Symptom network models in depression research

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CHAPTER 11

DISCUSSION

In this final chapter, it is time to draw up the balance. What has been accomplished so far and where can we take the network approach on psychopathology from here? These questions will be covered in this discussion. First, the findings in this thesis will be highlighted, followed by an overall conclusion. Second, reflecting upon results in the field so far, I will propose a research agenda for the network approach to psychopathology.

11.1 This thesis

11.1.1 A theoretical deepening of the network perspective on psychopathology

This thesis started with an explanation of the network perspective in **Chapter 2**. According to this perspective, psychopathology can be viewed as a complex dynamical system. **Chapter 2** also presented an introduction into graphical models, which can be used to model dependencies between a set of measured variables. This forms the basis of the network perspective to psychopathology.

Chapter 3 contained theoretical explorations of hypotheses following from the network perspective about characteristics of networks of individuals who are vulnerable to developing MDD. Simulation studies showed that (1) networks with stronger connections have a higher probability of ending up in a depressive state,

(2) exerting stress to the network results in *hysteresis*, and (3) vulnerable networks display *early warning signals* before shifting from one state to the other.

11.1.2 Methodological challenges for group-level analyses: network estimation and comparison

To test in empirical data whether vulnerability to develop or maintain MDD is related to local or global network connectivity, there were two challenges to be faced: (1) estimating the network structure with a formal model and (2) comparing network structures.

Facing the first challenge, we developed IsingFit, an R package to estimate the network structure from cross-sectional, binary data (see **Chapter 4**). This was the first method that was based on a formal model (i.e., the Ising model) and combines regularized logistic regression with a Goodness-of-Fit measure to reveal relevant connections of a network. Since the introduction of *eLasso* and its implementation in R package IsingFit, it has been frequently used to study various psychopathological disorders — such as PTSD, MDD, psychosis, substance use disorder (Afzali et al., 2017; Bekhuis et al., 2016; Boschloo, Schoevers, et al., 2016; Boschloo et al., 2015; Fried, Bockting, et al., 2015; Isvoranu, Van Borkulo, et al., 2016; McNally et al., 2015; Rhemtulla et al., 2016; Vega-Dienstmaier, 2015; Wigman, Vos, Wichers, Os, & Bartels-Velthuis, 2016) — and on attitudes (Dalege et al., 2016). Also, since the introduction of regularization in network estimation, it has become a standard and opened the way for applying it to Gaussian variables (Epskamp, 2016; Epskamp, Waldorp, et al., 2016) and variables of mixed type (Haslbeck & Waldorp, 2015) in a network estimation conte.

The second challenge in investigating differences in local or global network connectivity of individuals who are more vulnerable to develop or maintain MDD, was how to compare networks of such groups. With only visual inspection to rely on at the time, it was ambiguous to decide whether differences were meaningful. With NCT (see **Chapter 5**) we combined advanced network estimation techniques with permutation testing to provide a statistical procedure for comparing network structures from two independent, cross-sectional data sets. While NCT has only been just introduced, it has already been applied to pre- and post-treatment measurements of MDD and GAD symptoms of a large psychiatric sample (Beard et al., 2016) and to perinatal depressive symptoms in a sample of pregnant women

(Santos, Fried, Asafu-Adjei, & Ruiz, 2017). Moreover, since the development of NCT, resampling techniques have been used in various other projects that assess the variability of network estimates (e.g., bootnet; Epskamp, Borsboom, & Fried, 2017). With bootnet it is now possible to obtain information about how prone a network is to sampling variation and if interpretations of the network are stable with less observations.

11.1.3 Empirical studies relating local and global connectivity to vulnerability

In **Chapter 6**, we investigated the relationship between vulnerability and global network connectivity in an empirical study. We studied patients with MDD of whom we know the diagnostic status at two-year follow-up. Persisters (i.e., those who still have MDD at follow-up) are regarded as a more vulnerable group than remitters (i.e., those who recovered from MDD at follow-up). Comparing the networks of both groups at baseline, we showed that persisters indeed have a more densely connected network structure.

Another way of studying vulnerability is investigating healthy individuals of whom diagnostic status during six years of follow-up is known. In **Chapter 8**, we investigated the local symptom network connectivity of healthy individuals at baseline. We showed that healthy individuals with baseline symptoms that have the highest centrality in the baseline network were more likely to develop MDD than individuals with other symptoms at baseline. Network parameters at baseline were shown to be predictive for diagnostic status at follow-up.

11.1.4 Methodological challenge for individuals: predicting future course of patients

In **Chapter 9**, we built further on the hypothesis that an individual′s symptom network structure could be predictive for future course of the disorder. We made a first step, by presenting a method to use the *infection* and *recovery* rates of symptoms, combined with the ESM-based network structure. This method results in a *Percolation Indicator* (PI), which — according to percolation theory from physics — should have predictive value for the behavior of the network. A validation study showed that PI is a good estimator and that infection and recovery are

well retrieved from simulated data. Applying the method to empirical ESM data showed that some adjustments to handle real data are necessary. The second step would be to investigate whether PI indeed has predictive power. This needs to be investigated in ESM data of patients in a non-treatment period (to estimate the network structure and PI) and a follow-up measurement (to investigate how well PI predicts how the disorder evolves).

11.1.5 Conclusions

Finally, in **Chapter 10**, we provided an overview of the most important findings of empirical network studies between 2010 and 2016. According to three important themes (i.e., comorbidity, prediction, and clinical intervention), we showed that a wealth of information has been gained in a relative short period. In the light of this thesis, the review showed that the vulnerability hypothesis following from the network perspective — i.e., vulnerability is related to network density) — is supported by a number of studies. A consistent finding seemed to be that the group with *problems* has a more densely connected network (Bringmann, Pe, et al., 2016; Pe et al., 2015; Wigman et al., 2015). Moreover, network parameters are found to be informative for future course of depression, as we already saw when discussing **Chapter 6** and **8**. But what do these and other results mean, what can we learn from it, and how do we proceed from here?

11.2 Research agenda for the future

With all the promising results shown here and the vastly growing popularity of the network approach, I finish this thesis by proposing a research agenda for the network perspective on psychopathology. In my view, this agenda should consist of four main pillars for future research: 1) the validity of the network approach, 2) understanding psychopathology, 3) implications for clinical practice, and 4) methodological development.

11.2.1 Validity of the network theory

With the recently proposed theory of mental disorders (Borsboom, 2017), this approach is now formalized as a scientific theory of mental disorders. With a limited set of theoretical principles, the network theory offers an explanatory

model for mental disorders and has clear implications for diagnosis and treatment. Diagnosis, for example, would involve identifying the symptom network and treatment would involve changing or manipulating that network (e.g., through intervening on a specific symptom) (Borsboom, 2017). Although the network approach has an intuitive appeal and sits well with how clinicians and patients think of psychopathology (Borsboom & Cramer, 2013; Frewen et al., 2012, 2013; Kim & Ahn, 2002), this does not automatically mean that mental disorders behave as networks. Moreover, results from empirical studies have yielded plausible results, but do not provide evidence that mental disorders behave according to network theory (Epskamp, Kruis, & Marsman, 2016). Therefore, the validity of the network approach needs to be investigated (Fried & Cramer, in press; Wichers, Wigman, Bringmann, & De Jonge, 2017).

It is conceivable, at least for some disorders, that a common cause perspective is more appropriate. For example, Fried and Cramer (in press) argue that for PTSD there is a clear common cause, the traumatizing experience. In the DSM-5 criteria for PTSD, one can see that the trauma is implied to cause symptoms such as having nightmares and flashbacks *about* the trauma (Fried & Cramer, in press). Still, there are also criteria of PTSD that do not seem directly caused by the trauma (e.g. feeling emotionally numb and negative feelings about oneself). Perhaps, the reality of psychopathology is that it is a combination of both the common cause and network perspective (Fried & Cramer, in press; Wichers et al., 2017). In a so-called *hybrid model*, onset of a mental disorder could be dictated by a common cause (e.g., the traumatic event for PTSD), whereas continuation of the disorder constitutes direct relationships among the symptoms (Fried & Cramer, in press). The work of Epskamp, Rhemtulla, and Borsboom (2016) can be a good starting point for this. This work introduces two generalizations of the network model by means of combining the latent variable (or common cause) and network modeling.

A key assumption that should be tested is that symptoms are assumed to directly interact with each other. To investigate this, an experimental design would be a good opportunity. The common cause and the network model lead to different hypotheses about what happens upon intervening on a specific symptom. For example, take insomnia and fatigue. If insomnia → fatigue is the true mechanism, intervening on insomnia will reduce fatigue, whereas it will not if insomnia ← MDD → fatigue is the true mechanism (Fried & Cramer, in press). In the latter

(common cause) mechanism, only intervening on MDD will reduce both symptoms. Recently, however, Bringmann and Eronen (2017) argued that the contrast between the common cause and the network model creates a needless dichotomy. The common cause model — which is a very generic model — is often pictured as symptoms being caused by the disorder itself and in which interactions between symptoms is not allowed. According to Bringmann and Eronen (2017), this is a very simplistic and restrictive representation of common cause models and that, to validate the network perspective, a more fruitful road would be to test hypotheses following this perspective. A first test would then be, for example, to investigate whether symptoms indeed have causal interactions.

11.2.2 Understanding and predicting psychopathology

The second pillar of the research agenda would be the pursuit of understanding psychopathology. A big topic in clinical practice and research is, for example, the heterogeneity in symptomatology of patients (Boschloo, Schoevers, et al., 2016; Boschloo et al., 2015; Fried & Nesse, 2015a; Østergaard et al., 2011). Therefore, taking individual symptoms into account instead of using sum scores makes more sense; not all patients are sufficiently similar to assume the sum score adequately reflects their condition. The network approach naturally accommodates this property and is, consequently, an excellent tool to obtain a better understanding of psychopathology. It provides an opportunity to investigate why and/or how some people develop a mental disorder and others do not, and why and/or how some people recover from it and others do not.

From a network approach, research with the aim of understanding psychopathology could involve investigating whether there are certain characteristics in the dynamic network structures of patients (e.g., with MDD) that are associated with recovery or persistence of MDD. Already, there is evidence in the literature that network structure is related to worse outcome (see **Chapter 6** and **8**, but also Bringmann, Pe, et al., 2016; Pe et al., 2015; Wigman et al., 2016). In these studies, however, data — be it cross-sectional or ESM — were analyzed at the group level. What, then, do these results imply for an individual? The sobering answer is: we do not know. Taken to the extreme, individual networks might theoretically even be radically different from networks based on cross-sectional data. However, it does not seem plausible that individual networks will differ radically from group-level

networks, since it does not seem likely that for one patient with MDD feelings of worthlessness would cause suicidal ideation, but in another these feelings would result in cheerfulness. For future research, relevant questions are, for example, “How do individual networks relate to group-level networks?” and “Are there similarities in individual networks that are stable across patients?”

11.2.3 Networks in clinical practice

Since the network perspective on psychopathology has clear implications for diagnosis and treatment (Borsboom, 2017), the ultimate goal would be to have a place in clinical practice. Roughly, there are two ways in which the network approach can have an added value. First, it can help patients to gain insight into the dynamics of their own symptoms. In a pioneering study, Kroeze et al. (2016) implemented networks in clinical treatment. The dynamic network structure of a patient started a dialogue that motivated the patient to deal with a problem she was reluctant to deal with before. From this first result, Kroeze et al. (2016) concluded that using ESM and networks in clinical practice was feasible and provided useful additional insight, based on a data-driven source; it provided the patient a sense of control over her own recovery. Second, ESM and networks could guide micro-intervention. By establishing a patient$'$s dynamic symptom network through ESM measurements, those symptoms that can exert the strongest influence on the others could be revealed. A symptom that appears to have many causal influences on other symptoms and seems to be responsible for maintaining a disordered status would be a good candidate for intervention. Treating such an influential symptom would, according to the network approach, be the most efficient way to shift from a disordered to a healthy state.

Questions pertaining to the line of research as described above are more prediction oriented, such as "does intervening on symptom X decrease other symptoms efficiently? Micro-interventions have been advocated in many network studies and are posited as a big promise. But what do we currently know about this? So far, no studies have been conducted that intervened on a specific symptom and then monitored the subsequent changes on other symptoms to investigate whether this pattern of changes was as expected from th accompanying network structure. There are a few reasons for this, of which one might be that it is simply too soon. We first need to establish validity of the network approach. But even if we

have validated the network approach, there are several questions to be answered. For example, is it even possible to perform targeted intervention on a specific symptom? For some symptoms there are indeed symptom interventions, such as for sleep problems (Christensen et al., 2016), distress (Geraghty et al., 2016), suicidal behavior (Aseltine, James, Schilling, & Glanovsky, 2007), and worrying (Khoury et al., 2013; Klainin-Yobas, Cho, & Creedy, 2012) in depressive disorder. For other symptoms it may be less workable to treat them in isolation, such as fatigue or change in appetite. The field of experimental psychopathology might provide valuable insights for symptom oriented interventions (see van den Hout, Engelhard, & McNally, in press). In this field, symptom manipulations have been investigated that reduce specific symptoms of, for example, vividness in PTSD (Engelhard, van den Hout, & Smeets, 2011) and compulsions in OCD (Arntz, Voncken, & Goosen, 2007).

Besides the question whether it is feasible to intervene on an isolated symptom, one can also think about at what stage of MDD micro-intervention can be of most value: in a full-blown or an earlier prodromal phase? In case of a full-blown MDD, an often applied treatment currently is a combination of medication and psychotherapy (Hollon et al., 2005). Since medication intervenes in the process of neurotransmission in the central nervous system (CNS; Bondy, 2002) and psychotherapy addresses current interpersonal relationships (Mello, Mari, Bacaltchuk, Verdeli, & Neugebauer, 2005), this strategy will likely indirectly affect multiple symptoms. Micro-intervention is aimed to target one specific symptom (i.e., the one which is hypothesized to be the optimal target according to network analysis). Available research suggests that low intensity interventions in patients with more severe depression have at least the same response as in patients with milder complaints (P. Bower et al., 2013). However, whether this strategy will provide similar reduction of symptoms or even outperform standard therapy needs to be established with clinical trials.

Instead of intervening during a full-blown MDD, micro-intervention informed by the network approach could, possibly, be of more value in an earlier phase of MDD (i.e., the prodromal phase; Fava & Tossani, 2007) or when MDD is not full-blown but moderate. The goal would then be to prevent the illness from fully developing by applying a targeted, less intensive individualized intervention. Intervening on a symptom that is crucial in the *spreading* of problems across the network, might be more fruitful than intervening on the same symptom when

it has already culminated in a full-blown disorder. Note, however, that intervening on symptom-symptom interactions that is aimed at weakening the network structure could also be a good treatment strategy, regardless of the phase of MDD (Borsboom, 2017). With cognitive behavioral therapy, for example, dysfunctional patterns of thoughts (e.g., extreme worrying about not sleeping well) are replaced by healthier ones. In network related terms, this would weaken the connection between "insomnia" and "worrying/feeling worthless".

11.2.4 Methodological development

The fourth pillar of the research agenda is about the development of methods within the network perspective of psychopathology. Although the development of network analytic techniques has made remarkable progress in recent years, the network perspective of psychopathology is relatively young and could benefit from considerable methodological advances. It is important to note that the following is by no means intended as an exhaustive list of research topics and that it is based on topics I plan to work on in future projects[1].

11.2.4.1 Elaboration on existing work

In future work, IsingFit could be extended to provide a solution to highly correlated variables in the model. Currently, *eLasso* assumes the network structure to be sparse and will suppress weaker edges to zero. But what if that assumption does not hold? What if some symptoms of mental disorders are strongly associated? The aggregated symptom "sleep problems", for example, consists of the strongly associated variables insomnia and hypersomnia. This strong association might *dominate* the network structure such that other, less strong but potentially interesting associations will be suppressed to zero. In my view, a possible solution might be found in combining IsingFit with an estimation method suited for dense network structures (Marsman, Maris, Bechger, & Glas, 2015). Combining these techniques would allow for a certain part of the network to be more densely connected than other parts of the network. This may provide a way to study, for example, a disaggregated symptom network, without the strong influence of regularizing highly correlated variables.

[1] For more ideas about future research relating to methodological development, I refer to the Discussion section of the dissertation of Epskamp (2017; `http://sachaepskamp.com/Dissertation`)

Another extension could be to allow for more than two *ordered* categories of variables. Currently, *eLasso* accommodates binary data, *GeLasso* Gaussian data (Epskamp, 2016), and *MGM* categorical data (Haslbeck & Waldorp, 2015). Unfortunately, neither option can accommodate ordinal data. Often, questionnaires in psychology and psychopathology contain Likert items in which the categories are ordered (e.g., IDS; Rush et al., 1996). Although `cor_auto()` in qgraph can handle ordinal data by computing polychoric correlations (Epskamp, 2016), the ultimate regularized GGM network requires two steps instead of one, as in current aforementioned methods (see p. 239 of the dissertation of Epskamp, 2017; `http://sachaepskamp.com/Dissertation`). A big improvement in network estimation will come from a method, which is based on a model that deals with ordinal data more appropriately.

An entirely different line of investigation based on the Ising model could be about finding optimal targets for intervention in clinical practice. Given a certain network structure (ultimately of a patient), intervening on a symptom, could be modeled as *turning off* a node in the Ising network. Turning off a node will increase the probability that neighboring nodes will turn off. Consequently, it seems natural to intervene on the node with most neighbors, as this will yield the highest number of nodes turning off. However, a node that connects different parts of the network might also be an efficient target. Intervening on such a node provides the possibility to reach the whole network instead of one part. A search algorithm could detect the minimal set of target nodes for intervention, which yields maximal symptom reduction. An inspiring hands-on simulation tool "VAX!" shows a related model, which is about preventing the spread of a contagious disease across a network of individuals (`http://vax.herokuapp.com/game`).

For NCT, I can see three ways to extend it in its current form. First, other network estimation methods could be implemented. Recently developed methods, such as for categorical and mixed variables data (i.e., with `corauto()` in package qgraph and `mgmfit()` in package mgm; Epskamp et al., 2012; Haslbeck & Waldorp, 2015) could be incorporated. Second, researchers may also want to compare groups of individuals with ESM data. This allows for comparing two *population* networks (Bringmann et al., 2013; Epskamp et al., 2015). By randomly assigning individuals' ESM data sets to one of the groups, one can generate a null distribution of the metric of interest (i.e., a metric expressing the difference). This would

allow, for example, to compare the network based on healthy people′s ESM data to that of patients.

Another extension of NCT is to include the thresholds of the variables into the test. In the Ising model, the thresholds represent the autonomous predisposition of the variable to be 0 or 1 and are estimated with the intercepts of logistic regression. Although they are largely ignored in network analyses, it could be interesting to investigate. For example, the networks of male and female patients in **Chapter 5** — which served as an illustration for NCT — look very similar and NCT did not detect differences. Although we did not investigate this, it could be that thresholds are higher in one group than the other. The group with the higher thresholds would be the one with higher symptom means. In other words, although the association patterns are similar, the group with higher means have symptoms that are inclined to be more severe compared to the other group. Therefore, it is important to also be able to test for differences in thresholds.

11.2.4.2 Future topics of investigation

To understand how MDD develops and why some people develop MDD and others do not, it is important to study this process over time. ESM studies, in which patients are tracked over time, seem ideal for this purpose. However, having to fill out a questionnaire five times a day for two weeks is a time-consuming and burdensome process for patients. Before employing this widely, it seems important to me to think about three topics carefully. First, we need to develop methodology to express similarities in group-level and individual-level networks. Say, for example, that we are interested in whether a single edge in a group-level network is also present in individual-level networks. When can we say that it does? Similarity could be defined as an edge being present in a certain percentage of the individual-level networks, similar to the Group Iterative Multiple Model Estimation procedure (GIMME; Beltz, Wright, Sprague, & Molenaar, 2016; Gates & Molenaar, 2012). Another strategy could be to look at edge distributions. Assuming that the group-level network represents an *aggregate* of the network of individual group members and that individuals differ a lot in their individual-level network structure, the edges in the group-level network might be more evenly distributed than in the individual-level networks. That is, one individual could have particularly strong connections between symptoms A, B, and C, while an-

other individual has a strongly connected cluster of symptoms C, D, and E. In the group-level network, symptoms are expected to be more evenly connected.

The second topic is concerned with ESM data. Such data commonly consists of repeated measurements of an individual′s emotions; several emotions are measured about 8 to 10 times a day during 5 to 7 consecutive days (Bringmann et al., 2013; Pe et al., 2015; Wigman et al., 2015). Ultimately, the goal of ESM studies is to gain a better understanding of the onset and recovery (or recurrence) of MDD at the level of an individual (Wichers, 2014). I wonder, however, whether we are measuring the right variables here. Variables that are currently used in ESM studies are often emotions such as feeling cheerful, anxious, and disgusted (Bringmann et al., 2013; Pe et al., 2015; Wigman et al., 2015). But what exactly is the relationship between these short-term fluctuations in emotions (i.e., micro-level moment-to-moment experiences) and the symptoms of MDD (i.e., macro-level experiences; Wichers, 2014)? For example, how does feeling cheerful, anxious, and disgusted relate to symptoms such as "depressed mood", "loss of interest", and "feelings of worthlessness"? Presently, it is unclear how emotions, measured with ESM, relate to symptoms of MDD. If the main goal is to gain a better understanding of the onset and recovery (or recurrence) of MDD, it is questionable whether emotions are the ideal level of measurement. A good opportunity to study this in the nearby future will be provided by NESDA (Penninx et al., 2008), in which 400 of its nearly 3000 participants are studied with Ecological Momentary Assessment over 2 weeks. Besides EMA, NESDA-400 also symptoms of MDD are assessed with the Composite International Diagnostic Interview (CIDI; Wittchen, 1994).

The third topic, which is related to the previous, entails whether the time scale of current ESM studies is optimally suited for achieving a better understanding of the course of MDD. Although a two-week measurement period (e.g., as in NESDA-400) contains interesting information, it may not be adequate to fully capture the dynamics of MDD. In my view, symptom dynamics should ideally be investigated on a time scale in which depression has a chance to develop in individuals prone to the disorder. This seems to imply measuring symptoms once a day, over a prolonged period of a year. Only then one can capture the dynamics of interest. Currently, the largest provider of mental health services in the province of Friesland, the Netherlands (GGZ Friesland) is performing innovative ESM studies. In the *ZELF-i* study, for example, individuals with depressive complaints can track themselves for four weeks and receive personalized feedback on their dynam-

ics at the start of treatment (see http://www.trialregister.nl/trialreg/admin/rctview.asp?TC=5707 for information about this trial). Another study, the MIRORR project, aims to investigate whether network characteristics of momentary mood states of subgroups of adolescents with increasing levels of psychopathology can predict the course of psychopathology (see Booij et al., 2017, for more information about this trial).

These studies were separately funded from this PhD project and offer an interesting opportunity to also investigate long-term symptom dynamics using the statistical methodology such as described in this thesis. Another interesting development is that the Netherlands Study of Depression and Anxiety (NESDA; Penninx et al., 2008) now also aims to set up a study in which depression symptoms are measured daily, during a year in a large number of subjects. Together with the existing, more traditional ESM study, as well as the elaborate phenotypic information of the subjects, this will yield a wealth of possibilities to further explore symptom networks and how they vary between and within persons.

Combining short-term momentary emotion fluctuation and long-term symptom dynamics of the same individuals, will allow studying the relationship between the two types of dynamics. I believe the *watershed* model could be a starting point for this (Cannon & Keller, 2006; Kievit et al., 2016). This model integrates different explanatory levels as emergent properties of a hierarchical system. According to this watershed model, psychopathology can be viewed as the downstream consequence of many small upstream effects. In case of momentary emotions and symptoms, this could imply that momentary emotion states (upstream) and interactions between them can *spill over* to problems at symptom level (downstream). The implementation of this model in a Structural Equation Modeling (SEM) framework by Kievit et al. (2016), could be used to adapt this model to a network framework. With such an implementation, I plan to study whether indeed dynamical relationships between emotion states are the micro-level patterns of the macro-level development of psychopathology, as is assumed in ESM studies (Lunansky, Van Borkulo, Wichers, & Cramer, 2017). See Wichers (2014) for the basis of this assumption.

To conclude, much has been accomplished in the network perspective to psychopathology in recent years, but there is even more work that remains to be done. Hopefully, the proposed research agenda for the network approach to psychopathology can help in focusing research activities in this field. In this

research agenda, I see a clear role for the development of methodology besides the more clinically driven topics. Methodological development can range from building on existing methods (e.g., including symptom thresholds in comparing network structures), to developing new estimation methods (e.g., to model ordinal data), to developing tests to be able to investigate more clinically related topics (e.g., to establish whether group-level and individual-level networks are similar). It is this interface between methodologically and clinically oriented topics and the inspiring collaboration between researchers from different backgrounds, to which I hope to contribute in the future.