character of this gap will undergo constant change. At our Center at the Karolinska Institutet, scientists have been instrumental in developing international databases: 1. GeneLynx (Wasserman et al.) which is a catalog of human genes with links to the internet. 2. Pfam (Sonnhammer et al.) which is a collection of protein domains. 3. UGBASE (Brookes et al.) which is a curated collection of human single nucleotide polymorphisms (SNPs). 4. RiboTag (Wahlestedt et al.) which is a growing catalog of acessible (binding) sites on RNA for functional genomics purposes.

S46.4

HUBIN – Human Brain Informatics: a database project on schizophrenia

H. Hall¹*, T. McNeil², S. Arnborg³, L. Terenius¹, G. Sedvall¹. ¹Karolinska Institute, Stockholm; ²Lund University; ³Royal Institute of Technology, Sweden

To explore the etiology and pathophysiology of schizophrenia, the HUBIN project - Human Brain Informatics Center - has been established at the Karolinska Institute. This study combines molecular genetic, psychiatric, physical, brain imaging and perinatal risk data in a relational database. Major HUBIN studies are conducted on a large Swedish national sib pair material of schizophrenia and on a case-control material of patients with schizophrenia. Data on more than 2000 clinical and biological variables from more than 1000 subjects have been entered into the database. Data mining procedures are used to search for relations between variables from patients and volunteers. The data will be used to classify subgroups within the schizophrenia materials, and reveal new information regarding genetic and environmental mechanisms for the etiology and pathophysiology in the group of schizophrenia patients. The database will also be used for a detailed characterization of the variability of a large number of entities of importance for the human brain and its functions in relation to health and psychiatric disorders.

S46.5

A brain database: architectonics, receptors, functional imaging

K. Zilles¹*, K. Amunts¹, S. Geyer², A. Schleicher², H. Mohlberg¹. ¹University of Düsseldorf, Research Center Jülich; ²University of Düsseldorf, Germany

The increasing number of projects on human brain databases demonstrates the growing need for such tools in neuroscience. In addition to in vivo imaging studies, post mortem data are a mandatory component of brain databases.

Nissl- or myelin-stained sections through complete human brains were analysed using a novel observer-independent technique for architectonic cortical mapping. Additionally, whole brain cryosections were processed using quantitative in vitro receptor-autoradiography. The regional and laminar distributions of 15 receptor types were quantified. An elastic multigrid deformation was used to warp individual post mortem brains, microscopically defined areas, receptor patterns, and results of structural and functional in vivo studies to a common spatial reference brain. This enabled the establishment of probabilistic distributions of cortical areas. This strategy provides new insights into the concordance or discordance micro- and macroscopic structure and function. Examples are described for the normal adult human brain and for patients with neurological and psychiatric diseases.

Supported by: DFG, Human Brain Project (P20-MHDA52176); the National Institutes of Mental Health, National Institute for

Drug Abuse, National Cancer Institute and National Institute for Neurological Disease and Stroke.

S47. Sleep and psychiatric disorders

Chairs: J. Hetta (S), B. Appelberg (FIN)

S47.1

Sleep physiology and behavior

T. Åkerstedt*. IPM & Karolinska Institute, Stockholm, Sweden

The polysomnographical description of sleep shows an oscillation between medium and low frequency EEG activity. Starting with the high frequency EEG of wakefulness, brain activity progressively decreases from stage 1, to stage 2 to stage 3 to stage 4 (Slow Wave Sleep), and then an abrupt change to Rapid Eye Movement Sleep (REM). This sequence is repeated 4–5 times with less SWS and more REM. The function of sleep is only partially known, but the last decade has provided new knowledge indicating major metabolic and immunological changes during sleep.

Sleep loss will essentially increase SWS, and decrease Stage 2, Stage 1, and REM sleep during recovery sleep. SWS is clearly given priority. Experimental partial sleep loss also leads to a reduced insuline response to glucose and to a reduced glucose clearance. Experimental studies sleep and memory suggest that SWS/GH (and HPA hippocampal inhibition) are involved in the formation of declarative memory, while REM/cortisol are involved in the formation of procedural memory. Furthermore, relations between the immune system and sleep are now intensively examined.

S47.2

Sleep disturbances in depression

M. Berger*. Department of Psychiatry and Psychotherapy, University Hospital of Freiburg, Germany

Polysomnographic sleep studies revealed REM-sleep abnormalities as specific for depressive disorders. This is especially true for the results of a cholinergic REM-induction-test (CRIT) with RS86. Only depressed patients show a shortening of REM-latency and an increase of REM-density. The sensitivity of these abnormalities is about 70-80%. Regarding the unspecific disturbances of sleep continuity in depression there seems to be a bidirectional relationship between insomnia and depression. That means depression is not only linked with insomnia, but chronic insomnia also increases the risk to develop a depressive disorder. Therefore adequate treatment of insomnia also seems to be relevant to prevent depression. The third part of the presentation will focus on the interesting topic, that sleep deprivation pocesses antidepressive potency and that sleep during the second half of the night exerts depressogenic properties. Own results revealed that the combination of sleep deprivation with consecutive sleep phase advance is a useful strategy to bridge the gap between the onset of an antidepressive medication and its antidepressive effect. Finally the biochemical background of sleepwake-regulation in normals and in depressives in regard to the adenosine system will be discussed.