



Essays in the Econometrics of Asset Pricing and Public Health

Matthias Schmidtblaicher

Thesis submitted for assessment with a view to obtaining the degree of
Doctor of Economics of the European University Institute

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Department of Economics

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I confirm that chapter 2 was jointly co-authored with Peter R. Hansen and I contributed 50% of the work. The chapter was also published in the *Journal of Business & Economic Statistics* in July 2019.

I moreover confirm that chapter 3 was jointly co-authored with Peter R. Hansen and Noel T. Brewer and I contributed 50% of the work. An article that we published in *Vaccine* in January 2020 draws upon the chapter.

Signature and Date:

A handwritten signature in blue ink that reads 'Matthias Schmidtlaicher'.

January 29, 2019

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Abstract

In this thesis, I employ techniques from time series econometrics to study the detection of asset price bubbles and the measurement of vaccine uptake.

In Chapter 1, I analyse some commonly used bubble detection tests that have been derived from simplified present value relationships. I apply the tests to a flexible, estimated present value model and study their size and power properties. I find that, under the null hypothesis of price being equal to fundamental value, no time series based test allows for correct inference under conventional critical values. Comparing tests under the alternative hypothesis, tests for periodically explosive growth minimize maximum regret.

The next two chapters analyse human papillomavirus (HPV) vaccinations in Denmark. In Chapter 2, coauthored with Peter R. Hansen, we develop a dynamic model of vaccine compliance, based on the score-driven paradigm. The model estimates a time-varying compliance parameter, controlling for age effects and changing seasonality, and can diagnose events that impacted vaccine compliance. We apply the model to the weekly HPV vaccination data and find that compliance fell sharply following the broadcast of a controversial TV documentary. We also find that vaccine-critical media stories predict drops in compliance.

Chapter 3, coauthored with Peter R. Hansen and Noel T. Brewer, analyses more recent HPV vaccination data that have been sampled monthly. The lower frequency enables us to employ a simple method to control for seasonal and age effects. The longer sample period allows for the evaluation of a national information campaign that advertised the HPV vaccine's safety and effectiveness. We find that, after the information campaign began, HPV uptake recovered to its baseline level. Still, we estimate that 26,000 fewer girls received their first HPV vaccination as a result of the period of low compliance studied in Chapter 2, compared to a scenario in which uptake had not declined.

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A large part of this thesis would not have materialized without the Statens Serum Institute's

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Chapter 1

How to Test for Bubbles

1.1 Introduction

In recent years, rational bubbles have become more prominent in macroeconomic models.¹ The decision as to whether to think of an asset price as containing a bubble or not should be guided by empirical findings. Inference about bubbles is, regrettably, not straightforward, because the fundamental value of the asset is unobserved. The prevalent econometric approach is, therefore, to test auxiliary hypotheses that are easier to evaluate than fundamental value. I evaluate four common procedures: tests for explosive (Diba and Grossman 1988) or periodically explosive (Phillips, Wu, and Yu 2011) price growth; tests for cointegration between prices and dividends (Campbell and Shiller 1987); and variance ratio tests (Shiller 1981; Cochrane 1992, 2011). In the following, I will refer to these tests of auxiliary hypotheses as “bubble tests.” I apply these bubble tests to prices generated by an estimated model of fundamental value and study their characteristics.

1. For some examples, see Farhi and Tirole (2012), Galí (2014), or Galí and Gambetti (2015).

Empirical evidence on the performance of bubble tests is mixed.² On the one hand, tests for periodically explosive price growth identify some historical episodes that are conventionally considered bubble episodes (Phillips, Wu, and Yu 2011; Phillips and Yu 2011). On the other hand, Giglio, Maggiori, and Stroebel (2016) show that inferences drawn from tests for explosive price growth or cointegration contradict findings from more direct tests of the fundamental value hypothesis. Comparing bubble tests, some tests seem to discover bubbles more frequently than others. For instance, tests for periodically explosive price growth detect statistically significant bubble characteristics in a variety of asset classes (Phillips, Wu, and Yu 2011), whereas a variance ratio test applied to a broad stock market index finds no evidence for bubbles (Cochrane 2011). In this chapter, I improve the understanding of these conflicting findings by eliciting the size and power properties of different bubble tests.

The reason why a bubble test might provide an incorrect inference is that it is derived under a specific asset pricing model and, therefore, suffers from the joint hypothesis problem described by Fama (1970) in the context of market efficiency tests: we cannot be sure that a rejection is a rejection of the fundamental value hypothesis because it could simply be a rejection of the specific model for fundamental value. Tests for explosive or periodically explosive price growth rely on dividends being a martingale and the discount rate being constant. Cointegration tests require a constant discount rate. The variance ratio test by Cochrane (1992, 2011) allows for time variation in both dividend growth and discount rate, but it requires the computation of long-horizon regressions. These regressions are difficult. In this chapter, I evaluate the joint hypothesis problem by applying the tests to prices that are generated by a flexible asset pricing model with time variation in dividend growth, the discount rate, and in volatility. These generalizations are central to modern asset pricing and a bubble test should account for them.

When applying the tests to fundamental prices generated from the general present value

2. For a critical review of a previous generation of bubble detection tests, see Gürkaynak (2008).

model, I find that neither cointegration tests nor tests for explosive price allow correct inference. The failure of cointegration tests is due to time variation in the discount rate, reproducing the finding by Timmermann (1995). Tests of periodically explosive price growth require both a constant discount rate and white noise dividend growth for correct size, with most overrejections occurring due to time-varying dividend growth. Tests for periodically explosive price growth are, however, robust to time-varying volatility, which mirrors findings by Phillips, Shi, and Yu (2015a).

Although inference under conventional critical values is misleading, bubble test statistics may still provide information about bubbles. To assess this claim, I introduce a simple bubble model and evaluate the trade-off between size and power. I find that, for most parameter configurations, tests for periodically explosive price growth have the highest power when controlling size. In terms of maximum regret loss, tests for periodically explosive price growth are superior to the other tests. Critical values, however, depend on the present value model at hand. Conventional critical values in tests for periodically explosive growth will be only appropriate if we interpret rejection as rejection of the hypothesis that prices are generated by a present value model with constant discount rate and white noise dividend growth.

The rest of the chapter is organized as follows. Section 1.2 recapitulates the theory of bubbles and of auxiliary hypotheses for bubble tests. Section 1.3 introduces the present value model and Section 1.4 explains the estimation approach. The specifications of the different bubble tests are introduced in Section 1.5. Section 1.6 reports the results from simulations under the null hypothesis. Section 1.7 evaluates the tests under both the null hypothesis and the alternative. Section 1.8 discusses the implications of the results. Technical derivations and robustness checks are in the appendix.

1.2 Tests for bubbles and tests of auxiliary hypotheses

Consider an infinitely lived asset with price P_t and dividend D_t . The expected return or discount rate over the next period is

$$R_t^e = \mathbb{E}_t \left[\frac{P_{t+1} + D_{t+1}}{P_t} \right].$$

Rearranging and iterating forward yields

$$P_t = \mathbb{E}_t \left[\sum_{i=1}^{\infty} \frac{D_{t+i}}{\prod_{j=0}^{i-1} R_{t+j}^e} \right] + B_t.$$

If we have $\lim_{i \rightarrow \infty} \mathbb{E}_t \left[\frac{P_{t+i}}{\prod_{j=0}^{i-1} R_{t+j}^e} \right] = 0$, then the price will be equal to the sum of discounted future dividend payments or fundamental value

$$F_t = \mathbb{E}_t \left[\sum_{i=1}^{\infty} \frac{D_{t+i}}{\prod_{j=0}^{i-1} R_{t+j}^e} \right]. \quad (1.1)$$

More generally, solutions are given by

$$P_t = F_t + B_t,$$

where B_t is the bubble component. It can be verified that B_t obeys

$$R_t^e B_t = \mathbb{E}_t[B_{t+1}]. \quad (1.2)$$

We want to test the hypotheses

$$\{H_0 : B_t = 0 \forall t\} \text{ vs. } \{H_1 : B_t > 0 \text{ for some } t\}.$$

This test is hard to perform because F_t is not observed. Various tests of auxiliary hypotheses have, therefore, been proposed. In the following, I will refer to H_0 as the “fundamental hypothesis” and to H_1 as the “bubble hypothesis.”

1.2.1 Explosive price growth

Because the discount rate is larger than one, (1.2) implies that B_t grows at the rate $a_t^B = R_t^e > 1$. Tests for explosive price growth (Diba and Grossman 1988) therefore check for explosive growth in the observed price

$$\{H_0^{\text{ex}} : a_t = 1 \forall t\} \text{ vs. } \{H_1^{\text{ex}} : a_t > 1 \text{ for some } t\}, \quad (1.3)$$

where a_t is the expected growth rate of P_t , $a_t = \frac{\mathbb{E}_t[P_{t+1}]}{P_t}$. As Diba and Grossman (1988) elaborate, without further assumptions regarding F_t , this test can only be used in a contrapositive manner: if we accept H_0^{ex} , we can accept the fundamental hypothesis because the presence of a bubble necessitates explosive price growth. Rejection of H_0^{ex} , on the other hand, does not imply rejection of the fundamental hypothesis because F_t may grow explosively. I next illustrate the latter point.

Maintaining that the fundamental hypothesis holds, the expected growth rate of fundamental prices is given by

$$a_t^F = \frac{\mathbb{E}_t[F_{t+1}]}{F_t} = R_t^e - \frac{\mathbb{E}_t[D_{t+1}]}{F_t}, \quad (1.4)$$

using (1.1). If the discount rate is constant $R_t^e = R > 1$ and dividends follow a martingale, we will have $F_t = \frac{R^{-1}D_t}{1-R^{-1}}$ and, therefore, $a_t^F = 1$. More generally, a_t^F can take values larger than one. As an example, consider the discount rate process

$$R_{t+1}^e - \bar{R}^e = \varphi(R_t^e - \bar{R}^e) + \nu_{t+1}, \quad \mathbb{E}_t[\nu_{t+1}] = 0,$$

and let dividends be independent of R_t^e with expected growth rate G : $\mathbb{E}_t[D_{t+i}] = G^i D_t$. Linearizing (1.1) around \bar{R}^e (see Timmermann 1995, and Appendix 1.A.1), we get

$$a_t^F = \frac{\mathbb{E}_t F_{t+1}}{F_t} \approx \frac{G(\gamma - \varphi(R_t - \bar{R}^e))}{\gamma - (R_t - \bar{R}^e)}$$

in a region around \bar{R}^e , with $\gamma = \frac{1}{\varphi} \frac{(\bar{R}^e)^2}{G} (1 - \varphi \frac{G}{\bar{R}^e})$. First, consider the case when $R_t = \bar{R}^e$. Then, we will have $a_t^F > 1$ if $G > 1$, so dividend growth implies a submartingale in fundamental prices. Second, letting $G = 1$ but letting $R_t > \bar{R}^e$ also implies $a_t^F > 1$. If discount rates are above their mean, they will fall, which will drive up prices.³ Hence, associating a rejection of H_0^{ex} with a rejection of the fundamental hypothesis presupposes both a constant discount rate and no submartingale in the dividend process.

1.2.2 Cointegration between prices and dividends

Under the fundamental hypothesis and a constant discount rate, (see Campbell and Shiller 1987, and 1.A.2 in the appendix), we can rewrite (1.1) as

$$F_t - \frac{R^{-1}}{1 - R^{-1}} D_t = \frac{1}{1 - R^{-1}} \sum_{i=1}^{\infty} \mathbb{E}_t [R^{-i} \Delta D_{t+i}]. \quad (1.5)$$

If dividends are integrated of order one (I(1)), the right-hand side of (1.5) will be I(0). In that case, D_t and F_t are cointegrated with cointegrating vector $(1, -\frac{R^{-1}}{1 - R^{-1}})$. On the other hand, if prices contain a bubble, we will have

$$P_t - \frac{R^{-1}}{1 - R^{-1}} D_t = \frac{1}{1 - R^{-1}} \sum_{i=1}^{\infty} \mathbb{E}_t [R^{-i} \Delta D_{t+i}] + B_t,$$

3. A similar mechanism is explored by Phillips and Yu (2011).

where, by (1.2), B_t is non-stationary. Under constant discount rates, a valid auxiliary test for a bubble is therefore

$$\{H_0^{\text{ci}} : (P_t, F_t)' \not\sim \text{CI}(1, 1)\} \text{ vs. } \{H_1^{\text{ci}} : (P_t, F_t)' \sim \text{CI}(1, 1)\}. \quad (1.6)$$

Note that the null hypothesis of no cointegration H_0^{ci} corresponds to the bubble hypothesis and that H_1^{ci} corresponds to the fundamental hypothesis.

1.2.3 Variance ratio test

Cochrane (1992) derives a second order approximation to the fundamental price-dividend ratio

$$\text{Var}(FD_t) = \frac{1}{1-\Omega} \sum_{j=1}^{\infty} \Omega^j \text{Cov}(FD_t, \Delta d_{t+j}) - \frac{1}{1-\Omega} \sum_{j=1}^{\infty} \Omega^j \text{Cov}(FD_t, r_{t+j}),$$

where $\Delta d_t = \log D_t - \log D_{t-1}$, $r_t = \log R_t$, and $\Omega = e^{\mathbb{E}[\Delta d_t] - \mathbb{E}[r_t]}$. Dividing by $\text{Var}(FD_t)$, we have

$$1 = \frac{1}{1-\Omega} \sum_{j=1}^{\infty} \Omega^j \beta_j^d - \frac{1}{1-\Omega} \sum_{j=1}^{\infty} \Omega^j \beta_j^r,$$

where β_j^i is the coefficient of a univariate regression of the j 'th lead of variable $i \in \{d, r\}$ on FD_t .

Under the bubble hypothesis, such a regression on the price-dividend ratio PD_t will yield

$$1 > \frac{1}{1-\Omega} \sum_{j=1}^{\infty} \Omega^j \beta_j^d - \frac{1}{1-\Omega} \sum_{j=1}^{\infty} \Omega^j \beta_j^r,$$

maintaining that $\text{Cov}(PD_t, B_t) > 0$. A test of the fundamental hypothesis in this framework is therefore given by

$$H_0^{\text{vdlev}} : \frac{1}{1-\Omega} \sum_{j=1}^{\infty} \Omega^j (\beta_j^d - \beta_j^r) = 1 \quad \text{vs.} \quad H_1^{\text{vdlev}} : \frac{1}{1-\Omega} \sum_{j=1}^{\infty} \Omega^j (\beta_j^d - \beta_j^r) < 1. \quad (1.7)$$

Unlike the tests for explosive price growth or cointegration, the variance ratio test explicitly allows for a time-varying discount rate. Cochrane (2011) derives an analogous test in logs that uses the Campbell and Shiller (1988) return approximation, which I describe in 1.A.3 in the appendix.

The relative merits of the tests in evaluating the fundamental hypothesis is unclear. In order to draw an inference about the presence of a bubble, the test for explosive price growth relies on a constant discount rate and a martingale in dividends. The cointegration test assumes a constant discount rate. The variance ratio test allows for a time-varying discount rate, but is based on an approximation and requires computations of long-horizon regressions. Accordingly, the analytical viewpoint cannot determine which of the tests should be preferred. Therefore, I compare the tests using simulated data, generated by a flexible present value relationship. The present value model is described next.

1.3 Model

Dividend growth Δd_t and the log discount rate $r_t^e = \log(R_t^e)$ follow first order autoregressive processes with heteroscedastic innovations

$$\Delta d_{t+1} = \mu_d + \varphi_d(\Delta d_t - \mu_d) + \sigma_d \sqrt{h_t} \epsilon_{d,t+1} \quad (1.8)$$

$$r_{t+1}^e = \mu_r + \varphi_r(r_t^e - \mu_r) + \sigma_r \sqrt{h_t} \epsilon_{r,t+1}. \quad (1.9)$$

A period is one month. The conditional variance evolves as

$$h_{t+1} = \alpha(1 - \beta)\epsilon_{h,t+1}^2 + \beta h_t. \quad (1.10)$$

We also observe a proxy of the latent variance process

$$RV_{t+1} = \gamma + h_{t+1} + \sigma_{rv}\epsilon_{rv,t+1}. \quad (1.11)$$

The distributional assumption is

$$(\epsilon_{d,t+1}, \epsilon_{r,t+1}, \epsilon_{h,t+1}, \epsilon_{rv,t+1})' | \mathcal{F}_t \sim \mathcal{N}(0, \mathbf{I}_4)$$

and the parameters are further restricted as $(\varphi_d, \varphi_r, \beta)' \in (-1, 1)^3$ and $(\sigma_d, \sigma_r, \alpha, \sigma_{rv})' \geq \mathbf{0}$.

I now discuss the modeling choice. First, both dividend growth and the discount rate are allowed to exhibit persistence. Persistence in the discount rate is key for a present value model to describe asset price data (see e.g., Cochrane 2011). Dividend growth is often modelled as white noise. Recent empirical evidence (Van Binsbergen and Koijen 2010; Schorfheide, Song, and Yaron 2018), however, points at persistence in dividend growth, so I allow for it. The conditional variance (1.10) resembles the functional form of a generalized autoregressive conditional heteroskedasticity (GARCH) process (Bollerslev 1986). It differs from a GARCH in two ways: first, $\epsilon_{h,t}$ is an independent shock rather than a residual, so (1.10) is parameter driven rather than observation driven; second, the innovations to h_t are homoscedastic. I use (1.10) to model the conditional variance because it ensures a positive h_t while allowing for a tractable price expression. Finally, (1.11) defines a linear measurement equation for the realized variance RV_t as a proxy of h_t .

In deriving expressions for the fundamental price-dividend ratio, I follow Ang and Liu (2004).

1.B.1 in the appendix verifies that the fundamental price-dividend ratio can be written as

$$FD_t = \sum_{i=1}^{\infty} \exp(a_i + b_{d,i}\Delta d_t + b_{r,i}r_t^e + c_i h_t), \quad (1.12)$$

where the coefficients obey the recursions

$$a_{i+1} = a_i + (b_{d,i} + 1)\mu_d(1 - \varphi_d) + b_{r,i}\mu_r(1 - \varphi_r) + c_i\tau(1 - \beta) - 0.5 \log(1 - 2c_i\alpha(1 - \beta)) \quad (1.13)$$

$$b_{d,i+1} = (b_{d,i} + 1)\varphi_d \quad (1.14)$$

$$b_{r,i+1} = b_{r,i}\varphi_r - 1 \quad (1.15)$$

$$c_{i+1} = (0.5b_{d,i} + 1)^2\sigma_d^2 + 0.5b_{r,i}^2\sigma_r^2 + c_i\beta \quad (1.16)$$

with initial conditions $a_1 = \mu_d(1 - \varphi_d)$, $b_{d,1} = \varphi_d$, $b_{r,1} = -1$ and $c_1 = 0.5\sigma_d^2$. Conditions for the existence of (1.12) are derived in 1.B.2 and the strategy for approximating the infinite recursion is explained in 1.B.3 in the appendix.

1.4 Estimation

Estimation is not straightforward for at least three reasons: first, the discount rate is unobserved, precluding direct estimation of the transition equations; second, the measurement equation (1.12) is nonlinear, so Kalman filtering as in Van Binsbergen and Kojien (2010) is not feasible; third, dividends are highly seasonal and their non-seasonal component needs to be inferred from yearly aggregates. To overcome these complications, I use the simulated score method (Gallant and Tauchen 1998). First, an auxiliary model or score generator is fitted to the data, yielding auxiliary parameter estimates $\hat{\eta}$. Second, estimates of the present value model parameters, $\hat{\theta}$, are chosen to minimize a quadratic form of the first order conditions of the score generator at $\hat{\eta}$ when computed on simulated data.

1.4.1 Data

The monthly realized measure RV_t is computed from daily ex-dividend returns of the value-weighted New York Stock Exchange (NYSE) index from 1926 to 2016 as obtained from the Center for Research in Security Prices (CRSP). All further data on returns and dividends are taken from the annual dataset for the same index and time period. Inflation data are taken from Federal Reserve Economic Data (FRED). For details, see 1.C.1 in the appendix.

1.4.2 Score generator

I next present the score generator. Realized variance RV_t is described by an autoregressive-moving-average (ARMA) type observation driven model

$$\begin{aligned} g_t &= \eta_0 RV_{t-1} + \eta_1 g_{t-1} \\ RV_t &= \eta_2 + g_t + \eta_3 v_t; \quad v_t \sim \mathcal{N}(0, 1). \end{aligned} \tag{1.17}$$

The model closely resembles (1.10) and (1.11), with the difference that, in (1.17), the latent variable is observation driven. (1.17) can be seen as a linear Realized GARCH (Hansen, Huang, and Shek 2012) with a measurement equation for RV_t only.

The second part of the score generator is an annual vector autoregression (VAR) in returns and log price-dividend ratios, as in a standard return forecasting VAR (Campbell 1991)

$$\begin{pmatrix} r_t^a \\ pd_t^a \end{pmatrix} = \begin{pmatrix} \eta_4 \\ \eta_5 \end{pmatrix} + \begin{pmatrix} \eta_7 & \eta_8 \\ \eta_9 & \eta_{10} \end{pmatrix} \begin{pmatrix} r_{t-12}^a \\ pd_{t-12}^a \end{pmatrix} + \begin{pmatrix} u_{1,t} \\ u_{2,t} \end{pmatrix}, \quad \begin{pmatrix} u_{1,t} \\ u_{2,t} \end{pmatrix} \sim \mathcal{N} \left(\mathbf{0}, \begin{pmatrix} \eta_{11} & \eta_{12} \\ \eta_{12} & \eta_{13} \end{pmatrix} \right), \tag{1.18}$$

where $t \in \{12, 24, \dots\}$, r_t^a is the cumulative return over the year and pd_t^a is the ratio of the price at t with respect to the dividends over the previous year. When computing yearly dividend measures from monthly simulated data, I follow the construction of the CRSP in assuming that

dividends are reinvested over the year (Chen 2009). For all formulas for transformation and aggregation of model output, see 1.C.2 in the appendix. For the expressions of the scores, see 1.C.3 in the appendix.

1.4.3 Simulated Score method

In the estimation, I first obtain estimates of the parameters of the auxiliary models (1.17) and (1.18), $\boldsymbol{\eta}_1 = (\eta_0, \eta_1, \eta_2, \eta_3)'$ and $\boldsymbol{\eta}_2 = (\eta_4, \eta_5, \dots, \eta_{13})'$. Afterwards, estimation proceeds in two stages. In the first stage, the volatility parameters $\boldsymbol{\theta}_1 = (\alpha, \beta, \gamma, \sigma_{rv})'$ are determined. For that purpose, I simulate from (1.10) and (1.11) and compute the scores with respect to (1.17)

$$\mathbf{m}_1(\mathbf{RV}^*, \hat{\boldsymbol{\eta}}_1, \boldsymbol{\theta}_1) = \frac{\sqrt{n}}{B} \sum_{t=1}^{12B} \mathbf{s}_t(RV_t^* | RV_{t-1}^*, \hat{\boldsymbol{\eta}}_1). \quad (1.19)$$

\mathbf{RV}^* is the stacked sequence of $\{RV_t\}_{t=1}^{12B}$, B is the length of the simulation in years and

$$\mathbf{s}_t(RV_t^* | RV_{t-1}^*, \hat{\boldsymbol{\eta}}_1) = \frac{\partial \ell(RV_t^* | RV_{t-1}^*, \hat{\boldsymbol{\eta}}_1)}{\partial \boldsymbol{\eta}_1},$$

with $\ell(RV_t^* | RV_{t-1}^*, \hat{\boldsymbol{\eta}}_1)$ being the conditional quasi-log-likelihood of RV_t^* , evaluated at the estimates of the auxiliary model. $\hat{\boldsymbol{\theta}}_1$ is then chosen to minimize the quadratic form

$$Q_n(\boldsymbol{\theta}_1) = \mathbf{m}_1(\mathbf{RV}^*, \hat{\boldsymbol{\eta}}_1, \boldsymbol{\theta}_1)' \mathbf{W}_1 \mathbf{m}_1(\mathbf{RV}^*, \hat{\boldsymbol{\eta}}_1, \boldsymbol{\theta}_1).$$

To simplify notation, dependence of the criterion on \mathbf{RV}^* and $\hat{\boldsymbol{\eta}}_1$ is omitted. The weighting matrix \mathbf{W}_1 is the inverse of an estimate of the long-run variance of $\mathbf{s}_t(RV_t^* | RV_{t-1}^*, \hat{\boldsymbol{\eta}}_1)$. In the second stage, I simulate from (1.8), (1.9), (1.10), and (1.12) and compute the scores with respect

to (1.18)

$$\mathbf{m}_2(\mathbf{Y}^*, \hat{\boldsymbol{\eta}}_2, \boldsymbol{\theta}_2, \hat{\boldsymbol{\theta}}_1) = \frac{\sqrt{n}}{B} \sum_{t \in \{12, 24, \dots, 12B\}} \mathbf{s}_t(\mathbf{y}_t^* | \mathbf{Y}_{t-1}^*, \hat{\boldsymbol{\eta}}_2), \quad (1.20)$$

where \mathbf{Y}^* are the simulated data and

$$\mathbf{s}_t(\mathbf{y}_t^* | \mathbf{Y}_{t-1}^*, \hat{\boldsymbol{\eta}}_2) = \frac{\partial \ell(\mathbf{y}_t | \mathbf{Y}_{t-1}; \hat{\boldsymbol{\eta}}_2)}{\partial \boldsymbol{\eta}_2},$$

which is the score of the conditional quasi-log-likelihood of \mathbf{y}_t^* , evaluated at $\hat{\boldsymbol{\eta}}_2$. Estimates of the remaining parameters of the data generating process (DGP) $\boldsymbol{\theta}_2 = (\mu_d, \mu_r, \varphi_d, \varphi_r, \sigma_d, \sigma_r)'$ are chosen to minimize

$$Q_n(\boldsymbol{\theta}_2) = \mathbf{m}_2(\mathbf{Y}^*, \hat{\boldsymbol{\eta}}_2, \boldsymbol{\theta}_2, \hat{\boldsymbol{\theta}}_1)' \mathbf{W}_2 \mathbf{m}_2(\mathbf{Y}^*, \hat{\boldsymbol{\eta}}_2, \boldsymbol{\theta}_2, \hat{\boldsymbol{\theta}}_1).$$

\mathbf{W}_2 is the inverse of an estimate of the long-run variance of $\mathbf{s}_t(\mathbf{y}_t^* | \mathbf{Y}_{t-1}^*, \hat{\boldsymbol{\eta}}_2)$. Standard errors for the two-stage procedure are derived in 1.C.4 and computational aspects of the estimation are discussed in 1.E.1 in the appendix.

1.4.4 Estimates

The estimates are:

$$\Delta d_{t+1} = \underset{(0.0009)}{0.0022} + \underset{(0.0316)}{0.9552}(\Delta d_t - \underset{(0.0009)}{0.0022}) + \underset{(0.0544)}{0.0219} \sqrt{h_t} \epsilon_{d,t+1}$$

$$r_{t+1}^e = \underset{(0.0012)}{0.0071} + \underset{(0.0029)}{0.9970}(r_t^e - \underset{(0.0012)}{0.0071}) + \underset{(0.0058)}{0.0023} \sqrt{h_t} \epsilon_{r,t+1}$$

$$h_{t+1} = \underset{(0.0023)}{0.0080}(1 - \underset{(0.0825)}{0.7442})\epsilon_{h,t+1}^2 + \underset{(0.0825)}{0.7442}h_t$$

$$RV_{t+1} = \underset{(0.0021)}{-0.0056} + h_{t+1} + \underset{(0.0008)}{0.0024}\epsilon_{rv,t+1}.$$

The results show that the discount rate is highly persistent and less volatile than dividend growth. Dividend growth is also persistent, but much less so than the discount rate. Time-varying volatility is moderately persistent, in line with the behavior of monthly realized variance. The model fits the score generator well: the null hypothesis of overidentifying restrictions in the second stage is not rejected by the Sargan-Hansen test, with a p -value of 0.45. The first stage parameters are exactly identified.

The closest quantitative comparison to these estimates is Van Binsbergen and Koijen (2010), who estimate a log-linearized, homoscedastic version of the present value relationship using a shorter sample period. The estimated annualized expected return here is 8.5%, statistically indistinguishable to the 8.6% that they obtain in the case of market-reinvested dividends. The annualized dividend growth rate here is 2.7%, compared to 6.0% in their paper. The likely reason for the latter discrepancy is that, in my model, reinvested dividends accumulate over the year at the realized market return.⁴

1.5 Test specifications

I now describe the procedures implemented to test the auxiliary hypotheses (1.3), (1.6), and (1.7).

4. Van Binsbergen and Koijen (2010) model dividend reinvestment through positive correlation between dividend growth and the *unexpected* component of the return. In contrast, the reinvestment procedure as detailed in 1.C.2 in the appendix reinvests dividends at the *realized return*, which includes both the expected return and the innovation in the return. Because the expected return is positive, for a given dividend growth process, reinvested dividends over the year are higher with the reinvestment strategy that I use.

1.5.1 ADF test

Diba and Grossman (1988) propose to test H_0^{ex} versus H_1^{ex} in (1.3) by testing for $\beta^{\text{ADF}} > 0$ in the augmented Dickey-Fuller regression (ADF)

$$\Delta P_t = \alpha^{\text{ADF}} + \beta^{\text{ADF}} P_{t-1} + \sum_{i=1}^k \phi_i^{\text{ADF}} \Delta P_{t-i} + \epsilon_t^{\text{ADF}}, \quad \epsilon_t^{\text{ADF}} \sim \text{i.i.d.} \quad (1.21)$$

(1.21) includes an intercept but no time trend, as recommended by Phillips, Shi, and Yu (2014). Following the results by Phillips, Shi, and Yu (2015b), I set $k = 0$. In the appendix, I also report results for picking the specification with the lowest Bayesian information criterion (BIC) or by the rule proposed by Schwert (1989) and for running (1.21) on log prices. Critical values are interpolated from Table 4.2 in Banerjee et al. (1993).

1.5.2 SADF test

Arguably, the most prominent test for explosive price growth is the sup ADF (SADF) test (Phillips, Wu, and Yu 2011). The reason is that it has been found to have power against periodic explosive episodes created by a bubble that crashes from time to time. On the other hand, the simple test (1.21) has low power against those episodes (Evans 1991). The statistic of the SADF test is the supremum over t -statistics of estimate of β^{ADF} in (1.21) for a fraction of the sample $\tau = \lfloor rn \rfloor$, with $r \in [r_0, 1]$

$$\text{SADF} = \sup_{r \in [r_0, 1]} \text{ADF}_r.$$

I use the same range of specifications as for the ADF test. The starting value of the recursion is set to the closest integer of $0.01n + 1.8\sqrt{n}$, following Phillips, Shi, and Yu (2015a). Critical values are interpolated from Table 1 in their paper. I use recursive formulas to increase computational

speed, see 1.E.2 in the appendix.

1.5.3 Cointegration test

The cointegration test employs the two-step procedure by Engle and Granger (1987). In the first stage, estimate

$$P_t = \alpha^{\text{CI}} + \beta^{\text{CI}} D_t + u_t, \quad u_t \sim \text{i.i.d.}$$

by OLS and obtain the residuals \hat{u}_t . Next, run the ADF regression without drift on the residual,

$$\Delta \hat{u}_t = \beta^{\text{CI}} \hat{u}_t + \sum_{i=1}^k \phi_i^{\text{CI}} \Delta \hat{u}_{t-i} + \epsilon_t^{\text{CI}}, \quad \epsilon_t^{\text{CI}} \sim \text{i.i.d.} \quad (1.22)$$

A test of (1.6) takes the form of testing $\beta^{\text{CI}} = 0$ against the alternative $\beta^{\text{CI}} < 0$. Note that (1.22) does not contain a drift term, as implied by (1.5). Critical values are, therefore, the same ones as for an ADF regression without drift.⁵ Critical values are computed from the response surface estimates of MacKinnon (2010).

1.5.4 Variance ratio test

Following Cochrane (2011), I compute covariances from the parametric specification

$$\begin{pmatrix} r_t \\ \Delta d_t \\ PD_t \end{pmatrix} = \begin{pmatrix} a_r \\ a_d \\ a_{pd} \end{pmatrix} + \begin{pmatrix} b_r \\ b_d \\ b_{pd} \end{pmatrix} PD_{t-1} + \begin{pmatrix} u_{r,t} \\ u_{d,t} \\ u_{pd,t} \end{pmatrix}, \quad \begin{pmatrix} u_{r,t} \\ u_{d,t} \\ u_{pd,t} \end{pmatrix} \sim \mathcal{N}(\mathbf{0}, \Sigma^{pd}).$$

The sums of regression coefficients in (1.7) are given in closed form as

$$\frac{1}{1 - \Omega} \sum_{j=1}^{\infty} (\Omega^j \beta_j^d - \Omega^j \beta_j^r) = \frac{(b_d - b_r)\Omega}{(1 - \Omega b_{pd})(1 - \Omega)}.$$

5. For a detailed discussion of the specification of (1.22), see Timmermann (1995).

Cochrane (1992, 2011) does not provide critical values. Therefore, I use the variance bound test only in the analysis under both the fundamental and the bubble hypothesis, in which I set the critical values.

1.6 Performance under the fundamental hypothesis

In this section, I evaluate how well the auxiliary hypotheses approximate the fundamental hypothesis. I apply the bubble tests described in Section 1.5 to fundamental prices generated by the estimated present value model, using conventional critical values.

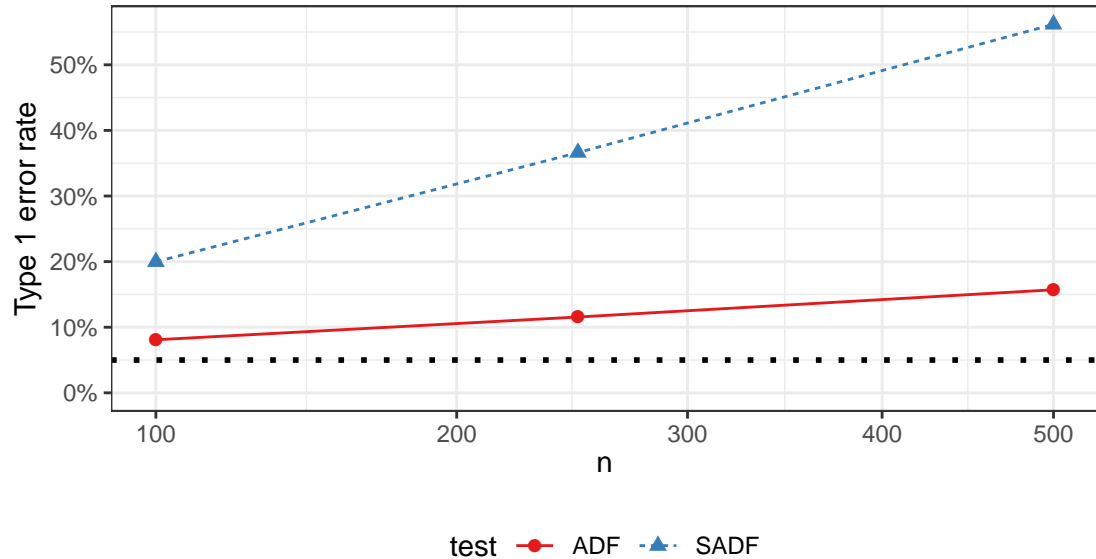
1.6.1 Simulations

To compute rejection frequencies, I simulate 30,000 sequences of different lengths from the model and apply the tests at their respective 5% level critical values. To mimic the seasonality in dividend data, observed dividends are computed as a twelve-month moving average of the dividends in model output. I do not use the variance ratio test here because there are no critical values for it.

Tests for explosive price growth

Figure 1.1 reports rejection frequencies of the tests for explosive price growth under the fundamental hypothesis, for different sample sizes. These tests should accept, because their null hypothesis corresponds to the fundamental hypothesis. The rejection frequencies, therefore, constitute Type 1 error rates. The horizontal line denotes the nominal level of 5%. Tests for explosive price growth reject far more frequently than their nominal level implies. Importantly, Type 1 error rates increase in sample size.

Figure 1.1. Type 1 error rates of tests for explosive price growth when prices are generated by the estimated present value model. The horizontal axis shows sample size in months, n . The horizontal dotted line denotes the nominal size of 5%.



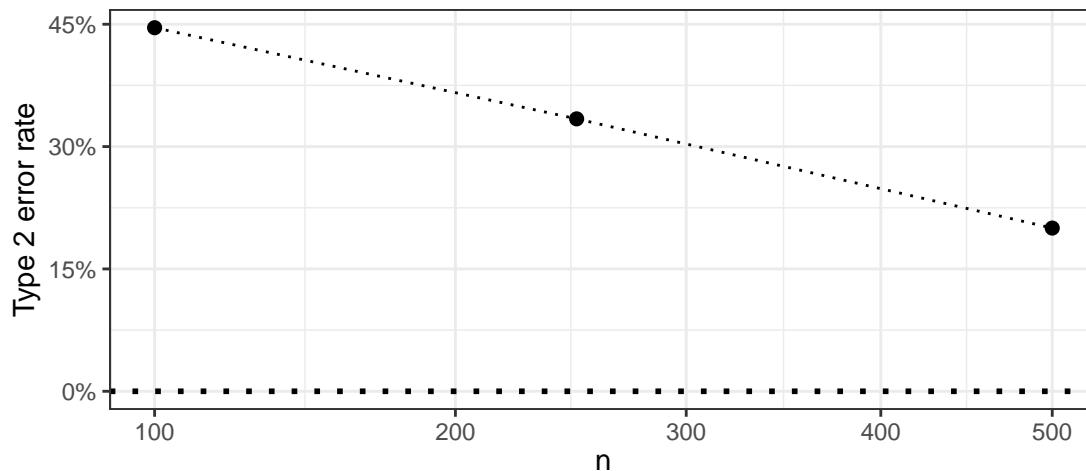
Cointegration test

The cointegration test's null hypothesis corresponds to the bubble hypothesis. It should, therefore, reject under the fundamental hypothesis, contrary to the tests for explosive price growth. Figure 1.2 shows the proportion of simulations in which the cointegration test fails to reject, i.e. the Type 2 error rate. Clearly, the test has low power against the bubble hypothesis.

1.6.2 Drivers of over- and underrejection

To check which features of the DGP impair inference, I rerun the simulations under restricted parameterizations and the corresponding pricing coefficients (1.13) through (1.16).

Figure 1.2. Type 2 error rate of the cointegration test when data are generated by the estimated present value model, as a function of sample size in months, n . The horizontal dotted line denotes the desired Type 2 rate of zero percent.



Tests for explosive price growth

Figure 1.3 plots rejection frequencies of the tests for explosive price growth under different configurations. I focus on two restrictions: setting the discount rate to a constant, that is, imposing $\varphi_r = \sigma_r = 0$ in (1.9); and letting dividend growth follow a white noise process, that is, $\mu_d = \varphi_d = 0$ in (1.8). The top left pane corresponds to Type 1 error rates at the estimated parameter values as reported in Figure 1.1. The panes on the right show Type 1 rates for white noise dividend growth and the bottom panes restrict the discount rate to a constant. Oversizing is most strongly associated with dividend growth not being white noise. Yet, even when dividend growth is white noise, a time-varying discount rate leads to inflated Type 1 error rates for the SADF test. In order to get Type 1 error rates close to nominal size, we need both the discount rate to be constant and dividend growth to be white noise, in line with the theoretical results from Section 1.2. I also simulated the DGP with homoscedastic innovations, ran the test on log-transformed variables and with different lag length selection procedures for k . I report results

for these configurations in 1.D in the appendix.

Cointegration tests

Figure 1.4 displays Type 2 error rates of the cointegration test for the same configurations as in the previous section. Underrejections for larger sample sizes can be mostly attributed to the time-varying discount rate, which conforms with the findings by Timmermann (1995). For the case of constant discount rate and white noise dividend growth, variation in price-dividend ratios is entirely driven by variation in h_t and the seasonal adjustment for dividends, such that the null hypothesis of no cointegration between prices and dividends is almost never rejected. In 1.D in the appendix, I report results for a variety of further configurations.

The results in this section have shown that, under the general fundamental price process, tests for explosive price growth and the cointegration test do not allow for correct inference. With regard to the tests for explosive price growth, overrejections are mostly due to dividend growth dynamics. The cointegration test underrejects because of discount rate dynamics. In the next section, I evaluate the tests under both the null and the alternative hypothesis in order to see if they can still distinguish between fundamental behavior and bubbles.

1.7 Performance under the fundamental and the bubble hypotheses

The simulations under the fundamental hypothesis have shown that inference from bubble tests at conventional critical values is likely misleading. Time variation in discount rates and in dividend growth rates not only contradicts the simplifying assumptions that bubble tests make, but also has a quantitative impact on the distributions of test statistics.

In this section, I investigate whether the test statistics under consideration still provide

Figure 1.3. Type 1 error rates of tests for explosive price growth when prices are generated by the estimated present value model. The horizontal axis shows sample size in months, n . The horizontal dotted line denotes the nominal size of 5%. The top left panel shows the results for the estimated present value process and corresponds to Figure 1.1. “dividend growth white noise” or “discount rate fixed” denote results for the setting where, respectively, $\mu_d = \varphi_d = 0$ or $\varphi_r = \sigma_r = 0$, while leaving other parameters fixed at their estimates.

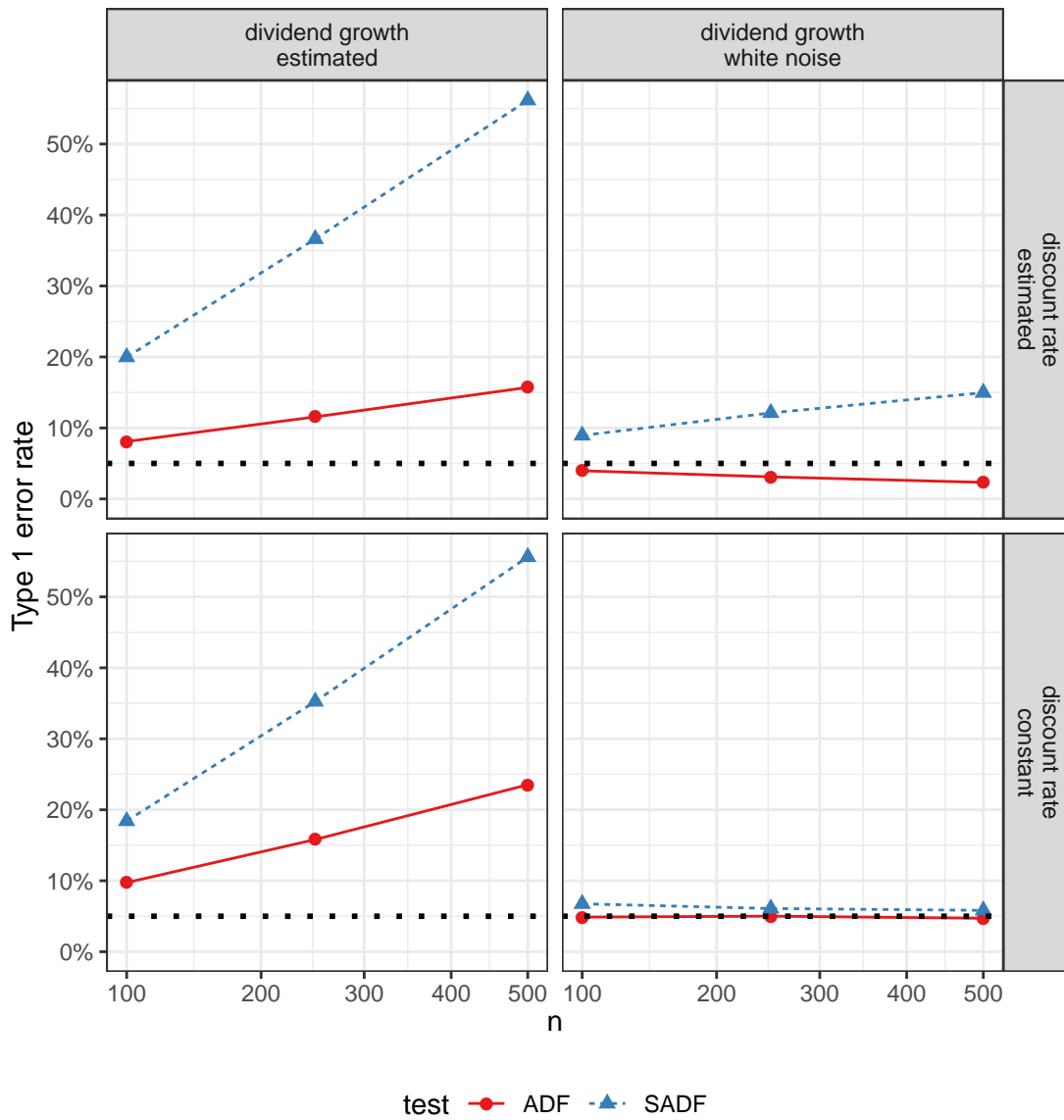
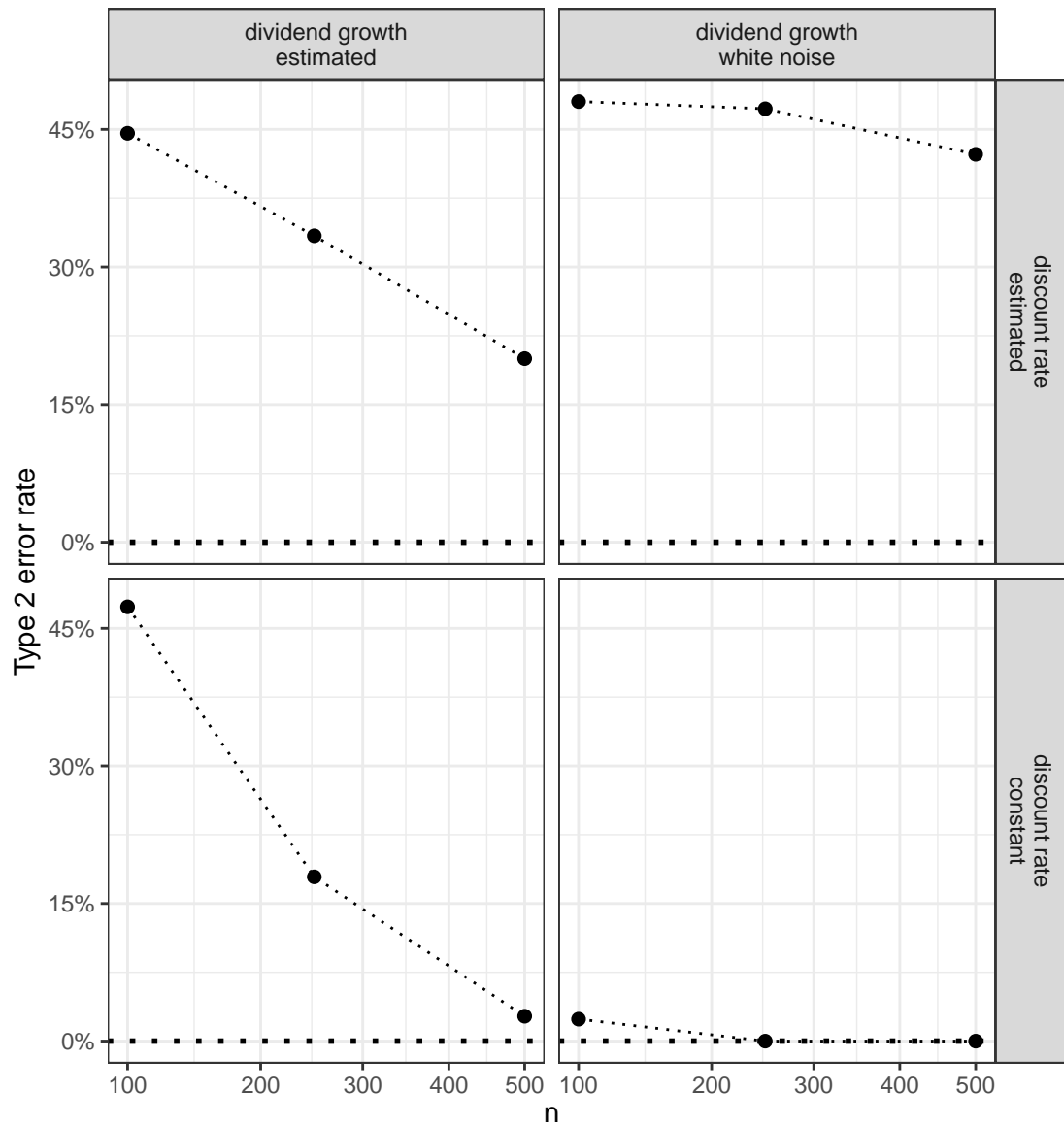


Figure 1.4. Type 2 error rate of the cointegration test when data are generated by the estimated present value model, as a function of sample size in months, n . The horizontal dotted line denotes the optimal Type 2 rate. The top left panel shows the results for the estimated present value process and corresponds to Figure 1.2. “dividend growth white noise” or “discount rate fixed” denote results for the setting where, respectively, $\mu_d = \varphi_d = 0$ or $\varphi_r = \sigma_r = 0$, while leaving other parameters fixed at their estimates.



information on bubbles, even when fundamental prices are generated from the flexible model. Heuristically, this seems to be the case. First, regarding explosive price growth tests, we have from (1.4) that the growth rate of fundamental price is

$$a_t^F = \left(1 - \frac{\mathbb{E}_t D_{t+1}}{F_t}\right) R_t^e < R_t^e = a_t^B.$$

Fundamental price grows at a smaller rate than the bubble, so too large a price growth statistic may still be indicative of a bubble. Second, for cointegration tests, although the cointegrating relationship (1.5) does not hold under time-varying discount rates, fundamental price-dividend ratios are stationary, contrary to bubbly price-dividend ratios. Examining the stationarity of residuals in a regression of prices on dividends could, therefore, still be worthwhile. Finally, the variance ratio test takes time variation in discount rates and dividend growth into account by construction.

1.7.1 Bubble process

To evaluate power, a process for the bubble needs to be specified. The bubble process is

$$B_{t+1} = \theta_{t+1} \pi^{-1} (R_t^e B_t - \underline{B}) + \underline{B}, \tag{1.23}$$

where θ_{t+1} is a Bernoulli random variable with parameter $\pi \in (0, 1]$ and $\underline{B} > 0$. B_t fulfills (1.2) as well as the additional requirement that a bubble should be prone to periodic collapses (Blanchard and Watson 1982). I set the initial value, B_0 , equal to \underline{B} .

1.7.2 Simulations

I fix the level of each test at 5% by adjusting the critical value. Power is computed from Type 2 error rates at the corresponding critical value. Computing Type 2 error rates requires parameter values of the bubble process. I deal with this dependence on nuisance parameters by simulating the DGP for a range of values for \underline{B} and π . Specifically, I let $\pi \in \{0.50, 0.70, 0.90, 0.95, 0.99\}$ and $\underline{B} \in \{0.01F_0, 0.25F_0, 0.50F_0, 0.75F_0, F_0\}$, where F_0 is the initial value of the fundamental price. Regarding sample size, I again consider $n \in \{100, 250, 500\}$. To ease comparison between the tests, the cointegration test is reformulated such that its null hypothesis corresponds to the fundamental hypothesis. The null is accepted when the cointegration statistic is below the 5% critical value and rejected otherwise.

Figure 1.5 shows Type 2 error rates. The horizontal axis represents π . The horizontal dimension of panes corresponds to different values of \underline{B} and the vertical dimension of panes displays sample size. To interpret the figure, note that a Type 2 error rate of 95% corresponds to the test statistic being of no value in distinguishing between the fundamental and the bubble hypothesis whereas a lower value shows that the test has power. We can discern the following patterns: in general, Type 2 error rates decrease as sample size increases or as \underline{B} , which governs the size of the bubble component, increases; second, all tests except the SADF test and the VD test have power smaller than level for some configurations and are, therefore, biased; third, no test uniformly dominates the others. Still, Type 2 error rates are mostly lower for the SADF test, except when the probability of no collapse is high and sample size is low.

A more concise measure of relative performance of the test is maximum regret. Regret is the difference between the Type 2 error rate of a test and the Type 2 error rate of the infeasible, most powerful test. Figure 1.6 displays maximum regret of the tests, the highest regret over values of \underline{B} and π , as a function of sample size. All tests except the SADF test show high maximum

regret.

The results of this section imply that bubble tests can be useful in distinguishing between the null and the alternative hypotheses, even when fundamental prices are generated by a flexible model. The SADF test, although not uniformly the most powerful, is the most promising among the tests considered.

1.8 Discussion

At least since the work by Shiller (1981), economists have tried to formally test the hypothesis that stock market prices are equal to the present value of dividends. Most tests have focused on running auxiliary tests that are less demanding than estimating the present value of dividends directly. Tests for periodically explosive growth appear to be the standard bubble tests in empirical work now (e.g., Brunnermeier, Rother, and Schnabel 2019). This is despite the discouraging findings by Giglio, Maggiori, and Stroebel (2016), who document that tests for periodically explosive growth spuriously detect bubbles in assets that fulfill the fundamental hypothesis, according to a model-free test.

In this chapter, I have shown that, under fairly standard assumptions about the data-generating process, no auxiliary test provides correct inference using standard critical values. Inference of tests for periodically explosive growth crucially depends on dividend growth being white noise. I also find that inference of tests for cointegration relies on the discount rate being constant, replicating a previous finding by Timmermann (1995). Comparing between bubble detection tests, I find that tests for periodically explosive growth provide the best size-power tradeoff in the case where the bubble process exhibits periodic crashes.

In order to make use of the favorable size-power properties of test for periodically explosive growth, one needs to control size. This is difficult, because, as I document, rejection frequencies

Figure 1.5. Type 2 error of bubble detection tests when controlling size. Fundamental value is generated by the estimated present value model and the bubble follows (1.23). The horizontal axis denotes the probability of no collapse, π , and panels correspond to different initial sizes of the bubble or different sample sizes. The dotted horizontal line depicts the Type 2 error rate of a test that has the same rejection frequency under the fundamental and the bubble hypothesis.

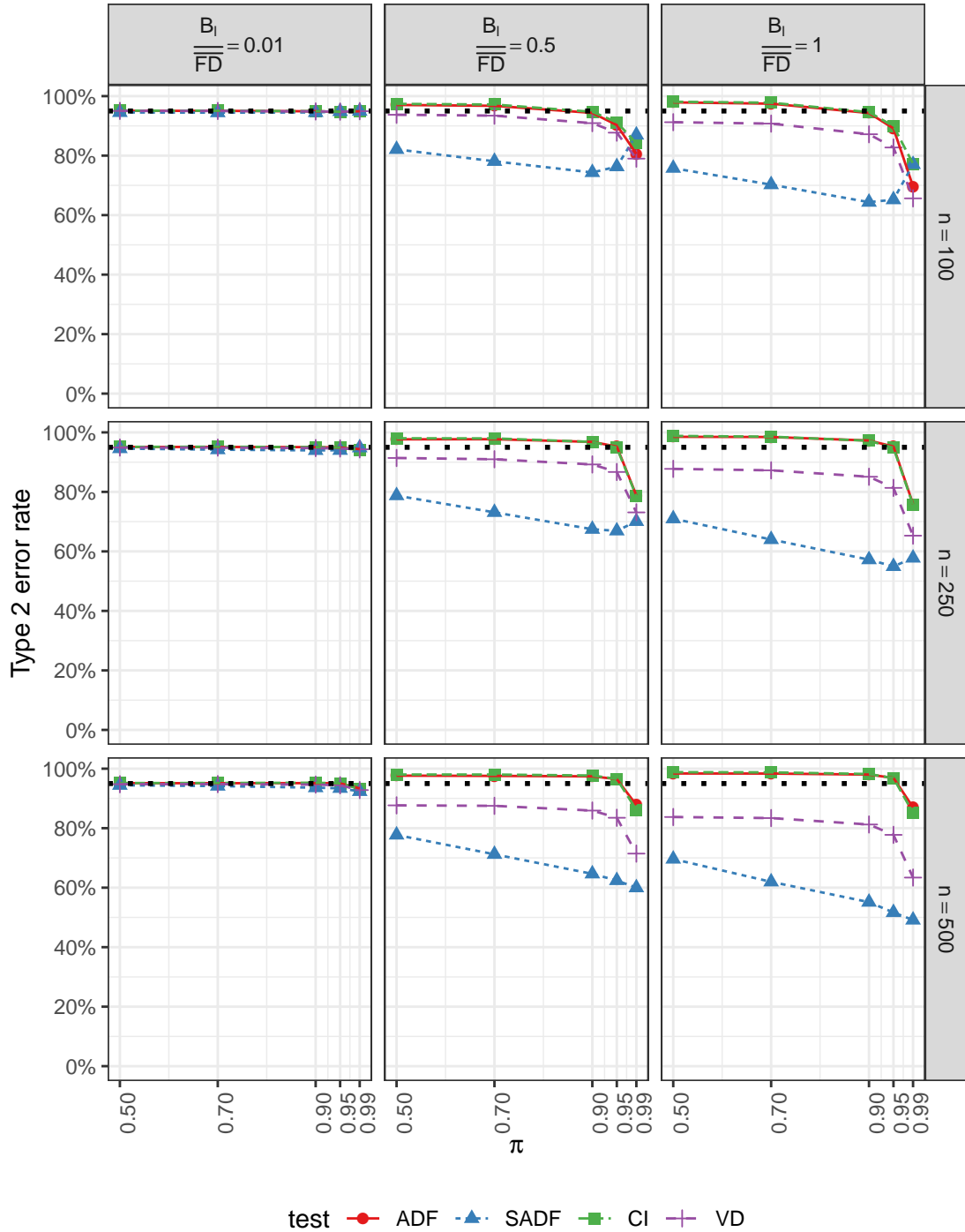
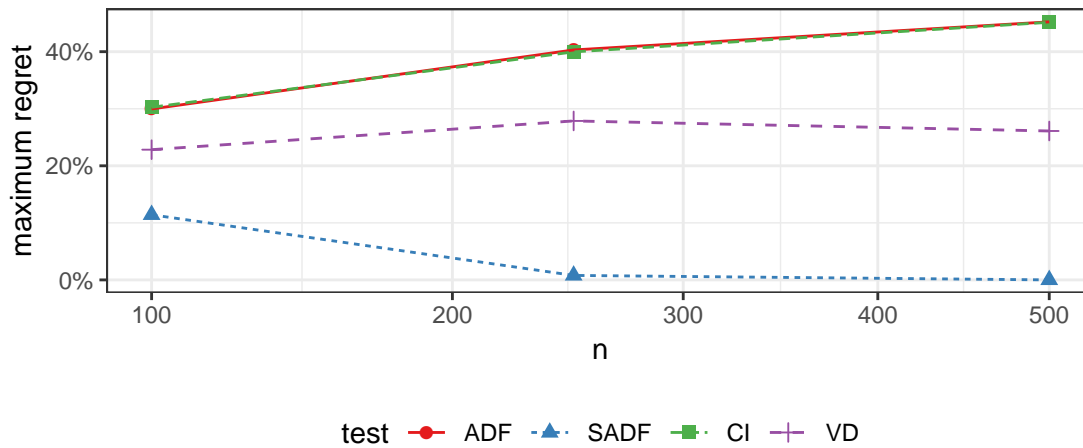


Figure 1.6. Maximum regret. Regret is the difference between the Type 2 error rate of a test and of the most powerful test for a given configuration. Maximum regret is the maximum over configurations of the bubble process (1.23). The horizontal axis denotes sample size.



depend on the configuration of the present value model. A researcher who tests for bubbles using a test for periodically explosive growth at conventional critical values should acknowledge that this is a test of a present value model with constant discount rate and white noise dividend growth and that a rejection might be evidence for misspecification of the simple model, rather than the finding of a bubble. In order to test a more general present value model, the researcher will need to estimate it. Recently, much progress on the estimation of asset pricing models has been made (e.g., Schorfheide, Song, and Yaron 2018).

Appendix to Chapter 1

1.A Derivations related to the auxiliary hypotheses

1.A.1 Fundamental price growth in linearized model

Linearize (1.1) inside the expectation operator around the mean expected return \bar{R}^e

$$F_t \approx \mathbb{E}_t \left[\sum_{i=1}^{\infty} (\bar{R}^e)^{-i} D_{t+i} + \sum_{i=1}^{\infty} \frac{\partial F_t}{\partial R_{t+i}^e} \Big|_{R_{t+i}^e = \bar{R}^e} (R_{t+i}^e - \bar{R}^e) \right].$$

Using that $R_{t+i}^e - \bar{R}^e = \varphi(R_t^e - \bar{R}^e) + \epsilon_{t+i}^r$ with $\mathbb{E}_t[\epsilon_{t+i}^r] = 0$ and that $\mathbb{E}_t[D_{t+i}] = G^i D_t$, D_t independent of R_t^e and $G < R_t^e$

$$\frac{\partial F_t}{\partial R_{t+i}^e} \Big|_{R_{t+i}^e = \bar{R}^e} = - \frac{D_t}{1 - \frac{G}{\bar{R}^e}} \frac{(G)^{i+1}}{(\bar{R}^e)^{i+2}},$$

so the approximation becomes

$$\begin{aligned} F_t &\approx \sum_{i=1}^{\infty} \left(\frac{G}{\bar{R}^e} \right)^{-i} D_t - \frac{D_t}{1 - \frac{G}{\bar{R}^e}} \frac{G}{(\bar{R}^e)^2} \sum_{i=1}^{\infty} \frac{(G)^i}{(\bar{R}^e)^i} \varphi^i (R_t^e - \bar{R}^e) \\ &= \frac{D_t}{1 - \frac{G}{\bar{R}^e}} \frac{G}{\bar{R}^e} \left(1 - \varphi \frac{G}{(\bar{R}^e)^2} \frac{1}{1 - \varphi \frac{G}{\bar{R}^e}} (R_t^e - \bar{R}^e) \right). \end{aligned}$$

The growth rate of fundamental price is, therefore,

$$a_t^F = \frac{\mathbb{E}_t F_{t+1}}{F_t} \approx \frac{G(\gamma - \varphi(R_t - \bar{R}^e))}{\gamma - (R_t - \bar{R}^e)}$$

with $\gamma = \frac{1}{\varphi} \frac{(\bar{R}^e)^2}{G} \left(1 - \varphi \frac{G}{\bar{R}^e}\right)$.

1.A.2 Cointegration

If we assume that $R_t^e = R > 1$ for all t we can rewrite (1.1) as

$$\begin{aligned} F_t &= \sum_{i=1}^{\infty} R^{-i} \mathbb{E}_t [D_{t+i}] \\ &= \sum_{i=1}^{\infty} R^{-i} D_t + \sum_{i=1}^{\infty} \mathbb{E}_t \left[R^{-i} \sum_{j=1}^i \Delta D_{t+j} \right] \\ &= \sum_{i=1}^{\infty} R^{-i} D_t + \sum_{i=1}^{\infty} \mathbb{E}_t \left[\sum_{j=1}^{\infty} R^{-j} \Delta D_{t+i} \right] \\ &= \frac{R^{-1}}{1 - R^{-1}} D_t + \frac{1}{1 - R^{-1}} \sum_{i=1}^{\infty} \mathbb{E}_t [R^{-i} \Delta D_{t+i}]. \end{aligned}$$

Subtracting $\frac{R^{-1}}{1 - R^{-1}} D_t$ on both sides yields (1.5).

1.A.3 Variance bound test in logs

Cochrane (2011) uses the approximate present value relationship derived using the Campbell and Shiller (1988) log-linear approximation of the return

$$pd_t = \mathbb{E}_t \left[\sum_{j=1}^{\infty} \rho^{j-1} \Delta d_{t+j} \right] - \mathbb{E}_t \left[\sum_{j=1}^{\infty} \rho^{j-1} r_{t+j} \right],$$

with $\rho = \overline{PD}/(1 + \overline{PD})$, \overline{PD} being the value around which one linearizes and imposing the transversality condition $\lim_{j \rightarrow \infty} \rho^j pd_{t+j} = 0$. Multiplying each side by $pd_t - \overline{pd}$ where $\overline{pd} = \mathbb{E}[pd]$

and take unconditional expectations

$$\text{Var}(pd_t) = \sum_{j=1}^{\infty} \text{Cov}(\rho^{j-1} \Delta d_{t+j}, pd_t) - \sum_{j=1}^{\infty} \text{Cov}(\rho^{j-1} r_{t+j}, pd_t).$$

Dividing by $\text{Var}(pd_t)$, we have

$$1 = \sum_{j=1}^{\infty} \rho^{j-1} \beta_j^d - \sum_{j=1}^{\infty} \rho^{j-1} \beta_j^r,$$

where β_j^i is the coefficient of a univariate regression of the j 'th lead of variable $i \in \{d, r\}$ on pd_t .

If the transversality condition fails to hold, we have

$$pd_t = \mathbb{E}_t \left[\sum_{j=1}^{\infty} \rho^{j-1} \Delta d_{t+j} \right] - \mathbb{E}_t \left[\sum_{j=1}^{\infty} \rho^{j-1} r_{t+j} \right] + b_t,$$

where b_t fulfills $\mathbb{E}_t[b_{t+1}] = \rho^{-1} b_t$. Repeating the same steps as in the main text

$$\text{Var}(pd_t) = \sum_{j=1}^{\infty} \text{Cov}(\rho^{j-1} \Delta d_{t+j}, pd_t) - \sum_{j=1}^{\infty} \text{Cov}(\rho^{j-1} r_{t+j}, pd_t) + \text{Cov}(pd_t, b_t).$$

Maintaining that $\text{Cov}(pd_t, b_t) > 0$, a test of whether the transversality condition in this approx-

imate present value model holds is given by

$$H_0^{\text{vd}} : \sum_{j=1}^{\infty} \rho^{j-1} \beta_j^d - \sum_{j=1}^{\infty} \rho^{j-1} \beta_j^r = 1 \quad \text{vs.} \quad H_1^{\text{vd}} : \sum_{j=1}^{\infty} \rho^{j-1} \beta_j^d - \sum_{j=1}^{\infty} \rho^{j-1} \beta_j^r < 1. \quad (1.24)$$

1.B Present value model

1.B.1 Derivation

For the purpose of the alternative parameterizations considered in Section 1.D, it is convenient to work with a more general variance process:

$$h_{t+1} = (\tau + \alpha \epsilon_{h,t+1}^2)(1 - \beta) + \beta h_t. \quad (1.25)$$

I now derive an expression for the fundamental price-dividend ratio. Rearranging (1.1) yields

$$FD_t = \sum_{i=1}^{\infty} \mathbb{E}_t \left[\exp \left(\sum_{j=1}^i (\Delta d_{t+j} - r_{t+j-1}^e) \right) \right]. \quad (1.26)$$

Denote the period t price of a cash flow i periods ahead as $P_{t,i}^D$. The conjecture is that

$$\frac{P_{t,i}^D}{D_t} = \exp(f_i(\Delta d_t, r_t^e, h_t)) = \exp(a_i + b_{d,i} \Delta d_t + b_{r,i} r_t^e + c_i h_t). \quad (1.27)$$

Verify this conjecture by induction. If (1.27) holds for a particular i , then (1.26) implies that

$$\begin{aligned} \exp(f_{i+1}(\Delta d_t, r_t^e, h_t)) &= \mathbb{E}_t \left[\exp(f_i(\Delta d_{t+1}, r_{t+1}^e, h_{t+1})) \exp(\Delta d_{t+1} - r_t^e) \right] \\ \exp(a_{i+1} + b_{d,i+1} \Delta d_t + b_{r,i+1} r_t^e + c_{i+1} h_t) &= \mathbb{E}_t \left[\exp(a_i + b_{d,i} \Delta d_{t+1} + b_{r,i} r_{t+1}^e + c_i h_{t+1} + \Delta d_{t+1} - r_t^e) \right]. \end{aligned}$$

Substituting the processes:

$$\begin{aligned}
& \mathbb{E}_t \left[\exp \left(a_i + (b_{d,i} + 1)(\mu_d + \varphi_d(\Delta d_t - \mu_d) + \sigma_d \sqrt{h_t} \epsilon_{d,t+1}) \right. \right. \\
& \quad \left. \left. + b_{r,i}(\mu_r + \varphi(r_t^e - \mu_r) + \sigma_r \sqrt{h_t} \epsilon_{r,t+1}) - r_t^e + c_i((\tau + \alpha \epsilon_{h,t+1}^2)(1 - \beta) + \beta h_t) \right) \right] \\
& = \exp(a_i + (b_{d,i} + 1)\mu_d(1 - \varphi_d) + b_{r,i}\mu_r(1 - \varphi_r) + c_i\tau(1 - \beta) + (b_{d,i} + 1)\varphi_d\Delta d_t + (b_{r,i}\varphi_r - 1)r_t^e + c_i\beta h_t) \\
& \quad \mathbb{E}_t \left[\exp \left((b_{d,i} + 1)\sigma_d \sqrt{h_t} \epsilon_{d,t+1} + b_{r,i}\sigma_r \sqrt{h_t} \epsilon_{r,t+1} + c_i\alpha(1 - \beta)\epsilon_{h,t+1}^2 \right) \right].
\end{aligned}$$

To develop the expectation, apply Lemma 1 from Ang and Liu (2004). If $c_i\alpha(1 - \beta) < 0.5$, then

$$\mathbb{E}_t \left[\exp(c_i\alpha(1 - \beta)\epsilon_{h,t+1}^2) \right] = \exp(-0.5 \log(1 - 2c_i\alpha(1 - \beta))).$$

So the whole expression becomes

$$\begin{aligned}
& \exp(a_i + (b_{d,i} + 1)\mu_d(1 - \varphi_d) + b_{r,i}\mu_r(1 - \varphi_r) + c_i\tau(1 - \beta) - 0.5 \log(1 - 2c_i\alpha(1 - \beta)) \\
& \quad + (b_{d,i} + 1)\varphi_d\Delta d_t + (b_{r,i}\varphi_r - 1)r_t^e + ((0.5b_{d,i} + 1)^2\sigma_d^2 + 0.5b_{r,i}^2\sigma_r^2 + c_i\beta)h_t),
\end{aligned}$$

yielding

$$a_{i+1} = a_i + (b_{d,i} + 1)\mu_d(1 - \varphi_d) + b_{r,i}\mu_r(1 - \varphi_r) + c_i\tau(1 - \beta) - 0.5 \log(1 - 2c_i\alpha(1 - \beta))$$

$$b_{d,i+1} = (b_{d,i} + 1)\varphi_d$$

$$b_{r,i+1} = b_{r,i}\varphi_r - 1$$

$$c_{i+1} = (0.5b_{d,i} + 1)^2\sigma_d^2 + 0.5b_{r,i}^2\sigma_r^2 + c_i\beta.$$

The initial condition is

$$\begin{aligned} \exp(a_1 + b_{d,1}\Delta d_t + b_{r,1}r_t^e + c_1h_t) &= \mathbb{E}_t \left[\exp(\mu_d + \varphi_d(\Delta d_t - \mu_d) + \sigma_d\sqrt{h_t}\epsilon_{d,t+1} - r_t^e) \right] \\ &= \exp(\mu_d(1 - \varphi_d) + \varphi_d\Delta d_t + 0.5\sigma_d^2h_t - r_t^e), \end{aligned}$$

yielding

$$a_1 = \mu_d(1 - \varphi_d)$$

$$b_{d,1} = \varphi_d$$

$$b_{r,1} = -1$$

$$c_1 = 0.5\sigma_d^2.$$

1.B.2 Stability

I follow an argument similar to that of Lettau and Wachter (2011). From (1.14), (1.15), and the restrictions on the coefficients, we have that $b_{d,i}$ and $b_{r,i}$ follow first-order difference equations that converge, respectively, to

$$\begin{aligned} \bar{b}_d &= \frac{\varphi_d}{1 - \varphi_d} \\ \bar{b}_r &= -\frac{1}{1 - \varphi_r}. \end{aligned}$$

Consequently, the steady state of (1.16) is given by

$$\bar{c} = \frac{0.5(\bar{b}_d + 1)^2\sigma_d^2 + 0.5\bar{b}_r^2\sigma_r^2}{1 - \beta}.$$

Therefore, from (1.13) we have that

$$a_{i+1} - a_i \rightarrow \mu_d - \mu_r - 0.5 \log(1 - 2\bar{c}\alpha(1 - \beta)) + c_i\tau(1 - \beta) \quad \text{as } i \rightarrow \infty.$$

Hence, a necessary condition for $f_i(g_t, r_t^e, h_t) \rightarrow -\infty$ as $i \rightarrow \infty$, is $\bar{a} < 0$, where

$$\bar{a} = \mu_d - \mu_r - 0.5 \log(1 - 2\bar{c}\alpha(1 - \beta)) + c_i\tau(1 - \beta).$$

Or, equivalently, that

$$\mu_r > \mu_d - 0.5 \log(1 - 2\bar{c}\alpha(1 - \beta)) + c_i\tau(1 - \beta),$$

that is the mean discount rate is larger than mean dividend growth plus a Jensen inequality adjustment for the mean variance of the variables. For the case of constant discount rate r and homoscedastic white noise dividend growth, this condition reduces to the expression $r > \mu_d + 0.5\sigma_d^2$ by Froot and Obstfeld (1991).

1.B.3 Approximation

The pricing coefficients (1.13) through (1.16) are computed exactly for n^* terms and the remainder is approximated by a geometric sum. For simplicity, define

$$d_t^i = a_i + b_{d,i}\Delta d_t + b_{r,i}r_t^e + c_i h_t.$$

We have that

$$\sum_{i=1}^{\infty} \exp(d_t^i) = \sum_{i=1}^{n^*} \exp(d_t^i) + \sum_{i=n^*+1}^{\infty} \exp(d_t^i) =: \sum_{i=1}^{n^*} \exp(d_t^i) + e_t.$$

As shown in Section 1.B.2, in the limit, $\Delta d_t^i = \bar{a}$. So we have

$$\begin{aligned}
e_t &\approx \exp(d_t^{n^*}) \sum_{i=n^*+1}^{\infty} \exp(\bar{a}(i - n^*)) \\
&= \exp(d_t^{n^*} - n^* \bar{a}) \left(\sum_{i=1}^{\infty} \exp(\bar{a})^i - \sum_{i=1}^{n^*} \exp(\bar{a})^i \right) \\
&= \exp(d_t^{n^*} - n^* \bar{a}) \left(\frac{\exp(\bar{a})}{1 - \exp(\bar{a})} - \exp(\bar{a}) \frac{1 - \exp(\bar{a})^{n^*+1}}{1 - \exp(\bar{a})} \right) \\
&= \frac{\exp(d_t^{n^*} + 2\bar{a})}{1 - \exp(\bar{a})}.
\end{aligned}$$

In the numerical computations, I set n^* to 500.

1.C Data and estimation

1.C.1 Data

Monthly dataset

I compute RV_t from daily ex-dividend returns (VWRETX) in the value-weighted dataset CRSP from 1926 to 2016

$$RV_t = \sum_{d \in t} (r_d^x)^2,$$

where r_d^x is the ex-dividend return in the daily dataset and $\{d\}$ is the index of days.

Annual dataset

In the CRSP data, returns R_t^a are given by the yearly value-weighted cum-dividend return index. The dividend series is constructed as $D_t^a = (R_t^a - R_t^{xa})P_{t-12}$, where R_t^{xa} is the yearly ex-dividend return index and P_{t-12} is the index value at the end of the previous year. All variables are converted to real terms using the ‘‘Consumer Price Index for All Urban Consumers’’ from

FRED.

1.C.2 Aggregation

Annual returns are the sum of monthly returns,

$$r_t^a = \sum_{i=0}^{11} r_{t-i},$$

for $t \in \{12, 24, \dots\}$. Annual reinvested dividends are given by

$$D_t^a = D_t + \sum_{i=1}^{11} D_{t-i} \exp\left(\sum_{j=1}^i r_{t-i+j}\right)$$

for $t \in \{12, 24, \dots\}$. To compute realized returns from the model output, I use that

$$\begin{aligned} R_{t+1} &= \frac{P_{t+1} + D_{t+1}}{P_t} = \frac{P_{t+1}/D_{t+1}}{P_t/D_{t+1}} + \frac{D_{t+1}}{P_t} = \left(\frac{P_{t+1}/D_{t+1}}{P_t/D_t} + \frac{D_t}{P_t}\right) \exp(\Delta d_{t+1}) \\ &= \exp(\Delta d_{t+1}) \left(\frac{P_t}{D_t}\right)^{-1} \left(1 + \frac{P_{t+1}}{D_{t+1}}\right). \end{aligned}$$

Taking logs,

$$r_{t+1} = \Delta d_{t+1} - \log(P_t/D_t) + \log(1 + P_{t+1}/D_{t+1}).$$

Similarly, for ex-dividend returns, we have

$$\begin{aligned} R_{t+1}^x &= \frac{P_{t+1}}{P_t} = \frac{P_{t+1}/D_{t+1}}{P_t/D_{t+1}} = \left(\frac{P_{t+1}/D_{t+1}}{P_t/D_t}\right) \exp(\Delta d_{t+1}) \\ &= \exp(\Delta d_{t+1}) \left(\frac{P_t}{D_t}\right)^{-1} \left(\frac{P_{t+1}}{D_{t+1}}\right). \end{aligned}$$

Taking logs,

$$r_{t+1}^x = \Delta d_{t+1} - \log(P_t/D_t) + \log(P_{t+1}/D_{t+1}).$$

The price-dividend ratio with respect to annual dividends is given by

$$pd_t^a = \log \left(\frac{P_t}{D_t} \times D_t \times (D_t^a)^{-1} \right).$$

1.C.3 Scores

Denote by T the number of monthly observations. The log-likelihood of the monthly observations \mathbf{RV} , conditional on an initial observation, is

$$\ell(\mathbf{RV}|\boldsymbol{\eta}_1, RV_0) \propto -\frac{1}{2} \sum_{t=1}^T \left(\log \eta_3^2 + \frac{(RV_t - \eta_2 - g_t)^2}{\eta_3^2} \right).$$

The derivative with respect to the scale parameter η_3^2 is

$$-\frac{1}{2} \sum_{t=1}^T \left(\frac{1}{\eta_3^2} - \frac{(RV_t - \eta_2 - g_t)^2}{\eta_3^4} \right).$$

The derivative with respect to η_2 is

$$\frac{\partial \ell(\mathbf{RV}|\boldsymbol{\eta}_1, RV_0)}{\partial \eta_2} = \sum_{t=1}^T \frac{RV_t - \eta_2 - g_t}{\eta_3^2} \left(1 + \frac{\partial g_t}{\partial \eta_2} \right),$$

and the derivative with respect to $\boldsymbol{\eta}_{01} = (\eta_0, \eta_1)'$ is

$$\frac{\partial \ell(\mathbf{RV}|\boldsymbol{\eta}_1, RV_0)}{\partial \boldsymbol{\eta}_{01}} = \sum_{t=1}^T \frac{RV_t - \eta_2 - g_t}{\eta_3^2} \frac{\partial g_t}{\partial \boldsymbol{\eta}_{01}}.$$

For $t > 1$, we have

$$\begin{aligned}\frac{\partial g_t}{\partial \eta_0} &= RV_{t-1} + \eta_1 \frac{\partial g_{t-1}}{\partial \eta_1} \\ \frac{\partial g_t}{\partial \eta_1} &= g_{t-1} + \eta_1 \frac{\partial g_{t-1}}{\partial \eta_1} \\ \frac{\partial g_t}{\partial \eta_2} &= \eta_1 \frac{\partial g_{t-1}}{\partial \eta_2}.\end{aligned}$$

Setting $g_1 = \mathbb{E}[g_t] = \eta_0 \eta_2 / (1 - \eta_0 - \eta_1)$ gives the initial conditions for $t = 1$

$$\begin{aligned}\frac{\partial g_1}{\partial \eta_0} &= \frac{\eta_2}{1 - \eta_0 - \eta_1} + \frac{\eta_0 \eta_2}{(1 - \eta_0 - \eta_1)^2} \\ \frac{\partial g_1}{\partial \eta_1} &= \frac{\eta_0 \eta_2}{(1 - \eta_0 - \eta_1)^2} \\ \frac{\partial g_1}{\partial \eta_2} &= \frac{\eta_0}{1 - \eta_0 - \eta_1}.\end{aligned}$$

Expressions for the scores of the VAR can be found in a textbook treatment of maximum likelihood estimation of a VAR (e.g., Chapter 3 of Lütkepohl 2005).

1.C.4 Two step standard errors

Denote the parameters and sample sizes of the first and second stage as $\boldsymbol{\theta}_1$ and $\boldsymbol{\theta}_2$, respectively.

The moment equations are given by

$$\begin{aligned}\mathbf{g}_1(\boldsymbol{\theta}_1) &= \mathbb{E} \frac{\partial}{\partial \boldsymbol{\eta}_1} \ell(RV_t | RV_{t-1}, \boldsymbol{\theta}_1, \hat{\boldsymbol{\eta}}_1) = 0 \\ \mathbf{g}_2(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) &= \mathbb{E} \frac{\partial}{\partial \boldsymbol{\eta}_2} \ell(\mathbf{y}_t | \mathbf{y}_{t-1}, \boldsymbol{\theta}_2, \boldsymbol{\theta}_1, \hat{\boldsymbol{\eta}}_2) = 0.\end{aligned}$$

Denote the index of years as $\mathcal{A} = \{12, 24, \dots, n \times 12\}$ so that n is the sample size in years. Also define year as

$$\text{year}(s) = \min_{\substack{t \in \mathcal{A} \\ t \geq s}} \{t\}$$

and denote the number of simulated paths by B . The sample counterpart of the moment conditions is

$$\begin{aligned} \mathbf{g}_{1,n}(\boldsymbol{\theta}_1) &= \frac{1}{n} \sum_{t \in \mathcal{A}} \frac{1}{B} \sum_b \sum_{\text{year}(s)=t} \frac{\partial}{\partial \boldsymbol{\eta}_1} \ell(RV_s^b | RV_{s-1}^b, \boldsymbol{\theta}_1, \hat{\boldsymbol{\eta}}_1) \\ \mathbf{g}_{2,n}(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) &= \frac{1}{n} \sum_{t \in \mathcal{A}} \frac{1}{B} \sum_b \frac{\partial}{\partial \boldsymbol{\eta}_2} \ell(\mathbf{y}_t^b | \mathbf{y}_{t-1}^b, \boldsymbol{\theta}_2, \boldsymbol{\theta}_1, \hat{\boldsymbol{\eta}}_2). \end{aligned}$$

Also let

$$\begin{aligned} \mathbf{H}_1(\boldsymbol{\theta}_1) &= \frac{\partial}{\partial \boldsymbol{\theta}_1} \mathbf{g}_1(\boldsymbol{\theta}_1) \\ \mathbf{H}_2(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) &= \frac{\partial}{\partial \boldsymbol{\theta}_2} \mathbf{g}_2(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) \\ \mathbf{H}_{2,1}(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) &= \frac{\partial}{\partial \boldsymbol{\theta}_1} \mathbf{g}_2(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) \end{aligned}$$

and

$$\begin{aligned} \mathbf{H}_{1,n}(\boldsymbol{\theta}_1) &= \frac{\partial}{\partial \boldsymbol{\theta}_1} \mathbf{g}_{1,n}(\boldsymbol{\theta}_1) \\ \mathbf{H}_{2,n}(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) &= \frac{\partial}{\partial \boldsymbol{\theta}_2} \mathbf{g}_{2,n}(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) \\ \mathbf{H}_{2,1,n}(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) &= \frac{\partial}{\partial \boldsymbol{\theta}_1} \mathbf{g}_{2,n}(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1). \end{aligned}$$

I now list some high-level assumptions that allow for a derivation of the asymptotic variance.

Assumption 1. *The moment conditions at the estimated parameter values permit the following asymptotic representations:*

1.

$$\mathbf{g}_{1,n}(\hat{\boldsymbol{\theta}}_1) = \mathbf{g}_{1,n}(\boldsymbol{\theta}_1) + \mathbf{H}_{1,n}(\hat{\boldsymbol{\theta}}_1)(\hat{\boldsymbol{\theta}}_1 - \boldsymbol{\theta}_1) + o_p(1)$$

2.

$$\mathbf{g}_{2,n}(\hat{\boldsymbol{\theta}}_2, \hat{\boldsymbol{\theta}}_1) = \mathbf{g}_{2,n}(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2) + \mathbf{H}_{2,n}(\hat{\boldsymbol{\theta}}_2, \hat{\boldsymbol{\theta}}_1)(\hat{\boldsymbol{\theta}}_2 - \boldsymbol{\theta}_2) + \mathbf{H}_{2,1,n}(\hat{\boldsymbol{\theta}}_1)(\hat{\boldsymbol{\theta}}_1 - \boldsymbol{\theta}_1) + o_p(1).$$

Assumption 2. *The scores obey a central limit theorem*

$$\sqrt{n} \begin{pmatrix} \mathbf{g}_{2,n}(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2) \\ \mathbf{g}_{1,n}(\boldsymbol{\theta}_1) \end{pmatrix} \xrightarrow{d} \mathcal{N} \left(\mathbf{0}, \begin{pmatrix} \boldsymbol{\Sigma}_{g,1} & \boldsymbol{\Sigma}'_{g,1,2} \\ \boldsymbol{\Sigma}_{g,1,2} & \boldsymbol{\Sigma}_{g,2} \end{pmatrix} \right).$$

Assumption 3. *The following quantities converge in probability:*

1.

$$\mathbf{H}_{1,n}(\hat{\boldsymbol{\theta}}_1) \xrightarrow{p} \mathbf{H}_1(\boldsymbol{\theta}_1).$$

2.

$$\mathbf{W}_{1,n} \xrightarrow{p} \mathbf{W}_1.$$

3.

$$\mathbf{H}_{2,n}(\hat{\boldsymbol{\theta}}_2, \hat{\boldsymbol{\theta}}_1) \xrightarrow{p} \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2)$$

4.

$$\mathbf{H}_{2,1,n}(\hat{\boldsymbol{\theta}}_2, \hat{\boldsymbol{\theta}}_1) \xrightarrow{p} \mathbf{H}_{2,1}(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2).$$

5.

$$\mathbf{W}_{2,n} \xrightarrow{p} \mathbf{W}_2.$$

First step

This step is the standard derivation of the asymptotic variance for the generalized method of moments (GMM) estimator and only repeated here for completeness. The criterion to be minimized is

$$\mathbf{g}_{1,n}(\boldsymbol{\theta}_1)' \mathbf{W}_{1,n} \mathbf{g}_{1,n}(\boldsymbol{\theta}_1).$$

So, the estimates $\hat{\boldsymbol{\theta}}_1$ fulfill the first order conditions

$$0 = \frac{\partial}{\partial \boldsymbol{\theta}_1} \mathbf{g}_{1,n}(\hat{\boldsymbol{\theta}}_1)' \mathbf{W}_{1,n} \mathbf{g}_{1,n}(\hat{\boldsymbol{\theta}}_1) = \mathbf{H}_{1,n}(\hat{\boldsymbol{\theta}}_1)' 2 \mathbf{W}_{1,n} \mathbf{g}_{1,n}(\hat{\boldsymbol{\theta}}_1).$$

Using Assumption 1:

$$0 = \mathbf{H}_{1,n}(\hat{\boldsymbol{\theta}}_1)' \mathbf{W}_{1,n} \left[\mathbf{g}_{1,n}(\boldsymbol{\theta}_1) + \mathbf{H}_{1,n}(\hat{\boldsymbol{\theta}}_1)(\hat{\boldsymbol{\theta}}_1 - \boldsymbol{\theta}_1) \right] + o_p(1).$$

Rearranging

$$\hat{\boldsymbol{\theta}}_1 - \boldsymbol{\theta}_1 = \left[-\mathbf{H}_{1,n}(\hat{\boldsymbol{\theta}}_1)' \mathbf{W}_{1,n} \mathbf{H}_{1,n}(\hat{\boldsymbol{\theta}}_1) \right]^{-1} \mathbf{H}_{1,n}(\hat{\boldsymbol{\theta}}_1)' \mathbf{W}_{1,n} \mathbf{g}_{1,n}(\boldsymbol{\theta}_1) + o_p(1).$$

Under Assumptions 2 and 3, we have that

$$\sqrt{n}(\hat{\boldsymbol{\theta}}_1 - \boldsymbol{\theta}_1) \xrightarrow{d} \mathcal{N}(\mathbf{0}, \boldsymbol{\Sigma}_{\theta_1}),$$

where

$$\boldsymbol{\Sigma}_{\theta_1} = [\mathbf{H}_1(\boldsymbol{\theta}_1)' \mathbf{W}_1 \mathbf{H}_1(\boldsymbol{\theta}_1)]^{-1} \mathbf{H}_1(\boldsymbol{\theta}_1)' \mathbf{W}_1 \boldsymbol{\Sigma}_g \mathbf{W}_1 \mathbf{H}_1(\boldsymbol{\theta}_1) [\mathbf{H}_1(\boldsymbol{\theta}_1)' \mathbf{W}_1 \mathbf{H}_1(\boldsymbol{\theta}_1)]^{-1}.$$

If we choose $\mathbf{W}_1 = \hat{\Sigma}_g^{-1}$, this simplifies to

$$\Sigma_{\theta_1} = [\mathbf{H}_1(\theta_1)' \mathbf{W}_1 \mathbf{H}_1(\theta_1)]^{-1}.$$

Second step

The criterion to be minimized is

$$\mathbf{g}_{2,n}(\theta_2, \hat{\theta}_1)' \mathbf{W}_{2,n} \mathbf{g}_{2,n}(\theta_2, \hat{\theta}_1).$$

So, the estimates $\hat{\theta}_2$ fulfill the first order conditions

$$0 = \frac{\partial}{\partial \theta_2} \mathbf{g}_{2,n}(\hat{\theta}_2, \hat{\theta}_1)' \mathbf{W}_{2,n} \mathbf{g}_{2,n}(\hat{\theta}_2, \hat{\theta}_1) = \mathbf{H}_{2,n}(\hat{\theta}_2, \hat{\theta}_1)' \mathbf{W}_{2,n} \mathbf{g}_{2,n}(\hat{\theta}_2, \hat{\theta}_1).$$

Substituting Assumption 1 into the first order condition

$$0 = \mathbf{H}_{2,n}(\hat{\theta}_2, \hat{\theta}_1)' \mathbf{W}_{2,n} \left[\mathbf{g}_{2,n}(\theta_1, \theta_2) + \mathbf{H}_{2,n}(\hat{\theta}_2, \hat{\theta}_1)(\hat{\theta}_2 - \theta_2) + \mathbf{H}_{2,1,n}(\hat{\theta}_1)(\hat{\theta}_1 - \theta_1) \right] + o_p(1).$$

Rearranging,

$$\hat{\theta}_2 - \theta_2 = \left[-\mathbf{H}_{2,n}(\hat{\theta}_2, \hat{\theta}_1)' \mathbf{W}_{2,n} \mathbf{H}_{2,n}(\hat{\theta}_2, \hat{\theta}_1) \right]^{-1} \mathbf{H}_{2,n}(\hat{\theta}_2, \hat{\theta}_1)' \mathbf{W}_{2,n} \left[\mathbf{g}_{2,n}(\theta_1, \theta_2) + \mathbf{H}_{2,1,n}(\hat{\theta}_1)(\hat{\theta}_1 - \theta_1) \right] + o_p(1).$$

Substituting the approximation for $\hat{\theta}_1 - \theta_1$ from the first step

$$\hat{\theta}_2 - \theta_2 = \left[-\mathbf{H}_{2,n}(\hat{\theta}_2, \hat{\theta}_1)' \mathbf{W}_{2,n} \mathbf{H}_{2,n}(\hat{\theta}_2, \hat{\theta}_1) \right]^{-1} \mathbf{H}_{2,n}(\hat{\theta}_2, \hat{\theta}_1)' \mathbf{W}_{2,n} \left[\mathbf{g}_{2,n}(\theta_1, \theta_2) + \mathbf{H}_{2,1,n}(\hat{\theta}_1) \left(\left[-\mathbf{H}_{1,n}(\hat{\theta}_1)' \mathbf{W}_{1,n} \mathbf{H}_{1,n}(\hat{\theta}_1) \right]^{-1} \mathbf{H}_{1,n}(\hat{\theta}_1)' \mathbf{W}_{1,n} \mathbf{g}_{1,n}(\theta_1) \right) \right] + o_p(1).$$

Under Assumptions 2 and 3

$$\sqrt{n} \begin{pmatrix} \hat{\boldsymbol{\theta}}_1 - \boldsymbol{\theta}_1 \\ \hat{\boldsymbol{\theta}}_2 - \boldsymbol{\theta}_2 \end{pmatrix} \xrightarrow{d} \mathcal{N} \left(\mathbf{0}, \begin{pmatrix} \boldsymbol{\Sigma}_{\theta_1} & \boldsymbol{\Sigma}'_{\theta_{1,2}} \\ \boldsymbol{\Sigma}_{\theta_{1,2}} & \boldsymbol{\Sigma}_{\theta_2} \end{pmatrix} \right),$$

where

$$\begin{aligned} \boldsymbol{\Sigma}_{\theta_2} &= \mathbf{M}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2)' \mathbf{W}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2) \mathbf{M}_2 \\ &+ \mathbf{M}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2)' \mathbf{W}_2 \mathbf{H}_{2,1}(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1)' \boldsymbol{\Sigma}_{\theta_1} \mathbf{H}_{2,1}(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) \mathbf{W}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2) \mathbf{M}_2 \\ &+ \mathbf{M}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2)' \mathbf{W}_2 \boldsymbol{\Sigma}_{g,1,2} \mathbf{W}_1 \mathbf{H}_1(\boldsymbol{\theta}_1) \boldsymbol{\Sigma}_{\theta_1} \mathbf{H}_{2,1}(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1)' \mathbf{W}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2) \mathbf{M}_2 \\ &+ \mathbf{M}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2)' \mathbf{W}_2 \mathbf{H}_{2,1}(\boldsymbol{\theta}_2, \boldsymbol{\theta}_1) \boldsymbol{\Sigma}_{\theta_1} \mathbf{H}_1(\boldsymbol{\theta}_1)' \mathbf{W}_1 \boldsymbol{\Sigma}'_{g,1,2} \mathbf{W}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2) \mathbf{M}_2 \end{aligned}$$

and

$$\boldsymbol{\Sigma}_{\theta_{1,2}} = \mathbf{M}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2)' \mathbf{W}_2 \boldsymbol{\Sigma}_{g,1,2} \mathbf{W}_1 \mathbf{H}_1(\boldsymbol{\theta}_1) \boldsymbol{\Sigma}_{\theta_1} - \mathbf{M}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2)' \mathbf{W}_2 \mathbf{H}_{2,1}(\boldsymbol{\theta}_1) \boldsymbol{\Sigma}_{\theta_1},$$

letting $\mathbf{W}_1 = \hat{\boldsymbol{\Sigma}}_{g,1}^{-1}$ and $\mathbf{W}_2 = \hat{\boldsymbol{\Sigma}}_{g,2}^{-1}$ and defining $\mathbf{M}_2 = [\mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2)' \mathbf{W}_2 \mathbf{H}_2(\boldsymbol{\theta}_1, \boldsymbol{\theta}_2)]^{-1}$.

1.D Additional simulation results

1.D.1 Present value model without time-varying heteroscedasticity

Figure 1.D.1 reports Type 1 error rates of tests for explosive growth when innovations to state variables are homoscedastic, that is, letting $\alpha = \beta = 0$ and $\tau = 0.0080$ in (1.25). Compared to Figure 1.1, rejection frequencies fall slightly when homoscedasticity to innovations is imposed. In case of the test for periodically explosive growth, this confirms the finding by Phillips, Shi, and Yu (2015b), who report little oversizing for a random walk with heteroscedastic innovations.

Figure 1.D.2 shows Type 2 error rates of the cointegration test under the same restriction. Again, imposing homoscedastic innovations does not alter rejection frequencies much. Note that, for the case of constant discount rate and white noise dividend growth, the fundamental price-dividend ratio (1.26) is constant and the only variation in measured price-dividend ratios is due to the seasonal aggregation of dividends.

1.D.2 Tests of log transformed variables

Although the hypotheses derived in the main part of this chapter are about price levels, similar hypotheses can be derived for log prices. The variance decomposition test in logs by Cochrane (2011) has been described in Section 1.A.3. Phillips, Wu, and Yu (2011) derive an analogous condition to (1.2) for log prices using the log linearization of the return by Campbell and Shiller (1988). As for the cointegration test, the ratio of prices and dividends will be stationary even in the general present value model (1.12), so a cointegration test applied to the log price-dividend ratio might be promising.

Implementation

When running the tests for explosive growth in log prices, I follow the same guidelines as in the tests in price levels.

Following Timmermann (1995), when applying the cointegration test in logs, I allow for a drift. In order to mitigate the effect of the resulting nuisance parameter in the distribution, the first stage regression includes a trend,

$$p_t = \alpha_0^{\text{ci}} + \alpha_1^{\text{ci}} t + \beta^{\text{ci}} d_t + u_t^{\text{ci}}, \quad u_t^{\text{ci}} \sim \text{i.i.d.}$$

Figure 1.D.1. Type 1 error rates of tests for explosive price growth when prices are generated by a present value model without time-varying heteroscedasticity in state variables.

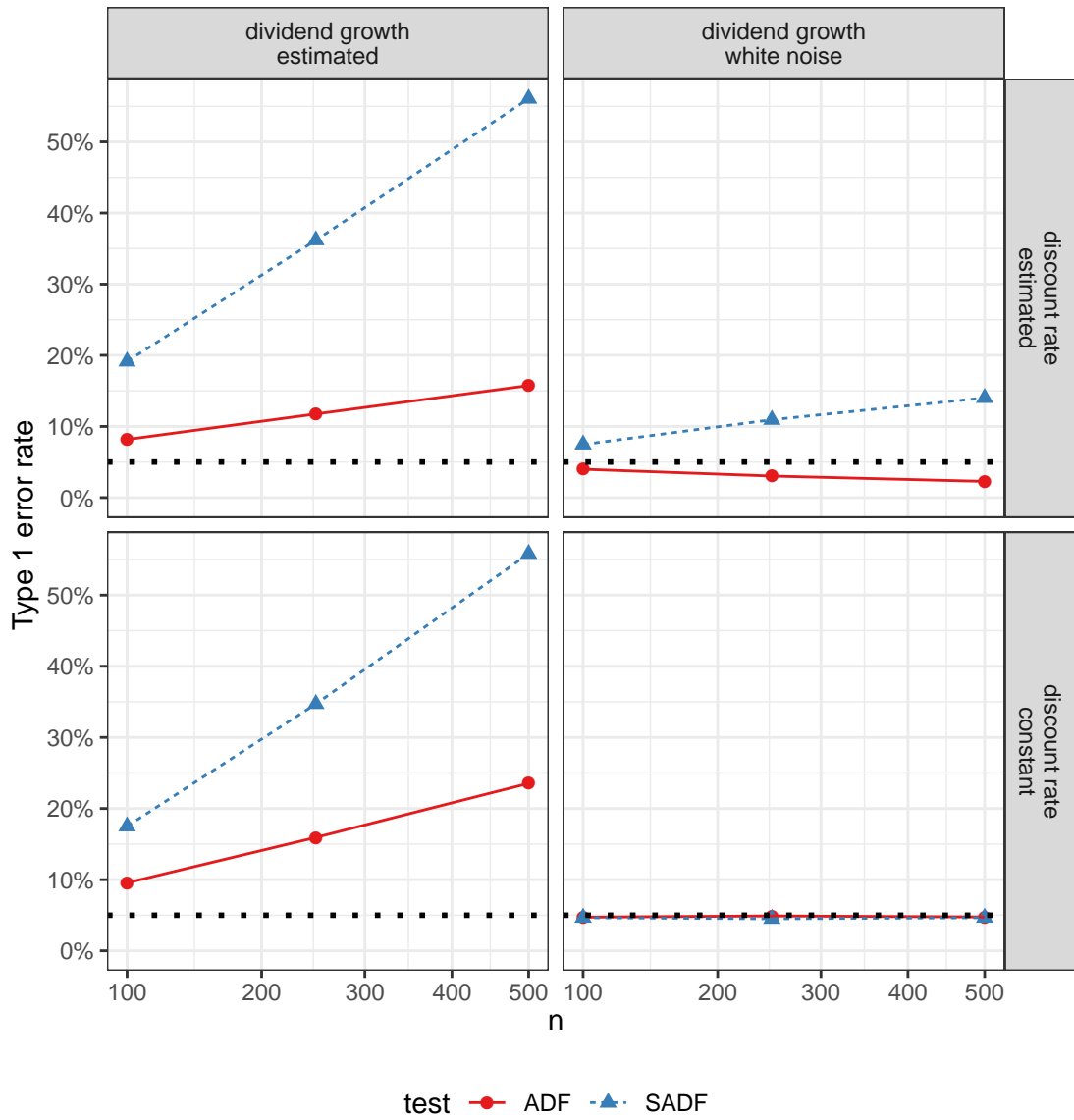
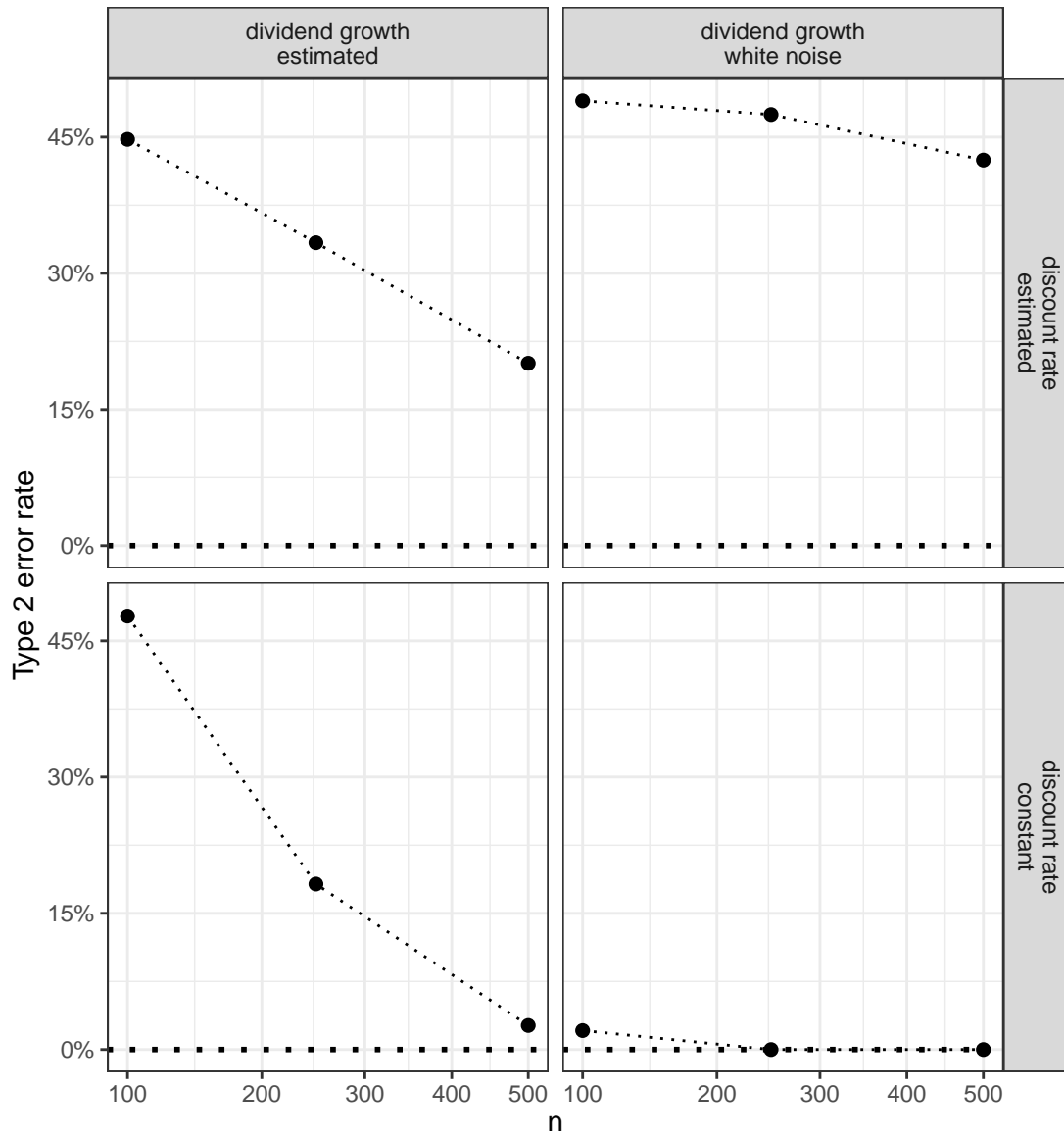


Figure 1.D.2. Type 2 error rate of the cointegration test when data are generated by a present value model without time-varying heteroscedasticity in state variables.



The second stage is again an ADF regression on the residual,

$$\Delta \hat{u}_t^{\text{ci}} = \beta^{\text{ci}} \hat{u}_t + \sum_{i=1}^k \phi_i^{\text{ci}} \Delta \hat{u}_{t-i}^{\text{ci}} + \epsilon_t^{\text{ci}}, \quad \epsilon_t^{\text{ci}} \sim \text{i.i.d.}$$

Critical values are taken from the corresponding Table in MacKinnon (2010).

For the variance ratio test in the log linear approximation, I estimate

$$\begin{pmatrix} r_t \\ \Delta d_t \\ pd_t \end{pmatrix} = \begin{pmatrix} a_r \\ a_d \\ a_{pd} \end{pmatrix} + \begin{pmatrix} b_r \\ b_d \\ b_{pd} \end{pmatrix} pd_{t-1} + \begin{pmatrix} u_{r,t} \\ u_{d,t} \\ u_{pd,t} \end{pmatrix}, \quad \begin{pmatrix} u_{r,t} \\ u_{d,t} \\ u_{pd,t} \end{pmatrix} \sim \mathcal{N}(\mathbf{0}, \mathbf{\Omega}^{vd}). \quad (1.28)$$

Under (1.28), the sums of regression coefficients in (1.24) are given in closed form as $\sum_{j=1}^{\infty} \rho^{j-1} \beta_j^d = b_d / (1 - \rho b_{pd})$ and $\sum_{j=1}^{\infty} \rho^{j-1} \beta_j^r = b_r / (1 - \rho b_{pd})$.

Results

Figure 1.D.3 plots the Type 1 error rates of tests for explosive growth in log prices. Compared to Figure 1.3, rejection frequencies for the estimated present value model are markedly reduced but still clearly higher than nominal size. Results under model restrictions imply that, overrejections seem to be mostly due to dividend growth not being white noise, as in the case of the explosive growth tests in levels. Note that power simulations exploring the sensitivity against bubble alternatives reported elsewhere employ price levels. The benchmarks to impressive power results for tests of explosive growth are the Type 1 error rates from Figure 1.3, not those from Figure 1.D.3.

Before concluding that tests for explosive growth in log prices are superior to those in levels, one needs to investigate their performance under the alternative. Figure 1.D.4 shows size-controlled Type 2 error rates of the bubble detection tests in logarithms. Compared to Figure 1.5,

there is little change in Type 2 error rates. The reason is that, under the alternative, the test statistics take lower values as well.

Figure 1.D.5 plots the Type 2 error rates of the cointegration test between log prices and log dividends. Despite the theoretical appeal of the test, no improvements in power can be discerned, compared to Figure 1.4. This finding corroborates with the one by Timmermann (1995).

1.D.3 Lag length selection procedures

I reran the simulations employing different procedures to choose the lag length k in the ADF regressions (1.21) or (1.22). The procedures were selection by BIC or by the rule proposed by Schwert (1989).

Figure 1.D.6 shows Type 1 error rates of tests for explosive growth when using BIC to select lag length. Oversizing of the SADF is exacerbated, relative to the results reported in Figure 1.3, for which $k = 0$ was imposed. Increased Type 1 error rates conform with the findings by Phillips, Shi, and Yu (2015a), who report that size distortions increase in k . Figure 1.D.7 reports Type 2 error rates of the cointegration test when selecting k by BIC in the second stage regression (1.22). Compared to Figure 1.4, the rates do not change much.

Figure 1.D.6 displays Type 1 error rates of tests for explosive growth when setting k to the closest integer of $12\sqrt[4]{\frac{n}{100}}$, as recommended by Schwert (1989). The SADF test oversizes even more with this rule. This should not be surprising, because the rule picks, on average, a higher k than BIC does. Figure 1.D.9 shows Type 2 error rates when selecting k by Schwert's rule in the cointegration test. The changes compared to Figure 1.4 do not exhibit a clear pattern.

Figure 1.D.3. Type 1 error rates of tests for explosive growth in log prices when prices are generated by the estimated present value model.

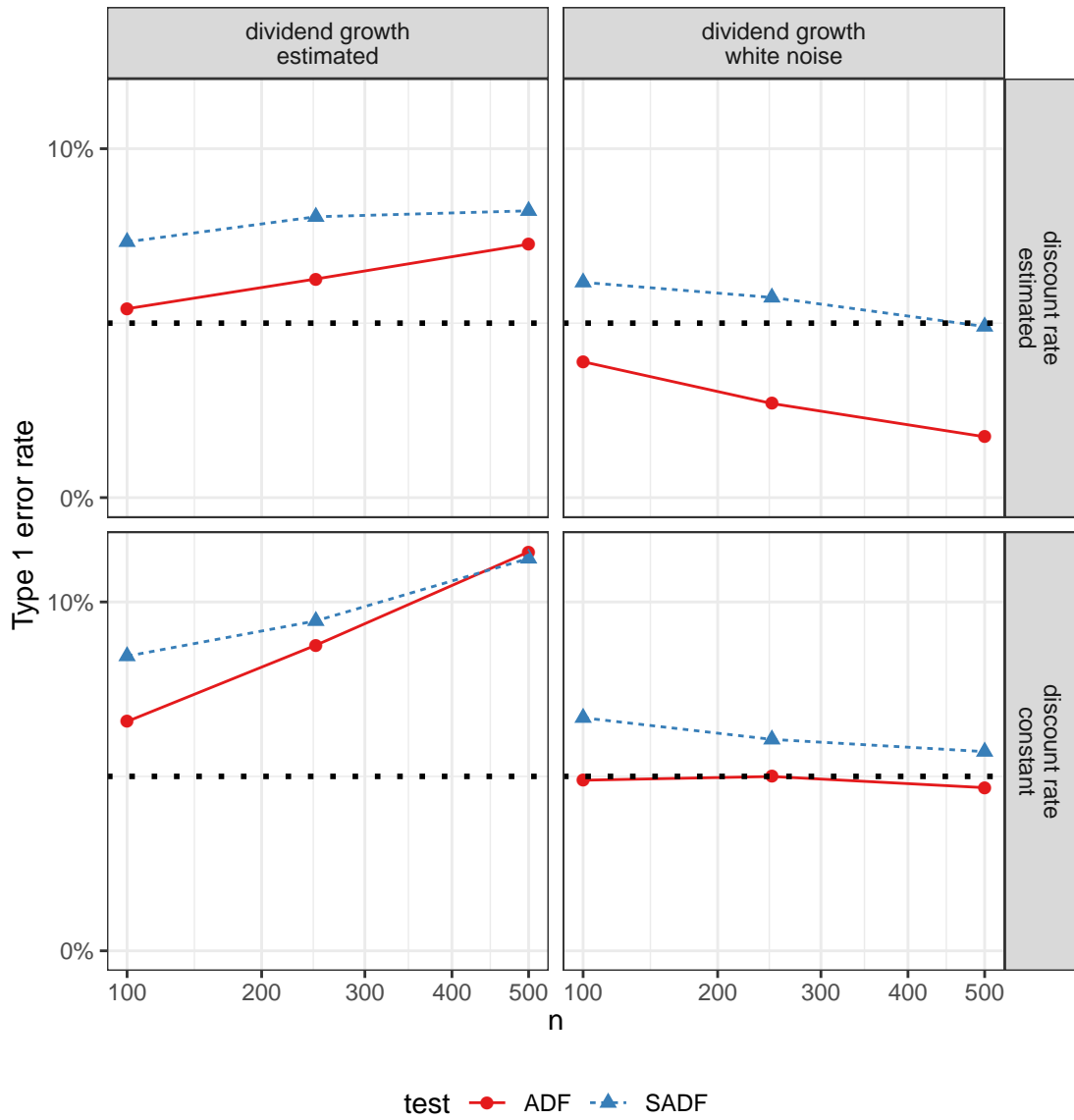


Figure 1.D.4. Type 2 error of bubble detection tests applied to log transformed variables when controlling size. Fundamental value is generated by the estimated present value model.

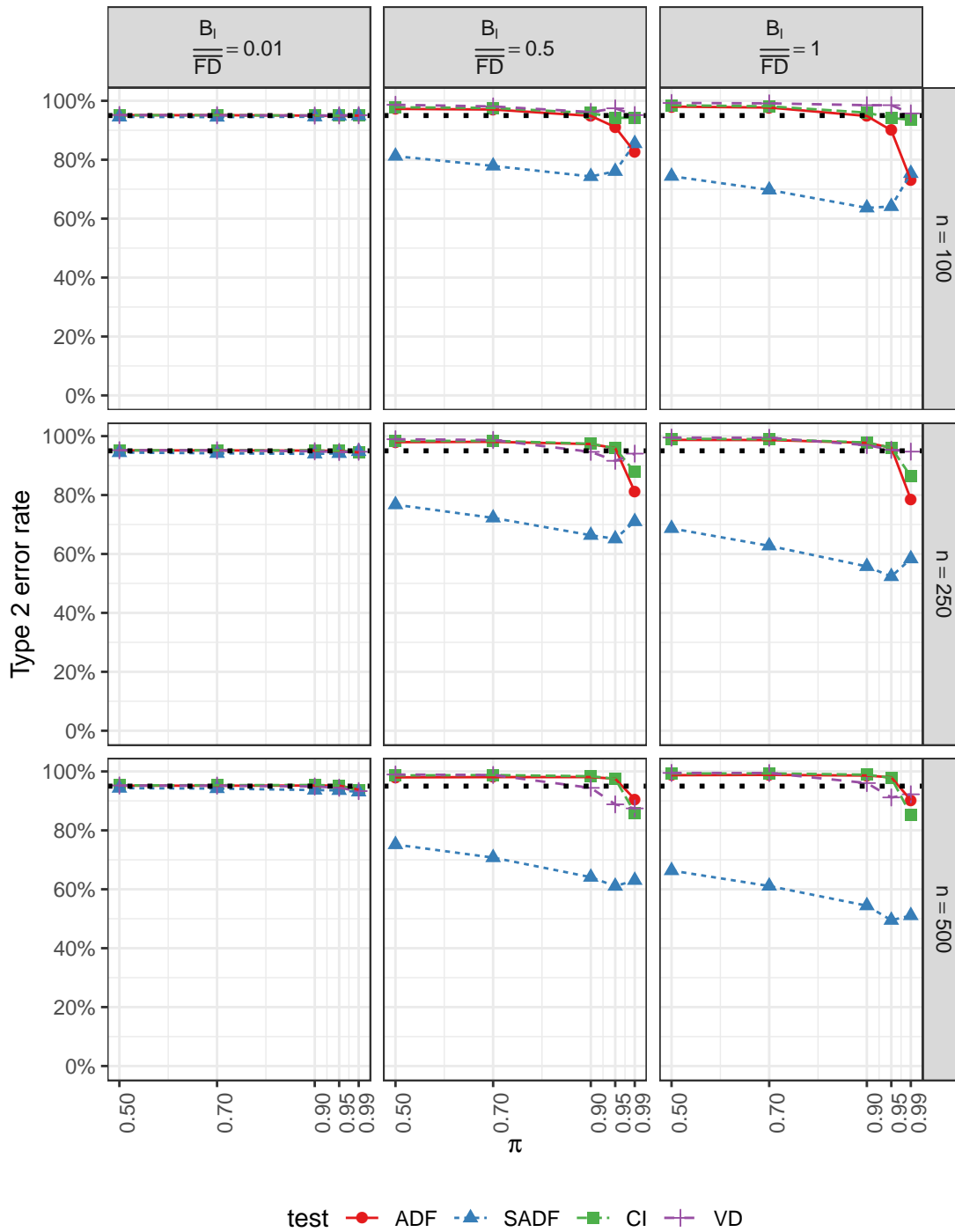


Figure 1.D.5. Type 2 error rate of the cointegration test between log prices and log dividends when data are generated by the estimated present value model.

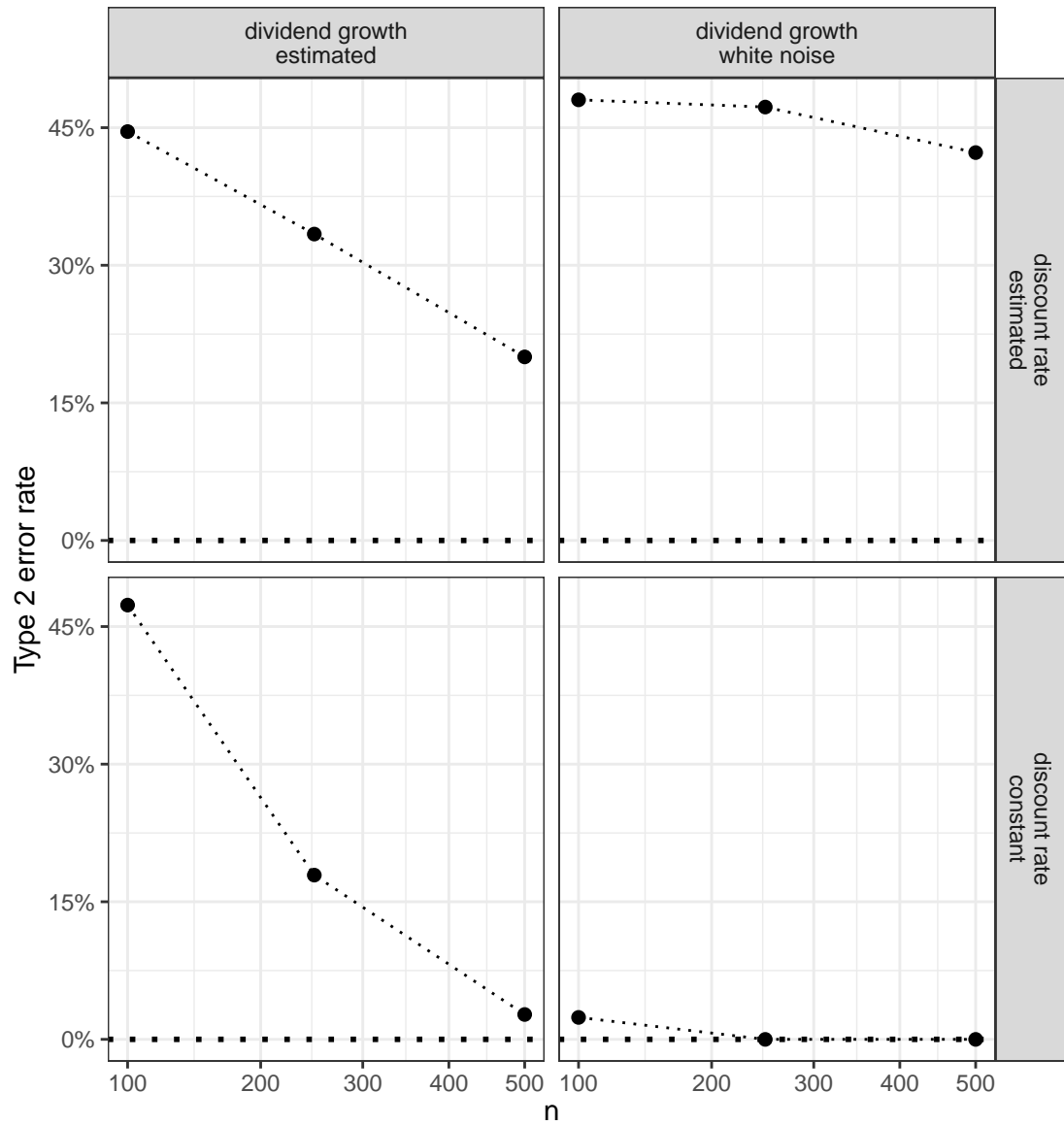


Figure 1.D.6. Type 1 error rates of tests for explosive price growth when prices are generated by the estimated present value model and k is selected by BIC.

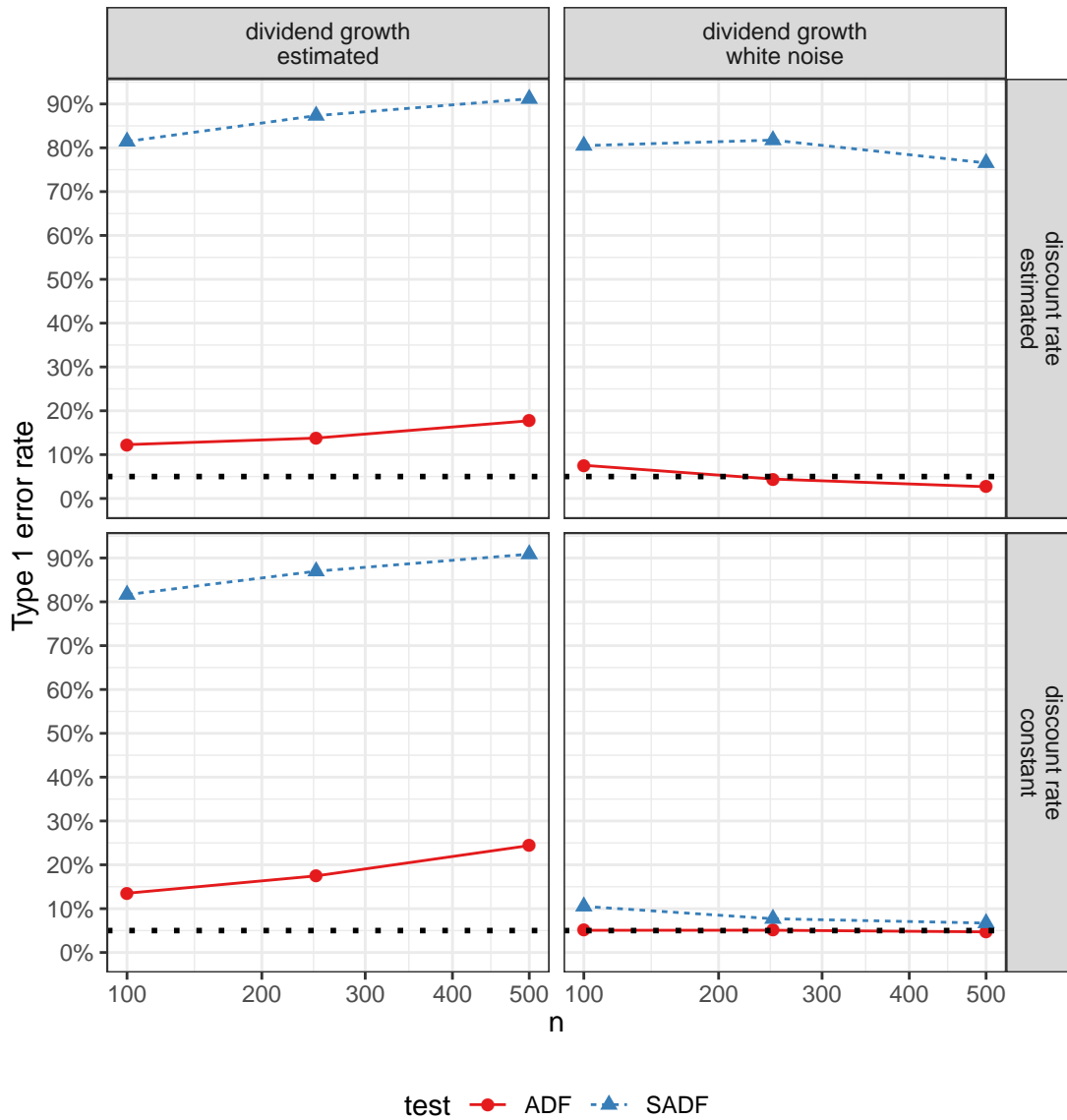


Figure 1.D.7. Type 2 error rate of the cointegration test when data are generated by the estimated present value model and k is selected by BIC.

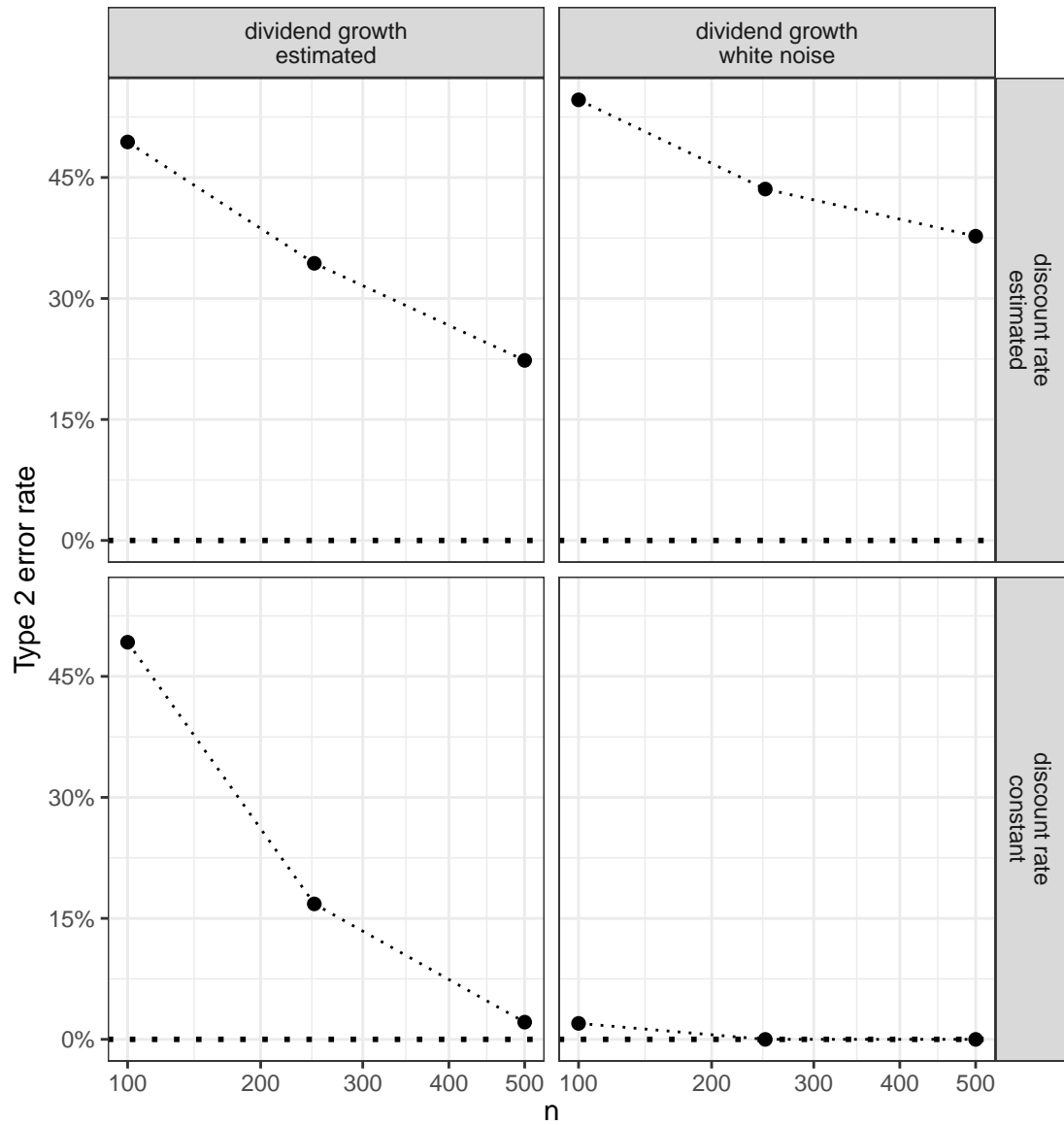


Figure 1.D.8. Type 1 error rates of tests for explosive price growth when prices are generated by the estimated present value model and k is selected by Schwert's rule.

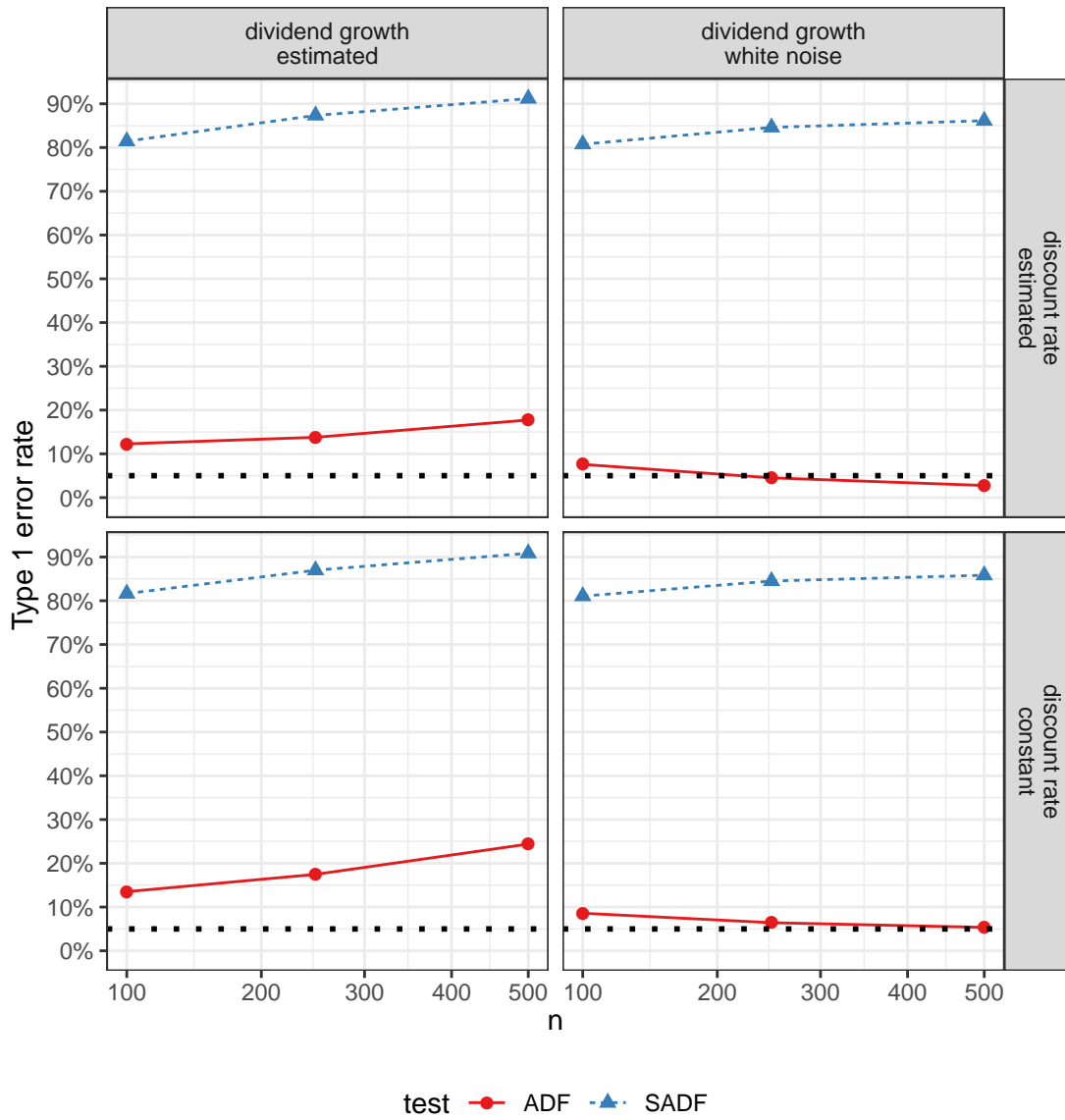
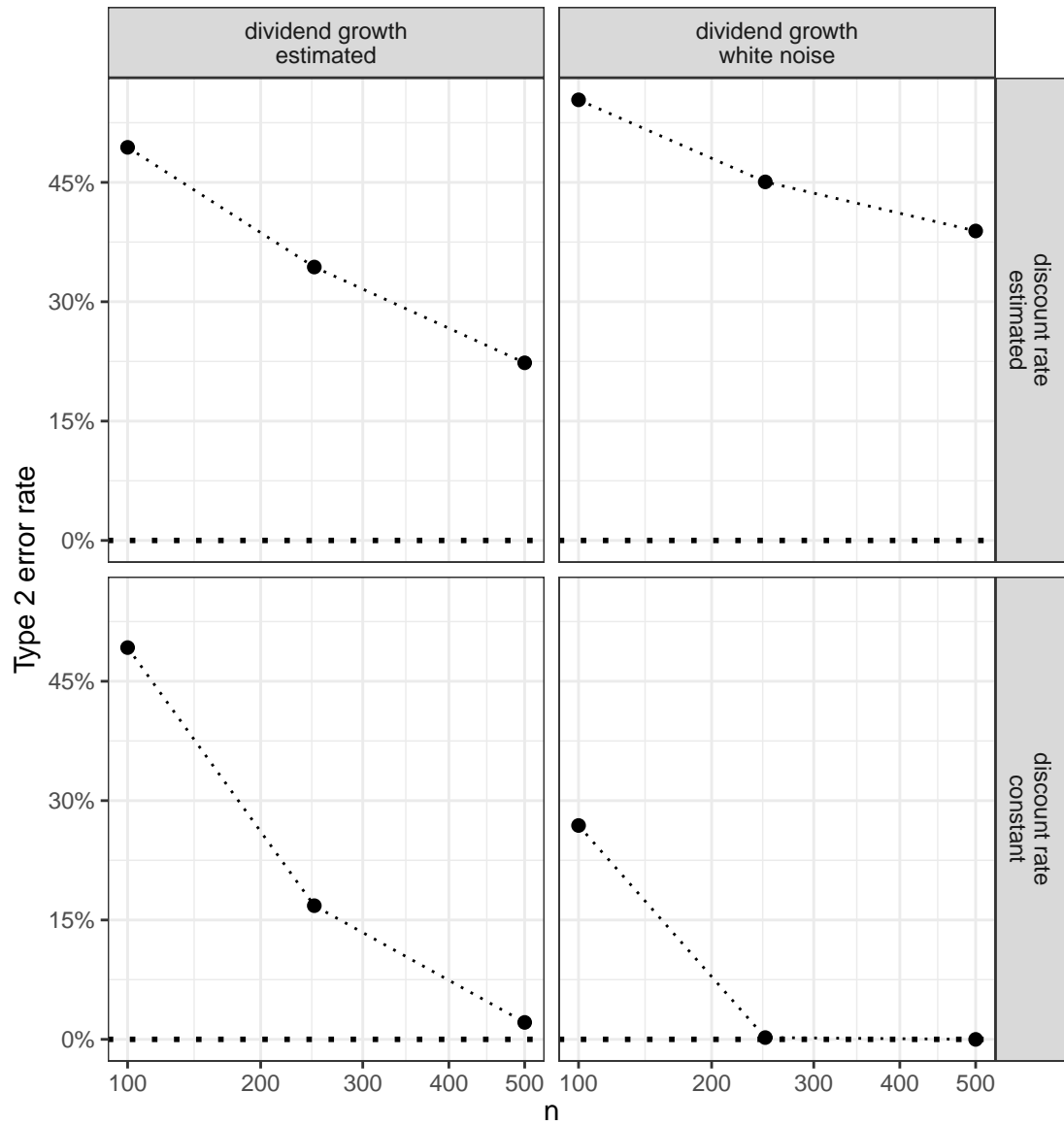


Figure 1.D.9. Type 2 error rate of the cointegration test when data are generated by the estimated present value model and k is selected by Schwert's rule.



1.E Computational aspects

1.E.1 Optimization and software

I set the length of the simulated sequence in years, B , equal to 500. In order to account for local minima and non-smoothness in (1.19) and (1.20), minimization is performed with the differential evolution algorithm (Storn and Price 1997) using the implementation by Mullen et al. (2011). When experimenting with tuning parameters of the optimization, I find that the differential evolution algorithm reliably converges to the same minimum for both criteria. The software implementation of all programs for this thesis is in R (R Development Core Team 2019), with computationally intensive operations performed in C++ using Rcpp (Eddelbuettel and Francois 2011).

1.E.2 Recursive OLS formulas for SADF test

For easy reference, I derive recursive formulas for computation of OLS here. Assuming for convenience that up to k periods before period 0 are observed, define the regressor matrix and the vector of dependent variable up to period t , respectively, as

$$\mathbf{X}_{(t)} = \begin{pmatrix} 1 & P_{t-1} & \Delta P_{t-1} & \Delta P_{t-2} & \dots & \Delta P_{t-k} \\ & & \vdots & & & \\ 1 & P_0 & \Delta P_0 & \Delta P_{-1} & \dots & \Delta P_{-k+1} \end{pmatrix} \quad \text{and} \quad \mathbf{y}_{(t)} = \begin{pmatrix} \Delta P_t \\ \Delta P_{t-1} \\ \vdots \\ \Delta P_1 \end{pmatrix}$$

and

$$\mathbf{X}_{(t+1)} = \begin{pmatrix} 1 & P_t & \Delta P_t & \Delta P_{t-1} & \dots & \Delta P_{t-k} \\ & & & \vdots & & \\ 1 & P_0 & \Delta P_0 & \Delta P_{-1} & \dots & \Delta P_{-k+1} \end{pmatrix} \quad \text{and} \quad \mathbf{y}_{(t+1)} = \begin{pmatrix} \Delta P_{t+1} \\ \Delta P_t \\ \vdots \\ \Delta P_1 \end{pmatrix}.$$

The OLS estimator at a given iteration is

$$\hat{\boldsymbol{\beta}}_{t+1} = (\mathbf{X}'_{(t+1)} \mathbf{X}_{(t+1)})^{-1} \mathbf{X}'_{(t+1)} \mathbf{y}_{(t+1)}. \quad (1.29)$$

We can check that

$$\mathbf{X}'_{(t+1)} \mathbf{X}_{(t+1)} = \mathbf{X}'_{(t)} \mathbf{X}_{(t)} + \mathbf{x}'_{(t+1)} \mathbf{x}_{(t+1)}. \quad (1.30)$$

Similarly,

$$\mathbf{X}'_{(t+1)} \mathbf{y}_{(t+1)} = \mathbf{X}'_{(t)} \mathbf{y}_{(t)} + \mathbf{x}'_{(t+1)} \mathbf{y}_{(t+1)}.$$

Using (1.29), we have

$$\begin{aligned} \mathbf{X}'_{(t+1)} \mathbf{X}_{(t+1)} \hat{\boldsymbol{\beta}}_{t+1} &= \mathbf{X}'_{(t+1)} \mathbf{y}_{(t+1)} \\ &= \mathbf{X}'_{(t)} \mathbf{y}_{(t)} + \mathbf{x}'_{(t+1)} \mathbf{y}_{(t+1)} \\ &= \mathbf{X}'_{(t)} \mathbf{X}_{(t)} \hat{\boldsymbol{\beta}}_{(t)} + \mathbf{x}'_{(t+1)} \mathbf{y}_{(t+1)} \\ &= (\mathbf{X}'_{(t+1)} \mathbf{X}_{(t+1)} - \mathbf{x}'_{(t+1)} \mathbf{x}_{(t+1)}) \hat{\boldsymbol{\beta}}_{(t)} + \mathbf{x}'_{(t+1)} \mathbf{y}_{(t+1)}. \end{aligned}$$

Solving for $\hat{\boldsymbol{\beta}}_{t+1}$,

$$\hat{\boldsymbol{\beta}}_{t+1} = \hat{\boldsymbol{\beta}}_{(t)} + (\mathbf{X}'_{(t+1)} \mathbf{X}_{(t+1)})^{-1} \mathbf{x}'_{(t+1)} (-\mathbf{x}_{(t+1)} \hat{\boldsymbol{\beta}}_{(t)} + \mathbf{y}_{(t+1)}).$$

Because inversion of $(\mathbf{X}'_{(t+1)}\mathbf{X}_{(t+1)})^{-1}$ is computationally costly, we can apply the Woodbury matrix identity to (1.30) and get

$$(\mathbf{X}'_{(t+1)}\mathbf{X}_{(t+1)})^{-1} = (\mathbf{X}'_{(t)}\mathbf{X}_{(t)})^{-1} - (1 + \mathbf{x}_{(t+1)}(\mathbf{X}'_{(t)}\mathbf{X}_{(t)})^{-1}\mathbf{x}'_{(t+1)})^{-1}(\mathbf{X}'_{(t)}\mathbf{X}_{(t)})^{-1}\mathbf{x}'_{(t+1)}\mathbf{x}_{(t+1)}(\mathbf{X}'_{(t)}\mathbf{X}_{(t)})^{-1}.$$

For the t statistics, we need an estimate of the error variance. We have

$$\hat{\sigma}_{(t+1)}^2 = \frac{1}{t+1-k} \sum_{i=1}^{t+1} (\mathbf{y}_i - \mathbf{x}'_i \hat{\boldsymbol{\beta}}_{t+1})^2,$$

for which there is little benefit in terms of computational speed from recursive computation, so

I reestimate the residuals at each step in the recursion.

Chapter 2

A Dynamic Model of Vaccine

Compliance: How Fake News

Undermined the Danish HPV

Vaccine Program

JOINT WITH PETER R. HANSEN

2.1 Introduction

Increased opposition to vaccine programs presents an important challenge to public health. Improving the understanding of the causes of vaccine hesitancy is of paramount importance to

health authorities. Vaccine scares have, historically, been accompanied by vaccine-critical media stories, prompting the question of how media contributed to vaccine hesitancy, see Offit (2011). More generally, media have been conjectured as drivers of a variety of personal decisions, ranging from asset allocation to migration, see DellaVigna and La Ferrara (2015). Vaccination is an important personal choice and the decision to get vaccinated or not has a clearly defined binary outcome. This makes vaccination data an interesting case study for measuring the influence of media. In this chapter we model time variation in vaccine uptake and analyze the extent to which this variation can be explained by related media coverage.

The chapter makes a number of econometric contributions. The first is a time series model for binomial variables with a time-varying success probability. Our model builds on the score-driven framework proposed by Creal, Koopman, and Lucas (2013), and we are, to the best of our knowledge, the first to develop a score-driven model for binomially distributed time series data. The framework implies an intuitive dynamic structure for the time variation in the binomial coefficient. If the empirical frequency exceeds the expected frequency, the binomial coefficient is subsequently adjusted upwards, and if the empirical frequency falls short of the expected frequency, the binomial coefficient is adjusted downwards. The magnitude of the adjustment is determined empirically. The score-driven model we develop is not specific to vaccination data and could easily be adapted to other time series that involve aggregated binary outcomes, for instance, the modeling of credit rating transitions and default intensities, in which time-varying parameter models were proposed using a different approach, see Koopman, Lucas, and Monteiro (2008) and Koopman, Lucas, and Schwaab (2011). A second econometric contribution is the flexible treatment of seasonality. Institutional changes in the way vaccinations were recorded compelled us to model seasonal variation at the monthly frequency, in addition to annual seasonal effects. We introduce a score-driven model of intra-monthly seasonality using an approach that is similar to that in Caivano, Harvey, and Luati (2016).

We apply the model to Danish human papillomavirus (HPV)¹ vaccine data. The data are weekly HPV vaccine initiations by birth year cohort from January 2009 to June 2017. The structure of the data calls for a model of vaccine uptake with two components. The first component characterizes baseline vaccine uptake and is identical for all birth year cohorts. The second component characterizes the variation in vaccine uptake over time, which is key in our analysis. It can be used to monitor vaccine compliance in real time, and this component is the measure that we relate to data on media coverage. The empirical analysis reveals a great deal of time variation in vaccine compliance, including a sudden drop in 2015. Our empirical results show that the decline in vaccine compliance began after negative coverage of the HPV vaccine appeared in the Danish media and, by augmenting our model to include media coverage, we show that media coverage significantly predicts declines in vaccine compliance. Some newspaper articles were blatantly false and misleading and warrant a *fake news* designation (in the original meaning of these words, which is “false news”). However, most media coverage was merely reporting on alleged side effects or on the declining vaccine uptake that had resulted from this concern. Although these news articles individually cannot be said to be false, they may collectively misrepresent the risk of vaccine-induced side effects. The largest drop in compliance occurred immediately after a TV documentary was aired on TV2 Denmark on March 26th, 2015. The program was entitled *De Vaccinerede Piger – Syge og Svigtede* (The Vaccinated Girls – Sick and Abandoned). After the program was aired, Danish HPV vaccine compliance fell from over 90% to less than 30%. TV2 Denmark has publicly acknowledged that their documentary contributed to the decline in HPV vaccinations. Our empirical analysis supports this conclusion, because of the sharp decline in HPV vaccine uptake immediately after the documentary aired. The model of HPV vaccine up-

1. HPV denotes a family with more than one hundred types of viruses, of which at least 13 can cause cancer, see WHO (2016). The HPV vaccine is expected to prevent many forms of cancer and the newest HPV vaccine, Gardasil 9, may reduce cervical cancer by as much as 90%. Evidence for the efficacy and safety of the HPV vaccine is strong. A recent Cochrane Systematic Review concluded: “There is high-certainty evidence that HPV vaccines protect against cervical precancer” and “We did not find an increased risk of serious adverse effects,” see Arbyn et al. (2018).

take also enables us to quantify the decline in vaccinations, relative to a counterfactual scenario in which compliance stayed at the level before the documentary. We estimate that nearly half of the girls born in 2003 postponed HPV vaccination following the TV2 documentary, and many of these girls are still unvaccinated. By the end of our sample period, we estimate that of the unvaccinated girls born in 2013, nearly 14,000 of these can be attributed to the declining HPV vaccine uptake that followed the TV documentary. For illustration, a 70% reduction in cervical cancer for 14,000 Danish females will, on average, translate into about one hundred fewer cases of cervical cancer and twenty-six fewer deaths. The full consequences of the decline in vaccine uptake may be substantially larger, because several other birth year cohorts, including girls born in 2004 and 2005, are also behind in vaccine coverage, relative to older cohorts.

A historical episode that is similar to the one that we investigate in this chapter is the decline in DTP vaccinations in the United States following the TV program *DPT: Vaccine Roulette*. The program initially aired in 1982 on an NBC affiliate, WRC-TV, and then nation-wide on *The Today Show*. The program falsely associated the pertussis component of the DTP vaccine with brain damage, and it was followed by extensive media coverage in the United States, which speculated that the pertussis vaccine was responsible for epilepsy, intellectual or physical disabilities, and even death, see Offit (2011, chapter 3). The Danish experience with the HPV vaccine program is similar to the onset of the pertussis vaccine scare in the United States. As had been the case for the *Vaccine Roulette* program in the United States, a large number of newspaper articles on the topic followed the Danish documentary.

It has been documented before that media can influence important personal decisions. For instance, Kearney and Levine (2015) showed that the MTV reality show *16 and Pregnant* reduced teen childbearing. Similarly, La Ferrara, Chong, and Duryea (2012) found that soap operas that portray small families had a significant impact on fertility (see also DellaVigna and La Ferrara 2015). There is also evidence that the revelation of questionable and malicious behavior by

health authorities can reduce care-seeking behavior. Specifically, the infamous Tuskegee syphilis experiment reduced the number of physician visits by black men, see Alsan and Wanamaker (2018), colonial era medical malpractice in central Africa is associated with higher levels of distrust in medicine today, see Lowes and Montero (2018), and vaccination uptake fell in Pakistan after the unmasking of CIA's involvement in a vaccination program that was used in the hunt for Osama bin Laden, see Martinez-Bravo and Stegmann (2018).

There is a nascent public health literature associating HPV vaccine hesitancy with media coverage. Faasse et al. (2017) documented that the number of monthly news articles on the HPV vaccine predicted the number of reported adverse events (AE) in New Zealand. In Denmark, we also observe a sharp increase in the number of reported AE in 2015. A recent study by Suppli et al. (2018) found that the monthly number of Danish HPV1 vaccinations was uncorrelated with media coverage before July 2013, but negatively correlated with it after July 2013. The study looks at the correlation between media activity and total number of HPV1 vaccinations, and determines a change point in July 2013. A great deal of the variation in the total number of HPV1 vaccinations can be attributed to changes in the Danish vaccination schedule. A catch-up program during the period from October 1, 2008 to December 31, 2010 has made the HPV vaccine available for free to girls born between 1993 and 1995. Another catch-up program in 2012 and 2013 made the vaccine freely available to women aged 19–26. Our analysis is based on cohort-specific weekly HPV1 vaccinations for girls aged between twelve and fourteen, which is not influenced by the catch-up programs. We relate vaccine uptake to media coverage by incorporating a measure of media coverage directly in the equation that drives the variation in uptake. In contrast, the analysis in Suppli et al. (2018) is based on a change point analysis of the correlation between the aggregate number of monthly vaccinations and media coverage.

The rest of this chapter is organized as follows. In Section 2.2 we present the core structure of our model in a simplified manner along with some preliminary empirical results for the Danish

HPV vaccination data. In Section 2.3 we present the econometric time-series model with a time-varying vaccine compliance. In Section 2.4 we incorporate media coverage of the HPV vaccine in the analysis, and show that the intensity of such coverage predicts the observed time variation in vaccine compliance. We summarize and conclude in Section 2.5. Mathematical derivations, supplementary empirical results, and information about media coverage can be found in Sections 2.A through 2.D in the supplementary material.

2.2 Data and preliminary analysis

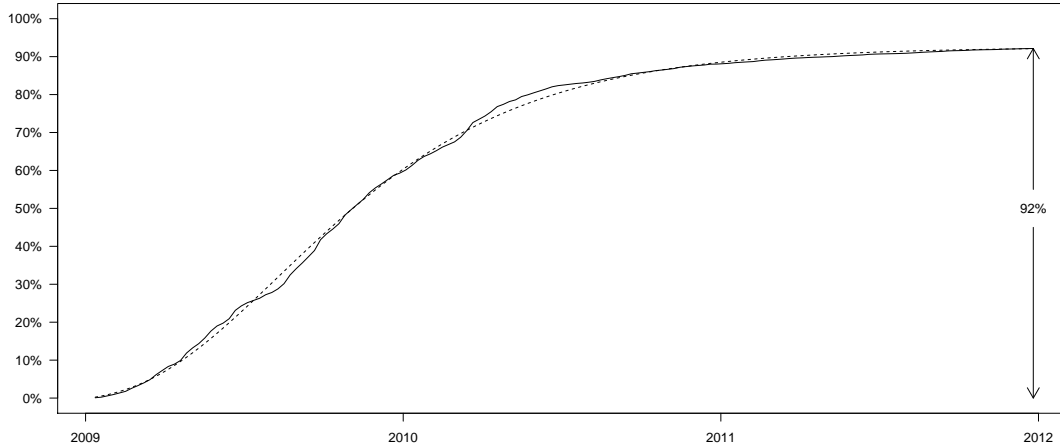
We obtained weekly birth year cohort-specific HPV vaccination data from the Statens Serum Institute (SSI) and birth year cohort size data from Statistics Denmark. SSI is responsible for the purchase and supply of vaccines to the Danish national vaccination programs and SSI collects data on vaccination uptake. Two types of HPV vaccines were administered during the sample period from early 2009 to mid-2017: Gardasil and Cervarix. HPV vaccines were initially licensed with three dose schedules, but are now administered with just two doses for young adolescents. Our empirical analysis will focus on the number of girls receiving the first dose of the HPV vaccine, which we denote by HPV1. Specifically, we will model the number of girls born in year $c = 1997, \dots, 2005$, who receive HPV1 in week t .

Let X_t denote the aggregate number of HPV1 vaccinated girls by week t , out of a birth year cohort with N_c girls. The basic idea is that the fraction of vaccinated girls, at time $t = 0, \dots, T$, is approximately given by

$$X_t/N_c \approx \delta \times \Lambda\left(\frac{t}{T}\right),$$

where $\Lambda(a)$ is an increasing function with $\Lambda(0) = 0$ and $\Lambda(T) = 1$ and where δ is a scalar between zero and one. The parameter δ is key in our analysis, because it can (in a steady state) be interpreted as vaccine compliance/coverage by the end of the sample period.

Figure 2.1. HPV vaccine uptake for birth year cohort 1997. The solid line displays the cumulative percentage of HPV1 vaccinated girls who were born in 1997, and the dotted line is a simple approximation fitted to the data. By the end of 2012, 92% of girls had been vaccinated.

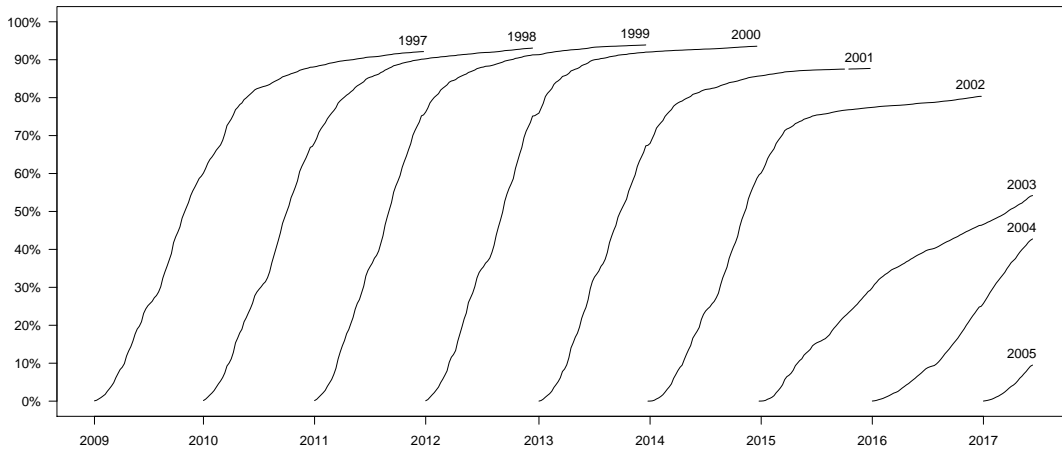


The simple structure, where δ is constant, is illustrated in Figure 2.1. The solid line shows the percentage of girls born in 1997 that have received the first dose of the HPV vaccine over a three-year period. The dotted line is a curve that is fitted to the data, using the shifted Gompertz distribution to specify $\Lambda(a_t)$, where a_t is the age of cohort 1997 at time t . In conjunction with the estimate of δ (about 92%) the simple model provides an approximation to the vaccine uptake over time for this cohort.

Figure 2.2 shows vaccine compliance over time for nine cohorts. We observe large discrepancies across birth year cohorts, with a much lower vaccine uptake for the youngest cohorts. For the four oldest cohorts, 1997-2000, vaccine adoption was high and slightly increasing over time. This was followed by a period of declining compliance starting with cohort 2001. Evidently, it is not possible to accurately describe all cohorts with a common specification such as in Figure 2.1. But, as we shall see, a modified specification that allows for time variation in the compliance rate describes the data well. The cornerstone of our model is the weekly number of vaccinations for each cohort, which (in a static model) has the expected value $\delta[\Lambda(a_t^c) - \Lambda(a_{t-1}^c)] \times N_c$, where a_t^c is the age of cohort c at time t and N_c is the cohort size. We generalized this model by allowing

for time-variation in δ , so that the expected number of vaccinations in week t for cohort c is given by $\delta_t[\Lambda(a_t^c) - \Lambda(a_{t-1}^c)] \times N_c$.

Figure 2.2. Cumulative HPV vaccine uptake by birth year cohort. The solid lines display the percentage of HPV1 vaccinated girls for each birth year cohort over a three-year period, starting in the year they turn twelve (lines labelled by birth year).



2.3 Statistical model

Let $x_{c,t}$ be the number of girls in cohort c that receive HPV1 in week t . The number of vaccinated girls in cohort c at time t is given by $X_{c,t}$, where $X_{c,t} = X_{c,t-1} + x_{c,t}$ with $X_{c,0} = 0$. The number of unvaccinated girls in cohort c that are eligible to receive the vaccine by the end of week t is denoted by $N_{c,t}$, see Appendix 2.C.2.

The age variable, a_t^c , for cohort c at time t is, without loss of generality, normalized so that $a = 0$ denotes the beginning of the three-year period and $a = 1$ by the end of it. To take an example, for those born in year 2000, we have the weekly number of vaccinations for the period primo 2012 to ultimo 2014, such that $a_t^{2000} = 0$ at the beginning of 2012 and $a_t^{2000} = 1$ by the end of 2014. The beginning of the three-year period is motivated by the design of the program, in which girls are only offered the vaccine for free once they turn twelve, this being the recommended

age at which HPV vaccination should start. The number of Danish girls vaccinated before their twelfth birthday is, therefore, negligible.

The basic structure of our model is that the number of vaccinated girls in week t is binomially distributed

$$x_{c,t} | \mathcal{F}_{t-1} \sim \text{bin}(N_{c,t-1}, p_{c,t}), \quad (2.1)$$

where the dependence across time and cohorts and seasonal effects are embedded in the structure of $p_{c,t}$. Our model for $p_{c,t}$ is given by

$$p_{c,t}(\theta) = \delta_t(\alpha) \lambda_{c,t}(\beta), \quad \text{with} \quad \lambda_{c,t}(\beta) = \frac{N_c}{N_{c,t-1}} [\Lambda(\beta; a_t^c) - \Lambda(\beta; a_{t-1}^c)],$$

where $\theta = (\alpha', \beta)'$ is the vector of unknown parameters.

The first component, $\delta_t(\alpha) \in (0, 1)$, defines vaccine compliance at time t , whereas the second component, $\lambda_{c,t}(\beta)$, only depends on t though the age of the cohort, a_t^c . So, the second term defines the part of $p_{c,t}(\theta)$ that all cohorts have in common, and if vaccine compliance were constant over time then all cohorts would have similar vaccine uptake. This is evidently not the case, as demonstrated in Figure 2.2.

From the binomial model structure in (2.1), it follows that the log-likelihood for cohort c in period t is given by

$$\ell_{c,t}(\theta) = \log \binom{N_{c,t-1}}{x_{c,t}} + x_{c,t} \log p_{c,t}(\theta) + (N_{c,t-1} - x_{c,t}) \log(1 - p_{c,t}(\theta)). \quad (2.2)$$

The maximum likelihood estimators are obtained by maximizing $\ell(\theta) = \sum_{c,t} \ell_{c,t}(\theta)$, with respect to the vector of parameters, $\theta = (\alpha', \beta)'$. To complete the model we need to adopt specifications for $\Lambda(\beta; a)$ and $\delta_t(\alpha)$. For $\Lambda(\beta; a)$ we adopt the cumulative distribution function (cdf) for the

shifted and truncated Gompertz distribution, which is given by

$$\Lambda(\beta; a) = \frac{1}{C(\beta)}(1 - e^{-\beta_0 a}) \exp(-\beta_1 e^{-\beta_0 a}), \quad a \in [0, 1],$$

where $C(\beta) = (1 - e^{-\beta_0}) \exp(-\beta_1 e^{-\beta_0})$ is a normalizing constant. Other, more flexible, specifications could be used, and in a preliminary analysis we also experimented with specifications based on the Weibull distribution and the Beta distribution. The shifted Gompertz was adopted because it had the best empirical fit.

Possible time-variation in $\delta_t(\alpha)$, which represents vaccine compliance across time, can be modeled in many ways. In the appendix we present results for several alternative approaches, such as the case where δ_t is piecewise constant with structural changes and cases where δ_t is a deterministic function of time. The best empirical fit is obtained with the specification for $\delta_t(\alpha)$ we present next.

2.3.1 A score-driven model for $\delta_t(\alpha)$

We model δ_t using an autoregressive model for the logit transformed variable, $\tilde{\delta}_t = \log\left(\frac{\delta_t}{1-\delta_t}\right)$

$$\tilde{\delta}_t = \alpha_0 + \alpha_1 \tilde{\delta}_{t-1} + \alpha_2 \tilde{s}_{t-1}, \quad (2.3)$$

where \tilde{s}_t (to be made precise later) signals the direction in which compliance may have changed as well as the magnitude. The logit transformation allows us to model $\tilde{\delta}_t$ as an unrestricted real-valued parameter, and the inverse transformation, $\delta = e^{\tilde{\delta}}/(1 + e^{\tilde{\delta}})$, will ensure that δ stays within its boundaries between zero and one. Equation (2.3) defines an observation-driven model based on the generalized autoregressive score framework by Creal, Koopman, and Lucas (2013). Score-driven models have been very successful in modeling time-varying parameters in econometric

models, and are the underlying structure of many empirically established models, such as the GARCH model, by Bollerslev (1986). In the present context, the score-driven model adjusts the value of δ_t directly in response to the number of vaccinations, $x_{c,t}$, deviating from the expected number. The adjustment is defined by the score which is the derivative of the log-likelihood, suitably scaled by the expected curvature of the log-likelihood function.

The vector of unknown parameters in $\delta_t(\alpha)$ is given here by $\alpha = (\alpha_0, \alpha_1, \alpha_2, \delta_0)'$, where δ_0 is the starting value for δ_t . Here, α_1 is a measure of the persistence in δ_t , and α_2 measures how strongly the model responds to the signal provided by \tilde{s}_t . Typically, there is a high degree of persistence in score-driven models, so we also consider the restricted variant of the model, where $(\alpha_0, \alpha_1) = (0, 1)$, which corresponds to the case where $\tilde{\delta}_t$ is a very persistent (unit root) process.

The signal, \tilde{s}_t , is key in this model. If, for instance, the number of vaccinated girls exceeds the expected number of vaccinations, it indicates that δ_t has increased in value and, intuitively, we would want (α_2 times) \tilde{s}_t to be positive in this situation. The score-driven framework by Creal, Koopman, and Lucas (2013) employs a natural signal that is deduced from the score of the log-likelihood function, $s_t = \partial \ell_t / \partial \tilde{\delta}$, weighted by a term that is defined by the curvature of the log-likelihood, $h_t = \partial^2 \ell_t / \partial \tilde{\delta}^2$. Specifically,

$$\tilde{s}_t = \frac{\sum_c s_{c,t}}{\sqrt{-\sum_c \mathbb{E}_{t-1} h_{c,t}}} = \frac{1}{\sqrt{\sum_c \frac{\lambda_{c,t}^2 N_{c,t-1}}{p_{c,t}(1-p_{c,t})}}} \sum_c \lambda_{c,t} N_{c,t-1} \left(\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right), \quad (2.4)$$

where $\hat{p}_{c,t} = x_{c,t}/N_{c,t-1}$. The expression (2.4) is derived in Appendix 2.A.1, but the interpretation is intuitive. In a week in which more individuals are vaccinated than expected, that is $\hat{p}_{c,t} > p_{c,t}$ for all c , then $\tilde{s}_t > 0$, and this would be an indication that δ_t may have increased in value, and visa versa in the event $\tilde{s}_t < 0$. So, we should expect the estimate of α_2 to be positive, which is indeed the case in our empirical analysis.

Our empirical analysis revealed that there was a need to account for seasonal variation in the

weekly vaccination rate. The most obvious seasonal effect is associated with the summer and winter vacations, during which the number of vaccinations is distinctly below that of neighboring weeks. This seasonal effect is pronounced and can be seen in Figures 2.1 and 2.2. The second seasonal effect is specific to the way in which the weekly vaccination data were collected over time. During the first part of the sample period, the reported number of vaccinations is higher towards the end of the month. After conferring with a medical professional, we discovered that the way in which vaccines are recorded has changed during the sample period. Before November 15, 2015, vaccines were recorded when physicians billed for the vaccines, which resulted in an overrecording of vaccines towards the end of the month. The introduction of an electronic vaccine registry (Det Danske Vaccinationsregister) resolved this issue when it started on November 15, 2015. Our modeling of seasonal effects is detailed in Appendix 2.B.

Table 2.1. Estimates for score-driven model

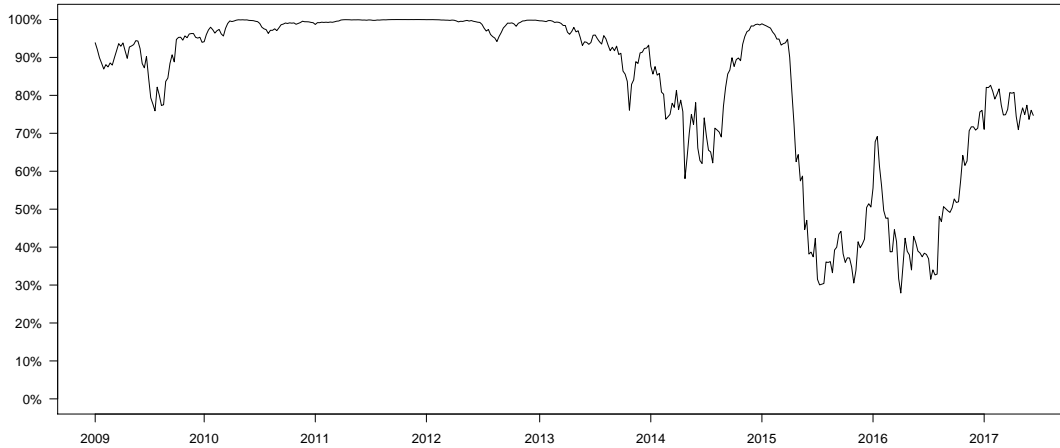
	$\hat{\alpha}_0$	$\hat{\alpha}_1$	$\hat{\alpha}_2$	$\hat{\beta}_0$	$\hat{\beta}_1$	$\ell(\hat{\theta})$
Unrestricted	0.000 (0.087)	0.992 (0.022)	0.054 (0.017)	7.250 (0.038)	3.030 (0.088)	-20122
$\alpha_0 = 0, \alpha_1 = 1$	0	1	0.066 (0.004)	7.240 (0.018)	3.010 (0.003)	-20318

Note: Standard errors in parentheses.

The key parameters of the estimated score-driven model are presented in Table 2.1, and the resulting time series for vaccine compliance is presented in Figure 2.3, where we plot δ_{t+1} against time t (because δ_{t+1} is observable at time t). In Table 2.1 we also report estimates of the restricted model in which we impose the restrictions $\alpha_0 = 0$ and $\alpha_1 = 1$, which resembles a local-level model for $\tilde{\delta}_t$. This specification is strongly rejected by the data. Estimates of all parameters in the model are presented in Table 2.B.1 in the appendix.

Figure 2.3 shows that vaccine compliance is estimated to be a bit lower during the first year in our sample (2009). Following this, compliance increases and stays above 95% for a three-year

Figure 2.3. The figure shows vaccine compliance, δ_t , as estimated by the score-driven model. Aside from the first year after the introduction of the HPV vaccine, the first noticeable decline in vaccine uptake is observed in 2013, followed by a rebound in 2014. The largest decline is observed in the second quarter of 2015, during which δ_t falls abruptly from about 95% to just over 30%.



period. The first noticeable decline in $\hat{\delta}_t$ is seen in 2013 and vaccine compliance is relatively low and volatile until the fall of 2014, after which compliance recovers to about 95% again. The most drastic shift in compliance is observed in the second quarter of 2015, during which $\hat{\delta}_t$ abruptly falls to just over 30%. Compliance stays low for an extended period, aside from a brief spike in early 2016. Only in late 2016 does compliance begin to recover. Towards the end of the sample period, June 2017, it hovers at about 75%. In Section 2.4 we analyze the variation in $\hat{\delta}$ in greater detail by relating it to media data.

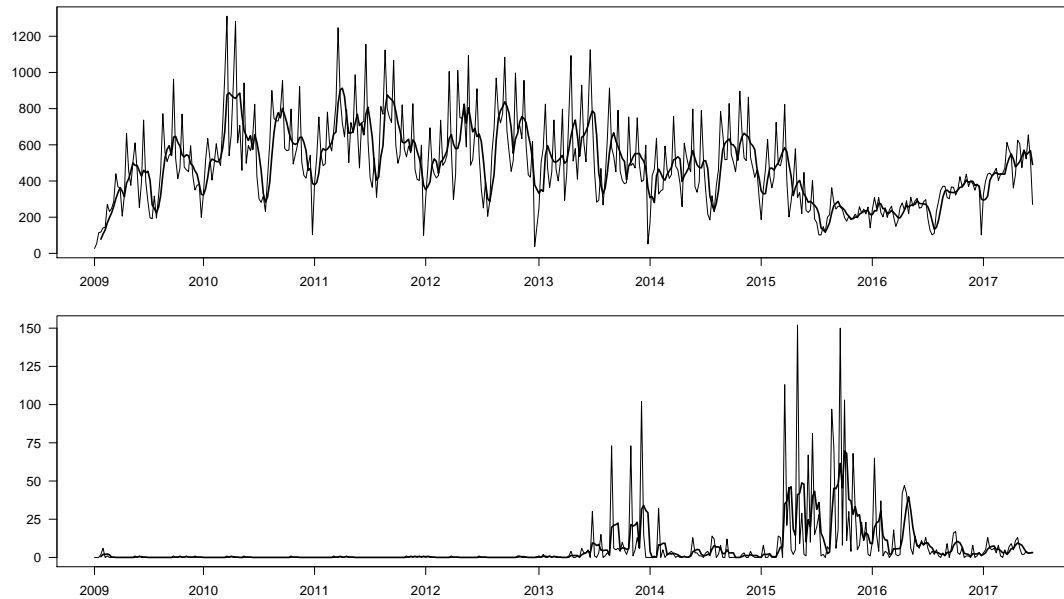
2.4 The influence of media

In this section we integrate the Danish media coverage of the HPV vaccine into the analysis. We obtained data on media coverage of the HPV vaccine from Infomedia, which is a searchable media database with comprehensive coverage of the Danish media. In order to gauge their effect on vaccine uptake, we focused on negative stories. Specifically, we obtained the weekly number

of media articles containing the keywords “HPV” and *bivirkning* (side effect) along with a list of excluding keywords that served to eliminate positive and irrelevant news stories. These are detailed in the supplementary material.

Figure 2.4 displays the weekly number of HPV1 vaccinations for girls born in 1997 or later (upper panel) and the weekly media count, denoted m_t (lower panel), along with the four-week moving averages for both variables. The weekly vaccination data in the upper panel of Figure 2.4 shows clear-cut seasonal effects, with a relatively low number of vaccinations during the summer and winter vacations. The vaccination data also display the pronounced end-of-month effects that existed prior to the introduction of the electronic vaccine registry on November 15, 2015. The lower part of Figure 2.4 shows that most of the media coverage occurred in 2015, starting in March 2015.

Figure 2.4. The upper panels show the weekly number of HPV1 vaccinations of girls born in 1997 or later, $\sum_c x_{c,t}$. The lower panel is the number of media stories, m_t , on HPV vaccine-related suspected side effects. The solid lines are four-week moving averages.



Most of the media articles that define m_t are merely reporting on alleged side effects or

commenting on the declining vaccine uptake as it occurred. Only a few articles can appropriately be labeled as “fake news,” for example, an article in *Metroxpress* on June 11, 2015, in which the headline read: “Doctors: One in 500 get seriously ill from the HPV vaccine.” Another example is the article in *Information* on May 30, 2016 that (contrary to scientific consensus) presented the view that “there is no scientific evidence that the HPV vaccine prevents cervical cancer.” Most articles were merely reporting on factual information, such as the declining vaccine uptake or the number of alleged side effects. An example is the article in *Berlingske* on August 31, 2015, “Now there are fewer being HPV vaccinated than MMR vaccinated,” in which it was speculated that stories about side effects might have caused the decline in HPV vaccinations. Another example is an article in *Ekstrabladet* on September 24, 2015: “More than 1,500 girls supposedly have HPV side effects.” This article reported that the total number of suspected HPV vaccine side effects had risen to 1,586. An extensive list of articles on this subject is presented in Table 2.D.3 in the appendix.

Below, we incorporate the media activity variable into the model, before turning our attention to a TV documentary that is referenced in many of the media stories after March 2015.

2.4.1 The effect of media coverage on vaccine compliance

In this section, we include the media variable, m_t , in the model to study whether some of the observed variation in vaccine compliance can be explained by media coverage of the topic. We achieve this by augmenting the score-driven model to include $\tilde{m}_t = \log(1 + m_t)$ as an explanatory variable in the dynamic equation for vaccine compliance, $\tilde{\delta}_{t+1} = \alpha_0 + \alpha_1 \tilde{\delta}_t + \alpha_2 \tilde{s}_t + \alpha_3 \tilde{m}_t$. The influence of media activity on vaccine compliance is measured by the parameter α_3 . If media activity were to reduce vaccine uptake, then we should expect $\alpha_3 < 0$, whereas if media activity had no effect on compliance then we should expect $\alpha_3 = 0$.

Estimating the augmented model by maximum likelihood yields:

$$\tilde{\delta}_{t+1} = \underset{(0.011)}{0.182} + \underset{(0.007)}{0.948}\tilde{\delta}_t + \underset{(0.006)}{0.050}\tilde{s}_t - \underset{(0.018)}{0.080}\tilde{m}_t, \quad (2.5)$$

where standard errors are given in parentheses below the estimates. The parameter of interest is estimated to be negative, $\hat{\alpha}_3 = -0.080$, and is significant. The significance of including the media variable in the model is also affirmed by the value of the log-likelihood function that increases from -20122 to -19954 . Additional estimates of this specification are presented in the first row of Table 2.2. The Table also reports the estimates for two alternative specifications, where the media variable is defined as, respectively, $\tilde{m}_t = m_t$ and $\tilde{m}_t = \sqrt{m_t}$. The complete set of parameter estimates for all specification are presented in Table 2.B.2 in the appendix. The qualitative results are the same for all specifications. The estimated coefficient for media

Table 2.2. Estimates for score-driven model with media variable

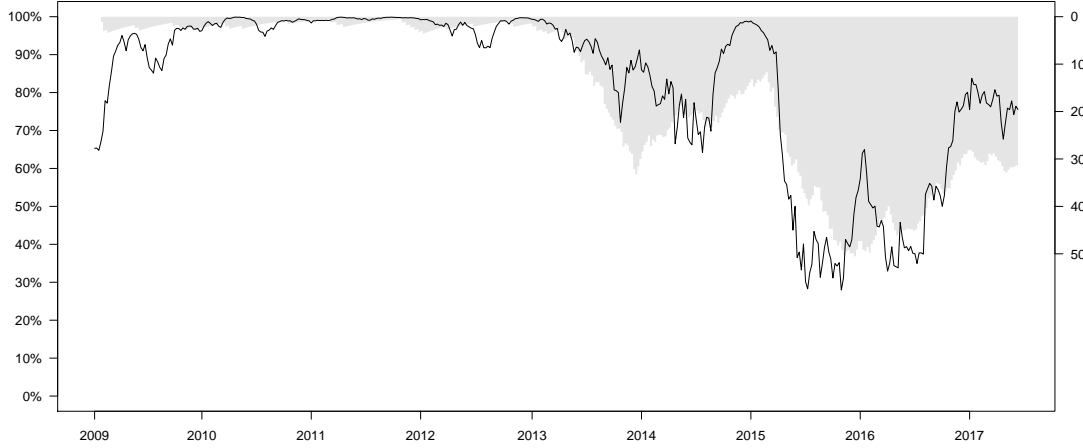
	$\hat{\alpha}_0$	$\hat{\alpha}_1$	$\hat{\alpha}_2$	$\hat{\alpha}_3$	$\hat{\beta}_0$	$\hat{\beta}_1$	$\ell(\hat{\theta})$
$\tilde{m}_t = \log(1 + m_t)$	0.182 (0.011)	0.948 (0.007)	0.050 (0.006)	-0.080 (0.018)	7.24 (0.019)	3.000 (0.003)	-19954
$\tilde{m}_t = m_t$	0.087 (0.003)	0.967 (0.014)	0.052 (0.012)	-0.004 (0.000)	7.24 (0.010)	3.000 (0.021)	-20033
$\tilde{m}_t = \sqrt{m_t}$	0.160 (0.007)	0.952 (0.004)	0.052 (0.012)	-0.045 (0.013)	7.24 (0.015)	3.000 (0.005)	-19971

Note: Parameter estimates and standard errors in parentheses, for the model $\tilde{\delta}_{t+1} = \alpha_0 + \alpha_1\tilde{\delta}_t + \alpha_2\tilde{s}_t + \alpha_3\tilde{m}_t$ for different specifications of \tilde{m}_t .

activity, $\hat{\alpha}_3$, is negative and significant in all cases. We focus on the results for the specification with $\tilde{m}_t = \log(1 + m_t)$ because it has the largest value of the log-likelihood function.

A detailed review of Danish media coverage of the HPV vaccine, detailed in the appendix, reveals that coverage was overwhelmingly positive until April 2013. The first article in mainstream media that associated the HPV vaccine with serious side effects was published in *Politiken*, a leading Danish newspaper, on April 17, 2013, which coincides with the first episode with rela-

Figure 2.5. Vaccine compliance in the model augmented with media coverage. The solid line (left axis) is the estimated vaccine compliance in the augmented score-driven model. The shaded area (right axis) is the exponentially weighted moving average of the media variable, m_t .



tively low values of δ_t . The article in *Politiken* featured a story on a girl presenting symptoms such as frequent headaches, dizziness, and tiredness, which her parents claimed were caused by the HPV vaccine. This article was followed by a series of others in the same newspaper that discussed the possibility of a link between the HPV vaccine and serious AE. An article in *Politiken* on May 3, 2013 raised doubt about the efficacy of the vaccine, and accused the Danish Health Authority (SST) for being “absolutely misleading” in their information about the HPV vaccine and cervical cancer.

In the model, (2.5), the media variable has a direct impact on $\tilde{\delta}_{t+1}$, and also a large indirect impact on $\tilde{\delta}_{t+2}$, $\tilde{\delta}_{t+3}$, etc., because the so-called *impulse response* function is given by $d\tilde{\delta}_{t+1}/d\tilde{m}_{t-j} = \alpha_3\alpha_1^j$, $j = 0, 1, \dots$, and α_1 is estimated to be close to 1. The aggregate media impact on $\tilde{\delta}_{t+1}$ is given by $\alpha_3 \sum_{j \geq 0} \alpha_1^j \tilde{m}_{t-j} = \alpha_3 M_t$ where $M_t = \sum_{j \geq 0} \alpha_1^j \tilde{m}_{t-j}$, which is an exponentially weighted average of past media activity. In Figure 2.5 we present the estimated time series of vaccine compliance, $\hat{\delta}_t$, and the aggregated media activity variable, M_t , based on the estimated model (2.5). The variation in $\hat{\delta}_t$ is similar to that in the model without the media variable, because $\hat{\delta}_t$ is primarily driven by the weekly number of vaccinations. The largest de-

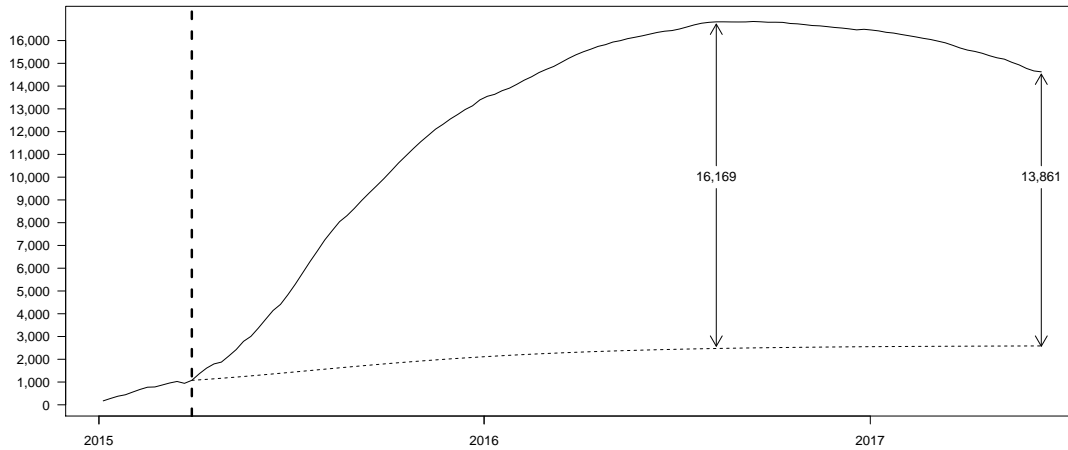
cline in vaccine compliance occurred in March 2015, which coincides with a large increase in the number of media articles that related the HPV vaccine to possible side effects. Many of these articles are related to the TV documentary that we focus on in the next section.

2.4.2 Vaccine uptake following the TV2 Documentary on March 26, 2015

On March 26, 2015, TV2 Denmark aired a documentary about the HPV vaccine. The program, which was entitled *De Vaccinerede Piger – Syge og Svigtede* (The Vaccinated Girls – Sick and Abandoned), presented personal and emotionally charged stories of girls who claimed that their illnesses were caused by the HPV vaccine. The documentary was viewed by nearly 10% of the population aged twelve and above (466,000 viewers). In a newspaper article, the TV2 editor responsible for the documentary acknowledged that it had contributed to the large decline in HPV vaccine uptake in 2015, see Sjöberg (2018). This view is supported by the raw vaccination data and our empirical analysis. Although our results cannot establish causality, the coincidence between the TV documentary and the drop in uptake represents a smoking gun in the absence of any good alternative explanation. The results based on the augmented model (2.5) show that the number of media stories significantly predicts the decline in vaccine uptake. Many of these media stories in 2015 were referencing the TV2 documentary. In fact, a media search on Infomedia reveals that the TV2 documentary was referenced 255 times in the Danish media during 2015. Of these references, 170 occurred before the end of April. At least forty-one of the references to the documentary were in stories produced by TV2. So, the spike in media activity, m_t , observed after March 2015 can be ascribed to the TV2 documentary.

The present framework enables us to compare actual vaccine uptake with counterfactual scenarios, such as one in which vaccine compliance had remained constant at the level it was at before the TV2 documentary aired. This is illustrated in Figure 2.6, in which the solid line shows

Figure 2.6. Missing vaccinations for girls born in 2003. The solid line displays the number of unvaccinated girls, born in 2003, relative to a hypothetical baseline with 100% compliance. The dotted line is the corresponding number of missing vaccinations had compliance been stable at 95%. The difference between the two lines is the additional number of unvaccinated girls resulting from the sharp decline in vaccine uptake starting in late March 2015.



the difference between the expected number of vaccinated girls under full compliance ($\delta = 1$) and the actual number of vaccinated girls (i.e., $\Lambda(a_t^c) \times N_c$ minus $X_{c,t}$) for girls born in 2003. The vertical line in early 2015 is the time at which the TV2 documentary was aired, and we observed a distinct break in the number of unvaccinated girls. The dotted line presents the corresponding number of unvaccinated girls in the hypothetical scenario in which δ_t remained constant at the value it had by the end of March 2015 (95%). Under the hypothetical scenario, we would have expected about 2,000 unvaccinated girls (born in 2003) by the end of our sample period. The actual number turned out to be substantially larger, and the discrepancy between the solid line and the dotted line shows the reduction in girls who were vaccinated. At the peak there were more than 16,000 additional unvaccinated girls in cohort 2003 alone. Some of these girls were vaccinated later, so that the additional number of unvaccinated girls, born in 2003, is over 13,800 by the end of our sample period (June 2017).

The implications of 13,800 unvaccinated girls can be estimated as follows. Annually, in Denmark, there are about 375 new cases of cervical cancer and about 100 deaths caused by the

disease. Moreover, about 6,000 cone biopsies are carried out annually as a preventive measure for cervical cancer. These Figures may be compared with a cohort size of 36,737 women (the average cohort size for women aged 35-49, the cohort with the highest incidence of cervical cancer). The HPV vaccines that were offered in the Danish vaccine program during our sample period protect against the HPV virus types 16 and 18, which are responsible for about 70% of the cases of cervical cancer. So, if we assume that the vaccine is 100% effective against HPV virus types 16 and 18, then 13,800 unvaccinated girls will translate into about 99 preventable cases of cervical cancer and 26 preventable deaths. Moreover, if we make the (quite conservative) assumption that HPV virus 16 and 18 are the cause of 50% of abnormal cell changes that are removed by a cone biopsy, then 13,800 unvaccinated girls translate into 1,127 preventable cone biopsies.

2.4.3 Some relevant international comparisons

We have established that Danish media coverage of the HPV vaccine and suspected side effects is a significant predictor of declining vaccine uptake in Denmark. This is further bolstered by the fact that the HPV vaccine programs in the neighboring countries Sweden and Norway did not see much variation in uptake. A recent study by Amdisen et al. (2018) compared the Danish HPV vaccine uptake for birth year cohorts 1998-2000 with that of cohorts 2001-2003. Interestingly, they found that the decline in vaccine uptake was significantly smaller for immigrants, which is consistent with Danish media coverage being a key determinant for the decline in HPV vaccine uptake.

It could be hypothesized that Danish media were merely reporting on an unusually high number of suspected side effects. This hypothesis is, however, not consistent with the data. We have obtained the monthly number of AE reports for Denmark and annual Figures for the same from Norway. It is important to note that an AE can be self-reported and does not establish that the reported symptoms were caused by the vaccine. As of 2018, only three cases were

deemed sufficiently plausible to justify compensation. Before 2015, the number of AE reports per 10,000 doses was similar for Denmark (between three and seventeen) and Norway (between eight and seventeen). In 2015, this statistic rose to 153 in Denmark, although nothing out of the ordinary was observed in Norway, see Table 2.3. From the monthly data, shown in the appendix, it is also clear that the high number of AE reports in 2015 pertains to the period after the TV2 documentary aired, not the period before. There was a total of 822 AE reports in Denmark in 2015, of which 11 and 15 were reported in January and February, respectively. In the following month, when the TV2 documentary first aired, this figure jumped to 53, then to 70 in April, before it peaked at 150 in May of 2015. The timing suggests that increased levels of media coverage preceded increased reporting of AE, similar to the case of New Zealand, see Faasse et al. (2017). Therefore, it supports the view that the high number of AE reports in 2015 is due to a hesitancy-induced false association between vaccinations and causally unrelated symptoms, rather than a localized occurrence of theretofore unrecognized actual side effects. In Appendix 2.D, we also investigate Google Search activity related to vaccine side effects in Denmark and Norway.

Table 2.3. Media coverage and number of AE reports

Year	Media Count	AE	AE per 10k doses	AE per 10k Norway
2010	4	67	13	17
2011	8	43	3	12
2012	5	95	3	11
2013	415	512	10	8
2014	140	192	17	11
2015	1329	822	153	12
2016	489	307	109	10

Note: Danish annual Figures on: HPV vaccine related newspaper articles; AE reported for the HPV vaccine; and AE per 10,000 doses of HPV vaccine given. Data retrieved from Lægemiddelstyrelsen (2016) and Danish Health Authority (2017). For comparison, we include the number of AE per 10,000 doses of HPV vaccine in Norway, based on an estimated 76,000 doses per year.

There is evidence that the TV2 documentary influenced vaccine uptake beyond Danish borders. An Irish documentary, *Cervical Cancer Vaccine - Is it safe?*, aired on December 14, 2015

on the channel TV3 in Ireland. It included many segments from the Danish documentary with English subtitles, and HPV vaccine compliance fell from 89.7% for the cohort vaccinated before the documentary aired to 55.8% for the cohort vaccinated afterwards. In addition, TV2 Denmark made their documentary available on YouTube, on which subtitles in various languages were added in due course. The decision to make the documentary freely available on YouTube was uncharacteristic for TV2 Denmark, because the station typically keeps its contents behind a paywall.

It is interesting to compare the Danish media coverage with a similar episode in the United States. In 2013 the TV network, CBS, aired a controversial TV program on the HPV vaccine in 2013, to which US media responded very differently from that in Denmark. On December 4, 2013, the CBS TV talk show *Katie* covered what the host referred to as the “HPV vaccine controversy.” Similar to the TV2 documentary, the program presented emotionally charged and personal stories of individuals who claimed to have been injured by the HPV vaccine, and only briefly mentioned the scientific consensus on the matter. The reaction from the US media was prompt with scolding criticism. On the day the program was aired, several media outlets criticized it in articles with castigating headlines such as: “Katie Couric Hands Her Show Over to Anti-Vaccination Alarmists” (*Slate*), “Katie Couric puts the anti-vaccination movement into the mainstream” (*LA Times*), and “Is Katie Couric the Next Jenny McCarthy?” (*TIME*). The *TIME* article concludes with: “Couric’s misdeeds are all the worse given that she’s taken much more seriously than [...]” Shortly after, on December 10, 2013, the TV host apologized and conceded that her program had been “too anti-vaccine and anti-science.” The instant media criticism in the United States and Katie Couric’s prompt apology may have averted a subsequent decline in HPV vaccine uptake in the United States, similar to the one observed in Denmark in 2015. In contrast, it was only after the TV2 documentary had been nominated for a prestigious journalistic prize, nine month later, that Danish media expressed the first criticism of it.

2.5 Summary and discussion

Vaccination is arguably one of the most important public health achievements in history. Critically, however, its success relies on the personal decisions to comply with recommended vaccine schedules. Increasingly, vaccine refusal is becoming a problem in many countries, as exemplified by ongoing outbreaks of measles in the United States and several European countries. Understanding the mechanisms that determine vaccine hesitancy is, therefore, of great importance to health authorities, including the influence that media have in the personal decision to comply with, or deviate from, recommended vaccine schedules.

In this chapter we have developed a dynamic model for vaccine compliance that is driven by discrepancies between expected and actual vaccination rate. The model could serve as a tool for health authorities to monitor vaccine hesitancy in real time. We applied the model to Danish HPV vaccination data, which has experienced a great deal of variation in vaccine uptake since the vaccine was introduced into the Danish childhood vaccination program in 2009. The econometric model we have proposed in this chapter is the first score-driven model for binomially distributed variables. The model could be adapted to other time series involving aggregated binary outcome variables and is, therefore, not specific to vaccine data.

Our empirical analysis supports the view that Danish media played an important role in the collapse of the Danish HPV vaccine program. Our results show that the first decline in HPV vaccine uptake coincides with the period in which Danish media began running stories that associated the HPV vaccine with serious side effects, and that the largest decline in vaccine uptake occurred immediately after a vaccine-critical TV documentary. The evidence that media coverage influenced vaccine uptake is strengthened further by the empirical results from the augmented specification that includes a media coverage variable. For this specification, we find that the media variable is a significant predictor of HPV vaccine uptake. The larger the number

of media stories that associate the HPV vaccine with side effects, the lower is the expected HPV1 vaccine uptake in subsequent weeks. The empirical evidence points to the TV2 documentary as the main culprit with regard to the sharp decline in vaccine uptake in 2015. This is primarily because the sharp decline in HPV vaccinations occurred immediately after the documentary aired, and because there is no good alternative explanation for the decline to have occurred at that particular point in time. It is also consistent with the empirical results from the augmented specification, because a very large number (255) of media stories in 2015 referenced the TV2 documentary, including 41 stories produced by TV2 Denmark. In February 2018, TV2 Denmark acknowledged that their documentary contributed to the failure of the HPV vaccination program, but emphasized that this had not been its intention.

The model framework introduced in this chapter makes it possible to quantify the effect that the decline in vaccine compliance has had on the vaccination program. We estimate that more than 16,000 girls born in 2003 delayed HPV vaccination because of the sharp decline in vaccine uptake after March 2015. Many of these girls have since been vaccinated. On May 10, 2017, the Danish Health Authority and the Danish Cancer Society started a campaign entitled “Stop HPV,” in an attempt to reinvigorate the HPV vaccine program. The latest data, as of March 2019, show that the campaign has partially succeeded in increasing vaccine uptake. For example, 37% of cohort 2005 received HPV1 during 2017 (the year they turned twelve), and 46% of cohort 2006 received HPV1 during 2018. These figures compare favorably to 30% and 25%, which were the corresponding percentages for cohorts 2003 and 2004, respectively. However, they fall well short of the vaccine uptake before 2013. For comparison, about 75% of the girls born in 1999 and 2000 were HPV1 vaccinated during the year they turned twelve. The campaign has also had a positive effect on cohort 2003, of which 80% have been HPV1 vaccinated as of March 2019. However, this figure is also well short of the coverage for older cohorts, which is 94% for girls born in 1998, 1999, or 2000.

Our empirical analysis of Danish HPV vaccination data is based on anonymized data, which limits a deeper analysis of the individuals who declined HPV vaccination. It would be interesting to investigate whether media effects vary with socio-economic characteristics. Following the infamous (and retracted) study that led the public to suspect a relationship between the MMR vaccine and autism, vaccine compliance fell throughout the United Kingdom (and elsewhere). Interestingly, Anderberg, Chevalier, and Wadsworth (2011) found that the decline in MMR vaccine compliance was more pronounced in areas with a higher number of educated individuals and a higher average income. It would also be interesting to investigate how prior knowledge about vaccines affects sensitivity with respect to media stories. In an online experiment with French voters, Barrera Rodriguez et al. (2018) found that respondents who had accurate beliefs about immigration statistics were not misled by fake news, whereas those with incorrect priors often were. Finally, social media may also have played an important role in the take-up or not of the HPV vaccine; however, there is mixed evidence as to its influence. For instance, Allcott and Gentzkow (2017) observed that survey respondents were as likely to believe in fake stories that had circulated on social media, as they were likely to believe in fake stories that had not. In the case of Danish HPV vaccination, social media likely played some role in the decline in HPV vaccine uptake and, in fact, social media were a catalyst for much of the media coverage. Links to the TV2 documentary (on YouTube) and articles that related the HPV vaccine to serious side effects were frequently shared on social media. We leave an investigation into the role of social media and HPV vaccinations for future research.

Appendix to Chapter 2

2.A Derivations of various results

2.A.1 Score for vaccine compliance

In this section we establish the result in (2.4). We seek the first and second derivatives of the log-likelihood function with respect to $\tilde{\delta}$. First observe that

$$\frac{\partial p_{c,t}}{\partial \tilde{\delta}_t} = \frac{\partial}{\partial \tilde{\delta}_t} \frac{e^{\tilde{\delta}_t}}{1+e^{\tilde{\delta}_t}} \lambda_{c,t} \eta_t = \frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} \lambda_{c,t} \eta_t,$$

where $\lambda_{c,t} = \frac{N_c}{N_{c,t-1}} [\Lambda(\beta; a_t^c) - \Lambda(\beta; a_{t-1}^c)]$. Thus from (2.2) we have that

$$s_{c,t} = \frac{\partial \ell_{c,t}}{\partial \tilde{\delta}_t} = \frac{\partial p_{c,t}}{\partial \tilde{\delta}_t} \left[\frac{x_{c,t}}{p_{c,t}} - \frac{N_{c,t-1} - x_{c,t}}{1-p_{c,t}} \right] = \frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} \lambda_{c,t} \eta_t N_{c,t-1} \left[\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right],$$

where $\hat{p}_{c,t} = x_{c,t}/N_{c,t-1}$. The score for $\tilde{\delta}_t$ is therefore given by

$$s_t = \sum_c s_{c,t} = \frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} \sum_c \lambda_{c,t} \eta_t N_{c,t-1} \left[\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right].$$

Next, for the second derivative, we have

$$h_{c,t} = \frac{\partial^2 \ell_{c,t}}{\partial \tilde{\delta}_t^2} = \frac{\partial}{\partial \tilde{\delta}_t} \frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} \lambda_{c,t} \eta_t N_{c,t-1} \left[\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right].$$

Now

$$\frac{\partial}{\partial \tilde{\delta}_t} \frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} = \frac{e^{\tilde{\delta}_t} (1+e^{\tilde{\delta}_t})^2 - e^{\tilde{\delta}_t} e^{\tilde{\delta}_t} 2(1+e^{\tilde{\delta}_t})}{(1+e^{\tilde{\delta}_t})^4} = \frac{-e^{3\tilde{\delta}_t} + e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^4} = \frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} \frac{1-e^{\tilde{\delta}_t}}{1+e^{\tilde{\delta}_t}},$$

and $\frac{\partial}{\partial \tilde{\delta}_t} \left[\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right] = -\frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} \lambda_{c,t} \eta_t \left[\frac{\hat{p}_{c,t}}{p_{c,t}^2} + \frac{1-\hat{p}_{c,t}}{(1-p_{c,t})^2} \right]$. Using that $\mathbb{E}_{t-1}(\hat{p}_{c,t}) = p_{c,t}$, we have

$$-\mathbb{E}_{t-1} h_{c,t} = \frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} \lambda_{c,t} \eta_t N_{c,t-1} \frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} \lambda_{c,t} \eta_t \frac{1}{p_{c,t}(1-p_{c,t})} = \left[\frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} \right]^2 \frac{\lambda_{c,t}^2 \eta_t^2 N_{c,t-1}}{p_{c,t}(1-p_{c,t})},$$

hence

$$\left(-\sum_c \mathbb{E}_{t-1} h_{c,t} \right)^{-1/2} = \left(\left[\frac{e^{\tilde{\delta}_t}}{(1+e^{\tilde{\delta}_t})^2} \right]^2 \sum_c \frac{\lambda_{c,t}^2 \eta_t^2 N_{c,t-1}}{p_{c,t}(1-p_{c,t})} \right)^{-1/2} = \frac{(1+e^{\tilde{\delta}_t})^2}{e^{\tilde{\delta}_t}} / \sqrt{\sum_c \frac{\lambda_{c,t}^2 \eta_t^2 N_{c,t-1}}{p_{c,t}(1-p_{c,t})}}.$$

Thus $\tilde{s}_t = (-\sum_c \mathbb{E}_{t-1} h_{c,t})^{-1/2} \sum_c s_{c,t}$ equals

$$\frac{\sum_c \lambda_{c,t} \eta_t N_{c,t-1} \left(\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right)}{\sqrt{\sum_c \frac{\lambda_{c,t}^2 \eta_t^2 N_{c,t-1}}{p_{c,t}(1-p_{c,t})}}} = \frac{\sum_c N_c \tilde{\lambda}_{c,t} \left(\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right)}{\sqrt{\sum_c \frac{N_c^2 \tilde{\lambda}_{c,t}^2}}{\tilde{\lambda}_{c,t}^2}} \approx \frac{\sum_c \tilde{\lambda}_{c,t} \left(\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right)}{\sqrt{\sum_c \frac{1}{N_{c,t-1} p_{c,t}(1-p_{c,t})} \tilde{\lambda}_{c,t}^2}},$$

where $\tilde{\lambda}_{c,t} = \Lambda(\beta; a_t^c) - \Lambda(\beta; a_{t-1}^c)$.

2.B Model of seasonal effects

In this appendix we detail the part of the model that accounts for the seasonal effect in the weekly vaccination data, and present the corresponding empirical results.

2.B.1 Score for seasonal component

We model seasonal effects by enhancing the model for $p_{c,t}$ with a third component. The binomial parameter is now decomposed as

$$p_{c,t}(\theta) = \delta_t(\alpha)\lambda_{c,t}(\beta)\eta_t(\gamma),$$

so that the parameter vector is given by $\theta = (\alpha', \beta', \gamma)'$.

To account for time variation in seasonality, we model $\tilde{\eta} = \log(\eta)$ by a separate score-driven model

$$\tilde{\eta}_t = g_{0,t} \sin(2\pi(z_t^m + g_{1,t}))(1 - z_t^a) + \gamma_2 z_t^a,$$

where $g_{i,t} = g_{i,t-1} + \gamma_i \tilde{s}_{g,i,t-1}$ for $i = 0, 1$. Analogous to (2.4), $\tilde{s}_{g,i,t-1}$ is the scaled score with respect to the seasonality parameters $g_{0,t}$ and $g_{1,t}$. Specifically,

$$\tilde{s}_{g,0,t} = \tilde{s}_t \text{sign}(\sin(2\pi(z_t^m + g_{1,t}))) (1 - z_t^a) \quad (2.6)$$

and

$$\tilde{s}_{g,1,t} = \tilde{s}_t \text{sign}(g_{0,t} \cos(2\pi(z_t^m + g_{1,t}))) (1 - z_t^a). \quad (2.7)$$

The expressions for (2.6) and (2.7) are derived below. In the estimation of the model, we treat the initial values for $(g_{0,0}, g_{1,0}) = (\gamma_3, \gamma_4)$ as free parameters, with domains $\gamma_3 \geq 0$ and $\gamma_4 \in [0, 1]$, respectively.

The seasonal variables, z_t^m and z_t^a , are defined as follows. First, z_t^m represents the location of week t in the month, as defined by the date of the Monday of that week, divided by the number of days in the months. For example, a week with a Monday on January 12 translates

into $z_t^m = 12/31$. Second, z_t^a is a binary variable that takes the value one during the summer vacation period (week numbers 28 to 31) as well as the two weeks around Christmas/New Year (week numbers 52 and 1).

Next, we derive (2.6) and (2.7) for the case $z_t^a = 0$. We seek the first and second derivatives of the log-likelihood function with respect to $g_{0,t}$ and $g_{1,t}$. First, note that $\frac{\partial p_{c,t}}{\partial g_{0,t}} = p_{c,t} \sin(2\pi(z_t^m + g_{1,t}))$ and $\frac{\partial p_{c,t}}{\partial g_{1,t}} = p_{c,t} g_{0,t} 2\pi \cos(2\pi(z_t^m + g_{1,t}))$. From (2.2) we have that

$$s_{g,1,c,t} = \frac{\partial \ell_{c,t}}{\partial g_{0,t}} = \frac{\partial p_{c,t}}{\partial g_{0,t}} \left[\frac{x_{c,t}}{p_{c,t}} - \frac{N_{c,t-1} - x_{c,t}}{1-p_{c,t}} \right] = p_{c,t} N_{c,t-1} \left[\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right] \sin(2\pi(z_t^m + g_{1,t}))$$

and $s_{g,2,c,t} = \frac{\partial \ell_{c,t}}{\partial g_{1,t}} = p_{c,t} N_{c,t-1} \left[\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right] g_{0,t} 2\pi \cos(2\pi(z_t^m + g_{1,t}))$. For the second derivatives, note that $\frac{\partial}{\partial g_{0,t}} \left[\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right] = -p_{c,t} \left[\frac{\hat{p}_{c,t}}{p_{c,t}^2} + \frac{1-\hat{p}_{c,t}}{(1-p_{c,t})^2} \right] \sin(2\pi(z_t^m + g_{1,t}))$ and

$$\frac{\partial}{\partial g_{1,t}} \left[\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right] = -p_{c,t} \left[\frac{\hat{p}_{c,t}}{p_{c,t}^2} + \frac{1-\hat{p}_{c,t}}{(1-p_{c,t})^2} \right] g_{0,t} 2\pi \cos(2\pi(z_t^m + g_{1,t})).$$

Because $\mathbb{E}_{t-1}(\hat{p}_{c,t}) = p_{c,t}$, we therefore have that

$$-\mathbb{E}_{t-1} \frac{\partial^2 \ell_{c,t}}{\partial g_{0,t}^2} = N_{c,t-1} \frac{p_{c,t}^2}{p_{c,t}(1-p_{c,t})} \sin^2(2\pi(z_t^m + g_{1,t}))$$

and

$$-\mathbb{E}_{t-1} \frac{\partial^2 \ell_{c,t}}{\partial g_{1,t}^2} = N_{c,t-1} \frac{p_{c,t}^2}{p_{c,t}(1-p_{c,t})} g_{0,t}^2 4\pi^2 \cos^2(2\pi(z_t^m + g_{1,t})).$$

Hence,

$$\left(-\sum_c \mathbb{E}_{t-1} \frac{\partial^2 \ell_{c,t}}{\partial g_{0,t}^2} \right)^{-1/2} = \left(\sin^2(2\pi(z_t^m + g_{1,t})) \sum_c \frac{p_{c,t}^2 N_{c,t-1}}{p_{c,t}(1-p_{c,t})} \right)^{-1/2}$$

and

$$\left(-\sum_c \mathbb{E}_{t-1} \frac{\partial^2 \ell_{c,t}}{\partial g_{1,t}^2} \right)^{-1/2} = \left(g_{0,t}^2 \cos^2(2\pi(z_t^m + g_{1,t})) \sum_c \frac{p_{c,t}^2 N_{c,t-1}}{p_{c,t}(1-p_{c,t})} \right)^{-1/2}.$$

Finally,

$$\begin{aligned}\tilde{s}_{g,0,t} &= \left(- \sum_c \mathbb{E}_{t-1} \frac{\partial^2 \ell_{c,t}}{\partial g_{0,t}^2} \right)^{-1/2} \sum_c s_{g,1,c,t} = \frac{\sum_c \lambda_{c,t} N_{c,t-1} \left[\frac{\hat{p}_{c,t}}{p_{c,t}} - \frac{1-\hat{p}_{c,t}}{1-p_{c,t}} \right] \sin(2\pi(z_t^m + g_{1,t}))}{\sqrt{\sin^2(2\pi(z_t^m + g_{1,t})) \sum_c \frac{\lambda_{c,t}^2 N_{c,t-1}}{p_{c,t}(1-p_{c,t})}}} \\ &= \tilde{s}_t \text{sign}(\sin(2\pi(z_t^m + g_{1,t}))),\end{aligned}$$

and, similarly, $\tilde{s}_{g,1,t} = \tilde{s}_t \text{sign}(g_{0,t} \cos(2\pi(z_t^m + g_{1,t})))$. In the case where $z_t^a = 1$, we define

$$\tilde{s}_{g,0,t} = \tilde{s}_{g,1,t} = 0.$$

2.B.2 Empirical results related to seasonal effects

Table 2.B.1. Estimates for score-driven model

$\hat{\alpha}_0$	$\hat{\alpha}_1$	$\hat{\alpha}_2$	$\hat{\delta}_0$	$\hat{\beta}_0$	$\hat{\beta}_1$	$\hat{\gamma}_0$	$\hat{\gamma}_1$	$\hat{\gamma}_2$	$\hat{\gamma}_3$	$\hat{\gamma}_4$	$\ell(\hat{\theta})$
0.000 (0.087)	0.992 (0.022)	0.054 (0.017)	0.939 (0.005)	7.25 (0.038)	3.03 (0.088)	0.001 (0.007)	0.002 (0.022)	-0.757 (0.087)	0.246 (0.042)	0.384 (0.081)	-20122
0	1	0.066 (0.004)	0.883 (0.000)	7.24 (0.018)	3.01 (0.003)	0.001 (0)	0.002 (0.000)	-0.679 (0.008)	0.285 (0.011)	0.465 (0.010)	-20318

Note: Standard errors in parentheses.

Table 2.B.2. Estimates for score-driven model with media variable

	$\hat{\alpha}_0$	$\hat{\alpha}_1$	$\hat{\alpha}_2$	$\hat{\alpha}_3$	$\hat{\delta}_0$	$\hat{\beta}_0$	$\hat{\beta}_1$	$\hat{\gamma}_0$	$\hat{\gamma}_1$	$\hat{\gamma}_2$	$\hat{\gamma}_3$	$\hat{\gamma}_4$	$\ell(\hat{\theta})$
(1)	0.182 (0.011)	0.948 (0.007)	0.050 (0.006)	-0.080 (0.018)	0.668 (0.004)	7.24 (0.019)	3.000 (0.003)	0.001 (0.000)	0.002 (0.000)	-0.755 (0.006)	0.304 (0.010)	0.488 (0.008)	-19951
(2)	0.087 (0.003)	0.967 (0.014)	0.052 (0.012)	-0.004 (0.000)	0.766 (0.003)	7.24 (0.010)	3.000 (0.021)	0.001 (0.000)	0.002 (0.000)	-0.756 (0.018)	0.287 (0.013)	0.491 (0.004)	-20033
(3)	0.160 (0.007)	0.952 (0.004)	0.052 (0.012)	-0.045 (0.013)	0.685 (0.003)	7.24 (0.015)	3.000 (0.005)	0.001 (0.004)	0.002 (0.000)	-0.753 (0.005)	0.284 (0.008)	0.489 (0.006)	-19971

Note: Standard errors in parentheses.

2.C Additional empirical results and details

2.C.1 Estimation of Figure 1

We set the estimate of the static parameter δ to match X_T/N_0 , that is, 92%. The estimate of $\Lambda(t)$ is determined by minimizing the discretized version of the Anderson-Darling statistic

$$\int_0^T \frac{(X_t/N_0 - \hat{\delta}\Lambda(t))^2}{\Lambda(t)(1 - \Lambda(t))} d\Lambda(t).$$

2.C.2 Birthday correction

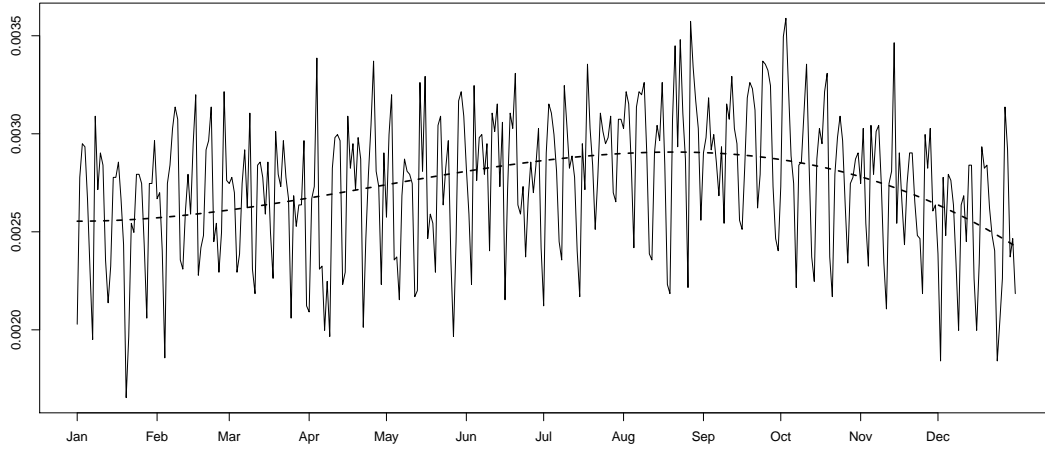
In Denmark, the HPV vaccine is given free when a female adolescent turns twelve. Therefore, the number of girls eligible for their first vaccine dose (HPV1) increases gradually over the year in which the cohort turns twelve. We adapt the definition of $N_{c,t}$ accordingly. We do not have information about the birthday of individuals in our anonymized data, but we can approximate the distribution of birthdays over the year for each cohort. Monthly birth statistics show that the birth distribution over the calendar year is almost identical across cohorts. Therefore, we estimate $g(a)$ using daily births for 2007. These data were obtained from Statistics Denmark.

Figure 2.C.1 plots daily births as a fraction of the total number of births over the year. We fit a fourth-order polynomial $b(\tau)$ to the fraction of daily births by least squares, where $\tau = \frac{i}{365}$, $i = 1, \dots, 365$ denotes the time of the year. The fitted values are drawn as a dotted line in the Figure. The daily measure of births is quite noisy and exhibits seasonal effects at the weekly and monthly frequencies. Still, the estimated function predicts a higher number of births in late summer and few births at the end of December, which is in line with stylized facts from other countries. The cumulative number of birthdays is given by $B(\tau) = \int_0^\tau b(u)du$, so that the fraction of girls in this who have turned twelve at time a_i^c is given by

$$g(a) = \frac{1}{B(1)}B(3a), \quad a \in (0, \frac{1}{3}],$$

with $g(a) = 0$ for $a \leq 0$ and $g(a) = 1$ for $a \geq \frac{1}{3}$. The number of girls in cohort c , which is eligible to receive HPV1 by the end of week t is given by the girls who have turned twelve, less those

Figure 2.C.1. Daily births as a fraction of total births in 2007



that have already received HPV1, that is

$$N_{c,t} = g(a_t^c)N_c - X_{c,t}.$$

It is worth mentioning that this time-of-birth correction has only a negligible effect on the estimation results. In fact, we also estimated the model using $g(a) = 1$ for $a \in [0, 1]$, which assumes all girls are eligible for the vaccine when $a = 0$, as well as with $g(a) = \min(3a, 1)$, which assumes an even distribution of births over the year. The alternative specifications for g did not change the empirical results in any substantive way.

2.C.3 Simple specifications for $\delta_t(\alpha)$

In this section we describe simple models for $\delta_t(\alpha)$. We use two simple approaches: (1) δ_t is piecewise constant, but subject to structural changes; (2) time-variation in δ_t is given by a deterministic function. In the first approach, the change-points are unknown parameters that are to be estimated from the data. Thus, α represents the (vector) of change point(s) during the

sample period and the values that δ_t takes before, between, and after change points. The second approach takes δ_t to be a simple deterministic function of time.

As explained in the main part of the chapter, we need to accommodate seasonality at both annual and monthly frequencies. For the simple specifications, we model seasonality as

$$\tilde{\eta}_t = \gamma_0 \sin(2\pi(z_t^m + \gamma_1))(1 - z_t^a) + \gamma_2 z_t^a,$$

where z_t^m , z_t^{sum} and z_t^{win} are the seasonal indicators described in the paper.

We estimated the piecewise constant model with one, two, three, and four breakpoints, and we consider the following four different deterministic specifications where δ is a simple function of t : hinge functions, Hermite polynomials, trigonometric polynomials, and natural cubic splines. Table 2.C.1 gives the exact expressions for the different specifications. The hinge function approximation is piecewise linear and the free parameters are the slopes and the knots at which the slope changes. The natural cubic spline basis function is cubic between knots and linear beyond boundary knots with knots placed at equally spaced intervals. For each function, we choose a flexible specification: the hinge model, the trigonometric polynomial and the natural cubic spline model each allow for nine free parameters in α . In the estimation of the Hermite polynomial model, four turned out to be the maximum order that was numerically feasible. Table 2.C.2 presents the estimates of all eight simple specifications for δ_t .

Figure 2.C.2 plots the estimated vaccine compliance, $\hat{\delta}_t$, for the different piecewise constant specifications. All models detect a break in the last week of March or the first week of April 2015. At that change point, compliance drops dramatically by forty to fifty percentage points. The models that allow for a second change point determine it in the first week of October 2016. At that second change point, compliance improves to about 70%. The models with three and four change points also detect a break in late 2013, when compliance falls from close to 100%.

The model with four break points also finds a break in late 2014, when compliance rises again to close to 100%. Estimates for $\hat{\delta}_t$ for the different approximations to deterministic functions are plotted in Figure 2.C.3. The estimated compliance paths all have similar shapes to the one for the model with three break points. The trigonometric polynomial model, however, also detects an initial uptake of compliance early in the sample.

Although these simple specifications give some insight into the dynamics of vaccine compliance in the data, the score-driven model has many advantages. First, for the simple specifications, the path of vaccine compliance depends on the arbitrary choice of basis function. Second, the simple specifications do not allow for dynamic updating of the compliance parameter. Third, the score-driven model fits the data (in terms of the log-likelihood) better, even though the score-driven model has fewer free parameters.

Table 2.C.1. Overview of simple specifications

Model	Specification
(1) One break	$\tilde{\delta}_t = \alpha_0 \mathbb{1}_{t < \alpha_2} + \alpha_1 \mathbb{1}_{\alpha_2 \leq t}$
(2) Two breaks	$\tilde{\delta}_t = \alpha_0 \mathbb{1}_{t < \alpha_2} + \alpha_1 \mathbb{1}_{\alpha_2 \leq t < \alpha_4} + \alpha_3 \mathbb{1}_{\alpha_4 \leq t}$
(3) Three breaks	$\tilde{\delta}_t = \alpha_0 \mathbb{1}_{t < \alpha_2} + \alpha_1 \mathbb{1}_{\alpha_2 \leq t < \alpha_4} + \alpha_3 \mathbb{1}_{\alpha_4 \leq t < \alpha_6} + \alpha_5 \mathbb{1}_{\alpha_6 \leq t}$
(4) Four breaks	$\tilde{\delta}_t = \alpha_0 \mathbb{1}_{t < \alpha_2} + \alpha_1 \mathbb{1}_{\alpha_2 \leq t < \alpha_4} + \alpha_3 \mathbb{1}_{\alpha_4 \leq t < \alpha_6} + \alpha_5 \mathbb{1}_{\alpha_6 \leq t < \alpha_8} + \alpha_7 \mathbb{1}_{\alpha_8 \leq t}$
(5) Hinge basis	$\tilde{\delta}_t = \alpha_0 + \sum_{j=1}^4 \alpha_{2j-1} \max(0, t - \alpha_{2j})$
(6) Hermite polynomial	$\tilde{\delta}_t = \alpha_0 + \alpha_1 \text{He}_1(t) + \alpha_2 \text{He}_2(t) + \alpha_3 \text{He}_3(t) + \alpha_4 \text{He}_4(t)$
(7) Trigonometric polynomial	$\tilde{\delta}_t = \alpha_0 + \sum_{j=1}^2 (\alpha_{2j-1} \cos(2\pi \times j \times t/T) + \alpha_{2j} \sin(2\pi \times j \times t/T))$
(8) Natural cubic spline	$\tilde{\delta}_t = \alpha_0 + \sum_{j=1}^8 \alpha_j B_j(t)$

Note: $\text{He}_j(t)$ refers to the j 'th Hermite polynomial in t .

$B_j(t)$ refers to the j 'th natural B-spline basis function in t . Knots of the spline are placed at equally spaced intervals.

2.C.4 Simple specifications for $\delta_t(\alpha)$ without monthly seasonality correction

In this section we document estimates for the simple specifications for $\delta_t(\alpha)$ that do not control for seasonality on a monthly basis. We include these results to demonstrate that the monthly

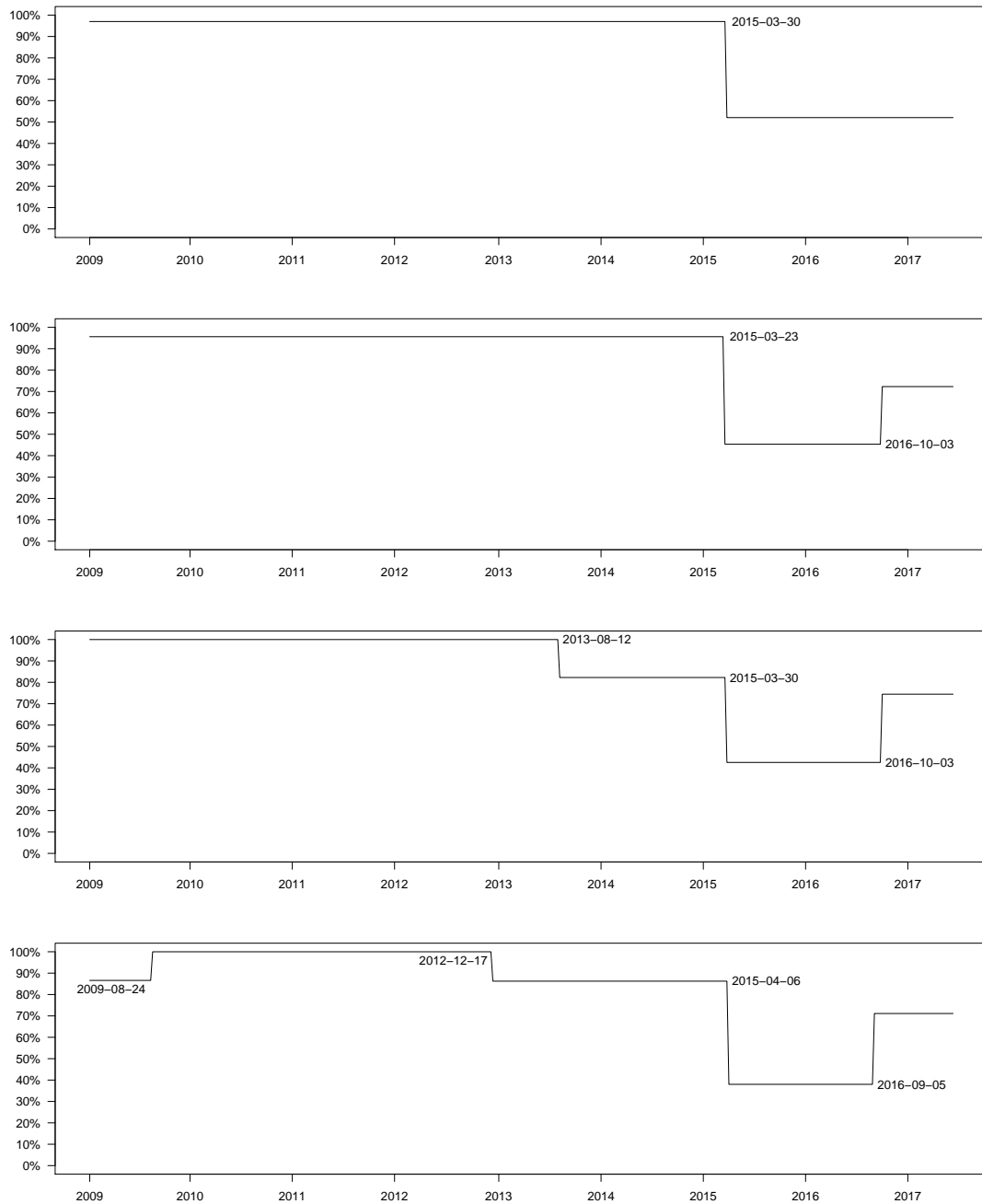
Figure 2.C.2. Models with structural changes in vaccine compliance, δ 

Figure 2.C.3. Simple models with time-varying vaccine compliance, δ

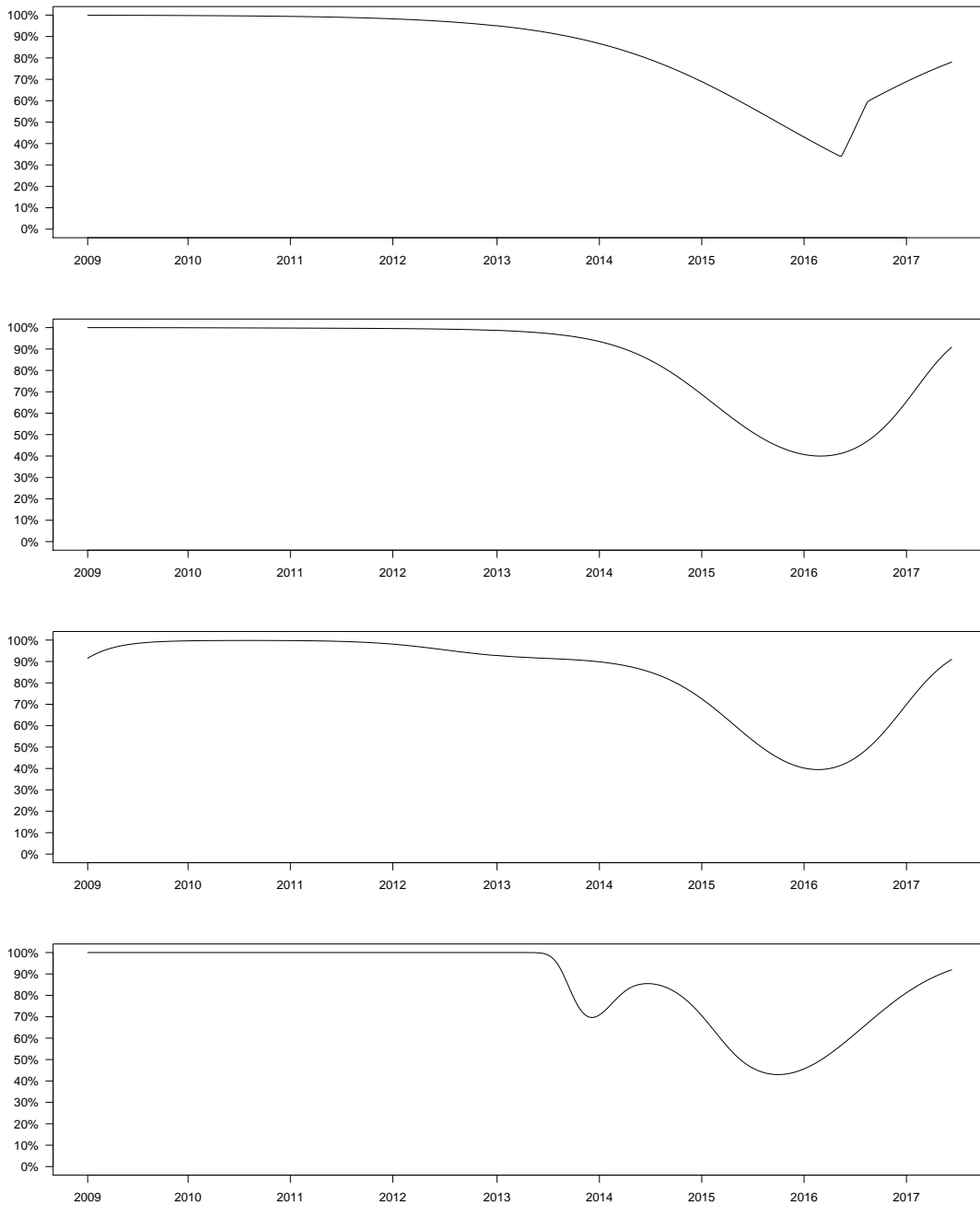


Table 2.C.2. Estimates for simple specifications

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
$\hat{\beta}_0$	7.1899	7.1861	7.2100	7.2170	7.1237	7.1597	7.1744	7.2092
$\hat{\beta}_1$	2.9937	2.9834	3.0230	2.9872	2.9574	2.9998	3.0030	3.0632
$\hat{\gamma}_0$	0.1874	0.1921	0.1896	0.1917	0.1921	0.1898	0.1932	0.1820
$\hat{\gamma}_1$	0.5333	0.5349	0.5337	0.5362	0.5363	0.5319	0.5344	0.5290
$\hat{\gamma}_2$	-0.8152	-0.8018	-0.7852	-0.7530	-0.7647	-0.8103	-0.7924	-0.8794
$\hat{\alpha}_0$	0.9700	0.9561	0.9999	0.8660	7.2544	3.7219	2.7401	62.2246
$\hat{\alpha}_1$	0.5207	0.4535	0.8224	0.9999	-0.0251	-58.7460	-0.0695	-51.8956
$\hat{\alpha}_2$	324	323	239	33	10.4847	6.7807	2.7068	30.4029
$\hat{\alpha}_3$		0.7229	0.4254	0.8624	0.0043	8.4769	-0.3602	-67.4860
$\hat{\alpha}_4$		403	324	206	58.1057	11.7334	1.0987	-58.3017
$\hat{\alpha}_5$			0.7444	0.3800	0.0999			-63.1225
$\hat{\alpha}_6$			403	325	382.8205			-77.9064
$\hat{\alpha}_7$				0.7113	-0.0586			-13.2806
$\hat{\alpha}_8$				399	396.2476			-91.3194
$\ell(\hat{\theta})$	-22992	-21910	-21089	-21097	-22116	-22151	-21962	-22524

Note: Standard errors in parentheses.

correction does not spuriously introduce patterns in the estimates, $\hat{\delta}_t$. The seasonal model is simply

$$\tilde{\eta}_t = \gamma z_t^a,$$

where z_t^a is defined as before.

The specifications for $\delta_t(\alpha)$ are those listed in Table 2.C.1. Figure 2.C.4 plots the estimated vaccine compliance, $\hat{\delta}_t$, for the different piecewise constant specifications. Figure 2.C.5 plots vaccine compliance for the simple specifications. The estimated time paths of $\hat{\delta}_t$ are very similar to those in 2.C.3. The only differences in terms of estimated break dates are that the last break is now determined to be one week later than in the model with monthly seasonality correction and that the model with three breaks sets the first break date a month earlier. Similarly, $\hat{\delta}_t$ in the different simple models are very much the same as those in Figure 2.C.3. Table 2.C.3 displays the estimates. For each specification, the log-likelihood is considerably lower than the respective value in Table 2.C.2, so even a static correction for monthly seasonality offers great improvements in the empirical fit of the data.

Figure 2.C.4. Models with structural changes in vaccine compliance without monthly seasonality correction, δ

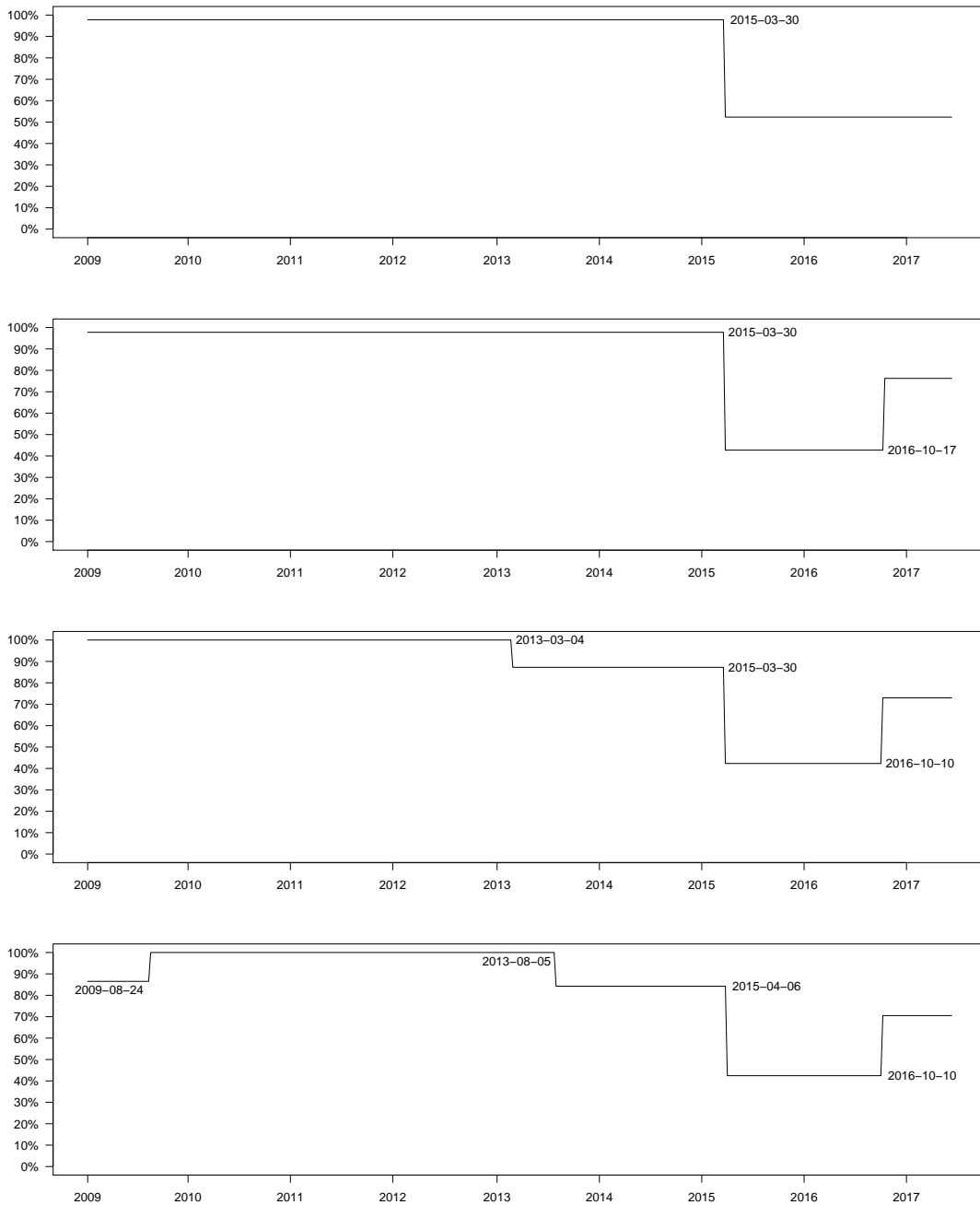


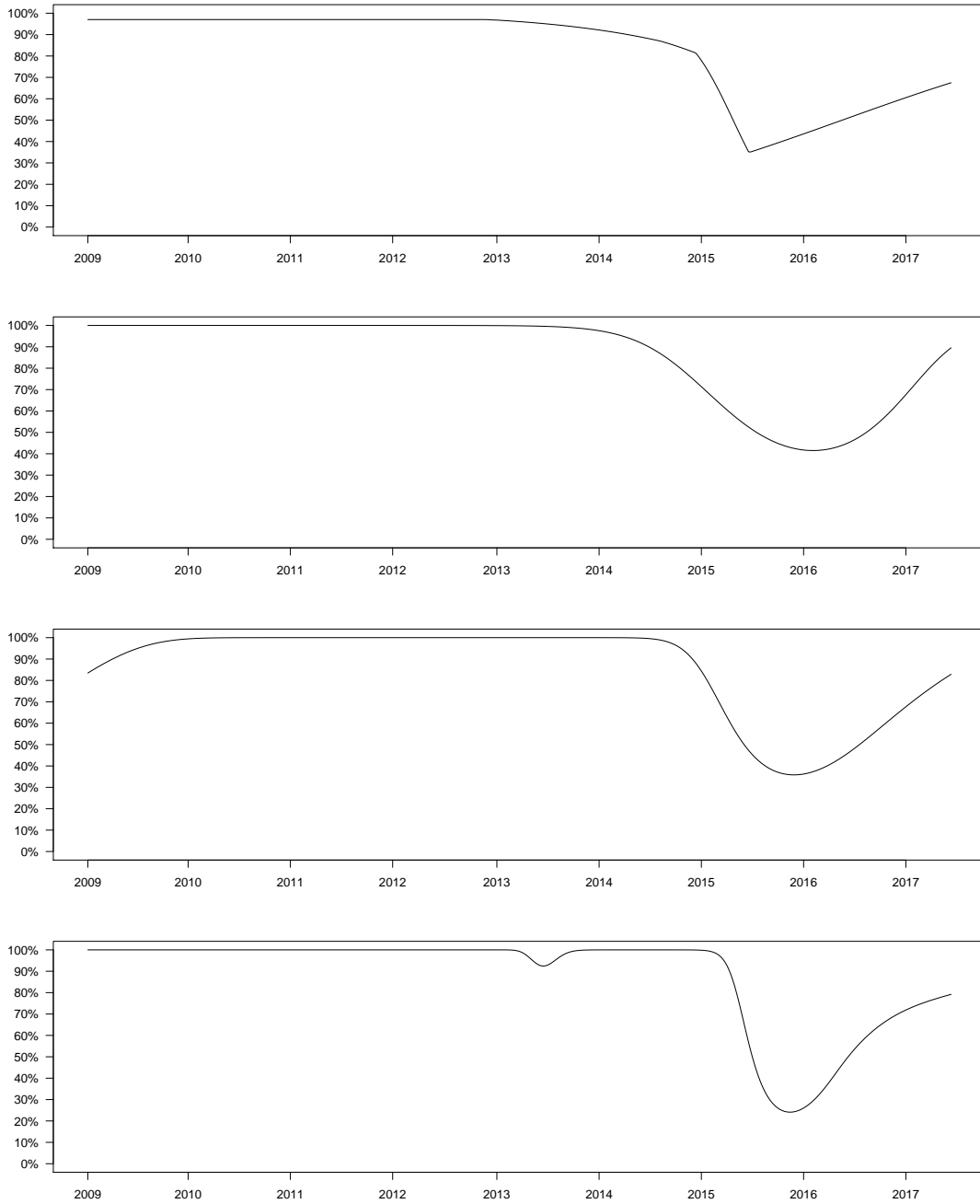
Figure 2.C.5. Simple models with time-varying vaccine compliance without monthly seasonality correction, δ 

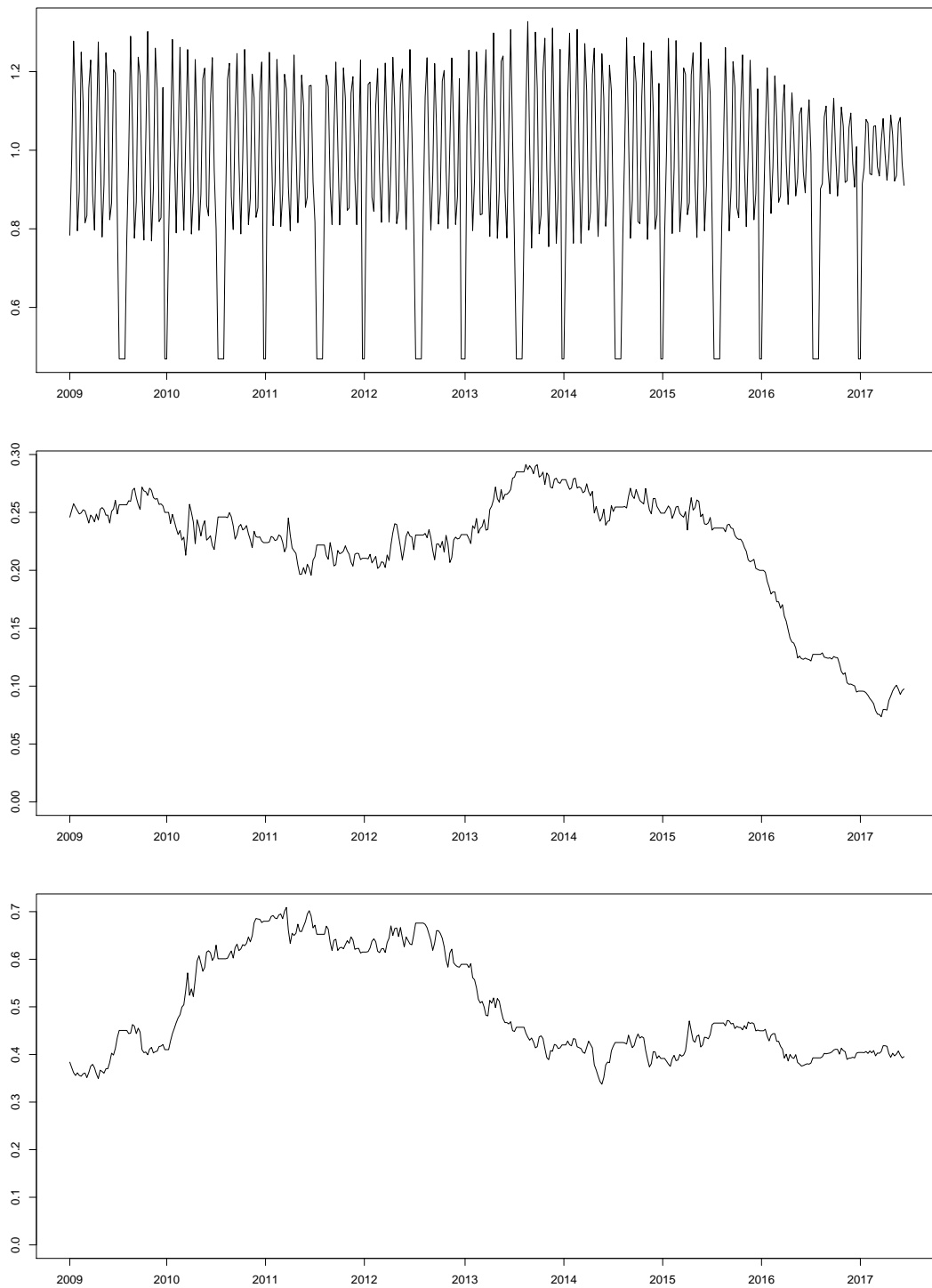
Table 2.C.3. Estimates for simple specifications without monthly seasonality correction

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
$\hat{\beta}_0$	7.1766	7.2157	7.2027	7.1994	7.1780	7.1533	7.1562	7.2312
$\hat{\beta}_1$	2.9848	3.0267	3.0128	2.9860	2.9886	3.0046	2.9675	3.0838
$\hat{\gamma}$	-0.8227	-0.8052	-0.7851	-0.7860	-0.7676	-0.8201	-0.8202	-0.8765
$\hat{\alpha}_0$	0.9782	0.9778	0.9999	0.8652	3.4871	6.2214	8.4378	62.4774
$\hat{\alpha}_1$	0.5233	0.4273	0.8726	1.0000	-0.0181	-98.1896	-8.2224	2.8881
$\hat{\alpha}_2$	324	324	216	33	203.4960	-2.5109	6.8286	6.3076
$\hat{\alpha}_3$		0.7625	0.4232	0.8424	-0.0053	35.4154	1.3612	-78.6869
$\hat{\alpha}_4$		405	324	238	291.2001	-0.8763	-1.9823	-38.1838
$\hat{\alpha}_5$			0.7297	0.4245	-0.0545			-66.4805
$\hat{\alpha}_6$			404	325	309.2410			-59.4491
$\hat{\alpha}_7$				0.7052	0.0911			-69.0183
$\hat{\alpha}_8$				404	336.1918			-56.0764
$\ell(\hat{\theta})$	-24868	-23492	-23019	-22923	-23914	-24158	-23890	-25009

Note: Standard errors in parentheses.

2.C.5 Seasonality correction in score-driven model

figure 2.C.6 plots the paths of $\hat{\eta}_t$ and of the time-varying seasonality parameters $\hat{g}_{0,t}$ and $\hat{g}_{1,t}$. During most of the sample period, monthly seasonality is strong, with estimated propensity during peaks being about 50% higher than during troughs. The monthly seasonal effect diminishes after 2015 and is not very pronounced towards the end of the sample period. The attenuation of the seasonal effect in the late sample period can also be seen in the graph of $\hat{g}_{0,t}$, the time-varying parameter that governs the amplitude of the sine function. The score-driven seasonal model reliably detects the change in the seasonal pattern induced by the end of the overrecording of vaccinations after the introduction of the electronic vaccine registry on November 15, 2015. The third panel of Figure 2.C.6 plots $\hat{g}_{1,t}$, the estimated time path of the phase shift in the sine function. $\hat{g}_{1,t}$ takes values between about 0.4 and 0.7 during the sample period, corresponding to overrecording towards the end of the month.

Figure 2.C.6. Time paths of $\hat{\eta}_t$ (upper panel), $\hat{g}_{0,t}$ (middle panel) and $\hat{g}_{1,t}$ (lower panel)

2.C.6 Further robustness checks

As an additional robustness check, we have also estimated the model where $x_{c,t}$ is assumed to be conditionally Poisson distributed (rather than conditionally binomially distributed). These results were also very similar to the ones reported here with the binomial specification. Finally, we also experimented with the specification for $\Lambda(\beta; a)$, and obtained very similar results with a truncated Weibull cumulative distribution function.

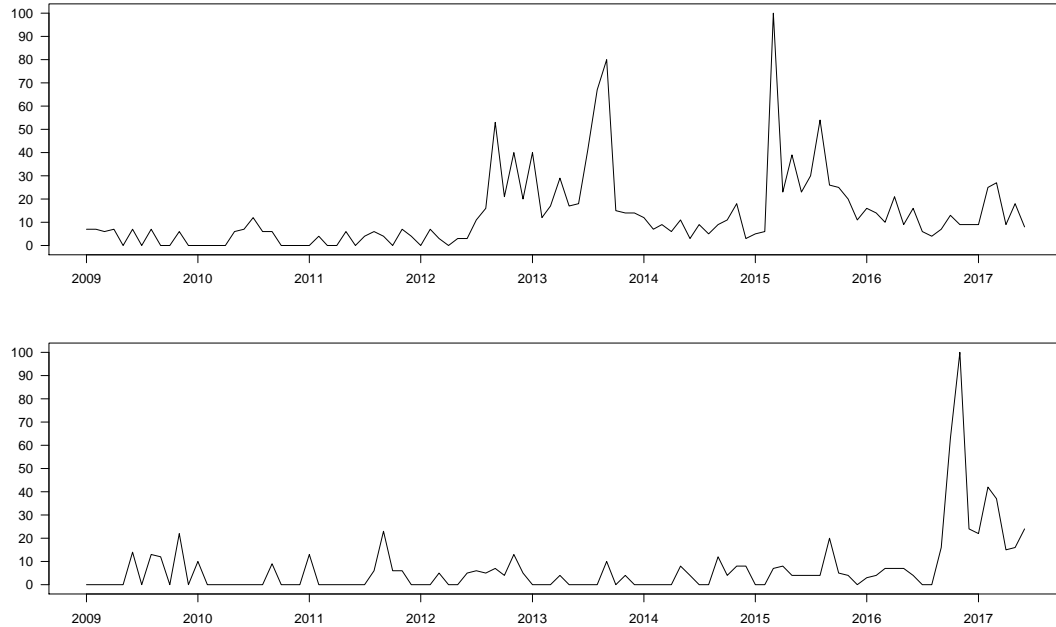
2.D Google searches, AE, and media activity

2.D.1 Google searches for HPV vaccine side effects

A potential concern about our findings is that lower vaccine compliance after March 2015 might reflect a global increase in anti-vax sentiment, rather than being just the effect of the TV2 documentary. Our analysis of reported AE in Denmark and Norway in Section 2.4 of the main chapter already points towards the increase in vaccine scepticism being specific to Denmark. Furthermore, we obtained monthly data on internet search intensity on side effects of the HPV vaccine from Google Trends.² Figure 2.D.1 displays the data for the sample period. The first time series is the search frequency for “bivirkninger hpv vaccine” in Denmark, normalized such that the maximum value is one hundred. The second time series is the corresponding time series for Norway (searches for “bivirkninger hpv vaksine”). The Danish data exhibit a prominent spike in March 2015, the month in which the documentary was aired. Norwegian search activity does not show much variation in 2015. The Figures, therefore, support the argument that the rise in Danish vaccine hesitancy during early 2015 was caused by an event that was specific to Denmark.

2. We would like to thank an anonymous referee for suggesting this exercise.

Figure 2.D.1. Search intensity for “HPV vaccine side effects” for Denmark (top panel) and Norway (bottom panel)



2.D.2 Monthly reported AE

Figure 2.D.2 shows the monthly count of reported AE by girls from the cohorts under study. The plot shows that the large number of reported AE in 2015 is mostly due to side effects reported after the airing of the documentary.

2.D.3 Media activities based on Google searches

Using simple Google searches we have also collected media data on the websites of the major Danish newspapers. We searched for keywords related to the HPV vaccine to identify articles on the topic, and to see which outlet had published stories that associated the HPV vaccine with serious side effects. The newspaper websites we searched are listed in Table 2.D.1 along with the number of articles (up until June 2017) and the readership in thousands in 2014 for each

Figure 2.D.2. Monthly number of AE reported

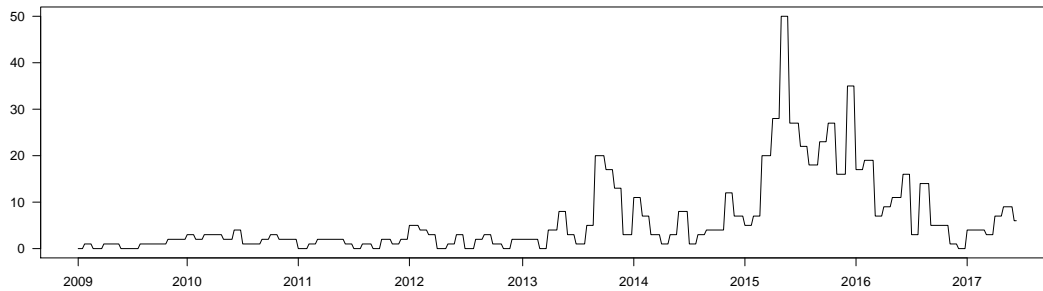


Table 2.D.1. Danish Media Articles: HPV Vaccine Efficacy and Side Effects

Source	Number of “negative” articles	Readership
<i>Metroxpress</i>	35	303
<i>Politiken</i>	21	90 (117)
<i>Kristeligt Dagblad</i>	17	26
<i>Berlingske</i>	12	76 (87)
<i>BT</i>	10	47 (66)
<i>Information</i>	7	20
<i>Ekstrabladet</i>	4	46 (61)
<i>Jyllandsposten</i>	1	84 (109)
<i>Weekendavisen</i>	0	– (46)

Note: Number of Danish media articles on the HPV vaccine that involve possible side effects or raising doubt about the vaccines effectiveness (2009-June 2017). Figures for weekday readership are in thousands (for 2014) with Sunday/weekend edition readership Figures in brackets. *Source:* Dansk Oplagskontrol.

newspaper. *Metroxpress*, which is a free newspaper, had the largest number of articles on the HPV vaccine with a negative sentiment and *Politiken* had the second most. Only one newspaper site, *Weekendavisen*, did not have any articles that met our criteria. The complete list of articles, along with date, source, and title, is presented in the appendix in Table 2.D.3. Most of these articles are merely reporting on alleged side effects or commenting on the declining vaccine uptake that has resulted from this concern. Other articles can appropriately be labeled as “fake news”. A prime example is an article in *Metroxpress* on June 11, 2015, whose headline read: “Doctors:

One in 500 get seriously ill from the HPV vaccine.”³ Another prime example of “fake news” is the article in *Information* on May 30, 2016, which (contrary to scientific consensus) presented the view that “there is no scientific evidence that the HPV vaccine prevents cervical cancer.”

Our internet search for HPV vaccine-related keywords on Danish language websites reveals that the Danish media coverage of the HPV vaccine was overwhelmingly positive until April 2013, and that negative stories about the vaccine were confined to anti-vaccine websites.⁴ The first article in mainstream media that associated the HPV vaccine with serious side effects was published in *Politiken*, a leading Danish newspaper, on April 17th, 2013. The article had a story about a girl who had a number of symptoms, including frequent headaches, dizziness, and tiredness, which her parents said were caused by the HPV vaccine.⁵ The article was followed by a series of articles in *Politiken* that discussed the possibility of a link between the HPV vaccine and serious AE. An article in *Politiken* on May 3, 2013 raised doubt about the efficacy of the vaccine, and accused the SST for being “absolutely misleading” in their information about the HPV vaccine and cervical cancer. The article quoted individuals labelled as, respectively, “one of the most recognized HPV experts in the world” and “a prominent medical professor”, and failed to mention that his views on vaccines are fringe views that contradicted those of the World Health Organization, the Centers for Disease Control and Prevention, and the European Medicines Agency (EMA). The two “experts” are quoted in several subsequent newspaper articles, including *BT* (Aug. 3, 2014), *Metroxpress* (May 19, 2015), and *Information* (May 30, 2016), and although the articles are rich with applauding credentials, none of them mentioned the controversial reputation of these individuals, nor their ties to the anti-vaccine movement.⁶

3. Translated from: “Lger: En ud af 500 piger bliver alvorligt syg af at f HPV-vaccinen”. The headline is not consistent with the quotes in the articles, and one of the two doctors that were quoted in the article, Jesper Mehlsen, has publicly denied having made this statement.

4. Earlier that year, on February 28th, *Politiken* contained an article that was critical of medical doctors who were advocating the HPV vaccine while receiving side income from GlaxoSmithKline, the producer of one of the HPV vaccine.

5. According to the mother, the parents learned from a homeopathy healer that their daughter’s illness had been caused by the HPV vaccines.

6. One of the “experts” had multiple articles on vaccines retracted, and is also a frequent speaker at Autism-

This was despite the fact that an 2003 article in *Journalisten*, which is published by the Danish Union of Journalists, had been critical of Danish media coverage of the HPV vaccine, and referred to one of these individuals as controversial, see Albrecht (2013). The timing of the first vaccine-critical newspaper articles coincides with the decrease of vaccine uptake.

2.D.4 Synopsis of TV2 documentary

The documentary was 36:04 minutes long, of which 22:23 minutes were spent on adverse events and their possible relation to the HPV vaccine, including 10:17 minutes relating the personal stories of three sick girls who all believed that their symptoms were caused by the vaccine. The documentary had no personal stories of women who had suffered from cervical cancer, and only 3:47 minutes of the documentary were spent on factual information about the vaccine, including a summary of the scientific studies that found no significant evidence of an association between the vaccine and AE. In the documentary, these scientific studies were immediately dismissed as irrelevant by the two doctors at the Synkocenteret, who suspected that there was an association between the vaccine and AE. 2:21 minutes were devoted to the way the SST had dealt with alleged side effects. The representative for SST, Henrik G. Jensen, was asked pointed questions, such as: “Is SST capable of judging whether a product, such as Gardasil, is safe?” and in response to an account of the vaccine monitoring procedure, the TV2 journalist Michael Bech commented “[this] sounds extremely bureaucratic.” 4:06 minutes had a conspiratorial connotation based on four emails that were missing from the material TV2 had received after a freedom of information request. In this segment, the TV2 journalist asked accusatory questions such as: “Did you omit the emails on purpose?” and “Is the Danish Health Authority in shambles or is this a cover-up?” 2:32 minutes were spent on considering how the girls who had experienced unexplained

One conferences that feature individuals such as Jenny McCarthy and Andrew Wakefield. The other individual was a speaker at a 2009 conference organized by the National Vaccine Information Center, which is an anti-vaccine organization in America that has its roots in the unfounded claims that the pertussis vaccine causes autism.

symptoms had been treated by the Danish health care system. Many of the girls were frustrated with the system: some felt they had been neglected whereas others felt they had been passed from one medical examination to the next without being given an explanation of their symptoms. A breakdown of the TV2 documentary into segments along with their type and duration is given in Table 2.D.2.

Figure 2.D.3. Vaccine compliance and media coverage during 2015

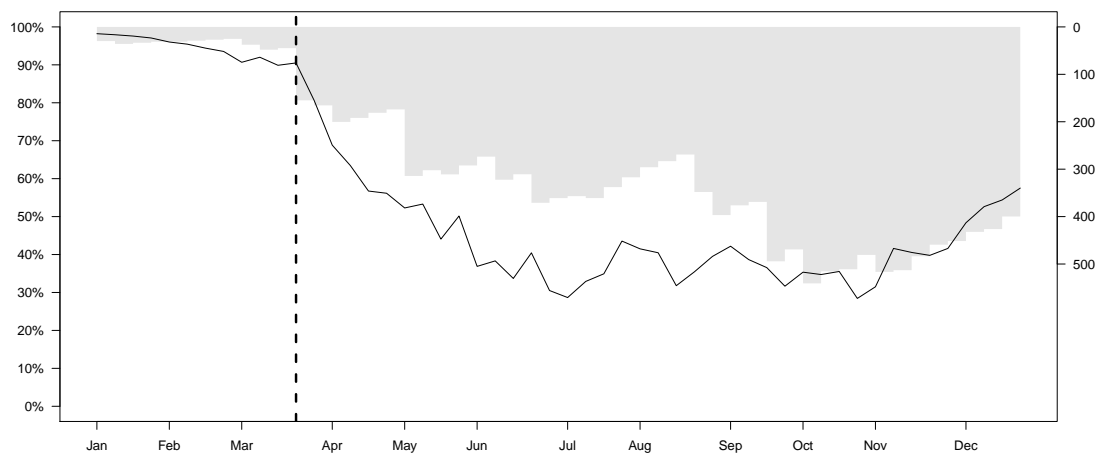


Figure 2.D.3 presents the estimated compliance for the year 2015, along with the media coverage of alleged side effects from the HPV vaccine (inverted axis). The shaded bars represent the exponentially weighted average of past media activity introduced in Section 2.4 of this chapter. The time of the pronounced decline in compliance is in agreement with the structural breaks models for δ_t , all of which found a large decline in $\hat{\delta}_t$ toward the end of March 2015. The vertical line denotes the week starting on March 30, and the abrupt decline in $\hat{\delta}_t$ occurs immediately after the airing of the TV2 documentary. Media coverage increased abruptly at the same time as the TV2 documentary; this is partly explained by media stories TV2 used to promote their documentary. Uncharacteristically for TV2 Denmark, which usually keeps its contents behind a

paywall, the company made the documentary freely available for on YouTube on which subtitles in various languages were added in due course.⁷ Given the attention the documentary attracted and the impact it had, the decline in vaccine compliance and the subsequent coverage of the subject in the Danish media can be reasonably attributed to it.

Table 2.D.2. TV2 Documentary: *De Vaccinerede Piger – Syge og Svigtede* (The Vaccinated Girls – Sick and Abandoned)

Start	Content	Dur.
0:00	PS Personal story of HPV vaccine harms (Amalie).	184
3:04	FI Factual information about HPV vaccine.	88
4:32	BG Denmark selected quadrivalent HPV vaccine. Information campaign to introduce the vaccine.	28
5:00	PS Personal story (Katrine).	175
7:55	AE Louise Brinth and Jesper Mehlsen (Synkopezenteret): Suspected AE.	172
10:47	SM MB: “Have the authorities been responsible?” Louise Brinth: “No, not yet.”	47
11:34	AE Louise Brinth and Jesper Mehlsen: Claim that HPV vaccine may be particularly risky for sports active girls.	41
12:15	PS Personal story (Laura).	186
15:21	TR Ulla Astman. Questions whether sick girls are getting sufficient help and treatment.	93
16:54	SM Henrik Jensen (DHA). MB: “Is DHA capable of evaluating whether a vaccine is safe?”	21
17:15	CU Freedom of Information Act request was submitted to DHA. Their reply did not include several emails.	33
17:48	AE Unapproved (detox) treatment by Dr. Downing (London). Cost about DKK 120,000.	253
22:01	FI Factual information about HPV vaccine safety studies. No difference in AE between vaccinated and not vaccinated.	80
23:21	AE Louise Brinth and Jesper Mehlsen: Safety studies are irrelevant for the symptoms they see.	43
24:04	AE Denmark awarded DKK 4 million compensation to three individuals for harms they believed were due to HPV vaccination. Other requests denied.	19
24:23	AE POTS (Amalie at Synkopezentret). POTS two cases in 1997 and fifty-seven cases in 2014.	139
26:42	CU Henrik Jensen (DHA) confronted. MB: “Did you omit the emails on purpose?”	118
28:40	CU Oluf Jrgensen: “It appears to be a smokescreen. DHA is not in compliance with the law.”	54
29:34	CU MB to Henrik Jensen (DHA): “Is the DHA in shambles or is this a cover-up?”	41
30:15	AE Sick girls want authorities to recognize that they got sick from the vaccine.	22
30:37	SM Henrik Jensen (DHA) explains how Denmark tracks warning signals about vaccine safety. MB: “It sounds extremely bureaucratic.”	73
31:50	AE EMA concludes that there is no evidence to link HPV vaccine to POTS, nor can it be ruled out.	36
32:26	FI Henrik Jensen (DHA): “Benefits of HPV vaccine greatly exceed (suspected) side effects.”	59
33:25	PS Personal story (Amalie).	72
34:37	TR Ulla Astman: “Clinics (HPV centers) for girls with symptoms will be established.”	58
35:35	BG Documentary concludes that it is neither proven nor disproven that HPV vaccine causes AE.	29
36:04	End.	

Note: The composition of the documentary in parts with the duration of each part listed in the last column. Acronyms refer to: BM: Michael Beck (the interviewing TV2 journalist); PS: personal story of individual who suspected sickness is caused by vaccine; FI: factual information about the vaccine; BG: background information about the introduction of the vaccine; AE: adverse events suspected to be caused by the HPV vaccine and details about unapproved coverage of these; SM: safety mechanisms for the vaccine, questioning whether warnings were being ignored/taken seriously; TR: treatment offered by the SST to individuals with suspected side effects; CU: cover-up in relation to the four emails that were omitted after a freedom of information request.

The list of media stories is given in Table 2.D.3.

7. TV2 removed the documentary from YouTube in October 2017, but unauthorized copies continue to be available here and elsewhere.

Table 2.D.3. Danish Media Articles: HPV Vaccine Efficacy and Side Effects

Date	Source	Title
17-Apr-13	<i>Politiken</i>	Frustrerede forældre: HPV-vaccine har gjort vores datters liv til et ...
3-May-13	<i>Politiken</i>	HPV-vacciner giver meget få, men alvorlige skader
3-May-13	<i>Politiken</i>	Internationale eksperter: Danmark oversælger fordelene ved HPV
3-May-13	<i>Berlingske</i>	Få men alvorlige skader efter HPV-vaccine
11-Jul-13	<i>Politiken</i>	Forening: Flere oplever bivirkninger efter HPV-vaccine
28-Jul-13	<i>BT</i>	400.000 piger i fare: HPV-Vaccinen kan give alvorlige bivirkninger
5-Sep-13	<i>Politiken</i>	Vi lader en rådden industri bruge vores børn som forsøgsdyr
5-Sep-13	<i>Kr.-Dagblad</i>	Vaccine skaber usikkerhed hos unge kvinder
13-Jul-14	<i>Berlingske</i>	Se Alvoren med HPV-vaccinen i øjnene
3-Aug-14	<i>BT</i>	Ramt af bivirkning: Signe får erstatning efter HPV-vaccine
3-Aug-14	<i>BT</i>	Ekspert: Danske piger har fået for høj dosis
25-Mar-15	<i>Politiken</i>	Efter voldsom kritik: Hjælp på vej til unge kvinder med smerter efter ...
25-Mar-15	<i>Kr.-Dagblad</i>	Regioner opretter særafdelinger for HPV-vaccinerede
25-Mar-15	<i>Metroxpress</i>	Læger advarer: Frygter bivirkninger ved HPV-vaccine
26-Mar-15	<i>Politiken</i>	Astrids far om hpv-bivirkninger: "Andre synes, at man er helt tosset ...
26-Mar-15	<i>Politiken</i>	Læge: Piger med vaccinebivirkninger skal tages alvorligt
26-Mar-15	<i>Kr.-Dagblad</i>	Læge om HPV: På tide at bivirkninger tages alvorligt
26-Mar-15	<i>Information</i>	Læge om HPV: På tide at bivirkninger tages alvorligt
26-Mar-15	<i>Information</i>	Vi kan aldrig være skråsikre på vacciner'
26-Mar-15	<i>Metroxpress</i>	HPV-vaccinen gjorde Katrine kronisk syg
17-Apr-15	<i>Information</i>	Hvornår tager man HPV-vaccinens ofre alvorligt?
5-May-15	<i>Berlingske</i>	Overlæge: Advar sportsaktive piger mod HPV-vaccinen
5-May-15	<i>Berlingske</i>	16-årige Natasja blev voldsomt syg efter HPV-vaccine: Har ikke været ...
5-May-15	<i>BT</i>	Overlæge advarer: Disse piger får flest bivirkninger af HPV-vaccinen ...
5-May-15	<i>BT</i>	HPV-vaccine eller ej? Her er forskerens fire 'forbudte' råd
5-May-15	<i>BT</i>	16-årige Natasja blev voldsomt syg efter HPV-vaccine: Har ikke været ...
5-May-15	<i>BT</i>	Politikere: Danskerne skal advares om HPV-vaccinen
5-May-15	<i>Kr.-Dagblad</i>	Overlæge: Myndigheder bør gøre mere for vaccineofre
6-May-15	<i>Ekstrabladet</i>	Trist pige: Fik HPV-vaccine og s virkede læber og tunge ikke
6-May-15	<i>Politiken</i>	Overlæge om HPV-advarsel: Vi er nødt til at undersøge bivirkninger
6-May-15	<i>Kr.-Dagblad</i>	Færre takker ja til vaccine mod livmoderhalskræft
6-May-15	<i>Metroxpress</i>	Far kæmper for sin datter: Så syg blev Astrid efter HPV-vaccine
18-May-15	<i>Berlingske</i>	Ekspert: Vi ved ikke, om HPV-vaccinens virkning er varig
19-May-15	<i>Berlingske</i>	Forsker bag HPV-vaccinen: Vil ikke give den til mine børn
19-May-15	<i>BT</i>	Forskeren bag omstridt HPV-vaccine: Jeg vil ikke give den til mine ...
19-May-15	<i>Metroxpress</i>	Forsker bag HPV-vaccinen: - Jeg vil ikke give den til mine børn
11-Jun-15	<i>Berlingske</i>	Læger: En ud af 500 piger bliver alvorligt syg efter HPV-vaccine
11-Jun-15	<i>Metroxpress</i>	Læger: En ud af 500 piger bliver alvorligt syg af at få HPV-vaccinen ...
12-Jun-15	<i>Metroxpress</i>	Nanna blev syg og tabte sig 18 kilo efter HPV-vaccinen: Læs hendes ...
25-Jun-15	<i>Kr.-Dagblad</i>	Indberetningsbunke over HPV-vaccines bivirkninger vokser
26-Jun-15	<i>Berlingske</i>	Frygt for bivirkninger får piger til at droppe HPV-vaccination
27-Jun-15	<i>Politiken</i>	Da besvimelser, kørestol og manglende skolegang blev hverdag for ...
27-Jun-15	<i>Berlingske</i>	Vaccinen der deler vandene
1-Jul-15	<i>Politiken</i>	Læger i stor strid om hpv-pigerne
6-Jul-15	<i>Metroxpress</i>	HPV-piger: Vi blev ikke advaret om bivirkninger
31-Aug-15	<i>Politiken</i>	Unge piger vælger i stigende grad HPV-vaccinen fra
31-Aug-15	<i>Berlingske</i>	Nu er der færre der får HPV-vaccinen end MFR
31-Aug-15	<i>BT</i>	Mette fik HPV-vaccinen for seks år siden og kan stadig ikke leve et
31-Aug-15	<i>Kr.-Dagblad</i>	1100 piger skal checkes for mistanke om HPV-bivirkninger
31-Aug-15	<i>Metroxpress</i>	Lange ventelister: Over 1.100 syge piger bestormer HPV-centre
1-Sep-15	<i>Metroxpress</i>	Blev afvist på HPV-center: 15-årige Katrine besvimer hele tiden
7-Sep-15	<i>Metroxpress</i>	Heidi HPV-vaccineret som 32-årig: Jeg er ikke den mor, jeg var før ...
8-Sep-15	<i>Metroxpress</i>	Læger: Stop med at HPV-vaccinere kvinder over 26 år
22-Sep-15	<i>Kr.-Dagblad</i>	Hundredvis med mistanke om HPV-bivirkninger venter på hjælp
24-Sep-15	<i>Politiken</i>	Indberettede HPV-bivirkninger slår alle rekorder
24-Sep-15	<i>Kr.-Dagblad</i>	Over 1500 piger har formodede HPV-bivirkninger
24-Sep-15	<i>Metroxpress</i>	Over 1500 piger har formodede HPV-bivirkninger
29-Sep-15	<i>Metroxpress</i>	Ekspert: Ny HPV-vaccine er ikke mere sikker
14-Oct-15	<i>BT</i>	Danske piger er ikke alene om HPV-plager: Udlandet oplever også ...
14-Oct-15	<i>Metroxpress</i>	SE KORTET: Hele verden har syge HPV-piger
21-Oct-15	<i>Metroxpress</i>	Vaccine-fortalere afgør syge HPV-pigers fremtid
26-Oct-15	<i>Berlingske</i>	Firma bag HPV-vaccinen underdrev omfanget af alvorlige bivirkninger
5-Nov-15	<i>Politiken</i>	Kronisk hovedpine, lammelser og krampeanfald er dagligdag for ...
5-Nov-15	<i>Metroxpress</i>	Førende svensk HPV-forsker: Mistanken om POTS er ikke undersøgt ...
7-Nov-15	<i>Kr.-Dagblad</i>	Færre vaccineres mod livmoderhalskræft
9-Nov-15	<i>Metroxpress</i>	TV: Her er HPV-pigernes symptomer
10-Nov-15	<i>Metroxpress</i>	Læge til Stine på HPV-center: Jeg er fløjtende ligeglad og ikke selv ...
10-Nov-15	<i>Metroxpress</i>	BLOG: Sådan er livet som POTS-syg mor til tre

continues...

(Table 2.D.3 continued).

Date	Source	Title
10-Nov-15	<i>Metroxpress</i>	Stor stigning: Over 100 danske piger har POTS
12-Nov-15	<i>Metroxpress</i>	HPV-kritisk læge: Styrelsen driver klapjagt på mig
12-Nov-15	<i>Metroxpress</i>	BLOG: Fyringer på HPV-center bekræfter min mistanke
12-Nov-15	<i>Metroxpress</i>	BLOG: Hvorfor ville I ikke undersøge mig for POTS?
17-Nov-15	<i>Metroxpress</i>	HPV-piger: Lægerne siger, vi er tossede
20-Nov-15	<i>Metroxpress</i>	BLOG: Er lægen styret af frygt eller lægeløftet?
24-Nov-15	<i>Metroxpress</i>	Smertesyndrom sendte 15-årig badminton-pige i kørestol
25-Nov-15	<i>Metroxpress</i>	BLOG: Piger med vaccineskader og læger på herrens mark
26-Nov-15	<i>Politiken</i>	Liselott Blixt affejer HPV-rapport: "Lavet af betalt lobby"
27-Nov-15	<i>Politiken</i>	Kritiseret HPV-center: "Hvilket ærinde har lægemiddelmyndighederne ...
1-Dec-15	<i>Metroxpress</i>	HPV: Alvorlige bivirkninger bliver aldrig indberettet
1-Dec-15	<i>Metroxpress</i>	Astrid opgav at indberette: Det var mega uoverskueligt
3-Dec-15	<i>Metroxpress</i>	Udskældt HPV-læge: Sundhedsstyrelsens uvidenhed er dybt...
8-Dec-15	<i>Metroxpress</i>	Førende forsker: Forsøg med HPV-vaccinen skjuler alvorlige bivirkninger
15-Dec-15	<i>Metroxpress</i>	Ekspert: Danske piger misinformeret i HPV-forsøg
17-Dec-15	<i>Politiken</i>	HPV-høring: Vi ser altså nye signaler
17-Dec-15	<i>Politiken</i>	Analyse: HPV-kritikken vil ikke dø
18-Dec-15	<i>Berlingske</i>	Trods videnskabelig modvind: Blixt tror stadig på HPV-bivirkninger
4-Feb-16	<i>Kr.-Dagblad</i>	Overlæge efterlyser behandling af HPV-pigers symptomer
4-Feb-16	<i>Information</i>	Overlæge efterlyser behandling af HPV-pigers symptomer
31-Mar-16	<i>Information</i>	Sundhedsstyrelsen er utroværdig
15-Apr-16	<i>Ekstrabladet</i>	Stadig flere piger sig nej til hpv-vaccinen
15-Apr-16	<i>Kr.-Dagblad</i>	Stadig flere piger sig nej til hpv-vaccinen
23-Apr-16	<i>Kr.-Dagblad</i>	Flere danske klager over HPV-vaccine end norske og svenske
23-Apr-16	<i>Kr.-Dagblad</i>	OVERBLIK: Danmark klager mest over hpv-vaccine
27-Apr-16	<i>Ekstrabladet</i>	Flere piger i hovedstadsområdet siger nej til hpv-vaccine
27-Apr-16	<i>Kr.-Dagblad</i>	Flere piger i hovedstadsområdet siger nej til hpv-vaccine
28-Apr-16	<i>Information</i>	Det er en svær beslutning at HPV-vaccinere sit barn
28-Apr-16	<i>Metroxpress</i>	Vi har nu set 500 piger med symptomer efter HPV-vaccinen
26-May-16	<i>Politiken</i>	HPV-rapport kaldes uacceptabelt, ringe videnskabeligt håndværk...
26-May-16	<i>Metroxpress</i>	Forskere og politikere klager over EU-frifindelse af HPV-vaccinen
30-May-16	<i>Kr.-Dagblad</i>	OVERBLIK: Hpv-vaccine til diskussion igen
30-May-16	<i>Information</i>	Ekspert anklager styrelse for at vildlede om HPV-vaccine
19-Jun-16	<i>Metroxpress</i>	Nyt HPV-kritisk magasin sætter fokus på bivirkninger ved vaccinen...
5-Aug-16	<i>Ekstrabladet</i>	Syg mor fortryder: Forkælede sig selv med HPV-vaccine til 3500 kr
4-Oct-16	<i>Kr.-Dagblad</i>	Brug af den kontroversielle hpv-vaccine er halveret
7-Apr-17	<i>Politiken</i>	Fortsat skepsis: Hver femte føler sig utryk ved hpv-vaccinen
28-Jun-17	<i>Politiken</i>	Overlæge afviser frikendelse af hpv-bivirkninger trods nyt studie...

Note: Danish media articles on the HPV vaccine that either reports on a possible association with serious side effects, or articles that raise doubt about the efficacy of the vaccine.

2.D.5 Media data collection: Infomedia search keywords

The media variable m_t was constructed by weekly statistics of media coverage using Infomedia keyword searches. Infomedia is a searchable media database with comprehensive coverage of the Danish media. Inclusive keywords were (('hpv' OR 'krftvaccine')) AND (('bivirkning' OR 'vaccineskade')) and a range of excluding keywords was used to eliminate articles that: reported on scientific studies that found the vaccine to be safe; argued in favor of including boys in the HPV vaccine program; announced a new and better version of the HPV vaccine; criticized the Danish media coverage of the HPV vaccine; and reported on scientific studies that showed girls with presumed vaccine injuries had higher morbidity and were more likely to have been hos-

pitalized because of a psychiatric disorder prior to HPV vaccination, see Mølbak, Hansen, and Valentiner-Branth (2016) and Lützen et al. (2017).

A comprehensive media search at Infomedia used the following keywords:

- Keywords: ('hpv' OR 'krftvaccine') AND ('bivirkning' OR 'vaccineskade')
- Excluding keywords:
 - Introduction of vaccine: 'kan redde 350' '350 kvinder kan reddes' '350 kvinder kan undg krft' '40 ddsfald kunne undgs' 'Vaccine kan redde mange flere' 'Vaccine kan redde kvinder' 'Siden den 1. oktober er piger fdt i 1993' 'I disse dage modtager alle 12-riige' 'Vaccin jeres brn mod HPV' 'Tre sm stik kan beskytte kvinder mod at udvikle livmoderhalskrft' 'Ny type af krftvaccine kan forlunge levetiden'
 - Vaccine for older cohorts: '1985-1992' '1985 og 1992' 'HPV-vaccinen vre gratis for alle' 'Kvinder over de 26 r burde ogs' 'hindre social ulighed' 'hindre ulighed'
 - Vaccine is safe: 'HPV-vaccinen er ikke farlig' 'Ingen beviser mod HPV-vaccine' 'ingen alvorlige bivirkninger' 'uden alvorlige bivirkninger' 'vaccination er sikker' 'HPV-vaccine giver ikke' 'vaccine er sikker' 'HPV-vaccine er fortsat sikker' 'vaccinen frikendes' 'HPV-vaccine er ikke skyld' 'vaccine frikendt' 'frikender HPV-vaccine' 'frikendte vaccinen' 'ger ikke risiko' 'HPVvaccination er sikker' 'HPV-vaccinen klarer frisag' 'studie afviser mistanke' 'ingen sammenhg mellem utilsigtede bivirkninger' 'ikke pvist alvorlige bivirkninger' 'afliver mistanke' 'Ikke get risiko for sclerose' 'ikke ud til at ge risikoen' 'Krftvaccine frikendt' 'Krftvaccine meldes ok' 'renset for mistanke' 'nsten uden bivirkninger' 'ikke er sammenhg' 'fr ikke alvorlige bivirkning' 'ingen grund til at frygte'
 - Information campaign: 'Informationsindsats' 'kampagne' 'www.stophpv.dk' 'Foredrag om HPV-vaccine' 'bliv klogere p livmoderhalskrft' 'HPV-vaccinen er effektiv' 'krftvaccine virker 100 procent' 'ja til HPV-vaccinen' 'cykler for HPV' 'Brev til forldre' 'Pmindelsesbreve' 'opfordrer nu forldre' 'Krfstens Bekmpelse sttter fuldt ud' 'livmoderhalskrft p trods af'
 - New vaccine: 'ny vaccine' 'ny HPV-vaccine' 'bedre HPV-vaccine p vej'
 - Other (irrelevant articles): 'HIV/AIDS' 'hepatitis B' 'omskring' 'lgemiddelagentur' 'influenzavaccination' 'p-piller' 'lungekrft' 'Hormonspiral' 'Strlebehandling' 'Frigiv hash' 'rsrapport' 'Morten Frisch' 'Jan Blaakr' 'Iben Holten' 'Anders Peter Hviid' 'Stinus Lindgreen' 'Lykkebo' 'Mads Koch Hansen' 'Milena Penkova' 'Jakob Schrder' 'David Budtz Pedersen' 'fredede olesen' 'social epidemi' 'March for Science' 'Norge' 'Sverige' 'kronprinsesse' 'Prostvac' 'stjerne i god sags tjeneste' 'kolesterol' 'Krftbehandlingen skal styrkes' 'Center for Krft og Sundhed' 'mslingeudbrud' 'Bavarian Nordic' 'hjernerodoping' 'Hiv-Danmark' 'Forskere satser' 'skabe en krftvaccine' 'hudkrft' '26 r og krfttram' 'Sverige er vaccinen billigere' 'hj niveau af antistoffer mod HPV' 'alvorlige bivirkninger ved den slags operationer'
 - Vaccine for boys: 'drengene skal tilbydes' 'Mnd m selv betale for vaccine' 'vaccinere drenge' 'hpv-vaccine til drenge' 'drenge br tilbydes' 'HPV-vaccination af drenge' 'Drenge br HPV-vaccineres' 'F drenge bliver vaccineret' 'ogs drenge fr HPV-vacciner' 'F drenge vaccineres' 'HPV-vaccine til alle drenge' 'HPV vacciner til mnd' 'vaccination af snner' 'alle unge mnd'
 - Girls with symptoms were sick before vaccination: 'oftere psykiatrisk' 'ramt psykisk' 'Syge inden vaccination' 'HPV-piger var oftere syge allerede inden vaccinen' 'HPV-piger var mere syge'
 - Critique of media: 'Mediernes dkning' 'journalistik koster liv' 'falsk videnskab' 'husk fakta' 'flelsesladet dokumentar' 'Medieomtalen af hpv-vaccinen' 'postfaktuelle' 'frygter boom i antal krfttilfide' 'frygter boom i krfttilfide' 'TV2 har et ansvar' 'Medierne har fortalt mange skrkthistorer' 'ude af proportioner'
 - Side effects vanishing: 'HPV-piger forsvundet' 'HPV-piger er som forsvundet' 'Tomme HPV-centre' 'Frre anmelder bivirkninger' 'frre bivirkninger' 'Mange flere hpv-vaccinerede' 'flere piger fr HPV-vaccination' 'Stigning i antallet af hpv-vacciner' 'flere HPV-vaccineret' 'Kurven er knkket'
 - Myths about the vaccine: 'Myter om srlige bivirkninger' 'Myter om vacciner' 'Myter om bivirkninger' 'alternative fakta' 'ekkokamre' 'faktaresistente'
 - Critique of Liselott Blixt: 'DF-ordfrer fr massiv kritik' 'sundhedsordfrer fr massiv kritik' 'Kritik hagler ned over Liselott Blixt' 'kritiserer Blixt i HPV-sag' 'Blixt er groft uansvarlig' 'Massiv kritik af DF' 'udsat for hrd kritik i HPV-sag'

Chapter 3

Resilience of HPV Vaccine

Uptake in Denmark: Decline and Recovery

JOINT WITH PETER R. HANSEN AND NOEL T. BREWER

3.1 Introduction

Vaccination is one of the most important public health achievements of the twentieth century (Centers for Disease Control and Prevention 2011). Central to realizing vaccination's full impact is ensuring high coverage that is timely and stable (Brewer et al. 2017) in order to ensure herd immunity (Anderson and May 1985). Unsubstantiated safety scares that spread through social

and traditional media can sometimes lead to sharp and lasting declines in vaccine coverage if not met with immediate and concerted efforts (Smith et al. 2008). Despite clear evidence of the HPV vaccine's safety and efficacy in independent reviews (Arbyn et al. 2018; Phillips et al. 2018), safety scares have been associated with large declines in HPV vaccine uptake in several countries, including Japan, Columbia, and Ireland (Hanley et al. 2015; Corcoran, Clarke, and Barrett 2018; Castro 2018). In Denmark, most girls were receiving the first dose of HPV vaccine in the first calendar year of eligibility, but this number declined substantially during an unfounded safety scare (Suppli et al. 2018). These declines in vaccination can be even more pronounced in vulnerable areas and populations (Hawker et al. 2007).

Few documented examples exist of vaccination programs recovering (National Health Service 2009) and, as a result, we have little understanding of resiliency in vaccination programs. Denmark's troubles with the HPV vaccine offer an important opportunity to understand a vaccine program's resilience in action. Although some have speculated that negative media coverage in Denmark led to the decline in uptake, we sought to confirm the role of two major media events. More importantly, we sought to establish whether a national information campaign about the HPV vaccine safety and effectiveness was associated with a recovery in uptake.

3.2 Methods

Setting and population

The HPV vaccine became part of the Danish national childhood vaccine program on January 1, 2009, delivered for free by family physicians to adolescent girls aged between twelve and fifteen and born in 1996 or later (Danish Health Authority 2009). The program first provided quadrivalent HPV vaccine (late 2008-January 2016), then bivalent HPV vaccine (February 2016-October 2017), and then nonavalent HPV vaccine (November 2017-current). Initially, the HPV

vaccine had a three-dose schedule, which dropped to a two-dose schedule for girls who initiated HPV vaccination before age fifteen (Danish Health Authority 2014). Our study population was all girls residing in Denmark who were born between 1997 and 2006 ($N=328,779$).

Patients and the public were not involved in the design, conduct or reporting of the research.

Procedures

Our retrospective cohort study examined four key time periods in the Danish media and the HPV vaccination landscape between 2009 and 2019. The first time period is *baseline*, from January 2009 to January 2013 (T_1). This period is when the HPV vaccine uptake was similar to uptake of other vaccines in the Danish childhood vaccination program and before the negative media coverage began.

The second time period is *some negative media*, from February 2013 to February 2015 (T_2). The period starts when the first stories that were critical of HPV vaccine appeared in mainstream media (Politiken 2013b). Several newspaper articles suggested that HPV vaccine had serious side effects or suggested that physicians who had advocated for HPV vaccination and its inclusion into the Danish vaccination program had conflicting interests (see also chapter 2). Research has not substantiated claims of serious AE from HPV vaccine beyond those typical for other adolescent vaccines (syncope and anaphylaxis, Arbyn et al. 2018).

The third time period is *extensive negative media*, from March 2015 to April 2017 (T_3). On March 26, 2015, TV2 Denmark aired a sensationalized documentary in Danish entitled: *De Vaccinerede Piger – Syge og Svigtede* (The Vaccinated Girls – Sick and Abandoned)(TV2 Denmark 2015). The documentary presented personal stories of young women who believed they had illnesses caused by the HPV vaccination, and the documentary suggested that the SST had not been forthcoming about serious AE from the vaccine. Prior to the documentary being aired, it was promoted on on TV2 Denmark along with trailers and related stories, and extensive

media coverage followed on the possible association between the HPV vaccine and severe AE (Suppli et al. 2018, see also chapter 2). By the end of April 2015, Danish media outlets had referenced the TV documentary 170 times (see chapter 2). A synopsis of the TV documentary appears in Section 2.D.4 in the supplementary material for chapter 2.

The fourth time period is *information campaign*, from May 2017 to February 2019 (T_4). The SST began a national information campaign on May 10, 2017 to share information on the safety and effectiveness of the HPV vaccination (Danish Health Authority 2019). The campaign also used personal stories and social media.

Measures

The SSI provided data on the monthly number of first doses of the HPV vaccine provided in Denmark by birth year cohort of recipients. Vaccination data were at the aggregate level and, thus, were fully anonymized. Statistics Denmark provided data on birth year cohort size, that is the number of girls residing in Denmark on January 1 in the calendar year in which they turned twelve. We used these data sources to calculate two measures of HPV vaccine initiation: *uptake*, the percentage of girls who had received at least one dose of HPV vaccine in a time period; and *coverage*, the percentage of girls who had received at least one dose of HPV vaccine by the calendar year they turned sixteen.

Data analysis

First, we characterized HPV vaccination coverage by birth year and age among all adolescent girls in Denmark born between 1997 and 2006. Next, we modeled the time variation in HPV vaccine uptake, adjusting for seasonal variation and age of unvaccinated girls.

We modeled HPV vaccine uptake using two components: a baseline component that is common for all birth year cohorts, and a time-specific component that captured time variation in

uptake that is driven by factors other than seasonality and age. Let N_c denote the size of cohort c , and let $x_{c,i}$ be the number of HPV vaccine initiation doses of cohort $c = 1997, \dots, 2006$ in month $i = 1, \dots, 48$. Here, $i = 1$ corresponds to January in the year in which the girls turn twelve, and $i = 48$ corresponds to December in the year in which they turn fifteen. The statistical model took $x_{c,i}$ to be Poisson distributed, with intensity given by

$$\mathbb{E}x_{c,i} = \delta_{\tau(c,i)} \gamma_i N_c, \quad 0 < \delta_{\tau(c,i)} \gamma_i < 1.$$

Here, γ_i is the baseline distribution of vaccinations over the forty-eight months we followed each of the cohorts. The variation in γ_i is tied to seasonality and age-specific uptake. The second term, $\delta_{\tau(c,i)}$, is the time-specific effect, where $t = \tau(c, i) = c + 12 + \frac{i-1}{12}$ is the calendar time in which cohort c is i months into the vaccination program. For example, the first month in which girls born in 2003 are eligible for HPV1 vaccination is, therefore, $\tau(2003, 1) = 2015.0$, which corresponds to January 2015. The calendar-specific component, δ_t , is key in our analysis. It shows how uptake varied over the sample period, and its time variation can be compared with events that may have influenced HPV vaccine uptake in Denmark. The parameters in the model are identified by a normalization of δ_t . In our empirical analysis we use the normalization $\sum_{t \in T_1} \delta_t = 1$, so that δ_t can be interpreted as uptake relative to the average uptake before February 2013 (baseline), where coverage was about 94% for Danish girls.

The Poisson assumption implies that the distribution of $x_{c,i}$, conditional on the sum $\sum_c x_{c,i}$ is a multinomial distribution with parameter

$$p_{c,i} = \frac{\delta_{\tau(c,i)} N_c}{\sum_c \delta_{\tau(c,i)} N_c}.$$

This, conveniently, does not depend on γ_i , so we can estimate and conduct inference about δ_t

by maximizing the multinomial likelihood function with respect to $p_{c,i}$. We computed robust standard errors (Wooldridge 1999), see Appendix 3.A.3.

Estimating relative uptake We computed average HPV vaccine uptake for each of the four periods as $\bar{\delta}(k) = \frac{1}{n_k} \sum_{t \in T_k} \hat{\delta}_t$, $k = 1, 2, 3, 4$, and the average uptake in period k relative to the baseline period, by investigating the ratio

$$r_k = \frac{n_k^{-1} \sum_{t \in T_k} \hat{\delta}_t}{n_1^{-1} \sum_{t \in T_1} \hat{\delta}_t}, \quad k = 2, 3, 4,$$

where T_k represents subperiod k . We computed confidence intervals around this ratio by inverting the Wald statistic for the nonlinear restriction $\log(r_k) - r = 0$, and solving for r .

Estimating cumulative missed opportunities We calculated missed opportunities for HPV vaccine initiation by comparing the actual number of vaccinated girls relative to a counterfactual number of vaccinated girls, for which the latter was based on the assumptions that all birth year cohorts had the same uptake as girls born in 1997, 1998, or 1999. The counterfactual number of vaccinated girls in cohort c in the i^{th} month of vaccination is simply

$$x_{c,i}^* = \frac{x_{1997,i} + x_{1998,i} + x_{1999,i}}{N_{1997} + N_{1998} + N_{1999}} N_c,$$

and by aggregating over cohorts for a given point in time, $X_t^* = \sum_{i:t=\tau(c,i)} x_{c,i}^*$, we arrived at the expected number of vaccinations in calendar month t , in the counterfactual scenario. We then compared this number to the actual number of vaccinations: $X_t = \sum_{i:t=\tau(c,i)} x_{c,i}$. A figure of their difference, $X_t - X_t^*$, appears in the appendix to this chapter. We then calculated the

evolution of the cumulative difference

$$\sum_{s \leq t} X_s - X_s^*.$$

Finally, we plotted this cumulative number of missed opportunities to show how far the country was behind or ahead of baseline.

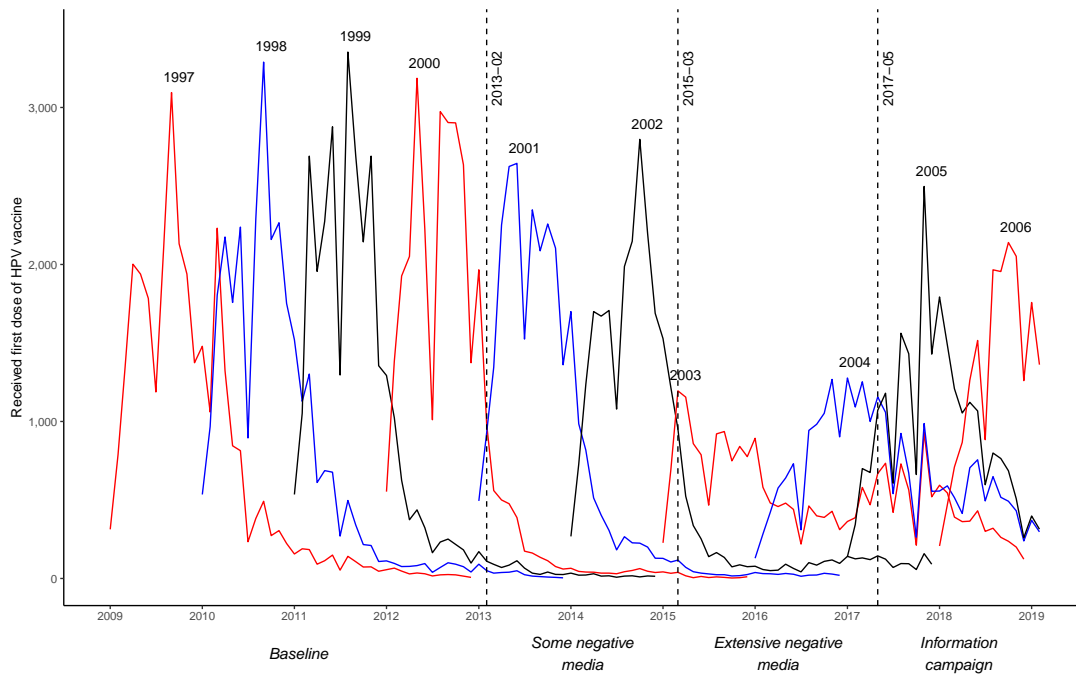
3.3 Results

3.3.1 HPV vaccine initiation

Of adolescent Danish girls who received HPV vaccine, most did so in the calendar year they became eligible (Table 3.1). HPV vaccine coverage in the calendar year the girls turned thirteen was near or above 90% for the older cohorts. During the negative media coverage, HPV vaccine coverage by age thirteen dropped to as low as 47% for the younger cohorts due for HPV vaccination. HPV vaccine initiation was particularly low during the years 2015 and 2016 (Figure 3.1). HPV vaccine initiation was also subject to seasonal effects, with relatively few doses delivered during the summer (July) and a peak during September.

During the baseline period, HPV vaccine uptake adjusted for seasonal and age effects fluctuated in a relatively narrow band (Figure 3.2). In the period with some negative media coverage, uptake decreased to 83.6% (95% CI: 78.0%, 89.7%) of baseline uptake. In the period with extensive negative media coverage, uptake fell even further, to 49.6% (95% CI: 44.5%, 55.2%) of baseline uptake. In the final period, when the information campaign was active, uptake increased again to a level indistinguishable from baseline (109.2%, 95% CI: 90.1%, 132.4%). This recovery was due to an unusually large number of girls being vaccinated at age fourteen and fifteen who had missed initiation at an earlier age (Table 3.1).

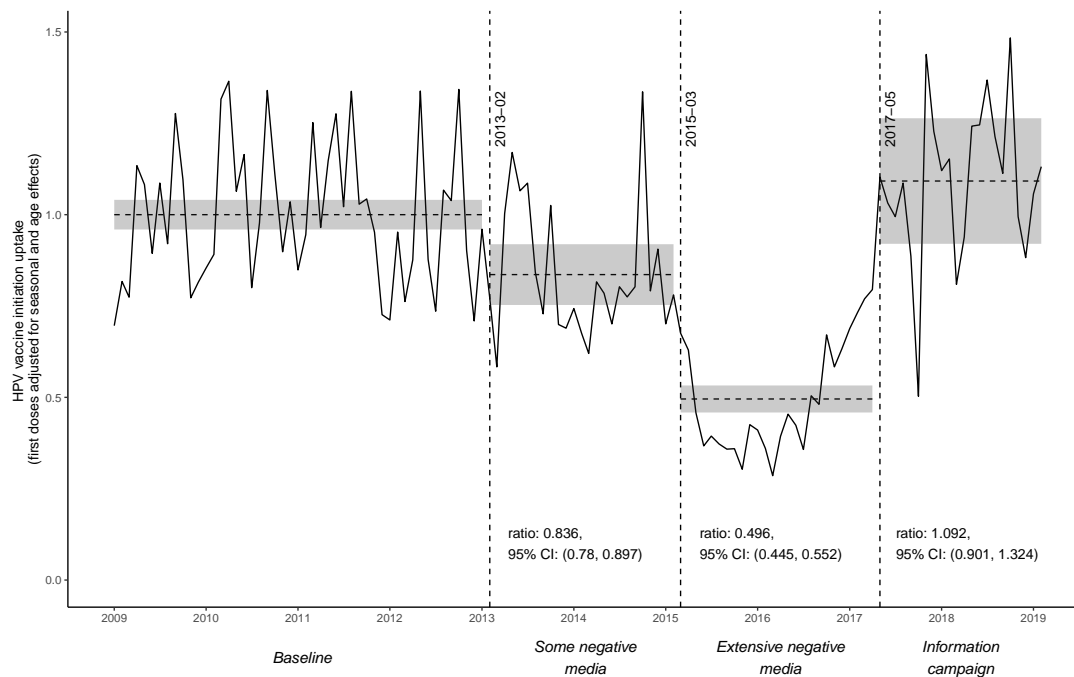
Figure 3.1. HPV vaccine initiation over time for each birth year cohort.



3.3.2 Cumulative missed opportunities

Initially, cumulative missed opportunities were slightly above the average of cohorts 1997-1999 (red area in first period of Figure 3.3). This was due to girls born in 1997 who were vaccinated at an older age than was the case for cohorts 1998 and 1999 (see also Table 3.1). The cumulative number of vaccinations quickly recovered (as shown in the blue area of vaccination surplus). In the period of some negative media coverage, cumulative missed opportunities followed a slight and steady increase. In the period of extensive negative media coverage, the number of missed doses continued to swell. By the end of the third period in May 2017, over 36,000 girls had missed the opportunity to receive the HPV vaccine as compared to vaccine delivery baseline. In the final period of the information campaign, the backlog of missed opportunities slowly shrank. A temporary increase in the number of missed opportunities in September and October 2017 was followed by an unusually large number of vaccinations in November 2017. Despite the recovery

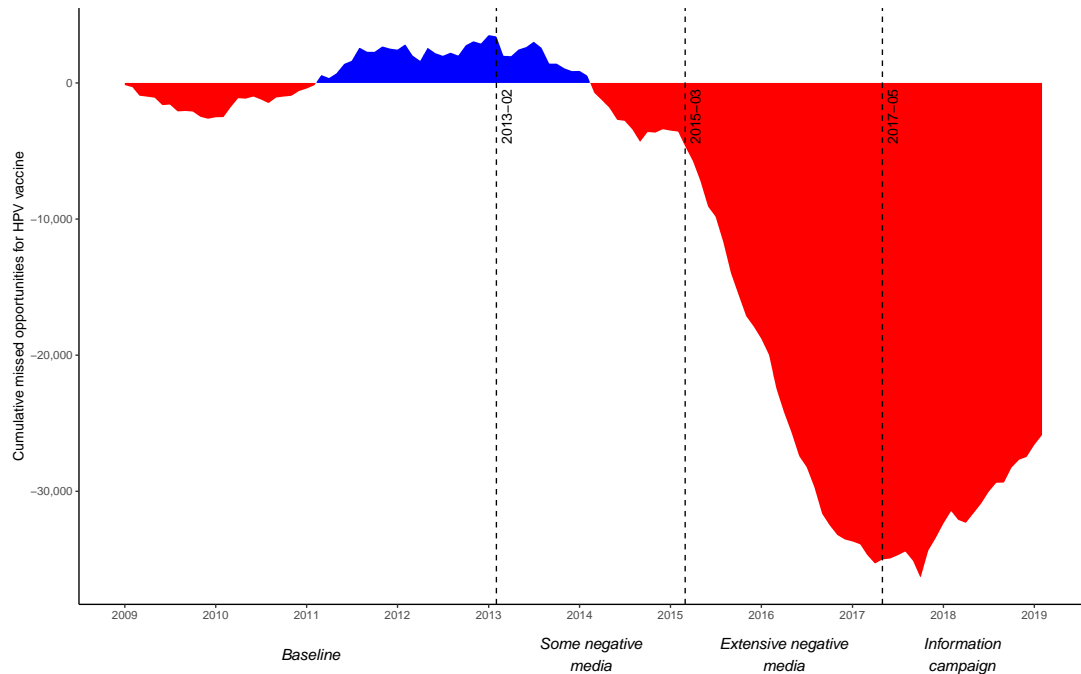
Figure 3.2. HPV vaccine initiation uptake in time periods defined by negative media coverage and national information campaign. Outcome was first HPV vaccine dose by the calendar year the girl turned sixteen, adjusted for seasonality and age effects. Solid lines show HPV vaccine initiation uptake, scaled to the baseline period's average. Dotted horizontal lines show average HPV vaccine initiation uptake for the time period, and shaded areas show 95% confidence intervals.



in vaccination provision by the end of the final time period in February 2019, the cumulative missed opportunities remained at around 26,000.

We translated these missed opportunities to additional cases of cervical cancer and deaths. Given a 0.9% lifetime prevalence of cervical cancer in Danish women a quarter of whom die from the disease (Engholm et al. 2010) and assuming 70% vaccine effectiveness against cervical cancer, these missed opportunities may lead to more than 180 cases of cervical cancer and more than 45 deaths that vaccination could have prevented.

Figure 3.3. Cumulative missed opportunities for HPV vaccine initiation in time periods defined by negative media coverage and national information campaign. Missed opportunities are relative to the average HPV vaccine initiation for birth year cohorts 1997-1999.



3.4 Discussion

Our study of over 300,000 Danish adolescent girls showed clear patterns of decline and recovery in HPV vaccination. Negative newspaper stories were associated with a 14% decline in HPV vaccine uptake relative to before the stories appeared, and the negative television documentary and media coverage that followed it were associated with uptake falling by half relative to baseline. The national information campaign coincided with the recovery to pre-crisis levels of uptake. Despite this recovery, the periods with negative media coverage left over 26,000 older girls unvaccinated who would have otherwise received the vaccine. The missed doses translate to over 180 avoidable cervical cancers and 45 deaths.

Coverage is generally high for vaccines globally, with HPV vaccination being an exception in facing substantial challenges (Bruni et al. 2016). Although declines in vaccination are un-

common, many ongoing crises are around HPV vaccination (Hanley et al. 2015; Castro 2018). Denmark's large decline in HPV vaccination may be attributable to an unsubstantiated safety scare generated and amplified through traditional media channels of newspapers and television, see Chapter 2. The underlying mechanisms for the decline remain unclear, but it is plausible that media reports generated concerns among the public that the Danish authorities inadequately addressed. The media coverage may also have led people to attribute existing or new unexplained symptoms to vaccination (Brewer, Hallman, and Kipen 2008). Such an explanation is consistent with events in New Zealand where the volume of news articles on the HPV vaccine was associated with AE reports (Faasse et al. 2017). Additional research is needed to understand the specific mechanisms through which unfounded safety scares undermine vaccine uptake, including the role of Denmark's decision early on to pay several people who had attributed their illnesses to HPV vaccination (Politiken 2013a).

Few studies have documented recovery in vaccination programs and, as a result, it is poorly understood. In the United Kingdom, a scandal generated by a research article purportedly linking autism to MMR vaccination triggered a decline in uptake that slowly recovered. Perhaps because of this experience, the United Kingdom was very proactive in managing public concern about the death of a young girl after HPV vaccination in 2008, during the early rollout of the vaccine. In contrast, Danish authorities were slow to respond to media stories questioning HPV vaccine safety, and early communication efforts relied on dry scientific information that did not engage to the public. The authorities did not have a social media presence, effectively ceding that method of communication to anti-vaccine activists. The Danish national information campaign, however, appeared to help HPV vaccination recover to precrisis levels. The introduction of the nonavalent HPV vaccine in November 2017 may have also helped to accelerate the recovery of the vaccination program, as our findings suggest that parents postponed vaccination to allow their daughters to receive the more effective vaccine.

Study strengths include the use of nationally representative vaccination data for an entire country. Study limitations include the correlational nature of the study. Interrupted time series study designs allow limited causal inference due, in part, to the possibility of other unknown historical events that could correlate with the focal events. The threat of selection is, however, unlikely given the representativeness of the sample and the low rate of migration in and out of the country (Rechel et al. 2013). Our analyses were ecological in nature, meaning that inference at the level of individual patients requires some caution.

In conclusion, the Danish vaccination program has successfully weathered a serious decline in HPV vaccine uptake that was associated with negative media coverage. The national information campaign appears to have been successful, although other events may account for some of the recovery. To ensure resilience of vaccination programs, researchers and practitioners have suggested specific steps that countries can take (Vorsters et al. 2017; Vorsters and Van Damme 2018). Countries should have established plans for addressing the inevitable safety scares that come at unpredictable times and from unpredictable sources. Tracking safety signals and public sentiment is also important to anticipating challenges to resilience. Countries should respond quickly and accurately to crises through a single spokesperson and use social media. Finally, it is important to rely on advocacy organizations and their networks (Saslow et al. 2018).

Table 3.1. HPV vaccine initiation from 2009 to 2019, by birth year cohort

Birth Year Cohort	N	First doses of HPV vaccine in year...											Cover age
		2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	
1997	33,630	60%	29%	4%	1%	-	-	-	-	-	-	-	94%
1998	32,937	-	67%	23%	3%	1%	-	-	-	-	-	-	94%
1999	33,034	-	-	75%	16%	3%	1%	-	-	-	-	-	95%
2000	33,387	-	-	-	75%	17%	2%	1%	-	-	-	-	95%
2001	32,548	-	-	-	-	68%	18%	2%	1%	-	-	-	89%
2002	31,863	-	-	-	-	-	60%	17%	3%	4%	-	-	84%
2003	32,463	-	-	-	-	-	-	30%	17%	20%	13%	-	80%
2004	32,741	-	-	-	-	-	-	-	25%	33%	19%	2%	79%
2005	32,925	-	-	-	-	-	-	-	-	37%	34%	2%	73%
2006	33,251	-	-	-	-	-	-	-	-	-	46%	9%	55%
Total first doses per year		20,014	31,788	33,861	31,669	28,793	25,908	15,915	15,044	31,000	37,124	4,499	

Note: Percentages are HPV vaccine initiation for birth year cohorts (rows). The last column shows HPV vaccine initiation coverage by the year girls in the cohort turned sixteen (or as of February 2019, whichever came first). Shading denotes the year the girls in the cohort turned twelve (darkest), thirteen, fourteen, and fifteen (lightest). 2019 data are for January and February.

Appendix to Chapter 3

3.A Statistical model

3.A.1 Model

We index observations by cohort $c = 1997, \dots, 2006$ and by month of eligibility $i = 1, \dots, 48$. Calendar time is given by $t = \tau(c, i) = c + 12 + \frac{i-1}{12}$. The cornerstone of the model is that the expected number of vaccinations follows

$$\frac{\mathbb{E}x_{c,i}}{N_c} = \delta_{\tau(c,i)} \gamma_i, \quad 0 < \delta_{\tau(c,i)} \gamma_i < 1,$$

and the statistical model is:

$$x_{c,i} \sim \text{Poi}(\lambda_{c,i}), \quad \lambda_{c,i} = \delta_{\tau(c,i)} \gamma_i N_c.$$

The parameters of interest are δ_t , where t runs from January 2009 to February 2019, whereas the seasonal and age specific parameters, γ_i , $i = 1, \dots, 48$, are nuisance parameters.

The conditional distribution of $x_{1997,i}, \dots, x_{2006,i}$ given $x_{\bullet,i} = \sum_{c=1997}^{2006} x_{c,i}$ has probability mass function,

$$\frac{\Pr\{x_{1997,i} = y_{1997}, \dots, x_{2006,i} = y_{2006}, \sum_c x_{c,i} = Y\}}{\Pr\{\sum_c x_{c,i} = Y\}}, \quad (3.1)$$

where the numerator equals

$$\begin{cases} e^{-\sum_c \lambda_{c,i}} \frac{\prod_c \lambda_{c,i}^{y_c}}{\prod_c (y_c!)} & \text{if } \sum y_c = Y \\ 0 & \text{otherwise,} \end{cases}$$

and the denominator equals

$$e^{-\sum_c \lambda_{c,i}} \frac{(\prod_c \lambda_{c,i})^Y}{Y!}.$$

If we define $\lambda_{\bullet,i} = \prod_c \lambda_{c,i}$, then it follows that the conditional mass function in (3.1) simplifies to

$$\frac{Y!}{\prod_c (y_c!)} \frac{\prod_c \lambda_{c,i}^{y_c}}{(\lambda_{\bullet,i})^Y} = \frac{Y!}{\prod_c (y_c!)} \prod_c \left(\frac{\lambda_{c,i}}{\lambda_{\bullet,i}} \right)^{y_c},$$

which is the multinomial distribution function with parameters

$$p_{c,i} = \lambda_{c,i} / \lambda_{\bullet,i} = \frac{\delta_{\tau(c,i)} N_c}{\sum_{c'} \delta_{\tau(c',i)} N_{c'}} = \frac{\delta_{\tau(c,i)} N_c}{W(\boldsymbol{\delta}, i)},$$

where $W(\boldsymbol{\delta}, i) = \sum_{c'} \delta_{\tau(c',i)} N_{c'}$. Conveniently, the parameters in the multinomial distribution do not depend on the nuisance parameters γ_i , $i = 1, \dots, 48$, and we can proceed to estimate the parameters of interest, $\boldsymbol{\delta} = \{\delta_t\}$, by maximizing the log-likelihood function defined by the forty-eight multinomial distributions.

3.A.2 Estimation

The conditional log-likelihood is given as

$$\sum_{i=1}^{48} \left[\log(x_i!) - \sum_c \log(x_{c,i}!) + \sum_c x_{c,i} \log(\delta_{\tau(c,i)} N_c) - \sum_c x_{c,i} \log(W(\boldsymbol{\delta}, i)) \right].$$

Multiplying every δ_t by the same positive constant does not change the likelihood. So, without loss of generality, we can introduce a normalization. We adopt the normalization $\frac{1}{n_1} \sum_{t \in T_1} \delta_t = 1$, which enables us to interpret δ_t as uptake in period t relative to average baseline uptake.

The derivative of the log-likelihood with respect to δ_t , which defines the first order condition for the estimation problem, is given by

$$\frac{1}{\delta_t} x(t) - \sum_i x_{\bullet,i} \frac{W'_t(i)}{W(\boldsymbol{\delta}, i)},$$

where

$$W'_t(i) = \frac{\partial W(\boldsymbol{\delta}, i)}{\partial \delta_t} = \sum_{c': \tau(c', i) = t} N_{c'},$$

and $x(t) = \sum_{i, c: \tau(c, i) = t} x_{c, i}$ denotes the number of girls being vaccinated in month t (across all cohorts) and $x_{\bullet, i} = \sum_c x_{c, i}$ is the number of girls that were vaccinated in the i -th month of eligibility across all cohorts.

For estimation and inference, it is useful to parameterize the model with unconstrained parameters. We achieved this with $\tilde{\delta}_t = \log \delta_t$, which is known as the canonical link for the Poisson distribution. Using the chain rule, we obtain the derivative of the log-likelihood function with respect to $\tilde{\delta}_t$, which is:

$$x(t) - \delta_t \sum_i x_{\bullet,i} \frac{W'_t(i)}{W(\boldsymbol{\delta}, i)}.$$

3.A.3 Inference

We seek confidence intervals for the average vaccine uptake in period k , $d_k = n_k^{-1} \sum_{t \in T_k} \delta_t$, $k = 1, \dots, 4$, and average vaccine uptake relative to baseline, $r_k = d_k/d_1$, $k = 2, 3, 4$. Standard errors for d_k and r_k are deduced from an estimate, $\hat{\mathbf{V}}$, of the variance-covariance matrix for $\boldsymbol{\delta}$. To this end, we use the misspecification-robust formula $\hat{\mathbf{V}} = \hat{\mathbf{A}}^{-1} \hat{\mathbf{B}} \hat{\mathbf{A}}^{-1}$, where $\hat{\mathbf{A}}$ and $\hat{\mathbf{B}}$ are both estimates of the Fisher information, based on the second derivative of the log-likelihood

and the variance of the score, respectively. Their elements are defined by

$$\hat{\mathbf{A}}_{s,t} = \frac{\partial}{\partial \delta_s} \left(\delta_t \sum_i x_{\bullet,i} \frac{W'_t(i)}{W(\boldsymbol{\delta}, i)} \right),$$

and

$$\hat{\mathbf{B}}_{s,t} = \sum_i \left(x_{c(s,i),i} - \delta_s x_{\bullet,i} \frac{W'_s(i)}{W(\boldsymbol{\delta}, i)} \right) \left(x_{c(t,i),i} - \delta_t x_{\bullet,i} \frac{W'_t(i)}{W(\boldsymbol{\delta}, i)} \right),$$

where $c(t, i)$ is the cohort index pertaining to calendar month t and the i 'th month of eligibility, and where we let $x_{c(t,i),i} = 0$ where $c(t, i)$ does not exist.

3.A.4 Confidence intervals for period averages

The time periods are indexed by $k = 1, 2, 3, 4$, and we seek confidence intervals for the average vaccine uptake,

$$d_k = n_k^{-1} \sum_{t \in T_k} \delta_t = n_k^{-1} \sum_{t \in T_k} \exp(\tilde{\delta}_t),$$

where T_k are the time periods pertaining to period k . For this, we invert the Wald statistic for the nonlinear restriction

$$a(\tilde{\boldsymbol{\delta}}) = d_k - d = 0,$$

solving for d . The Wald statistic is given by

$$a(\tilde{\boldsymbol{\delta}})^2 \left(\mathbf{a}'(\tilde{\boldsymbol{\delta}}) \hat{\mathbf{V}} \mathbf{a}'(\tilde{\boldsymbol{\delta}})' \right)^{-1} \stackrel{A}{\approx} \chi_1^2.$$

The elements of $\mathbf{a}'(\tilde{\boldsymbol{\delta}})$ are

$$\frac{\partial \log(d_k)}{\partial \tilde{\delta}_t} = \begin{cases} n_k^{-1} \delta_t, & t \in T_k, \\ 0 & \text{otherwise.} \end{cases}$$

The upper and lower end point of the $1-\alpha$ confidence interval is determined from the α -quantile, q_α , of the χ_1^2 distribution. We report 95% confidence intervals, which are based on $q_{5\%} = 3.84$.

$$\text{CI}_{d_k} = d_k \pm \sqrt{q_\alpha \left(\mathbf{a}'(\tilde{\boldsymbol{\delta}}) \hat{\mathbf{V}} \mathbf{a}'(\tilde{\boldsymbol{\delta}}) \right)}.$$

3.A.5 Confidence intervals for relative period averages

For uptake relative to baseline, the quantity of interest is

$$r_k = \frac{n_k^{-1} \sum_{t \in T_k} \delta_t}{n_1^{-1} \sum_{t \in T_1} \delta_t}.$$

We obtain confidence intervals about r_k by inverting the Wald statistic,

$$a(\boldsymbol{\delta}) = \log(r_k) - r = 0,$$

and solving for r , where $a(\boldsymbol{\delta})$ is the corresponding nonlinear restriction, for which $\mathbf{a}'(\boldsymbol{\delta})$ is given by,

$$\frac{\partial \log(r_k)}{\partial \tilde{\delta}_t} = \begin{cases} \frac{\delta_t}{\sum_{t \in T_k} \delta_t}, & t \in T_k, \\ -\frac{\delta_t}{\sum_{t \in T_1} \delta_t}, & t \in T_1, \\ 0 & \text{otherwise.} \end{cases}$$

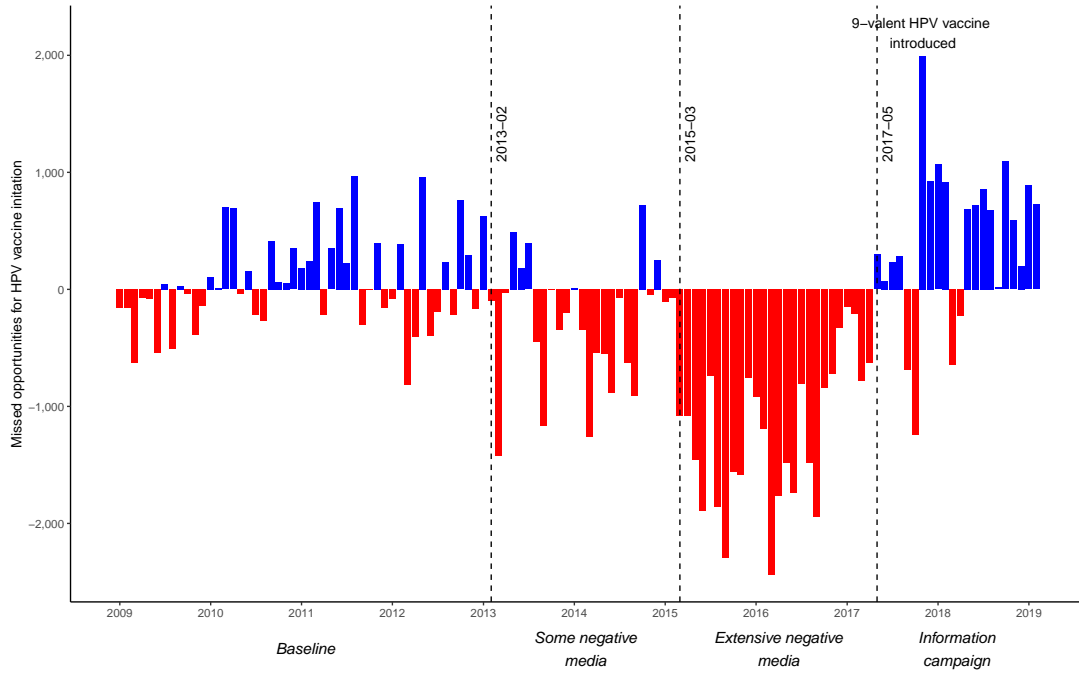
Following the same steps as with the confidence intervals for average uptake, we obtain confidence intervals for relative uptake:

$$\text{CI}_{r_k} = \exp \left(\log(r_k) \pm \sqrt{q_\alpha \left(\mathbf{a}'(\tilde{\boldsymbol{\delta}}) \hat{\mathbf{V}} \mathbf{a}'(\tilde{\boldsymbol{\delta}}) \right)} \right).$$

3.B Additional empirical results: missed opportunities

Figure 3.B.1 shows the missed opportunities for HPV vaccination per month, relative to the average time at which the first dose of HPV vaccine was received by birth year cohorts 1997-1999. Figure 3.3 presents the cumulative number of missed opportunities.

Figure 3.B.1. Missed opportunities



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