Towards a cognitive neuroscience of prosody perception and its modulation by alexithymia

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Chapter 4

Summary and conclusion

4.1. Recapitulation of research questions

Imagine again that you are sitting in a train, absorbed by a good book, and suddenly hearing a mother shouting angrily to her child: "Tim, you are going to annoy the other passengers!" As we have observed in Chapter 1 ('Introduction'), many people would be familiar with the involuntary tendency to turn one's head into the direction of Tim's mother, revealing that the utterance apparently constitutes a powerful social signal that our brain seems to process even we when we do not intend to.

In Chapter 1, I have explained that the communicative layer of speech that Tim's mother is using to convey anger is called 'prosody' in linguistics. Further, we have elucidated that prosody cannot only be used to convey emotional meaning (which we have called 'emotional prosody') but also part of the linguistic structure of an utterance (which we have called 'linguistic prosody'). The first empirical section of the present thesis was concerned with *what* network in the brain supports these two communicative functions of prosody, *how* the network does so (i.e., using what series of operations), *whether* this network indeed sometimes processes this information 'automatically' and, if so, *why* this might be the case. Concerning the last question, we have hypothesized that the existence of a 'hard-wired' system in the brain that prioritizes the processing of social signals that indicate potential harm to the organism (such as an angry mother, in the example above) can explain such automaticity of processing (Öhman & Mineka, 2001). To test this hypothesis, we have directly contrasted processing of emotional prosody with an artificial acoustic signal that has probably emerged more recently in evolutionary history (and is therefore probably not supported by a hard-wired dedicated system) that can also powerfully convey emotion – *music*.

Although the basic architecture of the prosody perception network would be expected to be relatively stable across individuals (otherwise, it would be hard to explain why communication through the prosodic layer of speech is relatively stable in its efficiency across individuals), there are also subtler differences in affective processing style among individuals (Canli, 2004). One personality trait that is associated with affective processing style is called *alexithymia* ('no words for feelings'), a trait that is associated with difficulties in recognizing, identifying and verbalizing emotions (Sifneos, 1973). As variation along the alexithymia continuum is associated with emotional processing differences, it is plausible that such differences are reflected in the emotional prosody perception network. Therefore, in the second empirical section of the present thesis, it was investigated whether (non-clinical) variation in alexithymia indeed modulates activity within the emotional prosody perception network, and if so, whether alexithymia is primarily reflected at a relatively early (and hence potentially 'automatic') or a relatively late stage of emotional processing (or both).

4.2. Towards a cognitive neuroscience of prosody perception and its modulation by alexithymia

4.2.1. Interhemispheric models of prosody perception

In Figure 1 (identical to Figure 1 of Chapter 1, repeated here for ease of reference), a schematic visualization is presented of the prosody perception pathway, as hypothesized by the two major models of (emotional) prosody perception (Schirmer & Kotz, 2006; Wildgruber, Ethofer, Grandjean, & Kreifelts, 2009) that existed when the work for the present thesis was initiated. As has been pointed out in Chapter 1 and will be explained in more detail in the next section, these models suggest that prosody perception is a multi-stage process, with elementary acoustic processing first taking place in the primary auditory cortex (Heschl's gyrus, or HG) and the middle part of the

superior temporal gyrus (m-STG). Subsequently, more abstract and complex auditory processing would take place in either the anterior STG (a-STG) or posterior STG (p-STG). Finally, abstract evaluation of prosody is hypothesized to be sub-served by the inferior frontal gyrus (IFG).

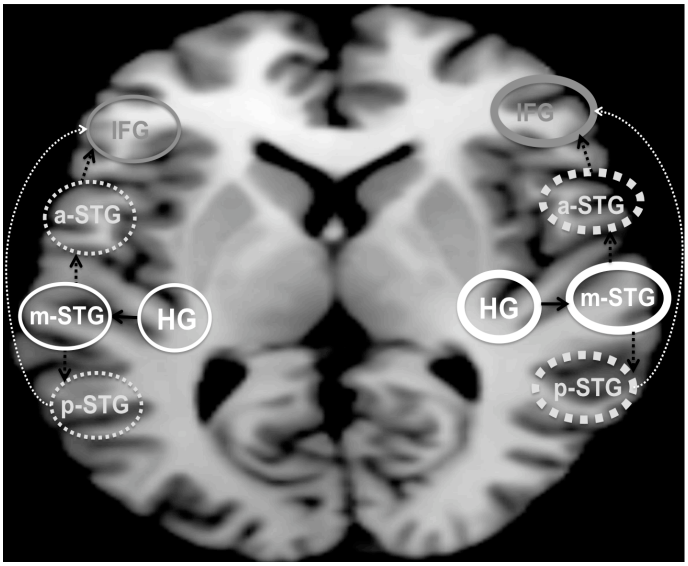

Figure 1 (repetition of Figure 1 of Chapter 1). Schematic visualization of the prosody perception pathway as hypothesized by two dominant models of (emotional) prosody perception, superimposed on an axial slice of an MRI scan. Solid circles and arrows indicate regions and connections that are hypothesized to be important in (emotional) prosody perception by both models. Dashed circles and arrows indicate areas and connections hypothesized to be involved in prosody perception by only one of the two models. White circles indicate areas hypothesized to be involved in the first stage, light-grey circles in the second stage and dark-grey circles in the final stage of prosody perception. Bold circles in the right hemisphere as compared to the left indicate hypothesized right hemispheric superiority for emotional prosody perception.
Abbreviations: HG = Heschl's Gyrus, m-STG = mid superior temporal gyrus, p-STG = posterior superior temporal gyrus, a-STG = anterior superior temporal gyrus, IFG = inferior frontal gyrus.

Early work in the cognitive neuroscience of prosody perception used the lesion-deficit approach in an attempt to answer the question of whether one cerebral hemisphere might be superior at (or 'specialized' in) processing of emotional prosody. As I have argued in Chapter 1, lesion-deficit data indeed provide the most powerful test of hemispheric specialization hypotheses since such data inform us about whether a hemisphere is *necessary* for a hypothesized mental function such as prosody perception. Three hypotheses concerning hemispheric specialization for prosody perception had been put forward:

(1) The right-hemisphere hypothesis (Ross, 1981) proposes that the right hemisphere is specialized in emotional prosody perception;
(2) The *functional* lateralization hypothesis (Van Lancker, 1980) adds that the hemispheres are specialized in the processing of different *functional categories* with the left and right hemisphere being specialized at the processing of linguistic and emotional categories, respectively;
(3) *Acoustic* lateralization hypotheses, in contrast, propose that hemispheric specialization for prosody perception can be traced back to specialization of the hemispheres for different acoustic dimensions of the speech signal. A prominent acoustic lateralization hypothesis states that the left hemisphere is better at processing of temporal information while the right is better at the processing of spectral information (Van Lancker & Sidtis, 1992). Because spectral information is important for the perception of emotional prosody, a right-hemisphere specialization for emotional prosody perception could then be explained by a low-level specialization for spectral processing.

Note that the functional lateralization hypothesis can be seen as an elaboration of the right-hemisphere hypothesis. Further note that the acoustic and functional lateralization hypotheses are not mutually exclusive – they could both be true and represent different stages of the prosody perception process (symbolized by the circles with different shades of grey in Figure 1).

When the work for the present thesis was initiated, the lesion-deficit literature had not provided consensus about whether there is hemispheric specialization for prosody perception and, if so, which of the above hypotheses can best explain it. Therefore, in Chapter 2.1, a test of these hypotheses was performed using quantitative meta-analysis of the lesion-deficit literature. Using the power of the combined sample size of all lesion-deficit studies on emotional and linguistic prosody perception published to date, it was found that *both* hemispheres are necessary for emotional and linguistic prosody perception (i.e., damage to each hemisphere significantly degraded linguistic and emotional prosodic perception performance). However, damage to the right hemisphere was more detrimental to emotional prosody perception performance than equivalent left-hemispheric damage. Thus, we found meta-analytic evidence for *relative* right-hemispheric specialization for emotional prosody perception and no evidence in favor of hemispheric specialization for linguistic prosody perception. In conclusion, based on the lesion-deficit studies to date, there is no support for the *functional* lateralization hypothesis (because no left-hemispheric specialization for linguistic prosody perception was found) and only evidence in favor of a weak (relative) version of Ross' (1981) right hemisphere hypothesis. However, the meta-analysis did not allow for a direct test of the acoustic lateralization hypothesis versus the functional lateralization hypothesis.

Therefore, in Chapter 2.2, a systematic test of the functional versus acoustic lateralization hypotheses of prosody perception was performed. This was achieved by presenting participants with bi-dimensional pseudowords that had both either angry or sad prosody and either linguistic stress on the first or second syllable. Exactly the same pseudowords were presented dichotically to participants while event related potentials (ERPs) were recorded. However, about half of the participants were instructed to categorize the *emotional* prosody dimension of the stimuli, while the other half categorized the *linguistic* prosody dimension. If the functional lateralization hypothesis were correct, we would expect to find a shift from a right to left hemispheric advantage in activation over the scalp for the emotional versus the linguistic task. Note that, since the acoustic material that was presented to both groups of participants was identical, such a shift would be evidence for the hypothesis that the functional processing mode (emotional versus linguistic mode of prosody perception) *pre se* can indeed drive hemispheric asymmetry. However, no such shift in hemispheric advantage was found, neither as indicated by the ear advantage nor on the electrophysiological level, even though the statistical power of our study was relatively high. Thus, in Chapter 2.2 we did not find evidence in favor of the functional lateralization hypothesis of prosody perception, which is in keeping with the results of the quantitative meta-analysis performed in the preceding chapter.

Can acoustic lateralization hypotheses, then, better account for the relative right-hemispheric specialization for emotional prosody found using meta-analysis of the lesion-deficit literature in Chapter 2.1? Note that, if the relative right-hemispheric specialization would indeed be driven by superiority of the right hemisphere in processing of acoustic cues relevant for emotional prosody perception, we would expect a right-hemispheric advantage to emerge somewhere relatively early (at the level of the auditory processing centers) in the prosody perception pathway visualized in Figure 1. In Chapter 2.3 of this thesis, a quantitative meta-analysis was performed on the neuroimaging literature of emotional prosody perception, which will be discussed in more detail in the next section. This meta-analysis revealed a bilateral temporo-frontal network involved in emotional prosody perception, again illustrating that *both* hemispheres are involved in emotional prosody perception. Formal meta-analyses of hemispheric asymmetry, however, revealed higher activation probability in the right transverse temporal gyrus (HG) and p-STG than in their left-hemispheric homotopes, albeit only at a liberal statistical threshold. Thus, meta-analytic evidence again points to *relative* hemispheric specialization for emotional prosody perception in the context of *bi*hemispheric involvement. Further, there is initial evidence that relative right-hemispheric specialization for emotional prosody perception is driven by specialization for elementary (HG) and more abstract (p-STG) acoustic processing within the prosody perception pathway, in line with *acoustic* lateralization hypotheses of emotional prosody perception. This result is also in line with our primary neuroimaging study of emotional prosody perception reported in Chapter 2.4, where we found a relative rightward hemispheric asymmetry for emotional prosody perception across tasks in the STG.

To summarize, regarding interhemispheric models of prosody perception, the present thesis finds evidence in support of *relative* right-hemispheric specialization for emotional prosody perception and no hemispheric specialization for linguistic prosody perception, based on meta-analysis of the lesion-deficit literature. Further, in a primary study using electroencephalography to systematically test the *functional* lateralization hypothesis, we do not find evidence for the functional lateralization hypothesis of

prosody perception. Quantitative meta-analysis of the neuroimaging literature further points to the involvement of a *bi*hemispheric network in emotional prosody perception. Formal meta-analytic analyses of hemispheric asymmetry within this network, suggest that relative right-hemispheric specialization for emotional prosody perception is driven by superiority of the right-hemisphere auditory processing centers, in line with *acoustic* lateralization hypotheses of emotional prosody perception. These results are in keeping with a mounting evidence base in support of hemispheric specialization for basic dimensions of the speech signal (Boemio, Fromm, Braun, & Poeppel, 2005) and hence bottom-up explanations of hemispheric specialization for more complex auditory signals, such as prosody.

In Figure 2, the insights gained from this thesis regarding the interhemispheric models of prosody perception are visualized by the bold circles that indicate a hemispheric advantage in the right prosody perception pathway as compared to the left, based on initial meta-analytic evidence.

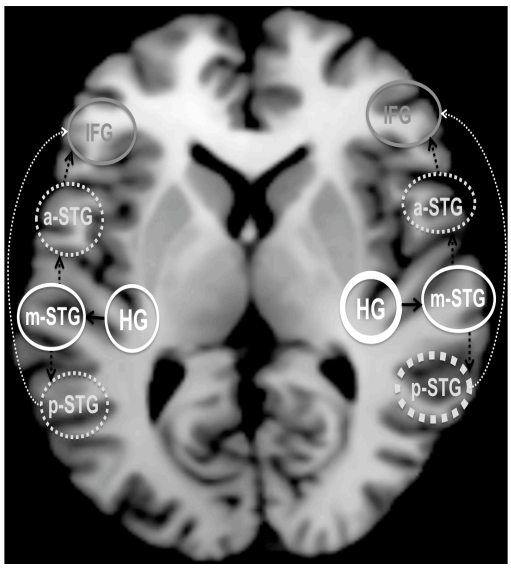

Figure 2. Adaptation of the model presented in Figure 1 based on the insights gained from the present thesis regarding interhemispheric models of prosody perception. The bold circles indicate right hemispheric processing centers in the emotional prosody perception pathway where initial evidence for a relative right hemispheric advantage has been found in this thesis (for further details, see Figure 1).

4.2.2. Intrahemispheric models of prosody perception

Functional magnetic resonance imaging (fMRI) allows us to further probe the brain to investigate which areas within each cerebral hemisphere are involved in prosody perception. When the work for this thesis was initiated, there already was a substantial body of fMRI literature available on emotional prosody perception. These neuroimaging studies had inspired two very similar three-stage models (Schirmer & Kotz, 2006; Wildgruber, Ethofer, Grandjean, & Kreifelts, 2009) of emotional prosody perception. In Figure 2, a schematic visualization can be found of the neural network of prosody perception these models propose (and the disagreement between the two models). Briefly, both models propose that in an initial stage (white circles in Figure 2) basic acoustic processing of (emotional) prosody takes place in the transverse temporal gyrus or Heschl's gyrus (HG) and the middle superior temporal gyrus (m-STG). Subsequently, in a second stage (light-grey circles in Figure 2), both models propose that more complex acoustic processing takes place to integrate the auditory information from stage one into more abstract units of representation, but the models disagree regarding the location of this processing stage. While the Wildgruber model places stage-two prosody perception in the posterior STG (p-STG), the Kotz model positions it further down the auditory object recognition pathway (the 'what' stream) in the anterior STG (a-STG). Finally, both models propose that in a third stage (dark-grey circles in Figure 2), abstract evaluation of prosody takes place in the inferior frontal gyrus (IFG).

In Chapter 2.3, a quantitative meta-analysis of functional neuroimaging studies was performed to test with high statistical power which of these two models can best account for the neuroimaging literature to date. Two functional contrasts were meta-analyzed, (i) a low-level contrast capturing all hypothesized phases of the emotional perception process, and (ii) a high-level contrast that captures more abstract (stage two and three) emotional prosody perception. The low-level contrast revealed above-chance convergence of activation likelihood in the bilateral m-STG continuing medially into HG, p-STG and the IFG. However, no significant convergence of activation likelihood was found in the a-STG. In the high-level contrast, significant convergence of activation likelihood was found in the right p-STG and IFG. Importantly, no significant convergence was found in either contrast for the amygdala, a structure hypothesized to be important for emotional perception (Schirmer & Kotz, 2006) and to be the evolved neural structure supporting automatic processing (Öhman & Mineka, 2001), an issue we will return to in the next paragraph. On the one hand, the amygdala might just not be crucial for emotional prosody perception, as suggested by lesion studies (Adolphs & Tranel, 1999; Bach, Hurlemann, & Dolan, 2013). However, as has been pointed out before, it is possible that the amygdala quickly habituates to emotional prosody, preventing its detection with fMRI paradigms that typically use sustained stimulation (Wiethoff, Wildgruber, Grodd, & Ethofer, 2009; but see Scheuerecker et al., 2007). Thus, the quantitative meta-analyses reported in Chapter 2.3 confirmed that HG and m-STG are likely to be involved in stage one and the IFG in stage three prosody perception, respectively, as proposed by both intrahemispheric models of prosody perception. Concerning stage-two prosody perception, however, our meta-analyses suggest that this more abstract acoustic processing stage is more likely to be supported by the p-STG as predicted by the Wildgruber model than the a-STG as predicted by the Kotz model. In Figure 3, a final modified schematic illustration of the prosody

perception pathway is provided based on our meta-analyses of the neuroimaging literature on emotional prosody perception.

Next, we asked whether automaticity of emotional prosody perception can be demonstrated in the emotional prosody perception network illustrated in Figure 3. As I have explained in Chapter 1, automaticity of prosody perception can be demonstrated at the neural level, by diverting attention away from the emotional prosody dimension of an utterance and observing whether there is continuation of above-threshold neural processing of emotional prosody (Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003). If we find such sustained neural processing of emotional prosody when participants do not intend to analyze the emotional prosody, this is evidence for the 'unintentionality feature' of automaticity of processing, as proposed by Moors and De Houwer (2001). Note that, based on Darwin's theory of evolution, it has been proposed that a hard-wired system dedicated to the perception of emotional prosody could explain automaticity for prosody perception, and that such a dedicated system would be particularly plausible for emotional information that signals potential harm to the organism (Öhman & Mineka, 2001; Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003; Schupp et al., 2004). If this hypothesis were true, we would particularly expect automatic perception of emotional prosody that signals *threat*, i.e., anger prosody. Thus in Chapter 2.4, using fMRI, we investigated whether continued supra-threshold activation could be found for anger prosody and, as a control, a non-threatening emotional category (surprise) when participants do not intend to analyze emotional prosody. It was found that for surprise no significant supra-threshold activation could be demonstrated when attention was diverted from the emotional prosody. For anger processing, however, continued supra-threshold activation was found in the right STG when subjects did not attend to the emotional prosody. Thus, in Chapter 2.4, we provide evidence for automatic processing of emotional prosody, but only when emotional prosody signals potential harm (i.e. anger), as predicted by models that assume an evolved dedicated system for the detection of social information that signals potential harm to the organism (Öhman & Mineka, 2001). However, our results suggest that the neural substrate does not reside in the amygdala but in the right superior temporal gyrus. Thus, also based on our meta-analyses of the lesion and neuroimaging literature above, it might be the specialized acoustic processing centers in the right hemisphere instead of the amygdala that have evolved to detect danger in the auditory modality. Alternatively, as has been pointed out above, it might be the case that these auditory processing centers are engaged by the amygdala first, but that activation of the amygdala itself is missed by fMRI because the amygdala quickly habituates to emotional prosody stimulation.

As I have elucidated in Chapter 1, if automaticity of emotional prosody perception can be explained by an evolved hard-wired system dedicated to the detection of emotional states of conspecifics, we would not expect automatic processing of a probably more recently invented artificial signal that can powerfully convey emotion – *music*. Alternatively, cross-cultural recognition of music by culturally disparate groups suggests that there might be a dedicated biological system for the recognition of emotion in music, too (Fritz et al., 2009) pointing to a potential fitness value of emotional music perception. Further, the 'super-expressive voices' hypothesis (Juslin & Västfjäll, 2008) proposes that emotional music obtains its powerful emotional expression capability by imitating and subsequently exaggerating the acoustic properties of emotional prosody. Thus, by imitating emotional prosody, music could engage a

system dedicated to the processing of emotional prosody and as such be processed automatically, too. If any of these two alternative hypotheses were true, we would expect automaticity of emotional music perception as well.

Recall that, in addition to the unitentionality feature of automaticity discussed above, rapid processing has been proposed to be a feature of automaticity (Moors & De Houwer, 2001). As was explained in Chapter 1, the affective priming paradigm (Fazio, 2001) takes advantage of the hypothesized fast processing of emotional information by first presenting an emotional stimulus (the affective prime) that is either positive or negative and almost immediately (typically 200 ms) thereafter presenting a second emotional stimulus (the target). Participants are required to categorize targets as positive or negative. The affective prime hence can be either congruent or incongruent with the valence of the affective target. If the affective prime is indeed processed very rapidly (i.e. in less than 200 ms), it should be able to facilitate responses to congruent targets but inhibit responses to incongruent targets. In Chapter 2.5 we presented participants with very short (800 ms) segments of emotional prosody and emotional music that were either happy or sad. Shortly (200 ms) after the onset of these affective primes, positive and negative affective target words were presented, which participants were required to categorize with respect to valence. While participants were engaged in the affective priming task, ERPs were recorded. We found a significant affective priming effect (APE) for prosody but not for music. Further, the so-called N400 effect was observed for incongruent vs. congruent trials for prosody and music, which had previously been proposed to be associated with automatic spreading of activation. However, in a second study, participants performed exactly the same tasks but with attention diverted from the affective dimension of the emotional targets. This time no APEs or N400's were found for prosody (nor for music). Thus, although mainly emotional prosody (but not emotional music) seems to fulfill the rapid processing feature of automaticity as evidenced by a significant APE in the first experiment, it did not fulfill the unintentionality criterion as measured in the second experiment. Moreover, the presence of the N400 in the first study combined with its absence in the second, suggested that such rapid APE effects are caused by response level interference.

Thus, to summarize the results regarding automaticity of processing, the present thesis does find evidence for the unintentionality criterion of automaticity for emotional prosody perception, but only when prosody signals threat, as predicted by phylogenetically inspired models that propose a hard-wired neural system dedicated to the detection of social information that signals potential harm to the organism. Further, we find evidence for the rapid processing criterion of automaticity for happy and sad emotional prosody but not for affective music. Further, happy and sad prosody do not fulfill the unintentionality criterion of automaticity as measured with the affective priming paradigm. Thus, we do not find strong evidence for a dedicated system for the perception of emotional music. This (though somewhat tenuously) could be understood within the theory of evolution, if we assume that emotional music perception either had no fitness value or emerged too late in evolutionary history to prompt the evolution of a dedicated system. Also, we do not find evidence in favor of the 'super-expressive voices' hypothesis, as on the basis of this hypothesis we would have expected stronger affective priming effects for music than prosody. Further, the result that APEs for prosody do not persist when attention is diverted away from the affective dimension of the stimuli, suggests that automaticity for emotional prosody perception is *relative* (i.e. processes can be more or less automatic, but even relatively

strong automatic processes will cease to operate when attentional resources are sufficiently depleted), as has been suggested previously (Pessoa, McKenna, Gutierrez, & Ungerleider, 2002; Mothes-Lasch, Miltner, & Straube, 2012).

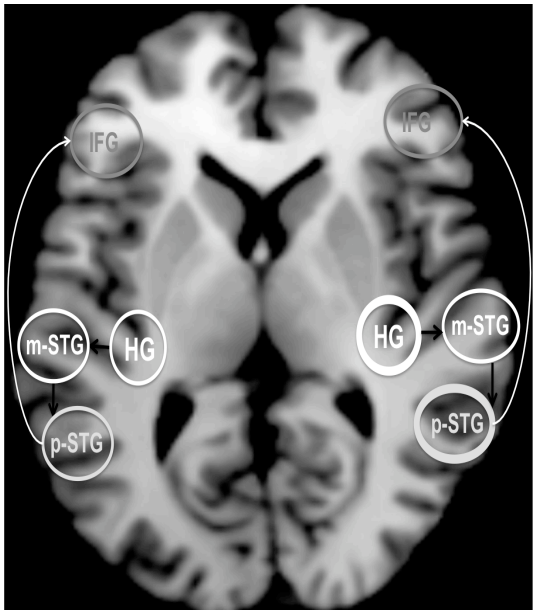

Figure 3. Final adaptation of the model presented in Figure 1 based on the insights gained from the present thesis regarding inter- and intrahemispheric models of prosody perception. White circles indicate the most likely neural substrate for stage-one, light grey circles for stage-two, and dark-grey circles for stage-three emotional prosody perception. Bold circles indicate right-hemispheric processing centers in the emotional prosody perception pathway where initial evidence has been found for greater activation likelihood as compared to their left hemispheric homotopes. For further details, see Figure 1.

4.2.3. Modulation of activity in the emotional prosody perception network by alexithymia

In the second empirical part of the present thesis, we asked whether the personality trait *alexithymia* might modulate processing within the emotional prosody perception network, and if so, whether relatively early (and possibly automatic) or relatively late emotional perception processes are associated with (normal variation in-) alexithymia.

In Chapter 3.1 we investigated whether alexithymia affects automatic processing of emotional prosody and music, as measured with the affective priming paradigm and concurrently recorded ERPs. Alexithymia did not significantly affect the behavioral APEs found during affective categorization. However, at the electrophysiological level alexithymia was associated with a reduced N400 component for affectively incongruent primes and targets. These results point to modulation by alexithymia of relatively automatic (and hence 'early') affective processing, without effects being evident at the behavioral level, possibly due to a higher level compensation mechanism.

In Chapter 3.2, we examined using fMRI whether alexithymia modulated the neural response to emotional prosody when attention was directed at emotional prosody and when attention was not directed at emotional prosody. Alexithymia did again not affect behavioral performance. However, on the neural level alexithymia was associated with a reduced response of the amygdala and the STG to emotional prosody, both when attention was directed to emotional prosody and when attention was diverted from the emotional prosody. Activation of the IFG, however, was not affected by alexithymia. Thus, alexithymia seemed to be associated with a relatively early stage of emotional prosody perception (stage-one and two processing in the STG) but not with a relatively late stage (stage-three processing in the IFG). Further, in keeping with the ERP results presented in Chapter 3.1, while modulation of automatic affective processing by alexithymia is evident at the neural level, it does not translate into effects at the behavioral level.

Together, then, these two studies suggest that (non-clinical) alexithymia primarily modulates relatively early emotional prosody perception stages. In the only other study on modulation of neural processing of emotional prosody by alexithymia, however, both modulation of relatively early and late ERP components was found during attended and unattended emotional prosody perception (Goerlich, Aleman, & Martens, 2012), without translating into effects at the behavioral level. In a recent meta-analysis of the neuroimaging literature on modulation of visual emotional processing by alexithymia (Van der Velde et al., 2013), it was found that alexithymia is associated with a *de*creased response of subcortical structures (such as the amygdala and the insula) but with an *in*creased response of the anterior cingulate cortex (ACC). The authors suggested that the decreased subcortical response might reflect disturbance of early emotional processing in individuals scoring high on alexithymia, while the increase in ACC activation may reflect a compensatory effort to allocate more attention to the emotional stimuli. Although admittedly speculative, this hypothesized primary early emotional deficit in combination with a higher order compensation mechanism may indeed explain the early emotional processing effects observed in this thesis, and why these effects do not translate into behavioral effects (due to higher level compensation). However, it should be noted that a limitation of the present research is that only modulation by normal (non-clinical) variation in alexithymia was studied and hence the conclusions may not generalize to clinical levels of alexithymia.

4.3. Summary of conclusions

We have started this concluding chapter by asking *what* network in the brain supports perception of prosody, *how* it does so, *whether* this network indeed sometimes processes prosody automatically, and if so, *why* this might be the case. Further, we asked whether alexithymia modulates processing in the emotional prosody network, and if so, whether early or late emotional processing is affected (or both).

The series of studies reported in the first empirical section of this thesis suggest that a bilateral temporo-frontal network comprising the HG, m-STG, p-STG and IFG analyzes prosody in at least three processing steps. Within this network, there is relative acoustic specialization of the right HG and p-STG for emotional prosody perception but no hemispheric specialization for linguistic prosody perception. Further, automatic processing of emotional prosody can indeed be demonstrated (but not for emotional music) and is particularly evident for anger prosody, suggesting that such automatic processing may be supported by a hard-wired neural substrate that has evolved to detect (social) threat in order to avoid harm to the organism.

Last, the studies reported in the second empirical section of this thesis confirmed that non-clinical variation in alexithymia modulates activity within the emotional prosody perception network. More specifically, alexithymia seems to primarily modulate early emotional prosody perception stages without translating into behavioral effects, pointing to a potential higher-level neural compensation mechanism.

4.4. Future directions

There are ample new avenues towards advancing our understanding of the cognitive neuroscience of prosody perception and its modulation by alexithymia. For instance, most of the neuroimaging literature to date has focused on emotional prosody perception. Future neuroimaging work could investigate to what extent the network identified in Figure 3 is also involved in various linguistic prosody functions. Regarding emotional prosody perception, it has hardly been directly tested yet whether there are indeed substantial structural and functional connections between the areas in Figure 3, and whether the information flow is indeed in the hypothesized directions. However, initial steps in this direction have been taken (Ethofer et al., 2013). Further, by combining the temporal resolution of ERPs and the spatial resolution of fMRI, it could be investigated whether the areas identified in Figure 3 are indeed active in early versus late stages of the prosody perception process as hypothesized. To gain knowledge regarding the *necessity* of the areas identified in Figure 3 for emotional and linguistic prosody perception, Transcranial Magnetic Stimulation (TMS) could be employed to investigate whether inhibition of activity in these areas does indeed degrade performance as hypothesized. Indeed, initial steps in this direction have been taken as well (Hoekert, Vingerhoets, & Aleman, 2010). Regarding automaticity of emotional prosody perception, future studies could investigate, with more emotional categories in addition to anger and with further reduced levels of attention to emotional prosody, how robust automaticity of emotional perception is and whether it is indeed specific for anger.

Last, concerning modulation within the prosody network by alexithymia, functional and structural connectivity studies may investigate whether alexithymia might be associated with altered structural connections between the ACC and early emotional prosody processing centers, and, using functional connectivity analyses, whether there

is altered control from the ACC over early emotional prosody processing centers along these pathways in alexithymia.

Such new directions will undoubtedly further advance our understanding of the cognitive neuroscience of prosody perception and its modulation by alexithymia.

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