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Predictive Model for Type 1 Diabetes.
A Case Study

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Predictive Models for Type 1 Diabetes. A Case Study
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Abstract
Linear models were identified from glucose-insulin data of a type 1 diabetic patient. The models were used for simulation and prediction with 10, 30, 60, 90 and 120 min prediction horizon. The predictions were able to track the measured blood glucose, at least for the lower prediction horizons, so that hypo-and hyperglycemic events can be prognosed. The best predictions were achieved with ARX and ARMAX models, predictions for the subspace-based models were not as good as for the autoregressive models.
Later a minimum variance controller was developed with an ARMAX model that was identified before. The controller could reduce the variance of the blood glucose concentration as well as eliminate hyperglycemic events. However it introduced the risk for hypoglycemia, which means that there is still some effort to be done. Linear models were identified from glucose-insulin data of a type 1 diabetic patient. The models were used for simulation and prediction with 10, 30, 60, 90 and 120 min prediction horizon. The predictions were able to track the measured blood glucose, at least for the lower prediction horizons, so that hypo-and hyperglycemic events can be prognosed. The best predictions were achieved with ARX and ARMAX models, predictions for the subspace-based models were not as good as for the autoregressive models. Later a minimum variance controller was developed with an ARMAX model that was identified before. The controller could reduce the variance of the blood glucose concentration as well as eliminate hyperglycemic events. However it introduced the risk for hypoglycemia, which means that there is still some effort to be done.

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1 Introduction

Diabetes mellitus is a wide-spread disease with more than 180 million sufferers worldwide [25]. Treatment is based upon insulin injections to force the blood glucose level within a lower and an upper threshold. Crossing these boundaries leads to hypo- or hyperglycemia with immediate effects like brain damage, coma and potentially death or chronic damages. The appropriate control of blood glucose is therefore of vital importance to the patient.
The aim of this thesis was to identify models of the glucose-insulin metabolism on the basis of type 1 diabetes patient data. Various models were compared with each other to see how much their structure deviates and which influence different methods have on the models. Later the models were used to develop a controller for the optimal insulin profile, so that the variations of the glucose level and thus the risk for hypo-or hyperglycemia can be reduced. In the following a basic insight into the physiological background of the disease diabetes mellitus will be provided to familiarize the reader with the subject. On the basis of that a literature survey with regard to previous work and current research topics will be presented. Section 4 and 5 describe the identification of the models and the control part respectively. The thesis completes with a final conclusion.

2 Medical Background

To understand the principle of the insulin-glucose-metabolism and the disease diabetes mellitus a basic insight into glucose, insulin, glucagon and diabetes mellitus will be given.
This chapter is based on [10].

2.1 Glucose

Glucose, also known as dextrose or grape sugar, is a monosaccharide and ingested into the body by carbohydrate food.
Glucose is one of the major energy deliverers in our body. Muscle cells are not permeable for glucose and therefore use fatty acids as energy sources. However during exercise muscle cells become permeable for glucose and can use this instead. Another way to enable glucose for entering the cell membrane is the availability of insulin which connects with receptors on the cell membrane. Thus a mechanism is ignited which among other things transports glucose into the cell. The only cells that don't need insulin to absorb glucose are brain cells, which can hardly use any other energy sources besides glucose. Therefore it is very important that the blood glucose concentration stays above a certain level to guarantee the required amount of glucose in the brain cells.
Insulin causes the excess glucose (about 60%), that cannot be taken up in cells after a meal, to be stored in the liver as glycogen. Glycogen is turned into glucose and secreted in times between meals when the blood glucose level is lower. When no further glucose can be stored in the liver as glycogen glucose is turned into fatty acids with the help of insulin and transported to the adipose cells.

A fasting person normally has a blood glucose concentration in the range of $4-5\frac{\text{mmol}}{\text{L blood}}$ ($70-90\frac{\text{mg}}{\text{dL blood}}$). After breakfast the concentration rises up to 7.8mmol/L (140mg/dL). Two hours after a meal the glucose concentration should be within the normal level again.
Implications of a hypoglycemic shock, which means a too low blood glucose level ($<$ 2.22-2.78mmol/L (40-50 mg/dL)), are nervous irritability, hallucinations, sweat, fainting, seizures and coma. Immediate treatment have to be large amounts of glucose or glucagon.
Hyperglycemia means too high blood glucose level (more than 7.8mmol/L (140 mg/dL)) and should be avoided, too. A higher glucose level in the blood than in the cells causes dehydration in the cells due to osmose. Furthermore a high glucose level in the urine leads to osmotic diuresis by the kidneys and

thus more fluids and electrolytes than usual are excreted. High glucose levels have long-term-effects that can impair tissues like blood vessels and increase the risk for heart attacks, strokes, end-stage renal diseases and blindness.

2.2 Insulin

Insulin is secreted in the islets of Langerhans in the β-cells in the pancreas and directly ingested into the blood. Insulin is a peptide hormone and has a half-life of about five minutes.
Whenever the blood glucose level is increasing (e.g. after a meal containing carbohydrates) insulin is secreted to enable glucose uptake and usage in cells (not in brain cells) and glucose storage. Insulin also prevents glucagon secretion and the release of fatty acids so that glucose is used for energy instead. Since insulin causes the surplus glucose to be converted into fat and supports the transportation into the fat cells in the same way as the transportation of glucose into the muscle cells, the storage of fat without insulin is nearly not feasible.
Further positive effects of insulin are the promotion of protein storage and the transportation of some amino acids into cells. Insulin also helps to produce new proteins and prevents degeneration of proteins.
For growth insulin is as important as the growth hormone is.
Factors that increase the insulin secretion are increasing blood glucose level, increasing amount of free fatty acids and/or amino acids in the blood and certain hormones whereas insulin secretion is prevented by decreasing blood glucose and fasting. Thus insulin is not only controlled by the blood glucose level.
While the carbohydrates are digested there should be a delay in the insulin secretion. Otherwise the newly ingested glucose would immediately be used in the muscles or stored in the liver and there would not be enough glucose for the brain cells. This is assured by a hormone called somatostatin.

Insulin for insulin-treatment is available in different forms. There is "regular" insulin which is effective for 3-8 hours and "long-lasting" insulin which is mixed with protein derivatives to increase the duration for absorption to 10-48 hours.

2.3 Glucagon

Glucagon is a peptide hormone and mainly has the opposite effect of that of insulin. Like insulin it is produced in islets of Langerhans in the pancreas but secreted when the blood glucose level is decreasing.

Due to glucagon being present in the blood, gluconeogenesis is initiated which converts the glycogen in the liver back into glucose and therefore the blood glucose level is rapidly increased. Glucagon also has an effect on fatty acids, so that these are used by the cells as energy sources.
Other causes for glucagon secretion besides a falling glucose level are certain amino acids and exercise. However the impact of exercise on glucagon secretion is not yet totally understood.
Generally the insulin mechanism is more important than the glucagon mechanism because glucose cannot be used or stored otherwise, but during times of starvation or exhaustive exercise the glucagon mechanism gains in importance due to a constantly low glucose level.

2.4 Diabetes mellitus

In diabetics insulin can either not be produced or not be utilized. Therefore glucose cannot be used nor stored except for the amount of glucose that is used in brain cells. This leads to a raise in blood glucose concentration and in the use of fats and proteins. There are two types of diabetes which will be explained in the following.

Type 1 Diabetes: In type I diabetic persons there is no insulin secretion and thus insulin has to be injected. Therefore this type is also called insulin-dependent diabetes mellitus (IDDM) or juvenile diabetes mellitus because it often occurs in young people. The disability to produce insulin is caused by damaged β-cells of the pancreas or a disease which blocks the insulin production. A major factor for type 1 diabetes might be heredity.
The blood glucose level in type 1 diabetes patients can be as high as 16-70 mmol/L (about 300-1200mg/dL) and is thus much higher than the level in a healthy person (up to 8-10 times higher).
The lack of insulin debilitates storage and production of proteins causing extreme weakness and deranged functions of the organs. The absence of insulin in the fat metabolism activates the release of fat into the blood and the usage as energy sources by almost all cells (besides the brain cells). This is not as threatening as the lack of insulin in glucose metabolism but possible consequential damages are atherosclerosis, heart attacks, cerebral strokes and other vascular accidents.
Further symptoms for type 1 diabetes are polyuria, intra-and extracellular dehydration, extreme thirst and weight loss. If not treated the disease can lead to death in a few weeks.
In the insulin-therapy usually one dose of long-lasting insulin is given each day to build up a basal insulin concentration and a bolus injection when a

meal is taken to regulate the increased amounts of glucose ingested with the meal.

Type 2 Diabetes: In contrast to type 1 diabetes patients type 2 diabetics are able to produce insulin. However their bodies are resistant to the effects of insulin. Large amounts of insulin are being secreted and existing in the blood, but cannot be used.
This type is also known as non-insulin-dependent diabetes mellitus (NIDDM) or adult onset diabetes and often appears after the age of 30.
Due to the insulin-resistance the amount of insulin in type II diabetics is increased enormously to compensate for that. Even in the early stages of the disease the increased level of insulin does not help to store and use glucose after a carbohydrate meal. As a consequences moderate hyperglycemia occurs. In later stages when the pancreas is exhausted and cannot produce these excessive amounts of insulin severe hyperglycemia cannot be avoided.
Since adiposity is one of the major risks for type 2 diabetes an efficient treatment, at least in the early stages, is exercise, caloric diet and weight loss. In later stages when these procedures don't have any more effect, drugs for increasing the insulin-sensitivity can be prescribed and if that does not help any more insulin has to be injected.

2.5 Link between diabetes mellitus and control theory

2.5.1 Conclusion

In non-diabetic persons insulin secretion is controlled by the amount of glucose in the blood. An increasing blood glucose level causes the release of insulin which supports the usage and storage of glucose and thus preventing the blood glucose level from rising too high. A decrease in blood glucose level prevents the secretion of insulin but promotes the secretion of glucagon to cause an increase in the glucose level again.
In Type I Diabetes mellitus patients insulin cannot be produced. Thus insulin has to be injected. To figure out the right amount of insulin is a very challenging task and often causes hyper- or hypoglycemia. A hypoglycemic shock occurs when the blood glucose level falls below a certain threshold. This has immediate effects like fainting and coma. Hyperglycemia instead refers to a too high glucose level and often remains unrecognized. Hyperglycemic effects are long-term consequences like dehydration, risk for strokes and heart-attacks and damages to tissues.

Since brain cells and muscle cells during exercise are able to take up glucose without the help of insulin and because the amount of ingested carbohydrates

is not easy to estimate, it is hard to figure out the impact of insulin on the blood glucose level.

2.5.2 Why is diabetes mellitus a control theory problem?

Since the glucose-insulin-metabolism in healthy bodies is a control loop inherently it is only natural to use control theory as a means in therapy for diabetic persons. To be able to mimic the natural regulation of insulin secretion the blood glucose concentration has to be measured and the appropriate amount of insulin has to be administered to the body. For this reason measuring devices for glucose level and insulin pumps for the injection of insulin have been developed.

3 Previous work, state of the art and research topics

3.1 Devices for glucose measuring and insulin administration

3.1.1 Glucose monitoring

Up to now diabetics measure the blood glucose for insulin therapy several times a day. With a blood glucose meter a drop of blood is tested for the amount of contained blood glucose [27].
Continuous glucose monitoring system (CGMS) measures the glucose concentration every 10 min for a duration up to 3 days. A sensor for measuring the glucose level is placed in the interstitial fluid in the subcutaneous tissues. Calibration has to be done several times a day with a conventional glucose meter [14]. CGMS are available in commercial use since 1999 [17].
Current research is done on noninvasive methods. The glucose is measured by means of for instance infrared light [23] or iontophoresis [31] through the skin without causing injury to the body. Noninvasive methods have the advantages that they are not causing any pain and that glucose can be measured continuously, but a disadvantage is that the glucose per penetrated tissue is measured and not glucose per blood volume [31].

3.1.2 Insulin delivery

Insulin is usually injected into the subcutaneous (SC) tissue with a syringe or an insulin pen. Insulin pumps for continuous subcutaneous insulin infusion (CSII) have been developed and allow a continously delivery of insulin instead of several injections a day. The basal insulin can easier be dispensed constantly than with a long lasting insulin which is injected once a day. The insulin boluses which are injected in times of meals can also be administered with the pump. However insulin pumps are far more expensive than conventional needles or pens and are thus not as widely used [26].
Even though the Intravenous (IV) route would be favorable from a control point of view the SC route is much easier to access and advantageous in safety aspects [2]. However the SC route introduces time delays in insulin absorption.

3.2 Type 1 diabetes modeling

A large number of models for the glucose-insulin interaction have been developed. Some models represent the physiological dynamics of glucose and insulin while others are empirical models. A few examples of both types will be presented in the following.

3.2.1 Physiological models

In 1981 one of the first physiological model of glucose-insulin dynamics was published by Bergman [4]. It is known as the "minimal model" or "Bergman model" and consists of three differential equations which describe the dynamics of glucose and insulin in the blood and of insulin in remote compartment. The drawback of this model is however that it does not consider SC insulin injection. For this reason the model has been extended in several studies [8]. A much more complex model with 19 compartments representing the glucose metabolism in a nondiabetic person was developed by Sorensen in 1985 [29]. A simpler model, but yet not as rudimental as the minimal model, was designed by Hovorka et al [16]. In 2002 they developed a nonlinear model with three subsystems for glucose kinetics and one subsystem for insulin action. The subsystems for glucose kinetics consist of two compartments, the "accessible" compartment or plasma compartment and the "non-accessible" compartment. The model was later revised with respect to the insulin absorption so that the model takes fast acting and slow acting insulin into account [14].

3.2.2 Empirical models

Empirical models have been identified both with simulated data from a physiological model as in the previous section and with measured data of type 1 diabetes patients.
In 1994 Bellazzi et al. [3] identified ARX models from type 1 diabetes patient data. The inputs were meal information (discrete values for low, medium or high), physical exercise (in discrete values for low, normal and high), insulin injection (continuous values) and special events (such as hypoglycemia). The models were used for predictions with horizons of 2h, 4h and 6h. The authors noticed a large variation in the prediction quality of different patients.
Five years later Bremer and Gough [5] estimated models from simulated data with literature models, non-diabetic patient data and type 1 diabetic patient data. The models were used for prediction of future blood glucose concentration. They conclude that even their simple and linear models are able to warn for hypoglycemia better than it would be possible with unfrequent

blood glucose measurements alone. However they attach importance to a daily update of the model's parameters for receiving reasonable predictions. In 2004 Hovorka and his group [15] used their physiological model for blood glucose prediction with data from 15 IDDM patients. Since the model parameters vary from patient to patient the parameters were re-estimated at each control step using Bayesian parameter estimation. They showed that adaptive nonlinear model predictive control is suitable for glucose control in fasting type 1 diabetes patients.
Sparacino et al. [30] identified first order polynomial models and first order AR models from continuously monitored data of 28 type 1 diabetics for the purpose of prediction. The study demonstrates that it is possible even with simple models to predict hypoglycemic events 20-25 min ahead in time which is just early enough to prevent the hypoglycemia by ingesting sugar.
Similar studies were accomplished by amongst others Reifman et al. [28] and Eren-Oruklu et al. [7].

3.3 Control strategies for type 1 diabetes

Traditional insulin therapy can be viewed as a partially closed-loop control [2]. The physician decides on the insulin dosage in a feed forward control based on the patient. A feedback control with blood glucose measurements specifies the present amount of insulin to be injected.
To improve the current way of insulin administration research is done on decision support systems and on a fully closed loop control with little needs of interaction by the patient.

3.3.1 Decision support systems

About 20 years ago the development of advisory systems for insulin treatment began. Model parameters for glucose-insulin dynamics are identified from measurements of the patient. Using adaption the parameters are updated every day. The ideal insulin amount is then calculated with the models and with information about the upcoming meals. Those systems are mostly based on infrequent glucose measurements with a glucose meter several times a day [14].
The Diabetes Advisory System (DIAS) [13] is based on a compartment model of the glucose metabolism whose parameters are estimated from self-monitored glucose data. Inputs are carbohydrate intake and insulin injection. The system works in three modes. The learning mode is used for estimating the parameters, the prediction mode is able to make predictions of the blood glucose with carbohydrate and insulin data. It is designated for detecting

hypo- or hyperglycemia and to see which influence changes in meals or in the insulin dose can have. The advisory mode should define the ideal insulin dosis for the least risk of hypo- or hyperglycemia.
The system AIDA (Automated Insulin Dosage Advisor) [22] uses a model of glucose-insulin interaction combined with a knowledge-based system to make predictions of the glucose level and to calculate the appropriate insulin dose.
UTOPIA (Utilities for optimizing insulin adjustment) [6] collects data in home measurements so that the physician can adjust the therapy in the next consultation.
There are also telemedicine systems that consist of a medical unit (MU) and a patient unit (PU). Both units communicate with each other via the internet. The PU is supposed to collect data in home measurements and to give daily proposals on the insulin dose. The physician can correct the therapy with the MU. Examples for these systems are Telematic Management of Insulin Dependent Diabetes Mellitus (T-IDDM) [1] and DIABTel [11].
Because of large paramater and response variations between patients and influences on the glucose metabolism like exercise and stress decision support systems are currently rarely used [3].

Besides training programs for teaching diabetic patients how to choose their amount of insulin dose according to the food have been developed as well [14]. In the 1980s a hospital diabetes teaching and treatment program had been carried out [24]. The program led to an improvement in the glucose control and to less hospitalization. The above mentioned AIDA system is also able to simulate a set of case studies for educational purposes [22].

3.3.2 Artificial pancreas

The artificial pancreas has been in the interest of research since the 1970s. The first systems were featured with intravenous (IV) glucose sampling and IV insulin infusion. Because of that they were not suitable for use in patients. When CGMS first was available more than 20 years later this was a breakthrough for the closed loop glucose control. Furthermore the technology of insulin pumps had been improved over the years as well [17]. Subcutaneous (SC) insulin infusion however introduced time delays because of the longer insulin absorption from subcutaneous tissue into the blood. This problem was solved when fast acting insulin was launched [14].
Components of the artificial pancreas are a CGMS, a controller and an insulin pump [17]. The closed loop system should be able to work as standalone system but might require additional information about meals because

of time delays in insulin absorption [18]. Most of the controllers use pole-placement, PID-control (proportional-integral-derivative) or MPC (model predictive control) [14]. For testing, the controller is often arranged in a laptop or a handheld computer. Intensions are to place it in the insulin pump or in an other handheld device for end product use [17]. Possibilites to use the artificial pancreas are to only switch it on during the night or to also use it during the day. Since there are no interactions like meal-intake or exercise, the glucose control is easier to achieve during the night [18]. Furthermore if hypoglycemia occurs during the night it mostly remains unrecognized. [17]. Therefore it seems reasonable to focus on an overnight controller first [18]. Only very few systems were actually implemented and tested [14]. That is the reason why there still is no closed loop glucose control system on the market right now even though research on this area had begun more than 30 years ago.

4 Identification

A set of carbohydrate, insulin and glucose data was given to the purpose of model identification. Several models and methods were investigated. Simulation and prediction of the output obtained with the models was compared with the real output.
The models and evaluation criteria will be explained below and a description of how to estimate the model order for different models will be given. After that the data set will be examined more closely and finally the identification results will be presented and discussed.

4.1 Description of models and evaluation criteria

Different types of models were identified, namely ARX, ARMAX and state space models. For judging the model's quality measures such as FIT, variance accounted for and standard deviation of simulation/prediction error were used.

4.1.1 ARX

Autoregressive models with exogenous input (ARX) are described by:

$$A(z^{-1})y_k = z^{-n_k}B(z^{-1})u_k + w_k \tag{1}$$

with

$$A(z^{-1}) = 1 + a_1 z^{-1} + ... + a_{n_a} z^{-n_a} \tag{2}$$

$$B(z^{-1}) = b_0 + b_1 z^{-1} + ... + b_{n_b} z^{-n_b+1} \tag{3}$$

and time-delay n_k [21].
y_k characterizes the model output, u_k characterizes the model input and w_k is white noise. n_a relates to the number of poles and n_b to the number of zeros + 1.

4.1.2 ARMAX

ARMAX models (Autoregressive moving average with exogenous input) have the following form [21]:

$$A(z^{-1})y_k = z^{-n_k}B(z^{-1})u_k + C(z^{-1})w_k \tag{4}$$

with $A(z^{-1})$ and $B(z^{-1})$ as described in (2) and (3) and

$$C(z^{-1}) = 1 + c_1 z^{-1} + ... + c_{n_c} z^{-n_c} \tag{5}$$

As it can be seen easily ARX models are a specialization of ARMAX models with $C(z^{-1}) = 1$.

4.1.3 State-space-models

State-space-models in innovation form are given by:

$$\begin{aligned} x_{k+1} &= Ax_k + Bu_k + Ke_k \\ y_k &= Cx_k + Du_k + e_k \end{aligned} \tag{6}$$

where $u_k \in \mathbb{R}^m$ denotes the input, $y_k \in \mathbb{R}^p$ the output, $x_k \in \mathbb{R}^n$ the state vector and $e_k \in \mathbb{R}^p$ is the innovation, i.e. one-step ahead prediction error [21].

4.1.4 Evaluation criteria

FIT
The FIT-value is calculated as

$$FIT = \left(1 - \frac{\|\hat{y} - y\|}{\|y - \bar{y}\|}\right) \cdot 100\% \tag{7}$$

where $\hat{y}$ is the predicted output, y is the measured output, $\|\hat{y} - y\|$ denotes the norm of the error between predicted and measured output and $\bar{y}$ denotes the mean of the measured output [19].

VAF
VAF means variance accounted for and is calculated by

$$VAF = \left(1 - \frac{var(y - \hat{y})}{var(y)}\right) \cdot 100\% \tag{8}$$

with $var(y - \hat{y})$ being the variance of the error between estimated and measured output and $var(y)$ the variance of the measured output [12].

Standard Deviation of simulation/prediction error (SDE)
The standard deviation of the simulation/prediction error $e_k = y_k - \hat{y}_k$ is calculated as [19]:

$$\hat{\sigma} = \sqrt{\frac{1}{N-1}\sum_{k=1}^{N}(e_k - \bar{e})^2} \tag{9}$$

with

$$\bar{e} = \frac{1}{n}\sum_{k=1}^{N} e_k \tag{10}$$

4.2 Software

The software used throughout this thesis was Matlab, in particular the System Identification Toolbox and the SMI-Toolbox by B. Haverkamp and M. Verhaegen [12].

4.3 Model determination

For each of the models the order was estimated as explained below. ARX models were obtained by least-squares-estimation, ARMAX by prediction-error-method. State-space models were identified with the Matlab command "n4sid" and with Multivariable Output-Error State Space Algorithm (MOESP) in the SMI-Toolbox.
Tables with the models' order are in Appendix B. Polynomials and matrices that were chosen for the prediction part are displayed in Appendix C.

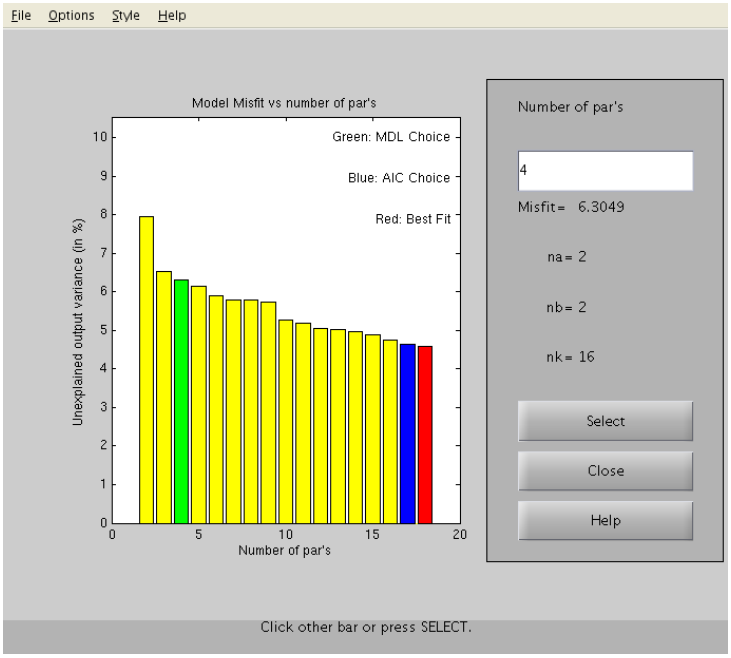


Figure 1: Order Selection for ARX: Identification of Day 1 and Input 1

4.3.1 Model order estimation

The Matlab command "delayestimation" was used to estimate the delay that each input most likely has. With the values that were received from the delay estimation the command "modelorderestimation" was applied for ARX models with $n_a = 1:10$, $n_{b_1} = 1:10$ and $n_{b_2} = 1:10$ (see Fig. 1). With the suggested orders the number of poles, the number of zeros and the delays were adjusted to receive as good results for simulation or prediction as possible.

Since ARMAX models differ from ARX models only in the description of the noise the results for delays and number of poles and zeros from the ARX models were used as a starting point to adjust them and the number of zeros for the noise input to receive as best ARMAX models as possible.

For subspace-based model identification the plot of singular values for different model orders suggests which model order most likely is the appropriate one (see Fig. 2).

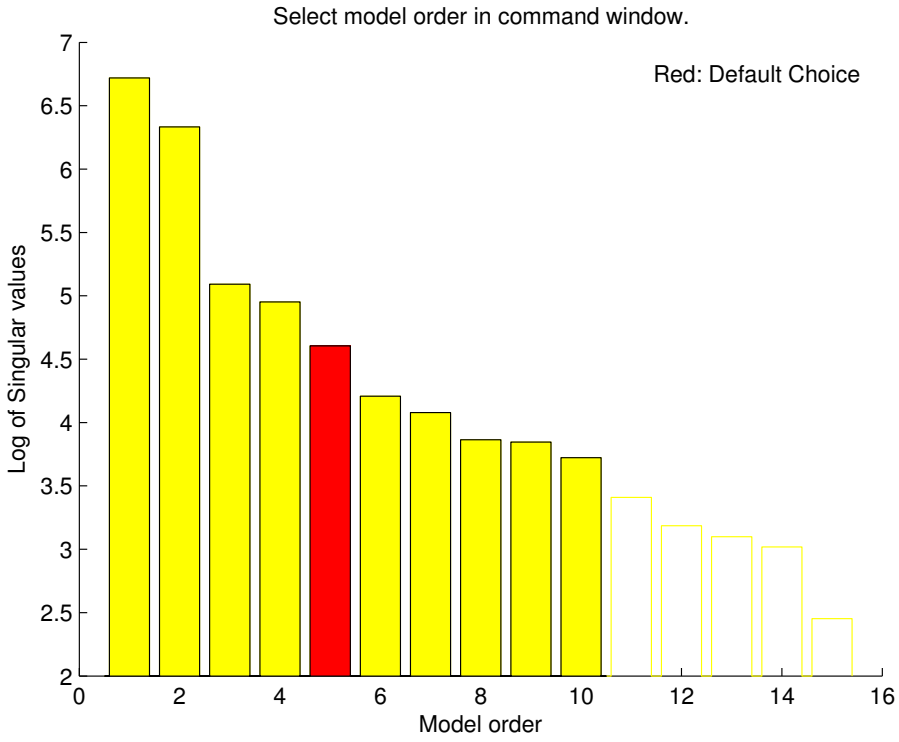


Figure 2: Singular values for the method n4sid for identification of Day 1

4.4 Data used for identification

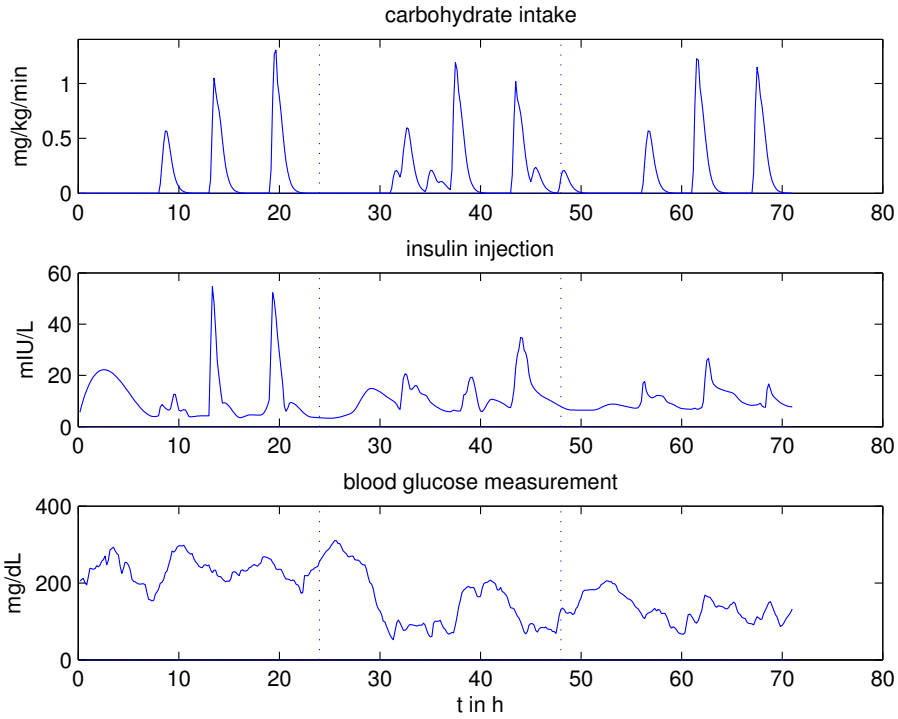


Figure 3: Measurement data used for identification

4.4.1 Description of the dataset

Fig. 3 shows the data used for identification. Input 1 is the appearance of glucose in plasma. Input 2 is the insulin concentration in the blood. The output are CGMS (continuous glucose monitoring system) measurements.
The data was collected from an IDDM patient during a clinical observation for the duration of 3 days. The patient received standard meals for breakfast (42g carbohydrates) at 8:00, lunch (70g carbohydrates) at 13:00 and dinner (70g carbohydrates) at 19:00. Sampling time of the data is 10 minutes.
It can be seen that the patient has a relatively high blood glucose level of more than 150mg/dL on the first day. However the patient went into hypoglycemia on the beginning and at the end of the second day. This can be seen in the low blood glucose level of almost 50mg/dL and on the additional carbohydrate intake between breakfast and lunch and after dinner on the second day. This could possibly have been extra glucose intakes to prevent

a hypoglycemic shock and make the blood glucose level rise again.

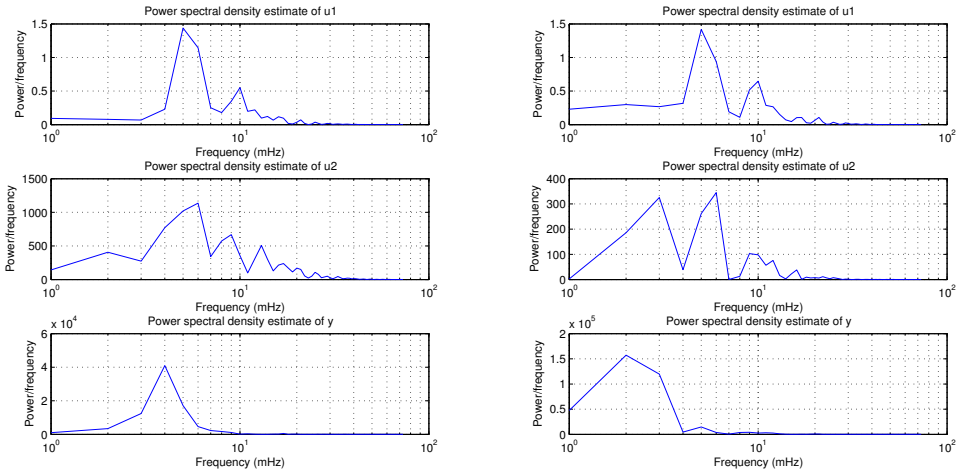
4.4.2 Pre-treatment of the data before identification

The time-scale was divided into three parts, so that each part represents one day. The partitioning can be seen as dotted lines in Fig. 3. Models identified from data of day 1 were validated with day 2 and models identified from data of day 2 were validated with day 3.
For the purpose of investigating whether the detrending of data has an influence on the quality of the models or not, the mean value of the output was removed before identification and added to the simulated/predicted output afterwards.

The natural logarithm of the data values was taken before identification. Detrending had to take place after the calculation of the logarithm. If the data was to be detrended first, positive and negative values with a mean value of zero would have appeared and caused trouble for the calculation of the logarithm. After the models had been identified the exp-function was applied to the predicted output to receive the correct output values.
First the logarithm was taken for input and output data (part A) and then for output data only (part B). Taking the logarithm of input data caused trouble where the input was zero. In this case the first 48 data samples, where the carbohydrate intake is zero, had to be removed.

4.4.3 Spectral analysis of the data

The power spectral densities (PSD) of inputs and output for day 1 and day 2 can be seen in Fig. 4. The PSD of input 1 has a peak at 5 mHz and a smaller one at 10 mHz. For frequencies between 10 and 20 mHz there are some small ripples but these are not as distinctive as the other two peaks. The PSD of input 2 has a peak at 6 mHz and two smaller ones at 9 mHz and at about 12 mHz for day 1. In the PSD of day 2 there are only those at 6 and 9 mHz visible and an additional peak at 3 mHz. The PSD of both days contain some smaller ripples in the frequency range of 10-20 mHz, like it was mentioned for input 1 already. This behavior is however not visible for the output's PSD. This has a peak at 4 mHz for day 1 and one at 2 mHz for day 2.
It can be deduced that the carbohydrate intake has similar frequency behavior for both days, whereas insulin injection and blood glucose measurement

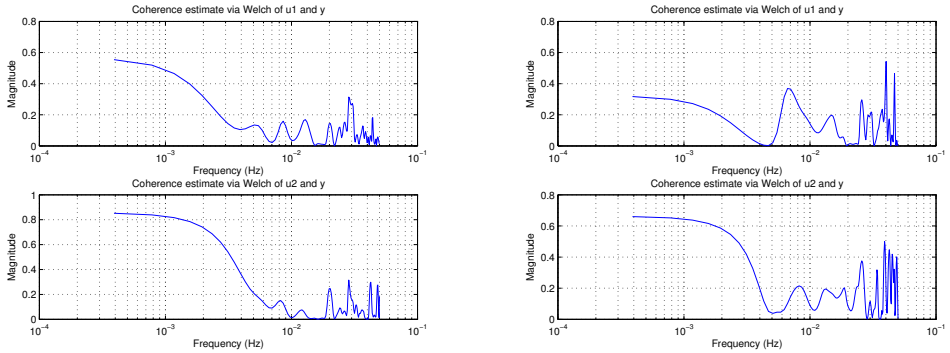


(a) day 1 (b) day 2

Figure 4: Power Spectral Densities of inputs and output for day 1 and 2

have little deviation in their frequency behavior for day 1 and 2. It is not surprising that the carbohydrate frequency behavior is similar, because the meals were standard meals on all days. However the profile of the insulin injection is different on day 2 than on day 1 (see Fig. 3).
A peak in the PSD characterizes that the respective signal is exciting for these frequencies [21]. In other words if the PSD is zero, the signal is not sufficiently exciting and unsuitable for identification.

The coherence is plotted in Fig. 5. Generally the coherence is quite low and it is lower for input 1 than for input 2. For day 2 the coherence is lower than for day 1 in the beginning, but has higher peaks for higher frequencies. The coherence is almost constant until about 1 mHz, but drops after that frequency and is close to zero. For higher frequencies the coherence has some peaks up to a magnitude of 0.3 for day 1 and 0.5 for day 2.
A coherence value differing from 1 suggests that there is noise interference, another input impacting the output, nonlinear behavior between input and output or non-zero initial values with low damping [21]. However it can not be distinguished which one of the above mentioned influences are affecting the system. Since there are two input signals, but only one is represented in each coherence plot, it is obvious that the coherence will not be equal to 1. If one coherence plot was close to 1 for all frequencies this would mean that the other input does not affect the output. This might be one explanation why there is low coherence between inputs and output, but other possibili-



(a) day 1 (b) day 2

Figure 5: Welch coherence estimates for day 1 and 2

ties like further not regarded inputs, non-linear input-output relationship or noise cannot be ruled out.

4.5 Simulation

Using the input data and the estimated initial state the output was simulated with each model and compared to the real output. For comparison the VAF and the standard deviation of the simulation error (SDE) were used.
To investigate the influence of the initial state, simulation of the validation day was performed with the estimation of the initial state from identification data and from validation data.
For ARX and ARMAX models the initial state was estimated with the Matlab command "findstates". For models estimated with the MOESP methods from the SMI toolbox the command "dinit" from the same toolbox was used. The initial state for models identified with "n4sid" were estimated with "findstates" and with "dinit" to determine which one of the two methods is more suitable.

4.5.1 Results

VAF and SDE can be seen in Table 1 and 2 in Appendix A. The selected model orders are to be found in Table 15 in Appendix B. For lack of space the polynomials and matrices which were identified are not displayed here. They are shown as example for the experiment "prediction" for the reason of being compared later.
The accordance of simulated output with measured output is ranging from not fitting at all (negative VAF or very high standard deviation) to more or less acceptable values (VAF more than 90% and low SDE). However good fits are mostly only achieved for simulating the same data that was used for identification.

The simulation results will be exemplified on two models.
Fig. 6 and 7 show the simulation of days 2 and 3 with models identified by the command "n4sid" from detrended data of day 2. In Fig. 6, where identification and validation day are the same, it can be seen that the simulation basically follows the measured output. The high frequency movements however are not caught with the simulation. Still the simulation is one of the best ones and trends for running into hypo- or hyperglycemia can be recognized. The VAF for this case is with a value of 93.39% quite high and the SDE has a relatively low value of 19.53 compared to other simulation standard deviations, indicating a good model quality.
Fig. 7(a), which displays the simulation of the following day with initial states estimated from day 2, shows that there is a great mismatch between

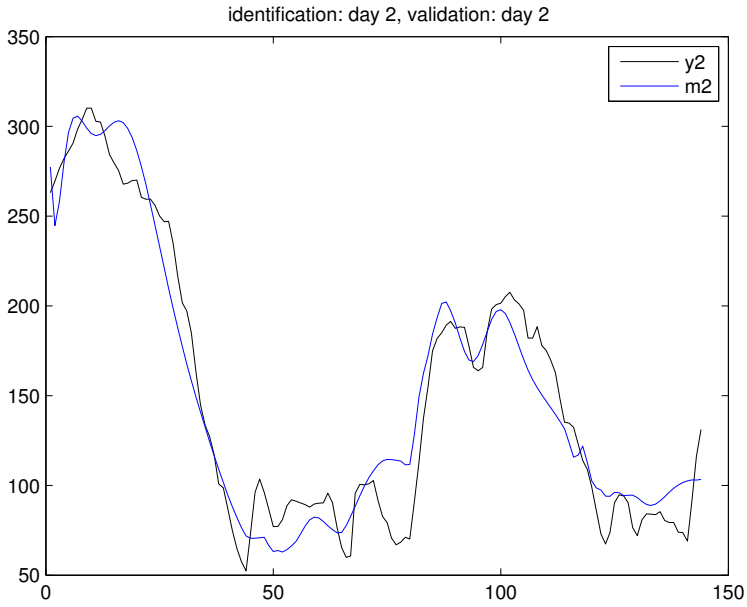
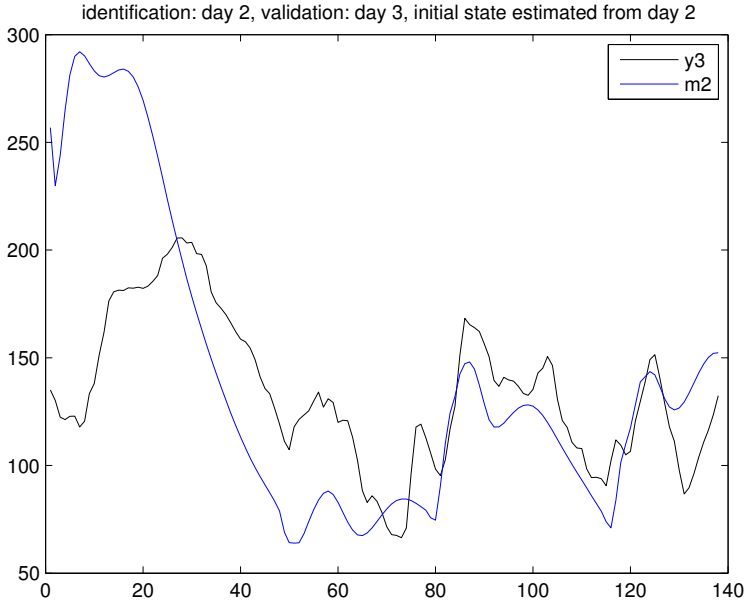
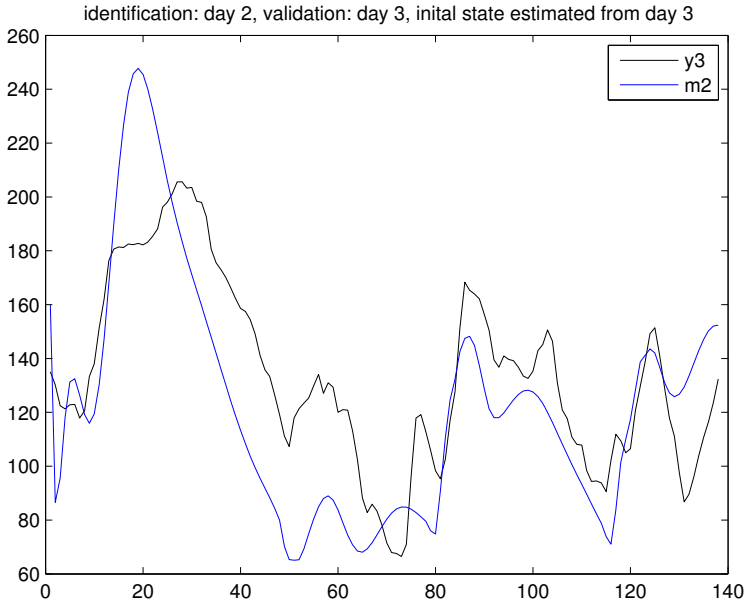


Figure 6: Simulation of day 2 with 5th order n4sid-model identified from day 2 after detrending the data, initial state estimated with dinit

simulated and measured output in the first half of the simulation. This is due to the wrong initial values. In the second half of the simulation, when the effect of the initial values is gone, the simulated output can keep track of the measured output much better. The VAF is negative here (-175.47%) which is a sign of poor simulation quality and the SDE is much higher than before (56.33).
In the case of taking the correct data for estimating the initial state (Fig. 7(b)) the first few values are in accordance with the measured output but after a few samples the simulated output is much too high and drops too early. After the second half of the simulation the simulated output looks similar to the one with wrong initial values. This shows that for longer simulations the effect of the correct initial states diminishes. The VAF is at least positive (29.03%) but still very low. The SDE is 28.59 and thus takes on only half of the value than in the case before.
The Bode plots in Fig. 8 show that the models Bode plot try to mimic the basic trend of the original data's Bode plot but that they do not coincide thoroughly. Especially the Bode plot of the model identified from day 1 is not able to follow the high frequency spectrum of the measured data. However the Bode plots shouldn't be interpreted too much because even the

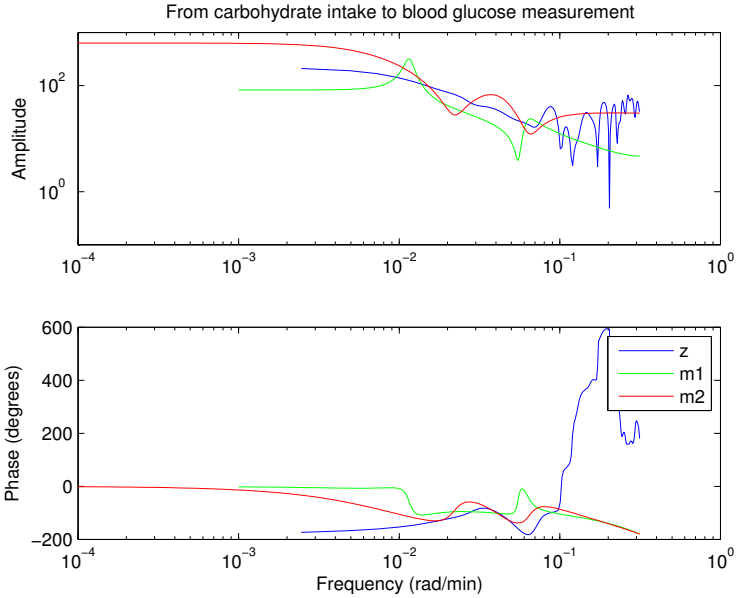


(a) Simulation of day 3, initial state estimated from day 2

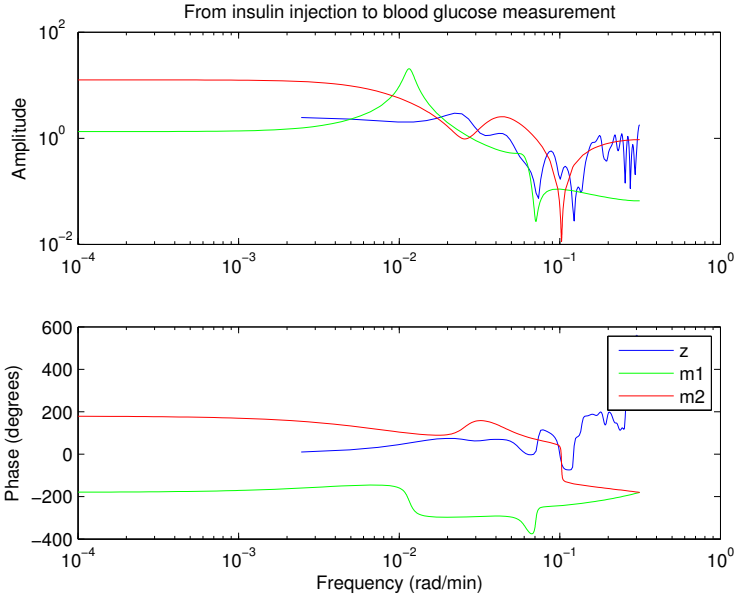


(b) Simulation of day 3, initial state estimated from day 3

Figure 7: Simulation of day 3 with n4sid-model of order 5 identified from day 2 after detrending the data, initial state estimated with dinit

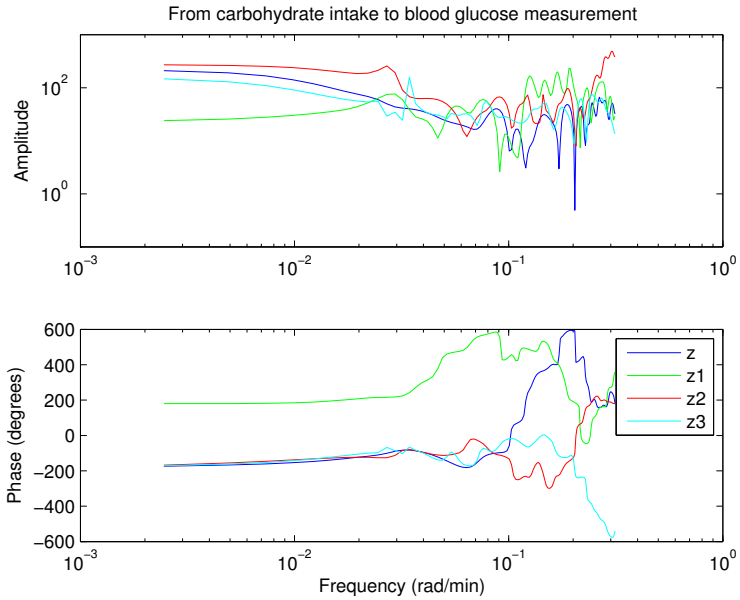


(a) input carbohydrates

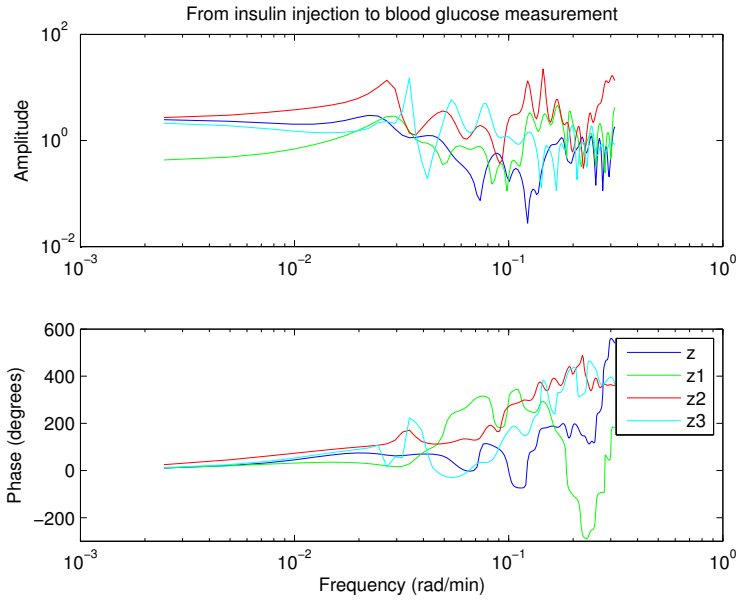


(b) input insulin

Figure 8: Bode diagram for models identified with n4sid compared to the Bode diagram of the measured data (m1 was identified from day 1 and m2 from day 2)



(a) input carbohydrates



(b) input insulin

Figure 9: Bode diagrams of the original data for the whole sequence and for each day

Bode plots of each day compared to each other deviate quite a lot (see Fig. 9).

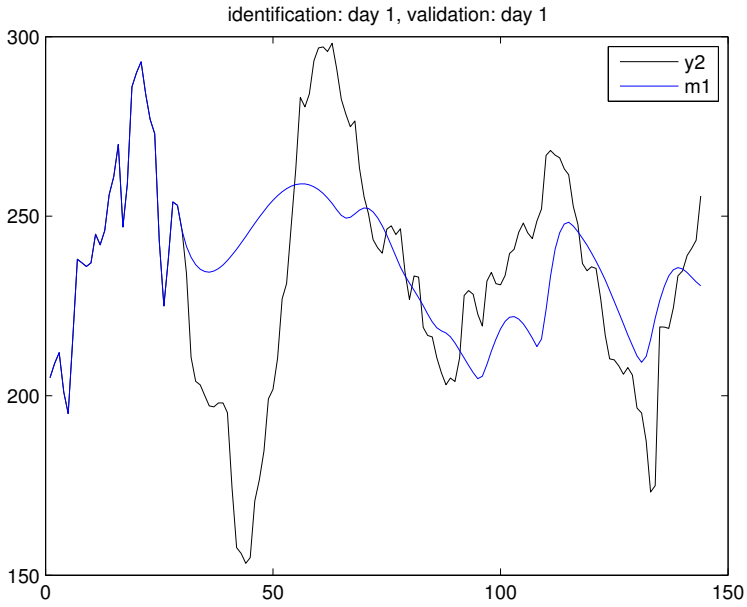
Fig. 10(a) shows the simulation of day 1 with an ARX model identified from day 1. The first values are matched perfectly, this is due to the known initial values. Later the simulation follows the measured output only scarcely. Some peaks are tracked but others are ignored completely. VAF (27.31%) and SDE (27.48) indicate a poor simulation quality. Fig. 10(b) displays the simulation of day 2 with the same model. Initial states were estimated with day 2. Again the beginning of the simulation is matched exactly because of the initial values. However the rest of the simulation does not follow the measured output at all. In this case the VAF is higher than for the simulation of day 1 (57.27%) but the simulation quality does not look any better than that for day 1. This shows that the measurement indicators like VAF do not always tell the truth. During simulation it occurred several times that the VAF suggested a good model quality but the simulation plot was poor or vice versa. The SDE is with 49.66 quite high, thus indicating bad quality.

An example for the ARMAX simulation can be seen in Fig. 11 and for MOESP models in Fig. 12.

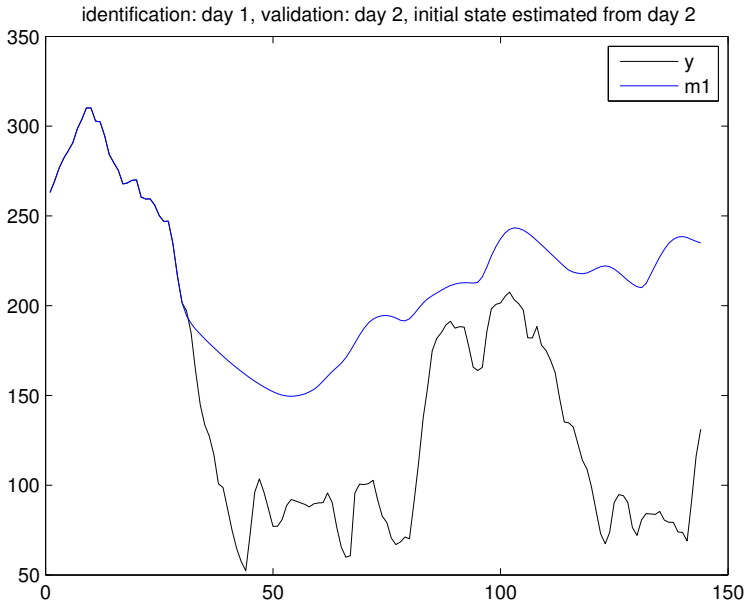
4.5.2 Discussion

Simulation of data for the next day with initial states estimated from the identification data results in very poor values for VAF and SDE. Using the validation data for estimating the initial states provides much better simulations. This is obvious because the initial state from the day before might be several mg/dL apart from the actual one. The simulation might then start much too high or too low. Therefore the initial value does have a great influence on the quality of simulated output and it is essential to estimate the initial values as close to the real ones as possible. However this is hard to achieve if the data that should be used for simulation are not available before the simulation, like for instance in an online surveillance of blood glucose level. In this case a few measurement samples have to be taken to estimate the initial value first.

The identification of day 2 provides much better results in simulation of day 2 than in day 3. This is an effect that will be seen later in the other experiments as well.
Regarding the detrending of data there can not be seen much improvements in comparison to not detrending the data. In some cases detrending results in better fits, but in others it seems more advisable not to detrend the data.

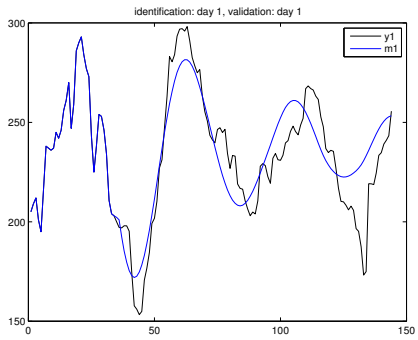


(a) day 1

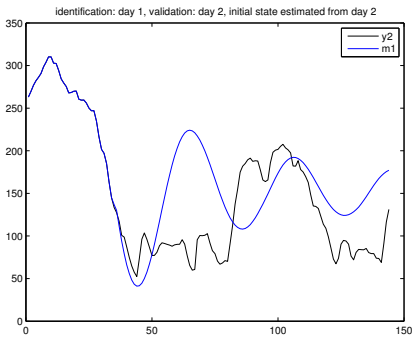


(b) day 2

Figure 10: Simulation of day 1 and 2 with ARX model identified from day 1 (order: $n_a = 2$, $n_{b1} = n_{b2} = 1$, $n_{k1} = 16$, $n_{k2} = 30$)

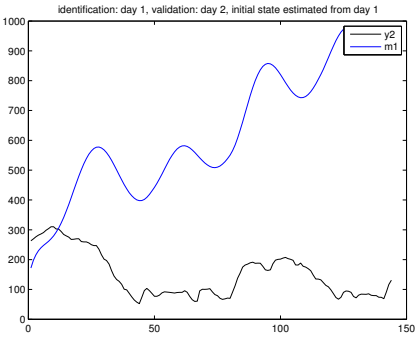


(a) day 1

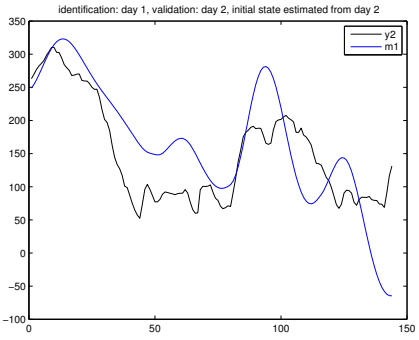


(b) day 2

Figure 11: Simulation of day 1 and 2 with ARMAX model identified from detrended data of day 1 (order:$n_a = 2$, $n_{b1} = n_{b2} = 1$, $n_c = 3$, $n_{k1} = 16$, $n_{k2} = 37$)



(a) initial state estimated from day 1



(b) initial state estimated from day 2

Figure 12: Simulation of day 2 with unstable MOESP model of order 4 identified from day 1

Low-order ARX models lead to unstable models and thus can not be used for simulation. Higher-order models (with input delays of around 20) however result in pole-zero-plots with some poles and zeros close to each other. Generally pole-zero-cancelations indicate a too high model order [21]. Still higher orders produce very poor simulations. For the first few values simulation and real output fit very nicely, this is due to the estimated initial state. After this however the simulation doesn't follow the real output at all.
ARMAX models are not really better than ARX although they are stable even for low orders.
For the n4sid command the estimation of the initial state with "findstates" is not much different to the one with "dinit", though it seems as if the simulations for "dinit" generally are a bit better than the others.
It was not possible to achieve stable models for all MOESP models. With the correct initial values this might not be such a big challenge, but with the wrong initial values the simulation is completely useless (see Fig. 12). For higher orders the Bode plots and the standard deviation of the simulation error was a bit lower, but there were plenty of pole zero cancellations. Since there was not much improvement in higher orders, it was tried to keep the order as low as possible.

The models' Bode plots try to mimic the general trend of the real Bode plots but are not capable of following the higher frequency changes. Mainly the models' Bode plots have a magnitude offset and are a bit lower in magnitude than the original ones.
The correlation of residuals mostly stay within the limits (except for MOESP models, where the residuals are a bit higher in the beginning but are decreasing into the limits for increasing lag). If the residuals were much higher than the margins this would indicate that there is something wrong with the model. See Fig. 14 as an example for the correlation of the residuals.

It is hard to say which one of the models is the best one. When a model seems to be best for some days this model produces poor simulations for other days. Furthermore even if VAF and SDE indicate good model quality the simulation plot might be of a very poor quality. Taking every aspect into consideration "n4sid"-models seem to be most appropriate for simulation, followed by ARMAX models and finally by ARX and MOESP models.

4.6 Prediction

For prediction a Kalman predictor with prediction horizons of 1, 3, 6, 9 and 12 steps according to 10, 30, 60, 90 and 120 min was used.

4.6.1 Results

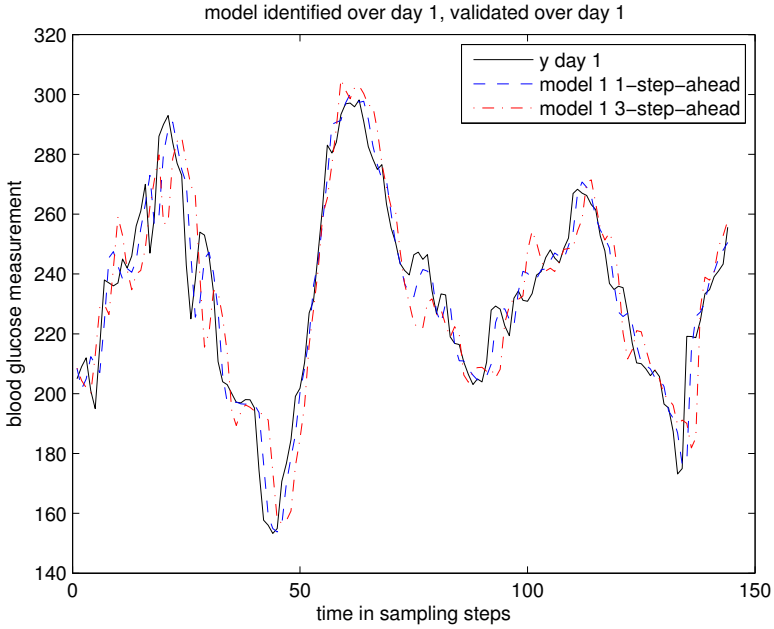
FIT, VAF and standard deviation of prediction error (SDE) were taken for each prediction and are displayed in Tables 3, 4 and 5 in Appendix A. The results for logarithmizing input and output data can be seen in tables 6 - 8 and for logarithmizing only output data in tables 9 - 11 in the same appendix. The models' orders can be found in tables 16 - 18 in Appendix B as well as the polynomials and A-matrices in Appendix C.

The prediction will be illustrated on the example of a 5th order model identified with n4sid from detrended data of day 1. Fig. 13 shows the 1- and 3-step-ahead predictions. For both days the 1-step-ahead prediction is very close to the original output. The 3-step-ahead prediction mainly follows the original output, there are however some overshoots and delays. The delay is quite large for the second day (Fig. 13(b)) from sampling steps 50 to 80, covering several sampling steps.
Since data and order are the same as in the part simulation the Bode diagram is the one that was already shown in Fig. 8.
The correlation of the residuals are displayed in Fig. 14. They are almost entirely in the limits, but don't have white noise properties.

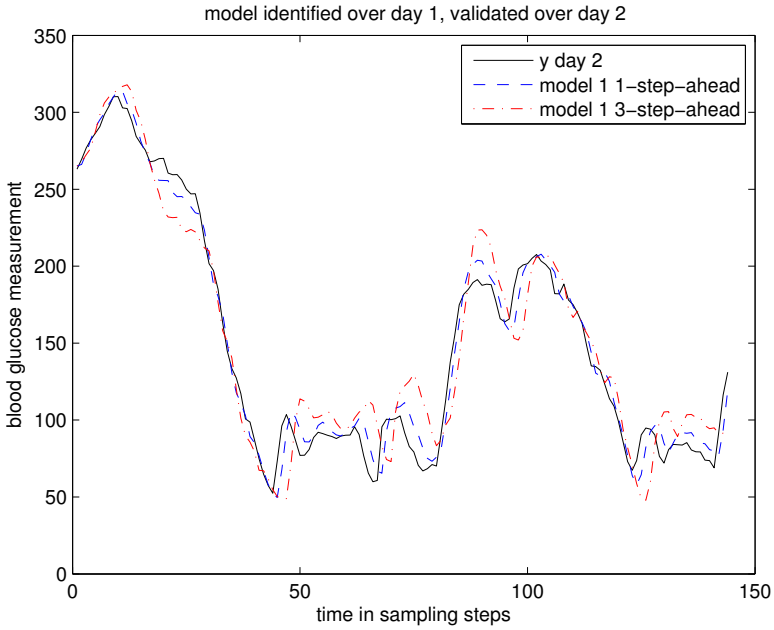
Fig. 15 - 17 show examples of the predictions with ARX, ARMAX and MOESP models.

There was no difference in taking the mean value first and dividing the data into three parts afterwards or vice versa.
The effect of removing the first order trends was also examined but this didn't result in different outcomes than only removing the mean value.

Regarding the calculation of the logarithm the prediction for both parts (part A: taking the logarithm of input and output data, part B: taking the logarithm of output data) is similar to the one without taking the logarithm. Sometimes normal prediction has better FIT, VAF and SDE values, but sometimes the values for the logarithmized data are better. The mean FIT values are about 5% higher for normal data up to a prediction horizon of 6

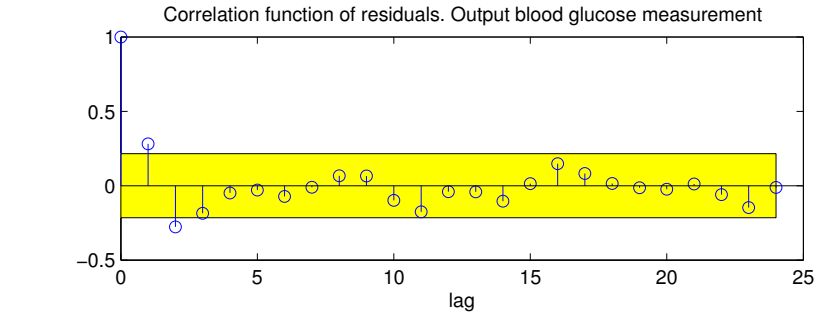


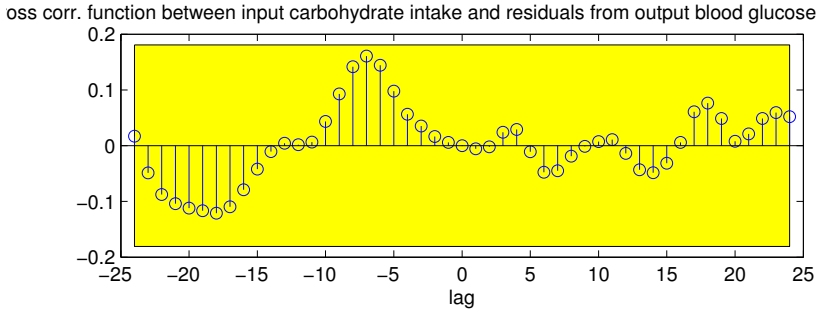
(a) day 1



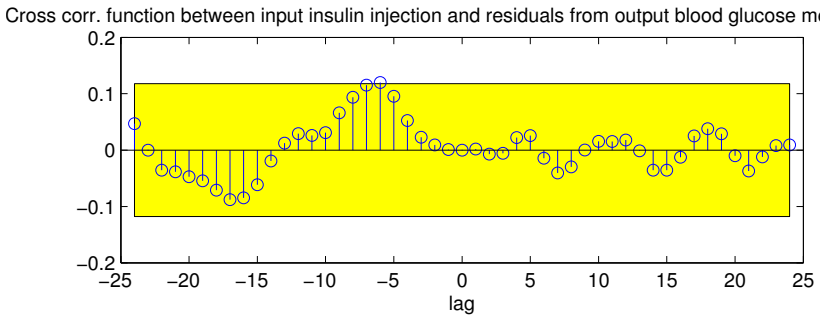
(b) day 2

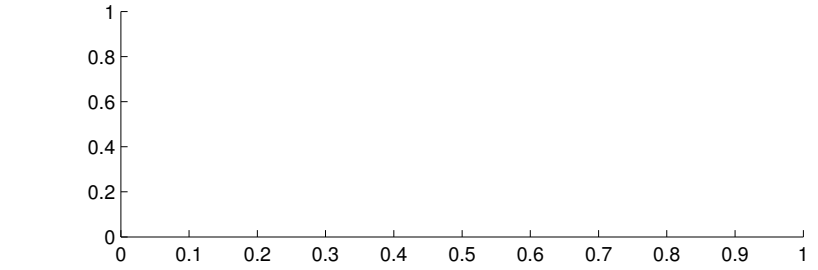
Figure 13: Prediction of day 1 and 2 with 5th order n4sid model identified from day 1 after detrending the data





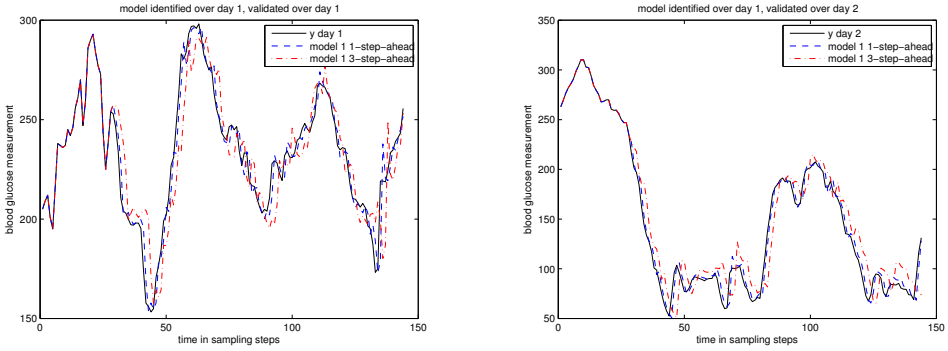
(a)





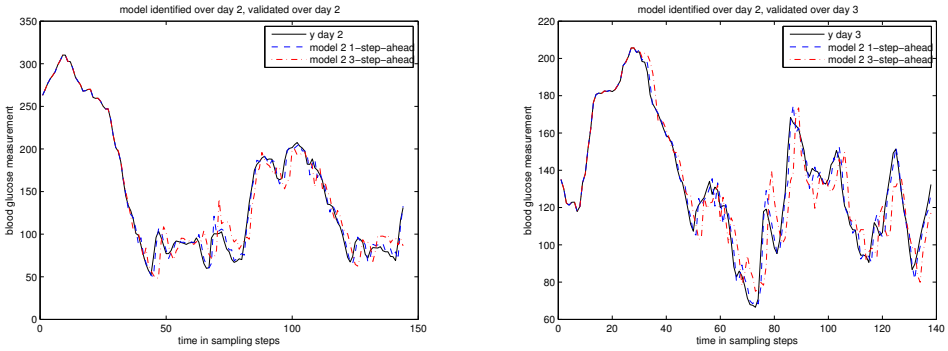
(b)

Figure 14: Correlation of the residuals of day 1 and n4sid model identified from day 1 after detrending the data



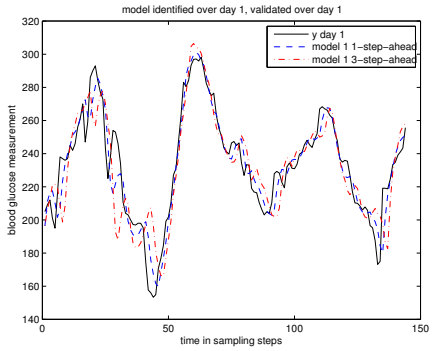
(a) day 1 (b) day 2

Figure 15: Prediction of day 1 and 2 with ARX model identified from day 1 (order: $n_a = 2$, $n_{b1} = n_{b2} = 1$, $n_{k1} = 16$, $n_{k2} = 28$)

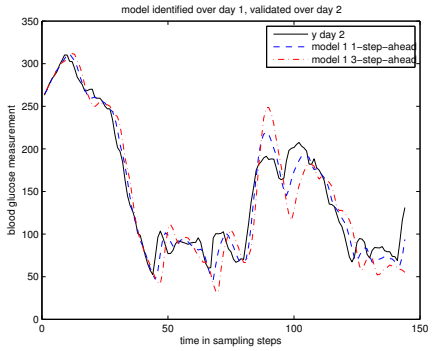


(a) day 2 (b) day 3

Figure 16: Prediction with ARMAX model identified from detrended data of day 2 (order: $n_a = 2$, $n_{b1} = 2$, $n_{b2} = 1$, $n_c = 5$, $n_{k1} = 10$, $n_{k2} = 34$)



(a) day 1



(b) day 2

Figure 17: Prediction of day 1 and 2 with 4th order MOESP model identified from day 1

steps. For larger prediction horizons the FIT values of logarithmized data become better than those of normal data (Fig. 29(b) in Appendix D).
Except for the n4sid models, the FIT values for part A are up to 50% better than those for part B. For n4sid models the FIT values of part B are 5% better than those of part A. For increasing prediction horizons the difference in FIT, VAF and SDE values is rising (see Fig. 29(a) in Appendix D).

4.6.2 Discussion

The FIT values for day 2 are up to 30% better than for day 1 and 3, regardless of which day was taken for identification (see Fig. 26 in Appendix D as example). The Bode plots for models identified from day 2 also seemed to be better fitting than those identified from day 1. This can for instance be seen in the Bode diagram of the ARMAX model (Fig. 18) where the model identified from day 1 has a larger offset than that of day 2. A simple reason for this phenomenon could be the higher mean value of blood glucose level on the first day leading to difficulties in prediction for the next day. In taking the mean value of every day before identification there should however be no difference between day 1 and 2, but still for detrended data the prediction for day 2 has better results. Probably the reason might be in the data itself. In day 2 there is an extra carbohydrate intake between breakfast and lunch and one after dinner, leading to a more exciting input signal for carbohydrates. This could be one explanation for the better results for day 2, but there could as well be plenty of others. To investigate this more data from several days would be required.

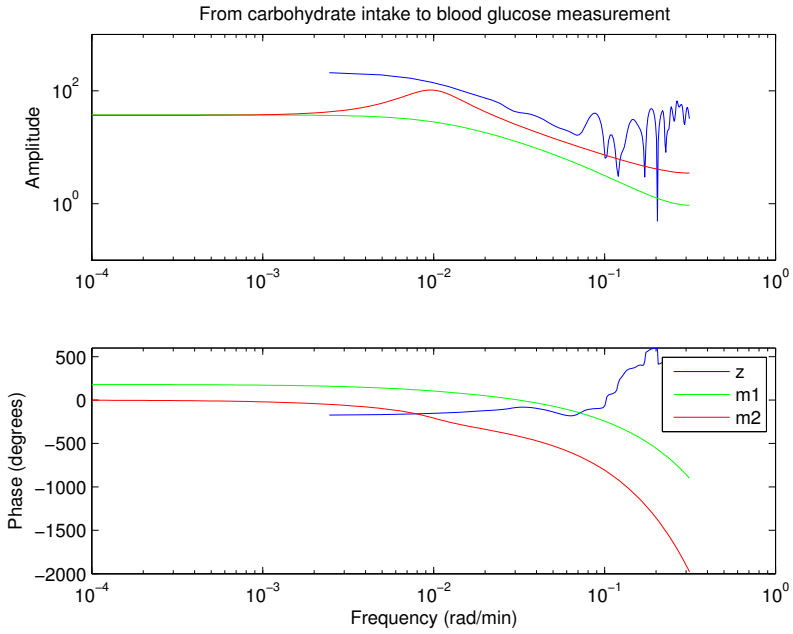


Figure 18: Bode diagram for input 1 with ARMAX model

The magnitude of the model's Bode plot is too low compared to the data spectrum. The difference between model 2's Bode plot and the data's Bode plot is 4dB and the difference between model 1's Bode plot and the system's is even 12dB (see Fig. 18). For ARX models, higher orders result in slightly better matching Bode plots but FIT, VAF, SDE and the prediction plots are not much better. Furthermore there were some pole-zero cancellations for higher order models. To achieve simpler models the smaller order was prefered.

The fact that the residuals don't have white noise properties is an indicator that there might be some effects not covered by the models. This could for instance be a too low model order, unaccounted for inputs or nonlinear behavior [21]. The residuals for input 1 are double as high as for input 2. This implies that input 1 probably is not represented appropriately in the model.

For increasing prediction horizons the quality of the prediction gets worse, as expected. The FIT takes on values of below 50% for prediction horizons greater than 3 or 6 steps and the SDE is more than 20 for larger prediction horizons. An example for FIT and SDE values can be seen in Fig. 27 in Ap-

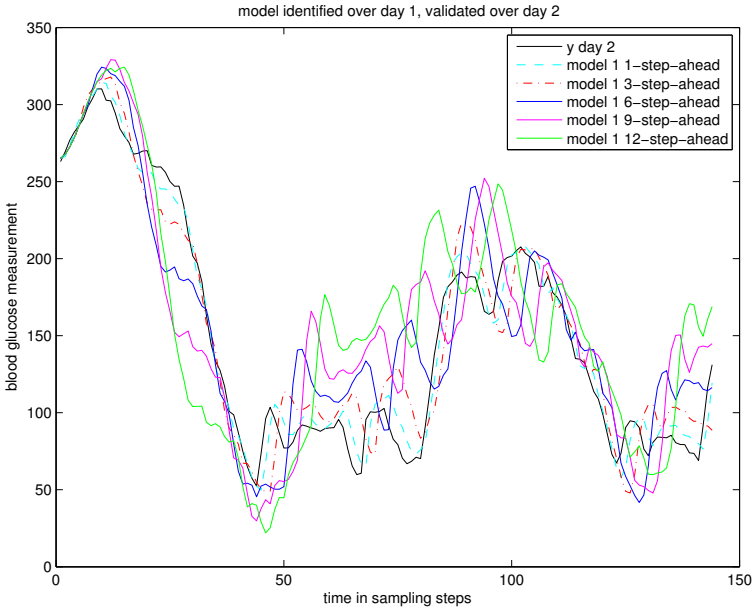


Figure 19: Prediction of day 2 with n4sid-model identified from day 2 after detrending the data for all investigated prediction horizons

pendix D for models identified from data of day 1 validated on day 2. Fig. 28 in Appendix D shows the FIT and SDE values that were obtained by taking the mean of the FIT and SDE values for models identified from day 1 and 2. In general the detrended models (dotted lines) have higher FIT and mostly lower SDE values and are thus of a bit better quality, except for MOESP models where the detrended data led to worse results than the normal data. The difference between detrended or not detrended data becomes stronger with increasing prediction horizon.
ARX and ARMAX models have the best FIT values and are quite close to each other, ARMAX models are a bit better for detrended data however. n4sid models for detrended data are of better quality for the SDE values. This shows that the quality indicators are not perfect and one should not trust a single indicator only. For judging the quality all indicators and the plotted output should be taken into consideration.
While the values for ARX and ARMAX are very close to each other and n4sid not being so far away, MOESP models have the lowest quality compared to the others. The mean FIT values for MOESP models are between 5% and 15% lower and the mean SDE values are between 5 and 25 higher than those of the other models.

An explanation for the better results with ARX and ARMAX models might be that there can be made more specifications for the order (n_a, n_{b1} and n_{b2}) and the most appropriate model can be picked by adjusting all these parameters for the order. Whereas for the state-space models only the order of the A-matrix can be changed.
Plotting the predicted and the measured output also shows that the quality for higher prediction horizons decreases (see Fig. 19). The larger the prediction horizon, the greater is the delay between prediction and measured output and the less can the prediction follow the high frequency changes. Although the VAF is decreasing for increasing prediction horizons this decrease is not as dramatically as for the other two indicators. This shows again that the values for VAF should not be trusted blindly.
If the delay is larger than the prediction horizon itself, it is hard for the patient to react fast enough to occurring hypoglycemia. Since larger prediction horizons not only have a bigger mismatch between measured and predicted output but also a larger delay (see Fig. 19), prediction horizons of more than 3 or 6 steps (30 or 60 min) should be avoided. On the other hand, longer prediction horizons would give enough time in case of an upcoming hypoglycemia to take precautions. Even though 30min is enough to prevent hypoglycemia by immediately ingesting carbohydrates a larger lead time would be much more convenient for the patient.

Regarding the logarithmic analysis it can be concluded that this experiment neither proved nor disproved a logarithmic behavior of the data. It showed at least that logarithmizing does not produce completely meaningless predictions.

4.7 Identification of models for different times of day

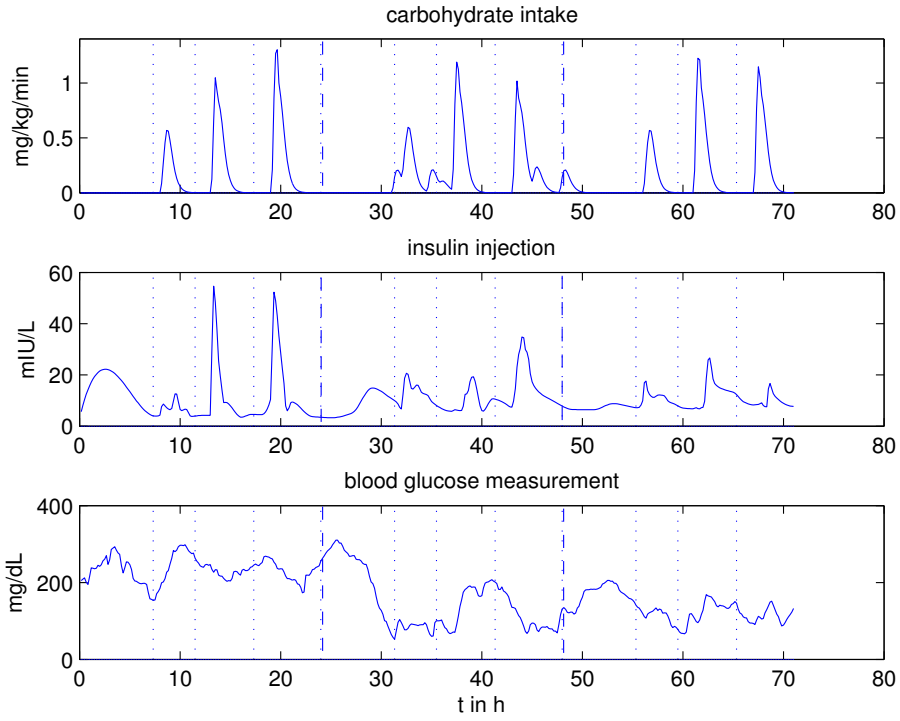


Figure 20: Division of the data into parts for night, breakfast, lunch and dinner

Since the blood glucose level is not only dependent on carbohydrate intake and insulin injection, but also on exercise, stress and other factors [26], the time of the day could play a role in the glucose metabolism. It might be that the model for breakfast differs from that for dinner because the body has rested during the night but is in motion during the day. To examine if it is profitable to identify models for different times of the day the data was divided into parts for night, breakfast, lunch and dinner for each of the three days (see Fig. 20).
The previous predictions had shown that there was not a remarkable difference between the predictions for detrended data or not detrended data. That is why it this experiment was only carried out with detrended data.

4.7.1 Results

The quality of the prediction turned out to be very poor, so that only ARMAX models for day 1 and 2 and n4sid models for day 1 were identified.
Models were validated with the corresponding time on the same or the following day. If a model e.g. was identified with breakfast data from day 1 then the predictions were compared to breakfast data on day 1 and 2.
There is no carbohydrate or insulin intake during the night, so the input signals are zero for the night parts. Therefore the input is not exciting enough to identify models for the night.
FIT, VAF and SDE values are displayed in Tables 12 - 14 in Appendix A and the model orders in Table 19 in Appendix B.

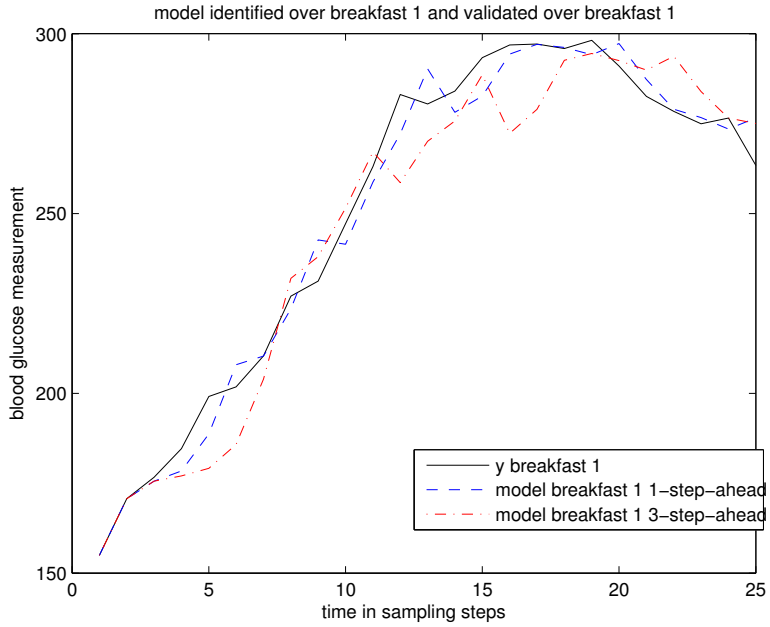
Fig. 21 shows the prediction for breakfast for day 1 and 2 for the ARX model identified with day 1. It can be seen that the prediction for day 1 (Fig. 21(a)) more or less follows the measured output. However the blood glucose does not undergo fast changes for this part and it is thus easier for the prediction to keep track with the measured output. The prediction for the second day (Fig. 21(b)) in contrast has more difficulties to match the measured output. The 1-step-ahead prediction can keep up with the measured data quite well with only a few overshoots and little delay. The 3-step-ahead prediction already has a large delay of 5 sampling steps (almost 1h delay) and predicts much higher values than they really are. Therewith the 3-step-ahead prediction is completely useless for the patient.

In Fig. 22 the prediction for breakfast of day 1 and 2 with the n4sid model is plotted. The prediction is as bad as for the ARX model.
The prediction for dinner time (Fig. 23) is slightly improved, however. The predicted output can keep track of the measured output much better, it's only the time-delay which is still too high.

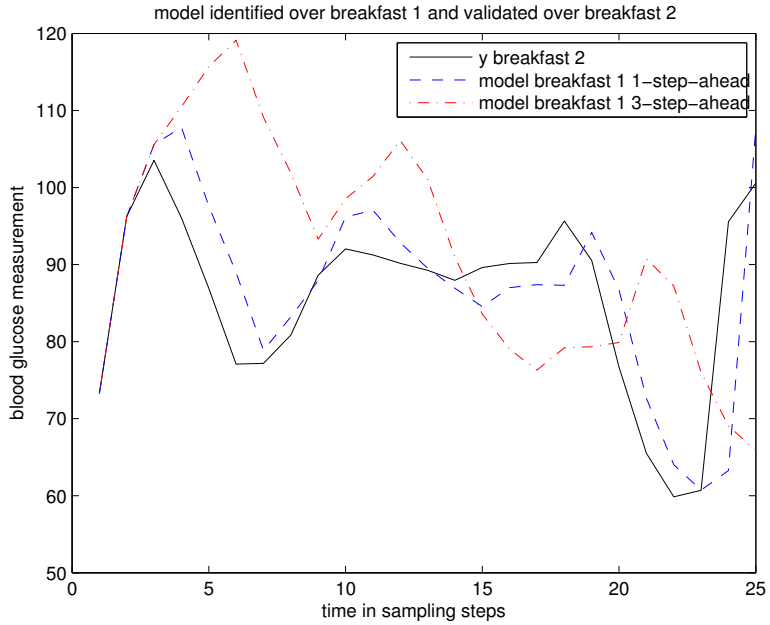
4.7.2 Discussion

The division of the data into small parts results in only a few sampling points for each part. For instance the part for breakfast contains only 24 values. It turned out that higher order models could not be identified because it was just not enough data. Furthermore the quality of the lower order models can be assumed to be very poor if there is so little information to do the identification on.

The reason for the predictions for dinner time being closer to the measured

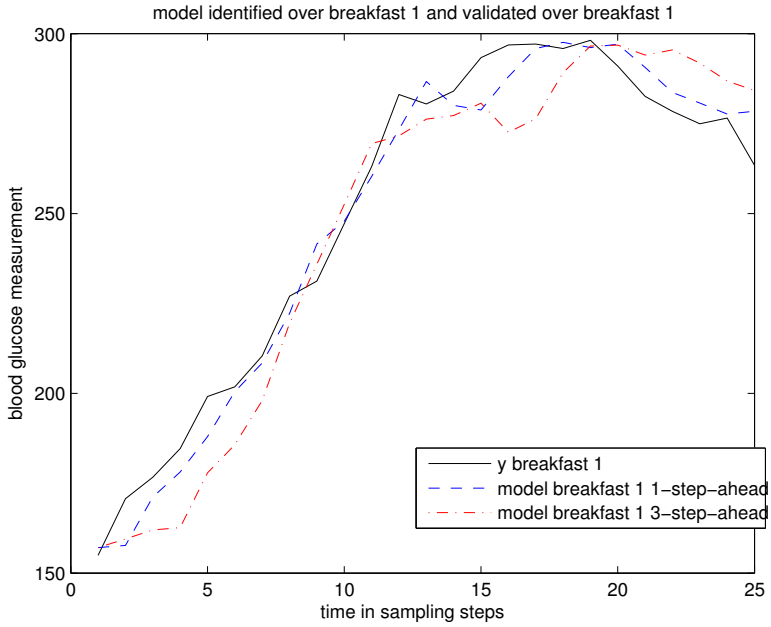


(a) Prediction for breakfast day 1

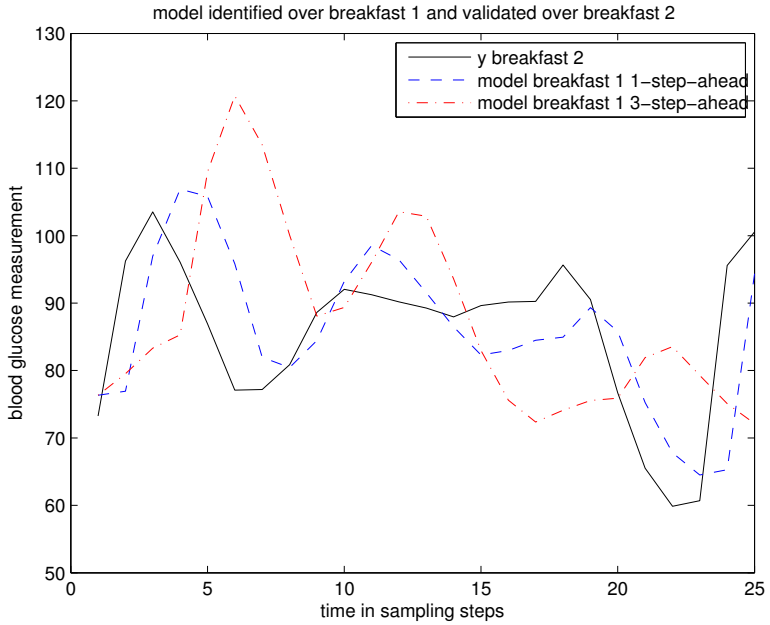


(b) Prediction for breakfast day 2

Figure 21: Prediction for breakfast with ARX model identified from breakfast day 1

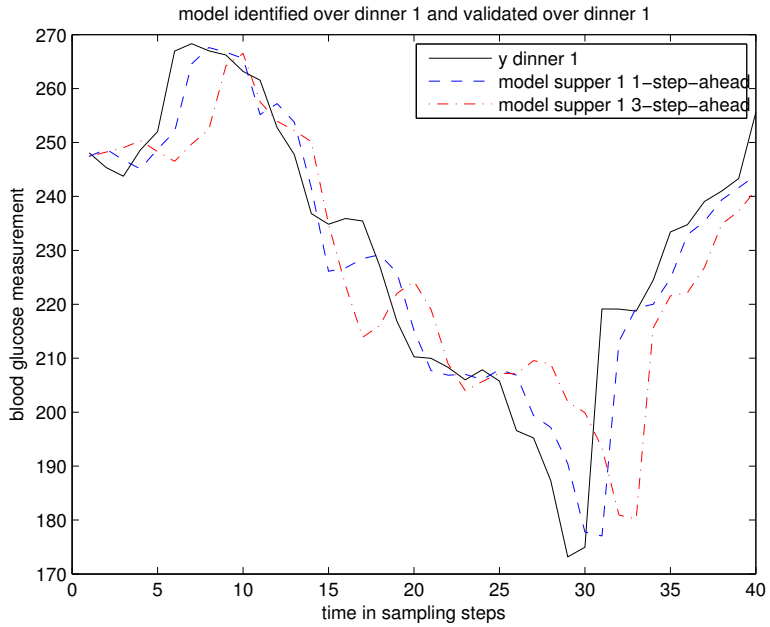


(a) Prediction for breakfast day 1

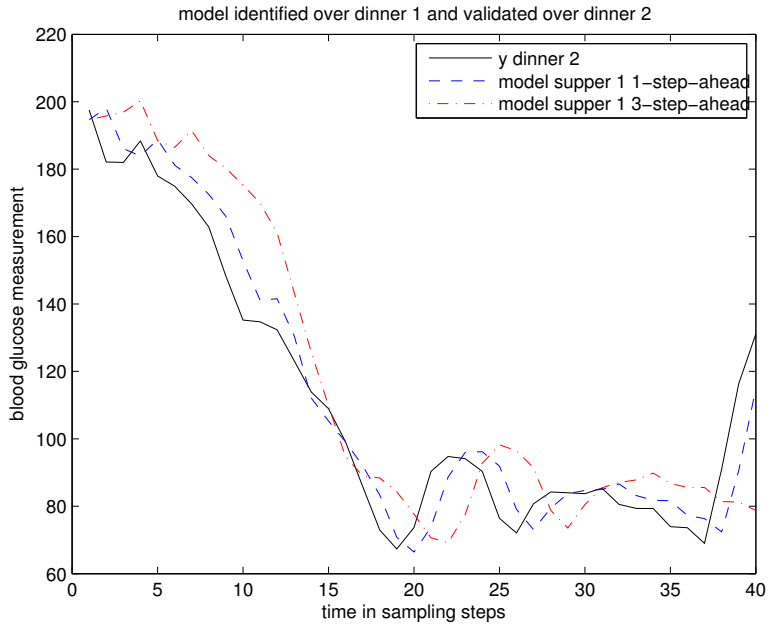


(b) Prediction for breakfast day 2

Figure 22: Prediction for breakfast with n4sid model identified from breakfast day 1



(a) Prediction for dinner day 1



(b) Prediction for dinner day 2

Figure 23: Prediction for dinner with n4sid model identified from dinner day 1

output than for breakfast time might be that dinner spreads a time span of 40 sampling points, which is nearly double as many as the breakfast time. However even though the predictions for dinner time look better than those for breakfast there still is a delay of about 3 steps for 3-step-ahead predictions. This means that if for instance hypoglycemia is predicted to occur in 30min it occurs already now and the patient does not have any time to take precautions for preventing it. Therefore even the predictions for dinner time, which look much better, are not more useful than those for breakfast.

Unfortunately there could not be gained any insights if the models depend on the time of the day or not. To do so more data samples are required. New measurements with a smaller sampling time would have to be taken.

4.8 Discussion

The prediction leads to much better results than the simulation. FIT and VAF have much more often values of more than 50% than simulation and the standard deviation of the error is much smaller than for simulation. This is because there is a comparison between predicted and actual output for the calculation of the next prediction value. Differences can thus much easier be corrected. Another advantage of this is that also unstable models can produce good predictions, unlike in simulation.

For simulation n4sid models are those with the best results followed by ARMAX and then ARX and MOESP. ARX and ARMAX are best for prediction, then n4sid and last are MOESP models. Since there is not a huge difference between the quality of ARX and ARMAX models the ARX models are to be prefered because of their simplicity. The method n4sid should be favoured in the identification of state-space-models because models identified with MOESP were not convincing.

Even though the models are quite reasonable for 1 and 3-step ahead predictions the quality could be further improved. There are several aspects that might have an influence on the models' quality, but also on the prediction itself.
The first aspect is the data. The appearance of glucose in plasma for input 1 was procured with a model. Input to the model was the amount of carbohydrates in the meal. Since it is hard to estimate the carbohydrate contents of a meal and since the model used for calculating the plasma glucose most probably does not exactly fit the reality, the data obtained for input 1 might deviate from the true plasma glucose. Furthermore the insulin concentration of input 2 was measured, interpolated and resampled. This might lead to falsified insulin behaviour. For all measurements taken (insulin concentration and monitoring of blood glucose level) there is no information about the sensors and their measuring error available.
Any nonconformity in the data first of all leads to erroneous models but since the input data is also used for simulation and prediction it has an effect of the simulation and prediction, too.
In addition there are other influences on the blood glucose concentration than carbohydrate intake and insulin concentration, which are not covered as inputs here. One such influence is exercise. The muscle cells take up glucose without the presence on insulin during heavy exercise. Since it was a hospital study the patient most probably did not go in for sports, so that these influences can be excluded for the identification of the models in this

thesis. For applying the models to the patient's daily life it would however be necessary to consider exercise as input. Yet this is hard to achieve because there had to be any means of quantifying the amount of exercise.
Besides, only linear models were examined, but it might be that the blood-glucose dynamics are nonlinear.

4.8.1 Comparison of polynomials and matrices of the different models

For the purpose of determining if and how much the different models deviate from each other the polynomials and matrices for the part Prediction were examined. They are displayed in appendix C.
The B-polynomials of the ARX models differ quite much from each other. This is mainly because the input delays were chosen so differently. If only those polynomials which have a similar input delay are compared, the factors for input 1 cover a range from -1 to -3 and are thus not so different. The factors for input 2 however differ from negative to positive values even for similar input delays. This might be an indicator for a not correctly chosen input delay for input 2. The A-polynomials for the ARX models of day 1 and 2 with and without detrending are quite akin. A mean A-polynomial could be $A(q) \approx 1 - 1.4q^{-1} + 0.4q^{-2}$.
The B-polynomials for the ARMAX models are again hard to compare. Those for input 2 vary strongly, but those for input 1 and day 2 are very close. The A-polynomials are similar except for that of day 1 without detrending. Taking the mean of the A-polynomials leads to $A(q) \approx 1 - 1.7q^{-1} + 0.7q^{-2}$, which is not so much different from the mean ARX-A-polynomial. The C-polynomials for day 1 are very different from each other, but those of day 2 are quite close with a mean polynomial of $C(q) \approx 1 - 0.3q^{-1} - 0.4q^{-2} - 0.2q^{-3}$.

Since it was hard to compare the matrices of the state-space models the eigenvalues were calculated for comparison. The n4sid models for day 1 and day 2 without detrending have different orders, so the number of eigenvalues is different, too. The eigenvalues of day 2 are however contained in a way in those of day 1:

$$0.68 \approx 0.78 \pm 0.5i$$
$$0.98 \approx 0.999$$
$$0.93 \approx 0.95 \pm 0.12i$$

The models for detrended data are comparable to those of not detrended data except that day 2 does only contain one complex pole, but an additional pole

at 0.45.
The MOESP models for not detrended data are of order 4 and the eigenvalues of day 1 and 2 are matching with those of the n4sid models.

$$1.01 \approx 1$$
$$0.87 \approx 0.8$$
$$0.956 \pm 0.19i \approx 0.964 \pm 0.19i$$

The model for day 1 with detrended data also has these eigenvalues. The model for day 2 does only have order 2, but the two poles are contained in those of the other models.

The analysis of the polynomials and matrices showed that n4sid and MOESP deliver similar poles for models of day 1 and 2 with and without detrending the data. Apart from a few exceptions ARX and ARMAX models also have alike poles. Since models for day 1 and day 2 are not really unlike it can be assumed that the models are not dependant on the days. If each day required a separate model it would be hard to use the models for prediction for other days than that used for identification. Detrending the data does also not seem to have a great influence on the models, which can be seen in the FIT, VAF and SDE values already. The fact that the models are not varying highly leads to the conclusion that they are quite reasonable and try to mimic the reality as good as possible.

4.9 Conclusion

- Simulation and prediction of the identified models have been performed. The simulations were rather poor, the simulated output does not follow the measured one at all. Prediction showed much better results.

- It seems as if predictions of more than 3 steps should however be avoided, since the time delay between predicted and measured output increases and the prediction quality becomes worse for increasing prediction horizons.

- Despite the fact that ARX models only consider white noise as input and are thus of less complexity than ARMAX models the quality of predictions with ARX models is nevertheless as good as those with ARMAX models. For the subspace-based models those predictions that were achieved with n4sid-models were better than those achieved with MOESP-models.

- Detrending the data before identification does not lead to completely different results than not detrending the data. In some cases the results were slightly better with detrended data, but it also occured that not detrended data produced better results. However in general it seemed more reasonable to detrend the data before identification.

- Identification of the normal data produced better results than taking the logarithm before identification and applying it back afterwards.

- The attempt to identify models for different times of the day has not been followed up after the first results showed that there are not enough data points to identify the models properly.

5 Controller

5.1 Minimum variance control

Given an ARMAX model for description of input-output system behaviour:

$$A(z^{-1})y_k = z^{-d}B(z^{-1})u_k + C(z^{-1})w_k \tag{11}$$

where

$$\begin{aligned} A(z^{-1}) &= 1 + a_1 z^{-1} + ... + a_{n_a} z^{-n_a} && (12)\\ B(z^{-1}) &= b_0 + b_1 z^{-1} + ... + b_{n_b} z^{-n_b+1} && (13)\\ C(z^{-1}) &= 1 + c_1 z^{-1} + ... + c_{n_c} z^{-n_c} && (14) \end{aligned}$$

and

$$\begin{aligned} y &\rightarrow \text{output}\\ u &\rightarrow \text{control action}\\ w &\rightarrow \text{noise}\\ d &\rightarrow \text{time delay} \end{aligned}$$

and solving the Diophantine equation

$$C(z^{-1}) = A(z^{-1})F(z^{-1}) + z^{-d}G(z^{-1}) \tag{15}$$

for the polynomials

$$\begin{aligned} F(z^{-1}) &= f_0 + f_1 z^{-1} + ... + f_{d-1} z^{-(d-1)} && (16)\\ G(z^{-1}) &= g_0 + g_1 z^{-1} + ... + g_{n_g} z^{-n_g} && (17) \end{aligned}$$

where n_g is the maximum of $(n_a - 1, n_c - 1)$

it is possible to compute a control law of the following type:

$$u_k = -\frac{G(z^{-1})}{B(z^{-1})F(z^{-1})}y_k \tag{18}$$

which minimizes the variance of the d-step-ahead output [20].

In this thesis an attempt to control the blood glucose in a type 1 diabetic patient was to use MV control to reduce the variance of the blood glucose concentration. Ideally the blood glucose should then fluctuate around a reference value within the limits that are considered as "normoglycemia" in healthy persons. The risk for hyper- and hypoglycemia should thus be reduced.

5.2 Structure of the control loop

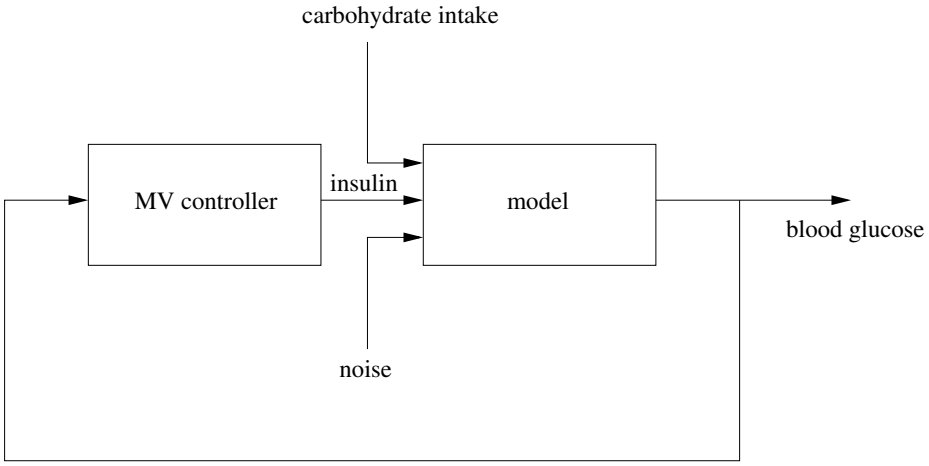


Figure 24: Control loop

The structure of the control loop can be seen in Fig. 24. Inputs to the model are the carbohydrate data, the calculated amount of insulin and a white noise sequence. The output is fed back to the controller. According to the control law in (18) the insulin sequence is calculated. Since it is not possible in reality to have negative insulin values, the insulin value was set to zero whenever a negative value was calculated by the controller.

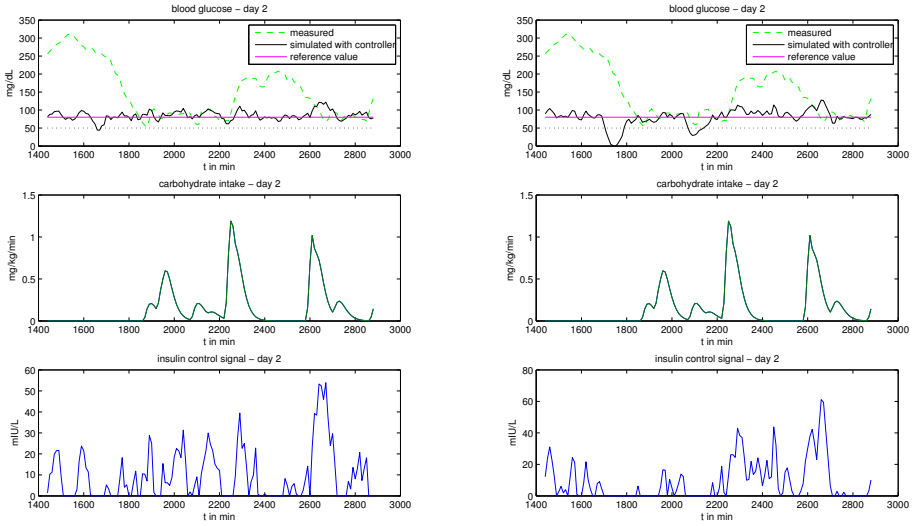
5.3 Results

To reduce the effort in solving the Diophantine equation an ARMAX model of order $n_a = n_{b1} = n_{b2} = n_c = n_{k1} = n_{k2} = 1$ was chosen as an example for the implementation of the controller. The model was identified with data from day 2. The reference value for the blood glucose concentration was set to 80 mg/dL, which is in the range of the "normal" blood glucose level in healthy persons [10]. Unless stated differently the initial value was assumed to match the reference value when the simulation was started.
The threshold for hypoglycemia was set at 50 mg/dL and is shown as dotted line in the plots. The blood glucose profile that was achieved with manual insulin injections in the hospital is drawn as dashed line in the plot for comparison.

The variance of the measured blood glucose for day 2 was calculated. It had a value of 5811 mg^2/dL^2. Minimum and maximum values of the blood

glucose concentration were 52.4 and 310.2 mg/dL. Day 3 had a variance of 1812 mg^2/dL^2 and minimum and maximum values of 0 and 205.6 mg/dL.

Fig. 30(a) in Appendix E shows the simulated blood glucose concentration and the input signals in the ideal case when the noise is disregarded. The blood glucose value is in the immediate vicinity of the reference value throughout the whole simulation and does only increase when carbohydrates are ingested. The maximum blood glucose concentration is 113 mg/dL and the variance is 58.4 mg^2/dL^2. The insulin profile basically follows the carbohydrate profile.
Since it is most likely to have noise acting on the process, all following simulations were performed with noise as additional input on the model. In Fig. 30(b) the simulation with the "ideal" insulin profile, which includes negative values, is shown. The blood glucose concentration is fluctuating around the reference value with a variance of 181 mg^2/dL^2 and minimal and maximal values of 46.7 and 118.2 mg/dL respectively.



(a) (b)

Figure 25: Simulations for day 3 with controlled insulin signal

Two examples for simulations with noise and only positive insulin values are shown in Fig. 25. Because of the noise the simulations are not repeatable and the blood glucose trajectory is varying highly in different simulations. Fig. 25(a) is an example for one of the best cases that could be achieved. The

blood glucose variance is 169.7 mg^2/dL^2. Minimum and maximum values are 43.8 and 121.9 mg/dL. The variance for the much worse example, which is shown in Fig. 25(b), is much higher with a value of 536.4 mg^2/dL^2. The minimum value is calculated to be -0.9 mg/dL, which of course is impossible in reality. The patient would have already died. The blood glucose exceeds the threshold of 50 mg/dL longer and more often than in the better case. The maximum value is with 127.5 still in the range of potential values that could occur in a healthy person [10].

In Fig. 30(c) and 30(d) the simulation with initial blood glucose values of 250 mg/dL and 0 mg/dL can be seen. In the case with the much higher blood glucose value compared to the reference, the blood glucose is corrected rapidly in the range of the reference value. The concentration is then similar to the one with correct initial values. The time until the blood glucose reaches the reference value is much longer for a too low initial value than with a too high one.

Since it was prohibited to have negative insulin values there is no means of increasing the blood glucose concentration with only an insulin controlled loop except when meals are taken. Therefore negative insulin values were scaled down with a factor to achieve the dimension of the carbohydrate unit and applied as positive values additional to the carbohydrate profile that was given with the meals. However the blood glucose concentration did not change in comparison to the one with only insulin control. An example can be seen in Fig. 31.

The simulation with the model identified from day 2 was also performed with data from day 3. The initial blood glucose value was chosen to match that of the measured data of day 3. Fig. 32(a) shows an example of one of the better simulations with a blood glucose variance of 306.6 mg^2/dL^2 and minimum and maximum values of 43.0 and 130.7 mg/dL. A rather poor simulation is plotted in Fig. 32(b). The variance is quite high with 1934.2 mg^2/dL^2, minimum and maximum values are -45.5 and 129.8 mg/dL.

5.4 Discussion

In the ideal case without noise the blood glucose variance could be reduced to 1% of the variance of the measured blood glucose that was achieved in manual insulin dose determination. For the noisy measurements the variance could be reduced between 3% and 10% of the measured blood glucose vari-

ance for day 2 and between 17% and 107% for day 3.
This shows that the variance can be diminished dramatically with the controller. However the blood glucose concentration highly depends on the noise and the variance flutuates a lot subject to the noise. This can be seen very well on day 3 where there is an improvement in the variance for the better case, but a change for the worse in the poor case. It seems crucial to reduce the noise as far as possible. The development of low noise sensors for blood glucose monitoring might be a step in that direction.
The MV control law is designed for applying both a positive and a negative control value, but in this case only positive insulin values are applicable. The controller does not have any knowledge of this contraint of the insulin values and can therefore not consider this when calculating the insulin profile. As a consequence the blood glucose variance might not be the minimal one.
Negative blood glucose values, that appear in some simulations, are of course not possible in reality. This happens because the controller cannot raise the blood glucose on its own. A real patient would already have taken carbohydrates or fallen into coma and died in the worst case.
While searching for an explanation for extreme low blood glucose values beyound the hypoglycemic threshold, two questions occured: Is this solely caused by noise or was the calculated insulin dose too high? The fact, that this situation does only appear when noise is added, could be an indicator for noise beeing the cause. Additionally, a restriction of insulin doses to a maximum value of 20 mIU/L did not result in less hypoglycemia than before, but in increased risk for hyperglycemia.

In the case of a too high initial value this is regulated fastly by setting the insulin dose to a high value until the blood glucose has reached the reference value. For too low initial values the controller does not have a chance to increase the blood glucose but to wait until the first meal is taken. Due to the noise it might happen that the blood glucose concentration is even falling further and cannot be raised by the controller alone. However the simulated scenario with an initial value of 0 mg/dL is rather exaggerated. The patient would most probably be dead already and even with an initial value between 20 and 50 mg/dL an immediate glucose intake can only be recommended.

The modification with altering the carbohydrate input did not lead to improved blood glucose concentration compared to only controlling the amount of insulin. Variance and number of overstepping the hypoglycemic threshold could not be reduced. Besides it is impractical for the patient having to ingest carbohydrates all day long whereas the insulin dose can easily be administered with an insulin pump 24h a day. It seems reasonable to operate

the controller with altering the insulin profile only but to ingest additional carbohydrates when hypoglycemia should occur.

The simulations for day 3 are in principal comparable to those of day 2. However the variance is higher for day 3. This reflects the fact that the predictions for day 2 also were of higher quality than those of the other days. To follow up this matter, data of more days would be required to do simulations on. Furthermore with additional data it could be examined if it is profitable to adjust the model's parameters adaptively every day as it is suggested by [9] and [15].

For the ideal case, when neglecting the noise, the insulin action is proportional to the ingested food. This is because the blood glucose does only increase during mealtimes, but is in accordance with the reference value the rest of the time. Therefore the blood glucose just has to be regulated by insulin when meals are occuring. For some of the other simulations with noise, e.g. Fig. 30(b) and Fig. 30(c), the insulin dose in times of meals is not proportional to the carbohydrate intake. The reason for this is that whenever the blood glucose is too low, the carbohydrates ingested with the meal are required to raise the blood glucose. A proportional insulin injection would only cause the blood glucose to fall even lower.

In spite of reducing the variance, the hypoglycemic threshold is passed more often with the controller than without. This is because the controller has no means of raising the blood glucose concentration by only having an influence on the insulin dosis. If the patient took some additional carbohydrates in the cases where the blood glucose is exceeding the hypoglycemic threshold there will most probably not be so long and intensive hypoglycemic phases as in some of the simulation plots. Therefore the quality of the controlled blood glucose concentration can most probably be increased in reality.
With the controller, hyperglycemia does not occur anymore. The controller can handle this by increasing the insulin dose. Hence, another way of increasing the controller's quality and decreasing the risk for hypoglycemia might be to raise the reference value to 90 or 100 mg/dL because there is still enough reserve before reaching hyperglycemia. Besides, a too high blood glucose value can easily be dealt with the controller, but not a too low value.

The calculated insulin profile is varying a lot during the day and does not only contain a few peaks. Therefore it is impracticable to try to achieve this insulin profile with single insulin injections. To be able to use a controller for the glucose control in a diabetic person, an insulin pump is inevitable.

5.5 Conclusion

- By introducing a controller, the blood glucose concentration's variance can be reduced. However the blood glucose is highly dependent on noise. Therefore the controller might lead to a very well regulated blood glucose profile, but it can as well be of poor quality.
- The controller can reduce the blood glucose by increasing the insulin dose, but has no means of raising the glucose level.
- Hyperglycemia can be avoided with the controller, whereas hypoglycemia seems to appear more often than by defining the insulin dose manually.
- Too high initial values can easily be regulated to the reference value. In the case of too low initial values the controller is powerless and has to wait for the next food ingestion.

6 Conclusion

Even with the utilized linear methods it was possible to make good predictions with the identified models for low prediction horizons.
The MV controller was able to reduce the variance of the blood glucose concentration and to eliminate hyperglycemia.

A challenge is that the amount of carbohydrates has to be numbered by the patient. On the one hand it is hard to estimate the content of carbohydrates in a meal and on the other hand especially snacks and alcohol intake are easy to forget and are then not represented in a meal diary.

Further research could be done on the following topics:

- Since the data that was used for this thesis originates from one patient only, an approach might be to collect data from different subjects and to identify models for each of these for determining if and how much the parameters deviate from patient to patient.
- The risk for hypoglycemia was increased with the controller. Modifications on the controller and a warning system for hypoglycemia are required before the controller can be tested on patients.
- Other control methods as PID control or Model Predictive control could be examined.
- It could be analyzed if better models and controllers can be achieved with nonlinear models and methods.
- By taking data for a longer period of time, e.g. more days, it could be studied if the models are varying between different days and if it is possible to apply the controller effectively to other days than that, that was used for identification. The effect of adapting the parameters at each day could also be investigated.
- If the data was collected with a smaller sampling time, the attempt to identify models for different times of the day could be resumed.
- To be able to implement a controller in a diabetic patient, other influences on the glucose level have to be taken into consideration. For instance the effect of excercise had to be quantified by some means and added as input to the model.

A Tables with identification quality criteria

x_{01} in Tables 1 and 2 indicates that the initial state x_0 was estimated with data from day 1, and from day 2 and 3 for x_{02} and x_{03} respectively.
n4sid 1 refers to the n4sid identification with estimation of x_0 with "dinit" from Verhaegen and n4sid 2 refers to the n4sid identification with estimation of x_0 with "findstates".

without detrending

		day 1	day 2		day 3	
model	day for. ident.		x_{01}	x_{02}	x_{02}	x_{03}
ARX	1	27.31	27.23	57.27		
	2			80.21	-200.25	54.32
ARMAX	1	11.37	22.57	35.70		
	2			80.77	-169.32	60.52
n4sid 1	1	62.77	14.38	77.70		
	2			94.10	-187.67	34.36
n4sid 2	1	60.65	23.83	77.19		
	2			94.57	-167.35	8.62
MOESP	1	83.85	-1000.00	29.79		
	2			91.40	-347.00	15.63

with detrending

		day 1	day 2		day 3	
model	day for. ident.		x_{01}	x_{02}	x_{02}	x_{03}
ARX	1	20.88	2.67	63.44		
	2			69.91	118.18	44.55
ARMAX	1	79.85	4.35	52.18		
	2			71.52	-125.24	42.13
n4sid 1	1	51.68	-12.29	21.17		
	2			93.39	-175.47	29.03
n4sid 2	1	59.56	-25.21	-58.92		
	2			91.16	-182.12	-182.12
MOESP	1	81.43	4.08	49.63		
	2			93.40	-1000.00	-23.97

Table 1: VAF values for "simulation"

without detrending

		day 1	day 2		day 3	
model	day for. ident.		x_{01}	x_{02}	x_{02}	x_{03}
ARX	1	27.48	64.81	49.66		
	2			33.80	58.81	54.32
ARMAX	1	30.35	66.85	60.92		
	2			33.31	55.70	21.33
n4sid 1	1	19.67	70.30	35.88		
	2			18.46	57.57	27.50
n4sid 2	1	20.22	66.31	36.28		
	2			17.70	55.50	32.45
MOESP	1	12.95	259.56	63.66		
	2			22.29	71.76	31.18

with detrending

		day 1	day 2		day 3	
model	day for. ident.		x_{01}	x_{02}	x_{02}	x_{03}
ARX	1	28.67	74.95	45.93		
	2			41.67	50.14	25.27
ARMAX	1	14.47	74.30	52.54		
	2			40.54	50.94	25.82
n4sid 1	1	22.41	80.50	67.45		
	2			19.53	56.33	28.59
n4sid 2	1	20.50	b 85.01	95.77		
	2			22.59	57.01	57.01
MOESP	1	13.89	74.40	53.92		
	2			19.52	65919.00	37.79

Table 2: standard deviation of simulation error

without detrending

	day	day 1					day 2					day 3				
		1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	78.81	54.29	26.09	5.79	-9.74	90.26	74.11	59.52	48.35	39.58					
	2						91.07	76.39	66.34	60.88	57.23	86.10	64.31	48.18	43.53	39.02
ARMAX	1	81.27	57.91	29.87	8.32	-7.21	88.47	72.45	58.11	47.17	38.46					
	2						91.38	78.75	71.86	70.31	70.53	84.86	57.26	36.71	29.83	27.33
n4sid	1	69.94	42.91	25.69	16.92	2.42	87.46	70.60	55.98	46.58	35.94					
	2						85.96	78.44	74.09	70.96	68.86	65.83	43.87	29.44	23.14	13.93
MOESP	1	66.08	46.47	34.75	25.14	15.56	79.54	61.97	40.38	23.20	12.86					
	2						86.24	77.44	72.77	69.70	67.54	63.17	32.25	2.71	-12.26	-24.92

with detrending

	day	day 1					day 2					day 3				
		1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	79.30	56.65	33.42	19.91	13.48	90.38	74.35	60.71	52.43	47.06					
	2						90.99	75.66	64.44	57.75	52.54	85.05	59.24	38.24	35.95	33.10
ARMAX	1	79.85	57.69	34.67	21.06	14.45	90.04	73.70	60.08	50.97	45.02					
	2						91.38	78.03	70.02	66.91	65.75	85.70	62.09	46.46	45.14	43.04
n4sid	1	72.35	54.15	41.55	30.54	23.44	86.35	70.25	53.21	40.24	30.40					
	2						86.50	79.07	74.64	70.90	68.89	69.60	48.45	31.58	23.50	16.22
MOESP	1	67.93	47.90	29.47	15.39	4.75	74.57	49.39	21.30	-4.74	-39.69					
	2						81.07	74.53	69.47	66.04	63.94	56.36	40.25	27.17	20.20	13.56

Table 3: FIT values for "prediction"

without detrending

	day	day 1					day 2					day 3				
		1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	95.51	79.11	45.39	11.26	-20.42	99.08	93.80	86.34	79.54	73.87					
	2						99.20	94.43	88.68	84.70	81.72	98.07	87.26	73.14	68.12	62.82
ARMAX	1	96.49	82.28	50.82	15.97	-14.84	98.68	92.63	83.86	75.51	67.98					
	2						99.26	95.49	92.08	91.19	91.32	97.72	81.90	60.58	51.82	48.55
n4sid	1	90.97	67.45	44.98	31.62	5.67	98.43	91.36	80.63	71.48	58.98					
	2						98.03	95.35	93.29	91.57	90.31	88.32	68.49	50.26	41.01	25.97
MOESP	1	88.51	71.45	57.90	45.24	30.51	95.89	85.81	65.27	42.69	26.73					
	2						98.11	94.91	92.59	90.83	89.48	86.52	54.46	6.63	-22.90	-48.84

with detrending

	day	day 1					day 2					day 3				
		1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	95.72	81.22	55.71	35.90	25.16	99.10	93.75	86.11	80.37	76.30					
	2						99.19	94.11	87.70	82.95	78.69	97.77	83.43	62.15	59.90	56.73
ARMAX	1	95.94	82.11	57.35	37.73	26.84	99.03	93.41	85.86	79.65	75.08					
	2						99.26	95.18	91.03	89.08	88.30	97.96	85.70	71.57	70.21	67.94
n4sid	1	92.36	78.99	65.94	52.08	41.79	98.15	91.26	78.70	65.69	53.94					
	2						98.19	95.64	93.59	91.58	90.39	91.10	74.61	56.41	47.46	39.17
MOESP	1	89.74	72.99	50.70	29.44	10.77	93.54	74.39	38.09	-9.61	-94.91					
	2						96.42	93.51	90.68	88.47	87.00	80.96	64.30	46.97	36.40	25.37

Table 4: VAF values for "prediction"

without detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	6.83	14.73	23.82	30.37	35.38	7.27	18.91	28.08	34.36	38.84					
	2						6.78	17.93	25.57	29.71	32.48	4.72	12.11	17.59	19.16	20.70
ARMAX	1	6.04	13.57	22.61	29.55	34.55	8.72	20.63	30.52	37.60	42.99					
	2						6.55	16.14	21.38	22.55	22.38	5.13	14.44	21.31	23.56	24.35
n4sid	1	9.69	18.39	23.91	26.66	31.31	9.53	22.34	33.44	40.57	48.66					
	2						10.66	16.38	19.68	22.06	23.65	11.60	19.05	23.94	26.07	29.20
MOESP	1	10.93	17.23	20.92	23.86	26.87	15.40	28.62	44.77	57.51	65.03					
	2						10.45	17.14	20.69	23.00	24.64	12.46	22.90	32.80	37.63	41.41

with detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	6.67	13.97	21.45	25.81	27.89	7.22	18.99	28.31	33.66	36.99					
	2						6.84	18.43	26.64	31.37	35.07	5.07	13.82	20.88	21.49	22.33
ARMAX	1	6.50	13.64	21.05	25.44	27.57	7.48	19.50	28.57	34.27	37.92					
	2						6.55	16.69	22.75	25.10	25.99	4.85	12.84	18.10	18.53	19.22
n4sid	1	8.91	14.78	18.81	22.32	24.60	10.34	22.45	35.06	44.50	51.56					
	2						10.23	15.87	19.23	22.05	23.55	10.13	17.10	22.41	24.60	26.47
MOESP	1	10.33	16.76	22.64	27.08	30.45	19.32	38.45	59.78	79.54	106.06					
	2						14.38	19.35	23.19	25.80	27.39	14.81	20.28	24.72	27.07	29.32

Table 5: standard deviation of prediction error

without detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	74.84	56.15	42.56	38.04	44.39	81.60	56.84	42.95	32.83	25.64					
	2						84.21	59.65	46.23	40.99	36.97	81.67	50.17	25.77	24.22	21.16
ARMAX	1	77.98	56.86	40.66	29.49	24.56	81.77	56.07	40.67	30.36	21.37					
	2						85.22	65.25	57.82	57.10	57.03	82.66	52.77	33.06	27.56	26.27
n4sid	1	52.59	29.33	5.89	-10.29	-16.84	64.94	45.65	29.76	17.74	6.69					
	2						78.32	62.98	47.84	38.99	31.17	61.02	32.88	11.24	1.96	-6.44
MOESP	1	66.23	35.26	-0.01	-30.27	-48.68	77.77	51.82	33.30	26.18	19.42					
	2						77.07	54.14	30.27	1.59	-30.50	62.05	15.95	-38.68	-79.15	-138.41

with detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	75.46	61.11	55.27	53.20	53.04	79.17	52.35	37.94	33.31	31.84					
	2						84.81	62.76	51.21	46.80	44.92	82.24	53.46	34.05	32.53	31.55
ARMAX	1	80.35	66.61	62.12	61.30	62.34	80.92	56.39	46.61	42.78	41.07					
	2						85.44	66.44	59.11	58.42	58.44	83.26	57.75	45.73	46.86	46.15
n4sid	1	62.36	43.43	28.14	19.89	17.08	65.38	44.96	31.09	22.90	18.89					
	2						75.04	60.82	51.91	48.20	46.63	66.96	47.42	37.70	36.06	34.61
MOESP	1	70.37	60.30	58.09	57.72	58.60	66.64	39.94	26.92	22.10	17.67					
	2						76.23	59.70	52.31	49.92	49.11	68.22	43.10	23.98	16.25	12.67

Table 6: FIT for prediction with logarithmizing input and output data

without detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	95.09	85.17	73.97	68.92	74.74	98.68	92.34	85.08	79.04	74.69					
	2						99.12	93.74	86.16	80.27	76.49	97.54	81.31	56.27	54.50	52.13
ARMAX	1	95.49	82.76	67.51	54.53	48.20	98.61	91.09	80.93	71.27	62.34					
	2						99.20	94.94	91.14	90.22	90.36	97.85	83.39	63.31	54.76	52.77
n4sid	1	78.52	50.92	9.25	-28.21	-47.00	92.44	80.73	61.34	39.24	16.00					
	2						97.36	91.73	80.89	71.74	64.12	87.75	63.84	36.09	19.45	2.75
MOESP	1	90.10	61.43	-2.54	-89.41	-169.22	97.89	89.47	75.19	64.60	56.80					
	2						97.15	86.03	50.37	-25.93	-165.99	88.44	42.69	-70.15	-219.91	-554.57

with detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	94.90	86.53	81.94	80.27	80.13	97.05	83.75	71.38	66.96	65.65					
	2						99.05	93.30	85.75	81.84	80.51	97.54	82.29	63.24	61.87	61.56
ARMAX	1	96.51	90.28	87.47	86.90	87.61	98.46	91.03	85.22	82.40	81.20					
	2						99.20	95.05	90.96	90.16	90.02	97.88	85.36	73.48	74.06	73.67
n4sid	1	86.63	68.92	47.94	34.70	29.89	90.30	74.99	56.64	44.91	39.36					
	2						96.27	90.06	82.88	79.28	78.27	91.28	77.71	68.39	67.02	65.35
MOESP	1	92.03	85.16	83.48	82.88	83.45	92.01	73.47	59.41	52.11	44.29					
	2						97.62	92.97	87.85	84.88	84.73	92.43	75.58	55.35	47.13	43.35

Table 7: VAF for prediction with logarithmizing input and output data

without detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	6.18	10.75	14.24	15.56	14.03	8.74	21.02	29.34	34.78	38.22					
	2						7.13	19.00	28.27	33.75	36.84	5.32	14.67	22.44	22.89	23.48
ARMAX	1	5.93	11.59	15.91	18.82	20.09	8.96	22.68	33.18	40.72	46.62					
	2						6.79	17.08	22.61	23.75	23.59	4.98	13.83	20.56	22.83	23.33
n4sid	1	12.93	19.55	26.58	31.60	33.84	20.88	33.35	47.24	59.22	69.63					
	2						12.34	21.85	33.21	40.38	45.51	11.88	20.41	27.13	30.46	33.47
MOESP	1	8.78	17.33	28.26	38.41	45.79	11.04	24.65	37.84	45.20	49.93					
	2						12.83	28.39	53.52	85.25	123.90	11.54	25.70	44.27	60.71	86.84

with detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	6.30	10.24	11.86	12.40	12.44	13.06	30.62	40.64	43.67	44.53					
	2						7.41	19.66	28.68	32.38	33.54	5.33	14.29	20.58	20.96	21.04
ARMAX	1	5.21	8.70	9.88	10.10	9.82	9.44	22.76	29.21	31.87	32.94					
	2						6.78	16.90	22.84	23.83	24.00	4.94	12.99	17.48	17.29	17.42
n4sid	1	10.21	15.56	20.14	22.55	23.37	23.66	37.99	50.03	56.39	59.16					
	2						14.67	23.95	31.44	34.58	35.42	10.02	16.02	19.08	19.49	19.98
MOESP	1	7.88	10.75	11.34	11.55	11.35	21.48	39.13	48.40	52.57	56.70					
	2						11.71	20.14	26.48	29.54	29.69	9.34	16.77	22.68	24.68	25.55

Table 8: SDE for prediction with logarithmizing input and output data

without detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	77.42	52.81	24.62	3.96	-12.44	83.69	58.79	43.58	33.80	25.13					
	2						84.17	59.41	43.64	35.97	30.28	81.73	49.67	23.06	19.05	12.40
ARMAX	1	76.71	51.11	33.00	24.69	19.50	83.94	60.58	46.75	39.97	36.06					
	2						84.89	63.52	53.45	49.91	48.20	81.48	46.79	21.11	11.68	7.09
n4sid	1	69.39	41.29	19.67	6.32	-10.89	80.17	54.75	40.58	33.14	22.17					
	2						80.49	64.44	58.02	52.33	47.55	62.25	25.42	7.26	-5.69	-21.79
MOESP	1	62.24	38.49	19.33	3.14	-9.84	70.64	48.24	29.03	17.43	14.06					
	2						77.25	64.30	62.96	63.59	60.39	59.42	30.23	8.88	2.33	-4.58

with detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	75.90	50.15	30.26	19.41	13.32	83.96	60.25	45.41	37.41	32.62					
	2						84.63	61.17	48.56	42.74	38.00	82.37	53.24	33.30	31.75	28.95
ARMAX	1	77.18	52.17	31.30	20.34	14.18	83.27	58.94	44.78	37.45	33.00					
	2						85.08	61.43	46.43	39.04	34.13	81.85	51.89	32.52	28.56	26.36
n4sid	1	70.55	51.44	40.75	35.73	32.19	78.21	56.71	41.55	32.10	26.27					
	2						79.81	67.53	65.04	63.99	63.62	59.43	29.77	17.43	11.46	6.74
MOESP	1	60.33	39.17	27.97	24.78	24.90	66.93	42.85	17.98	2.44	-2.22					
	2						71.30	63.56	59.75	56.92	54.30	49.22	33.20	20.11	12.50	4.93

Table 9: FIT for prediction with logarithmizing output data only

without detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	95.42	79.61	46.02	9.08	-25.38	99.04	93.21	84.60	76.31	68.64					
	2						99.09	93.43	83.76	74.69	68.13	97.53	80.98	53.52	47.39	38.20
ARMAX	1	94.92	78.35	58.64	46.42	38.40	98.89	92.04	81.74	74.84	71.12					
	2						99.14	94.27	89.13	86.64	85.68	97.37	77.06	46.13	30.45	23.18
n4sid	1	91.02	67.74	42.99	21.50	-12.62	98.51	92.02	82.84	74.59	64.95					
	2						97.71	91.84	86.98	82.45	79.44	86.95	48.43	19.86	-7.15	-43.51
MOESP	1	86.99	66.89	44.47	19.75	0.50	95.29	83.67	57.63	35.87	39.59					
	2						97.73	94.16	92.55	92.75	91.06	85.91	54.85	18.04	3.59	-22.18

with detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	94.44	75.55	50.96	32.96	22.10	98.94	92.49	83.38	77.25	73.84					
	2						99.13	93.43	85.37	79.83	76.05	97.78	83.94	66.36	66.16	64.14
ARMAX	1	94.84	77.14	52.47	34.99	24.47	98.82	91.95	82.75	76.88	73.59					
	2						99.08	92.73	83.58	77.11	72.77	97.60	83.13	66.40	62.28	60.27
n4sid	1	91.79	78.31	68.05	60.78	55.74	97.55	89.22	76.00	66.00	61.01					
	2						97.60	93.31	90.98	89.89	89.80	84.74	52.59	33.43	21.69	11.76
MOESP	1	84.84	65.42	52.94	48.97	50.38	93.46	76.98	31.55	-10.16	-3.07					
	2						95.92	93.17	90.75	89.28	87.91	80.14	65.29	49.57	35.51	13.27

Table 10: VAF for prediction with logarithmizing output data only

without detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	6.90	14.56	23.69	30.74	36.10	7.44	19.80	29.81	36.97	42.54					
	2						7.25	19.48	30.62	38.22	42.89	5.33	14.80	23.14	24.62	26.68
ARMAX	1	7.27	15.00	20.73	23.60	25.30	7.99	21.43	32.46	38.11	40.83					
	2						7.05	18.18	25.05	27.77	28.75	5.50	16.26	24.91	28.31	29.75
n4sid	1	9.66	18.31	24.34	28.56	34.21	9.27	21.47	31.47	38.29	44.98					
	2						11.49	21.70	27.41	31.82	34.45	12.26	24.37	30.38	35.13	40.66
MOESP	1	11.63	18.55	24.02	28.88	32.16	16.48	30.70	49.45	60.84	59.05					
	2						11.45	18.36	20.74	20.46	22.72	12.74	22.81	30.73	33.33	37.52

with detrending

		day 1					day 2					day 3				
	day	1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	7.60	15.94	22.58	26.40	28.45	7.81	20.82	30.97	36.23	38.86					
	2						7.08	19.47	29.06	34.12	37.18	5.06	13.60	19.69	19.75	20.32
ARMAX	1	7.32	15.41	22.23	25.99	28.02	8.24	21.55	31.56	36.53	39.04					
	2						7.30	20.49	30.79	36.35	39.64	5.26	13.94	19.68	20.85	21.39
n4sid	1	9.23	15.02	18.22	20.19	21.45	11.88	24.94	37.22	44.30	47.44					
	2						11.78	19.65	22.81	24.15	24.26	13.26	23.37	27.69	30.04	31.88
MOESP	1	12.55	18.96	22.12	23.03	22.71	19.43	36.45	62.85	79.74	77.13					
	2						15.35	19.85	23.11	24.87	26.42	15.13	20.00	24.10	27.26	31.61

Table 11: SDE for prediction with logarithmizing output data only

with detrending

			day 1					day 2					day 3				
	day		1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	breakfast	85.95	75.35	62.21	56.49	51.90	24.51	-67.37	-133.54	-203	-263.89					
		lunch	60.05	30.56	22.34	10.63	3.34	82.54	50.84	16.07	-3.22	-10.00					
		dinner	65.46	39.95	21.25	10.45	9.94	77.13	50.46	36.49	18.01	-3.14					
	2	breakfast						38.96	9.82	33.17	38.33	19.23	62.31	15.95	-11.17	-27.74	-43.80
		lunch						90.76	76.23	66.14	58.46	52.39	76.58	31.94	-16.79	-38.15	-48.75
		dinner						82.14	52.40	37.79	28.32	19.86	62.58	-15.28	-109.76	-149.42	-141.97
n4sid	1	breakfast	83.79	70.27	55.40	51.70	46.56	6.33	-66.30	-70.72	-98.84	-112.38					
		lunch	62.16	36.92	19.78	1.29	-12.94	70.65	29.78	-14.87	-41.48	-54.16					
		dinner	63.59	40.27	26.12	19.75	25.39	75.39	51.14	34.99	15.30	-2.59					

Table 12: FIT for prediction after identifying different times of the day

with detrending

			day 1					day 2					day 3				
	day		1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	breakfast	98.04	94.47	88.88	90.44	93.56	44.27	-154.89	-284.66	-497.40	-619.52					
		lunch	84.11	52.25	40.36	20.87	7.21	97.13	77.96	38.31	10.68	4.57					
		dinner	88.09	63.98	38.02	19.82	20.46	94.80	76.19	65.85	48.59	22.75					
	2	breakfast						62.77	19.11	56.56	62.77	36.44	88.40	48.61	16.07	-11.80	-47.61
		lunch						99.16	94.62	90.01	86.13	80.97	94.72	57.72	-18.59	-55.71	-68.18
		dinner						96.85	78.34	70.69	69.30	66.38	86.06	-31.32	-330.58	-495.23	-453.53
n4sid	1	breakfast	97.42	91.83	83.98	87.98	91.59	12.28	-174.10	-175.88	-269.42	-288.21					
		lunch	85.92	61.50	38.75	7.78	-19.18	92.07	55.73	-13.60	-64.50	-80.59					
		dinner	87.05	65.89	50.01	42.84	50.32	94.19	78.69	70.79	56.85	42.40					

Table 13: VAF for prediction after identifying different times of the day

with detrending

			day 1					day 2					day 3				
	day		1	3	6	9	12	1	3	6	9	12	1	3	6	9	12
ARX	1	breakfast	6.48	10.89	15.44	14.32	11.76	8.70	18.60	22.85	28.48	31.25					
		lunch	5.75	9.97	11.14	12.83	13.90	8.71	24.14	40.40	48.60	50.24					
		dinner	8.76	15.23	19.98	22.72	22.63	9.18	19.63	23.51	28.84	35.36					
	2	breakfast						7.11	10.48	7.68	7.11	9.29	6.07	12.78	16.33	18.85	21.66
		lunch						4.72	11.93	16.26	19.15	22.44	6.98	19.74	33.06	37.88	39.37
		dinner						7.14	18.72	21.78	22.29	23.32	6.39	19.60	35.50	41.74	40.25
n4sid	1	breakfast	7.44	13.24	18.54	16.06	13.43	10.91	19.29	19.35	22.39	22.96					
		lunch	5.41	8.95	11.29	13.86	15.75	14.48	34.22	54.81	65.96	69.11					
		dinner	9.13	14.82	17.94	19.19	17.89	9.70	18.57	21.74	26.43	30.53					

Table 14: SDE for prediction after identifying different times of the day

B Utilized model orders for identification

without detrending

model	day	n_a	n_{b1}	n_{b2}	n_c	n_{k1}	n_{k2}	model	day	order
ARX	1	2	1	1		16	30	n4sid (dinit)	1	5
	2	2	2	2		18	32		2	3
ARMAX	1	2	1	1	4	16	28	n4sid (findstate)	1	5
	2	2	1	1	2	5	34		2	4
								MOESP	1	4
									2	2

with detrending

model	day	n_a	n_{b1}	n_{b2}	n_c	n_{k1}	n_{k2}	model	day	order
ARX	1	2	1	1		15	26	n4sid (dinit)	1	5
	2	3	1	1		13	17		2	5
ARMAX	1	2	1	1	3	16	37	n4sid (findstate)	1	5
	2	2	2	1	2	13	20		2	5
								MOESP	1	3
									2	2

Table 15: Orders selected for simulation

without detrending

model	day	n_a	n_{b1}	n_{b2}	n_c	n_{k1}	n_{k2}	model	day	order
ARX	1	2	1	1		16	28	n4sid	1	5
	2	3	1	1		9	34		2	3
ARMAX	1	2	2	1	4	16	33	MOESP	1	4
	2	2	2	1	3	11	37		2	4

with detrending

model	day	n_a	n_{b1}	n_{b2}	n_c	n_{k1}	n_{k2}	model	day	order
ARX	1	2	1	1		5	36	n4sid	1	5
	2	2	1	1		8	38		2	5
ARMAX	1	2	1	2	4	6	34	MOESP	1	5
	2	2	2	1	5	10	34		2	2

Table 16: Orders selected for prediction

without detrending

model	day	n_a	n_{b1}	n_{b2}	n_c	n_{k1}	n_{k2}	model	day	order
ARX	1	3	1	1		11	28	n4sid	1	2
	2	2	1	1		16	27		2	4
ARMAX	1	2	2	1	3	20	20	MOESP	1	4
	2	2	2	1	3	11	33		2	3

with detrending

model	day	n_a	n_{b1}	n_{b2}	n_c	n_{k1}	n_{k2}	model	day	order
ARX	1	2	1	1		20	20	n4sid	1	5
	2	2	1	1		25	20		2	3
ARMAX	1	2	2	1	3	20	32	MOESP	1	4
	2	2	2	1	5	20	32		2	2

Table 17: Orders selected for prediction with logarithmized inputs and output

without detrending

model	day	n_a	n_{b1}	n_{b2}	n_c	n_{k1}	n_{k2}	model	day	order
ARX	1	3	1	1		11	28	n4sid	1	2
	2	2	1	1		16	27		2	4
ARMAX	1	2	2	1	3	20	20	MOESP	1	4
	2	2	2	1	3	11	33		2	4

with detrending

model	day	n_a	n_{b1}	n_{b2}	n_c	n_{k1}	n_{k2}	model	day	order
ARX	1	2	1	1		20	20	n4sid	1	5
	2	2	1	1		25	20		2	3
ARMAX	1	2	2	1	3	20	32	MOESP	1	4
	2	2	2	1	5	20	32		2	3

Table 18: Orders selected for prediction with logarithmized output

with detrending

model	day		n_a	n_{b1}	n_{b2}	n_{k1}	n_{k2}	model	day		order
ARX	1	breakfast	2	1	1	0	1	n4sid	1	breakfast	1
		lunch	2	1	1	0	0			lunch	1
		dinner	1	1	1	0	0			dinner	1
	2	breakfast	3	2	1	0	0				
		lunch	2	5	2	0	0				
		dinner	2	1	1	0	0				

Table 19: Orders selected for prediction after identification of different times of the day

C Identified polynomials and matrices for prediction

For the simulation experiment the polynomials and matrices of the different models are displayed here. m1 stands for models identified from day 1 and m2 from day 2, respectively. For the state-space-models only the A-matrix and the eigenvalues are shown.

ARX
without detrending:

$$\begin{aligned} m1: A(q) &= 1 - 1.358q^{-1} + 0.372q^{-2} \\ B_1(q) &= 4.94q^{-16} \\ B_2(q) &= 0.2049q^{-28} \\ m2: A(q) &= 1 - 1.631q^{-1} + 0.8141q^{-2} - 0.1555q^{-3} \\ B_1(q) &= -3.036q^{-9} \\ B_2(q) &= 0.3756q^{-34} \end{aligned}$$

with detrending:

$$\begin{aligned} m1: A(q) &= 1 - 1.341q^{-1} + 0.3884q^{-2} \\ B_1(q) &= -1.904q^{-5} \\ B_2(q) &= 0.03228q^{-36} \\ m2: A(q) &= 1 - 1.57q^{-1} + 0.5996q^{-2} \\ B_1(q) &= -0.9534q^{-8} \\ B_2(q) &= -0.01244q^{-38} \end{aligned}$$

ARMAX
without detrending:

$$
\begin{aligned}
m1 : A(q) &= 1 - 0.2007q^{-1} - 0.7812q^{-2} \\
B_1(q) &= -27.66q^{-16} + 34.96q^{-17} \\
B_2(q) &= 0.3247q^{-33} \\
C(q) &= 1 + 1.284q^{-1} + 0.275q^{-2} + 0.1777q^{-3} + 0.2771q^{-4} \\
m2 : A(q) &= 1 - 1.884q^{-1} + 0.8948q^{-2} \\
B_1(q) &= -6.663q^{-11} + 6.385q^{-12} \\
B_2(q) &= 0.1358q^{-37} \\
C(q) &= 1 - 0.343q^{-1} - 0.4313q^{-2} - 0.1992q^{-3}
\end{aligned}
$$

with detrending:

$$
\begin{aligned}
m1 : A(q) &= 1 - 1.282q^{-1} + 0.348q^{-2} \\
B_1(q) &= -2.464q^{-6} \\
B_2(q) &= 0.01755q^{-34} + 0.01642q^{-35} \\
C(q) &= 1 + 0.0506q^{-1} - 0.05201q^{-2} + 0.1848q^{-3} + 0.1418q^{-4} \\
m2 : A(q) &= 1 - 1.917q^{-1} + 0.9265q^{-2} \\
B_1(q) &= -6.517q^{-10} + 6.867q^{-11} \\
B_2(q) &= -0.02166q^{-34} \\
C(q) &= 1 - 0.3027q^{-1} - 0.3866q^{-2} - 0.1632q^{-3} - 0.003037q^{-4} - 0.1152q^{-5}
\end{aligned}
$$

n4sid

without detrending:

m1: A =

	x1	x2	x3	x4	x5
x1	0.97892	-0.038865	0.0077732	-0.020216	0.049224
x2	-0.08155	0.99777	-0.10111	-0.20103	-0.16882
x3	-0.042255	-0.055858	0.85675	-0.18723	0.35479
x4	-0.044346	0.28957	-0.011871	0.79259	0.5214
x5	-0.036226	0.085415	-0.24889	-0.26195	0.84069

$eig(A) = \{0.7843 \pm 0.5273i, 0.9991, 0.9495 \pm 0.1185i\}$

m2: A =

	x1	x2	x3
x1	1.0007	0.036976	-0.067476
x2	-0.019577	0.80441	0.103
x3	0.04423	0.15343	0.7917

$eig(A) = \{0.6828, 0.9845, 0.9295\}$

with detrending:

m1: A =

	x1	x2	x3	x4	x5
x1	0.95645	-0.1098	0.11809	0.014875	-0.010794
x2	0.1716	0.94963	-0.27526	-0.11587	-0.015236
x3	-0.008739	0.071186	0.86356	-0.48217	0.31533
x4	-0.059194	0.21313	0.30017	0.78549	0.2216
x5	0.10605	0.14964	-0.25681	-0.0089214	0.83135

$eig(A) = \{0.7692 \pm 0.5435i, 0.9852 \pm 0.1140i, 0.8776\}$

m2: A =

	x1	x2	x3	x4	x5
x1	0.91536	-0.060285	-0.061944	0.10353	-0.09881
x2	-0.060813	1.0526	-0.18746	0.048826	-0.03125
x3	-0.26525	0.35126	0.59699	0.17414	-0.25403
x4	-0.052271	0.29854	-0.25738	0.9032	0.20108
x5	-0.05445	-0.13029	0.13037	-0.42713	0.3784

$eig(A) = \{0.4504, 0.7877 \pm 0.3335i, 0.9235, 0.8972\}$

MOESP

without detrending:

m1: A =

	x1	x2	x3	x4
x1	1.0254	-0.14544	-0.063307	0.088451
x2	0.0029551	0.98245	0.3736	-0.048946
x3	0.0034266	-0.096358	0.94281	-0.11816
x4	-0.0039834	-0.008023	0.029959	0.84123

$eig(A) = \{1.0126, 0.8665, 0.9564 \pm 0.1906i\}$

m2: A =

	x1	x2	x3	x4
x1	1.0148	-0.15503	-0.067336	0.057644
x2	0.013548	0.96089	0.37062	0.077715
x3	-0.0026518	-0.065356	0.99643	-0.23293
x4	-0.0037382	0.0091471	0.078493	0.76338

$eig(A) = \{0.7990, 1.0086, 0.9639 \pm 0.1913i\}$

with detrending:

m1: A =

	x1	x2	x3	x4	x5
x1	0.94672	-0.14594	-0.19888	0.03148	-0.1537
x2	0.12258	1.0248	-0.2676	-0.030184	-0.18778
x3	0.020241	0.0049067	1.0085	0.17072	0.077289
x4	0.0022759	0.014042	-0.1007	0.81523	0.59839
x5	0.0099654	-0.030547	-0.010914	-0.5279	0.78855

$eig(A) = \{0.7996 \pm 0.5770i, 0.9630 \pm 0.1499i, 1.0586\}$

m2: A =

	x1	x2
x1	1.0111	-0.1639
x2	0.01777	0.95163

$eig(A) = \{0.9814 \pm 0.0450i\}$

D FIT and SDE plots for prediction

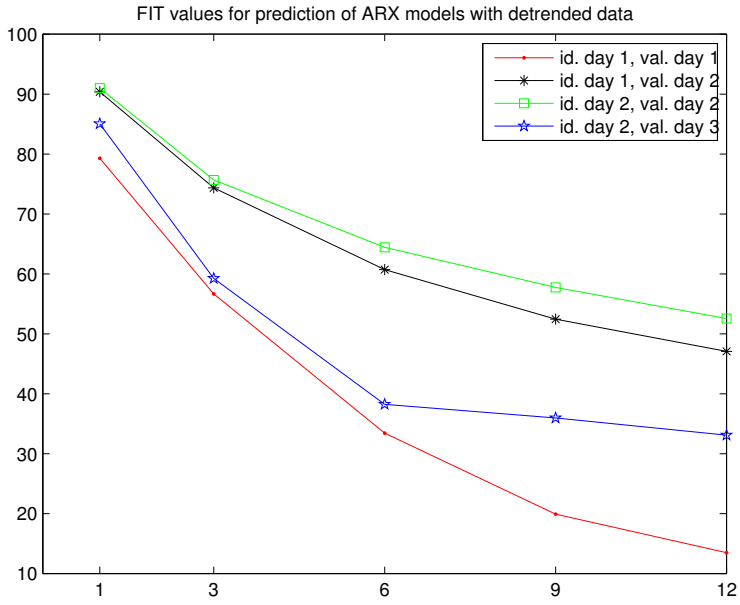
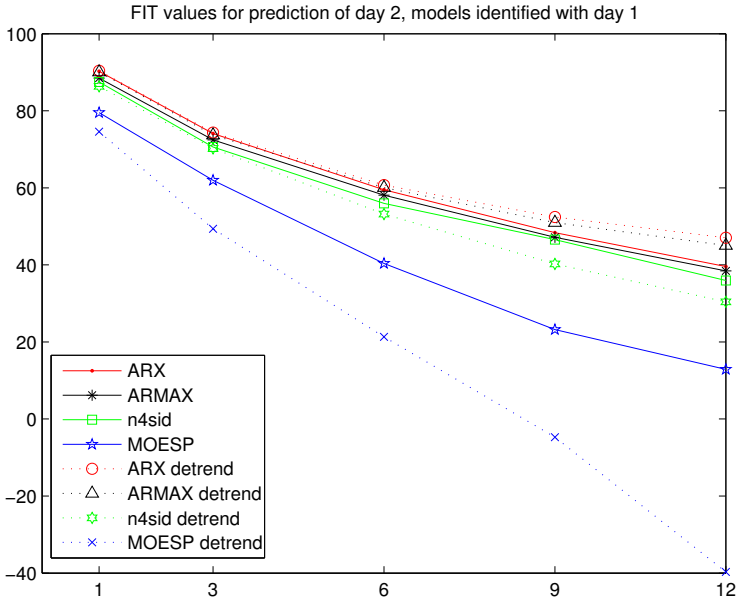
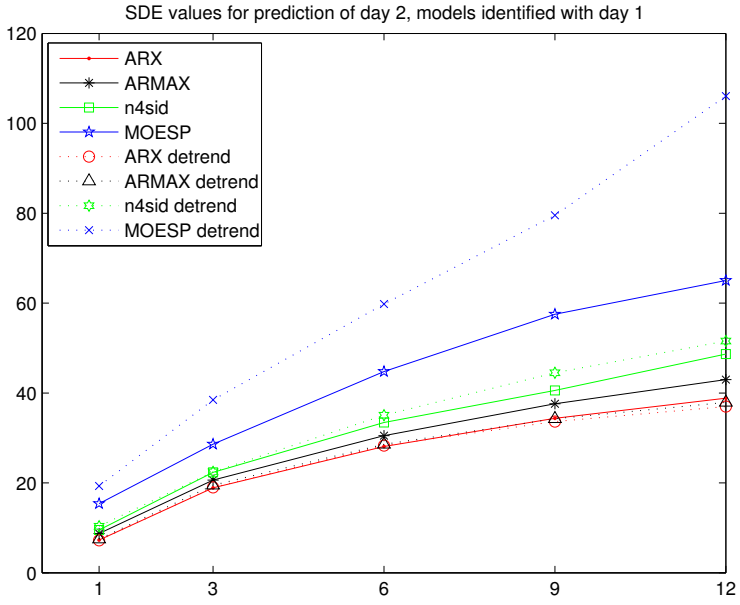


Figure 26: Prediction of day 1, 2 and 3 with ARX model identified from day 1 and 2 after detrending the data

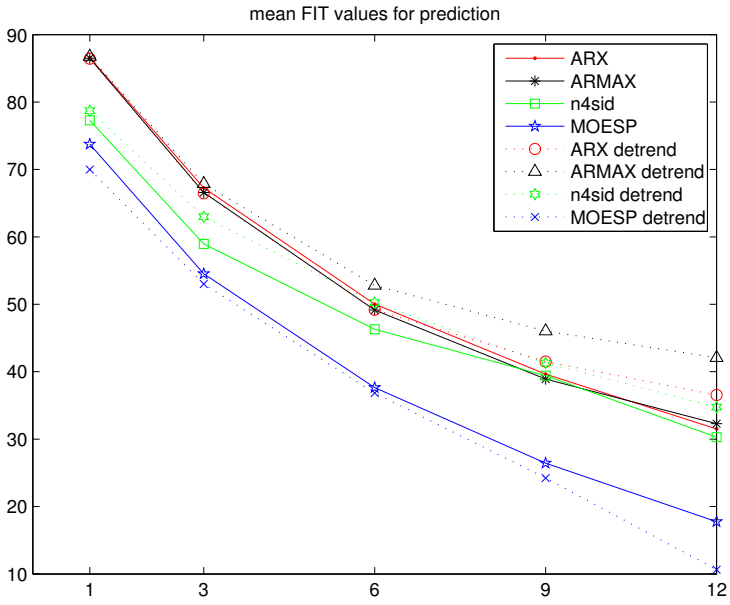


(a) FIT values

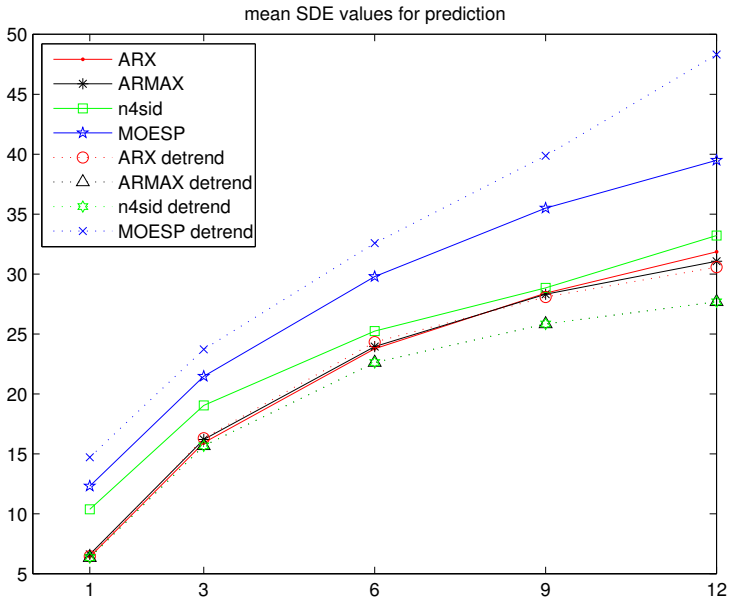


(b) SDE values

Figure 27: FIT and SDE values for all models with increasing prediction horizons, models identified with data from day 1 and validated on day 2

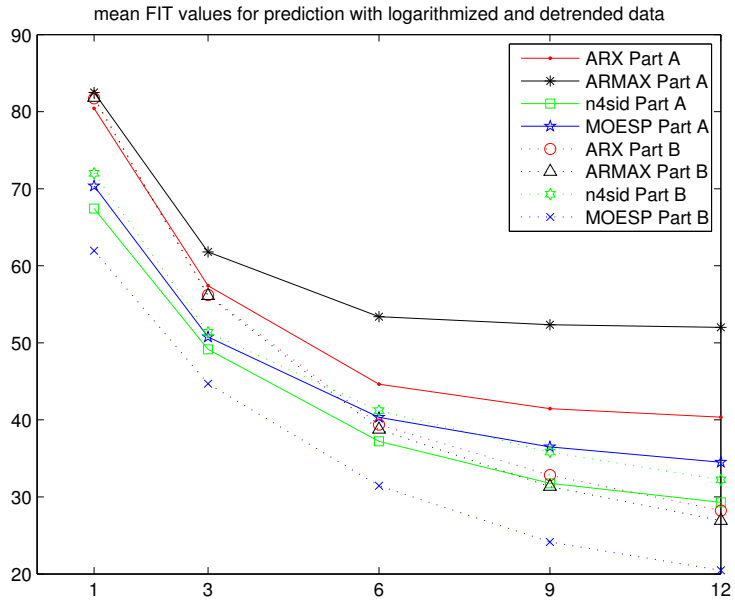


(a) mean FIT values

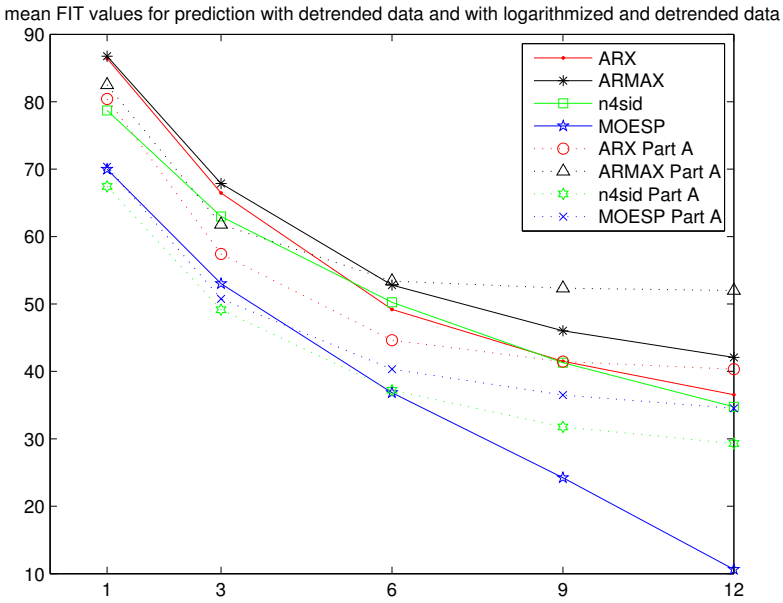


(b) mean SDE values

Figure 28: mean FIT and SDE values for all models with increasing prediction horizons



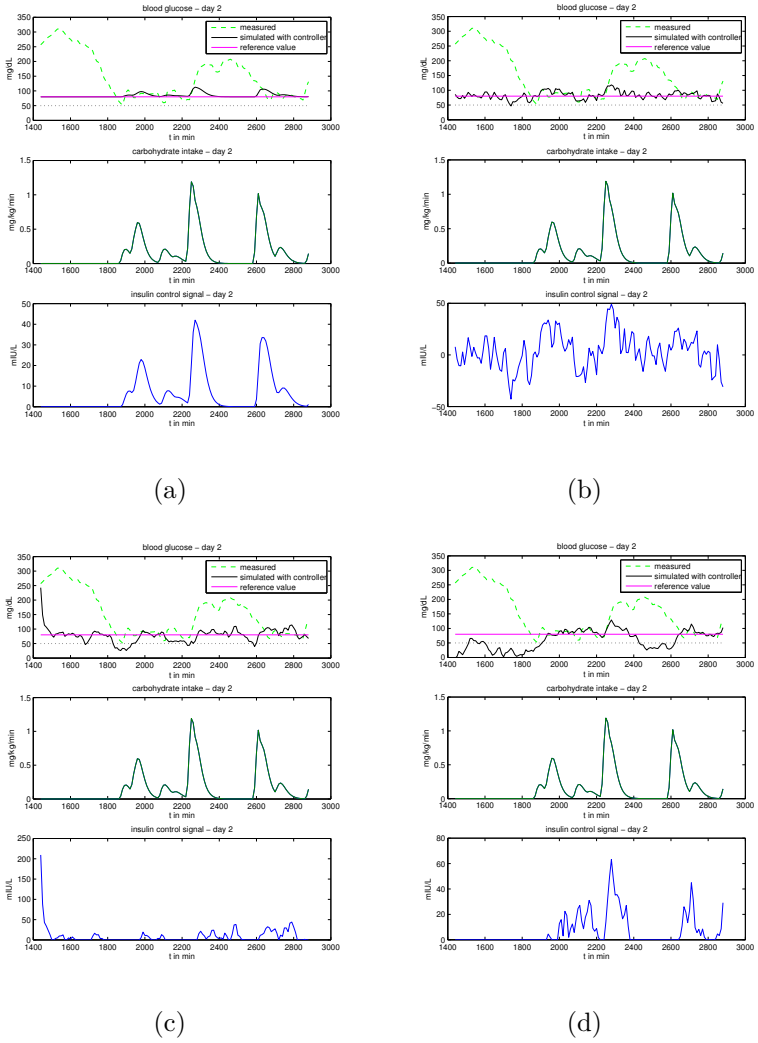
(a) part A and part B



(b) normal data without logarithmizing and part A

Figure 29: comparison of mean FIT values for logarithmized and detrended data

E Simulations with the controlled system



(a) (b)

(c) (d)

Figure 30: Simulations for day 2 with controlled insulin signal. (a) without noise; (b) with noise, negative insulin values are allowed; (c) with noise, initial value = 250 mg/dL; (d) with noise, initial value = 0 mg/dL

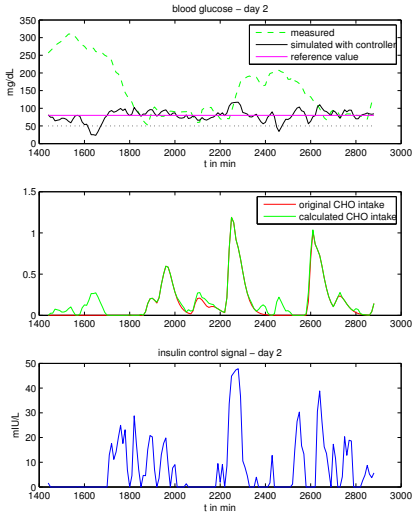
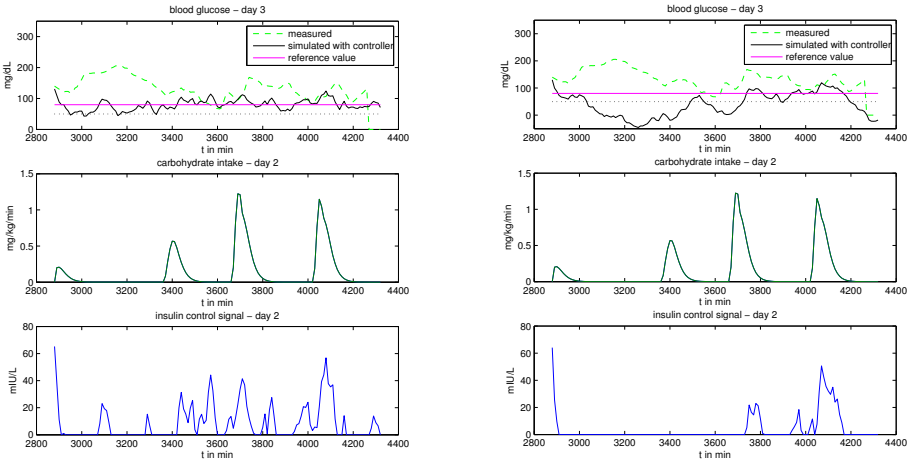


Figure 31: Simulations for day 2 with controlled insulin and carbohydrate signals



(a) (b)

Figure 32: Simulations for day 3 with controlled insulin signal

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