REVIEW ARTICLE

ACTA Vol. 10, No. 2, 2012, 291-305
NEUROPSYCHOLOGICA

Received: 03.04.2012
Accepted: 28.06.2012

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RESEARCH ON MEMORY IN SPECIFIC LANGUAGE IMPAIRMENT (SLI)

Grażyna Krasowicz-Kupis[1,2]
[1] Institute of Psychology, Maria Curie-Skłodowska University, Lublin, Poland
[2] Educational Research Institute, Warsaw, Poland.

SUMMARY

The term "Specific Language Impairment" (SLI) refers to disturbances in acquiring the skills needed for language, especially the syntactic and morphological components, in children who are not diagnosed with structural anomalies of the brain, hearing impairments, or significant general learning impairments, and who have not been deprived of contact with other people (Rapin 1996). Several important theories on the pathomechanisms of SLI include memory deficits, especially working and phonological memory. Of particular interest is the procedural deficit hypothesis advanced by Michael T. Ullman and Elizabeth I. Pierpont, which attempts to integrate neuronal, cognitive, and linguistic research on SLI and explain the heterogeneity of the deficits ascribed to this disorder, and will be described in this article. The importance of testing various aspects of memory in individual diagnosis will also be analysed, with particular reference to the co-occurrence of SLI and dyslexia.

Key words: short-term memory (STM), phonological memory, procedural deficit, co-occurrence

SPECIFIC LANGUAGE IMPAIRMENT AS A DEVELOPMENTAL DISORDER

The term "Specific Language Impairment" (SLI) refers to a problem in the development of language, without intellectual limitations, hearing impairments, or motor, social-emotional, or neurological problems (Stark & Tallal 1988).

SLI belongs to the category of specific disorders, which means that the demonstrated level of language competence is substantially below the level of non-verbal intelligence. In other words, the language deficits are not caused by an intellectual deficit. A review of the definitions advanced since the 1990s shows virtually unanimity in this respect.

A distinct majority of researchers also agree that in the case of SLI, the limitations in language skills are not explained by some obvious factor, such as hearing impairment, a low IQ, neurological damage, or psychological and emotional problems (Tallal, Stark & Mellits, 1985; Bishop, 2008). Thus the key diagnostic criteria are primarily the exclusion criteria, as is the case with other developmental disorders (e.g. dyslexia, cf. Krasowicz-Kupis, 2008).

Bishop (2008) defines SLI as a distinct speech deficit without other accompanying developmental deficits. Rapin (1996), in his definition, refers to the exclusion criteria and defines SLI as the abnormal acquisition of language in children who have not been diagnosed with structural brain damage, hearing impairments, or significant general learning disorders, and who have not been cut off from contact with other persons. SLI can also be described as a disturbance featuring a slow, abnormal pattern of language development, which appears when more than one element of the language system breaks down, thus blocking the system's natural compensatory potential (Parisse & Maillart, 2009). It should be emphasized that without intervention this disorder has a negative impact on academic achievement, and also affects the course of life, education, and professional career.

According to most of the definitions mentioned above, the symptoms of SLI result from language system deficits, which can involve all its subsystems, but especially those involved with the grammar of the native language. These deficits affect various aspects of speech, so the symptoms of SLI can be expressive or receptive in nature. The expressive symptoms include:

- errors in the articulation of phonemes (pronunciation);
- errors in inflection (in English, this pertains primarily to verb flexion);
- limited vocabulary;
- difficulty in learning new words;
- difficulty in accessing words in memory
- difficulty in building sentences with a degree of complexity appropriate to the child's age.

The receptive symptoms include:

- difficulties in understanding words;
- difficulties in understanding sentences, especially complex ones.

In particularly difficult cases the symptoms can involve all elements of the language system: phonological, morphological, syntactical, lexical, semantic, and pragmatic (Bishop, 2006; Leonard, 2006). It is a matter of some controversy, however, as to whether or not semantic and pragmatic symptoms should be included in SLI. Some authors (e.g. Cummings, 2008) distinguish a relatively independent disorder, called Pragmatic Language Impairment (PLI).

The clinical picture of SLI is not entirely clear, since it is characterized by several very different profiles of language development. Moreover, the profile of language competence in a particular child is dynamic in nature, and can undergo fundamental changes in the course of development.

Many researchers and clinicians argue that SLI cannot be treated as an isolated language problem, since in practice language deficits are rarely unaccompanied by other problems, motor and cognitive, such as problems with coordination or motor fluency, problems with working memory, or difficulties in processing time (Thomas, 2005; Ullman & Pierpont 2005; Pąchalska et al., 2007), though the language problems are typically dominant, and seriously interfere with the individual's normal functioning.

In characterizing children with SLI it is commonly stated that they begin to speak late, at preschool age they have still not caught up with their contemporaries in speech and language, and in the first years of primary school they can have problems with learning to read and write, so that throughout their schooling their academic achievements are below average (Smoczyńska, 2006). The problems of this group of children - especially their school difficulties - have a negative impact on their development, not only in the cognitive sphere, but also in respect to emotional and social functioning, which means that SLI can adversely affect the individual's entire life (Leonard, 2006).

The frequency of occurrence of SLI is a subject of constant controversy. Leonard (2006) showed, after analyzing the estimated frequency factors among preschool children, that the rate ranges from 1.5% (using very strict criteria), through 5% (DSM-IV), to somewhat over 7% (according to the very thorough analysis of Bruce Tomblin and associates in the 1990s; see Tomblin, Records, Buckwalter, Zhang, Smith et al., 1997), or even higher.

The problems involved in estimated the frequency of occurrence of SLI result from three different kinds of difficulties (Leonard, 2006; Snowling & Hulme, 2009). The first of these is the diversity of the not-always-clear diagnostic criteria, resulting from differing understandings of the concept of SLI (for example, should the definition include children with problems in the production and reception of speech, or only expression?), which led Hulme & Snowling (2009) to suggest that the percentage of children with SLI is somewhere between 3% and 6% of the population.

The second difficulty results from the fact that SLI is a dynamic disorder: some symptoms can be overcome, others persist or recur later in life. Thus one and the same person may be counted as suffering from SLI at one stage in their life, and not at another (Tomblin et al., 1997; Hulme & Snowling, 2009). The third difficulty pertains to the breakdown by gender. In this respect the literature is unan-

imous in one thing: SLI is more common in boys. However, the estimated proportions vary considerably. Tomblin et al. (1997), for example, give a figure of 1.33:1, boys over girls, though previously the figures had been much higher: 3:1 or 4:1 (Hulme & Snowling, 2009). Recent research in the US (Cheuk, Wong & Leong, 2005), which covered children from 4 to 8 years old, found a proportion 3 boys to 1 girl. The question would appear to be still open.

The problem of memory functions in SLI appears in the context of both symptoms and explanatory mechanisms. In descriptions of the symptoms we often encounter references to a deficit in verbal memory, manifesting as a problem in accessing words, due to a limited number of words available in the active lexicon. A short-term memory deficit is also listed as an accompanying symptom. On the other hand, memory deficits figure largely in hypotheses regarding the mechanisms underlying SLI, as will be discussed below.

THE CAUSES OF SLI

Genetic and neurodevelopmental etiology of SLI

In the effort to identify the primary causes of SLI, the search for its genetic and neurodevelopmental sources has become a priority, as is the case of other developmental disorders, is given to finding (Norbury et al., 2008).

The search for the genetic background of SLI has led researchers to focus on family studies, including twin studies. The results seem to confirm that there is indeed a genetic foundation, but the interpretation is still open to question (Parisse & Maillart, 2009; Leonard, 2006; Hulme & Snowling, 2009; Byrne, Delaland, Fielding-Barnsley, Quain, Samuelsson et al., 2002; Kovas, Hayiou-Thomas, Oliver, Dale, Bishop et al., 2005).

Some very interesting results have been provided by research in molecular genetics, looking for genetic mutations associated with SLI, including work on the famous KE family (Lai, Fisher, Hurst, Vargha-Khadem & Monaco, 2001; Hulme & Snowling, 2009). Detailed studies of the genotype of persons with SLI have not confirmed the existence of any connection between this disorder and a particular gene. It has been shown that persons with SLI present more risk factors than do healthy controls, in the form of different quantitative trait loci (QTLs, that is, chromosome regions particularly associated with a disorder) that may tend to produce a language disturbance. The QTLs most commonly indicated for SLI include chromosomes 16, 19, and 13, less often 10 (SLI Consortium, 2002; Monaco & Newbury, 2007; Hulme & Snowling, 2009). Thus the reported results indicate that SLI is a disorder with many causes, and the differentiation of the symptoms and forms of the disorder may reflect a diverse genetic etiology (Hulme & Snowling, 2009).

In the search for the neurostructural and neurofunctional background of SLI, one of the first major achievements occurred when Albert Galaburda and associates (Galaburda, Sherman, Rosen, Aboitiz & Geschwind, 1985) pointed to the

symmetry of the planum temporale in persons with SLI (as opposed to the asymmetry considered typical for the normal development of speech). Their conclusions were confirmed in neuroimaging studies in the 1990s (Plante. Swisher, Vance & Rapcsak, 1989; Jernigan, Hesselink, Sowell & Tallal, 1991; Plante, Swisher & Vance, 1991) using magnetic resonance imaging (MRI). Most of these studies have found atypical asymmetry or even symmetry in the perisylvian region, most often involving the planum temporale and the inferior frontal gyrus.

Elena Plante, Kenneth Shenkman, and Melinda M. Clark (1996) suggested that asymmetries take shape in the third trimester of pregnancy, and the level of testosterone may be of fundamental significance. John L. Locke (1994) also considered the role of atypical asymmetry of the planum temporale as a causal factor for language disturbances, and argued that these can be explained as the effect of the compensatory use of the right hemisphere for language functions, which causes increased anatomical and functional symmetry between the two hemispheres in children with SLI. However, the research done by Laurie Gauger, Linda Lombardino and Christiana Leonard (1997) did not confirm this hypothesis, since they found that the symmetry in question is the result rather of the smaller dimensions of both hemispheres. Their research found interesting anatomical differences in respect to the symmetry of the temporal lobes in a group of SLI children, and indicated that some parts of the brain associated with language functions in the left hemisphere – the planum temporale and the pars triangularis – differ in both shape and size in children with SLI, compared to their healthy contemporaries. These results point up the structural and functional connection of brain structures and language skills, and confirm earlier reports that developmental language disorders correlate with changes in the structure of the brain.

Some interesting research has also been conducted by Christiana Leonard et al. (2002), who found that Heschl's gyri in the left hemisphere of children with SLI have less surface area than in children with dyslexia, while the planum temporale shows a greater tendency to symmetry.

In recent years research has also been done on patients from 5 to 17 years of age, using the newest generation of three-dimensional MRI technology (Soriano-Mas et al. 2009). Measurements have been made of the volume of gray and white matter, and the anatomical structures of particular brain regions have been compared using voxel-based morphology (VBM). Patients with SLI presented with a greater volume of gray and while matter, especially in the subgroup of younger children (to age 11). A particular increase in the amount of gray matter was noted in the right perisylvian region and in the occipital lobes. Comparative studies with subjects matched for age found greater differences in younger children as regards expanded gray matter in the rhinoencephalon, the temporo-parietal cortex, the caudate nucleus, the pre-motor area, and the precuneus, as well as the white matter in the frontal and temporal lobes (Soriano-Mas et al. 2009). Of particular interest is the fact that in older children (over 11 years of age) these differences were not significant. This confirms the dynamic nature of SLI, and points up the necessity to take account of age in evaluating the neuroanatomical foundations of this disorder.

Further anatomical changes that have been explored in connection with SLI include the corpus callosum, which is smaller in those children with SLI who were at risk in the prenatal period. This may indicate that the brains of children at risk for disturbances of language development are particularly exposed to prenatal environmental risks. Studies of the basal ganglia have also found reduced volume and bilateral changes in the structure of the cerebellum in persons with SLI (Hulme & Snowling, 2009). Many neurofunctional studies have confirmed the existence of a pattern of insufficient or excessive activation during language processing of these same areas where structural differences have been found in neuroanatomical studies of children with SLI.

Neurofunctional studies most often discover atypical signals related to auditory perception in the differentiation of similar sounds, as in the case of children with dyslexia (McArthur, Atkinson & Ellis, 2010).

Christine Weber-Fox and associates (Weber-Fox, Leonard, Wray, Tomblin & 2010) analyzed neuronal activity in 15 persons ranging in age from 14 to 18 years with a childhood diagnosis of SLI, and found that the potentials evoked by grammatical language tasks are atypical in many adolescents with SLI, as opposed to the potentials for nonverbal tasks.

Neurofunctional studies monitoring changes in neuronal activity in the members of a family diagnosed with SLI (5 members of the same family, who were 10, 16, 38, 40, and 70 years of age when tested) presented with changed activation in the frontal and temporal lobes (Hugdahl, Gundersen, Brekke, Thomsen, Rimol et al., 2004). These activation differences proved to be the most obvious in the temporal lobes, especially the central temporal gyrus at the border with the superior temporal sulcus. In the control group, Brodmann field 44 was also active. These results, according to the authors, imply reduced activation in SLI of brain regions crucial for processing speech and phonological awareness.

Frederic Dick, Fiona Richardson, and Maria Cristina Saccuman (2008) have pointed out that in the case of neuroanatomical and neurofunctional changes implicated in disturbances in the development of language it is difficult to determine precisely what is cause and what is effect. On the one hand, it may be supposed that the changes in the brain may be the cause of the language deficits, but it cannot be ruled out that the language deficits and limited experience in the use of language could be causing structural and functional changes in the brain.

THE PATHOMECHANISMS OF SLI

Among the efforts to explain the psychological and linguistic mechanisms underlying the symptoms of SLI it is possible to distinguish three types of theories:

- linguistic theories, which focus primarily on deficits of the language system;
- psychological (cognitive) theories, which emphasize the cognitive deficits;
- mixed theories, which try to integrate both these approaches.

The linguistic theories originate from generative grammar, and reduce the nature and mechanisms of SLI to deficits in linguistic knowledge. In the linguistic

approach, SLI is understood as a deficit of linguistic competence or as a failure (partly congenital) to learn grammatical rules in the course of development (Hulme & Snowling 2009). Most of the conceptions that point to the linguistic pathomechanisms of SLI are based on research on English-speaking children. For most of them the largest problem is the application of morphemes and the arrangement of words to express the intended meaning. English-speaking children with SLI use grammatical morphemes much less often than do young, normally developing children at the same level of language development (Hulme & Snowling, 2009). This implies a certain qualitative difference in the acquisition of language. Although the linguistic theories differ among themselves in details, they have in common the tendency to link SLI with a deficit in grammar, understood as a formal set of rules underlying grammatical competence in adults using the given language. A detailed discussion of these theories would far exceed the bounds of the present study; at this point, suffice it to mention the most important such theories:

- the functional category deficit theory;
- Rice et al.'s concept of prolongation of the root infinitive stage;
- the theory of implicit rules deficit developed by Gopnik and many others (Rice & Wexler, 1996; Rice, 2000; Leonard. 2006).

From the psychological point of view, purely linguistic theories are unsatisfactory, since it seems essential to explain, not just what components are missing in the language system, but why these children encounter barriers in the acquisition of language. The authors of psychological theories appeal to nativistic theories of language acquisition.

Leonard (2006) argues that none of the linguistic theories can be confirmed empirically in any convincing way. Their main advantage is that they are very precise and provide detailed predictions regarding the symptoms of disturbances in the development of language. Their weak point, however, is that they deal with a very limited set of languages, primarily English. It should be added at this point that there is no one single universal deficit in SLI, and the symptoms may be associated to a very high degree with the nature and structure of a particular language. That in turn leads to a search for the cognitive mechanisms, above and beyond language, that could explain the mechanisms of SLI in a more universal way. Research done in different languages on the symptoms and mechanisms of SLI may suggest an "input" deficit for language information, which brings cognitive theories into play (Hulme & Snowling, 2009).

An alternative point of view, then, can be found in the cognitive approach that emerges from psychology. The linguistic problems experienced by a person with SLI are here interpreted as referring, not to knowledge of rules, but to the cognitive processes responsible for linguistic operations. In this approach, language, being a dynamic and inherently complex system, is continually interacting with other cognitive systems. Language deficits, then, should be viewed in conjunction with more basic cognitive functions, such as, for example, memory or attention. The broadest explanatory hypothesis points to deficits in processing

information. This means that the main problem lies in the type of cognitive operation, while the nature of the particular task in which they are put to use is not essential (Leonard, 2006). These theories may focus on the place (limited range of memory, e.g. working memory), energy (sufficient to complete a task that has been undertaken), and time, i.e. the pace of processing.

Detailed concepts have been formulated within this approach, pointing to the following deficits:

- the general retardation hypothesis;
- the temporal processing deficit hypothesis;
- the auditory processing deficit hypothesis;
- the word-learning deficit hypothesis;
- the hypothesis that children with SLI use the wrong mechanisms for grammatical analysis.

The mixed linguistic-cognitive theories, which combine selected elements of the linguistic and cognitive models, have been formulated thanks to research conducted to verify the hypotheses discussed above. Mixed theories focus on both the basic cognitive deficits observed in SLI and their linguistic manifestations, that is, symptoms in the form of language deficits. In this group of theories we can distinguish:

- the surface structure hypothesis;
- the procedural deficit hypothesis;
- the connectionist model (Leonard, 2006; Hulme & Snowling, 2009).

Above and beyond the division into linguistic and psychological theories is the procedural deficit theory proposed by Ullman and Pierpont (2005), who combine linguistic and cognitive deficits with the documented brain changes, demonstrating the common neuroanatomical and neurofunctional bases of both types of explanation, referring to both language and information processing, thus constituting a very important representation of the mixed theories.

THE CONCEPT OF A MEMORY DEFICIT IN SLI

The cognitive theories discussed above look for psychological mechanisms that could account for the disturbances in learning language. It should be emphasized that despite the assumption that children with SLI have a normal level of nonverbal intelligence, still, they display many non-linguistic, cognitive deficits that do not fundamentally affect the IQ (Thomas, 2005; Ullman & Pierpont, 2005; Parisse & Maillart, 2009).

Perhaps the most completely elaborated hypothesis referring to cognitive mechanisms concerns the role of working memory in SLI (Mainela-Arnold & Evans, 2005), with particular attention to auditory, or even phonological working memory. In theoretical considerations and research models we usually find references to Alan Baddeley's model of working memory, which postulates the existence of three components: a phonological buffer, a visuospatial sketch pad, and the central executive (Baddeley, 2003). There has been considerable re-

search done to confirm the connection between the occurrence of SLI and the weakening of only one of these components: the phonological buffer. Still, many doubts remain regarding the nature of this codependency. Some problems with auditory working memory seem to be congenital, while others are more conditioned by the environment (Bishop, Adams & Norbury, 2006). Moreover, there are reports of visual and executive working memory deficits in SLI children, though to a limited extent (Hoffman & Gillam, 2004). One hypothesis concerning the causes of the language problems experienced by children with SLI suggests that working memory and short-term phonological memory are compromised (Gatherole & Baddeley 1999; Ullman & Pierpont 2005; Vance, 2008). This pertains to the situation when material of a phonological nature is represented in working memory, which has a major impact on learning new words. This deficit makes itself felt most clearly in tasks requiring the repetition of non-words. There has been considerable research confirming the presence and importance of this deficit (cf. Adams & Gatherole, 2000), and difficulties in repeating non-words has been recognized as the best behavioral marker of SLI, persisting even after effective therapy (Bishop, 2006; Hulme & Snowling; 2009). This results from the fact that the attempt to repeat non-words has greater diagnostic accuracy than standardized language tests, especially when for various reasons the latter cannot be used. Research on twins has also shown that this capability is conditioned by inheritance (Hulme & Snowling, 2009). The repetition of non-words, more than the skills of auditory processing, predicts performance on tests evaluating the reception of grammatical structures, comprehension, memory for sentences, and looking for or finding words. Auditory processing, evaluated by Paula Tallal's ART method, has proven to be a good predictor only for tasks associated with the reception of grammar.

The most surprising thing in these findings is that the task of repeating non-words essentially involves only one language subsystem: namely, the phonological system, which is usually considered non-essential in differentiating SLI. The repetition of non-words is a complex task, which involves, to begin with, the phonemic segmentation of unknown forms, then the identification of the constituent phonemes, and finally the creation of the requisite executive representations and their articulation (Hulme & Snowling, 2009). Thus the process includes not only phonological components, but also auditory and kinesthetic functions. Primarily, however, the task uncovers a deficit in the storage time for phonological representations in memory, which in turn can be regarded as the cause of lexical disturbances. However – and this is worth remembering - there has also been research indicating that not all children with SLI experience difficulties in repeating non-words. Moreover, Maggie Vance (2008) has proven that memory and language deficits can have a common, cognitive cause of a more basic nature: for example, a problem with processing speech. Thus the converse interpretation is also possible: that the deficits in language processing are revealed in poorer performance of memory tasks, including precisely those involved in repeating non-words.

Short-term memory deficits, as measured by digit recall tests, have been clearly indicated in Danish research conducted by John van Daal, Ludo Verhoevena & Hans van Balkom (2009) on a large group of 4- and 5-year-olds with SLI. The repetition of digits correlates most strongly with syntactic skills, but with the remaining aspects of language functioning under consideration here - that is, phonological, lexical-semantic, and speech production - the correlation is only moderate.

In the context of the word learning deficit hypothesis, it may be well to return to an SLI-related problem mentioned earlier regarding the assimilation of the lexical and grammatical components of language (Hulme & Snowling, 2009). Research has shown that normally developing children in middle childhood learn an average of 10 words a day, while SLI children learn words at a much slower pace: they do learn new words, but the lexical representations they form in the process are not very stable. Children with SLI also have problems with working out the meaning of new words in the context of a sentence, or using them in grammatical relations. Most of the research has confirmed that, especially in English, most of the difficulties occur with verbs, which are involved with abstract grammatical relations. Semantic knowledge or lexical elements seem more transparent, which means that they do not cause these kinds of problems. Research focusing on the word learning deficit has shown that the previous hypotheses referring only to the phonological memory deficit constitute an overly narrow explanation of the mechanism underlying these problems. Word learning depends on both phonological and grammatical features, which are connected with morphological structures (Hulme & Snowling, 2009).

Longitudinal research conducted since the 1980s has confirmed that phonological short-term memory is weaker in children with SLI. These deficits limit progress in the development of key components in language development, but the cause-and-effect relationships in this case are not obvious. Short-term memory conditions the acquisition of vocabulary in the preschool period, but later on lexical knowledge can support the performance of phonological tasks. Conti-Ramsden and Durkin (2008) conducted longitudinal research on children with SLI up to the age of 14. Early measures of short-term memory explain the acquisition of vocabulary and linguistic expression, whereas early measures of reading explain later results in short-term memory. The authors came to the conclusion that the connection between the processes of language acquisition, reading and writing, and memory in SLI over the course of development manifests a bidirectional (reciprocal) interaction.

THE PROCEDURAL DEFICIT HYPOTHESIS

Ullman and Pierpont (2005) have formulated the procedural deficit hypothesis (PDH), which integrates neuronal, linguistic, and cognitive research on SLI, and refers to the heterogeneity of linguistic and non-linguistic deficits ascribed to this disorder, as well as the co-occurrence of different developmental disorders.

The most important assumption of the PDH is the premise that the configuration of linguistic and non-linguistic deficits observed in persons with SLI is the effect of the interaction of deficits occurring in two primary memory systems: procedural and declarative. The procedural system, which features implicit learning and a slow pace of acquisition, along with rapid, automatic action and sequential processing, is the foundation for the normal development of grammar. This system is also involved in working memory, complex sequential and hierarchical processing, and rapid time processing. The operation of this system in adults involves brain structures associated with the so-called "dorsal pathway," creating a connection region for the basal ganglia, and including, inter alia, the frontal cortex and Broca's area, the premotor cortex, and the caudate nucleus.

The second system, declarative memory, constitutes the foundation – from the perspective of language development – for the development of vocabulary. This system is characterized by parallel processing, slow recall, and explicit, conscious learning; it is also engaged in the acquisition of semantics, including the creation of semantic representations. The brain structures responsible for the declarative system are make up part of the so-called "ventral pathway," a network of connections that join the medial temporal regions (especially the hippocampus), the temporo-occipital regions of the neocortex, and selected parts of the frontal regions, along with a portion of the right lobe of the cerebellum (Thomas, 2005; Ullman & Pierpont 2005).

Generally speaking, the rule-bound components of the language system, such as grammar, are significantly conditioned by the functioning of procedural memory, which explains their dominant presence in SLI, whereas lexical functions are more dependent on declarative memory.

In many persons with SLI there are changes in the brain structures responsible for the procedural memory system, especially in Broca's area and the basal ganglia, especially the caudate nucleus, as confirmed by a great deal of research. These changes provide the justification, according to Ullman and Pierpont, for formulating the hypothesis that the basic deficit in SLI is not so much linguistic as procedural, which accounts for the well-known language deficits in SLI, especially grammatical, as well as the concomitant motor and cognitive problems.

According to the theoretical approach presented here, the mechanism underlying disturbances in language development is rooted not so much in language as in procedural memory, which affects selected aspects of the language system, while also causing other symptoms involving non-linguistic skills that are also dependent on procedural memory. These include those functions involved in tasks requiring working memory or temporal processing. The basis for the familiar behavioral manifestations are neurostructural and neurofunctional changes in the brain structures responsible for this system. A deficit in procedural memory disrupts the use of language rules, regardless of the specific subsystem: phonological, morphological, or syntactical. The PDH is based on the mechanisms of procedural memory, which are not a specific language mechanism, but rather affect the way information is processed. This enables us to explain the co-oc-

currence of other cognitive deficits in SLI (Hulme & Snowling, 2009), in which processes are disturbed that are not associated with language, but are also dependent on procedural memory, such as learning and performing complex sequences, or using hierarchical cognitive or motor structures. Functions based on declarative memory, however, such as the activation of semantic representations or declarative knowledge, should not show any deficit.

Another important premise of the PDH is that there is a compensatory connection between the procedural and declarative systems. In persons with SLI, the grammatical problems can be compensated by support from declarative memory.

One of the interesting aspects of the PDH model is the way it accounts for the co-occurrence of different disturbances. Ullman and Pierpont (2005) point out that structural and functional abnormalities in brain structures involved in the procedural system, including the basal ganglia, especially the caudate nucleus and the striate nucleus, are found in various different developmental disturbances: in SLI, to be sure, but also in ADHD or Tourette's Syndrome. Similarly, the manifestations of procedural deficits observed on the behavioral level are present in many developmental disturbances, but especially in ADHD and SLI (according to Ullman and Pierpont's review of the literature, the rate of co-occurrence may be as high as 45%).

This concept should be regarded as particularly promising - despite many limitations (especially those associated with the unclear assumption that a procedural memory deficit occurs in all children with SLI; cf. Hulme & Snowling, 2009) and despite the fact that not everyone agrees with the assumption that there is compensation between the procedural and declarative systems (Thomas, 2005). The PDH has important implications for differential diagnosis and therapy, including both behavioral and pharmacological methods (Ullman & Pierpont 2005).

SUMMARY

The foregoing review of the most important information concerning SLI should serve to make it clear that many questions remain unanswered. The greatest controversy surrounds the theories that explain the mechanisms and causes of this disturbance. This is why it seems urgently necessary to conduct research that would make it possible to resolve the numerous doubts and verify the proposed solutions. Bishop (2006) has pointed out the many and diverse risk factors for SLI, referring primarily to cognitive deficits, mostly associated with the functioning of memory, which, in combination with other deficits, create problems in the acquisition of language. An example of this might be the combination of weak short-term memory with difficulties in differentiating speech sounds.

At the same time, a direction of exploration has emerged that is making it possible to find the brain mechanisms responsible for the linguistic and cognitive deficits present in SLI. This direction, marked out by the PDH, seems very promising, and indicates that it may be possible after all to reconcile many hitherto

unclear and superficially irreconcilable facts concerning SLI – such as significant heterogeneity, the variability of symptoms, and co-occurrence.

The problem of memory functioning in SLI is a promising field of study, and - of particular importance - it integrates the results of research from different specializations: neurobiology, genetics, psychology, etc.

It is also interesting that research on memory deficits in SLI has cast so much light on the co-occurrence of developmental disturbances. This is the approach we find in the PDH, which is based on research on the the structural and functional abnormalities of brain structures associated with the procedural system, including the basal ganglia, especially the caudate and striate nuclei. These abnormalities are found not only in SLI, but also in ADHD, dyslexia, Tourette's Syndrome, and others. From this perspective the co-occurrence of ADHD and SLI reaches 45% (Ullman & Pierpont, 2005). The results of genetic research also seem to be tending in the direction of the co-occurrence of disorders, rather than searching for isolated units.

Looking from a wider perspective, it is precisely the reference to memory deficits that seems to unite research results from different areas and makes it possible to find the common ground for many developmental disorders.

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Address for Correspondence:
Prof. Grażyna Krasowicz-Kupis
Institute of Psychology, Maria Curie-Skłodowska University
Plac Litewski 5, 20-080 Lublin, Poland
e-mail: gkrasowiczkupis@wp.pl