

Sleep deprivation and intrinsic brain connectivity – a near-systematic review

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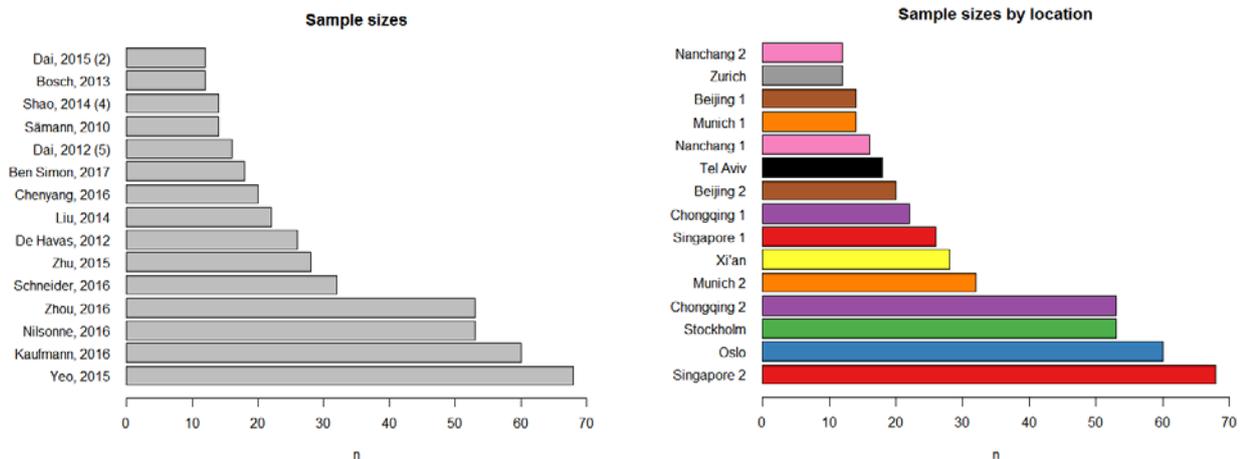
In the [Stockholm Sleepy Brain project](#), we have investigated the effect of partial sleep deprivation on intrinsic brain connectivity (i.e. functional connectivity in the resting state). What this means is that we took research participants, kept them awake, and then put them in the MR scanner. Once they were inside, we asked them to look at a fixation cross and do nothing else in particular for eight minutes. We measured blood flow with the MR scanner and then investigated the correlations between changes in blood flow between different brain areas. And then we compared that between the conditions when the participants were sleep deprived and well rested. The motivation was that this would shed new light on neurophysiological correlates of sleepiness. We have reported our findings as a [preprint](#).

Our manuscript does not allow space for a complete review of the earlier literature. Therefore, I am writing it here instead. Thus, the aim of this text is to review the published literature on the effects of sleep deprivation on intrinsic brain connectivity in humans.

Studies reported so far

Inclusion criteria for studies reviewed here were: resting state fMRI study comparing sleep deprivation to a control condition in humans. My search strategy was to query the PubMed database using the terms “sleep AND fMRI”, and assess the resulting records by title. I get weekly update e-mails with studies matching these search terms, and have kept adding studies to the list below; hence I believe it is up to date as of 2017-05-11. I have also added studies that were referenced from those included here. I have extracted information from the studies detailed in the table at the end. The assessment of records and assessment has not been checked by anyone else.

I have identified 22 published reports, based on what I believe are 15 unique datasets, ranging in size from 12 to 68 participants. Sometimes it was not easy to determine whether a dataset was unique (see determinations case-by-case below). Based on my preliminary appraisal, a total of 395 participants have been included in analyses in these reports, after excluding participants due to head motion and for other reasons. The following figures show the 15 datasets sorted by size. On the left, the datasets are labeled by author and year of the first publication, and the number of publications based on the same dataset is given in a parenthesis if > 1 . On the right, I have tentatively grouped the studies by location.



Data and code to make these plots are available at [GitHub](#).

I will now review these studies, grouped by location. I will pay particular attention to the risk of bias. There are three biases in particular, which to my mind are important in appraising studies in this area. The first is bias due to low statistical power (leading to increased risk of type 1 and type 2-errors and overestimation of effect sizes). The second is bias due to participants falling asleep during scanning. This is based on the assumption that we want to know from these experiments is what happens in the brain when people are sleepy, not when they are actually asleep. The two most important factors for this determination are whether participants were instructed to keep their eyes open or closed, and whether there was any monitoring to make sure they stayed awake. Thirdly, I will try to assess bias due to region selection. This is one instance of what can be known as researcher degrees of freedom, or forking paths, or undisclosed outcome switching. If a researcher reports analyses focusing on some particular brain area without a strong theoretical justification, then one wonders whether other brain areas were similarly investigated but the results not reported.

The Munich studies (#1, #19)

The first study of sleep deprivation and resting state connectivity was reported by Philipp Sämann et al. in 2010 (#1). Our research group is on friendly terms with Dr Sämann; among other things, he [gave a talk at a symposium we hosted in 2014](#). Study #1 included 16 participants who were scanned with eyes closed and no monitoring. The default mode network was identified with independent component analysis (ICA), and sleep deprivation was found to cause reduced connectivity within this network and reduced anticorrelation to the so-called anticorrelated network. This study was seminal and I believe it has been highly influential in the field. Several following studies have reported results consistent with these initial findings.

Study #19 originated from the same group in Munich and included 32 participants with mild partial sleep deprivation (2 h), undergoing resting state scans with eyes open and eye-tracking. This study is included here because resting state data were acquired after sleep deprivation, but the paper reports no analyses of functional connectivity. Instead, the study aimed to investigate activity related to pupil size.

Because study #1 used ICA as main approach, the main results were at low risk of region selection bias. Follow-up analyses used regions of interest selected based on inspection of ICA results, inflating the risk of bias somewhat. As may perhaps be expected from an initial study in a new field, sample size was small. Vigilance state was uncontrolled. I therefore conclude that study #1 was at high risk of bias. Study #19 is noninformative about functional connectivity.

The Nanchang studies (#2, #10-12, #18)

I have tentatively grouped these studies into two clusters because I believe they are based on two unique datasets. In the first cluster, Study #10 cites study #11, confirming the identity of the dataset. Study #2 is also grouped in this cluster, based on similarities in the descriptions of procedures and sample characteristics. This experiment included 16 participants who were scanned

three times: once after full sleep with acupuncture, once after sleep deprivation with acupuncture, and once after sleep deprivation with sham acupuncture (needle besides acupuncture point on right foot). Scanning was performed with eyes closed and wearing blinders.

Study #2 reported differences in amplitude of low-frequency fluctuations (ALFF), stratified by sex. Study #11, like study #2, reported differences in ALFF, and the results appear on inspection to agree fairly well with those from study #2, although study #2 reported results stratified by group only. Study #10 presented connectivity differences from the left posterior cerebellar lobe, with a lenient threshold of $p < 0.001$ for individual voxels and a cluster threshold of 10 voxels, finding small scattered foci in different cortical areas.

In most cases, an unbalanced intervention design, i.e. lacking the sham condition with full sleep, would be considered to introduce a high risk of confounding. It is not possible to separate effects of acupuncture from effects of full sleep. However, if one is skeptical towards effects of acupuncture on the central nervous system, then one must consider that the imbalanced design may not be a major problem.

The second cluster consists of studies #12 and #18. Here, 16 participants were scanned after full sleep and after 72 h total sleep deprivation, and 12 were analysed, mainly for ALFF again. Results do not agree well with those from studies #2 and #11. Both studies performed predictive modelling to attempt to classify data from the two conditions.

I conclude that these studies were all at high risk of bias due to low power and uncontrolled vigilance state. Study #10 was also at high risk of region selection bias. If the grouping is correct, the extensive reporting on the same dataset without making clear that the data are identical risks giving the impression that there is more evidence in the field of sleep deprivation and resting state connectivity than is actually the case.

The Singapore studies (#3, #15)

Study #3 analysed 26 participants with and without total sleep deprivation, with 8 minutes unmonitored eyes-open scanning. The default mode network and the anticorrelated (task-positive) network was identified using a pre-defined seed region in the PCC. From the resulting maps, 13 seed regions were selected. Sleep deprivation was found to cause reduced correlation between DMN nodes and reduced anticorrelation between DMN and CAN nodes.

Study #15 analysed 68 participants from three different datasets. Resting state data were acquired with different lengths of the runs, with eyes open, and monitored by eye-tracking. 114 regions of interest covering the whole gray matter were investigated. Sleep deprivation caused reduced connectivity within the DMN and attention networks, and reduced anticorrelation between DMN and attention networks. Additionally, increased global signal variability was reportedly observed after sleep deprivation, although no formal hypothesis testing is reported to support this interpretation; two time-courses from one participant are shown.

Study #3 is at high risk of bias due to low power, due to region selection (since regions were selected based on the same dataset), and due to unmonitored wakefulness. Study #15 is not at high risk of bias from any of these three sources. It is the largest, and in my estimation probably the most trustworthy and informative, of all the studies reviewed here.

The Zurich study (#4)

Study #4 included 12 participants, who were scanned after full sleep and one night total sleep deprivation, with concurrent fMRI and EEG. Thus, EEG allowed for excellent monitoring of wakefulness. A seed-based analysis was performed to study the functional connectivity from the PCC and “dorsal nexus” to the entire brain. Sleep deprivation reduced functional connectivity between the PCC and the bilateral ACC (BA 32), yet increased connectivity between the DN and two areas within the right DLPFC (BA 10). Seed regions were apparently a subset of previously used seeds from the same group. I conclude that the study was not at high risk of bias due to uncontrolled vigilance, but it was at high risk of bias due to low power and region selection.

The Beijing studies (#5-8, #20)

The Beijing studies #5-8 appear to be based on one dataset of 16 participants, scanned with eyes closed; monitoring was not mentioned. Studies #6 and #8 cite study #5, confirming the identity of the dataset, and study #7 is highly similar in design. Each study used its own preprocessing strategy, and the final number of analysed participants was 13 in two of the studies and 14 in the other two.

Studies #5-7 used seed region analyses including the thalamus and sub-regions of the amygdala. Study #5 claims to show increased anticorrelation of several regions to the thalamus following sleep deprivation. But this finding is supported by statistical tests only on regions of interest chosen after inspection of whole-brain results. This analysis approach greatly increases the risk of region selection bias. Study #6 compared connectivity maps between conditions and found some areas with increased connectivity and some areas with decreased connectivity to basolateral and centromedial amygdala. In case you wonder what happened to the superficial amygdala, the third major part, the answer is that those results are reported in study #7, in which the authors followed a similar procedure as in study #6, finding certain areas with increased and certain areas with decreased functional connectivity. Study #8 found that ICA-based DMN and salience network time-series were more strongly correlated after sleep deprivation. It is not clear why this particular network comparison was selected.

Study #20 was similar in design but appears to have been performed at a later date with a sample of 20 participants. Connectivity from the hippocampus was compared between conditions, and lower connectivity was found in scattered foci in the cortex.

I conclude that studies #5-8 and #20 are at high risk of bias due to low power, uncontrolled vigilance state, and region selection bias.

The Chongqing studies (#9, #13, #21)

Studies #9 and #13 appear to be based on the same dataset. 23 participants were sleep deprived for one night and underwent 5 min resting state imaging with no instructions about whether to keep eyes open or closed. Study #9 analysed time series from 160 regions covering the whole brain, thus protecting from region selection bias. However, I am not able to understand the results. The main finding was that anticorrelation decreased after sleep deprivation. This appears to be based on an analysis of 22 pairwise correlations, but I am unable to tell from the manuscript what these pairs are and how they were selected. There are 22 negative and 22 positive correlations drawn from the full matrix of 160 regions. While the regions are clustered into six major networks, only 15 pairwise correlations could be calculated between these networks, so that can't be it. The methods section describes that correlations were chosen for further comparison if they were significant at $p < 0.05$ in the full sleep condition, but in that case there ought to be more positive than negative correlations (according to figure 2), and the positive correlations ought to have a magnitude > 1 , while they are reported to be mainly in the range 0.2-0.3. Study #13 analysed connectivity between 13 DMN nodes, and found it to be lower following sleep deprivation.

In study #21, the same procedures for inclusion and scanning were used, except that monitoring by eye-tracking was added. The final number of participants included was 53. Thus, this is one of the largest datasets reported. Time courses were extracted from 264 areas covering the whole gray matter, and were clustered into 13 networks which were compared between conditions. Lower connectivity was found after sleep deprivation in the subcortical and the cerebellar network.

In conclusion, study #9 is at high risk of bias due to low power and uncontrolled vigilance state, and the results are beyond my comprehension. Study #13 is at high risk of bias due to low power and uncontrolled vigilance state, but in my opinion not at high risk of bias due to region selection. Study #21 is at low risk of bias from all three sources, and is in my opinion one of the most trustworthy and informative studies reviewed here.

The Xi'an study (#14)

This study included 28 participants, who were scanned for 7 minutes with eyes closed after full sleep and after 1 night of sleep deprivation. Voxel-mirrored homotopic connectivity (VMHC) was investigated. This is a measure of how much each voxel correlates to the contralateral voxel. Small areas in thalamus and cortex were seen with increased connectivity. I judge that this study was at high risk of bias due to low power and due to uncontrolled wakefulness, but not due to region selection.

The Stockholm study (#16)

This one is our own, to which I referred at the beginning of this post. We used 3 h partial sleep deprivation and resting state scanning for 2 x 8 minutes with eyes open, monitored by eye-tracking. We investigated several measures of connectivity, including ICA-derived networks, cross-correlation analysis following study #3, and seed-based analyses of amygdala and thalamus following studies #5-7. To reduce the risk of bias, we [published hypotheses](#) ahead of data analysis. With 53 participants included in analyses, we found effects in the same direction as reported in study #3, i.e. reduced connectivity within the default mode network and reduced anticorrelation between DMN nodes and the anticorrelated task-positive network. But the effects observed in our study were weaker and not statistically significant. We were not able to replicate results of studies #5-7.

The Oslo study (#17)

In this study, participants were scanned three times (morning, evening, next morning) with 8.3 minutes long resting state runs with eyes open. 41 participants were sleep deprived overnight and 19 were allowed to sleep as usual. ICA networks were estimated, and 19 out of 40 were determined to be meaningful. The authors then investigated correlations between these network components, and how these correlations were affected by sleep deprivation. This strikes me as unexpected, since connectivity within rather than between ICA components is more commonly investigated, and is easier to interpret. To the extent that network components are inversely correlated in the full sleep condition, reduced connectivity within components would be expected to lead to increased connectivity between components. The manuscript reports that 17 pairwise connections were affected by sleep deprivation, 13 of which changed to more positive correlations and 4, consequently, which changed in a negative direction.

Machine learning algorithms were tested and had some predictive ability. Interestingly, the classifier previously developed by Enzo Tagliazucchi to predict whether resting state scans were made while participants were awake or sleeping predicted a less than 50% likelihood that participants were awake while they were being scanned following sleep deprivation.

With 41 participants in the sleep-deprived group, I judge that the study was not at high risk of bias due to low statistical power. Since wakefulness was not monitored, it was at risk of bias due to uncontrolled vigilance state. But since ICA analyses covered the whole brain, the study was at low risk of region selection bias.

The Tel Aviv study (#21)

This study included 18 participants, who were scanned after one night TSD and after full sleep. Resting state runs were 6:50 minutes with eyes open, monitored by eye-tracking. Analyses were heavily graph theoretical. The main outcomes were modularity, which decreased after sleep deprivation, and participation, which increased after sleep deprivation. This would seem to be consistent with a general reduction in connectivity. I conclude that this study was at high risk of bias due to low power but not due to uncontrolled wakefulness nor region selection.

Assessment of findings

Much of the literature reviewed here has focused on the default mode network and its anticorrelated networks, variously identified as the anticorrelated network (ACN), the task-positive network (TPN), or the attention network(s). The finding that sleep deprivation reduces connectivity in the default mode network was first reported in study #1, and subsequently investigated in many of the following studies (#3, 4, 8, 12, 13, 15, 16, 17, 21, 22). Only a subset of these following studies (#3, 4, 13, 13, and 17) reported statistically significant findings supporting

that sleep deprivation caused reduced connectivity within the default mode network and/or reduced anticorrelation to the ACN/TPN. But all of the studies demonstrated effects in the same direction. Taken together, these studies in my appraisal provide strong and consistent evidence.

Other outcomes have been investigated by fewer of the studies. ALFF was investigated in four studies (#11, 12, 16 18), but findings were not consistent.

Biological interpretation

It is challenging to interpret the biological meaning of the findings reported in this literature. Many connectivity metrics (e.g. ALFF, ReHo) have unknown relationships to information processing in the brain. The studies reviewed here make interpretations that may be broadly classified as follows:

1. Observed connectivity differences may be responsible for the subjective experience of sleepiness or the behavioral effects of sleep deprivation (e.g. studies # 1, 5, 6, 7, 8, 21).
2. Observed connectivity differences may counteract the effects of sleepiness (e.g. study # 2).
3. Observed differences may represent a biomarker for sleepiness (e.g. studies #12, 17).
4. Observed connectivity differences are similar to another state, suggesting that sleep deprivation is an instance of that other state (study #17).

In study # 17 (Zhou 2016), the authors write: "we speculated that sleep deprivation is a simplified version of aging". This was based on the finding that aging was associated with reduced connectivity in 8 networks and sleep deprivation was associated with reduced connectivity in 2 of these 8 networks. This interpretation is however not easily reconciled with the dominant theory to explain age-related reductions in connectivity, which is that white matter tracts that transfer information between gray matter areas are damaged progressively, mainly from vascular causes. Age-related decline in connectivity appears, therefore, to be caused by long-term structural changes, whereas a decline in functional connectivity in sleep deprivation appears to be a reversible functional state.

A parsimonious explanation for observed findings in general among these papers is that participants fell asleep. Events of falling asleep may be associated with changed neural activity, head motion, changed breathing patterns, and changes in heart rate, all of which are expected to affect the measured signal. Most studies addressed these possible effects, especially head motion, and attempted to reduce their influence in different ways. One perspective on the risk that participants fell asleep is that effects may be confounded. Another perspective is that if sleepiness is a propensity to fall asleep, then this is what we are out to measure. Notably, episodes of falling asleep may be partial (involving only some regions of the brain) and brief, and are not necessarily always noticed by the participants themselves.

Where next?

The studies reviewed here provide strong and consistent evidence for effects of sleep deprivation on the connectivity of the default mode network. Several other outcomes have been investigated, but only in a relatively small number of the reviewed studies. Thus, there exist a number of proposed associations that require confirmation in additional datasets. Since a lot of data have been gathered, it would be possible in principle to test hypotheses on existing data, which would save a lot of resources compared to gathering new data from scratch.

Among the studies reviewed here, not one has published data openly. The data from the Stockholm Sleepy Brain project will be published at openfmri.org, and much of the metadata is [already available](#). None of the other studies mentioned data availability, except #12 (Gao et al. 2015), which gives an accession number to the [Harvard Dataverse](#). There is however [no dataset associated with this number](#).

Studies reviewed here

#	doi	Title	1st author, year	n analysed	RS paradigm	Sleep deprivation	Main analysis method and outcomes	Main results	Remarks
1	10.1007/s10334-010-0213-z	Increased sleep pressure reduces resting state functional	Sāmān, 2010	14	6 min, eyes closed, no monitoring	Partial sleep deprivation (PSD) with 3.5 h sleep.	- ICA with unrestricted number of components, 2 default mode network (DMN) components identified, anticorrelated network (CN) defined as	PSD caused decreased connectivity within DMN and ACN and between the two networks.	

#	doi	Title	1st author, year	n analysed	RS paradigm	Sleep deprivation	Main analysis method and outcomes	Main results	Remarks
		connectivity				scanning next evening at 19:00	negative connectivity to these components. - 25 ICA components and matching to a template. - Seed regions defined from same dataset's ICA for DMN and ACN.		
2	10.1016/j.sle.2011.09.019	Gender differences in brain regional homogeneity of healthy subjects after normal sleep and after sleep deprivation: A resting-state fMRI study	Dai, 2012	16	Time not specified, eyes closed, wearing blinders	Total sleep deprivation, scanning next evening at 19:00	Regional homogeneity (ReHo). Analysis stratified by sex (male/female).	Increased ReHo following SD mainly in frontal and parietal areas and mainly in females	Presumed same dataset as in studies 10 and 11
3	10.1016/j.neuroimage.2011.08.026	Sleep deprivation reduces default mode network connectivity and anti-correlation during rest and task performance	De Havas, 2012	26	8 min, eyes open, no monitoring	Total sleep deprivation, scanning in the morning of the deprivation night at about 05:00	Analysis of default mode network and anticorrelated network	SD was associated with reduced connectivity within DMN and anticorrelation between DMN and ACN.	
4	10.1073/pnas.1317010111	Sleep deprivation increases dorsal nexus connectivity to the dorsolateral prefrontal cortex in humans	Bosch, 2013	12	8 min, eyes closed, monitored by 32-channel EEG	Partial sleep deprivation with 3h42min mean sleep time in deprivation condition, starting about 3 AM, scanning next evening	A seed-based analysis was performed to study the functional connectivity from the PCC and "dorsal nexus" to the entire brain	Sleep deprivation reduced functional connectivity between the PCC and the bilateral ACC (BA 32), yet increased connectivity between the DN and two areas within the right DLPCF (BA 10)	Seed regions said to be used on previous work (http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0044799). This previous paper however also includes rois in amygdala and subgenual ACC.
5	10.1371/journal.pone.0078830	Decreased Thalamocortical Functional Connectivity after 36 Hours of Total Sleep Deprivation: Evidence from Resting State fMRI	Shao, 2013	14	6 min 36 s, eyes closed	36 h TSD, scanning at 08:00 in the morning	Thalamus defined using WFU pickatlas. Whole-brain results compared between conditions.	After TSD, multiple temporal and prefrontal regions were negatively correlated to thalamus seed region.	Studies 5-8 based on same dataset
6	10.1371/journal.pone.0112222	Altered Resting-State Amygdala Functional Connectivity after 36 Hours of Total Sleep Deprivation	Shao, 2014	13	6 min 36 s, eyes closed	36 h TSD, scanning at 08:00 in the morning	Basolateral (BLA) and centromedial (CMA) amygdala defined from Jülich atlas. Whole-brain results compared between conditions.	TSD caused changed connectivity mainly to ACC and PCC.	Studies 5-8 based on same dataset
7	10.1002/jnr.23601	Altered superficial amygdala-cortical functional link in resting state after 36 hours of total sleep deprivation	Lei, 2015	14	6 min 36 s, eyes closed	36 h TSD, scanning at 08:00 in the morning	Superficial amygdala (SFA) defined from Jülich atlas. Whole-brain results compared between conditions.	L SFA: TSD caused decreased connectivity to R dorsal PCC, L thalamus, R dorsal ACC, R middle occipital gyrus, L culmen. R SFA: TSD caused increased connectivity to R medial PFC and decreased connectivity to R dorsal PCC. Amygdala-mPFC connectivity correlated to POMS scores.	Studies 5-8 based on same dataset
8	10.1371/journal.pone.0133959	Large-Scale Brain Network Coupling Predicts Total Sleep Deprivation Effects on Cognitive Capacity	Lei, 2015	13	6 min 36 s, eyes closed	36 h TSD, scanning at 08:00 in the morning	ICA followed by "As a measure of cross-network coupling, we calculated absolute values of Pearson correlation coefficients (CC) between component time courses derived from the SN, ECN, and the DMN (CCSN, DMN and CCSN, ECN) to represent the coupling strength between different networks."	Increased salience-DMN correlation after TSD. This correlation was also weakly associated with VAS sleepiness rating and with reaction times in a working memory test.	Studies 5-8 based on same dataset
9	10.1111/jsr.12147	Enhanced brain small-worldness after sleep deprivation: a compensatory effect	Liu, 2014	22	5 min, no specific instructions	34 h TSD, scanning in afternoon or evening	Correlation matrix of 160 brain regions	"strength of the average positive functional connectivity tended to increase after SD, and the negative correlation was significantly less suppressed"	Presumed same dataset as in study 13 22 pairwise correlations selected? Unclear.
10	10.2147/NDT.S84204	Connectivity pattern differences bilaterally in the cerebellum posterior lobe in healthy subjects after normal sleep and sleep deprivation: a resting-state functional MRI study	Liu, 2015	16	Not specified	Total sleep deprivation, scanning next evening at 19:00	Cross-correlation analysis with seeds determined from functional activation during acupuncture	Small scattered foci at lenient threshold of voxel-wise $p < 0.001$	Presumed same dataset as in studies 2 and 11
11	10.1371/journal.pone.0120323	Frequency-Dependent Changes of Local Resting Oscillations in Sleep-Deprived Brain	Gao, 2015	16	Not specified	Total sleep deprivation, scanning next evening at 19:00	ALFF and ReHo in whole brain	SD caused increased ALFF in occipital and sensorimotor areas, decreased in mPCF, dIPCF, and inferior parietal lobule. SD caused increased ReHo in SMC and visual cortex.	Presumed same dataset as in studies 2 and 10
12	10.2147/NDT.S78335	Long-term total sleep deprivation decreases the default spontaneous activity and connectivity pattern in healthy male subjects: a resting-state fMRI study	Dai, 2015	12	Not specified	72 h TSD	ALFF and DMN connectivity	SD caused reduced connectivity in small foci of the DMN, and different ALFF in some areas.	Presumed same dataset as in study 18
13	10.1016/j.brainres.2014.04.019	Module number of default mode	Wang, 2014	22	5 min, no monitoring	34 h TSD, scanning in	Connectivity between DMN seed regions	SD caused reduced connectivity	Presumed same dataset as in study 9

#	doi	Title	1st author, year	n analysed	RS paradigm	Sleep deprivation	Main analysis method and outcomes	Main results	Remarks
	11.007	network: Inter-subject variability and effects of sleep deprivation				afternoon or evening			
14	10.1007/s11682-015-9490-5	Increased interhemispheric resting-state functional connectivity after sleep deprivation: a resting-state fMRI study	Zhu, 2015	28	7 min, eyes closed	One night TSD	Voxel-mirrored homotopic connectivity (VMHC)	SD caused increased VMHC in thalamus and some cortical areas	
15	10.1016/j.neuroimage.2015.02.018	Functional connectivity during rested wakefulness predicts vulnerability to sleep deprivation	Yeo, 2015	68	Varying lengths, eyes open, eye-tracking	One night TSD	Connectivity between 114 regions of interest	Sleep deprivation caused reduced connectivity within the DMN and attention networks, and reduced anticorrelation between DMN and attention networks.	
16	10.1101/073494	Intrinsic brain connectivity after partial sleep deprivation in young and older adults: results from the Stockholm Sleepy Brain study	Nilsson, 2016	53	2 x 8 minutes, eyes open, eye-tracking	3 h PSD	Analysis of ICA-derived networks, replicatory analysis of specific seed regions, ALFF, global signal	Increased global signal variability, no other notable effects	Our own study; published as preprint
17	10.1016/j.neuroimage.2015.12.028	The brain functional connectome is robustly altered by lack of sleep	Kaufmann, 2016	60	8.3 min, eyes open	Between-group design with 41 sleep deprived (one night TSD) and 19 non-deprived	ICA and subsequent analysis of connectivity between (not within) components. Classification by machine learning.	Several network intercorrelations with effects of sleep deprivation.	
18	10.2147/NDT.S99644	Sleep deprivation disturbs regional brain activity in healthy subjects: evidence from a functional magnetic resonance-imaging study	Wang, 2016	12	Not specified	72 h TSD	ALFF	SD caused different ALFF in some areas.	Presumed same dataset as in study 12
19	10.1016/j.neuroimage.2016.06.011	Spontaneous pupil dilations during the resting state are associated with activation of the salience network	Schneider, 2016	32	2x12 min eyes open, monitored with eye-tracking	PSD with 2h earlier wake-up time than usual; in-home setting	Main effects of sleep deprivation were not analysed. Activity related to pupil size was investigated.	Increase in pupil size was associated with activity in ACC, insula, dIPFC, and other areas.	
20	10.1007/s11682-016-9570-1	Short-term memory deficits correlate with hippocampal-thalamic functional connectivity alterations following acute sleep restriction	Chenyang, 2016	20	Not specified	TSD	Connectivity from hippocampus seeds was compared between conditions	SD caused mainly reduced connectivity to scattered foci in cerebral cortex	
21	10.1089/bra-in.2016.0452	Sleep deprivation make the young's brain like elderly: A large-scale brain networks study	Zhou, 2016	53	5 min, eyes open, eye-tracking	TSD 36 h	Time courses extracted for 264 areas covering the whole brain, clustered into 13 networks. Connectivity within these networks compared between conditions	Sleep deprivation caused reduced connectivity in subcortical and cerebellar networks.	
22	10.1002/hbm.23596	Tired and Misconnected: A Breakdown of Brain Modularity Following Sleep Deprivation	Ben Simon, 2017	18	6:50 min, eyes open, eye-tracking	One night TSD	Modularity index and participation coefficient	SD caused reduced modularity and increased participation	

This text was originally posted at http://nilsonne.net/2017/05/11/sleep_deprivation_and_connectivity/