MORPHOLOGICAL CHANGES OF ORGANS IN DIABETES

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Abstract
In this article, the morphological changes that occur during diabetes mellitus in humans are studied and analyzed.

Kyewords: morphological changes, diabet, human body

What is Morphological changes?

Morphological change refers to change(s) in the structure of words. Since morphology is interrelated with phonology, syntax, and semantics, changes affecting the structure and properties of words should be seen as changes at the respective interfaces of grammar. On a more abstract level, this point relates to linguistic theory. Looking at the history of morphological theory, mainly from a generative perspective, it becomes evident that despite a number of papers that have contributed to a better understanding of the role of morphology in grammar, both from a synchronic and diachronic point of view, it is still

seen as a "Cinderella subject" today. So there is still a need for further research in this area.

Generally, the field of diachronic morphology has been dealing with the identification of the main types of change, their mechanisms as well as the causes of morphological change, the latter of which are traditionally categorized as internal and external change. Some authors take a more general view and state the locus of change can be seen in the transmission of grammar from one generation to the next (abductive change). Concerning the main types of change, we can say that many of them occur at the interfaces with morphology: changes on the phonology–morphology interface like i-mutation, changes on the syntax–morphology interface like the rise of inflectional morphology, and changes on the semantics–morphology like the rise of derivational suffixes. Examples from the history of English (which in this article are sometimes complemented with examples from German and the Romance languages) illustrate that sometimes changes indeed cross component boundaries, at least once (the history of the linking-s in German has even become a prosodic phenomenon). Apart from these interface phenomena, it is common lore to assume morphology-internal changes, analogy being the most prominent example.

A phenomenon regularly discussed in the context of morphological change is grammaticalization. Some authors have posed the question of whether such special types of change really exist or whether they are, after all, general processes of change that should be modeled in a general theory of linguistic change. Apart from this pressing question, further aspects that need to be addressed in the future are the modularity of grammar and the place of morphology.

What this excursus has shown then is that on closer inspection morphological change is not that easy to define, which depends on the fact that the characteristics of morphology interrelate with phonology, syntax, and semantics. So it is not isolated from other parts of the grammar, and it cannot be entirely divorced from phonological, syntactic, and semantic concerns. But this is also exactly why morphology and morphological change are so fascinating.

Studying morphological change can provide a window on the human mind from a historical perspective, at least for those who are also interested in cognitive and theoretical aspects of language. For example, speakers of Middle English who were presented with the Old French loan word crevice (Modern French écrévisse) for the first time tried to find a formal correspondence in their mother tongue.1 on the basis of the semantics of the word and changed the shape of the word accordingly: this is how crayfish came into being (and even developed into a verb via conversion!). From examples like these we see what speakers do when they are exposed to (new) data, how they process and produce language which, after all, is the basis for acquiring linguistic

competence. What we see again is that borrowing can be seen as being part of morphological change because borrowed items affect the content of the lexicon.

Patients with diabetes experience vitreous degeneration, characterized by "precocious" liquefaction and posterior vitreous detachment. Biochemical studies have detected that hyperglycemia alters vitreous collagen, changes that might be responsible for the observed vitreous degeneration. This study was undertaken to identify if there are morphological changes within the vitreous of diabetic patients that are consistent with the biochemical data and to identify how these could underlie the observed clinical phenomena.

In normals, a transition was observed from a homogeneous structure in youth to one that contained fibers in middle-age, which degenerated and were associated with significant liquefaction in old age. In the diabetic child, the vitreous structure contained prominent fibers whose appearance was similar to middle-aged normals and not the agematched controls. This study characterizes the morphological manifestations of precocious senescence of vitreous in a patient with diabetes. The abnormal vitreous fibers are likely the result of biochemical changes in collagen that are related to hyperglycemia - a phenomenon that could be inhibited by drug therapy.

Diabetes has profound effects on extracellular matrices and connective tissues throughout the body, primarily via non-enzymatic glycation and abnormal crosslinking of collagen. These biochemical changes induce so-called "precocious senescence" of various tissues and organs in the diabetic patient.

It is known that vitreous in diabetic patients undergoes precocious liquefaction and posterior vitreous detachment. Furthermore, abnormal collagen crosslinking and non-enzymatic glycation have been detected in vitreous of diabetic humans. Such destabilization of the molecular network within vitreous contributes to the aforementioned clinical observations. Since new blood vessels arising from the retina grow into the posterior vitreous cortex, any structural abnormalities within vitreous could result in traction upon these new blood vessels. Subsequent vitreous hemorrhage and traction retinal detachment are the sequelae causing vision loss in advanced proliferative diabetic retinopathy. This investigation was undertaken to determine whether structural abnormalities exist within vitreous of patients with diabetes that are consistent with the biochemical changes and the observed clinical abnormalities.

Such morphologic abnormalities in the corpus vitreous of a child with only a 5-year history of diabetes and no diabetic retinopathy are quite striking. However, studies have shown that 40-52% of children with 5-year duration of diabetes have joint contractures that result in limited joint mobility. This is particularly interesting when one considers that vitreous and articular cartilage are both composed of type II collagen. Furthermore,

there is a strong positive correlation between the extent of limitation in joint mobility and the degree of diabetic retinopathy.

The reported morphological findings in vitreous are consistent with clinical observations of vitreous degeneration in diabetes. Such changes are also consistent with the phenomenon of "precocious senescence" of other tissues in diabetic patients. Studies by Hamlin et al. and Monnier et al. have linked the development of precocious aging changes to biochemical abnormalities of collagen related to diabetes and hyperglycemia. The findings in vitreous presented herein may also be the result of abnormal collagen cross-linking and nonenzymatic glycation of vitreous, phenomena that have been identified in diabetic patients and that have been described as the cause of collagen fibril aggregation in other tissues.

In a recent morphological study on the hypothalamus of male rats one year after induction of diabetes by streptozotocin, we described lesions in the arcuate nucleus and median eminence. Following a preliminary investigation on the testes of the same animals, we found various degrees of testicular change. In order to investigate further the possible relationship between these two findings, we have performed a systematic light and electron microscopic study of the hypothalami, pituitaries and testes of rats after one year of streptozotocin-induced diabetes. Immunohistochemical identification of luteinizing hormone (LH) and luteinizing hormone-releasing hormone (LHRH) was also carried out on semi-thin and thin sections of the pituitaries and hypothalami.

Further elucidating the molecular events underlying this process is important in view of the role that vitreous synchisis (liquefaction) and syneresis (collapse) can play in exacerbating proliferative diabetic retinopathy. New vessels that have grown into the vitreous cortex prior to these developments will experience traction, inducing vitreous hemorrhage and/or traction retinal detachment. Therapeutic regimens designed to inhibit or limit the degree of vitreous degeneration in diabetes could thus have salubrious effects in preventing severe visual loss, since studies have shown that separation of the vitreous cortex from the internal limiting lamina of the retina is associated with these blinding sequelae. Alternatively, an innocuous method to induce posterior vitreous detachment prior to the growth of new vessels into the posterior vitreous cortex could be very beneficial as preventive therapy. This concept is supported by the findings that new vessels that grow in areas where vitreous is already detached have an "abortive" appearance and are not likely to be clinically significant. Indeed, part of the therapeutic effect of panretinal laser photocoauglation may be the induction of posterior vitreous detachment, so that any subsequent neovascularization will not be able to grow into the vitreous cortex, thus having a better prognosis.

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