DOWNLOAD PDF NGF AND RELATED MOLECULES IN HEALTH AND DISEASE

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There are also chapters on the involvement of NGF and related molecules in neurological diseases, including Huntington's disease, the multiple sclerosis-like model of experimental allergic encephalomyelitis, peripheral neuropathies, neuroblastoma, Parkinson's disease, Alzheimer's disease, and even motion sickness syndrome, also psychiatric.

This article has been cited by other articles in PMC. Abstract Promising studies suggest that defects in synaptic plasticity detected in schizophrenia may be linked to neurodevelopmental and neurodegenerative abnormalities and contribute to disease-associated cognitive impairment. We aimed to clarify the role of the synaptic plasticity regulatory proteins, nerve growth factor NGF and its receptor NGFR in the pathogenesis of schizophrenia by comparative analysis of their blood levels and functional single nucleotide polymorphisms SNPs in genes encoding these proteins NGF and NGFR in schizophrenia-affected and healthy subjects. In conclusion, our results demonstrate association of schizophrenia with the rs, rs and rs, rs, rs as well as with the decreased blood levels of corresponding proteins. This complex disorder with still unclear etiology and molecular pathomechanisms is characterized by both neurodevelopmental [3] and neurodegenerative abnormalities [4] , [5] , [6] and cognitive impairments [7] linked to behavioral changes [8]. Promising studies suggest that defects in synaptic plasticity detected in schizophrenia [9] may be linked to neurodevelopmental and neurodegenerative abnormalities [10] , [11] , [12] , [13] and contribute to cognitive impairment associated with this disease [14] , [15] , [16] , [17]. Therefore, study of synaptic plasticity regulatory genes in schizophrenia represents a special interest, as it can provide insight into molecular mechanisms of schizophrenia-associated cognitive dysfunction and sufficiently contribute to development of target-oriented therapy for this disorder. In addition, neurotrophins play an important role in the immune response [19] , which is upregulated in schizophrenia [20] , [21]. In our recent study we demonstrated implication of genetic variation of brain-derived neurotropic factor, modulators of brain plasticity in cognitive processes [15] , in pathogenesis of schizophrenia [12]. Other important members of neurotrophin family are nerve growth factor NGF and its receptor NGFR , the essential mediators of synaptic and morphological plasticity, neuronal growth, survival, and differentiation, especially in the developing brain [18] , [22]. The mature form of nerve growth factor NGF derives from a precursor, proNGF, which was recently discovered to exert crucial brain functions responsible for mood and cognitive activities [23]. Moreover, decreased blood levels of NGF among first-episode schizophrenia patients compared to healthy subjects have been observed [25] , [26]. Interestingly, it has been shown that chronic cannabis abuse raises NGF serum concentrations in drug-naive patients with schizophrenia compared to healthy control subjects [27]. The potential implication of NGFR in schizophrenia either at protein or genetic levels has not been studied yet. Materials and methods 2. Study population A total of unrelated Caucasian individuals of Armenian nationality living in Armenia chronic schizophrenia patients, 25 first-episode schizophrenia patients and healthy subjects were enrolled in this study. They were diagnosed as paranoid schizophrenics ICD code: Chronic patients were treated with haloperidol and first-episode patients were antipsychotic-free. They passed a special examination by two independent experienced psychiatrists to establish no personal or family history of mental disorders. Fifty-two schizophrenia-affected subjects and sixty-seven healthy subjects were nicotine-dependent tobacco cigarette smokers.

Chapter 2 : Nerve growth factor and its receptor in schizophrenia

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This article has been cited by other articles in PMC. Abstract Nerve growth factor NGF is the firstly discovered and best characterized neurotrophic factor, known to play a critical protective role in the development and survival of sympathetic, sensory and forebrain cholinergic neurons. NGF promotes neuritis outgrowth both in vivo and in vitro and nerve cell recovery after ischemic, surgical or chemical injuries. Recently, the therapeutic property of NGF has been demonstrated on human cutaneous and corneal ulcers, pressure ulcer, glaucoma, maculopathy and retinitis pigmentosa. NGF eye drops administration is well tolerated, with no detectable clinical evidence of systemic or local adverse effects. The aim of this review is to summarize these biological properties and the potential clinical development of NGF. A Brief Overview NGF is a neurotrophic factor discovered in for its properties of promoting growth and survival of peripheral sensory and sympathetic nerve cells of mammalians, human included [1 , 2]. Subsequent studies carried out in laboratory animals have shown that peripherally innervated tissues produce and release NGF which is retrogradelly transported by specific receptors, to finally provide a protective action and a functional neuronal integrity [3]. The effect of NGF on target cells depends on the ratio of these two receptors co-distributed on cell surface [7]. Subsequent studies carried out in animal models showed that exogenous NGF administration can promote peripheral nerve growth and re-establish the functional activity of peripheral nerve fibers and damaged neurons [8 - 11]. However, the attempts of using exogenous NGF in human neuropathies were not encouraging since intravenously NGF peripheral administration caused unwanted side-effects, in addition to the desired stimulation of nerve fiber outgrowth [12]. The first evidence that NGF might have therapeutic properties, was obtained with the topical administration on human cutaneous, corneal and pressure ulcers [13] while the effects on Glaucoma [14], Maculopathy [15] and Retinitis Pigmentosa represent recent outcomes [16]. The topical application provided successful outcomes and resulted to be well tolerated, since no detectable clinical evidence of systemic or local adverse effects were observed [17 , 18]. These results paved the way for the development of clinical trials of NGF in Ophthalmology and cutaneous ulcers. Since the discovery, NGF attracted clinicians for potential application in different fields, firstly in the treatment of neurological disorders and later on as healing-promoting agent in the ulcer management [13]. The reparative properties of NGF have been tested in several conditions of healing. Successful NGF effects were obtained in impaired healing due to experimental injury or autoimmune disorders and during the topical long-term NGF application in experimental as well as human pressure and cutaneous ulcers [21 - 24]. The profibrogenic activity of NGF was later on confirmed in several in vitro studies, indicating the fibroblast as target cells for the NGF-driven healing [25]. A consistent number of patients suffering from corneal ulcers due to neurotrophic impairments associated with herpetic infections, chemical burns, local surgery, topical anesthetic abuse and diabetes have been treated until now [13 , 17]. The topical application of murine NGF promoted a complete healing of the ulcer associated with a recovery of the corneal sensitivity, providing a long term benefit to trigeminal innervations [26]. This observation is in line with a previous study showing that NGF is expressed during fibrotic liver injury and may regulate the number of activated Hepatic Stellate Cells myoFBs-like cells via induction of caspase3-mediated apoptosis [28]. In a pilot study carried out in mice with acute self-limiting pro-fibrotic liver injury, NGF administration allowed recovery from injury by means of myoFB apoptosis and absence of trkANGFR [29]. Due to the lack of effective pharmacological agents for balanced tissue repairs except for the short-term steroid application , these new findings suggest that NGF might be a suitable therapeutic tool in conditions with impaired tissue healing. These findings were also provided by a previous study on cultured keratocytes, supporting the new therapeutic strategies to overt

fibrosis by modulating myoFB survival in fibrotic tissues [30].

Chapter 3 : Nerve Growth Factor: A Focus on Neuroscience and Therapy

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NGF and Related Molecules in Health and Disease. Luigi Aloe, and Laura Calza. Volume , Pages () Previous volume. Next volume. Nerve growth factor.

Nerve growth factor NGF is known for playing a critical protective role on a number of brain neurons in mammals, including humans. NGF can be delivered to the CNS via nasal route and has a neuroprotective action in case of neurodegenerative diseases. The aim of this study is to investigate for the first time whether purified NGF can play a neuroprotective role on human brain neurons affected by neurodegenerative diseases when administered via nasal route. Two female patients, both affected by frontotemporal dementia FTD associated with corticobasal syndrome CBS at different stages of disease progression, received a daily intranasal NGF spray for one year. These findings suggest the potential neuroprotective role of IN-NGF administered in case of neurodegenerative diseases. INTRODUCTION Nerve growth factor NGF is the first discovered and best characterized member of the neurotrophin family [1], known for playing a critical protective role in the development and survival of sympathetic, sensory and basal forebrain cholinergic neurons in mammals, including humans [2â€”4]. For over three decades, studies conducted on NGF have been focusing on its biochemical and molecular characterization. Since the discovery by Rita Levi-Montalcini, other researchers demonstrated the potential therapeutic role of NGF in the treatment of human diseases in the central nervous system CNS and peripheral nervous system PNS [5â€”7], and some trials have shown encouraging results. Inevitable loss of neurons and synapses in neurodegenerative diseases occurs over time, leading to cognitive decline. NGF does not cross the blood-brain barrier if injected subcutaneously or intravenously; therefore, an alternative delivery method is required. Intranasal delivery of NGF has so far been sufficiently investigated in animal models [11â€”21] and only recently in humans, as demonstrated in a recent study on intranasal administration of NGF in a brain injury [22]. Some of these studies demonstrated that NGF has the ability to rescue recognition memory deficits in mice [16] and to slow amyloid neurodegeneration [12]. Furthermore, recent studies have shown an active link between the nasal pathway and the spinal cord in the delivery of NGF to the CNS, thus demonstrating the neuroprotective ability of NGF to support injured neurons in a model of spinal cord injury [13â€”15, 21]. Different ways of direct delivery of NGF to the CNS have been investigated in humans and animal models, including direct CNS infusion [26, 28], gene therapy approaches [24â€”25], cell-based delivery using stem cells [29], and application of an encapsulated cell biodelivery device [30]. All these approaches have the restriction of being invasive. Hence, the aim of this study is to investigate for the first time whether purified NGF would play a neuroprotective role on human brain neurons affected by neurodegenerative diseases when administered via nasal cavity. What is nerve growth factor? NGF is the founding member of the neurotrophins family of proteins. In , while working in the Victor Hamburger laboratory at Washington University, Rita Levi-Montalcini discovered that sarcoma tissue in mice produced a soluble factor that promoted the growth of nearby sensory and sympathetic ganglia [1, 5]. Working on a team with Stanley Cohen, they isolated the substance responsible for this phenomenon and named it nerve growth factor. Many cells of the immune-hematopoietic system also produce and utilize NGF: Furthermore, the largest source of NGF is inside the mouse salivary glands [33]. The effect of NGF on target cells depends on the ratio of these two receptors co-distributed on cell surface [35]. These data place CD2AP as a major intracellular signaling molecule coordinating NGF signaling to regulate collateral sprouting and structural plasticity of intact adult axons [36]. FTD is now one of the most common forms of dementia in persons younger than 65 years [40]. Recent studies have identified the presence of TAR DNAâ€”binding protein TDP , which may be the primary disease protein underlying the ubiquitin-positive cases [42]. Corticobasal syndrome CBS is a progressive neurological disorder that may involve the motor system, cognition or both [48â€—49]. Typically, it starts as a movement disorder, with affected individuals showing a unilateral paucity of movement and muscle rigidity with or without a tremor. Previously considered

as a distinct clinicopathologic entity [50], then termed corticobasal degeneration CBD [51]. Clinicopathologic studies have since revealed that the originally described clinical features of CBD, now called CBS, are often due to other pathologies. From recent studies, 4 CBD phenotypes emerged: The cognitive associated signs [49] are alien hand syndrome, apraxia, acalculia, and visual-spatial impairment. CBS language associated signs [49] include progressive aphasia. METHODS The rationale of nasal route delivery Nasal delivery is easy and non-invasive and the olfactory mucosa is in direct contact with the brain and cerebrospinal fluid by the olfactory and trigeminal pathways [57]. An accurate literature review shows that NGF and other large molecules can reach neurons of the CNS, particularly the brain, via nasal route [11, 17â€”19]. These studies Frey II et al. Subsequent studies about intranasal delivery of NGF and others molecules NGF-like to the brain have reinforced the concept [15, 16, 20, 21]. The anatomical connection between the nasal cavity and central nervous system structures consists of two possible routes. One is associated with the peripheral olfactory system connecting the nasal passage with the olfactory bulbs and rostral brain regions; the other goes through the peripheral trigeminal system connecting the nasal passage with the brainstem and spinal cord region [15, 21, 57, 58]. For further details of purification see: Informed consent was provided by their caregivers. History, neurological examination, review of magnetic resonance brain imaging, and PET-scan within recent years, clinical dementia rating, Mini-Mental State Examination MMSE , Simpson-Angus scale, and verbal fluency test were performed. Memantine and idebenone therapy was discontinued 6 months before the beginning of NGF-treatment in the experimental NGF-study Group Case 1; Case 2 , in order to be clearly and sufficiently convinced that the observed effect was due to the action of NGF. The long-term administration of NGF was limited only 1 time daily, 1 spray per nostril two daily sprays in the morning to avoid the undesired effects. Increasing dosage some adverse effects have occurred as reported in Table 1. All adverse effects were attenuated h after onset. Start of disease Heart arrhythmia in treatment with beta-blockers. Non-L-dopa responsive extrapyramidal syndrome on the right side with a mild right hand tremor, dysgraphia, dyscalculia, speech disorder progressive aphasia. Absence of other associated pathologies. Aged 60, in early stage CDR scores 1. Signs and symptoms overlapping with Case 2. Aged 64, in middle stage CDR scores 2. Signs and symptoms overlapping with Case 1. Absence of other associated pathologies in both patients. Amyloid PET-scan was negative in all patients. In recent years, evidence of the microbial origin of various chronic inflammatory disorders, including several neurodegenerative, neuropsychiatric and other systemic disorders, has been steadily growing. The same hypothesis could be applied to all neurodegenerative diseases. Images of tracer distribution were acquired over a 30â€”60 min period. The tomographic images were acquired in the axial, coronal and sagittal sections with a thickness of 4. Red color indicates more 18 FDG-use than blue. Regionally specific effects were compared using linear contrasts. Resulting foci were characterized in peak height u. Patients were more responsive, had slight improvement in speech and in motor rigidity. A greater responsiveness mood and eye opening was observed during the first 15â€”20 days of each monthly treatment. Over time the denaturation of the molecule pushed down neurological performances mood and eye opening. In the post-treatment phase, the mean rate of MMSE decline was 2. During the first 3 months of stopping NGF-treatment, a decrease in levels of attention and mood tone was observed. After 1 year of stopping NGF-treatment and no other conventional medication, there was a general worsening of the patients. MMSE scores worsened again with a mean annual rate of decline 4. This marked a clear return to pre-treatment conditions. The hypothesis of the positive effect due to NGF is also suggested by the case of two other patients in the control group who were treated with memantine and idebenone therapy and did not show similar improvements. Rigidity assessment Rigidity assessment was performed using the Simpson-Angus Scale, currently used to assess pseudoparkinsonism. The severity grades of each item are rated using a 5-point scale. We focused only on the evaluation of elbow rigidity. The resistance to this procedure has also been rated. Post-treatment assessment after a month period of NGF-treatment showed a slight improvement in rigidity: In Case 1 patient in middle stage , rigidity improved from marked to moderate. In Case 2 patient in early stage , rigidity improved from moderate to slight. The

control group did not show similar improvements. After 1 year of stopping NGF-treatment and no other medication, there was a clear return to pre-treatment conditions. In detail, Case 1 middle stage showed improvement in word recruitment and a decrease in phonemic paraphasias; e. Case 2 early stage showed improvement in fluency and word recruitment. After 1 year of stopping NGF-treatment and no other medication, speech assessment showed a score worsening of 2. Dose-dependent adverse effect The following undesirable effects were observed: All adverse effects were attenuated within h maximum following the administration. No side effects were observed in a 18â€”24 months period of follow-up after the last dose of NGF was administered. One year after the interruption of NGF treatment and no other medication , PET-images show a clear deterioration of the global framework, compared to the previous PETs. The brain areas show a significant reduction of the activities Figs. Red color indicates more FDG-use than blue. A Before treatment, note the FDG-uptake reduction in the following brain areas: D After 1 year of stopping NGF-treatment, the brain areas showed a significant reduction in global activities. A Before treatment, note FDG-uptake reduction in the following brain areas: DISCUSSION As reported above, NGF is an endogenous neurotrophin that exerts trophic and differentiative activity on neurons of the central and peripheral nervous systems with neuroprotective and regenerative effects observed in neurodegenerative diseases or following injury. Numerous data show the protective role of NGF in case of neurodegenerative diseases in human models too. Despite that, invasive routes of administration are not optimal for clinical use [24â€”26, 28, 30], i. Any adverse effect was attenuated h after the administration and no side effects were observed in a 18â€”24 months period of follow-up after the last dose of NGF was administered. However, the clinical observation presented in this report needs to be confirmed on a large scale clinical trial to better understand the potential neuroprotective role of NGF in neurodegenerative diseases and other pathologies of central nervous system brain injury, spinal cord injury, ischemic damage when administered via nasal route. In the last years some authors have demonstrated the possibility to synthesize in a laboratory a recombinant human NGF for clinical use [64].

Chapter 5 : NGF and Related Molecules in Health and Disease: Volume : Luigi Aloe :

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Chapter 7 : Meeting titles and venues - NGF MEETINGS

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