1. A. The regions of the tracks that are dense with silver grains correspond to those segments of DNA that were replicated when the concentration of the label was high. The less dense regions mark segments of DNA that were replicated when the concentration of label was low.

B. The difference in the arrangements of the dark and light sections of the tracks derives from the difference in the labeling schemes in the two experiments. In the first experiment $^3$H-thymidine was added immediately after release of the synchronizing block. Thus, replication initiated at origins in the presence of label, giving a continuous dark section on both sides of the origin. When the concentration of label was lowered, replication proceeded in both directions away from the origin, leaving light sections at both ends of the dark sections. In the second experiment replication began at origins in the absence of $^3$Hthymidine so that the origin was unlabeled. Addition of a high concentration of label followed by a low concentration gave rise to a dark section with a light section at one end. Adjacent dark sections are part of the same replicating DNA molecule; they are linked by the unlabeled (therefore invisible) segment that contains the replication origin.

C. The approximate rate of fork movement can be estimated from the labeling times and the lengths of the labeled sections. In the first experiment, segments roughly 100 um in length were labeled during the 45-minute labeling period. Because two replication forks were involved in synthesizing each labeled segment, each replication fork synthesized about 50 um of DNA in 45 minutes. Therefore, the rate of fork movement is about 1.1 um/min (50 um/45 min = 1.1 um/min). In the second experiment segments roughly 50 um in length were labeled; however, each was synthesized by only one replication fork. Thus, the rate of fork movement was also about 1.1 um/min. This information is not sufficient to estimate the time required to replicate the entire genome. The missing information is the number of active origins of replication and their distribution.

2. A. The three density peaks represent, from light to heavy, unreplicated DNA, once replicated DNA, and twice replicated DNA (Figure below). The injected DNA is labeled with $^3$H but is otherwise normal DNA, which is “light.” Each newly synthesized strand incorporates $^{32}$P label and BrdU, which increases its density. Thus, after one round of replication, the DNA will be intermediate in density, containing one “light” $^3$H-labeled strand and one “heavy” $^{32}$P-labeled strand. After the second round of replication, the hybrid DNA will give rise to one hybrid density duplex and to one duplex that contains two $^{32}$P-labeled “heavy” strands. The fully “heavy” duplex will appear at the densest position in the gradient. Since the fully “heavy” DNA contains two new strands, it will contain no $^3$H label. The formation of discrete peaks in this experiment makes an important point: most of the observed labeling is due to replication and
not to repair synthesis. If the incorporation of label were due to repair synthesis, which is patchy, the label would be smeared through the gradient rather than concentrated in discrete peaks.

B. The injected DNA mimics the expected behavior of chromosomal DNA in one very important way: it undergoes only a single round of replication in one cell cycle. This behavior is apparent from the lack of fully “heavy” DNA after one cell cycle. In another way, however, the injected DNA behaves very differently from chromosomal DNA: a large fraction of the injected DNA does not replicate even after two cell cycles. The lack of replication is apparent from the persistence of a fully “light” peak of DNA. It is not clear why some of the injected DNA does not replicate. Perhaps some of the eggs have been rendered incompetent for replication by the experimental protocol.

C. Since cycloheximide is an inhibitor of protein synthesis, it should have no direct effect on the synthesis of DNA. And, indeed, in the presence of cycloheximide one round of replication is completed normally, though no more occur thereafter. Cycloheximide apparently blocks further progress through the cell cycle because a key cell-cycle event depends on protein synthesis. The important point is that the DNA will not replicate again unless the cells progress through the cell cycle.