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Titel der Diplomarbeit

„Determinants of pharmaceutical R&D: The impact of regional price regulation”

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## Acronyms and Abbreviations

<table>
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<th>Acronym</th>
<th>Description</th>
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<tbody>
<tr>
<td>EMEA</td>
<td>Europe, Middle East and North Africa</td>
</tr>
<tr>
<td>FEM</td>
<td>Fixed effects model</td>
</tr>
<tr>
<td>GBP</td>
<td>Pound sterling</td>
</tr>
<tr>
<td>OECD</td>
<td>Organisation for Economic Co-operation and Development</td>
</tr>
<tr>
<td>OLS</td>
<td>Ordinary least squares</td>
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<tr>
<td>PPRI</td>
<td>Pharmaceutical Pricing and Reimbursement Information</td>
</tr>
<tr>
<td>QALY</td>
<td>Quality adjusted life year</td>
</tr>
<tr>
<td>R&amp;D</td>
<td>Research and development</td>
</tr>
<tr>
<td>UK</td>
<td>United Kingdom</td>
</tr>
<tr>
<td>US</td>
<td>United States</td>
</tr>
<tr>
<td>USD</td>
<td>United States dollar</td>
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</table>
Abstract (English)

The purpose of my thesis was to illustrate how the regulation of drug prices affects pharmaceutical companies R&D investments. Several publications have been made dealing with this topic, which I briefly summarized in the first part of my thesis. After that I summarized the prevailing regulatory schemes that are used to regulate drug prices and discussed their possible effects. The most important part of my thesis is the empirical part where I try to verify the impact of price regulation on R&D spending. For my estimation I used a panel data set containing the 20 biggest pharmaceutical companies in terms of sales in the period of 2000 to 2008. As a regulation proxy I used the share of sales made in the EMEA region, share of sales made in the US and share of sales in the rest of the world based on the assumption, that in the EMEA region prices underlie stricter price regulation compared to the US. The main result out of my fixed effects estimation is that price regulation has a significant negative impact on pharmaceutical companies’ R&D investments; this negative impact is even reinforced the more sales a company makes in the EMEA region. By way of comparison I also performed a random effects estimation with the same variable, which provides consistent results.
Chapter 1

1.1. Introduction

Pharmaceuticals play a very important role in health care. A general trend towards more consumption per capita in the industrialized countries can be observed due to society’s demographic change. More consumption causes more costs for the purchasers of drugs, which are usually governmental health insurances in the case of prescribed drugs. Since total costs of health care system are increasing due to an aging society policy-makers try to reduce costs among other things by regulating drug prices. This is an important issue, because a balancing act has to be made between low drug prices and keeping the incentive and resources for pharmaceutical companies to invest in R&D. In the first chapter of my thesis I want to provide an overview of existing empirical studies dealing with the issue how the regulation of drug prices can affect firms’ R&D investments. In the second part I want to say a few words about price regulation and the most common techniques to regulate drug prices. In the third part I estimated the effects of drug price regulation on R&D spending using the share of sales in the different regions as regulation proxies based on the assumption that compared to Europe, drug prices in the US are largely unregulated and could derive some conclusions out of my estimation.
1.2. Review of the literature

The relationship between price regulation and profit margin has just been recently in the focus of some econometrical papers. One very important work by Vernon (2003)\(^1\) focused on the relationship between price regulation and profit margin. He used a panel data set containing the 20 biggest pharmaceutical companies from 1994 to 1999. The main assumption was that drug prices in the US largely remain unregulated compared with the rest of the world, therefore he used the share of companies’ sales made in the non US market as indicator of regulation. According to his results an increase of 10 percentage points in share of sales made in the non US market would result in a decline of 2.7 to 3.5 percentage points in profit margin, which might have a negative influence in terms of R&D expenditures.

In a next step Vernon (2005)\(^2\) established the lagged cash flow and the expected profit to be key determinants of pharmaceutical companies’ R&D spending, which both might be influenced by price regulation. For his estimation he used a panel data set of the 14 biggest pharmaceutical companies in the period of 1994 to 1997. Again, he assumes drug prices in the US to be less regulated compared to the rest of the world. Then he tried to simulate the impact if the US theoretically implemented a new regulation policy, which would cut drug prices. He concluded this implementation would result in a decline in firms’ R&D expenditures between 23.4 and 32.7 percent. These results cannot be used to make a statement on the impact of social welfare, because it is impossible to quantify the innovative productivity of the R&D loss, but nevertheless he could show that price regulation might have a negative impact on R&D investments.

In a very recent study Civan and Maloney (2009)\(^3\) tried to estimate the price elasticity of drug development out of the fact, that the number pharmaceuticals which are standing in the development process feature a highly positive correlation with the price of already existing pharmaceuticals within this therapeutic area. A cross

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sectional analysis of the prices of 600 drugs was used in order to estimate the average price elasticity which accounts for 35 percent on average and is even 0.51 in their preferred specification. An elasticity of 0.51 would mean that if there was a price cut of 50 percent on existing drugs in a certain therapeutic area it would reduce the number of drugs in the development pipeline of this therapeutic area by 25 percent, keeping all other factors constant. Of course some caveats have to be considered, for example the findings might not be valid for across-the-board price cutting, but estimating the price elasticities of drugs in different therapeutic areas is an interesting approach to illustrate the loss of new pharmaceuticals due to price cutting.

Another interesting work with a different approach performing a prospective micro-simulation was published by Abbot and Vernon (2007)\(^4\). In order to do this simulation Monte Carlo techniques were used. They could show that price controls have a negative impact on pharmaceutical companies’ willingness to invest capital in early-stage R&D. According to their results, a price cut of 40 to 50 percent of drug prices in the US will lead to a decline between 30 and 60 percent of investments in R&D projects concerning early-stage development.

When summarizing these empirical studies, it seems that there is indeed a negative impact of drug price regulation on R&D investments to a certain extent.

Chapter 2

2.1. Price regulation

The main goal of price regulation is to maximize society’s total welfare, no matter what kind of price regulation applies. There is a tradeoff between static efficiency and dynamic efficiency. Static efficiency focuses on maximizing the present welfare, where in the most extreme case prices equal short-run marginal costs by reason of price regulation. This approach only aims at the present welfare and can have an adverse effect on future periods since companies would not be able to make any profits meaning that there is no capital available for investments. Without investments a technological progress, which might raise welfare, would not be possible. Assuming that drug prices would equal their marginal costs, no resources would be available to be invested in R&D and that would result in a loss of new innovative drugs that would contribute to raise society’s total welfare. On the other hand dynamic efficiency is focused on maximizing long-term welfare, thus prices equal long-term marginal costs, which consist of the marginal costs where long-term expenditures such as R&D and investments are included to some extent. Compared to static efficiency consumers most likely will face higher prices, at least in the short-term. Nowadays it seems to be common that politicians are in favor of static efficiency in order to profit from lower prices rather than focusing on future welfare.

2.2. Mechanisms of regulating drug prices

2.2.1. External price benchmarking

External price benchmarking is the most used technique in order to cut prices of pharmaceuticals within the OECD countries. The basic idea of this regulation scheme is to introduce a price cap for a newly launched drug, which depends on the price of this drug in predetermined countries. The criteria of the selection of these so-called benchmark countries might differ substantially, but usually the benchmark countries are selected based on their economic and/or geographic proximity, thus European countries are prone to use other European countries as reference countries. For example, Austria uses the average drug price of all European union’s member states
as reference where the medicinal product is approved (PPRI, 2007, p.36). The practice of external price benchmarking can be explained as follows: once a new pharmaceutical is brought to a country’s market, the manufacturing pharmaceutical company submits a price proposal to the regulatory authority. The authority evaluates the adequacy of the proposed price by comparing it to the price of the same drug in the selected benchmark countries. The manufacturing pharmaceutical company’s proposed price is rejected if it is higher than the average drug price in the reference countries; it is obvious that external price benchmarking is only partly suitable for first- or early launch countries, which face the problem of directly negotiating the price with the pharmaceutical company. This might be difficult due to the correct evaluation of the degree of differentiation and the level of substitutability from already existing drugs in the same therapeutic area.

2.2.2. Internal reference pricing

Internal reference pricing is a commonly used regulation scheme where pharmaceuticals are grouped in therapeutic areas and priced by comparing the price with competitor drugs within this therapeutic area. The requirement for the realization of this form of price regulation is that there are already similar drugs available on the domestic market, so it is particularly appreciable to generics and “me-too” products. A generic is an almost identical copy of an original drug, whose patent has expired, whereas a “me-too” product might be the best within its therapeutic area due to an increase in efficacy of an already explored mechanism of action. There are of course differences of internal reference pricing among countries, but the main principals are equal. The price setting process is similar to external price benchmarking, the regulatory authority evaluates the key characteristics of the pharmaceutical that wants to enter the market and puts it into a therapeutic area which contain similar or (in case of generics) identical drugs. The price of these similar or identical drugs is used as reference and the pharmaceutical company’s proposed price is adapted accordingly. For the special case of generics, many countries use a separate internal reference system, which constrains generic prices to be a fixed fraction of the original

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5 ARTS DANIELLE, HABL CLAUDIA, LEOPOLD CHRISTINE, WINDISCH FRIEDERIKE, (2007). Pharmaceutical Pricing and Reimbursement Information Austria

6 Differences are only allowed in the manufacturing process and supplying ingredients
product, which patent has expired. 17 member states\textsuperscript{7} of the European Union used internal reference pricing in 2008, especially countries with a high quantity of generics on the market. A very difficult question when applying internal price referencing is how to evaluate the additional benefit of one drug relative to another, already existing drug. If there are vast improvements towards existing drugs of the same therapeutic area, the evaluation might be very hard as it is based on the evaluation of life\textsuperscript{8}. Another problem of this regulation scheme is to value gradual improvements of existing drugs. Some countries do not allow a premium price to be gained for drugs whose improvement is not vast compared to existing therapeutic alternatives. In other countries a premium is allowed to be obtained for gradual improved drugs and thus the question is raised who should pay this premium, either the payer due to a higher reimbursement price or the patient through additional cost sharing.

\subsection*{2.2.3. Pharmaco-economic assessment}

To use pharmaco-economic assessment in order to evaluate the appropriate price of newly developed pharmaceuticals is a rather new approach, which was first of all introduced in Australia in 1993. To the present, many countries have employed this evaluation system to confirm their pricing and reimbursement decisions. Pharmaco-economic assessment usually describes a cost-effectiveness analysis, where the additional costs of new pharmaceutical products are contrasted with the additional effect related to health outcomes. If there is already a therapeutic alternative on the market, incremental cost-effectiveness is employed in order to determine if the new drug is “worth” the additional cost. In case there is no comparable drug on the market, an implicit or explicit cost-effective threshold has to be set\textsuperscript{9}. Among pharmaco-economic assessment, cost-effectiveness studies are the most prevalent, but in particular circumstances cost-benefit or cost-utility analyses are also performed. While a cost-benefit analysis only captures the quantity of life a drug would save, a cost-effectiveness analysis is more considerable including the quality of life.

\textsuperscript{7} According to PPRI 2008: Austria, Belgium, Bulgaria, Czech Republic, Estonia, Greece, Finland, France, Hungary, Italy, Lithuania, Latvia, Poland, Portugal, Slovenia, Slovakia and the UK.

\textsuperscript{8} Additional utility of new drugs is often measured in improvements in the quality of life and the quantity of life.

of life as well. The quality of life can be scaled as quality-adjusted life years (QALY), which is commonly used in pharmaco-economic assessment. The idea behind QALY is to attach weight to life years in terms of medical condition. A year of life without any disability is rated with a factor of 1; in contrast a year of life with a physical impairment is weighted with a value lower than 1. In the next step, it is to monetarily quantify one QALY in order to use it as parameter of a pharma-economic assessment. The value of a QALY can differ among countries, for example a QALY in the UK is considered to be 30 000 GBP where in the US it is considered to be 50 000 USD\(^{10}\). When taking in account all these factors it might be clear that pharmaco-economic assessment itself is multidisciplinary and technically challenging. Therefore smaller or lower-income countries might face the problem of providing adequate resources and often settle for other countries’ pharmaco-economic assessment or pharmaceutical companies’ assessment. Many countries have established government institutes which attend to pharma-economic assessment.

**Risk-sharing**

Sometimes it might be difficult for payers to make a decision whether to purchase drugs due to a lack of robust outcome analysis at the time the purchase decision is made. In order to cushion this risk a risk-sharing agreement can be made to set targets for the outcome of a pharmaceutical product, where for one medical indication an expected outcome is determined. When these goals cannot be reached, the pharmaceutical company has to partly rebate the costs\(^{11}\). This “outcome guarantee” is especially interesting for new costly medication where the outcome is questionable. Due to the shared risk between purchaser and pharmaceutical company patients gain access to new expensive pharmaceutical products and for the pharmaceutical firms it is easier to sell their products.


\(^{11}\) CHAPMAN STEVE, DURIEUX PIERRE, WALLEY TOM, (2004). Good Prescribing Behavior, in Regulating Pharmaceuticals in Europe: Striving for Efficiency, Equity and Quality, *Open University Press, Maidenhead*
2.3. Trends and conclusions

The prior overview of different approaches to regulate drug prices gives an idea how policy makers can influence drug prices. Of course the regulation schemes differ to some extent among countries, which is in contrast to the global process of discovering and developing new drugs. Pricing and reimbursement policies have to manage the balancing act between encouraging pharmaceutical innovation and holding down drug prices. A general trend towards cost-effectiveness evaluation can be observed among countries meaning that the pricing process is more transparent and evidence-based, which results in a better predictability of regulation decisions for pharmaceutical companies. The advantages of this kind of transparent regulation are obvious, but on the other hand it is more costly and can be made responsible to make the regulation process even more complex for both, payers and pharmaceutical companies.

2.4. Effects of price regulation

When reviewing the existing literature that deals with the effects of price regulation there is a broad consensus that it has detrimental effects on the costs and quality of medical care, but in theory ambiguous effects can be discussed. There is an indirect and a direct way how price regulation can influence the costs and quality of medical care. The former influences medical care through R&D spending. As Vernon (2004) identifies the lagged cash flow\textsuperscript{12} and the expected profits to be the most important determinates for pharmaceutical companies’ R&D spending, both of these determinates are influenced by price regulation. If firms’ expected profits are revised downwards due to price regulation, less external capital will be available to be spent on R&D. Cash flows are also lowered due to price regulation, with the difference that this effect operates with a lag of one year, thus the allocation of resources to R&D might be reduced. Reduced R&D spending is supposed to result in less innovative new drugs. According to Danzon (1997, chapter 5)\textsuperscript{13} this effect is reinforced by

\textsuperscript{12} It is a challenging process for a pharmaceutical company to decide how much to spend on R&D and in order to simplify this question the cash flow of the previous year is used as one of the determinants to define the current period’s R&D investment.

applying most kinds of price regulation. It seems to be comprehensible that less R&D spending will result in a decline of newly discovered chemical entities, which might affect society in two different ways. Assuming that the costs of regulation in terms of higher mortality or morbidity are higher than the savings in R&D spending might result in a less cost-effective health care. In case R&D savings are higher than the costs of regulation, it might result in a more cost-effective health care, but the former seems to be more feasible.

Price regulation can also directly influence medical care by reducing the price of existing drugs. Lower drug prices could cause greater use and therefore might contribute to higher quality and reduction of overall costs. Cutting the prices of drugs might lead to adverse effects on the availability of new drugs, because pharmaceutical countries will first launch their new pharmaceuticals in countries, where drug prices are subject to less regulation. Another reason for firms to delay the launch in strictly regulated markets is that low drug prices in a country could be referenced for other countries, as being used in external price benchmarking.

As the effects of price regulation seem to be ambiguous to a certain degree, it is a very important issue for policy-makers. Several publications have been made dealing with this topic and in the following chapter I want to summarize the most important empirical findings.
Chapter 3

3.1. Hypothesis part

I wanted to examine the influence of the regulation of prices for drugs on pharmaceutical companies’ R&D expenditures. I expected the R&D expenditures of companies, which make most of their profits in Europe to be lower than the R&D expenditures of companies that make most of their profits in the US. This expectation seems quite rational considering that there is generally more price regulation on pharmaceuticals in Europe meaning that drugs are cheaper in Europe compared to the US. As a consequence, companies are able to make more profit in the US and therefore they have more disposable capital to be invested in R&D to explore new medication. In figure 1 I plotted the relation between the logarithmic R&D expenditures divided by sales (l_s_rd_exp) of the 20 pharmaceutical companies used in the model and their market share in the EMEA region\textsuperscript{14} (share_emea).

\textsuperscript{14} Europe (East and West), Middle East and Africa. Of course the most important market within the EMEA market is Europe, so for simplicity the EMEA market can be equated with the European market.
The vertical axis represents the logarithmic R&D expenditures and the horizontal axis represents companies’ market shares in the EMEA region. Obviously, there seems to be a non-linear relationship between these two variables. The graph shows an inverted U shape, that is to say that at lower shares in EMEA R&D expenditures are increasing with the increment of market shares in the EMEA region up to a certain share of sales (“critical market share”) as from that R&D expenditures seem to be decreasing. According to the plot the critical market share seems to be around 30 to 35 percent meaning that a pharmaceutical company that makes more than 30 to 35 percent of its sales in the EMEA region invests less or equal in R&D than a company, whose revenues have a lower ratio of revenues in the EMEA region of total sales. This finding is attributable to price regulation in the EMEA region, which is more distinctive than in the US and can be denoted as “regulation effect”. The reason for this regulation effect might be that it is not optimal for a pharmaceutical company in terms of profits and R&D expenditures to make most of its sales in the EMEA region due to the strict price regulation of drugs. An appropriate strategy to avoid the R&D loss might be to explore new markets where drug prices are not subject to regulation. Of course the EMEA market is important for multinational pharmaceutical companies due to its size and potential profits and, thus, at the beginning, R&D expenditures increase with the share of sales in the EMEA region, but if a pharmaceutical company is too concentrated on the EMEA market, it has a decreasing effect on R&D spending that is likely to be due to price regulation of drugs.

By way of comparison I also plotted the logarithmic R&D expenditures divided by sales (l_s_rd_exp) of the selected 20 pharmaceutical companies and their market share in the North America region (figure 2).
The vertical axis represents the logarithmic R&D expenditures and the horizontal axis represents companies’ market shares in the North America region\textsuperscript{15}. Again, there seems to be a non-linear relationship between these two variables, whereas it is not as distinctive as in EMEA. A significant difference can be observed comparing figure 1 to figure 2. According to the plot, an increasing trend of R&D expenditures can be assessed with increasing companies’ share of sales. Drug prices in the US are not as regulated as in Europe, so companies can make more profit because they are able to sell their drugs at a higher price. Accordingly companies can attain more profit and they have more money to spend on R&D. Again, the graph shows that in case a pharmaceutical company is too concentrated on the US market and makes most of its sales in the US, it has a negative effect on R&D spending. However, the numbers seem to differ from the European case: Here, the “optimal” value of a firm’s engagement in the US lies probably around fifty to seventy percent.

For the sake of completeness, I also plotted the logarithmic R&D expenditures of the selected 20 pharmaceutical companies and their market share in the rest of the world (figure 3).

\textsuperscript{15} For simplicity the North American market can be equated with the US market, since this is the most important market within the North American region.
Again, there seems to be a non-linear relationship between these two variables. It is
striking to see that the rest of the world is not that important compared to the EMEA
and the North American market related to the share of sales, because there seems to
be a peak of frequency at 10 to 20 percent. According to the plot R&D expenditures
are increasing up to a certain share of sales from that a decreasing trend can be
observed.

In summary it can be stated that it seems to be better for a pharmaceutical company
to make more sales of its total sales on the US market in terms of R&D expenditures,
because higher profits can be made selling their drugs due to the less regulated
market. The EMEA region is also important for pharmaceutical companies, but profits
are not that high due to drug price regulation. It might be optimal for a pharmaceutical
company to be present on both markets, more focusing on the US market
respectively in terms of profits and therefore R&D expenditures.
3.2. The Data

The panel data set contains annual information of the world’s biggest 20 pharmaceutical companies \(^{16}\) between 2000 and 2008. The following variables are included: R&D expenditures, cash flow, Tobin’s q, number of employees, debt, the growth of sales, share of the firms’ sales in EMEA region, share of sales in North America and share of sales in the rest of the world \(^{17}\). The data set is unbalanced meaning that there are some missing data for selected companies and years.

3.3. Summary statistics

In table 1, the basic summary statistics for my key variables of my model are briefly shown. The columns contain the mean, standard deviation and number of observations of the variables. The number of observations sometimes differs among the variables due to their availability within the dataset.

In the first row reveals that the mean of the variable R&D expenditures accounts for 2 958 356 016 USD and has a standard deviation of 2 011 252 779 USD. The expenditures for R&D range from 252.83 million USD to 8.3 billion USD. This very broad range within the world’s biggest pharmaceutical companies is represented by the quite large standard deviation.

An examination of the second row shows that the mean of cash flow is 4 618 063 880 USD with a standard deviation of 3 988 683 270 USD. Again, the variable’s broad range is responsible for the large standard deviation.

Tobin’s Quotient is a dimensionless ratio between market value and total assets of a company that is used for the evaluation of a company. The resulting ratio measures the value of one unit capital relatively to its actual purchase price. The higher Tobin’s Q, the higher profit potential of deployed capital is estimated by financial markets. If the value of Tobin’s Q is bigger than 1, the company should do additional investments. In the used dataset the mean of Tobin’s Q is 2.43 and has a standard deviation of 1.69.

\(^{16}\) i.e. as measured in sales Abbot Laboratories, Amgen Inc., Astellas, AstraZeneca, Baxter, Bayer, Bristol Myers Squibb, Eisai, Eli Lilly, Glaxo Smith Kline, Johnson&Johnson, MSD Sharp&Dohme, Novartis, Novo Nordisk, Pfizer, Roche, Sanofi-Aventis, Schering-Plough, Takeda, Wyeth

\(^{17}\) share of sales in Latin America + share of sales in Asia Pacific region
The number of employees is on average 58,996 and varies between 7,196 and 166,900, with a standard deviation of 33,671.

The debt of the companies has an average of 4,232,144,830 USD and a standard deviation of 4,041,986,580 USD.

The companies’ mean annual growth of sales is 9 percent with a standard deviation of 15 percent.

With a share of 46 percent and a standard deviation of 17 percent, companies generate most of their revenues in North America, followed by the EMEA region with a share of 29 percent and a standard deviation of 12 percent. Lastly, the share of sales in the rest of the world contributes 23 percent to their total sales with a standard deviation of 16 percent.

Table 1: Summary statistics of the key variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard deviation</th>
<th>Number of observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>R&amp;D expenditures</td>
<td>2,958,356,016</td>
<td>2,011,252,779</td>
<td>177</td>
</tr>
<tr>
<td>Cash flow</td>
<td>4,618,063,880</td>
<td>3,988,683,270</td>
<td>165</td>
</tr>
<tr>
<td>Tobin’s Q</td>
<td>2.43</td>
<td>1.69</td>
<td>162</td>
</tr>
<tr>
<td>Number of employees</td>
<td>58,996</td>
<td>33,671</td>
<td>169</td>
</tr>
<tr>
<td>Debt</td>
<td>4,232,144,830</td>
<td>4,041,986,580</td>
<td>177</td>
</tr>
<tr>
<td>Growth of sales</td>
<td>0.09</td>
<td>0.15</td>
<td>177</td>
</tr>
<tr>
<td>Share of sales in EMEA region</td>
<td>0.29</td>
<td>0.12</td>
<td>162</td>
</tr>
<tr>
<td>Share of sales in North America</td>
<td>0.46</td>
<td>0.17</td>
<td>162</td>
</tr>
<tr>
<td>Share of sales in the rest of the world</td>
<td>0.23</td>
<td>0.16</td>
<td>162</td>
</tr>
</tbody>
</table>
I also examined the correlations between the explanatory variables used in the model (table 2). The growth of sales is positively correlated with the logarithmic lagged cash flow with a correlation coefficient of 0.16, positively correlated with the share of sales obtained in the EMEA region with a correlation coefficient of 0.07, positively correlated with the and positively correlated with the share of sales made in the rest of the world with a correlation coefficient of 0.02.

The growth of sales is negatively correlated with logarithmic debt divided by sales with a coefficient of -0.12, negatively correlated with Tobin’s Q with a coefficient of -0.02, negatively correlated with the logarithmic number of employees divided by sales with a coefficient of -0.02 and negatively correlated with the share of sales obtained in the US.

The logarithmic debt divided by sales is positively correlated with the logarithmic number of employees divided by sales with a correlation coefficient of 0.20, slightly positively correlated with the share of sales made in the EMEA region with a correlation coefficient of 0.004 and is positively correlated with the share of sales obtained in the US with a positive correlation coefficient of 0.22.

The logarithmic debt divided by sales is negatively correlated with Tobin’s Q with a correlation coefficient of -0.14, negatively correlated with the logarithmic lagged cash flow with a coefficient of -0.13 and is negatively correlated with the share of sales made in the rest of the world with a coefficient of -0.30.

Tobin’s Q is positively correlated with the logarithmic number of employees divided by sales with a correlation coefficient of 0.22, positively correlated with the logarithmic lagged cash flow with a correlation coefficient of 0.33 and positively correlated with the share of sales made in the US.

The logarithmic number of employees divided by sales is positively correlated with the share of sales obtained in the EMEA region with a correlation coefficient of 0.61 and positively correlated with the share of sales made in the rest of the world with a correlation coefficient of 0.08.

The logarithmic number of employees divided by sales has a negative correlation with the logarithmic lagged cash flow with a correlation coefficient of -0.08 and a
negative correlation with the share of sales made in the US with a correlation coefficient of -0.48.

The logarithmic lagged cash flow divided by sales is negatively correlated with the share of sales made in the EMEA region with a correlation coefficient of -0.13, negatively correlated with the share of sales obtained in the rest of the world with a correlation coefficient of -0.09. The logarithmic lagged cash flow divided by sales has a positive correlation with the share of sales made in the US with a correlation coefficient of 0.16.

The share of sales in the EMEA region have a slight positive correlation with the share of sales made in the rest of the world with a correlation coefficient of 0.0063 and have a negative correlation with the share of sales made in the US with a correlation coefficient of -0.68.

<table>
<thead>
<tr>
<th>Table 2: Correlation coefficients between the explanatory variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>salesgr</td>
</tr>
<tr>
<td>salesgr</td>
</tr>
<tr>
<td>l_s_debt</td>
</tr>
<tr>
<td>tobin</td>
</tr>
<tr>
<td>l_s_employ</td>
</tr>
<tr>
<td>l_lag_s_cash_op</td>
</tr>
<tr>
<td>share_emea</td>
</tr>
<tr>
<td>share_emea2</td>
</tr>
<tr>
<td>share_rest</td>
</tr>
<tr>
<td>share_us</td>
</tr>
</tbody>
</table>
3.4. Econometric Background

Having a panel data set, I used the fixed effects model for my estimation. First I want to shortly discuss the advantages of a panel data set and then I want to argue, why a fixed effects model is more appropriate than a random effects model in this case.

In general, there are three different types of data available for empirical analysis: time series, cross section and panel data. In a time series data set, values of selected variables are collected over a period of time, in a cross section data set values of one or more variables are collected at the same point in time for different subjects, such as individuals, firms or countries. Usually cross section data are used to compare the differences among the subjects. Panel data sets combine these two types of data sets by collecting data for different subjects (i.e. firms for my estimation) over a period of time.

According to Baltagi there are some important advantages of panel data\textsuperscript{18}:

- There is heterogeneity within the data set, because variables for different subjects are observed over time.

- Due to the combination of time series and cross-section observations, panel data provide more “informative data, more variability, less collinearity among variables, more degrees of freedom and more efficiency.”\textsuperscript{19}

- The dynamics of change can be better investigated using panel data because of the availability of repeated cross section observations.

- There are effects that simply cannot be detected when using time series or cross section data.

- Panel data allow examining more complicated behavioral models. For example, appearances like economies of scale or technological progress can be better described by panel data.


pp. 3-6
- Based on the availability of many data for different subjects’ variables over time, panel data might minimize the bias when aggregating subjects into broad aggregates.

To sum it up, the use of panel data can enhance econometric analysis in such a manner that may not be possible using simply cross section or time series data, but we have to keep in mind, that there are also some caveats using panel data which will be addressed at the end of the chapter.

3.5. The fixed effects model (FEM)

I used the fixed effects model, because the pharmaceutical companies were not randomly chosen but chosen on their size related to sales. Another reason was that the individual error term \( u_i \) might be correlated with the explanatory variables, because obviously the selected firms are no random sample. Additionally, there is no omitted variable problem with the fixed effects model.

However, the fixed effects model has the disadvantage that the comparison can only take place within the observed firms (that’s why it is also called within-model), but not between the firms. For the latter, a between-model (or random-effects model) should be used. However, as argued above, these would yield biased results.

To get a basic idea of the fixed effects model it can be regarded as OLS regression with a dummy variable for each company. There are of course many different approaches of the fixed effects model depending on the assumptions made about the intercept, the slope coefficient and the error term \( \varepsilon_i \), but in the literature the following model is known as the general FEM:

\[
Y_{it} = \beta_{1i}^{20} + \beta_2X_{2it} + \beta_3X_{3it} + \ldots + \beta_nX_{nit} + \varepsilon_{it}
\]

\( t = 1,2,\ldots,T ; i = \text{different subjects (i.e. companies)} \)

Where \( \beta_1 \) denotes the intercept term, \( \beta \) are the different coefficients of the different variables \( X \) and \( \varepsilon_{it} \) denotes the error term. The subscript \( i \) on the intercept term indicates that the intercepts of the companies might differ across companies which represents the individuality of each company, but the intercept does not vary over

---

20 \( \beta_{1i} \) can be also written as \( \alpha_i + u_i \), where \( \alpha_i \) denotes the constant and \( u_i \) denotes companies’ fixed effects
time (therefore the term “fixed effects”) and the slope coefficients are still regarded as constant. This difference might be explained through special features of each company like for example managerial style (i.e. fixed effects). In this fixed effects model the intercept is not considered to deviate over time, which is called time invariant. To take variation of the intercept into account dummy variables are implemented in the model for each company represented by \( u_i \). Just as dummy variables are used to represent an individual company effect, time dummy variables for each year can be added to take a time effect into account because of factors like technological changes or changes in government regulatory policies which are both very important for pharmaceutical companies.

3.5.1. Caveats on the use of the fixed effects model

Of course there are some problems that have to be faced using the fixed effects model:

- There is a considerable loss of degrees of freedom when introducing too many dummy variables. Therefore, one needs a considerable amount of observations to use the fixed effects model.

- Multicollinearity may occur when using many variables, which can make the precise estimation of one or more parameters difficult.

- The error term \( \varepsilon_{it} \) might be a problem when it is assumed to follow the classical assumptions \( \varepsilon_{it} \sim N(0, \sigma^2) \). This assumption may be modified because the \( i \) index represents cross-sectional observations and the \( t \) index represents time series observations. This problem can be solved by assuming that the error variance is heteroscedastic or that the error term is not autocorrelated.

3.6. The random effects model

The random effects model provides an approach not to lose many degrees of freedom due to the use of dummy variables expressing this ignorance through the disturbance term \( u_{it} \). The basic model is:

\[
Y_{it} = \beta_{1i} + \beta_{2}X_{2it} + \beta_{3}X_{3it} + \ldots + \beta_{n}X_{nit} + u_{it}
\]
Compared to the fixed effects model $\beta_{1i}$ is not considered as fixed, but it is assumed to be a random variable with a mean of $\beta_1$. The intercept value of $\beta_{1i}$ for each company can be described as:

$$\beta_{1i} = \beta_1 + \varepsilon_i$$

where $\varepsilon_i$ denotes the random error term fulfilling the criteria $N(0, \sigma^2)$. For this reason the random effects model contains two (or more) errors: $\varepsilon_i$ which denotes the cross-section, or individual specific, error component and $u_{it}$, which denotes the combined time series and cross-section error component. The main idea behind this model is that the selected companies for the model are a randomly chosen from a very large sample of such companies and that they have a mean value for the intercept $\beta_1$ and the individual differences in the intercept term are considered by the error term $\varepsilon_i^{21}$. Since $\varepsilon_i$ is not directly observable, it is called an unobservable or latent variable.

### 3.7. Fixed effects model or random effects model

To pick the “correct” model is sometimes quite challenging and strongly dependent on the assumptions that have been made about the error component $\varepsilon_i$. If $\varepsilon_i$ is not considered to be correlated with the independent variables, then the random effects model might be the suitable model and if $\varepsilon_i$ is considered to be correlated with the independent variables, then the fixed effects model might be the suitable model.

One main assumption of the random effects model is that the unobserved effect $\varepsilon_i$ is a random drawing from a very large sample. A simple example can show that sometimes this assumption might be violated. In case we want to do research on the unemployment rate across the 50 states in the US, the assumption that the 50 states are a random sample is not feasible. This was also one of the reasons I decided to use the fixed effects model for my estimation, because the pharmaceutical companies were not randomly chosen but chosen on their size related to sales.

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21 “In FEM each cross-sectional unit has its own (fixed) intercept value, in all $N$ such values for $N$ cross-sectional units” see GUJARATI DAMODAR N., PORTER DAWN C., (2009) Basic Econometrics: Fifth Edition, Mcgraw-Hill Higher Education p.648
Of course there are some more criteria to pick the correct choice between the fixed effects model and the random effects model, but listing them all would go beyond the scope of my thesis, nevertheless I want to briefly comment on the most important\footnote{GRIFFITHS WILLIAM E., HILL CARTER R., JUDGE GEORGE G., LEE TSOUNG-CHAO, LÜTKEPOHL HELMUT, (1988). Introduction to the Theory and Practice of Econometrics: Second Edition, *Wiley* pp. 489 - 491}:

- Significant differences in the estimates of the fixed effects model and the random effects model can occur when N (number of cross-sectional units) is large and T (number of time series data) is small. Consider that in the random effects model $\epsilon_i$ is the cross-sectional random component, while in the fixed effects model $\beta_{1i}$ is treated as fixed. The assumption that $\beta_{1i}$ is fixed seems valid when the individual, or cross-sectional units in the sample are to be thought of as not randomly drawn from a larger sample. If this is the case, the fixed effects model is suitable. On the other hand, if the cross-sectional units in the sample are considered to be randomly drawn from a very large sample, the random effects model is suitable. Since the selected companies for my estimation were not randomly chosen but on their size related to sales, I chose the fixed effects model.

- If $\epsilon_i$ is correlated with one or more independent variables, the random effects model provides biased results, whereas in the fixed model $u_i$ is allowed to be correlated with the independent variables. Since it might be possible, that $u_i$ is correlated with my regressors, I decided in favor of the fixed effects model.

- The random effects model is more efficient than the fixed effects model, when N is large, T is small and its assumptions are not violated.

### 3.8. Empirical Part

In my model my dependent variable is the logarithmic R&D expenditures divided by sales (\texttt{l_s_rd_exp}), the set of explanatory variables includes:

- salesgr: growth of sales in percent
- \texttt{l_s_debt}: logarithmic debt (USD) divided by sales (USD)
- tobin: Tobin’s Q
- \texttt{l_s_employ}: logarithmic number of employees divided by sales (USD)
• \text{l\_lag\_s\_cash\_op}: logarithmic cash flow (USD) divided by sales (USD) of period t-1
• \text{share\_emea}: share of sales in EMEA region
• \text{share\_emea2}: squared share of sales in EMEA region
• \text{share\_us}: share of sales in North America
• \text{share\_rest}: share of sales in the rest of the world

The variable share of sales in North America is also not included\(^{23}\), because it is used as reference variable meaning that the coefficients of share\_emea, share\_emea2 and share\_rest represent a comparison with the share of sales made in the US.

The equation of my fixed effects model reads as follows:

\[ \log(\text{rd\_exp/sales})_{it} = \alpha_1 + \beta_1 \log(\text{salesgr})_{it} + \beta_2 \log(\text{debt/sales})_{it} + \beta_3 \text{tobin}_{it} + \beta_4 \log(\text{employees/sales})_{it} + \beta_5 \log(\text{lagged cash flow/sales})_{it} + \beta_6 (\text{share\_emea})_{it} + \beta_7 (\text{share\_emea2})_{it} + \beta_8 (\text{share\_rest_rev})_{it} + u_i + \varepsilon_{it} \]

\( t = 2000, 2001, \ldots, 2008 \) and \( i = 1, 2, \ldots, 20 \)

where \( \alpha_1 \) denotes the constant, \( \beta_n \) denote the different coefficients of the independent variables, \( u_i \) denote the companies’ fixed effects and \( \varepsilon_{it} \) denotes the error term which is independent and identically distributed. Table 3 illustrates my empirical findings. By way of comparison I also performed a random effects estimate in order to compare my results out of the fixed effects estimation.

\(^{23}\) There can always only put (n-1) dummy variables into a model, using the omitted variable as reference variable
Table 3: Differences between random and fixed effects

<table>
<thead>
<tr>
<th>Dependent Variables</th>
<th>random effects</th>
<th>fixed effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>l_s_rd_exp</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>explanatory variables</th>
<th>random effects</th>
<th>fixed effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>constant</td>
<td>-2.7894***</td>
<td>-3.2974***</td>
</tr>
<tr>
<td></td>
<td>(0.000)</td>
<td>(0.000)</td>
</tr>
<tr>
<td>salesgr</td>
<td>0.0978</td>
<td>0.1153*</td>
</tr>
<tr>
<td></td>
<td>(0.134)</td>
<td>(0.089)</td>
</tr>
<tr>
<td>l_s_debt</td>
<td>-0.0585***</td>
<td>-0.0631***</td>
</tr>
<tr>
<td></td>
<td>(0.000)</td>
<td>(0.000)</td>
</tr>
<tr>
<td>tobin</td>
<td>-0.0197</td>
<td>-0.0159</td>
</tr>
<tr>
<td></td>
<td>(0.145)</td>
<td>(0.252)</td>
</tr>
<tr>
<td>l_s_employ</td>
<td>-0.1107*</td>
<td>-0.1057*</td>
</tr>
<tr>
<td></td>
<td>(0.053)</td>
<td>(0.071)</td>
</tr>
<tr>
<td>l_lag_s_cap</td>
<td>0.0062</td>
<td>0.0242</td>
</tr>
<tr>
<td></td>
<td>(0.780)</td>
<td>(0.471)</td>
</tr>
<tr>
<td>share_emea_v</td>
<td>2.1176</td>
<td>3.8502*</td>
</tr>
<tr>
<td></td>
<td>(0.179)</td>
<td>(0.058)</td>
</tr>
<tr>
<td>share_emea2</td>
<td>-3.9456*</td>
<td>-5.7044**</td>
</tr>
<tr>
<td></td>
<td>(0.066)</td>
<td>(0.028)</td>
</tr>
<tr>
<td>share_rest_v</td>
<td>-0.8670**</td>
<td>0.0786</td>
</tr>
<tr>
<td></td>
<td>(0.015)</td>
<td>(0.919)</td>
</tr>
<tr>
<td>number of observations</td>
<td>121</td>
<td>121</td>
</tr>
</tbody>
</table>

Notes: ***, **, and * indicates statistical significance of 1, 5, and 10 respectively. p-values in parentheses
We can see that there are some significant variables. The variable salesgr with a p-value of 0.08 is significant at 10 percent-level with a coefficient of 0.11, which means that an increase of 1 percentage of salesgr will result in an increase of 11 percent of R&D expenditures. This result is quite comprehensible, because an increase of pharmaceutical companies’ sales is usually accompanied with an increase of profits, thus there is more capital that can be invested in R&D. The variable l_s_debt is highly significant at 1 percent-level with a p-value of 0.00 with a coefficient of -0.06. If there is an increase in l_s_debt of one percent, there will be a decline of R&D expenditures of 0.1 percent. This finding goes along with rational expectations that an increase of companies’ debts yields a decrease in R&D investments, because less capital is disposable for investments. The regressor l_s_employ is significant with a p-value of 0.07 at 10 percent-level with a coefficient of -0.10. Increasing l_s_employ by one percent goes hand in hand with a 0.1 percent decrease of R&D expenditures. This result meets my expectations, because the number of employees is an important cost factor for the pharmaceutical industry and therefore is consistent that more employees will result in less available money to be invested in R&D.

The most important variables for my research are the share of sales in the EMEA and the squared shares of sales in the EMEA region. I used these two variables as regulation proxies, and we can see, that both of the variables are significantly different from zero, whereas share_emea is significant with a p-value of 0.05 at 5 percent-level with a positive coefficient of 3.85 and share_emea2 is highly significant with a p-value of 0.02 at 5 percent-level with a negative coefficient of 5.70. These two coefficients completely meet my expectations, because the R&D investments are increasing until a critical market share in the EMEA region and from that value R&D investments are decreasing compared to the shares of sales made in the US. Referred to the R&D expenditures for a pharmaceutical company it is better to have higher shares of sales in the US market up to a certain value, because higher prices can be achieved for their drugs due to a lack of price regulation. This can be interpreted as evidence that the strict price regulation of drugs in the EMEA region has a negative impact of pharmaceutical companies’ R&D spending and can be made responsible for a loss of new innovative drugs. The higher the share of sales made in the EMEA region, the higher is the negative impact of the squared term, in other words the more sales a company makes in the EMEA region beyond the optimal market share, the higher is the R&D investment loss compared if a company
had made more shares of its sales in the US. In order to avoid losses of R&D investment due to the strict price regulation in the EMEA region, a company should not make more sales beyond the optimal or critical market share in the EMEA region. Since my model contains a share of sales in the EMEA region term and its squared term, the optimal market share for a company in the EMEA region can be computed easily through differentiation:

First, recall my model:

\[
 y = \alpha_1 + \beta_1 \log(\text{salesgr}) + \beta_2 (\text{debt/sales}) + \beta_3 \text{tobin} + \beta_4 \log(\text{employees/sales}) + \beta_5 \log(\text{lagged cash flow/sales}) + \beta_6 (\text{share\_emea}) + \beta_7 (\text{share\_emea})^2 + \beta_8 (\text{share\_rest}) + \epsilon
\]

Differentiating y with respect to \(share\_emea\) yields:

\[
\frac{\partial y}{\partial x} = \beta_6 + 2\beta_7 (share\_emea) = 0
\]

In order to maximize, I set the equation zero and rearrange it:

\[
(share\_emea) = -\frac{\beta_6}{2\beta_7}
\]

If we plug in the value of the coefficients of \(\beta_6\) (3.85) and \(\beta_7\) (-5.70) the equation yields 0.33. The optimal market share for the EMEA region of a pharmaceutical company is therefore 33 percent. This optimal market share also meets my expectations, because when we go back to fig 1 in the hypothesis part we can see, that a share of sales beyond 33 percent in the EMEA region results in decreasing R&D expenditures.

By way of comparison I also performed a random effects estimation with exactly the same variables as to use it as “robustness check”. My findings are also illustrated in table 3.

The independent variable salesgr now has turned insignificantly, but with a p-value of 0.13 it can be regarded as trend. With a positive coefficient of 0.09 it almost equals the coefficient of salesgr in the fixed effects estimation. The regressor \(l_s\_debt\) is again significantly different from zero with a p-value of 0.00 with a coefficient of -0.05 it almost equals the value of the coefficient of the fixed effects estimation. The variable \(l_s\_employ\) is significant at the 5 percent-level with a coefficient of -0.11,
which closely matches with the coefficient of the fixed effects estimation. The regulation proxy share_emea has turned insignificantly, but the algebraic sign remains the same compared to the fixed effects estimation. The squared share_emea term is again significantly different from zero with a p-value of 0.06. The coefficient value of -3.94 is similar to the value estimated by the fixed effects approach. The squared term can be considered to have more weight than the regular share_emea term, because the higher the share of sales made in the EMEA region, the higher is the negative impact of the squared term.

To sum it up the findings of the random effects estimation go along with the results of the fixed effects estimation, the value of the coefficients are quite similar and coefficients’ algebraic signs equal. Hence the results of the fixed effects approach can be regarded as robust.
Chapter 4

4.1. Conclusion

Price regulation of pharmaceuticals can theoretically contribute to society’s total utility by lowering drug prices that might result in lower costs per use and therefore greater use, which could lead to reduced costs of care. Conversely, the costs of regulation might exceed this effect when considering the reduced R&D spending and the delayed launches of new drugs. In my thesis I tried to find an approach to make the effect of price regulation on R&D expenditures detectable. Usually pharmaceutical companies are present on both markets, the EMEA market and the US market. To use the shares of sales in the different regions as regulation proxy was quite a good approach to show that in a regulated market R&D investments are decreasing compared to a less regulated market. The negative influence of price regulation on pharmaceutical companies’ R&D investments cannot be denied and is shown empirically in my thesis. I could also show, that for a pharmaceutical company the optimal market share in the EMEA region is 35 percent related to R&D expenditures, beyond this value the “regulation effect” becomes important and reduces companies´ R&D spending. It also seems consistent that a less regulated drug market makes it possible for companies to realize higher prices for their drugs and therefore more capital can be invested in R&D, which is important for the development of new innovative drugs. The more difficult question is to exactly quantify the loss of R&D spending due to price regulation in the EMEA market. Of course pharmaceutical companies are globally acting and the EMEA region is an important sales market, but for a firm it seems better not to be too concentrated on the EMEA market because there the prices of drugs are more regulated compared to the US market.
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Abstract (German)

Curriculum Vita

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