematic grant 2019: cardiovascular diseases in women’. The funders had no role in the study design, data collection, data analysis, decision to publish, or drafting of the manuscript.

**Conflict of interest:** S.K. reports, outside the submitted work, non-financial support from Lilly France, Novonordisk, Novartis Pharma, Roche diabetes care, Lifescan, Abbott France, Sanofi, ViVi Healthcare, Servier, Becton Dickinson and personal fees from Icomed, Pascaleo, BT3SI, M3global research. C.M.-V. reports, outside the submitted work, personal fees and non-financial support from Servier, Lundbeck, AstraZeneca, MSD, and Boehringer. J.B. reports, outside the submitted work, personal fees from Abbott, Bayer, Bottu, Ferring, Steripharma, Kantar, Teriak, personal fees and non-financial support from Pfizer, Quantum Genomics, personal fees from Sanofi and Servier. All other authors declared no conflict of interest.

**Data availability**

We cannot share National Health Data System data as they are only available on a secure portal. Authorisation to access this portal needs registration and clearance.

**References**


