Evidence for symmetric chromosomal inversions around the replication origin in bacteria

ABSTRACT
X-alignments are thought to arise from large reversals of the genomic sequence symmetrically around the origin of replication, which is most likely caused by these inversions. The discovery of these chromosomal invertions also suggests that bacterial genome evolution involves many such changes.

INTRODUCTION
Most vertebrate organs and tissues experience development during embryogenesis, while postnatal changes are primarily concerned with growth. The CNS is unique in that postpostnatal development involves significant morphological development, cell differentiation, and acquisition of function. The molecular mechanisms behind these complex developmental processes remain unclear. We have recently conducted a DDRT-PCR analysis of genes expressed in the murine postnatal developing brain. A study that would continue to follow the same baseline was able to produce sequences of "rnf" (RNA fingerprints) of genes transcribed at various stages of brain development, which we then compare at four different timepoints: newborn (day 1), daily 10, day 20 and adult (Day 42). Our experimental aim was matched by the discovery of numerous highly developmentally regulated genes specifically found in the wild-type mouse brain. We use this data to help others identify specific transcripts with significantly accelerated expression in their postnatal brains that require minimal screening efforts. About 1% of the 200-300 transcripts displayed, out of a total of around 25,000, had expression profiles that were developmentally regulated. In this paper, we describe RNA fingerprints that display a subset of developmentally regulated genes. These groups can be used as cloning replicates to select specific cDNAs and were selected for use in selecting at least two different batches of radiated ARN (DDRT-PCR) profiles; therefore each fingerprint contains at most three developmentically significant rRNAs. At least three bands of DNA have been identified on each fingerprint group, representing the highly regulated genes during development. Our classification of marked transcripts into three broad groups is based on their frequency of expression: genes that increase in mRNA levels during brain development, those with lower rRNA rates during development and those that peak during this stage of development. In order to verify that alterations in DDRT-PCR profiles are authentic, two cDNA fragments were recovered and used in downstream expression analysis. The complete process as illustrated in Figure 1 (and see) is described below. The original DDRT-PCR expression profiles were replicated precisely by the northern blot expression profiling of both transcripts, reinforcing our approach.

CONCLUSION
According to the new findings, clonal expansion is a prevalent feature in the B-cell repertoire of patients with rheumatoid arthritis. This expansion involves resting memory B cells and activated B cell repertoires, some of which are derived from the memory Brain tumor's internal compartment. As the range of clonal expansions increases, from the bloodstream to the synovial compartment, the narrowing of diversity indicates that these antigens located in the brain are responsible for these "antigen-receptor biases," and evidence suggests that some of these expansion patterns

may be joint-specific. Due to the rarity of identical clones in two distinct joints, immune reactions are likely unique to each joint. Additionally, B cells from this joint are unlikely to contain a different foreign antigen, so they are reacting with autoantigens produced locally, potentially by local tissue breakdown. Lymphoid aggregates that contain the cellular components of an ectopic germinal center can be formed in synovial tissue of rheumatoidic arthritis patients, as new research has shown, and can maintain B-cell clonal expansion and diversification. It is likely that the B cells that mature in these 'pseudogerminal centers' and those that we have identified in the current studies are responding to specific (auto)antigens. Hence, the identification of antigenic reactivations of these B cells, and specifically of those within the memory compartment that have probably "passed over (auto)antigen and T cell selection and rescue"; in this case, they may provide important clues about the role of B lymphocytes and their immunoglobulin molecules in the immunopathogenesis of rheumatoid arthritis.