NEUROCHEMISTRY

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Introduction

The title of the chapter, neurochemistry is not contemporary in nature, at a time when multidisciplinary approach to any subject area is in the vogue. We have carefully considered the existing literature and tried the best to include all the relevant material published from India during the past two decades on chemical processes in the neural tissue. Reports which encompass solely pharmacology, toxicology, physiology, behavior, and anatomy of the brain were painstakingly mined for valuable information contained therein in terms of chemistry of the brain in its true value and added herein. Last one decade saw a plethora of activities in the field of neurogenetics in India, in relation to neurological, psychiatric, neurodegenerative and developmental disorders. Since a separate chapter for neurogenetics was not planned in this volume, information made available in this upcoming field during the past two decades has also been included in this chapter.

The time is ripe with a lot of enthusiasm for search of truth on how the brain works in chemical terms, and with a lot of sophisticated technologies to back, the focus being shifted from simple higher functions of the brain such as thought processes and memory retention, to more complicated super level functions such as mind and consciousness. Two decades ago a similar volume listed about eight and odd centers in whole of India engaged in neurochemistry research, which today accounts for similar numbers, but for each metropolitan city alone, with several more small cities and towns adding one or more research centers on CNS to the list. Private medical institutions contributed a lot to this increasing numbers recently, but with little output. At this juncture it is interesting to mention that even philosophical institutions like Sree Ramakrishna Institute of Culture, Kolkata has already organized two international conferences on brain, mind and consciousness, with participation from neurochemists, neurophysiologists and molecular biologists brushing shoulders with philosophers and monks with deep traditional knowledge on the powers of mind and consciousness. Therefore it was not surprising that many sessions were discussing functional MRI and superbly controlled experiments on premonitions, dreams, subconscious actions, and near death experiences of individuals resuscitated from clinically dead conditions.

We have tried to include all the relevant materials and for the purpose we have resorted to a systematic search in the public library of medicine database, and also requested all the members of the Society for Neurochemistry (India), and the Indian Academy of Neurosciences to send in their contributions in neurochemistry during the past twenty years. To the latter request many complied, and we have included all the pertinent information received from these two sources.

Protein metabolism and gene expression

Neural development

Studies on tubulin gene expression in brains from three different species viz., rat, mouse and chick, varying with respect to their periods of synaptogenesis, show that the rise and fall in the level of tubulin during synaptogenesis is closely parallel to that of tubulin mRNA (Bhattacharya and Sarkar, 1991). Significant increase in the expression of cerebral NF-M, NF-L and NF-H mRNA level, a decrease in expression of NF proteins during the first 3-4 postnatal weeks of rat brain development has been found (Ghosh et al, 1999).

Poddar et al (1996) have investigated the molecular mechanism of thyroid hormone action on actin and tubulin gene expression in the developing rat brain, and shown that the developmental profile of these cytoskeletal mRNAs in both normal and hypothyroid brains display a biphasic pattern, increasing progressively during the first week after birth and declining thereafter. The relative sensitivity of the two isotypes of actin mRNA (beta and gamma) to thyroid hormone was examined (Sarkar et al 1997) in cerebra from normal and hypothyroid rats of different ages, covering the period of synaptogenesis, and has been shown that hypothyroidism is associated with a reduction in the steady state level of beta-actin mRNA but levels of gamma-actin mRNA were reduced only during the early phase of synaptogenesis. It has been further shown that thyroid hormones regulate the expression of actin gene by stimulating the rate of synthesis as well as intracellular distribution of actin during the mid phase of the second trimester of gestation (Pal et al 1997). T3 has been shown to stimulate actin mRNA by acting at the level of transcription, whereas tubulin mRNA is enhanced primarily by post-transcriptional regulation in the cerebral cortex (Sarkar et al 1999). An iodine-deficient rat model was studied for the pattern of T3 receptor genes expression in the brain during postnatal development, and reported that an increase in expression of c-erbA-alpha 2 and -beta 1 T3 receptor transcripts while c-erbA-alpha-1 remains unchanged in the brain of the iodine-deficient neonates compared with their euthyroid counterparts (Chattopadhyay et al 1995).

The levels of isoforms of Na-K-ATPase mRNA have been found to increase significantly during the first three postnatal weeks representing the critical period of synaptogenesis and myelination (Chaudhury et al 1996). Further investigations revealed that transcription of all alpha mRNA isoforms of Na^+K^+-ATPase were sensitive to T3 and at post-transcriptional level T3 enhanced the half life of alpha-3 mRNA by 1.5-fold with no discernible effect on alpha-1 and alpha-2 mRNA (Bajpai and Chaudhury, 1999). Glial cells as the target cells for the regulation of Na^+K^+-ATPase by T3 in the developing brain has been established since glial cells of developing rat brain showed progressive increase in the expression of the enzymes four isoforms (alpha-1, alpha-2, beta-1 and beta-2) (Banerjee and Chaudhury, 2001).

Hypothyroidism has been shown to be linked to cerebral cortex development by regulating apoptosis through the caspase pathway involving Bax, Bcl-2 and Bcl-x(L), linked to pro-nerve growth factor/p75 neurotrophin receptor (Kumar et al 2006b). It has been found that T3 plays a positive role in maintaining glutathione homeostasis in astrocytes and in protecting the brain from oxidative stress by increasing the activity of glutamate cysteine ligase (Dasgupta et al 2007).

Enzymes

A series of studies conducted in India have brought to light presence of distinct cytochrome P-450 enzymes in the brain that are different from well-characterized hepatic forms, and their role in the metabolism of xenobiotics and drugs in the brain by pathways different from those known to occur in liver. The first report from this series claimed localization of cytochrome P-450 dependent monooxygenases in neuronal and glial cells, and their inducibility and specificity in the brain (Dhawan et al 1990). Phenobarbital has been found to induce P450 2B1/2B2 in brain microsomes which specifically catalyzed the O-dealkylation of 7-pentoxy- and 7-ethoxyresorufin (Parmar et al 1998; Dhawan et al 1999). Tirumalai et al (1998) have demonstrated the diversity in the distribution of cytochrome P450 and associated mono-oxygenase activities in brain which may reflect the differential capability of various regions of the brain to detoxify or bioactivate diverse xenobiotics. Constitutive expressions of several major cytochrome P450s, which were induced to the administrations of drugs such as ethanol (P4502E1), phenobarbital (P4502B), and several psychoactive drugs (P4502D6 and P4502D1), their activities and differential induction in rat brain regions by several drugs including ethanol and phenobarbital, and their topographic distribution in rat and human brains have been demonstrated later (Upadhya et al 2000; 2002; Chinta et al 2002; 2005a).

The expression and induction by ethanol of cytochrome P4502E1 in rat cerebellum and hippocampus, but not in other brain regions, and its presence in astrocytes and glia indicated that this enzyme may be involved in the regulation of ethanol induced free radical damage and neuronal degeneration (Yadav et al 2006a; Kapoor et al 2006a). CYP1A1, a cytochrome P-450 enzyme, bioactivates polycyclic aromatic hydrocarbons has been shown to localize predominantly in neurons of cerebral and granule cell layers of cerebellum, and pyramidal neurons of CA1, CA2, and CA3 subfields of the hippocampus (Chinta et al 2005b). The expression of CYP1A1/1A2 isoenzymes in the primary cultures of rat brain neuronal and glial cells revealed significant difference in the activity of CYP1A-dependent 7-ethoxyresorufin-o-dealkylase (EROD) in microsomes prepared from both rat brain neuronal and glial cells (Kapoor 2006b). Kommaddi et al (2007) provided evidence that a unique P450 enzyme, generated by alternate splicing in a histiospecific manner can modify genotoxic potential of carcinogens such as benzo(a)pyrene by altering their biotransformation pathway. It has also been shown that constitutive mRNA and protein expression of cytochrome P4503A in brain may not only be

involved in the process of detoxication mechanism but also in the metabolism of endogenous substrates in the brain (Yadav et al 2006b).

Certain enzymes in the brain have been implicated in regulating the damage control in toxic events. Thioltransferase (glutaredoxin), member of the family of thiol-disulfide oxido-reductases that maintain the sulfhydryl homeostasis in cells, has been localized in neurons of different brain regions, such as cerebral cortex, purkinje and granule cell layers of the cerebellum, granule cell layer of the dentate gyrus and in the pyramidal neurons of CA1, CA2 and CA3 subfields of hippocampus (Balijepalli et al 2000). Glutaredoxin has been shown to mediate recovery of complex I by regenerating protein thiols utilizing reducing equivalents of glutathione (Kenchappa and Ravindranath, 2003). Usha et al (2000) have demonstrated a region-specific regulation of trypsin-like proteases and caspase-3 activation in the brain, to control the regulation of apoptosis following neurotoxic insults. Similarly, mitochondrial glutaredoxin has been shown to transiently get up-regulated in midbrain and striatum after neurotoxic insults (Karunakaran et al 2007). Cu-Zn-superoxide dismutase, an important enzyme in the detoxification of reactive oxygen radicals, has been shown to be up-regulated by the well-known monoamine oxidase-B inhibitor and antiparkinsonian drug, L-deprenyl (selegiline; Saravanan et al 2006). Interestingly voltage activated calcium channel activity has been shown to positively influence monoamine oxidase activity in the striatum of rodents, significantly influencing the levels of dopamine (Samataray et al 2003). Golden hamsters were found to have very low activities of monoamine oxidase-A and -B, and have been shown to be resistant to certain neurotoxins, that are to be metabolized into an active form through these brain enzymes (Mitra et al 1994).

The role of monoamine oxidase type B has been shown preferentially, if not exclusively to metabolize dopamine in certain discrete regions of monkey brain (Lakshmana et al 1998). Age related changes in the antioxidant defense enzymes in brain have been reported by Sahoo and Chainy (1997), and it has been demonstrated that thyroid hormone influenced the antioxidant defense parameters of rat brain (Das and Chainy, 2004). Studies on the role of glial cells in the management of oxidative stress indicated that glial cells have a greater poteintial in combating oxidative stress as compared to neurons since they have enriched antioxidant enzymes (Khanna and Nehru, 2007).

Lipid metabolism

Phospholipid and cholesterol

Many of the studies on lipid metabolism in the brain from India during the past couple of decades arise from neurotoxicological studies, which are addressed separately elsewhere in the book. Two examples of toxin-induced defined human disorders are available and are briefed below. Diabetes-induced by alloxan accompanied a decrease in contents of total phospholipids in microsomes and increase in both phospholipids and cholesterol in the mitochondria, which responded

to insulin treatment (Patel and Katyare, 2006). A significant decrease was observed in the cholesterol, phospholipids, sphingomyelin, phosphatidylserine and phosphatidylethanolamine content of myelin (Swapna et al 2006a), that accompanied structural disarray in myelin membrane (Swapna et al 2006b) in thioacetamide-induced hepatic encephalopathy in rats.

Influence of diet

Diet and fatty acid metabolism in the brain was one of the major areas covered during the past two decades. It has been reported that iron deficiency could lead to alterations in the functions related to phosphoinositide-linked receptor system, including phosphatidylinositol, phosphatidylinositol-4-phosphate and phosphatidylinositol-4,5-bisphosphate, the precursors for the second messengers, diacylglycerol and inositol phosphates. Iron supplementation for several days could restore these functions in the brain (Mohindra et al 1990). It has been shown that depending on the nature of dietary fat, the fatty acid composition of subcellular membranes is altered, which in turn could regulate the activity of membrane-bound enzymes that are vital for brain function (Srinivasarao et al 1997). Following protein deficient diet phosphatidyl choline contents was shown to be increased, phosphoinositides turnover reduced and phosphorylation of proteins increased markedly, suggesting that dietary protein deficiency causes alterations in transmembrane signaling mechanism in rat brain (Bansal et al 2000). Diet of sodium has been shown to affect the lipid content differentially in various parts of the mouse brain (Kaur et al 2003).

Neurotransmitters and their receptors

It has been demonstrated that polyunsaturated fatty acids mediated decrease in dopamine and serotonin in hypothalamus and cerebral cortex could be corrected by thyroid hormone administration (Varghese et al 2001). It is reported that long-term use of the dopamine receptor agonists can effect the organization of synaptic membrane by inhibiting the biosynthesis of all major phospholipids including phosphatidyl inositol (Singh and Shankar, 1996). Seasonal variation in the membrane fluidity, membrane lipid components, fatty acid composition of membrane phospholipid, positional distribution of fatty acids at Sn-1 and Sn-2 position of phosphatidylcholine and phosphatidylethanolamine has been reported in myelin, synaptosomes, and brain mitochondria of a tropical air breathing teleost, *Clarias batrachus* (Roy et al 1997).

Studies on serotonin-1A [5-HT(1A)] receptor provided evidence that cholesterol is necessary for ligand binding and G-protein coupling of this important neurotransmitter receptor (Pucadyil and Chattopadhyay, 2004). The same group has demonstrated modulatory role of cholesterol on the ligand binding of the bovine hippocampal 5-HT(1A) receptor by cholesterol complexation in native membranes (Paila et al 2005). The reduction of membrane cholesterol significantly attenuates the antagonist-binding function of the serotonin-1A receptor (Pucadyil and

Chattopadhyay, 2005), and oxidation of membrane cholesterol significantly inhibited the specific binding of the agonist and antagonist to 5-HT(1A) receptors (Pucadyil et al 2005). These results comprehensively demonstrated the importance of cholesterol in the serotonin-1A receptor function and formed the basis for understanding lipid-protein interactions involving this important neuronal receptor.

Glycolipids

Sugar-specific binding of bovine brain (14 kDa) galactose-binding lectin (BBL) to individual endogenous gangliosides (GM1, GM2, GD1a, GD1b and GT1b) was studied and the results indicated that a terminal sialic acid moiety covering a galactose moiety may at times enhance BBL recognition of the latter, and that changes in ganglioside pattern is a possible modulator of lectin function in vivo (Kannan and Appukuttan, 1997). The developmental profiles of sialidase, beta-galactosidase, beta-hexosaminidase and beta-glucosidase were compared to those of the gangliosides in rat brain and spinal cord and the rapid phase of increase in enzyme activities between 0-7 and 14-21 days and a decrease thereafter was found to be consistent with the turnover rate of gangliosides, which in rat brain is reported to be the highest between 10 and 20 days (Prasad, 1996).

Teratogenic effects of maternal alcohol consumption on cell number, gangliosides and ganglioside-catabolizing enzymes in the central nervous system of the offspring have been investigated. It has been demonstrated that there was significant increase in the concentration of total ganglioside, N-acetyl-neuraminic acid and in the proportions of individual ganglioside fractions in CNS of the pups, which was, at least partly, due to the decreased activities of ganglioside catabolizing enzymes (Prasad, 1992). Similarly, maternal protein deficiency caused significant cell loss in cerebrum, cerebellum, brain stem and spinal cord of pups at weaning, which was accompanied by significant region-specific changes in the proportions of individual gangliosides, due to higher activities of sialidase, beta-galactosidase, beta-glucosidase, and beta-hexosaminidase, which are involved in the catabolism of gangliosides (Prasad, 1991).

The functional significance of ammonia production in the brain in relation to neuronal membrane gangliosides has been investigated, and reported an increase in the content of gangliosides along with a rise in the content of GD1A and GD1B, without any change in beta-galactosidase and N-acetylhexosaminidase in cerebral cortex, cerebellum, and brain stem (Modi et al 1994). Insulin sensitive increase in free fatty acid, gangliosides and lipid proxides were found in the brains of diabetic animals (Kumar and Menon, 1993).

Aging

Sex steroids and signaling

Estrogen receptor (ER) level decreases in the cerebral cortex of old female mice and is downregulated by estradiol. The level of ERá is significantly higher than that of ERâ (Sharma and Thakur, 2006; Thakur and Sharma, 2007). The binding of transcription factors to promoter and co-regulators play a key role in estrogen action (Thakur and Ghosh, 2008; Thakur and Sharma, 2007; Asaithambi et al 1997). The level and synthesis rate of androgen receptor (AR) decrease but its phosphorylation increases in the cerebral cortex with advancing age (Mukherjee et al 1999; Thakur et al 2000b). AR mRNA expression and its core promoter methylation are inversely regulated by testosterone and estradiol in adult mice (Kumar and Thakur, 2004a,b). The binding of 40kDa nuclear protein to AR promoter decreases in old (Thakur and Kumar, 2007). Taken together, such age-related changes in AR might lead to reduced responsiveness of androgens in old age (Thakur et al 2000a,b). The expression of norbin (Mani et al 2001), a neurite-outgrowth-related brain protein, is also age- and sex-specific. The expression of PS1 (Ghosh and Thakur 2008a) and PS2 (Ghosh and Thakur 2008b) protein and mRNA is modulated by age, sex and sex steroids.

Reproductive aging in females is marked by distinct stages characterized by alterations in the secretion of pituitary-gonadal hormones and development of mammary tumors, autoimmune diseases, etc. Involvement of factors that influence these changes in the hormone release are investigated and reported that norepinephrine and dopamine in the medial preoptic area and arcuate nucleus of the hypothalamus play a crucial role in the surge in luteinizing hormone while a reduction in DA release augmented the secretion of PRL, which were dependent on the age-related decline in these parameters or their synthetic machinery (MohanKumar et al 1994; 1997; ThyagaRajan et al 1995).

Nucleic acid metabolism

RNA polymerase activities were found to be decreased in the nuclear fractions of neuronal, astroglial, and oligodendroglial cells obtained from rat cerebral cortex of young, adult, and old ages (Venugopal and Rao, 1991). An alkaline endodeoxyribonuclease (Venugopal and Rao, 1993) and a deoxyribonuclease (Suvarchala and Rao, 1994) from young and aging rat brain were purified and found to be decreased with age. Altered conformation and increased strand breaks in neuronal and astroglial DNA of aging rat brain has also been demonstrated (Bhaskar and Rao, 1994). It has been shown that DNA-damage accumulates in neurons, and the cerebral cortex is the most vulnerable region (Mandavilli and Rao, 1996). Age-dependent changes and regional distribution of topoisomerase II alpha and beta (Kondapi et al 2004), and DNA-polymerase alpha, beta, delta and epsilon activities in isolated neuronal and astroglial cell fractions from developing and aging rat cerebral cortex have been examined (Raji et al 2002). The results indicated

that while DNA-polymerase alpha and delta/epsilon has some role in long patch base excision repair and in other modes of DNA repair, topoisomerase II beta activity in ageing may contribute to the genomic instability in the ageing brain.

DNA repair

Age-related decline in nonhomologous end joining activity in rat neurons was demonstrated in cell-free extract from cortical neurons by an *in vitro* double strand break repair assay (Sharma, 2007), indicating gradual loss with age of the DNA repair in the brain.

Accumulation of b-actin and p53 gene-specific DNA lesioning during brain aging has been demonstrated (Sen et al 2007a), which were sensitive to hydroxyl radical scavengers like mannitol and sodium benzoate or by catalase. Chronic exposure to microwave radiation on developing rat brain has been shown to cause significant increase in DNA single strand breaks (Paulraj and Behari, 2006). Age-associated accumulation of oxidative DNA damage products such as 8-OHdG and DNA protein cross-links were reported in various brain regions by Haripriya and colleagues (2005). They have shown that L-carnitine has an inhibitory effect on the accumulation of this age-related oxidative DNA damage.

Cholesterol

Age-specific sensitization of corticosterone over phospholipid and cholesterol regulation in discrete brain regions have been studied by Bhargava et al (1991). They reported that the alteration in lipids was associated with decrease in plasma beta-hydroxy butyrate levels and increase in beta-hydroxy butyrate dehydrogenase. The content of brain mitochondrial total phopholipid, cholesterol and their ratio increases with age and affect the mitocondrial membrane system, and in turn respiration-related parameters in the brain (Modi et al 2008). Thyroid hormone has been reported to affect phosphatidyl ethanolamine and phosphatidic acid specifically in brain mitochondria, implying enhanced phospholipid turnover without affecting its or cholesterol content (Bangur et al 1995). The levels of phosphatidylinositol and phosphatidylinositol-4,5-bisphosphate were found to be decreased following acute stress, which was reversed back to control levels following repeated stress (Subramoniam et al 1990).

Lipid peroxidation

While a decreased peroxidative potential in rat brain microsomal fractions during ageing has been reported (Devasagayam, 1989), lipid peroxidation has been found to be maximum in brain followed by liver, kidney and heart (Pushpendran et al 1998), indicating higher susceptibility of the brain to xenobiotic toxicity. It has been demonstrated that the endogenous lipid peroxides in different regions of guinea pig or rat brain increases in old animals (Vohra et al 2001; Rani and Panneerselvam 2002), which was shown to be correlated with a decline in Na-K-

ATPase activity (Kaur et al 1998). L-Carnitine has been found to be useful in age-associated increase in lipid peroxidation in cerebral cortex, cerebellum, and hypothalamus of rat (Kaur et al 2001; Rani and Panneerselvam 2002). In crude rat brain synaptosomal fractions exposed to iron and ascorbate, enhanced lipid peroxidation followed by protein oxidation was observed (Chakraborty et al 2001), which was not inhibited by catalase but by alpha-tocopherol indicating relevance in the aetiopathogenesis of several neurodegenerative disorders as well as ageing of brain. Loss of cardiolipin in aged brain mitochondria linked to a loss of mitochondrial membrane potential during aging has been demonstrated, which may lead to uncoupling of oxidative phosphorylation, ATP depletion and activation of apoptotic cascade in aged rat brain (Sen et al 2007b).

Neurotransmitters and their receptors

Several studies have been published from this subcontinent on neurotransmitters and their receptors . most of which deal with neurotoxicity of metals and certain chemicals entities as well as neuropharmacological investigations on drugs. In this chapter we addressed only those which are within the scope of neurochemistry in relation to behavior, and functions of the CNS during normal physiology.

Behavior

Dopamine in nucleus accumbens and caudate nucleus has been shown to be involved in feeding and drinking behavior mediated through central D2 receptors, since intracerebral injections of the transmitter increased water intake which was suppressed by known D2 receptor antagonists (Pal and Thombre, 1993). Tremor has been shown to be directly associated with central nervous system serotonergic function, in terms of its synthesis and release, since 5-HT synthesis inhibition in the brain failed to evoke tremor response in rats (Mohanakumar and Ganguly, 1989; Mohanakumar et al 1990; Sarkar et al 2000; Mehta et al 2001;2003;2005). It has also been demonstrated that basal ganglia and olivo-cerebellar system participate in this motor dysfunction (Mohanakumar and Ganguly, 1989; Mehta et al 2001; 2003). Cholinergic modulation of serotonin synthesis has been demonstrated for the first time in literature (Kumari et al 2007), and it is suggested that many of the pharmacological interpretations in the past arising out of cholinergic and serotoninergic drugs should be reassessed in this light. Swim ability has been shown to be directly related to the intact nigrosrtiatal dopaminergic pathway, and swim inability has been correlated positively with striatal dopamine levels (Haobam et al 2005). It has been demonstrated that unilateral bias in dopaminergic function is achieved only following median forebrain bundle, but not after nigral or striatal lesion by certain neurotoxins (Sindhu et al 2006). Kokare et al (2006) have demonstrated that GABA-A receptors mediate orexin-A stimulated food intake.

Sleep and sleep deprivation on neurotransmitter function

It has been shown that rapid eye movement (REM) sleep loss increases acetylcholinesterase activity (Thakkar and Mallick, 1991; Mallick and Thakkar, 1991; Mallick and Thakkar, 1992), decreases neuronal calcium (Mallick and Gulyani, 1996), increases Na-K ATPase and chloride ATPase activities (Mallick and Gulyani, 1993), and activation of 5'nucleotidase activity (Thakkar and Mallick, 1996), all of which mediated through noradrenaline. Decreased breakdown of NA (Thakkar and Mallick, 1993) or increasing tyrosine hydroxylase activity (Majumdar and Mallick, 2003) resulted in increased availability of noradrenaline at the synapses. At the molecular level it has been observed that the noradrenaline(NA) acted on á-1A adrenoceptor and by activating calmodulin increased Na-K ATPase activity by dephosphorylation of the enzyme (Mallick et al 2000; Das et al 2008a). REM sleep loss increases apoptosis and results in reduced structural proteins in neurons suggesting function of REM sleep is to prevent apoptosis and cell loss (Biswas et al 2006a). Since REM sleep loss induced effects were mediated by NA acting on á1 adrenoceptors, and its active site has been modelled in silico and using point mutation studies, the amino acid that is most crucial for binding of the neurotransmitter and its agonist/antagonist have been identified (Ramachandran et al 2007).

Hormones and neurotransmitters

The brain neurotransmitter levels and their receptor activity, and hormonal pathways control or regulate many physiological functions in the body. A likely interaction between CNS insulin receptors and brain monoamines or thyroid hormone, has been reported *in vivo* following intracerebroventricular administration of insulin in rat (Bhattacharya and Saraswati, 1991), or *in vitro* in the total membranes, synaptosomes and choroid plexus (Azam and Baquer, 1990, Azam et al 1990). Gene expression and receptor binding studies indicated that muscarinic M1 and M3 receptors showed reciprocal changes in the brain stem during regeneration of pancreas, which in turn would regulate sympathetic and parasympathetic activity, and control the islet cell proliferation and glucose homeostasis (Renuka et al 2004). Conversely, muscarinic M1 receptor showed increase in the number of receptors and their affinity in diabetes implying modulation of receptor binding by insulin in the brain (Gireesh et al 2008). One of the studies has shown that the liver cell proliferation influences the brain stem GABAergic neurotransmission and these changes regulate the hepatic proliferation through the sympathetic stimulation (Biju et al 2002). Alterations of GABA receptors influencing levels of GABA (Balarama Kaimal et al 2007), and serotonin-2C receptor binding (Pyroja et al 2007) in the brain stem of rats during pancreatic regeneration have also been reported. It is suggested that the up-regulated receptor function might induce hepatocyte proliferation mediated through sympathetic stimulation.

Immunohistochemical or histoenzymological studies on localization of LH-RH, acetylcholinesterase, neuropeptide Y, calcitonin, serotonin, dopamine, b?-endorphin, gonadotropin-releasing hormone, neuronal nitric oxide synthase, neuropeptide Y, glucagon-like peptide-1 and GnRH in the discrete brain regions of several vertebrate species especially teleost have been carried out during ontogeny, reproductive cycle and following castration (Subhedar and Rama Krishna, 1988; 1990; Subhedar et al 1997a,b; Sarkar and Subhedar, 2000; 2001; Singru et al 2003; Gaikwad et al 2004; 2005; Biju et al 2005; Sakharkar et al 2005; 2006; Mazudar et al 2005; 2007).

Involvement of neuropeptide Y Y1 receptors in the regulation of LH and GH cells in the pituitary of the catfish has been shown (Mazumdar et al 2006). Interestingly cocaine- and amphetamine-regulated transcript peptide in the brain of the catfish has been demonstrated in a recent study (Singru et al 2007).

Dietary influence

The effect of nutritional deficiencies on neurotransmitter levels of the brain were studied by one group and reported that the levels of 5-hydroxytryptamine, 5-hydroxyindoleacetic acid, GABA, glutamate and tryptophan were found to be decreased in weaning rats with iron deficient diet (Shukla et al 1989a,b).

Dopamine synthesis and autoxidation

It has been shown that dopamine can cause dose-dependent inhibition of Na+, K+-ATPase activity of rat brain crude synaptosomal-mitochondrial fraction and this enzyme inactivation could be prevented by catalase, metal-chelators and quinine scavengers but not by superoxide dismutase or hydroxyl-radical scavengers like mannitol and dimethylsulphoxide, respectively (Khan et al 2003). Studies have also revealed that the damaging effects of DA on brain mitochondria are apparently mediated by quinone oxidation products generated by auto-oxidation of DA as well as catalyzed by a mitochondrial activity (Jana et al 2007) since in contrast to dopamine, its metabolite 3,4-dihydroxyphenylacetic acid caused only marginal inhibition of rat brain mitochondrial respiratory chain activity. It has been demonstrated that long-term levodopa treatment would result in significant loss of 5-HT in serotonergic and dopaminergic regions of the brain, without any significant change in L-DOPA metabolism,, dopamine synthesis would be severely impaired in all the regions causing overt cognitive, motor, and psychological functional aberrations (Borah and Mohanakumar, 2007).

Excitotoxicity & neuroprotection

A recent study demonstrated that both NMDA and non-NMDA sub-types of glutamate receptors are involved in glutamate mediated neurotoxicity but their relative contribution is highly dependent on the age of the animal (Sanganahalli et al 2006). Regulation of p75 (NTR) by NMDA receptors exert a control in inducing

apoptosis (Hota et al 2008a) and NR1 and GluR2 expression mediates excitotoxicity (Hota et al 2008b) in hypobaric hypoxia, that causes neural degeneration and memory loss, especially at high altitude. This study indicated that excitotoxicity occurs in hypobaric hypoxia and the drug administration against ionotropic receptors of glutamate could be a potential therapeutic value for ameliorating high-altitude-induced cognitive dysfunction. Conversely, transcriptional regulation of polysialylated neural cell adhesion molecule expression by NMDA receptor activation in retinoic acid-differentiated SH-SY5Y neuroblastoma cultures (Singh and Kaur, 2007) has been demonstrated to underlie the basis of the neuroprotective effect of subtoxic dose of NMDA in these cells (Singh and Kaur, 2005).

Miscellaneous

Relative DNA cleavage efficiency and copper binding ability of serotonin and structurally related molecules (tryptophan and melatonin) has been demonstrated, and indicated that the phenolic group in serotonin regulate strand cleavage activity (Hadi et al 2001).

Nitric oxide

Shukla et al (1995) have suggested that during infections, nitric oxide synthase induction causes the release of large quantities of nitric oxide, resulting in increased blood-brain barrier permeability, which could be blocked by inhibiting the enzyme (Shukla et al 1996). It has been further shown that nitric oxide itself can cause an increase in the permeability of blood-brain barrier, however, arginine-induced opening is not nitric oxide mediated. Contrarily, Shyamaladevi and colleagues (2002) have demonstrated that high nitric oxide concentrations in the brain following L-Arg administration could increase the permeability of blood-brain barrier to peripheral GABA. Paul and Jayakumar (2000) have reported the gamma-aminobutyric acid transaminase inhibitory action of nitric oxide in the brain.

At a time when a lot of evidences are in favour of nitric oxide as a mediator of neurotoxicity and the initiator of cascade of events that culminate in neurodegeneration, there are groups that fiercely contest their claims by providing evidences for a novel neuroprotective role for this endogenous gaseous neuromodulator (see the review by (Mohanakumar et al 2002b). From India, the role of nitric oxide in 3-nitropropionic acid (Deshpande et al 2006) or 6-hydroxydopamine (Barthwal et al 2001) induced toxicity was investigated using *in vivo* and *in vitro* models and reported that the neurotoxin generates nitric oxide both directly as a donor and indirectly by enhancing nitric oxide synthase expression in astrocytes and neurons. At a time when Singh et al (2005) claimed that nitric oxide synthase inhibition could effectively protect against neurodegeneration, it has been demonstrated convincingly that nitric oxide in the brain is extremely useful to scavenge reactive oxygen species generated (Mohanakumar et al 1994; 1998; Banerjee et al 2008; Thomas et al 2008).

Depletion of the cellular antioxidant glutathione in dopaminergic neurons could contribute oxidative and nitrosative stress (Vali et al 2007). Nitration of mitochondrial complex I subunits by peroxynitrite is reported (Mythri et al 2007) which could be blocked by antioxidant such as curcumin.

CNS Disorders : Neurochemistry

Japanese Encephalitis

Japanese Encephalitis virus infects neural progenitor cells and decreases their proliferation (Das and Basu, 2008), releases proinflammatory mediators (Ghosal et al 2007), induces IL10, IL-18 and IL-1b in microglia and astrocytes (Swarup et al 2007; Das et al 2008b), TNF receptor-associated death domain mediated microglial activation and subsequent release (Swarup et al 2008), through TRADD (Swarup et al 2007a). These results not only provide a new and general principle for cure of the whole spectrum of neuroinflammatory diseases that include infection of the CNS, but also other neurodegenerative disorders that affect millions of people worldwide.

Glioblastoma

Understanding the link between inflammation and oxidative stress in the progression of glioblastoma multiforme has been investigated in detail. Evidences suggest that the increased presence of inflammatory cytokines in the tumor microenvironment is a major factor in inducing malignancy (Agarwal et al 2008). Moreover, prolonged exposure of cells to inflammatory cytokines induces reactive oxygen species production which is often considered as an adverse factor due to their stimulating effects on cell proliferation, genetic instability, and senescence evasion (Sharma et al 2007).

Glioblastoma multiforme (GBM) is the most common, infiltrative and highly vascularized tumor with a very poor prognosis. Elevated levels of pERK in grade IV and PARP cleavage in both grade II (astrocytoma) and grade IV (GBM) tumors and predominant increase in μ-calpains in GBM tumor tissues compared to low grade tumor along with increased LDH activity and elevated procaspase-3 supports necrotic cell death in GBM. (Bhaskara et al 2005; 2006).

Cerebral malaria

Increases in cerebrospinal fluid protein and malondialdehyde were observed in 73 patients with cerebral malaria, compared to values in 23 control subjects, and it is proposed that cerebral oedema due to enhanced permeability of vascular endothelium induced by increased lipid peroxidation plays a crucial role by allowing the parasite to enter the brain (Das et al 1991). Changes in brain neurotransmitters, 5-HT, norepinephrine, and dopamine were studied (Roy et al 1993) in cerebral malaria.

It has been shown that both necrosis and apoptosis exist during cerebral malaria in the mouse brain. Calpains, calpastatin and spectrin breakdown products (Shukla et al 2006), intrinsic cell death proteins-Bcl_2, Bax, cytochrome-c and p53 were elevated (Kumar and Babu 2002; 2003) in mice brain during experimental fatal murine cerebral malaria**.** Further, the activity and the levels of PKC isoforms were investigated in the mouse brain in an experimental model of fatal murine cerebral malaria (Kumar et al 2003). This study shows that cerebral malaria alters the levels of more than one PKC isoforms and also suggests the degradation or decreased levels of all PKC isoforms. Then decreased levels of these isoforms might play an important role in dysfunctional apoptosis. The study also qualitatively analyzed the possible putative protein candidates serving as substrates for PKC during CM pathology (Kumar et al 2003).

Paraplegia

It has been shown that there is a temporal progressive loss of mitochondria function during experimental spinal cord injury (Sullivan et al 2007), a finding which would be helpful in limiting secondary damage in spinal cord by devising treatment to salvage mitochondrial function.

Alzheimer's disease

Alzheimer's disease (AD) is a neurodegenerative disorder, characterized by progressive loss of cognitive function. An invariant pathological hallmark of AD is the deposition of plaques consisting largely of the 40-42 amino acid long amyloid ß (Aß) peptide. Available literature indicates lower rates of incidence of AD in both rural and urban populations of India than in developed countries. To determine whether limitation of Aß concentration may be responsible for lower rate of incidence, the levels of Aß in CSF collected from non-demented individuals ranging in the age from 20 years to 65 years were measured (Subramanian et al 2006). For the first time, it is reported that CSF Aß levels are low in Indian population, which may be contributing to lower rate of AD incidence.

A simple and economical procedure for production of bulk amounts of Aß by employing bacterial expression system has been developed (Subramanian and Divya Shree, 2007) as a first step towards the development of Alzheimer's vaccine. Recent active immunization studies with fibrillar Aß in humans had to be halted because 6% of the AD patients developed acute meningoencephalitis, likely due to anti-Aß specific autoimmune Th1 cells. In an attempt to develop safer vaccines, the influence of oligodeoxynucleotides as adjuvant on the Th1 and Th2 immune response to Aß in aged rats and DNA prime – protein boost strategy could elicit a more robust Th2 response were investigated (Subramanian and Divya Shree 2008). Significantly, the IgG subclasses of the antibodies generated by DNA prime – protein boost regimen with oligodeoxynucleotides as adjuvant were primarily IgG2b and IgG1 isotypes, suggesting that heterologous immunization strategy along

with oligodeoxynucleotides would be advantageous in eliciting more beneficial Th2 type humoral immune response.

Expression and regulation of genes associated with the pathogenesis of Alzheimer's disease and mechanism of sex steroid hormone action during brain aging have been studied extensively in India in animal models during the past two decades. The genes associated with the pathogenesis of AD include amyloid precursor protein (APP) (Mani and Thakur, 2006; Asaithambi et al 1999), and presenilin (PS) (Thakur and Ghosh, 2007). The expression of APP mRNA (Thakur and Mani, 2005), its alternative splicing pattern and protein level changes with age and by sex steroids in adult and old mice have been investigated. Methylation of APP promoter is higher in females (Mani and Thakur, 2006).

Suram and colleagues (2002; 2007) have for the first time provided evidences for helical transitions in supercoiled DNA by amyloid-b-peptide (1-42) and aluminium, and demonstrated its immunoreactivity in apoptotic nuclei of degenerating AD brain hippocampal neurons (Suram et al 2007). These data suggested endonuclease action of A beta and its biological significance in terms of causing direct DNA damage.

Parkinson's disease

Hegde et al (2006) have postulated DNA strand breaks in the midbrain, caudate nucleus, putamen, thalamus, and hippocampus as a mechanism of neuronal death in Parkinson's disease (PD). This study provided a comprehensive database on stability, damage, and conformations of DNA in different regions in brains of PD patients. A clinical prospective study has been undertaken in PD patients for finding the cause and effects of Indian traditional medicine for the first time and reported therapeutic doses of L-DOPA contained in the preparation (Nagashayana et al 2000; Sankaran Kutty et al 2001). Gangopadhyay et al (2000) have reviewed antioxidant protective therapy and its usefulness in PD. Blood proteome profiling of human PD samples revealed involvement of haptaglobin related protein precursor and â-globin truncation in the pathogenesis of PD and demonstrated that plasma proteome profiling may assist as a parameter in disease diagnosis (Sinha et al 2007).

A number of animal models of PD have been developed and tested in Indian laboratories. Toxin induced lesions following systemic administration or unilateral intracranial infusion to achieve hemiparkinsonian animals have been tried. MPTP, the parkinsonian neurotoxin that causes parkinsonism in humans and has been employed to obtain PD model in mice (Mitra et al 1992; 1994), which has been shown to be sensitive to many of the antiparkinsonian drugs currently in use (Muralikrishnan and Mohanakumar 1998; Muralikrishnan et al 2003). It has also been demonstrated that the voltage gated calcium channels have a direct role in dopaminergic neuronal death (Mohankumar et al 2002a). Non streroidal antiinflammatory mediators have been shown to be useful in PD therapy employing

this model (Mohanakumar et al 2000; Sairam et al 2003; Maharaj et al 2004). The mitochondrial electron transport chain inhibitor at the complex-I level, rotenone has also been used for modeling a hemiparkinsonian model in rats which is progressive in nature and has protein aggregation in the dopaminergic neurons (Saravanan et al 2005; Sindhu et al 2005). This model also responded to clinically used antiparkinson's drug, selegiline (Saravanan et al 2006). It has been demonstrated for the first time that homocysteine, a byproduct in dopaminergic catabolic pathway following folate deficiency could be a mediator for specific dopaminergic neuronal death in PD (Chandra et al 2006).

The involvement of toxicant responsive genes and vesicular monomine transporter-2, complexin-I, á-enolase and glia maturation factor-â, as well as alterations in the expression of several transcripts associated with various pathways such as energy metabolizing pathways in experimentally induced parkinsonism in mice were demonstrated recently from India (Patel et al 2007; 2008; Singh et al 2008). The results indicated that defective energy metabolism could play a critical role in inducing PD. Estimations of antioxidant defense enzymes, inducible nitric oxide synthase and differential expressions of CYP2E1, CYP1A1, GST-ya, GST-yc, and GSTA4-4 genes suggested the involvement of these genes in the augmentation of lipid peroxidation pathways in neurodegeneration (Kumar et al 2006a; Patel et al 2005; 2006; Singh et al 2006).

Huntington's disease

Huntington's disease is a progressive neurodegenerative disorder caused due to expansion of polyglutamine repeats in exon 1 of huntingtin gene, which leads to neuropsychiatric disturbances, motor abnormalities and striatal neurodegeneration. Oxidative stress was shown to cause proteasomal dysfunction in cells expressing mutant huntingtin (Goswami et al 2006a). The potent antioxidant curcumin caused proteasomal dysfunction and increased cell death in cells expressing mutant huntingtin (Dikshit et al 2006). NF-kappaB dependent transcription was decreased and NF-kappaB subunits were found to have abnormal interaction with polyglutamine protein and sequestered as aggregates in cells expressing mutant huntingtin (Goswami et al 2006b). The co-chaperone was found to be over-expressed in cells expressing polyglutamine repeats (Dikshit and Jana, 2007). A new ubiquitin ligase, called E6-AP, was able to promote the proteasomal degradation of misfolded polyglutamine proteins and suppress the polyglutamine protein aggregation and polyglutamine protein-induced cell death (Mishra et al 2008). HIPPI, a molecular partner of huntingtin interacting protein HIP1 was found to interact with the putative promoter sequence of caspase-1, 8 and 10 (Majumder et al 2007a). Increased calpain and caspase-2 activity and decreased mitochondrial complex-II activity in cells expressing the exogenous exon 1 of huntingtin were also reported (Majumder et al 2007b). HYPK, a huntingtin interacting protein, reduced aggregates and apoptosis induced by N-terminal huntingtin with 40 glutamines in Neuro2a cells and exhibited chaperone-like activity (Raychaudhuri et al, 2008a).

In a further study it was found that HYPK is an intrinsically unstructured protein (IUP) with premolten globule like conformation (Raychaudhuri et al 2008b).

Administration of an inhibitor of the mammalian mitochondrial enzyme succinate dehydrogenase, 3-nitropropionic acid has been shown to cause striatal lesions and phenotype similar to HD. It was shown that 3-NP could also cause complex-I and –IV deficits and decrease in NAD+ linked state 3 respiration in rat brains (Pandey et al 2008). Tiagabine, a GABA uptake inhibitor, attenuated 3-nitropropionic acid-induced alterations in various behavioral and biochemical parameters in HD rats (Dhir et al 2008). Resveratrol, an antioxidant with cyclooxygenase I inhibitory activity was also found to be protective in the 3-nitropropionic acid-induced model of Huntington's disease (Kumar et al 2006).

Leigh Syndromes

A novel, homoplasmic T11984C missense mutation in ND4 gene, is speculated to interplay this mitochondrial mutation along with nuclear gene(s) in the pathogenesis of Leigh syndrome (Vanniarajan et al 2006).

Anxiety & Depression

Nair et al (2006) reported that alterations in CREB/BDNF may contribute to the generation of individual differences in stress neurocircuitry, providing a substrate for altered vulnerability to depressive disorders. It has been found that during anxiety, amygdal neuropeptide Y and alpha-melanocyte stimulating hormone interacts each other (Kokare et al 2005).

Leprosy

Leprosy is one of the most intriguing diseases of the mankind where a non-cultivable bacterium, i.e. *Mycobacterium leprae* invades the peripheral nervous system to play havoc but the central nervous system is spared. In a study carried out in human leprous nerves, using SDS-PAGE, Western immuno-blot and immuno-histochemistry we have been able to demonstrate abnormality in the neurofilament proteins and their phosphorylation state (Save et al 2004a). *M. leprae* induced alteration in the phosphorylation high and medium neurofilament that led demyelination (Shetty et al 1999). Save et al (2004b) have investigated compartmentalization if any in the kinases wherein immunohistochemical localization of phosphorylated NF and ERK 1/ 2 was done in the dorsal root ganglia of *M leprae* infected mice.

CNS Disorders : Neurogenetics

Parkinson's disease

Even though epidemiological, pharmacological and biochemical studies on PD have been continuing in India for a long time, the first genetic study was reported

by Thelma and group (Nagar et al 2001), where they reported that the polymorphisms, G88C and G209A in alpha-synuclein gene in Indian patients with PD comprising of 18 familial, 3 juvenile, 48 early onset and 100 sporadic cases (in total 169 patients) do not seem to be genetic determinants of the disease. In a subsequent study the association of seven SNPs and SNP haplotypes in the phase II conjugating enzyme N-acetyl transferase 2 gene (*NAT2*) and slow acetylator phenotype, with the development of young onset and late onset PD (YOPD & LOPD) amongst Indians they suggested significant association of SNPs and specific SNP haplotypes in *NAT2* and slow acetylator phenotype with YOPD, but to a lesser extent with LOPD (Chaudhary et al 2005). Their group has also carried out association studies of leucine-rich repeat kinase 2 (*LRRK2*) and *parkin* genes (Punia et al 2006; Chaudhary et al 2006), searching for mutations of *LRRK2* gene in Indian PD patients. Their findings revealed that these mutations are either rare or absent in this population, and as such these SNPs may not be useful for diagnostic screening and genetic counseling. In the case of *parkin* gene, they observed a mutation frequency of 8.5% in Indian PD patients. Their studies on nine point mutations suggested that these mutations accounted for 14.3% familial, 6.9% young onset, and 5.9% late onset sporadic PD in India.

Two other groups also explored the possibility of *parkin* gene as a potential candidate gene for PD (Madegowda et al 2005; Biswas et al 2006b, 2007). The first study was on mutation screening of *parkin* gene from a non-white, non-oriental population of early onset PD (Madegowda et al 2005). The second study identified 7.24% mutations of *parkin* gene among eastern Indian PD patients and reported that two SNPs, Ser167Asn and Val380Leu are associated with PD (Biswas et al 2006, 2007). A study carried out on twenty markers on four dopamine receptors (*DRD1*, *DRD2*, *DRD3*, and *DRD4*) and dopamine transporter (*DAT*) genes in northern and southern India PD subjects showed that a 120 bp duplication marker of *DRD4* is associated with PD in both the two datasets (Juyal et al 2006). A case-control analysis to investigate the association of polymorphism in the genes that encode proteins involved in toxication-detoxication and dopaminergic pathways with susceptibility to PD (Singh et al 2008a) on seventy PD patients and one hundred healthy controls suggested the polymorphism in the genes involved in detoxification and dopamine regulation may modulate the susceptibility to PD and could be important risk factors for PD.

Alzheimer's disease

AD is a complex disease caused by an interaction of multiple environmental and genetic factors. Chandak et al (2002) studied the association of presenilin-1 (*PS-1*) and apolipoprotein E (*ApoE* gene variants) in 49 cases and 100 matched controls from western India. The genetic analysis confirmed the association of APOE epsilon 4 allele with AD that was reported by others. The *PS-1* variant allele 1 demonstrated no association in this case. In a later report, Luthra et al (2004) showed that the odds of developing AD or vascular dementia were 4.4 and 3.7

times higher, respectively, in the presence of even a single epsilon4 allele of *ApoE*. A case-control approach by Kaur and Balgir (2005) on 100 cases and 36 healthy controls also supported the above findings. They reported that the odds of having AD significantly increased among those with one or more copies of the E4 allele compared to individuals with the APOE3/3. Recently Pandey et al (2007) studied the association of PS1 intron 8 and ApoE epsilon4 gene polymorphism in degenerative and vascular dementia patients in the northern Indian population. Based on the results they concluded that ApoE epsilon4 allele increased the susceptibility to both degenerative as well as vascular dementia, but PS1 allele 1 increased the susceptibility to degenerative dementias only.

Huntington's disease (HD)

The first report on genetics of HD in India was published in 2000 (Pramanik et al 2000), in which the authors have analyzed the distribution of polymorphic CAG and CCG repeats in huntigtin gene in clinically diagnosed unrelated HD and normal individuals from Indian population. Their studies showed that the expanded CAG repeats in HD patients were in the range of 41 to 56 and in the control subjects it ranged from 11 to 31 repeat alleles. The same group in 2003 reported that GluR6 kainate receptor gene and polymorphic CCG repeat genotypes influence the age of onset of HD in genetically susceptible individuals (Chattopadhyay et al 2003a). Further studies on variation of age of onset of the disease revealed the interaction of CAG repeats of *Htt* with the *TP53* (transcription factor p53) and *hCAD* (human caspase activated DNase) genes in modulating the age of onset in HD (Chattopadhyay et al 2005). Subsequently they studied the involvement huntingtin interacting proteins in the formation of huntingtin aggregates and mechanism of apoptosis using cell model of HD (Majumder et al 2007; Raychaudhuri et al 2008a,b). Muthane and his collaborators in 2001 made a comparison of clinical and genetics data of HD and they found that the mean CAG repeat allele was 48.4 in the Indian HD cohorts (Murgod et al 2001). Another study investigated the population variation and haplotypes of HD in India, for which they analysed CAG repeats at the HD locus and the closely linked polymorphisms in 30 HD families and 250 ethnically matched controls (Saleem et al 2003). Haplotype analysis of their study suggested the presence of a founder mutation in a subset of Indian families, which provided evidence for multiple and geographically distinct origins for the HD mutation in India.

Ataxias

Similar to HD, spinocerebellar ataxias (SCAs) are also caused by expansion of (CAG)n triplet repeats and the repeat number beyond a threshold size are pathogenic in nature. Genetic analysis of ataxias carried out in 42 Indian families in the year 2000 for the first time and found that SCA2 is more frequent in this population. They observed an intergenerational increase in CAG repeat size along with horizontal increase with the birth order of the offspring in the SCA2 families,

suggesting the significance of parental age in repeat instability (Saleem et al 2000). Subsequent work by the same group on 215 normal and 64 expanded chromosomes identified and characterized two novel SNPs in exon 1 of the SCA2 gene and their results demonstrate that CAA interruptions in the repeat region play a vital role in conferring stability to SCA2 repeat. They also showed that the absence of CAA interruptions predisposes repeat alleles towards instability and pathological expansion (Choudhry et al 2001). Following this in 2003, another group examined the variation in the RAI1 gene in 30 spinocerebellar ataxia patients and normal individuals in order to understand the possible influence of this gene on the age at onset. Their study indicated that in SCA2 patients RAI1 might explain about 13% of the variability in the age at onset of the disease (Chattopadhyay et al 2003b).

Machado-Joseph disease 1/SCA3 is the most common type of spinocerebellar ataxia (SCA) worldwide. In order to understand the low prevalence of the disease among SCA patients from eastern India, Chattopadhyay et al (2003b) analyzed the CAG repeats and two bi-allelic intragenic markers at SCA3 locus among 412 normal individuals and 10 patients. Their study suggested that the low prevalence of SCA3 could be due to the low prevalence of large normal alleles, which might act as the reservoir for the expanded alleles. As there is a wide variation in the prevalence of spinocerebellar ataxia type 1 (SCA1) in different populations, Mittal and colleagues (2005) made an attempt to study the mutational history and prevalence in the Indian population. They observed SCA1 in approximately 22% (37/167 families) of the autosomal dominant cerebellar ataxias (ADCAs) in the Indian population. Their analyses using markers linked to SCA1 suggested that the prevalence of SCA1 in the Indian population is dependent on both repeat length and number of interruptions. The spectrum of these repeat alleles also implicated the antiquity of SCA1 mutation in the Indian population (Mittal et al 2005). The same group also carried out research on spinocerebellar ataxia type 12 (SCA12), an autosomal dominant cerebellar ataxia associated with the expansion of an unstable CAG repeat in the 5' region of the PPP2R2B gene on chromosome 5q31-5q32. They found that SCA12 accounts for approximately 16% (20/124) of all the autosomal dominant ataxia cases diagnosed in AIIMS, Delhi. They identified four novel SNPs and a dinucleotide marker spanning approximately 137 kb downstream of CAG repeat in the PPP2R2B gene. The analysis of 20 Indian SCA12 families and ethnically matched normal unrelated individuals provided evidences for the presence of a common founder for SCA12 in the Indian population (Bahl et al 2005).

Alluri et al (2007) have evaluated the sizes and distributions of the repeats at the SCA1, SCA2, SCA3, SCA7 and DRPLA loci by molecular analysis and also the association of larger normal (LN) alleles with disease prevalence. The studies conducted by Krishna et al (2007) have shown that SCA1 represents the more common mutation in southern India. They have also identified large numbers of SCA3 probands in this group. Friedreich ataxia (FRDA) is characterized by homozygous expansion of GAA repeats in the first intron of the frataxin gene.

Chattopadhyay et al (2004) have studied the distribution of polymorphic GAA repeats in the frataxin gene among 6 clinically diagnosed patients and 160 ethnically matched normal individuals, in order to have information on the prevalence of FRDA in the eastern part of India. They found one novel rare haplotype, ACCT, among the expanded alleles as well as among normal individuals and they suggested that this haplotype might be characteristic of Indian population.

Wilson disease (WD)

WD is an autosomal recessive disorder characterised by hepatolenticular degeneration as a result of accumulation of copper. It is caused by defects in ATP7B gene located in chromosome 13q14. Gupta et al (2003) have reported a PCR-based molecular diagnostic test to identify presymptomatic siblings of WD affected individuals in families with multiple offspring. In a later study they identified prevalent mutations in the ATP7B of Indian WD patients and attempted to correlate the genotype with the disease phenotype. Based on their findings they speculated a potential role for yet unidentified modifying loci for the observed phenotypic heterogeneity among the WD patients (Gupta et al 2005). Further work on WD by the same group suggested that the some of the intragenic SNP markers of the WD gene are highly heterozygous across most world populations and this could be used in combination with analysis of prevalent mutations as a comprehensive strategy for determining presymptomatic and carrier sibs of WD patients (Gupta et al 2007a). In a comparative study conducted on different WD mutations between different regions of India, they suggested the presence of a high genetic heterogeneity and the absence of a single or a limited number of common founder mutations (Gupta et al 2007b). Santhosh et al (2006) studied the spectrum of ATP7B mutations including 11 novel mutations in Indian WD patients and documented lack of a single dominant mutation. The presence of identical WD phenotype among siblings in only 6 of 8 families with >1 child affected by WD suggests that factors other than ATP7B mutations influence WD phenotype. A recent study on this disease suggested that R778W and I1102T variation in ATP7B are most common mutations in the Indian WD cohorts and thus might provide the basis of genetic (PCR-RFLP) diagnostic tool for Indian WD patients as well as in siblings/parents where biochemical parameters are ambiguous (Kumar et al 2007).

Schizophrenia

In an attempt to investigate the association of the cytosolic phospholipase A2 locus (cPLA2) with schizophrenia, Chowdari et al (2001) failed to show any significant association in the Indian population. A number of linkage, association and cytogenetic studies have implicated chromosome 22 in schizophrenia and bipolar disorder. Saleem et al (2001) investigated whether 8-repeat allele of a CAG repeat tract at this locus is overrepresented in both schizophrenia and bipolar patient groups and based on their results they suggested that the repeats within this gene or other genes in the vicinity of this locus are likely to be associated with

bipolar disorder and schizophrenia in Indian population. This region also harbors synaptogyrin 1 (*SYNGR1*) gene located on 22q13.1, within 1 million base pairs of this marker and the encoded protein is associated with synaptic vesicles. Verma et al (2004) identified a novel nonsense mutation (Trp27Ter) in exon 2 of the *SYNGR1* in a family multiply affected with schizophrenia. Since involvement of SYNGR1 is implicated in presynaptic pathways, they suggested that reduced levels of this protein might play some role in the pathogenesis of schizophrenia (Verma et al 2004). Subsequently, they performed the sequencing of all exonic and flanking intronic regions of the SYNGR1 gene in 198 BPAD and 193 SCZ cases and they observed a novel mutation Lsy99Glu in one BPAD patient and two other novel common polymorphisms. Their result supported SYNGR1 as a probable susceptibility gene for SCZ and BPAD. From the observed positive association with both SCZ and BPAD, they concluded that a common pathway might possibly be involved in the etiology of both the disorders (Verma et al 2005).

In a different study, Semwal et al (2002) analysed eight genes for possible association with schizophrenia and two markers, one in the promoter region of the serotonin 2A receptor gene and the other in the tryptophan hydroxylase gene have shown positive genetic correlation with schizophrenia through population based study. As dysfunction of dopamine D2 receptor signaling is implicated in schizophrenia, Kukreti et al (2005) studied the association of two synonymous polymorphisms (His313 and Pro319) in the dopamine D2 receptor (*DRD2*) gene with schizophrenia. Their results provided suggestive evidences for a positive association of these SNPs with the disorder (Kukreti et al 2005). *DRD2* Taq1B, Taq1D, S311C, H313H and Taq1A polymorphisms were also studied by Vijayan et al (2007) in relation to both disease association and antipsychotic drug response in patients and control subjects from South India. Their results indicated that certain polymorphisms could be defined for their critical functions in disease pathology and drug response in South Indian population based on genotype phenotype correlations. A recent report of two-point linkage analysis performed by Mukherjee et al (2006) on a series of 52 multiplex pedigrees with 23 polymorphic markers demonstrated a positive linkage and association finding at 18p11.2 for psychosis.

Autistic disorder

Autism is a childhood neurodevelopmental disorder characterised by behavioural problems. It is a highly heritable complex disorder. The first report on genetics of autism from India was published by Guhathakurta and colleagues in the year 2006. Hyperserotoninemia of platelets is one of the well characterised endophenotypes for autism. Therefore, they selected serotonin transporter gene (*SLC6A4*) as one of the potential candidates for the disorder. In their first paper, they reported the findings on the genetic association analysis of a promoter region polymorphism (5-HTTLPR) of serotonin transporter gene (*SLC6A4*) with autism in the Indian population. Even though this study failed to provide any positive association using population and family based approaches, a recent study by the

same group (Guhathakurta et al 2008) on two polymorphisms, VNTR of 17 bp at intron2 (STin2) and an SNP at 3'UTR (HTT-3'UTR-SNP) reported a contrasting linkage disequilibrium (LD) pattern between the two markers in cases and controls. They also observed significant disease-specific distortion in the distribution of HTT-3'UTR-SNP genotypes and the specific haplotypes of the two markers, with higher frequencies of T/T genotype and 10-T haplotype in autistic cases. Overall their study suggested that either these markers or nearby markers of SLC6A4 that are in LD, might pose a risk towards autism in the eastern Indian population. Since autism is a polygenic disorder, they selected several candidate genes based on the importance of its gene product in neurodevelopment and physiology and the significant location of these genes in autism susceptibility regions in human genome.

Their preliminary studies on reelin gene (*RELN*) suggested that CGG repeat polymorphism in the 5'UTR of *RELN* might have a role in the susceptibility towards autism with the paternal transmission and non-transmission respectively of 10- and > or =11-repeat alleles, to the affected offspring (Dutta et al 2007b). Additionally, in a recent study they analyzed six more SNP markers of *RELN* using case-control and family based association approaches (Dutta et al 2008b), but the findings suggested that these markers are unlikely to be associated with autism spectrum disorder in the Indian population. They have also analysed different markers of homeobox genes such as *HOXA1* and *HOXB1*as well as the glutamate receptor 6 gene (*GLUR6* or *GRIK2*) for possible association with the disorder (Sen et al 2007, Dutta et al 2007a), but failed to detect any positive findings.

Attention Deficit Hyperactivity Disorder (ADHD)

ADHD is neurobehavioral disorder having strong genetic component and a childhood onset. A preliminary genetic association study on dopamine beta hydroxylase (DBH) gene by Bhaduri et al (2005) was the first report on genetics of ADHD from the Indian population. They conducted a family based study on two markers of DBH gene, where they observed a trend towards association in the case of DBH444g/a marker and lack of association in the case of intron 5 (Taq I) polymorphism (Bhaduri et al 2005). They further extended their work including one more marker (-1021C/T) and the results of the association analysis showed lack of significant association. However, they observed low D' values and corresponding log of odds scores in the control group as compared to the ADHD families indicating the higher chance of the two polymorphisms being transmitted together in ADHD families (Bhaduri and Mukhopadhayay 2006).

Since dopaminergic system is highly implicated in ADHD, they considered dopamine D4 receptor (*DRD4*), monoamine oxidase A (*MAOA*) and dopamine transporter gene (DAT1) as candidate genes for the disorder. In the case of *DRD4*, they observed association of a higher repeat allele 48-bp VNTR of exon 3 in Indian ADHD children (Bhaduri et al 2006). Their work on *MAOA* gene showed a significant overrepresentation of 3.5 repeat allele in the cases in comparison to controls and

preferential transmission of the short allele (3.5 repeat) from mothers to male ADHD probands. From these findings they concluded that the short 3.5 repeat allele of the MAOA-m VNTR might be associated with ADHD in our population and possibly the biased transmission could be the reason for boys to be more prone to ADHD as compared to girls (Das et al 2006). A 40 bp variable number of tandem repeats (VNTR) in the 3'-untranslated region (3'-UTR) of the *DAT1* was investigated by this group for possible association with ADHD in Indian children. The data obtained from this study revealed that the DAT1 3'-UTR 9R allele might confer risk of ADHD in this population (Das et al 2007). Since serotonin transporter gene (*SLC6A4*) polymorphisms are variously implicated in mediating susceptibility to ADHD, Banerjee et al (2006) studied the association of SLC6A4 promoter (5-HTTLPR) and intron-2 (STin2) polymorphisms with ADHD for the first time from the Indian population. The family-based study design indicated significant preferential transmission of 12-repeat allele of the STin2 to ADHD cases suggesting a possible positive correlation. Maternal inheritance of this allele was also significant suggesting a novel role for epigenetic mechanisms in the etiology of ADHD.

Epilepsy

Juvenile myoclonic epilepsy (JME) is common idiopathic generalized epilepsy, which has a complex pattern of inheritance. The study by Vijai et al (2003) provided suggestive evidence of allelic association between JME and KCNQ3. Subsequently they investigated the Ala322Asp mutation in GABRA1, which showed lack of association with autism. They also examined two marker loci, D5S2118 and D5S422, flanking GABRA1 and found that the allele frequencies at D5S422 exhibited a significant difference between the cases and controls suggesting genetic association between JME and genes located in the proximity of this marker (Kapoor et al 2003). In 2007, they reported identification of a novel epilepsy locus at chromosome 5q12-q14 in a family exhibiting autosomal dominant form of JME from south India (Kapoor et al 2007). Following this, they mapped a novel idiopathic epilepsy locus on chromosome 3q13.3-q21 through linkage analysis and identified a rare missense variant at an evolutionary-conserved region of calcium sensing receptor (CASR) in epilepsy patients (Kapoor et al 2008). These two findings along with CASR expression in various subregions of human brain provide suggestive evidences for the involvement of CASR in pathophysiology of epileptic disorders.

In a different study on generalized tonic clonic seizures, Soundararajan et al (2006) carried out a case-control analysis to understand the genetic correlation of two SNP markers of debrisoquine hydroxylase (CYP2D6) with the disease and they observed a significant association of 2850C > T with the disease. Kumar et al (2006c) investigated the relationship of apolipoprotein E (apoE) genotype, plasma levels of apoE and lipids in temporal lobe epilepsy (TLE) patients in Asian Indians. Outcome of their case control study revealed that apoE might play an important role in the etiology of epilepsy.

Mental Retardation (MR)

Mental retardation (MR) is a common disorder, affecting 1-3% of the total population and it arises from failure to develop age appropriate cognitive abilities and intelligence level. It is an etiologically heterogeneous type of condition with the involvement of both genetic and non-genetic factors. Hyperhomocysteinemia and / or homocystinuria, are often associated with mental retardation (MR). One of the possibilities of increased homocysteine level can be due to the inhibition of cystathionine beta-synthase (CBS), which mediates conversion of homocysteine to cystathionine. Several polymorphisms are reported for *CBS* and Dutta et al (2005, 2007c) analyzed three markers such as 34bp VNTR in exon-intron 13 junction and T833C/844ins68 in exon 8 for possible association with MR. The markers, T833C and 844ins68 are always associated together. The results of their preliminary study suggested lack of association of T833C/844ins68 with MR in the Indian population. However, they obtained a significant maternal transmission of the risk alleles (Dutta et al 2005). Their study on 34bp VNTR also demonstrated lack of association of this marker with MR through population and family based analyses.

Another reason behind decreased homocysteine is the reduced methylene tetrahydrofolate reductase (MTHFR) enzyme of the folate metabolic pathway. Converging evidences from biochemical and nutritional studies support the role of *MTHFR* gene in the etiology of MR. Therefore, they also investigated two polymorphisms of *MTHFR,* C677T and A1298C in idiopathic MR (Dutta et al 2008a). It has been concluded that these polymorphisms are not contributing directly to the aetiology of MR in Indian population since both case-control and family-based analysis revealed no significant transmission of the mutated allele. Mental retardation is often associated with chromosomal abnormalities. Moghe et al (1981) performed a cytogenetic study to evaluate the karyotype abnormalities in 74 MR patients. Of these, fourteen had chromosomal abnormalities, which included seven sex chromosomal and seven autosomal abnormalities.

One of the inherited forms of MR is the one seen in Fragile X syndrome. In a study conducted on 146 Indian patients (174 X chromosomes) with unexplained mental retardation, three of the 118 males were found to have the FMR1 full mutation (Pandey et al 2002). Since Down Syndrome (DS) patients suffer from cognitive dysfunction and dopamine as well as serotonin are known to control cognitive and behavioral attributes, Das Bhowmik et al (2008) recently reported the association of a variable number of tandem repeat (VNTR) in the exon 3 of DRD4 and an insertion/deletion polymorphism in the promoter region of 5HT transporter (5HTTLPR) with DS. Their family based study demonstrated significant over-transmission of a DRD4 VNTR allele to DS patients, whereas no association was observed in the case of 5HTTLPR.

Developmental Neurobiology

Serotonin (5-HT) is an important neurotransmitter known to act as a trophic factor for its own development as well as its target neurons. It has been shown that exposure to 5-HT antibodies during neonatal stage (Mohanakumar et al 1995) or at adulthood (Shravani et al 1998) respectively affect adversely the development of the serotoninergic system and thereby lead to specific long lasting changes in behaviors, and levels of 5-HT in the brain.

An interesting study on the effect of sound given prenatally on the developing brain of chickens has been carried out in India by a group, involving the auditory pathway, higher auditory imprinting area and hippocampus. Patterned prenatal auditory enrichment from embryonic day 10 to hatching at 65 dB in frequency range of 100-6300 Hz by species-specific maternal and hatchling sounds and sitar music in the domestic chick were given under the experimental conditions. Early embryonic auditory stimulation has shown an increase in the number and size of neurons in brainstem auditory nuclei: the nucleus magnocellularis, NM; nucleus laminaris, NL (Wadhwa et al 1999). The enhanced sound stimulation favored the survival of neurons by reducing physiological cell death and increasing the relative amounts of Bcl-2, an anti-apoptotic protein (Alladi et al 2005a). Prenatal patterned acoustic stimulation enhanced synaptic protein synthesis (Alladi et al 2002) and modulated the expression of the immediate early gene products, c-Fos and c-Jun, in NM and NL (Alladi et al 2005b). There was a change in the proportion of glutamate and GABA in the auditory nuclei in the auditory stimulated groups compared to the controls as determined by NMR spectroscopy (Aggarwal et al 2007).

Furthermore, the sound-stimulated groups showed an increase in neuronal size and the proportion of neurons containing calcium-binding proteins, calbindin D-28K and parvalbumin in the mediorostral nidopallium hyperpallium ventrale, which is an auditory imprinting area of chick forebrain (Panicker et al 2002). Prenatal exposure to both species-specific sounds and sitar music facilitates the postnatal auditory responsiveness and preference of the chicks to species-specific maternal calls (Jain et al 2004). Both auditory stimulated groups showed a significant increase in the proportions of CALB and PV-immunopositive neurons as well as the levels of protein expression when compared to the control in the embryonic period and on posthatch day one (Chaudhury et al 2006; 2007). It is thus observed that intermittent chronic auditory stimulation given in the prenatal period brings about an early maturation of the auditory pathway.

The developing human visual system was studied to describe the chronology of proliferation, migration, synaptogenesis as well as development and maturation of neurons of the retina, lateral geniculate body and visual cortex in the human fetuses. The neuronal differentiation in the central retina begins earlier than that occurring elsewhere completes by midgestation (19-21 weeks) and continues in the periphery until 30 weeks of gestation (wg). During differentiation, the neurons

express an array of molecules, such as neurotransmitters (Wadhwa et al 1988a; Masood et al 1993) neuropeptides (Wadhwa and Bijlani, 1988; Wadhwa et al 1988b; 1990) and calcium-binding proteins (Wadhwa and Jotwani, 1993).

Ganglion cells differentiate at 8-9 wg and arrange into a ganglion cell layer (GCL) in the central retina at 13-14 wg. Immunoreactivity to markers such as GABA, glutamate, tyrosine hydroxylase, calretinin, calbindin, taurine, parvalbumin, Trk B and Trk C is localized in the prospective ganglion cells from 10-11 wg onward (Jotwani et al 1994; 1998; Nag and Wadhwa, 1996; 1997; Nag et al 1998; Nag and Wadhwa, 1999). A transient increase of neuronal nitric oxide synthase in ganglion cells occurs between 18-25 wg of fetal human retina (Wadhwa and Nag, 1999). Synaptic development in fetal human retina by labeling with markers of synaptogenesis (synaptophysin and syntaxin-1) showed that IR to both markers is present in the central inner plexiform layer at 11-12 wg (Nag and Wadhwa, 2001). These salient features of human retinal development have been recently reviewed (Nag and Wadhwa, 2006).

Expression of Cdc2-kinase, a cell cycle regulator, showed a typical developmental profile starting from embryonic day 16- to 3w postnatal age. Its expression was evident in post mitotic neurons also where it was found to make a functional complex with an adult age specific cyclin D1 and thus, suggesting implication of cdc2-kinase in neuronal functions of mature brain also (Tamaru et al, 1993).

Expression of PFK2, a critical glycolytic factor, was found to undergo a change from a liver type PFK2 isoform expressed at embryonic stage to a typical brain type PFK2 after 1w postnatal age (Pandey et al, 2005a), implying that transition from a foetal anaerobic environment to a typical aerobic respiration in the neonates induces a series of metabolic events associated with brain development. In addition, it has also been described that neonatal hypothyroidism renders developing brain susceptible for poor growth and oxidative stress during the critical period of brain development in mice (Pandey et al, 2005b).

Insect neurochemistry

Allatinhibin and allatotropin two neuropeptides from the insect brain *Manduca sexta* were isolated by *in vitro* technique and its further characterization were done by different chemical and enzymatic and other molecular separation techniques (Unni et al 1991; 1993). Two analogues of allatotropin were synthesized, which correspond to active fragment, amino acids 5-13 of the natural Mas-AT with substitution of Nor-leucine for methionine at 7^{th} and 8^{th} positions (Unni, 1995). Various stimulation of juvenile hormone biosynthesis by juvenile hormone acids and larval haemolymph of *Manduca sexta* has been further investigated (Unni, 1999; 2004). Influence of juvenile hormone on Eri silkworm, *Philosamia ricini* with special reference to carbohydrate metabolism has also been investigated (Choudhury and. Unni, 1998). Acetylcholinesterase activity in the head of semi

domesticated tropical silkworm, *Antheraea assama* has also been investigated (Das et al 2004). Insect hormone analogues and neuropeptides-induced upregulation of general metabolism in silkworms suggests improvement in the silk protein/fiber production.

Conclusions

Despite having several institutions with deep commitment to neurochemistry research, easy and abundant availability of funds from Govt. agencies and private sources for modern research equipments, consumables and other infrastructure facilities, it is painful to have to conclude that Indian neurochemistry still lags far behind at the international scenario. This area of research is fraught with lack of imagination and serious initiatives, and the body of literature amounts to mere follow-up or search for implications on discoveries elsewhere, as is reflected on the impact of papers published from India in terms of its citations and follow-up by others in the field. Equipped with a rich traditional knowledge on superior functions of the nervous system, diversity in population, and having unique nervous systems health problems in the country, we are at an advantageous position to contribute significantly in this filed. It is time for a serious reconsideration on our priorities, responsibilities, and accountability, and to consolidate on the resources, concentrate on relevant areas, and gear up our activities in this filed.

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