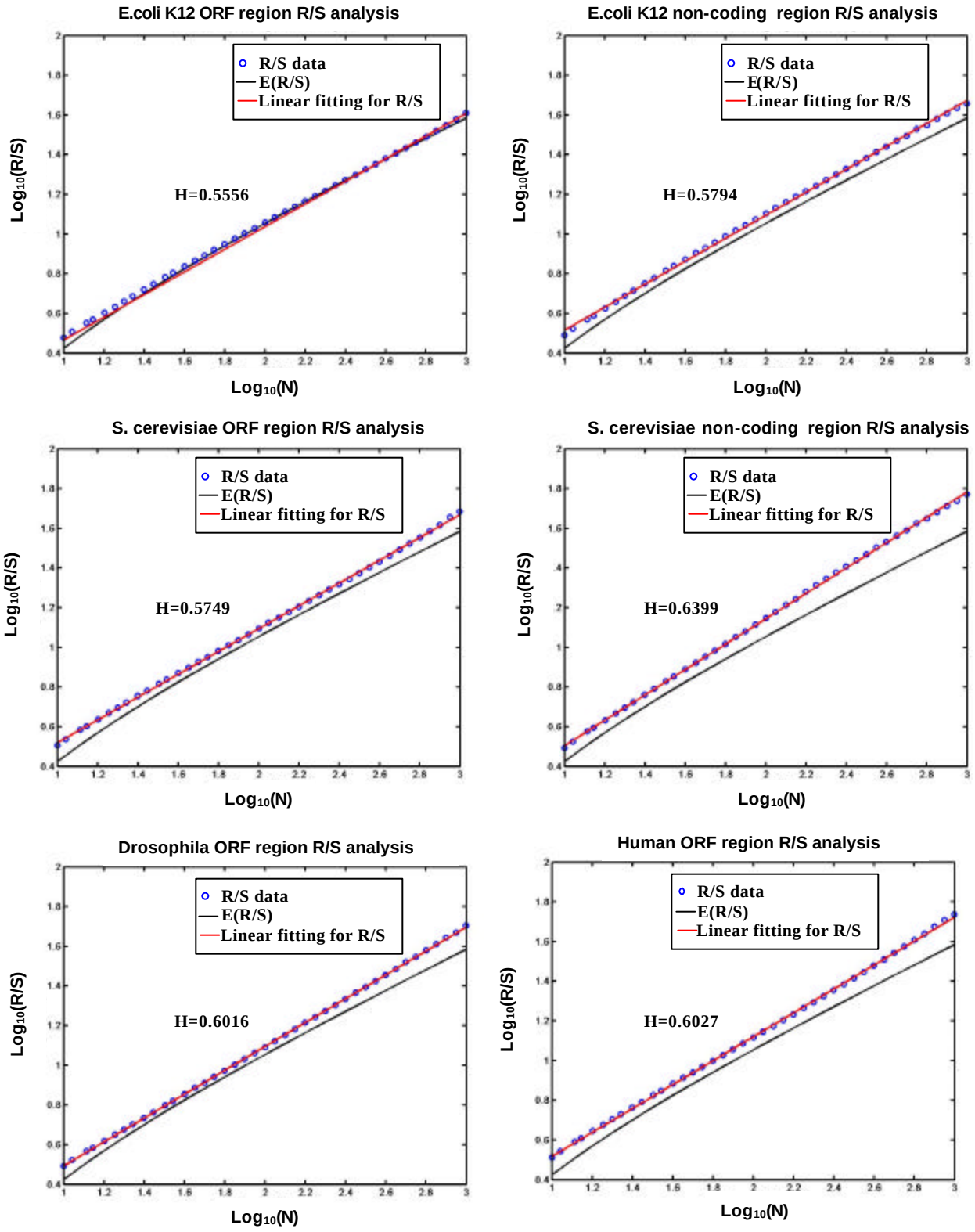

Figure1. R/S analysis to estimate Hurst exponents (H) for various organisms and in various regions of their genomic sequences. R/S analysis algorithm is run on the sequence data from various organisms, distinguishing coding regions (open reading frames, ORF) and non-coding regions. Results are shown for both the coding and non-coding regions from E.coli k12, S.cerevisiae (budding yeast), and the coding regions from Drosophila (fruit fly) and human. Expected value of R/S in a Brownian motion is estimated by an empirical equation corrected for finite sampling (see Apendix I). The Hurst exponents (H) estimated by linear fitting of the data points are shown in each plot (see Table1 for statistical test).

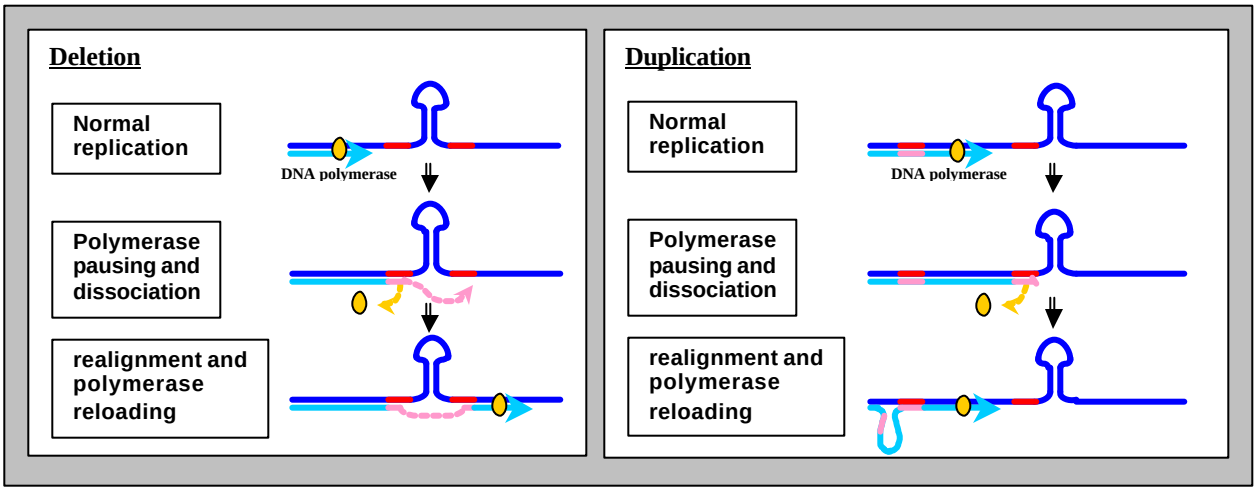

Figure 2. Replication slippage causes deletions and duplications. During DNA replication, duplex DNA is dissociated into single stands. Daughter strand is synthesized by DNA polymerase complementarily to the template strand. The single stranded DNA are prone to form secondary structures (for example, hairpins) when there are complementary sequences or repeats in the strand. Those secondary structures hinder the synthesis process of DNA ploymerase, and can cause it to pause or even dissociate from the DNA. The absence of DNA polymerase can free the end of the daughter strand from its template. When there are short repeats in the sequence, the freed end may mistakenly realign to a "wrong" repeat in the template strand. Thus, when DNA polymerase is reloaded onto such misalignment, it will continue the previously aborted synthesis, and cause deletion or duplication in the daughter strand. If the changes in daughter DNA strand escape DNA repair mechanism, they can be inherited by progenies.

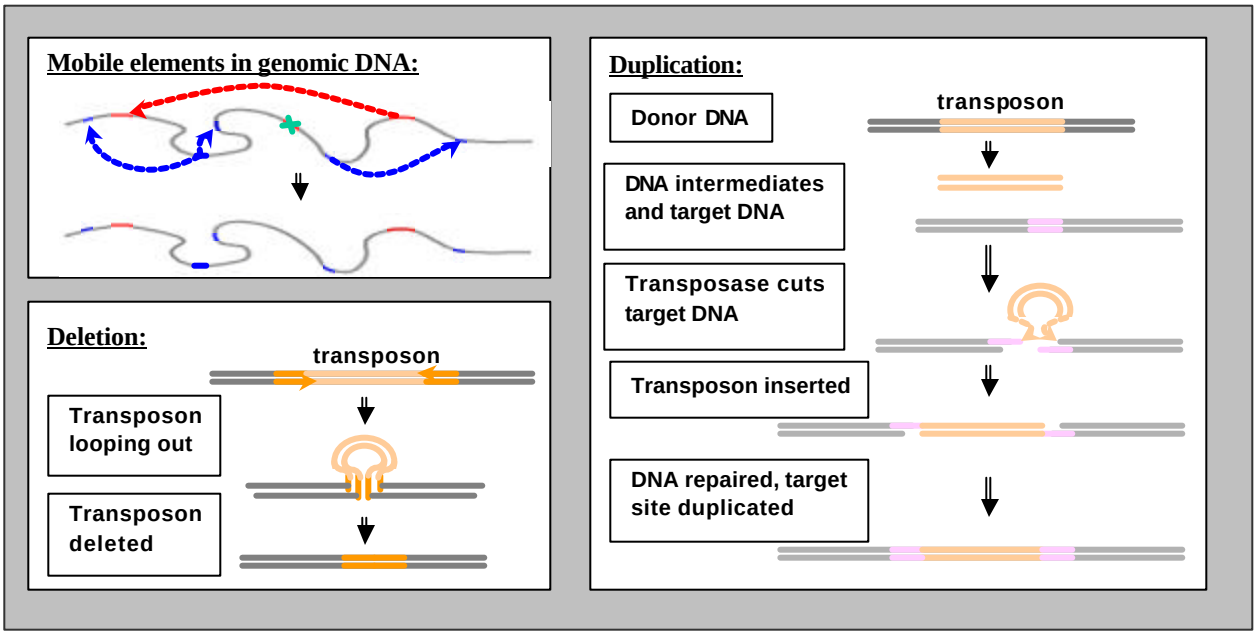

Figure 3. Mobile elements cause deletions and insertions in genomic DNA. In the figure we show for an example how transposons can cause deletions and duplications. Transposons (as most of the mobile elements) contain sequence that encodes for an enzyme, transposase, and two flanking inverted repeats. Transposase contributes to the deletions and insertions of the transposons either by cleaving out the loop structures formed by transposons in DNA or by cleaving the target sites for transposon insertions. The inverted repeats flanking the transposons are formed by duplication of the target sites during transposon insertion. Those repeats also make the transposons prone to form loop structures, thus susceptible for deletion. Some transposons can replicated themselves, through either DNA intermediates (replication of the deleted fragments) or RNA intermediates (reverse transcriptase) , they can propagate themselves in the genomic sequences.

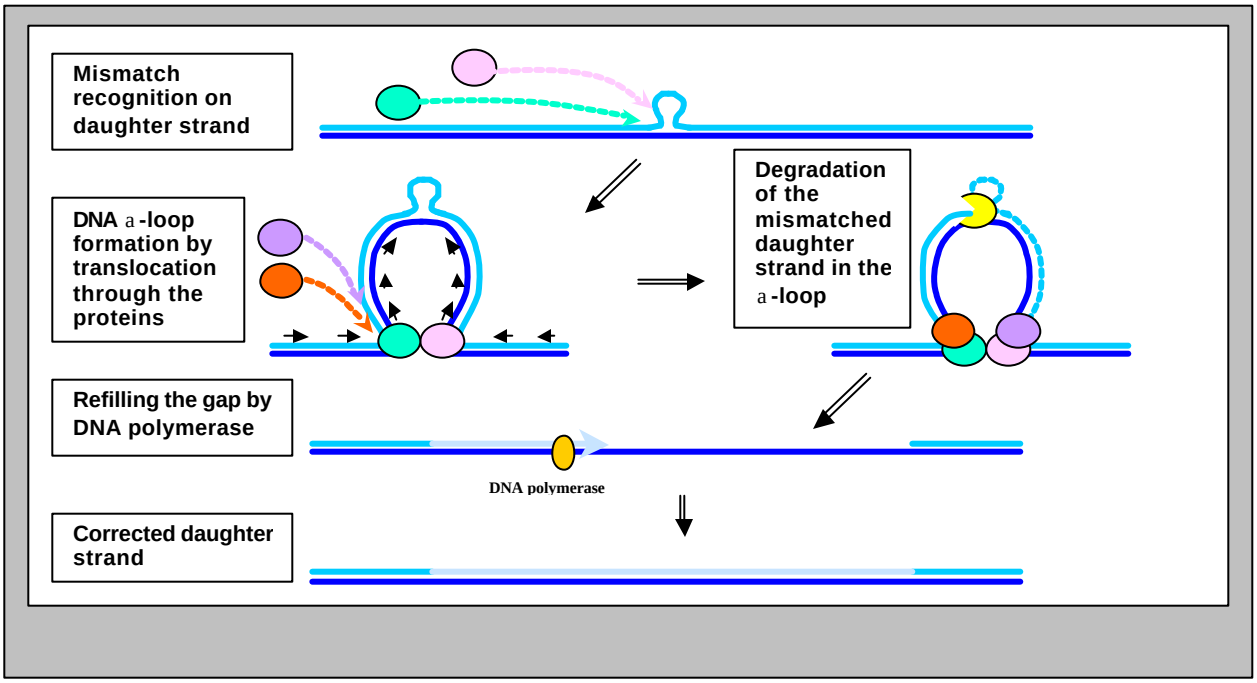

Figure 4. DNA mismatch mechanism prevents deletions and insertions. DNA mismatch machinery is composed of multiple proteins with different functions. A dimeric protein complex can specifically recognize and bind to the positions on DNA that contain mismatches. They further recruit other members of the machinery. Together, they thread the region of the DNA containing the mismatch through them and form a α-loop structure. Exonuclease, later recruited, digests specifically the daughter strand in the α-loop. Finally, when the DNA is let loose from the α-loop, DNA polymerase refills the gap on the daughter strand caused by the digestion of exonuclease and finish the correction work.

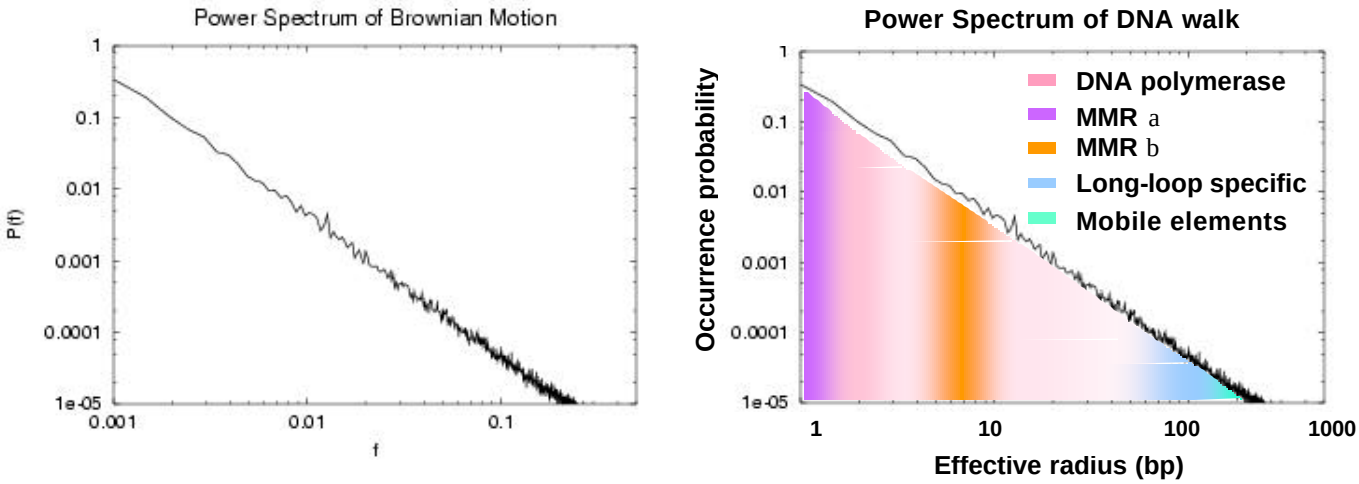

Figure 5. Power spectrum explanation of the Brownian or Fractal Brownian motion in DNA walk. The right plot shows the power spectrum of a Brownian motion (see text for more detail), and the left plot shows the suggested power spectrum of DNA walk using biological events as building blocks. Brownian and Fractional Brownian motion can be generated by a series of events with different frequencies and with their amplitudes following the inverse power law. During DNA evolution, various cellular events (listed in the text and some of them on the graph) are the forces that cause DNA sequence to become a realization of Brownian or Fractional Brownian motion. Therefore, we suggest that the combination those cellular events also follow the inverse power law and form a power spectrum that look like the one in the figure.

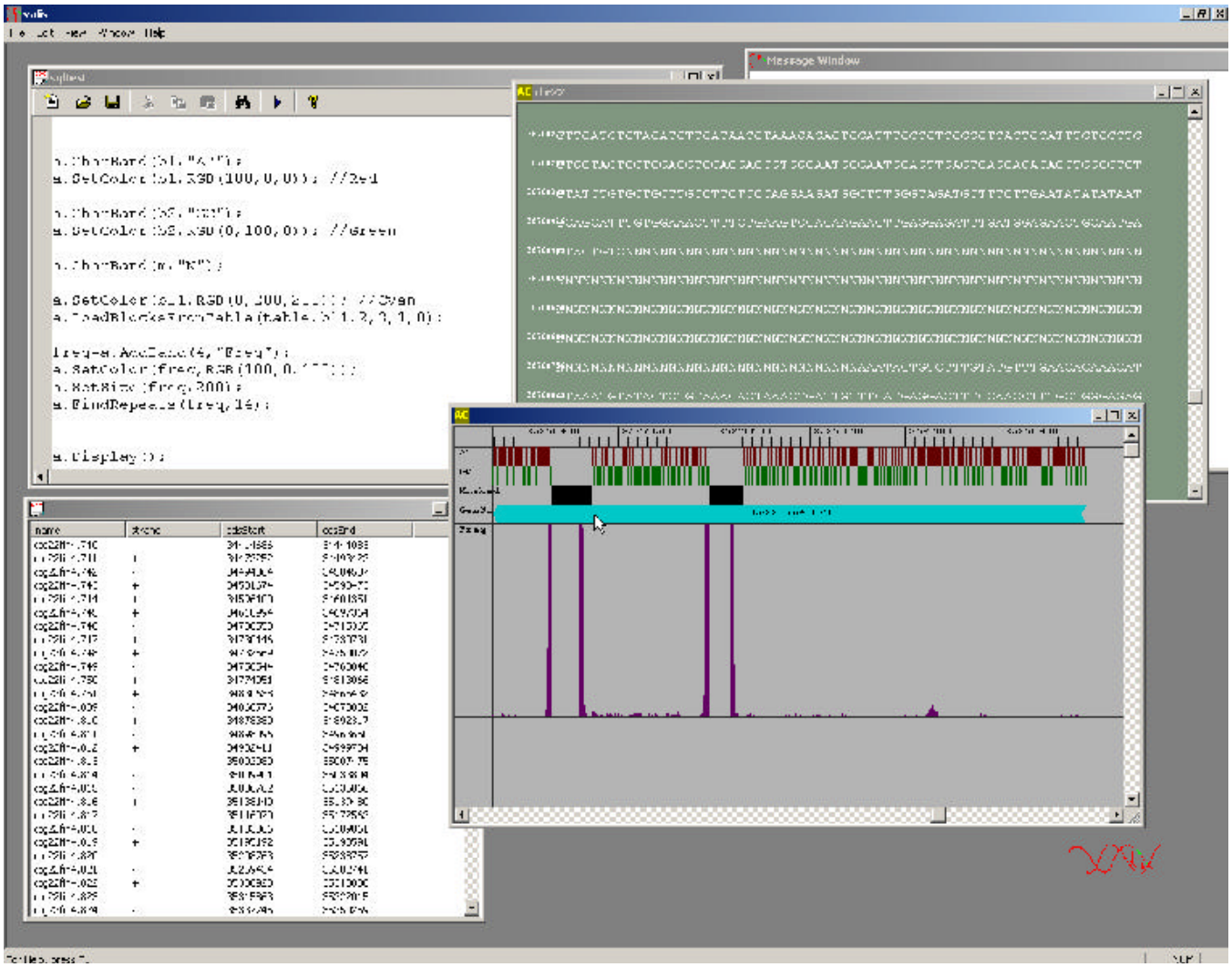

Figure 6: Valis Screenshot showing the analysis of human chromosome 22.