Biosimilar Entry and the Pricing of Biologic Drugs*

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Abstract

Biosimilars are the generic equivalent for biologic drugs, a group of complex pharmaceutical products that cannot be exactly replicated. The US passed regulation promoting biosimilar entry in 2010. Since then, more than 30 biosimilar products have been approved. We study how biosimilar entry has affected the price, volume sold, and formulary placement of biologic drugs, finding that the reaction of reference biologic products to biosimilar entry is much more aggressive than the reaction of traditional brand drugs to generic entry. We then present a simple model to argue that differences in the perception of biosimilar products can explain this discrepancy. The model and empirical results suggest a positive outlook for biosimilars, provided that the current medical evidence on their safety and efficacy is confirmed.

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1 INTRODUCTION

Pharmaceutical market regulation grants temporary monopoly power to new drugs. Once this protection expires, identical generic competitors quickly enter the market at much lower prices, capturing all but a small share of the market previously held by the original brand drug. The economic rationale behind the regulation is well known: society trades off the temporary inefficiency of a monopoly in order to incentivize the development of valuable innovation. The natural life-cycle of pharmaceutical products however, breaks down in the case of biologic drugs: large molecules that are grown from organic tissue (as opposed to the more traditional chemically-synthesized small-molecule drugs). The production process of biologic drugs is so complex that it is impossible to replicate exactly. This prevents the manufacturing of identical generic copies even after patent expiration. As a result, original biologic manufacturers can maintain monopoly power indefinitely.

The US government attempted to address this issue by passing the Biologics Price And Competition Act (BPCIA) in 2009. The BPCIA created a preferential approval pathway—similar to the one already in place for generic drugs—for so-called *biosimilar* products: copies of a reference biologic that are identical up minor differences in clinically inactive components. While generics can claim to be exact copies of their reference product, biosimilars cannot. The existence of these differences, however small, leaves uncertainty over the impact of biosimilars and the competitive reaction of reference biologics.

In this paper, we study how reference biologic products in the US have reacted to the approval and entry of biosimilar products. Our evidence suggests that reference biologics actively fight biosimilar entry by lowering prices in an attempt to maintain volume and formulary status. This response stands in stark contrast to how brand products react to generic entry: brand drugs never attempt to compete on price with generic entrants, preferring instead to maintain prices and sell to a small number of patients with strong brand preferences. Building on the model of brand and generic competition by Frank and Salkever (1992), we show that greater differences in perceived value and similarity between an incumbent drug and a same-molecule entrant can lead to lower equilibrium prices but higher quantity for the incumbent, and lower quantity and profits for the entrant. The model suggests that reference biologics will be less likely to compete on price with biosimilar products for which there is greater available evidence of efficacy. We exploit the fact that some biosimilars had to go through a more rigorous approval process to show empirical evidence consistent with this prediction.

To test the impact of biosimilar entry we use a generalized difference-in-differences framework with heterogeneous timing of treatment. A drug in our treatment group is

"treated" when its first biosimilar is launched on the US market. Other biologics and brand drugs in the same therapeutic class serve as the control group. The outcomes we examine come from a rich dataset on pricing and access to biologic drugs that includes information on list prices, estimated net prices, volume sales, and formulary status.

We find that after biosimilar entry reference biologics experience a significant fall in net price of about 20%, a noisy decline in volume sales, and a small but significant decline in formulary coverage. The decline in net price comes entirely from increased discounts: we do not see any change in list prices. These results suggest that reference biologics react to biosimilar entry by increasing concessions to PBMs and payers in order to maintain their formulary status, and, in turn, sales volume. Overall, their efforts seem to be relatively successful, as we detect only small changes in formulary status, and noisy changes in volumes.

These results suggest that biosimilars generate a very different competitive response than generic drugs. We explore possible economic explanations for these differences using a model of biosimilar entry that builds on the seminal model of brand and generic competition in Frank and Salkever (1992). In the model, an incumbent serves both a "loyal", and a "price-sensitive" segment of the market, and faces competition from a same-molecule entrant in the price-sensitive segment. We model competition in the price-sensitive market as a Hotelling model where the entrant differs both because of travel cost and because it can have lower vertical quality.

The model shows that the incumbent will retreat to the loyal segment of the market if the there is not enough differentiation between its product and that of the entrant. Combined with the empirical results, this intuition suggests that reference biologics may compete more aggressively with biosimilar entrants because of a perceived gap in quality, and potential horizontal differentiation between the two products.

An important implication of the model is that same-molecule competitors that are very similar to their originator should capture a larger market share, and potentially elicit a weaker price response. We test this prediction by comparing price and volume changes for four reference biologics of biosimilar products approved through the regular approval process.[1] We find that in these four cases, the reference biologics lost a much higher share of their pre-entry volume, and barely changed their price.

The model and empirical analysis highlight the important role played by reference biologics in limiting market penetration of biosimilars, a key factor in determining their

[1]There are four such products. Two of these were approved before the BPCIA was passed. The other two are insulin product, which—despite insulin being a biologic drug in nature—the FDA did not classify as biosimilar until recently. See Section 3.2 for more details on these four drugs.

profitability. Medical studies however, suggest a high degree of substitutability between reference biologics and their biosimilar counterparts. If this is true, our results suggest disseminating this information could go a long way towards ensuring profitability of biosimilars. Even without specific interventions, we expect that as patients and providers become more familiar with biosimilars over time, their impact on equilibrium outcomes will more closely resemble that of generic drugs.[2]

This paper adds to the growing literature on the impact of biosimilars by being, to our knowledge, the first systematic study looking at the US market. This literature contains several works on biosimilars in Europe (Scott Morton et al., 2018), as well as a number of case studies (Atteberry et al., 2019; Karaca-Mandic et al., 2019). We contribute to this literature by focusing on reference biologics and how their competitive choices affect the impact of biosimilar products.

More broadly, this work contributes to the literature on competition in pharmaceutical markets. In particular, we join a series of recent papers showing that strategic behavior of pharmaceutical companies is rarely reflected in widely available list prices, but instead becomes apparent only through movements in net price, which are much harder—but not impossible—to track (see e.g. Feng et al., 2020; Kakani et al., 2020).

The rest of the paper proceeds as follows. Section 2 provides some background on biologic and biosimilar drugs. Section 3 presents the data, the main empirical analysis, and the results. Section 4.1 introduces the model of competition between an incumbent and a same-molecule entrant, and discusses its implications. Section 5 concludes.

2 Characteristics and Regulation of Biologic Drugs

Biopharmaceutical products, more commonly known as biologic drugs or biologics, are large, complex molecules derived from living sources. Examples of biologic drugs include insulin, interferons (used to treat multiple sclerosis, some types of cancers, and hepatitis), and monoclonal antibodies (used to treat rheumatoid arthritis, psoriasis, and ulcerative colitis).[3]

[2]Pockets of the US health care system have already managed to achieve penetration rates on par with those of generic drugs. See e.g. the example of Kaiser Permanente in California: https://biosimilarsrr.com/2019/11/07/how-did-kaiser-permanente-reach-95-utilization-of-biosimilar-herceptin-and-avastin-so-quickly/, retrieved November 12, 2020.

[3]The oldest examples of biologic products are vaccines, the first of which was the smallpox vaccine developed in 1796 by Edward Jenner, a British doctor. It consisted of a injection of a milder virus in the same family. In the late 19th century, American Home Products—a predecessor of the pharmaceutical company Wyeth—became the first company to mass-produce a version of the smallpox vaccine. Called Dryvax, their product was a live-virus preparation of another virus in the smallpox family: the vaccinia virus, from which all vaccines take their name.

Biologics cannot be replicated exactly, due to their complexity. This makes them fundamentally different from more traditional, "small-molecule" drugs, which are synthesized using perfectly replicable chemical processes. Biologics are grown inside living organisms that have very specific cell lines, different from those of other organisms of the same species. Only the company that manufactures the reference biologic has access to the specific cell line that produced the original drug, and therefore no other company can ever create an exact copy. It is still possible to create a "biosimilar" version of an existing biologic drug: a slightly different drug, containing the same active ingredient, but that may present small differences in clinically inactive components from its reference biologic.[4]

The impossibility to reproduce exact copies of biologic drugs means that most existing regulation managing drug competition over the life-cycle of pharmaceutical products does not apply to biologics. For example, generic drugs have been able to take advantage of an accelerated approval process since the passage of the Hatch-Waxman Act of 1984. The Abbreviated New Drug Application (ANDA) process waives the requirement to provide clinical trial data for generic products. Biosimilar drugs cannot apply through the ANDA process, and, until recently, would have had to undergo testing as if they were a completely new drug.

The Biologics Price Competition and Innovation Act (BPCIA) of 2009 attempts to address the gaps in the regulation of biosimilars by creating an abbreviated approval pathway for these drugs. Under the BPCIA, there are two main requirements for approval of a biosimilar product: (i) a presentation of chemical data showing the product is highly similar to the reference biologic, (ii) evidence of equivalence with the reference biologic from a clinical trial (Timmis, 2015). Crucially, this is a requirement that does not apply to generic drugs. Even though equivalence trials are generally smaller and cheaper to run than the pivotal trials for new molecule applications, they represent a significant barrier for potential entrants.

While the BPCIA makes it easier to bring them to market, biosimilars still face three significant hurdles relative to generic drugs. First, the cost of developing a biosimilar is still much higher than the cost of developing a generic. Informal estimates place the cost of developing a biosimilar drug anywhere between from $100 million to $250 million, while the development of a generic small molecule drug costs about $2-$5 million

[4]As defined by the Food and Drug Administration (FDA), a "biosimilar" is a biologic that is highly similar to and has no clinically meaningful differences from an existing reference or innovator biologic that is already in production. The phrase "meaningful differences" refers to the efficacy and safety of the biosimilars in comparison to the reference biologic.

(Grabowski et al., 2014).[5] Second, biosimilars face much higher risk of litigation from the company producing the reference biologic. Biologic drugs are protected by a much greater number of patents relative to small molecule drugs, and infringement of any of these patents is enough to prevent the biosimilar from coming to market (Chen et al., 2017). Third, biosimilars cannot take advantage of regulation that promotes use of generic products, such as mandatory substitution laws.[6]

3 Impact of Biosimilar Entry on Price, Volume, and Formulary Placement of Reference Biologics

3.1 Data

We combine four different data sources to create a comprehensive database of drug prices, sales, and formulary placement.

First, we use data on invoice sales, net sales, and volume sales from SSR Health. The SSR Health database covers roughly 1,000 brand drugs and contains data quarterly data on Wholesale Acquisition Cost (WAC), quantity sold, and net sales from 2007 to 2019.[7] WAC is an estimate of the manufacturer's list price for a drug to wholesalers or distributors, but does not take into account any rebates or discounts. To obtain an estimate of net price we divide reported net sales—which are comprehensive of all discounts and rebates—by units sold. The main downside of this dataset is that it only includes data on branded drugs—it does not report data on biosimilar sales or price.

As a second source of price information, we use Average Sales Price (ASP), a publicly available measure of net price published by the Centers for Medicare and Medicaid Services (CMS). Data on ASP is available starting in 2005 and on quarterly basis. Unlike other measures of price, ASP incorporates rebates to commercial payers, though it excludes rebates to Part D, the Department of Veteran Affairs, and Medicaid, as well as a few other minor exceptions.[8] CMS also aggregates ASP at the level of a Healthcare Common Proce-

[5]For the estimation of developing a biosimilar drug see remarks made by FDA Commissioner Scott Gottlieb to the America's Health Insurance Plans' National Health Policy Conference on March 7, 2018 (https://www.fda.gov/news-events/speeches-fda-officials/capturing-benefits-competition-patients-03072018, retrieved November 3, 2020).

[6]In general, substitution laws cannot have the same impact on biosimilars even if passed, because they apply to sales at point of retail in pharmacies, and most biologic drugs are not sold through pharmacies, but administered by physicians in outpatient settings.

[7]SSR Health sources data on WAC and units sold from Symphony Health, and collects net sales figures from the SEC filings of publicly traded companies. For further discussion of the coverage of the SSR Health data see the data section of Feng et al. (2020).

[8]This exclusion is stated in the Code of Federal Regulations: "In calculating the manufacturer's average sales price, a manufacturer must exclude sales that are exempt from inclusion in the determination of the

dure Coding System (HCPCS). The HCPCS code for drugs is usually unique for a given molecule.[9] This is usually not an issue for brand products that are still patent protected, but it can be an issue for products whose patent has expired.

Our third source of information comes from MMIT Analytics, a pharmacy advisory company, from which we obtain formulary coverage data spanning January 2011 through June 2020. A formulary is a list of drugs covered by an insurance plan. Formularies divide drugs into several categories of coverage called tiers. While tier design can change from plan to plan, most formularies include at least two tiers of brand coverage: a preferred tier for brands that grant higher discounts, and a separate tier for all remaining ones. Drugs in the preferred tier have lower copays, while drugs in higher tiers have the higher copays, or, occasionally, coinsurance rates. Formularies can also place non-monetary restrictions on drugs. Two frequent restrictions are Prior Authorization (PA), which requires physicians to submit a written form to the insurer before authorizing a prescription, and Step Therapy (ST) which authorizes using a drug only only after an alternative therapy has failed. Using the MMIT data we construct three measures of coverage: the fraction of lives whose formulary offers (i) any coverage, (ii) unrestricted coverage, or (iii) preferred coverage of a given drug. To create these measures, we follow a procedure outlined in Geruso et al. (2019).[10]

Fourth, and finally, we collect biosimilar approval and launch dates from the FDA's website and from public announcements of pharmaceutical companies, respectively.

3.2 *Empirical Strategy and Summary Statistics*

Since different drugs experience biosimilar entry in different periods, we use a generalized difference-in-difference approach (Imbens and Wooldridge, 2009). Our main specification is

$$y_{it} = \beta \times \mathbb{I}\{\text{Biosimilar has entered}\} + \theta_i + \gamma_a + \delta_t + \varepsilon_{it} \tag{1}$$

Our dependent variables are measures of price, volume sales, and formulary coverage. We control for drug fixed effects θ_i in all regressions, which is important given the large variance in outcomes across drugs. In regressions where the dependent variables measure price or formulary coverage we also control for quarter fixed effects δ_t. In regres-

best price under section 1927(c)(1)(C)(i) of the Act" (42 CFR § 414.804 (a)(4)(i)). The cited section refers to 42 CFR § 447.505, which defines best price for the purpose of Medicaid reimbursement, and specifies the exemptions we mention in the main text.

[9] One of the most important exceptions to this rule is insulin, which has a unique HCPCS code combining all rapid-acting and long-acting insulin products.

[10] For a more detailed discussion of the MMIT data or of how we create these variables please refer to Appendix A.

Table 1: Summary Statistics for Treatment and Control Groups

	Biologics w/ Biosimilar		Control Group	
	Mean	Std. Dev.	Mean	Std. Dev.
# of Drugs	7		31	
Invoice Sales in 2018 (million USD)	1,152.06	897.26	643.16	968.51
Net Sales in 2018 (million USD)	456.29	368.82	431.22	645.02
% Covered	87.86	7.77	87.15	9.73
% Preferred	18.75	18.14	15.41	16..17
% Unrestricted	27.23	17.41	23.76	17.20

sions where the dependent variable measures volume we control for age fixed effects γ_a instead.[11] We also test slightly different specifications where we add a post-entry linear trend, and indicators for the number of biosimilars in the market. We cluster standard errors at the drug level throughout our analysis.

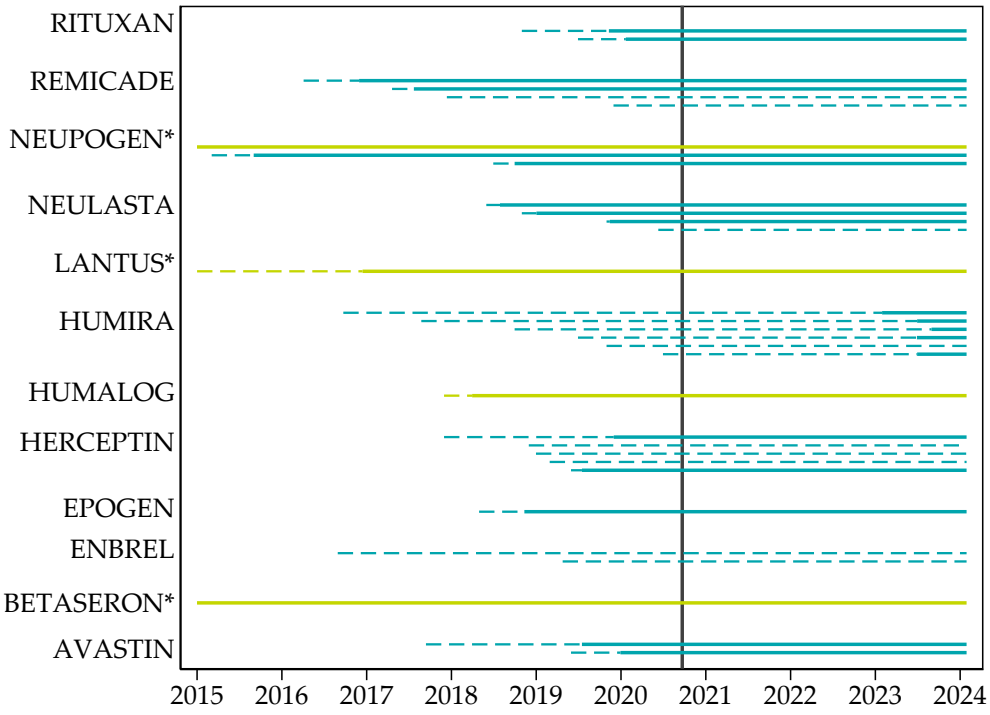
We include in the control group all drugs in the same therapeutic class as the biologics that experience biosimilar entry. Overall, our regressions include seven biologic drugs that experienced biosimilar entry at some point between 2007Q1 and 2019Q3, and thirty-one drugs that did not. Table 1 displays some basic summary statistics for these two groups. Biologics that experience biosimilar entry tend to have slightly higher net sales and formulary coverage, and much higher invoice sales. This is not too surprising, as we would expect biosimilars to challenge more successful products.

Figure 1 plots the approval and launch timeline of all thirty-one biosimilars approved for use in the United States as of July, 2020. A dashed line in the graph represents a product that has been approved, but not launched, while a solid line represents a launched product. One noticeable pattern is the long delay between approval and launch, which often lasts multiple years. Only twenty of these biosimilars are currently available for sale. Some of the drugs, like Amjevita (a biosimilar of Humira), have not launched due to agreements with the producer of the reference biologic (Barlas, 2019). Others are still tied up in litigation.

Four of the products we include are biosimilars but were not approved through the accelerated pathway introduced by the BPCIA. These are Admelog (biosimilar of Humalog), Basaglar (Lantus), Extavia (Betaseron), and Granix (Neupogen). Extavia and Granix

[11] We measure age as quarters since US launch date. While controlling for both age and quarter FE may be preferable, the two sets are highly collinear, and can lead to overfitting on limited data. Prices tend to increase over time, while formulary coverage tends to decrease over time, regardless of age. Conversely, volume follows an inverted-U shape, making it more important to flexibly control for age effects.

Figure 1: Biosimilar Approval and Launch Timeline



Note: Solid lines represent a biosimilar's presence in a market, while dashed lines represent a biosimilar's approval by the FDA. Biosimilars represented by blue lines were approved through BPCIA's abbreviated pathways, while those represented by green lines were approved through a different process.

applied for marketing approval before the abbreviated pathway was introduced.[12] Admelog and Basaglar are insulin products, which the FDA did not consider as biologics until 2020.[13] As a result they were approved through a pathway called 505(b)(2), which is usually reserved for follow-on products of existing brands.

3.3 Impact on Price and Volume

We start by examining the effect of biosimilar entry on price and volume of reference biologics. We use three measures of price. WAC is our measure of list price. To measure price net of rebates we use both data from SSR Health (net sales divided by volume sales)

[12]Extavia was approved in 2009. Granix was approved in 2012, but submitted its first application to the FDA in 2009 (see https://www.drugs.com/nda/xm02_091202.html, retrieved November 10, 2020).

[13]While insulin *is* a biologic drug, it was first marketed in the US before the word "biologic" entered in the official US regulation terminology. As a result, it was not considered as such. The FDA closed this loophole in February of this year, by publishing a rule that included insulins among the products eligible for approval under the abbreviated BPCIA pathway (see https://www.fda.gov/news-events/press-announcements/fda-works-ensure-smooth-regulatory-transition-insulin-and-other-biological-products, retrieved November 10, 2020).

and ASP data from CMS. We measure volume simply as a count of units sold.[14]

Table 2 displays our results. The overall takeaway from the analysis is that biosimilar entry is associated with a borderline significant decrease in net price and ASP, but no significant change in list price (measured by WAC) or volume.[15] Results vary in significance across specifications, but overall, it appears that net price drops immediately after biosimilar entry, and increasingly so as more biosimilars are approved. ASP also drops, but appears to do so more gradually, and by a lower amount. Entry of multiple biosimilars also appears to have some impact on WAC.

Since biosimilar entry is not random, it's possible that selection into biosimilar development could bias our estimate. However, these concerns generally suggest that we will probably underestimate the coefficients we are interested in. We expect biosimilars to enter in markets with relatively higher and faster-growing sales. We can partially control for the level of sales using drug fixed effects, but we cannot adequately control for future growth. Drugs can have unobservably higher future sales because of growing volume, growing price, or both. This means that any negative impact of biosimilars on the reference biologic's volume and price will be attenuated by the growing path of the market for that molecule, which would lead to a downward bias on our estimated coefficient.

To verify that the regression results are not merely capturing secular trends in price or volume, we run a non-parametric version of equation 1, where we include indicators for each quarter post-biosimilar entry. We plot our results for price measures in Figure 2, and for volume in Figure 3.[16] The results confirm our original impression. Net price falls somewhat faster than ASP, though they both end up at a similar level. WAC is essentially flat. Neither line seems to display significant pre-trends. We do see a negative pre-trend in volume, though the line seems to flatten out about two years prior to biosimilar entry. This pre-trend is also the opposite of what we would expect based on economic intuition: biosimilars should enter in markets with growing volume. Regardless, the plot confirms the overall impression from the regressions, though volume appears to fall slightly two

[14]Units vary across drugs, but this does not matter for our regression because we analyze percentage effects and also control for drug fixed effects. In one case, the units of a product (Herceptin) changed in the middle of our sample as the original 440mg unit was replaced by a smaller 150mg unit in 2017Q2. To keep prices consistent we adjust units by a $44/15$ factor in all quarters following the change.

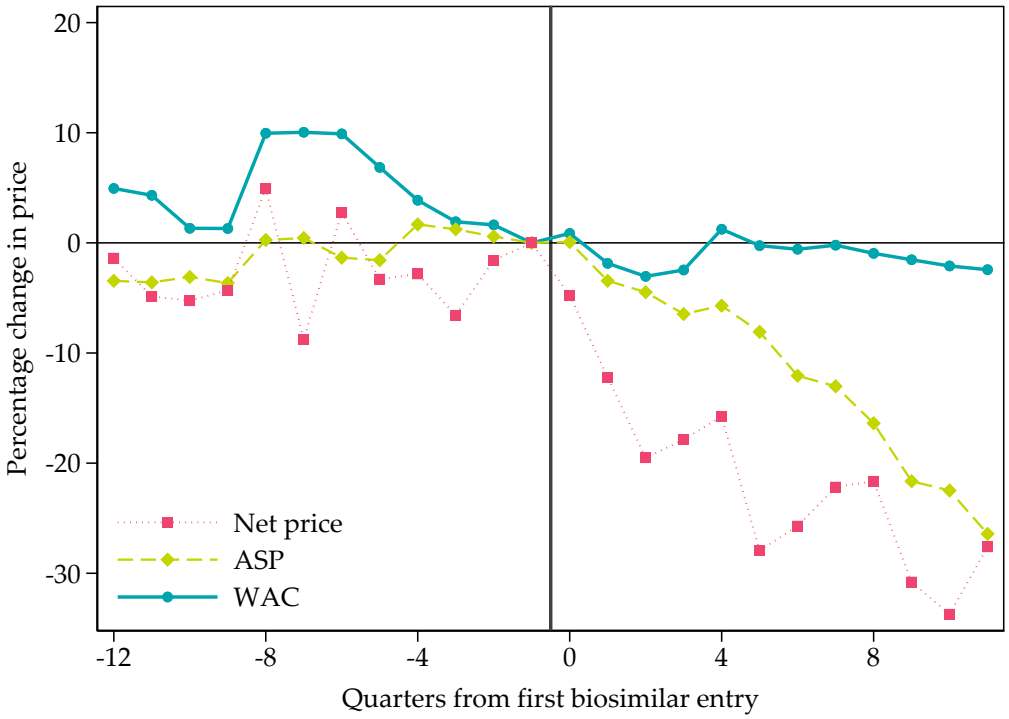
[15]The phenomenon of WAC, or list price, not falling after a biosimilar enters has been widely reported in the media and may have contributed to skepticism about biosimilars. For example, Atteberry et al. (2019) use the absence of response in WAC after the entry of a filgrastim biosimilar as evidence that biosimilars are ineffective.

[16]We do not report standard errors for these quarterly plots because we do not have enough data to estimate them precisely in such a flexible model. However, we want to be transparent about the fact that almost none of these coefficients in these plots are likely to be significantly different from zero. This is due to the small sample size relative to the number of coefficients estimated.

Table 2: Impact of biosimilar entry on price and volume of biologics

	Log WAC			Log net price			Log ASP			Log units sold		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
Biosimilar has launched	-0.044	-0.080		-0.220	-0.271		-0.079	-0.026		-0.097	0.421	
	(0.127)	(0.088)		(0.149)	(0.114)		(0.071)	(0.068)		(0.324)	(0.248)	
Biosimilar launch trend		0.004			0.006			-0.014			-0.056	
		(0.007)			(0.009)			(0.006)			(0.005)	
1 Biosimilar launched			0.022			-0.150			-0.039			-0.331
			(0.113)			(0.187)			(0.073)			(0.310)
2 Biosimilars launched			-0.218			-0.288			-0.127			-0.028
			(0.123)			(0.147)			(0.082)			(0.471)
3 Biosimilars launched			-0.372			-0.548			-0.190			-0.668
			(0.138)			(0.164)			(0.091)			(0.490)
Product FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Quarter FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	N	N	N
Age FE	N	N	N	N	N	N	N	N	N	Y	Y	Y
N	1,482	1,482	1,482	1,410	1,410	1,410	679	679	679	1,482	1,482	1,482
R^2	0.981	0.981	0.981	0.968	0.968	0.968	0.999	0.999	0.999	0.955	0.959	0.956

Figure 2: Price Quarterly DID Coefficient Plots



years or so after biosimilar entry.

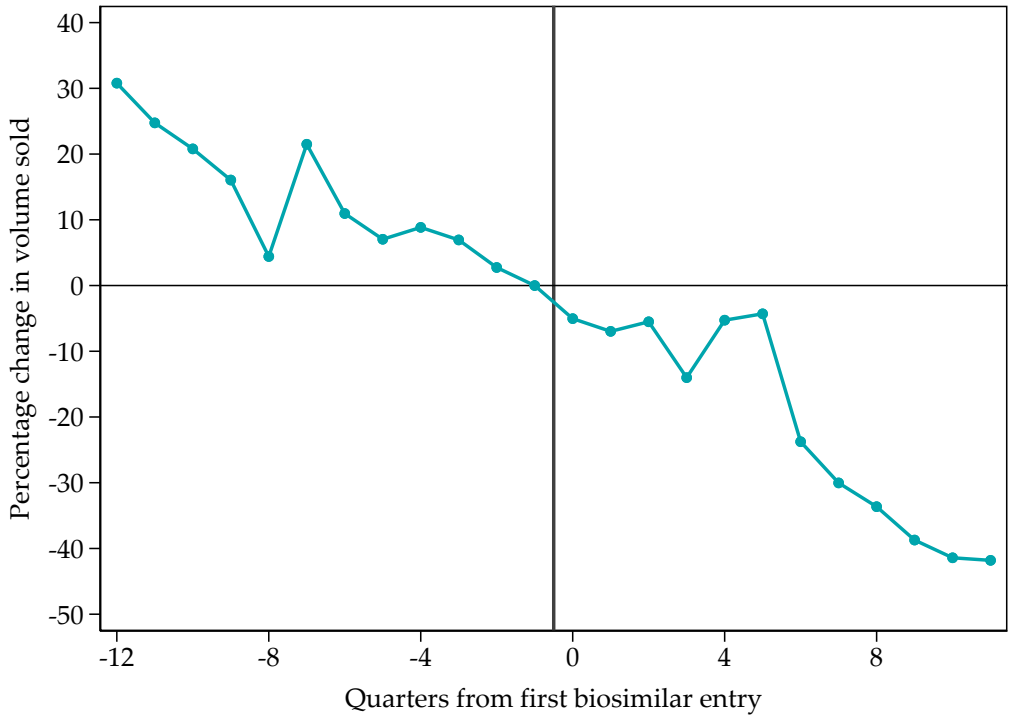
We also test the robustness of these results by running two sets of checks, which we report in Appendix C. First, we test whether our estimates are sensitive to including all biologic drugs. We find that even in this larger sample, our results are virtually unchanged. Second, we perform a placebo test where we add an indicator for biosimilar *approval*. If behavior prior to launch but after a biosimilar has been approved is correlated with any underlying trends in prices, volume, or formulary status then the approval indicator should pick up such trends. We find that approval of biosimilar is not associated with a significant change in any of our dependent variables.

3.4 Impact on Formulary Placement

Finally, we examine the impact of biosimilar entry on formulary placement. The dependent variables are measures of access to a given drug. We express these as the fraction of patients whose health plan offers any coverage, unrestricted coverage, or preferred coverage of a given drug (three measures in total). As controls we use drug and quarter fixed effects.

Table 3 shows our results. Biosimilar entry leads to worse formulary coverage across

Figure 3: Volume Quarterly DID Coefficient Plots



all three of our measures, though the most apparent impact is on preferred status, which falls by around 6 percentage points (off of a lower baseline than either preferred or unrestricted). As in the price and volume regressions, the effect of entry increases over time, and as more biosimilars are introduced.

We repeat the same robustness tests used for the price and volume regressions, and find broad consistency across all of them. Figure 4 plots quarterly coefficients from a non-parametric regression, and confirm the overall impression we get from the regressions results. Appendix C reports regression results using a broader sample of biologic drugs—we estimate slightly larger, but qualitatively consistent coefficients—and a placebo test that adds an indicator for biosimilar approval—which we find is not associated with a significant change in formulary coverage.

4 An Economic Theory of Competition with Biosimilar Drugs

Two main results emerge from our analysis of biosimilar entry. First, reference biologics react by lowering net price. Second, biosimilar do not appear to make a meaningful dent in the volume or formulary placement of reference biologics. Taken together, these results suggest that reference biologics are actively fighting biosimilar competitors, and are will-

Table 3: Impact of biosimilar entry on formulary coverage of biologic drugs

	% Covered			% Unrestricted			% Preferred		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
Biosimilar has launched	-5.733	-4.440		-2.900	-2.664		-6.282	-4.893	
	(3.885)	(4.002)		(1.858)	(2.314)		(3.203)	(3.370)	
Biosimilar launch trend		-0.249			-0.0455			-0.268	
		(0.223)			(0.213)			(0.224)	
1 Biosimilar launched			-7.658			-1.606			-6.203
			(4.033)			(1.870)			(4.204)
2 Biosimilars launched			-2.060			-4.462			-5.877
			(3.569)			(2.352)			(3.443)
3 Biosimilars launched			-16.87			-11.58			-15.74
			(2.552)			(2.674)			(2.678)
Constant	91.68	88.12	91.72	19.14	56.03	19.19	10.94	25.64	10.99
	(0.267)	(1.615)	(0.258)	(0.340)	(1.978)	(0.338)	(0.279)	(1.335)	(0.276)
Product FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Quarter FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
N	792	792	792	792	792	792	792	792	792
R^2	0.708	0.713	0.722	0.931	0.931	0.932	0.957	0.958	0.958

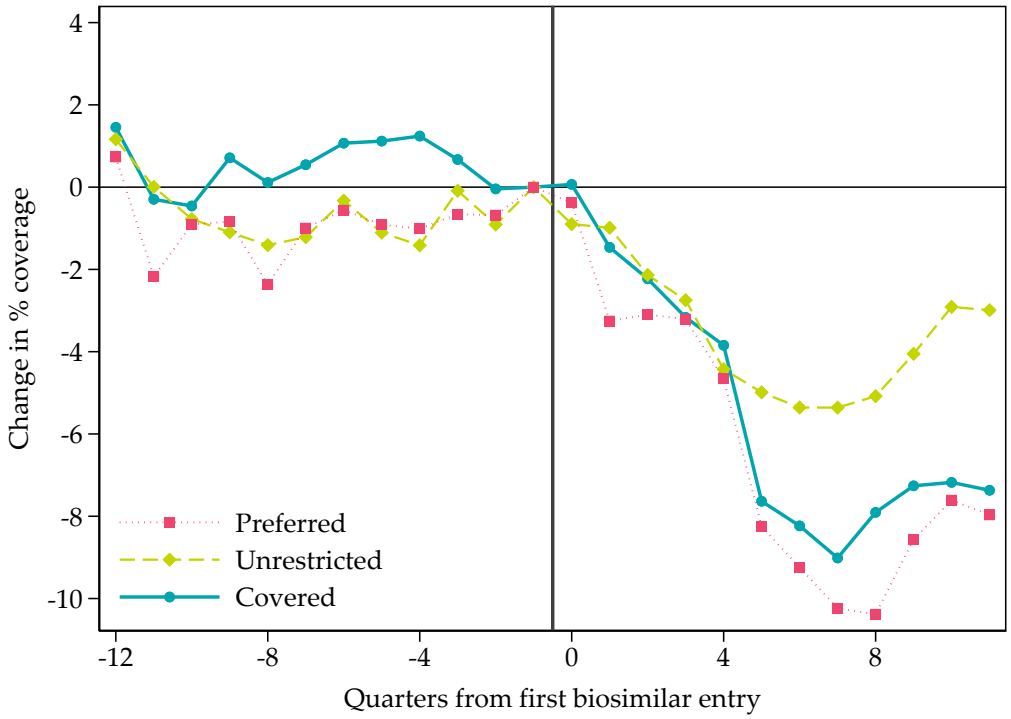
Note: Each of the outcomes is a percentage of lives on a health plan with a formulary status of covered, preferred, or unrestricted. Covered means that a drug is on the formulary and does not fall under an explicit "Not Covered" tier. The low constant for preferred models can be explained by the fact that drugs we flag as preferred are both preferred and unrestricted. For a further explanation of our tier assignment see Appendix A.

ing to grant large discounts in order to maintain their volume, and formulary position even after biosimilar entry.

This reaction stands in stark comparison to the reaction of small-molecule brand drugs to generic competitors. Empirical research dating back to the time of the Hatch-Waxman Act has consistently found that brand drugs essentially "acquiesce" to generic entry. Brand prices generally do not change or even increase after generic entry (Grabowski and Vernon, 1992; Berndt and Aitken, 2011), marketing activity ceases (Scott Morton, 2000), and volume falls by up to 90% within the first year of generic competition (Grabowski et al., 2016).

To rationalize this reaction, we present a model of competition between an incumbent originator and a same-molecule entrant, and argue that small differences between the incumbent and the entrant—perceived or otherwise—can justify a very different range of equilibrium outcomes. We then present suggestive summary statistics to support the model, and use it to provide an outlook for the future of biosimilars.

Figure 4: Formulary Quarterly DID Coefficient Plots



4.1 A Model of Strategic Response to Biosimilar Entry

The seminal model of competition between brand and generic products comes from Frank and Salkever (1992) (hereafter, FS), which conjectures that brand products may react to generic entry by keeping price constant, or even increasing it, in order to focus on the inelastic part of the demand curve. The paper models this "loyal" segment of the demand curve as having an inelastic preference for brand products. We build on the model by FS to explain why reference biologics may react differently than small-molecule brands to a same-molecule competitor.

The main intuition of the model is that perceived differences between an incumbent originator and a same-molecule entrant could weaken competition in the market, making it marginally more profitable for the incumbent to fight the entrant by competing on price. These perceived differences could be real—e.g. because biosimilars are not exactly identical to their reference biologic—or driven by an informational gap on the effectiveness and safety of the entrant, consistent with recent empirical evidence by Bronnenberg et al. (2015) on the purchasing patterns of informed and misinformed consumers. We present a full derivation of the paper in Appendix B, and focus on the setup and main results here.

We adopt the same generic setup as FS, but where FS leave demand functions largely

unexpressed, we make specific functional assumptions in order to parametrize the difference between generic and biosimilar competitors.

In the original setup of FS, the originator's demand function is divided in two segments, $D_L(p_b)$ and $D_S(p_b, p_g)$. The subscripts L and S respectively indicate a "loyal" segment of patients—who will never switch to a generic alternative—and a price-sensitive segment. p_b and p_g denote the price of the incumbent (brand, or biologic) product and the price of the (generic or biosimilar) entrant.

We model demand in the loyal market as a measure α of homogeneous consumers with valuation V_b for the brand product, and 0 for the generic product. We model demand in the price sensitive market as a simple Hotelling model of price competition. A measure 1 of consumers is distributed uniformly along a unit interval. The incumbent is exogenously located at 0, while the entrant is exogenously located at 1. Utility of purchasing the incumbent or the entry product for a consumer in location $x \in (0,1)$ is given by

$$U_b(x) = V_b - p_b - tx$$
$$U_g(x) = V_g - p_g - t(1-x)$$

where t is the travel cost, and $V_g \leq V_b$.

The overall maximization problem of the incumbent is[17]

$$\max_{p_b} p_b \left(D_L(p_b) + D_S\left(p_b, p_g^\star(p_b)\right)\right)$$

where

$$D_L(p_b) = \begin{cases} \alpha & \text{if } p \leq V_b \\ 0 & \text{otherwise} \end{cases}$$

and

$$D_S\left(p_b, p_g^\star(p_b)\right) = \min\left\{\max\left\{0, \frac{1}{2} + \frac{V_b - V_g - p_b + p_g^\star(p)}{2t}\right\}, \frac{V_b - p_b}{t}, 1\right\}$$

The segmented nature of the market implies that the incumbent has three options:

1. Capture only the loyal market, and stay out of the price-sensitive market

2. Capture the loyal market, and compete with the entrant over the price-sensitive

[17] Relative to the original problem in Frank and Salkever (1992) we omit the cost function, whose inclusion would not alter any of the implications of the model, and limit the number of generic competitors to one, since we are not interested in studying how number of competitors affected the response of the the firm.

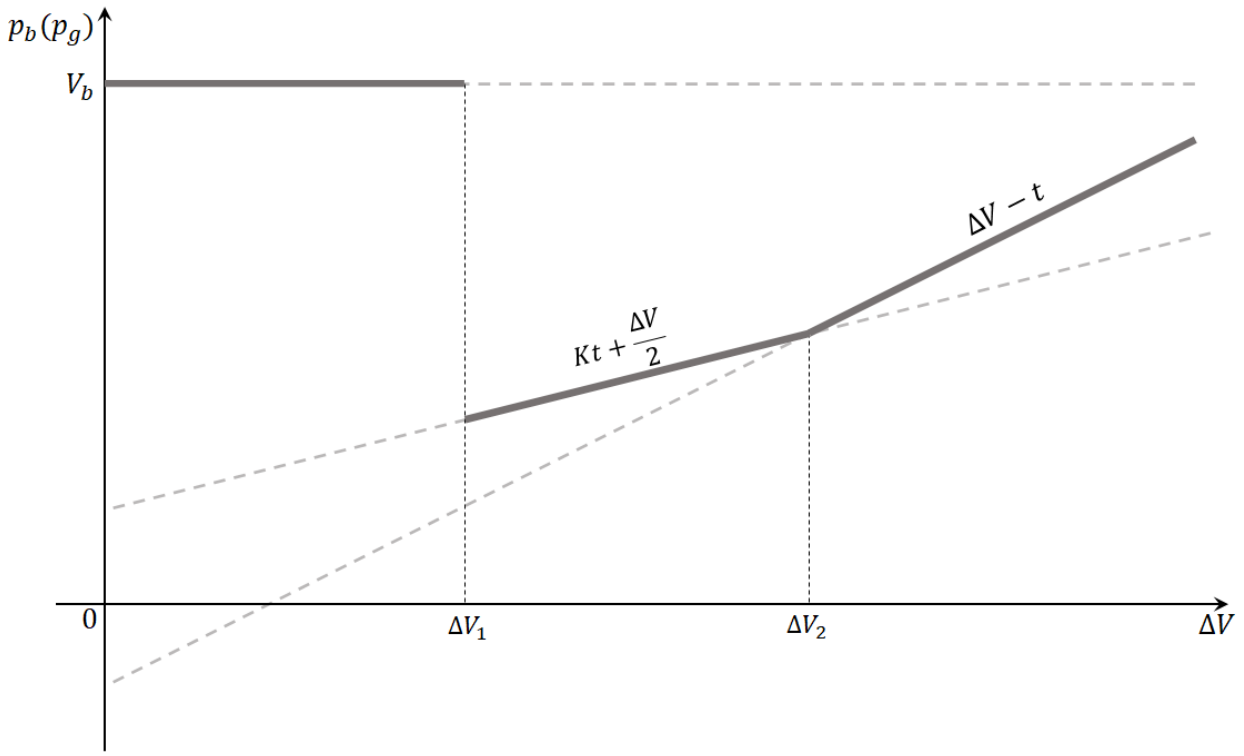


Figure 5: Optimal price of the incumbent as a function of ΔV

market

3. Capture the entire market

Which option is optimal depends on t and ΔV.[18]

Equilibrium price and profits are straightforward to calculate for options 1 and 3. If the incumbent focuses on the loyal market only, it will set price $p_1 = V_b$, netting profit $\Pi_1 = \alpha V_b$. To capture the entire market instead, the incumbent has to set price $p_3 = \Delta V - t$. Doing so nets profits $\Pi_3 = (1 - \alpha)\, p_1$. The equilibrium outcome in the competitive case is more complicated, but we show in Appendix B that the optimal price can be written as $p_2 = Kt + \frac{\Delta V}{2}$, with $K > 0$.

Figure 5 displays the optimal price as a function of ΔV for visual clarity. The key result is that, while price is generally increasing with respect to ΔV, a low enough value of ΔV generates a discontinuous jump in price as the incumbent retreats to the loyal segment of the market. The following theoretical results describe the best-response function of the incumbent and the thresholds ΔV_1 and ΔV_2 on the graph.

[18] It also depends on α, but we are not interested in the impact of market size.

Proposition 1. *There exist thresholds $\Delta V_1(t)$ and $\Delta V_2(t)$ such that for all $\Delta V < \Delta V_1(t)$, the incumbent sells only to the loyal market, and for all $\Delta V > \Delta V_2(t)$ the incumbent captures the entire market.*

We provide a formal proof of Proposition 1 in Appendix B, and discuss the intuition here. The first part of the proposition depends on the fact that a lower ΔV lowers profits and forces the incumbent to compete more aggressively on price in the price-sensitive market. At some point, the price required to compete will be low enough that it makes more sense to abandon the market altogether in order to be able to extract the maximum surplus from the loyal segment of the market. The second part of the proposition is equally intuitive. For large enough ΔV, the entrant stops representing credible competition in the price-sensitive market, and the incumbent can capture the entire market without needing to lower the price too much.

Corollary 2. *ΔV_1 is a weakly decreasing function of t, while ΔV_2 is a weakly increasing function of t. There exists a threshold $\bar{t}$ such that for all $t < \bar{t}$, $\Delta V_1(t) = \Delta V_2(t)$.*

The proof of this Corollary is also in Appendix B. The intuition behind this result is that a decline in t has two effects. On the one hand, it makes it easier for the entrant to compete and decreases overall profits for the incumbent. This makes it less worthwhile for the entrant to compete in the price-sensitive market, and makes it more likely that the incumbent will stay out of it altogether. On the other hand, it makes it easier for the incumbent to compete for consumers that are very close to the entrant. Hence, capturing the entire market becomes a relatively more attractive proposition than just competing with part of it. The combination of these two effects implies that a fall in t makes option 2 look worse than both option 1 and option 3. For t small enough option 2 will never be optimal, and the incumbent will either capture the entire market, or leave the price-sensitive market to the entrant altogether.

4.2 Evidence of Heterogeneous Response to Biosimilar Entry

One of the takeaways of the model is that a small differences in either vertical or horizontal quality between an incumbent and a same-molecule entrant can lead to vastly different price responses from the incumbent. Finding empirical variation in quality in this setting is difficult, because demand patterns are likely driven by perceived differences rather than objective measures. Our solution is to use variation in the amount of information available at the time of biosimilar approval to test some of the implications of the model.

Of all biologics that experienced biosimilar entry, three were competing against biosimilar products approved through the accelerated channel introduced by the BPCIA, but

four were competing against products that used different avenues to approval: Betaseron, Neupogen, Humalog, and Lantus.[19] It is likely that the difference between these four products and their biosimilars is perceived to be smaller. The biosimilars for Betaseron and Neupogen went through the same standard FDA approval process for all biologics, so they reached the market having cleared the same standard of evidence required of their reference biologics. The biosimilars for Humalog and Lantus were approved through an abbreviated pathway called 505(b)(2), which is reserved for follow-on products such as line extensions. Since follow-on products are generally acknowledged to be identical (and sometimes superior) to their originators, the perceived difference between these two products and their biosimilars is likely also smaller.

Our conjecture is that entry of these products led to a greater fall in the volume of their originator biologic products, and possibly also a more muted response in price, akin to that of brand products after generic entry.[20]

Figure 6 shows price and volume trends for biologics whose biosimilars were approved through the accelerated BPCIA procedure vs. biologics whose biosimilars were approved through alternative procedures. These figures mirror the Figures 2 and 3, but split the effect across the two samples. Moreover, because the two groups are quite small, we aggregate the effects at the yearly level instead of the quarterly level. Year 1 is defined as the first four quarters after biosimilar entry (including the actual quarter of entry), and Year -1 is the last four quarter before biosimilar entry. All coefficients are normalized relative to Year -1.

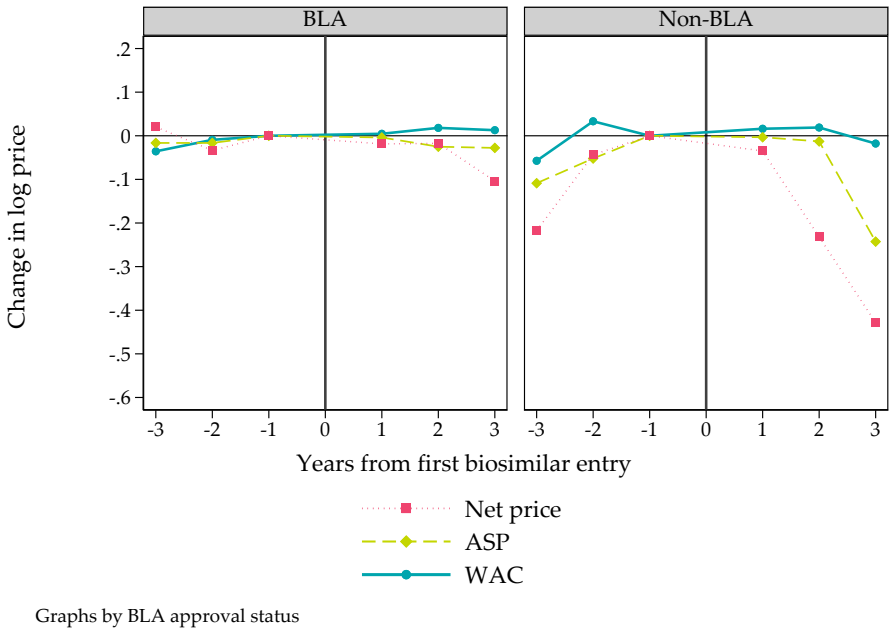
We find that price and volume trends are consistent with the predictions of our model. Entry of biosimilars approved through a regular BLA or the 505(b)(2) procedure is associated with a steeper drop in volume, and a more muted price response. Conversely, it appears that entry of biosimilars that were approved through the accelerated procedure actually had virtually no effect on volume, but elicited a very aggressive price response.

4.3 Policy Implications and Future Outlook of Biosimilar Products

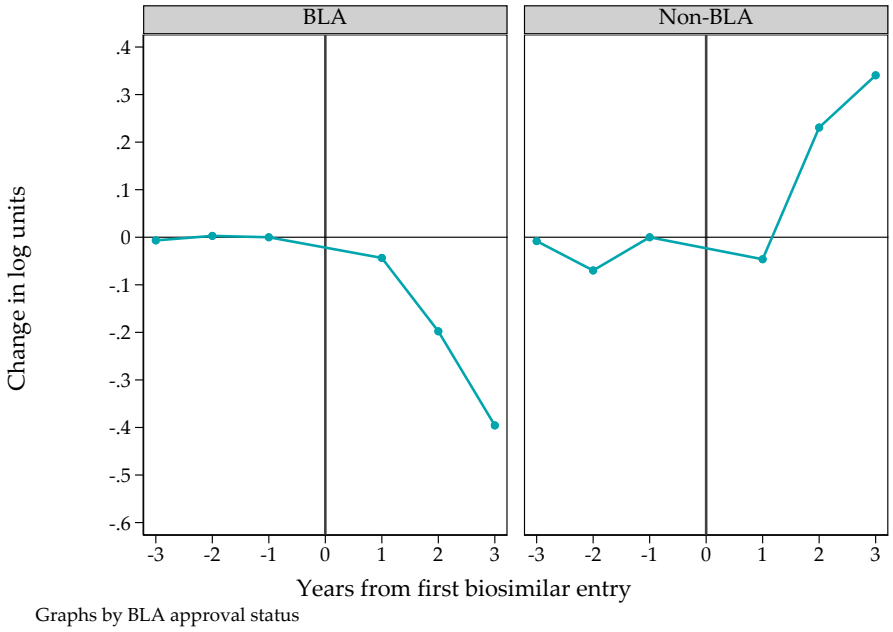
Our empirical and theoretical analysis suggests that while biosimilars are facing greater hurdles than generics, there are reasons to be optimistic about their potential promise. In this section we outline the main challenges that biosimilars have to face, and discuss the how potential policy levers may ease these challenges and increase the likelihood of

[19] Neupogen, competed initially against a biosimilar that was approved through a regular BLA (Granix), but later also competed against additional biosimilars approved through the abbreviated procedure.

[20] The model makes an unambiguous prediction on volume, but not on price. As long as $\Delta V > \Delta V_1$, a fall in ΔV leads to a lower price for the incumbent. The price increases only when ΔV falls below ΔV_1.



(a) Price



(b) Volume

Figure 6: Comparison of reference biologic responses: BLA vs. non-BLA biosimilars

biosimilar success in the future.

The two main challenges facing biosimilars are (i) high development costs and (ii) lingering uncertainty about their effectiveness and quality—which allows reference biolog-

ics to maintain a dominant position in the market by granting additional discounts. These two issues can interact with one another to create a particularly challenging environment for biosimilars. High development costs limit the number of biosimilars, which in turn makes it more profitable on the margin for reference biologics to fight biosimilars with lower prices and maintain a dominant position. The converse also applies: if reference biologics commit to fighting biosimilar entry through aggressive pricing the potential profits of launching a biosimilar will be low, further discouraging potential entrants.

Each of these two challenges can be addressed using a specific set of policies targeted at either lowering fixed costs of entry, or promoting biosimilar use over that of reference biologics.

Policies that aim to lower the cost of developing and launching a biosimilar product can target two main sources of cost: the FDA approval process, and the risk of potential litigation. The FDA approval process of biosimilars has already been streamlined by the BPCIA. However, the accelerated biosimilar pathway still requires biosimilars to run clinical trials with the goal of showing equivalence with the reference biologic. While these trials are smaller and cheaper to run than traditional pivotal trials for NDAs and BLAs, they can still be very expensive. As evidence accumulates that biosimilars are indeed broadly equivalent to biologics, the government may move to remove this requirement altogether; perhaps replacing it with a pharmacovigilance requirement for the first few years post-approval. The government could also take steps to limit the exposure of biosimilars to litigation from manufacturers of reference biologic drugs.[21]

A second set of policies should be aimed at fostering biosimilar adoption, either by collecting and disseminating evidence of equivalence with reference biologics, or by promoting biosimilar use. Policies falling under the first umbrella could subsidize studies comparing biosimilars and biologics, and regulate the spread of information that some biosimilar manufacturers have recently argued harms the diffusion of biosimilars (Cohen and McCabe, 2020).

Policies to promote biosimilar use could include favorable government reimbursement policies and mandatory substitution laws. Reimbursement policies for Medicare Part B currently do favor biosimilar drugs, but generate somewhat weak incentives. According to current regulation, biosimilars are reimbursed at Average Sales Price (ASP), plus 6 percent of the ASP of the reference biologic.[22] In theory, this policy provides the same incentive to prescribe a biosimilar or its reference biologic. In practice however,

[21] There is currently bipartisan legislation, the Biologic Patent Transparency Act, which would force manufacturers of biologics to register all patents relevant to a given product, akin to the FDA Orange Book for small molecule drugs.

[22] Until 2018, biosimilars were reimbursed using the standard Part B formula of ASP plus 6%.

hospitals whose acquisition cost for biologics is below ASP will still face financial incentives that favor reference biologics. For example, recent evidence from IQVIA suggests that 340B clinics—which have access to favorable pricing—have lower adoption rates for biosimilar Bevacizumab (IQVIA, 2020). One way to reinforce the incentive to switch to biosimilars would be to adopt the same policy in place for brand and generic drugs: when a generic drug enters, Medicare Part B calculates a molecule-specific sales-weighted ASP, and then uses it to reimburse both brand and generic versions. A less extreme version of this policy was recently introduced to congress: HR 6179 would establish a "shared savings" program encouraging physicians to prescribe biosimilars by offering them a percentage of any net realized savings.[23]

Finally, we note that these problems might slowly resolve themselves over time. Current medical evidence strongly suggests that biologics and biosimilars are basically identical (see e.g. Barbier et al., 2020; Luttropp et al., 2020). At the same time, public awareness of biosimilars—at least among patients—appears to be low: according to a recent HarrisX poll, 65% of Americans admit to not being familiar with the term "biosimilars".[24] As knowledge spreads to physicians and patients we can expect insurers and providers to promote biosimilar switching more aggressively.[25] This learning process occurred with generics as well. Early evidence of generic adoption suggests that brand drugs were able to maintain significant market share even one or two years after generic entry (Grabowski and Vernon, 1992). Today, this is no longer the case, as generics capture almost the entire market within a few months (Grabowski et al., 2016). Policies targeted towards greater acceptance of biosimilars however, may speed up this process, though policymakers should be wary of adding to the already large development costs that biosimilar manufacturers face.

5 Conclusion

In this paper we examine how biologic drug react to the entry of biosimilar competitors. We find that so far, the modal reaction of biologic manufacturers has been to compete on price with these new entrants. By aggressively cutting prices, reference biologics have been able to limit losses in volume and formulary coverage, which in turn has prevented

[23]The bill can be accessed at https://www.congress.gov/bill/116th-congress/house-bill/6179?s=1&r=5, retrieved November 13, 2020.

[24]For poll results see harrisx.com/wp-content/uploads/2020/03/Biosimilars-topline-poll-public-memo-VF.pdf, retrieved November 13, 2020.

[25]This is already happening in some contexts, such as Kaiser Permanente in California (https://biosimilarsrr.com/2019/11/07/how-did-kaiser-permanente-reach-95-utilization-of-biosimilar-herceptin-and-avastin-so-quickly/, retrieved November 10, 2020).

biosimilars from gaining meaningful market share.

The main conceptual question that arises from this result is why reference biologics do not behave more like small-molecule brand drugs, which rarely compete on price with generic drugs. Our hypothesis is that perceived differences in the quality of biosimilar drugs can soften competition in the market, making it relatively more profitable for biologics to compete on price. We say perceived differences, because the medical evidence available on biosimilars has so far suggested that biosimilars are just as safe and effective as their reference biologics.

Our exercise, therefore, has two takeways. First, widespread dissemination of information about the safety and efficacy of biosimilars should increase their competitiveness in the market, and eventually convince reference biologics to focus on loyal consumers only. Second, even in the absence of such nudges, we expect, as providers and patients become more familiar with biosimilars, that the market will eventually reach an equilibrium where the reaction of any brand drug to a same-molecule competitor will be to keep prices constant, and cede market share.

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A Data

In this section we describe the formulary data we obtained from MMIT Analytics, and explain how we constructed the coverage variables from that data. For a description of the SSR Health data, see instead Online Appendix A to Feng et al. (2020), particularly Section A.1.

A.1 Formulary Data

MMIT Analytics collects monthly data on formulary coverage from health plans. This data includes information about the coverage tier of a specific drug, as well as the plan sponsor, and the number of lives it covers.

We have data on around 300 drugs—including all biosimilars and reference biologics—from January of 2011 until April of 2020. Throughout this period, MMIT's coverage grew from around 100 million lives to over 300 million lives, representing almost the entirety of the insured US population. The data includes health plans across all possible channels: commercial plans, Medicare, Medicaid (both State-sponsored and Managed Medicaid), as well as the ACA health exchanges (Figure 7).[26]

A.2 Construction of coverage variables

We followed Geruso et al. (2019) in assigning different formulary tiers to buckets of covered, preferred, and unrestricted. MMIT analytics processes the tier information for all formularies to create a harmonized "universal status" variable that describes each formulary's tiers in a way that makes them comparable across plans and years. Moreover, each drug is flagged for three possible non-monetary restrictions: Prior Authorization (PA), Step Therapy (ST), and Quantity Limit (QL). Of these three, only the first two represent meaningful obstacles to the prescription of a drug.[27] We use these two variables to create mutually exclusive coverage bins following a procedure first used in Geruso et al. (2019).

The procedure has two steps. First, we create six categories of coverage based on the "universal status" variable (plus one empty category for missing data). Table 4 displays the map that we use. Next, we put all drugs that are flagged for PA or ST into a separate group—to which we assign the number 2. This overrides any previous group assignment.

From these seven bins, we create three indicator variables as follows:

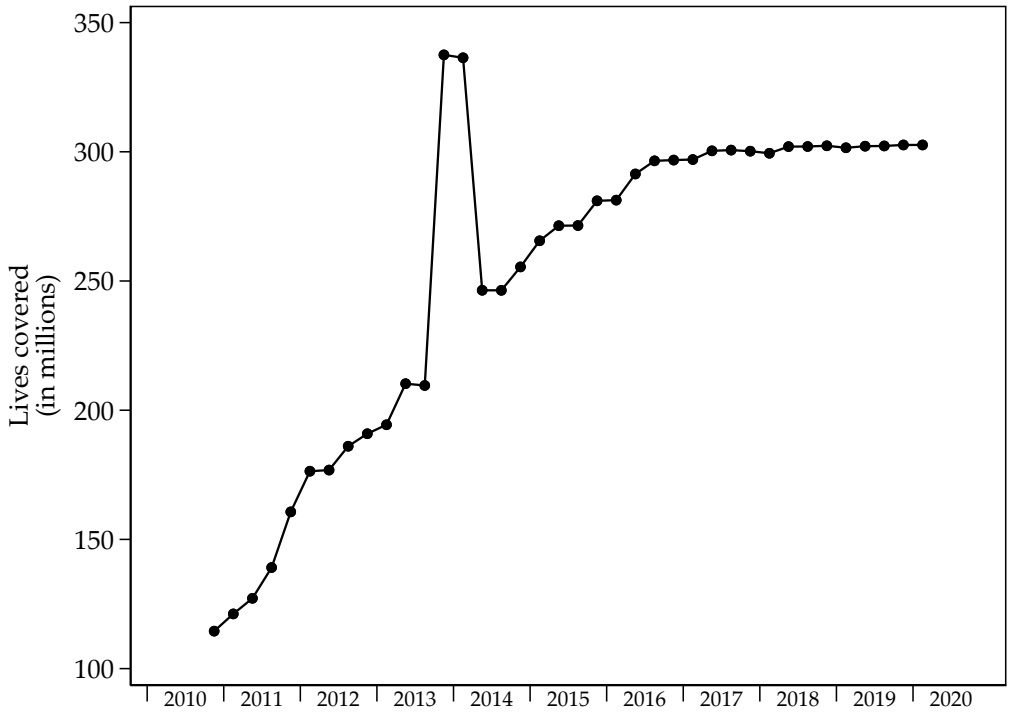
[26] The spike in 2014Q1 and 2014Q2 is caused by double-counting of lives in certain (unidentifiable) commercial plans. The issue does not affect other quarters.

[27] Quantity limit refers to the maximum number of doses that can be prescribed, and is used to prevent abuse of medications like opioids—e.g Vicodin or Oxycontin —or amphetamines—e.g. Adderall or Ritalin.

Table 4: Drug tiers assignment

Group	Category	Universal Status
0	Not listed/Uncategorized	
1	Not covered	Not Covered Drugs Tier
		Unknown Coverage
3	Medical	Medical
4	Non-preferred specialty	Injectable Drugs Tier
		Non-Preferred Specialty Drugs Tier
		Specialty Drugs Tier
5	Covered/non-preferred brand	Covered Drugs Tier
		Non-Prefered Brand Drugs Tier
		Non-Preferred Generics and Non-Preferred Brands
6	Preferred specialty	Preferred Specialty Drugs Tier
7	Preferred brand	Generic Drugs Tier
		Non-Preferred Generic Drugs Tier
		Non-Preferred Generics and Preferred Brands
		Preferred Brand Drugs Tier
		Preferred Generic Drugs Tier
		Preferred Generics and Preferred Brands
		Value Brand Drugs Tier
		Value Generic Drugs Tier
		Zero Co-pay Tier

Figure 7: MMIT lives covered



1. Any coverage: groups 2-7
2. Unrestricted coverage: groups 3-7
3. Preferred coverage: groups 6-7

The dependent variables in our formulary regressions are weighted averages (using number of lives covered as weights) of these three indicators for each drug and quarter, across all health plans. Another way to interpret them is as the percentage of lives with a certain level of coverage for a given drug.

B Model Derivation

We assume that demand for the originator product can be micro-founded using a Hotelling model. Each consumer has distance d from the product, and earns

Assuming that the value and price of the generic competitor are such that all consumers prefer consuming the generic option to nothing, the Hotelling model is set up as in the figure below.

We make the following assumptions on model parameters:

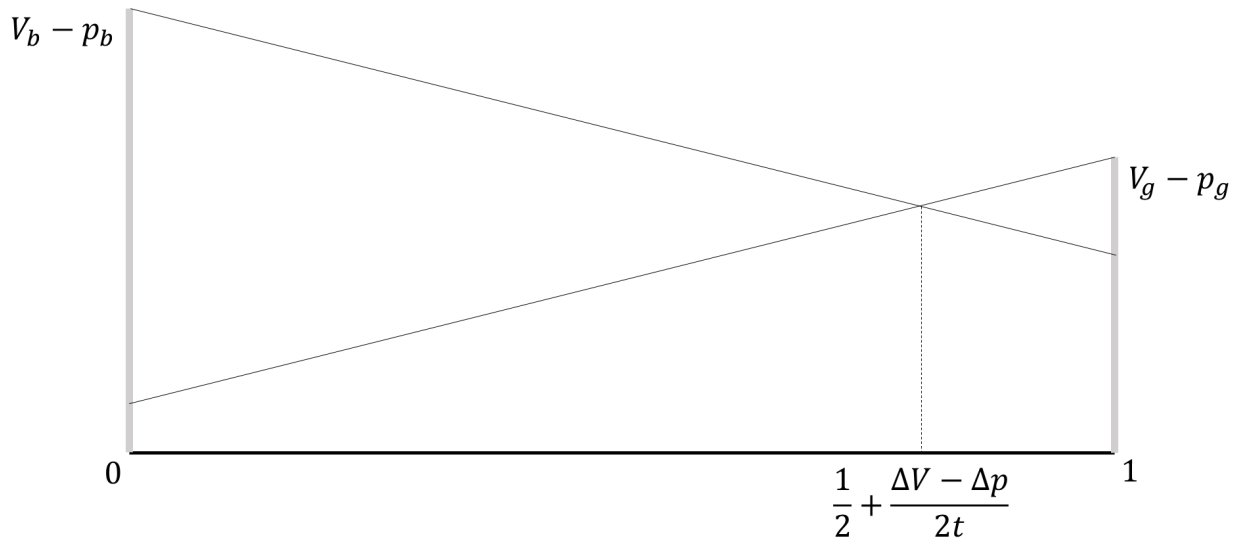


Figure 8: Hotelling model setup for the price-sensitive segment

- $V_b \geq V_g$. This simply ensures that the generic follower cannot be perceived a strictly better than the originator, just as its equal.
- $V_g > t$. This ensures that all consumers would prefer the generic to the outside option if the generic had a price of 0.

Let's also assume that prices are announced sequentially, and that the entrant announces after observing the price of the originator incumbent. This is not necessary for the result of the model, but it does simplify exposition by allowing us to set up the incumbent's FOC as a direct expression for p_b instead of a best-response function.

Problem of the entrant

As long as the incumbent remains competitive in the market (i.e. as long as p_b is set such that there are some consumers with location $x \in (0, 1)$ who are indifferent between the incumbent and the entrant, and prefer both to the outside option), the entrant faces residual demand function

$$D_g\left(p_g\right) = \max\left\{\frac{1}{2} - \frac{V_b - V_g - p_b + p_g}{2t}, 0\right\}$$

If instead the incumbent is not competitive (i.e. sets a p_b such that the marginal voter for the entrant is indifferent between the entrant and the outside option), the residual

demand function (assuming price $p_g < V_g$) is

$$D_g\left(p_g\right) = \min\left\{\frac{V_g - p_g}{t}, 1\right\}$$

Combining the two, we can write the overall demand function as

$$D_g\left(p_g\right) = \min\left\{\max\left\{0, \frac{1}{2} - \frac{V_b - V_g - p_b + p_g}{2t}\right\}, \frac{V_g - p_g}{t}, 1\right\}$$

The solutions to the possible problems of the entrant are as follows:

Competitive incumbent:

$$\max_{p_g} p_g \times \left(\frac{1}{2} - \frac{V_b - V_g - p_b + p_g}{2t}\right)$$

which yields the FOC

$$\begin{aligned}\left[p_g\right] &: \left(\frac{1}{2} - \frac{V_b - V_g - p_b + p_g}{2t}\right) - \frac{p_g}{2t} = 0 \\ \implies p_g^{\text{comp}} &= \frac{t - V_b + V_g + p_b}{2}\end{aligned}$$

Non-competitive incumbent:

$$\max_{p_g} p_g \times \left(\frac{V_g - p_g}{t}\right)$$

which yields the FOC

$$\begin{aligned}\left[p_g\right] &: \left(\frac{V_g - p_g}{t}\right) - \frac{p_g}{t} = 0 \\ \implies p_g^{\text{nc}} &= \frac{V_g}{2}\end{aligned}$$

Boundary between the competitive and non-competitive case The last boundary that we need to consider is that between the competitive and non-competitive case. In other words, it may occur that p_b implies

$$\frac{1}{2} - \frac{V_b - V_g - p_b + p_g^{\text{comp}}}{2t} > \frac{V_g - p_g^{\text{comp}}}{t}$$

which is impossible, based on the demand function we derived. In these situations, the problem has a corner solution, which is given by p_g such that

$$\begin{aligned}
\frac{1}{2} - \frac{V_b - V_g - p_b + p_g}{2t} &= \frac{V_g - p_g}{t} \\
\implies t - V_b + V_g + p_b - p_g &= 2V_g - 2p_g \\
\implies t - V_b + p_b &= V_g - p_g \\
\implies p_g &= V_g - t + V_b - p_b
\end{aligned}$$

Notice that this implies that the best-response function of the entrant is *increasing* in p_b, which breaks monotonicity. We show later what assumption we need on V_g that excludes this possibility.

Unit interval boundaries: The truncation of the demand function at 1 implies that if the optimal price would imply demand > 1, we have a corner solution. The firm will increase the price until it reaches $D\left(p_g\right) = 1$. This price is

$$p_g = V_g - V_b + p_b - t$$

when the incumbent is competitive and

$$p_g = V_g - t$$

when the incumbent is not competitive.

Putting together all these moving parts, the best-response function can be written as

$$p_g^{\text{comp}}\left(p_b\right) = \max\left\{0, \frac{V_g - V_b + p_b + t}{2}, V_g - V_b + p_b - t\right\}$$

and

$$p_g^{\text{nc}}\left(p_b\right) = \max\left\{0, \frac{V_g}{2}, V_g - t\right\}$$

Notice that in the second case, the best response will be $\frac{V_g}{2}$ if $V_g \in [t, 2t]$ and $V_g - t$ if $V_g > 2t$. To simplify the analysis, we assume the latter: $V_g > 2t$.[28]

Then, we can combine these two results to write down the full best-response function:

$$p_g^{\star}\left(p_b\right) = \min\left\{\max\left\{0, \frac{V_g - V_b + p_b + t}{2}, V_g - V_b + p_b - t\right\}, V_g - t\right\} \tag{2}$$

[28] $V_g \in [t, 2t]$ is what leads to the non-monotonicity result mentioned earlier.

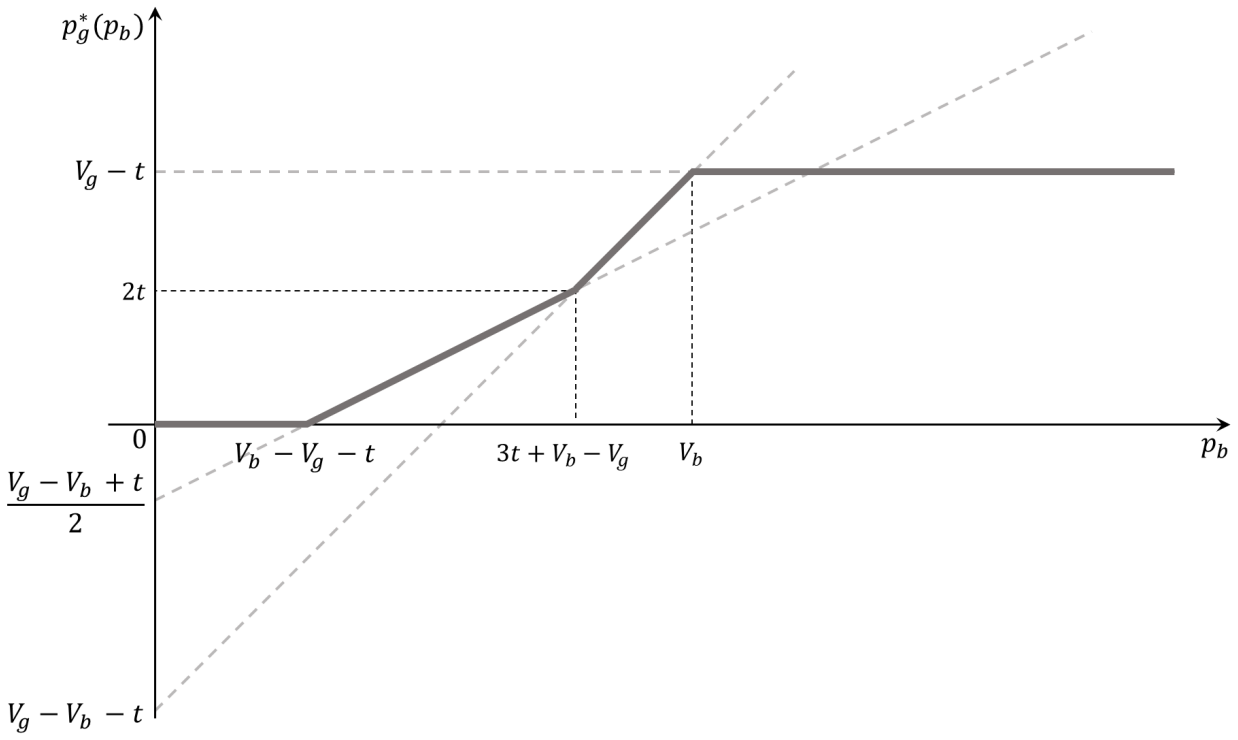


Figure 9: Best response function of the entrant

Figure 9 plots this best response function for clarity. Intuitively, for p_b low enough, the generic entrant won't be able to capture any demand, and will set a price of 0. As p_b increases, the entrant will be able to set a price p_g that puts her in competition with the incumbent, and demand is split. In this segment, the best response function has slope $\frac{1}{2}$. If p_b increases further, the entrant will be able to capture the entire demand, and the limit on what price allows her to do that is increasing one-to-one with respect to p_b. At some point though, the natural limit to the amount that the entrant can charge will kick in. This limit is $V_g - t$, i.e. the price that makes the consumer in location 0 indifferent between entrant and the outside option.

The derivative of $p_g^\star(p_b)$ with respect to p_b is

$$\frac{\partial p_g^\star(p_b)}{\partial p_b} = \begin{cases} 0 & \text{if } p_b < \Delta V - t \\ \frac{1}{2} & \text{if } p_b \in (\Delta V - t, \Delta V + 3t) \\ 1 & \text{if } p_b \in (\Delta V + 3t, V_b) \\ 0 & \text{if } p_b > V_b \end{cases} \tag{3}$$

where $\Delta V = V_b - V_g > 0$.

Problem of the incumbent

The demand function of the incumbent in the price-sensitive segment is analogous to that of the entrant:

$$D_b\left(p_b\right) = \min\left\{\max\left\{0, \frac{1}{2} + \frac{V_b - V_g - p_b + p_g^\star\left(p\right)}{2t}\right\}, \frac{V_b - p_b}{t}, 1\right\}$$

We then assume that the loyal segment of the market has a measure α of consumers, who are willing to pay up to V_b in order to purchase the product. Hence–

$$D\left(p\right) = \begin{cases} \alpha & \text{if } p \leq V_b \\ 0 & \text{otherwise} \end{cases}$$

The firm has three options:

1. It can set whatever price allows it to capture the entire market (including the price-sensitive option)
2. It can set a price that is competitive in the price-sensitive market (meaning it will split that market with the entrant)
3. It can sell only in the loyal market, with a price that implies that it will not compete in the price-sensitive market

We explore each option separately, leaving option 2 for last, as it is the most complicated one.

Pricing to capture all demand

Because we have already calculate the best-response function of the entrant, we know what price will lead to this option: $\Delta V - t$. Any price higher than that will allow the entrant to cut into the demand of the incumbent. With that price, the firm earns

$$\Pi_b\left(\Delta V - t\right) = \left(\Delta V - t\right) \times \left(1 + \alpha\right)$$

Notice that this option only ever makes sense if $\Delta V > t$, so as ΔV shrinks, this option is no longer feasible.

Pricing to the loyal market only

In this case, the optimal price is V_b, yielding profits

$$\Pi_b(V_b) = \alpha V_b$$

There will be no profits from the price-sensitive market because no consumers in that market are willing to pay V_b.

Pricing to both markets

First, consider the optimal price in the price-sensitive market. This will allow us to make some comparisons later. Under this scenario, the entrant is competitive in the market (i.e. $p_b > \Delta V - t$), hence the maximization problem becomes

$$\max_p p \times \left(\frac{1}{2} + \frac{V_b - V_g - p + \frac{V_g - V_b + p + t}{2}}{2t} \right) \equiv p \times \left(\frac{3t + V_b - V_g}{4t} - \frac{p}{4t} \right)$$

which implies

$$p_b = \frac{3}{2}t + \frac{\Delta V}{2}$$

And in turn,

$$\begin{aligned} p_g^\star(p_b) &= \frac{V_g - V_b + t + \frac{V_b - V_g}{2} + \frac{3}{2}t}{2} \\ &= \frac{\frac{1}{2}V_g - \frac{1}{2}V_b + \frac{5}{2}t}{2} = \frac{5}{2}t - \frac{1}{4}\Delta V \end{aligned}$$

To check that this equilibrium makes sense, recall from Figure 9 that $p_g \in (0, 2t)$. Hence, this equilibrium makes sense as long as $\Delta V < 5t$. If ΔV is too large, the entrant will set $p_g = 0$, and capture no demand, while the incumbent will set $p_b = \Delta V - t$ and capture the entire market.

Now let's calculate the optimal price when pricing to both markets. Since we have assumed that the two firms are both competing in the market, the incumbent must set $p_b \in (\Delta V - t; 3t + \Delta V)$. This in turn implies $p_g^\star(p_b) = \frac{p_b + t - \Delta V}{2}$ (to see why, check equation 2). Hence, we can write the maximization problem as

$$\max_p p \times \left(\alpha + \frac{1}{2} + \frac{\Delta V - p + \frac{p + t - \Delta V}{2}}{2t} \right) = p \times \left(\alpha + \frac{1}{2} + \frac{\Delta V - p + t}{4t} \right)$$

The FOC yields:

$$\begin{aligned}
&[p] : \alpha + \frac{1}{2} + \frac{\Delta V - p + t}{4t} - \frac{p}{4t} = 0 \\
&\implies \alpha + \frac{\Delta V + 3t}{4t} = \frac{p}{2t} \\
&\implies p = 2t\alpha + \frac{\Delta V + 3t}{2} \\
&\implies p = \frac{4\alpha + 3}{2} t + \frac{\Delta V}{2}
\end{aligned}$$

This price implies a best-response price

$$\begin{aligned}
p_g &= \frac{\frac{4\alpha+3}{2} t + \frac{\Delta V}{2} + t - \Delta V}{2} \\
&= \frac{(4\alpha + 5)\, t - \Delta V}{4} = \left(\alpha + \frac{5}{4}\right) t - \frac{\Delta V}{4}
\end{aligned}$$

The difference in price is

$$\begin{aligned}
p_b - p_g &= \frac{4\alpha + 3}{2} t + \frac{\Delta V}{2} - \left(\alpha + \frac{5}{4}\right) t + \frac{\Delta V}{4} \\
&= \left(\frac{4\alpha + 1}{4}\right) t + \frac{3}{4} \Delta V > 0
\end{aligned}$$

and profits

$$\begin{aligned}
\Pi &= \left(\frac{(4\alpha + 3)\, t}{2} + \frac{\Delta V}{2}\right) \times \left(\alpha + \frac{1}{2} + \frac{\Delta V - \left(\frac{4\alpha+1}{4}\right) t - \frac{3}{4}\Delta V}{2t}\right) \\
&= \left(\frac{(4\alpha + 3)\, t + \Delta V}{2}\right) \times \left(\frac{(4\alpha + 3)\, t + \Delta V}{8t}\right) \\
&= \frac{\left((4\alpha + 3)\, t + \Delta V\right)^2}{16t}
\end{aligned}$$

We now proceed to the analysis of how changes in ΔV and t affect the optimal choice of the incumbent firm.

Comparison of profits as a function of ΔV

We start by discussing how the best response of the incumbent changes as ΔV changes. Throughout this section we will hold t constant and we will assume that it is strictly positive.

Notice that when the entrant and the incumbent are competing in the price-sensitive market,

$$\frac{1}{2} + \frac{\Delta V - p_b + p_g}{2t} \in (0, 1)$$

which implies

$$p_b - p_g \leq t + \Delta V$$

(otherwise demand is fully captured by the entrant), and

$$p_b - p_g \geq \Delta V - t$$

(otherwise demand is fully captured by the incumbent. Recall that

$$p_b - p_g = \left(\frac{4\alpha + 1}{4}\right) t + \frac{3}{4}\Delta V$$

Hence, these conditions, if expressed in terms of ΔV, imply

$$\begin{aligned} \left(\frac{4\alpha + 1}{4}\right) t + \frac{3}{4}\Delta V &\leq t + \Delta V \\ \implies (4\alpha - 3)\, t &\leq \Delta V \end{aligned}$$

and

$$\begin{aligned} \left(\frac{4\alpha + 1}{4}\right) t + \frac{3}{4}\Delta V &\geq \Delta V - t \\ \implies (4\alpha + 5)\, t &\geq \Delta V \end{aligned}$$

i.e.

$$\Delta V \in (4\alpha t - 3t; 4\alpha t + 5t)$$

At the upper bound, demand will be fully captured by the incumbent, and at the lower bound, demand will be fully captured by the entrant.

At the boundaries, profits are

$$\begin{aligned}\Pi\left(\left(4\alpha-3\right)t\right) &= \left(\frac{\left(4\alpha+3\right)t}{2}+\frac{\Delta V}{2}\right)\times\left(\alpha+\frac{1}{2}+\frac{\Delta V-\left(\frac{4\alpha+1}{4}\right)t-\frac{3}{4}\Delta V}{2t}\right)\\ &= \left(\frac{\left(4\alpha+3\right)t}{2}+\frac{\left(4\alpha-3\right)t}{2}\right)\times\left(\alpha+\frac{1}{2}+\frac{\frac{1}{4}\left(4\alpha-3\right)t-\left(\frac{4\alpha+1}{4}\right)t}{2t}\right)\\ &= 4\alpha^2 t\end{aligned}$$

and

$$\begin{aligned}\Pi\left(\left(4\alpha+5\right)t\right) &= \frac{\left(8\alpha+8\right)^2 t^2}{16t}\\ &= \left(4\alpha^2+8\alpha+4\right)t\\ &= 4\left(1+\alpha\right)^2 t\end{aligned}$$

Notice that for $\Delta V=\left(4\alpha+5\right)t$ the profit of charging $\Delta V-t$ is

$$\begin{aligned}\Pi\left(\Delta V-t\right) &= \left(1+\alpha\right)\times\left(\left(4\alpha+5\right)t-t\right)\\ &= 4\left(1+\alpha^2\right)t\end{aligned}$$

which matches.

The other boundary is not necessarily determined by $\Delta V=\left(4\alpha-3\right)t$. Instead, the incumbent will switch to only serving the loyal market whenever

$$\alpha V_b > \Pi\left(\Delta V\right)=\frac{\left(4\alpha+3\right)^2 t^2+\left(4\alpha+3\right)t\Delta V+\Delta V^2}{16t}$$

which implies

$$\Delta V^2+\left(4\alpha+3\right)t\Delta V+\left(4\alpha+3\right)^2 t^2-\alpha V_b=0$$

Using the quadratic formula:

$$\begin{aligned}\Delta V &= \frac{-\left(4\alpha+3\right)t\pm\sqrt{\left(4\alpha+3\right)^2 t^2-4\left(\left(4\alpha+3\right)^2 t^2-\alpha V_b\right)}}{2}\\ &= \frac{-\left(4\alpha+3\right)t+\sqrt{4\alpha V_b-3\left(4\alpha+3\right)^2 t^2}}{2}\end{aligned}$$

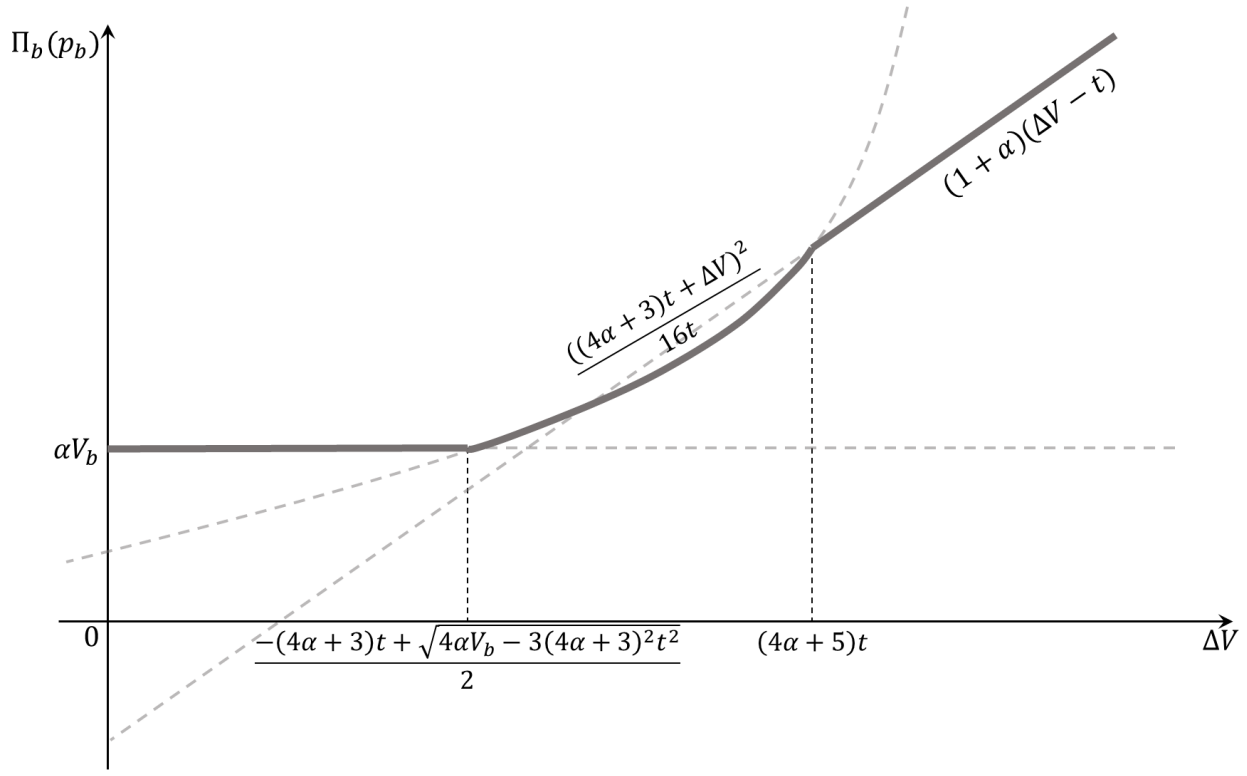


Figure 10: Incumbent profits as a function of ΔV

where the other solution is ruled out because $\Delta V > 0$.

Figure 10 displays the profits of the incumbent graphically for intuition. As ΔV decreases, the incumbent is slowly driven out of the price-sensitive market and into the loyal market.

The two thresholds in the figure map to ΔV_1 and ΔV_2 in Proposition 1. Hence, the derivation in this section serves as the proof of that Proposition.

Impact of t on the best-response of the incumbent

We can then prove Corollary 2 by simply checking how changing t would affect the two thresholds ΔV_1 and ΔV_2.

A fall in t has two effects. First, it makes it easier for the entrant to compete and decreases overall profits, which increases the minimum threshold ΔV that makes it worthwhile for the entrant to compete in the price-sensitive market. This is reflected in the graph by the fact that the intersection point A shifts right whenever t falls (because the expression denoting its x coordinate is unambiguously decreasing in t). Second, however, it makes it easier for the incumbent to compete for consumers that are very close to the entrant. Hence, capturing the entire market becomes a relatively more attractive

proposition. This is reflected in the graph by the fact that the intersection point B shifts left whenever t falls (because the expression denoting its x coordinate is unambiguously increasing in t). This proves Corollary 2.

The combination of these two effects implies that for t small enough the incumbent will either capture the entire market, or leave the price-sensitive market to the entrant altogether. Which option is better depends on ΔV.

Table 5: Impact of biosimilar entry on formulary coverage of biologic molecules

	% Covered			% Preferred			% Unrestricted		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
Biosimilar has launched	-2.235	-2.022		-3.582	-3.817		6.209	6.080	
	(4.486)	(4.261)		(4.032)	(3.710)		(3.118)	(2.747)	
Biosimilar launch trend		-0.0412			0.0452			0.0247	
		(0.104)			(0.109)			(0.118)	
1 Biosimilar launched			-5.333			-6.854			4.459
			(5.024)			(3.854)			(3.407)
2 Biosimilars launched			2.459			1.325			8.871
			(2.050)			(2.958)			(2.878)
3 Biosimilars launched			1.519			1.306			8.135
			(2.205)			(2.540)			(2.943)
Constant	91.91	88.01	91.87	11.00	26.00	10.96	19.30	56.14	19.28
	(0.219)	(1.487)	(0.228)	(0.267)	(1.383)	(0.277)	(0.360)	(2.062)	(0.371)
Product FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Quarter FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
N	792	792	792	792	792	792	792	792	792
R^2	0.789	0.789	0.799	0.958	0.958	0.960	0.924	0.924	0.925

Note: Each of the outcomes is a percentage of lives on a health plan with a formulary status of covered, preferred, or unrestricted. Covered means that a drug is on the formulary and does not fall under an explicit "Not Covered" tier. The low constant for preferred models can be explained by the fact that drugs we flag as preferred are both preferred and unrestricted. For a further explanation of our tier assignment see Appendix A.

C Additional Results and Robustness Checks

C.1 Additional results

We check whether biosimilars expand access to the reference molecule. To do so, we look at how coverage changes at the *molecule* level before and after biosimilar entry. When a plan covers both the reference biologic and its biosimilar, we select the product with the more generous coverage tier. We present our results in Table 5. Entry of a biosimilar does not seem to have a large impact on overall coverage. We detect a small but not significant decrease in preferred coverage, and in any coverage, and a small and insignificant increase in unrestricted coverage. Entry of multiple biosimilars is associated with a large and significant increase in unrestricted coverage however.

Alternative control group

We check whether our results are robust to including all biologics in the control group. Table 6 shows results for price and volume regressions. Overall, the results are qualitatively very similar, though we find slightly smaller results for net price, and slightly larger results for ASP and volume.

Table 7 shows results for coverage regressions. Once again, the results are very similar, though slightly lower in magnitude across the board. This confirms our impression that the formulary position of reference biologic drugs did not change much after biosimilar entry.

Placebo test

In Table 8 we report results from our placebo test. We include an indicator for whether the biosimilar has been approved. The idea is to exploit the somewhat lengthy delay between approval and launch to check that our main specification is not picking up any spurious effects due to pre-existing trends. We do not detect any significant correlation between approval and any of our outcomes of interest.

Table 6: Price and Volume Regression Results (all biologics)

	Log WAC			Log net price			Log ASP			Log Units sold		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
Biosimilar has launched	0.109	0.009		-0.092	-0.166		-0.096	-0.028		-0.116	0.342	
	(0.117)	(0.073)		(0.125)	(0.077)		(0.070)	(0.068)		(0.268)	(0.194)	
Biosimilar launch trend		0.011			0.008			-0.015			-0.0479	
		(0.007)			(0.008)			(0.005)			(0.00572)	
1 Biosimilar launched			0.172			0.054			-0.050			-0.235
			(0.137)			(0.139)			(0.075)			(0.275)
2 Biosimilars launched			-0.022			-0.193			-0.143			-0.144
			(0.085)			(0.122)			(0.079)			(0.384)
3 Biosimilars launched			-0.139			-0.453			-0.240			-0.838
			(0.090)			(0.106)			(0.078)			(0.250)
Product FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Quarter FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	N	N	N
Age FE	N	N	N	N	N	N	N	N	N	Y	Y	Y
N	4,606	4,606	4,606	3,928	3,928	3,928	2,382	2,382	2,382	4,595	4,595	4,595
R^2	0.979	0.979	0.979	0.955	0.955	0.955	0.996	0.996	0.996	0.946	0.946	0.946

Table 7: Formulary Regression Results (all biologics)

	% Covered			% Unrestricted			% Preferred		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
Biosimilar has launched	-3.981	-2.537		-3.296	-3.779		-4.749	-2.971	
	(2.492)	(2.766)		(1.884)	(2.369)		(1.637)	(1.903)	
Biosimilar launch trend		-0.248			0.0831			-0.306	
		(0.281)			(0.211)			(0.250)	
1 Biosimilar launched			-2.039			-0.983			-0.909
			(2.284)			(2.456)			(1.789)
2 Biosimilars launched			0.330			-2.003			-5.121
			(1.591)			(2.229)			(1.823)
3 Biosimilars launched			-13.47			-8.373			-14.54
			(1.192)			(1.966)			(1.333)
Constant	81.47	84.20	81.50	18.33	56.10	18.35	5.559	18.81	5.579
	(0.225)	(1.050)	(0.230)	(0.262)	(1.779)	(0.261)	(0.158)	(1.119)	(0.159)
Product FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Quarter FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
N	1,603	1,603	1,603	1,603	1,603	1,603	1,602	1,602	1,602
R^2	0.792	0.794	0.792	0.917	0.917	0.917	0.903	0.905	0.904

Note: Each of the outcomes is a percentage of lives on a health plan with a formulary status of covered, preferred, or unrestricted. Covered means that a drug is on the formulary and does not fall under an explicit "Not Covered" tier. The low constant for preferred models can be explained by the fact that drugs we flag as preferred are both preferred and unrestricted. For a further explanation of our tier assignment see Appendix A.

Table 8: Approval placebo test results

	Net Price	ASP	WAC	Units	% Covered	% Unrestricted	% Preferred
Biosimilar has launched	-0.343	-0.085	-0.100	-0.229	-6.300	-2.190	-4.667
	(0.110)	(0.060)	(0.106)	(0.494)	(3.647)	(1.959)	(3.472)
Biosimilar has been approved	0.158	0.008	0.0767	0.320	1.005	-1.259	-2.863
	(0.135)	(0.058)	(0.130)	(0.226)	(1.589)	(2.295)	(2.336)
Product FE	Y	Y	Y	Y	Y	Y	Y
Quarter FE	Y	Y	Y	Y	Y	Y	Y
Age FE	N	N	N	Y	N	N	N
N	1,410	679	1,482	1,380	792	792	792
R^2	0.969	0.999	0.981	0.951	0.709	0.932	0.958