Research has shown that cells in the suprachiasmatic nucleus (SCN) are heterogeneous and several cell types in the SCN are able to sustain circadian rhythms outside the SCN. In the LeSauter and Silver (1999) paper, they try to determine what cells in the SCN control locomotor rhythms in mammals. LeSauter and Silver hypothesize that the cells responsible for the locomotor rhythms are the calcium-binding protein calbindin-D28K (CaBP) cells that make up a compact subnucleus in the SCN.

In order to test whether the CaBP cells are responsible for locomotor rhythms, a lesion and transplant experiment was conducted. The study used wild-type hamsters with a free-running period of ~24 hours and mutant hamsters with a free-running period of ~22 and 20 hours. All animals were housed individually and each cage had a running wheel. At ~7 weeks of life, all animals were transferred from a LD to a DD cycle. In the lesion experiment, hamsters were given partial electrolytic lesions (lesions made by electrical current) in the SCN and locomotor activity (wheel running) was monitored for 4 weeks in animals that sustained rhythms and 12 weeks in animals that became arrhythmic after surgery. In the transplantation experiment, tau mutant hamsters were monitored for 1-3 weeks prior to partial electrolytic lesions and those that were arrhythmic after the SCN lesion received transplants. After transplantations, locomotor activity was monitored for 16-20 weeks. In addition to monitoring locomotor activity, histology (looking at SCN tissue under a microscope) tests, were done in order to determine the number and presence/absence of CaBP cells in SCN after the lesion and transplantation experiment.

In the partial SCN lesion experiment, the hamsters that had one or both of the CaBP subnucleus in the SCN continued to have rhythms. However, animals with lesions that bilaterally destroyed the CaBP subnucleus were arrhythmic. In the transplantation experiment, hamsters

that received grafts (donor tissue) with SCN CaBP cells present had the rhythms of the donor restored. Six hamsters lacking SCN CaBP cells in the graft were arrhythmic after transplantation. In general, the restoration and persisting locomotor rhythms were significantly correlated with the number of SCN CaBP cells remaining in SCN of hamsters after the partial lesion experiment and the number of SCN CaBP cells in the graft in the transplant experiment.

The results indicate that the CaBP subnucleus located in the SCN of the hamster are controlling and maintaining the locomotor rhythms. Ablation of CaBP cells resulted in loss of locomotor rhythmicity and replacement of CaBP cells can reinstate locomotor rhythmicity. Additionally, even when 67% of the SCN tissue remained, without the presence of CaBP cells, these remaining regions were not sufficient in maintaining locomotor rhythms. Therefore, regional specialization of function are present in the SCN and this negates the former belief that as long as ~25% of the SCN survive ablation, circadian rhythms persist.