Background:

Current models of spoken language production postulate that the linguistic system is composed of a network of lexical and semantic nodes that automatically spread activation to each other as part of linguistic processing. A cardinal feature of spreading activation models is that activtation spreads automatically to related nodes and, accordingly, this process is considered to be implicit, in that it occurs outside of conscious awareness or control. Intact implicit processing is thought to underlie the automatic, fluent, rapid use of language in typical adults. Implicit processing is also a component of learning and, therefore, is likely to be an important function of all rehabilitation approaches undertaken to remediate language deficits following stroke (i.e., aphasia).

Previous research has raised the question of whether automatic activation of the linguistic system is preserved or impaired in aphasia. Prather and colleagues (Prather, Zurif, Stern and Rosen, 1992; Prather, Zurif, Love and Brownell, 1997) examined implicit priming effects in individuals with aphasia and found that individuals with non-fluent aphasia were found to show priming at longer prime-target intervals than typical adults, which was interpreted to mean that these individuals had typical patterns of automatic spreading activation once it began, but that it was slow to begin. On the other hand, individuals with fluent aphasia were found to prime at both short and long intervals, which was interpreted to mean that these individuals showed typical initiation of automatic spreading activation, but that it did not damp over time as it does in the typical system. These findings are significant because, if there is a deficit in the spread of automatic activation in individuals with aphasia, this may account for some of their language deficits, and/or may influence their response to treatment.

The current investigation addressed the time-course of automatic spreading activation in aphasia using a lexical decision task with each target item preceded by a masked prime. Priming was assessed at eleven different prime-target intervals.

Participants:

Participants were 21 individuals with aphasia between the ages of 21-80 who were at least nine months post-stroke. Aphasia profiles included both fluent and non-fluent aphasias, and spanned the range of severity from mild to severe. Individuals with aphasia had no other significant neurological or psychiatric history, no history of speech and language disorders, and were native speakers of English. A control group of 31 typical adults with the same exclusionary criteria was also assessed.

Methods:

Participants completed a lexical decision task with masked primes. Prime items were all lower-case, and were either identity primes (the same word as the target) or strings of alternating x's and g'x matched in number of letters to the upcoming target. All targets were upper-case. Eleven different prime-target intervals were assessed: 30, 50, 70, 100, 150, 200, 250, 500, 750, 1000, and 1500 msec. Eleven different experimental lists were presented, each containing 80 prime-target pairs with a single prime-target interval in each list. Assignment of targets to the primed or unprimed condition was randomized for each participant, as was the sequence of target presentation

within each list. Reaction time data were collected for all responses, and analyzed for real-word targets that were correctly identified by a specified response deadline.

Findings:

Overall, the control group showed a gradual increase in priming up to the 200 msec prime-target interval, at which priming effects were statistically significant, with priming effects maintained at a somewhat lower level at longer intervals (see Figure 1). In contrast, individuals with non-fluent aphasia demonstrated weaker priming effects overall, and no clear trends. Individuals with fluent aphasia also showed weak priming effects as a group, but demonstrated a trend toward stronger priming specifically at the 250 and 750 msec prime-target intervals. An effect of age was seen in the control group, with participants over the age of 70 showing no reliable priming at any prime-target interval, whereas participants who were 70 and younger showed the pattern of priming at the 200 msec prime-target interval described above (see Figure 2). A trend toward this same pattern was evident for the individuals with non-fluent aphasia, but not for those with fluent aphasia (see Figures 3 and 4, respectively). In addition to these observations, it is notable that both groups of individuals with aphasia demonstrated evidence of inhibition, with trends toward negative priming effects, at some prime-target intervals, while the control group, overall, demonstrated only facilitation.

Conclusions:

Results from this study indicate that individuals with aphasia differ from typical adults on a subliminally primed lexical decision task in a number of ways. First, while the control group showed significant priming at one prime-target interval (200 msec.), with a logical progression of gradually increasing and decreasing priming effects in the preceding and following intervals, both groups of participants with aphasia showed less of a logical pattern. This may reflect particularly high variability in implicit processing in individuals with aphasia as compared with typical adults. Second, while typical adults, as a group, showed only facilitative priming effects, both fluent and non-fluent aphasia groups showed some inhibitory effects, which may indicate differences in the level of information activated by the sub-perceptual primes, or differences in the ability of individuals with aphasia to effectively use the information provided by the primes.

While further study is needed, these data indicate clearly that individuals with aphasia do manifest differences in automatic spreading activation when compared with typical adults. Because automatic spreading activation underlies and supports the implicit, rapid processing of language, better understanding of these differences will shed light not only on the underlying deficits of aphasia, but also on whether and/or how timing or other parameters should be considered when designing treatment tasks in order to maximize the cognitive-linguistic system's response to treatment. In addition, further exploration of these findings will be applicable to investigations of the appropriateness of using treatment techniques that may rely heavily on implicit processing mechanisms (e.g., errorless learning techniques) versus treatments that rely more on explicit skills (e.g., errorful learning).

References:

Prather, P., Zurif, E., Stern, C. and Rosen, T. J. (1992). Slowed lexical access in nonfluent aphasia: A case study. *Brain and Language*, **43**(2): 336-348.

Prather, P. A., Zurif, E., Love, T. and Brownell, H. (1997). Speed of lexical activation in nonfluent Broca's aphasia and fluent Wernicke's aphasia. *Brain and Language*, **59**(3): 391-411.

Figure 1:

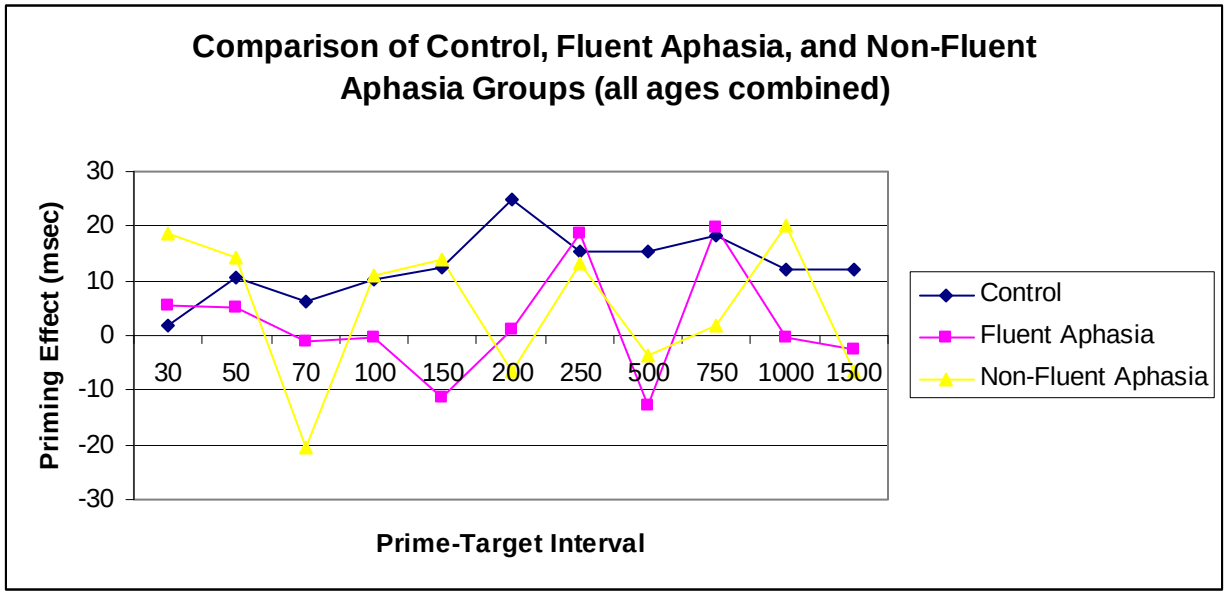

Figure 2:

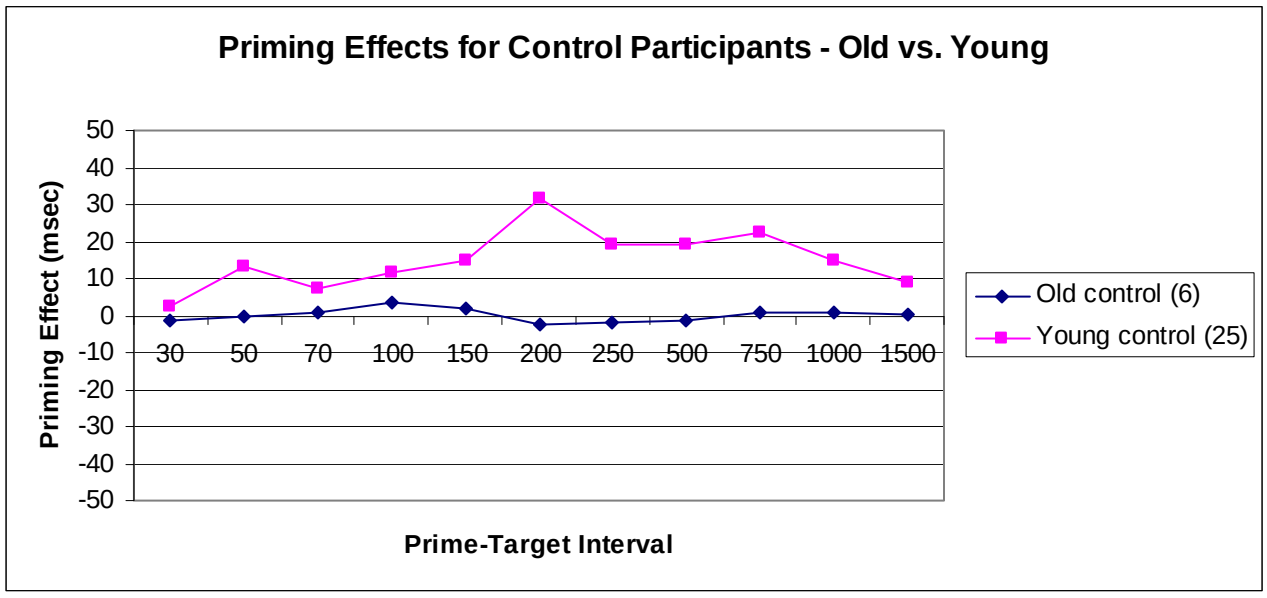

Figure 3:

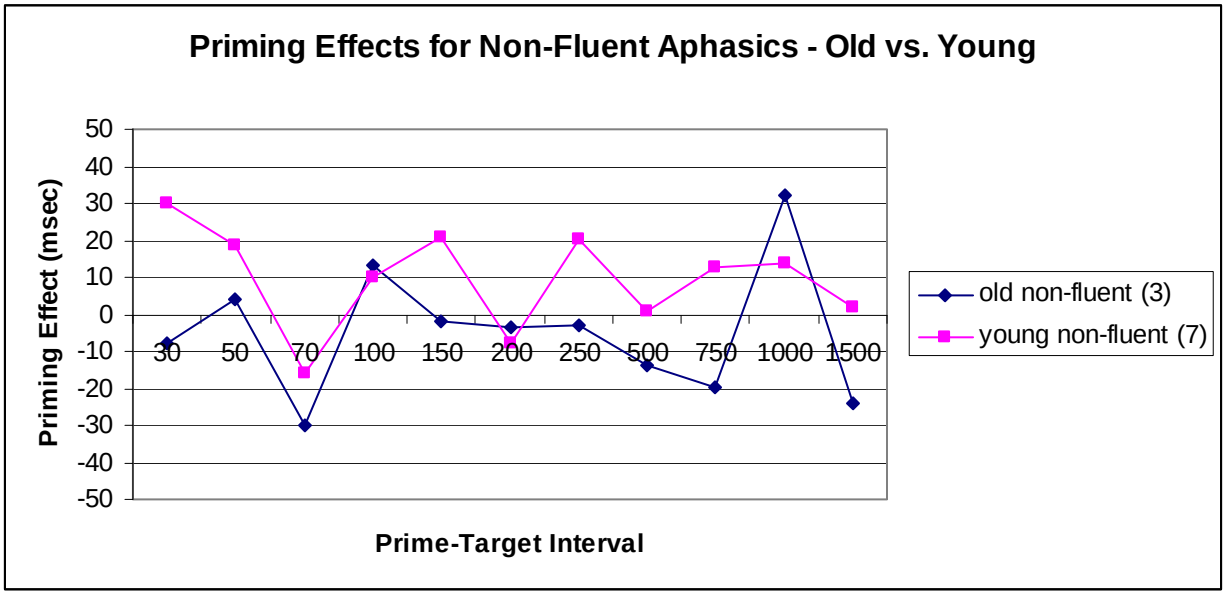

Figure 4: