The manuscript looks better after the revision. However, current version is still full of mysteries. Obviously, the authors have excellent background of genetic association study in psoriatic arthritis (PsA) and cutaneous-only psoriasis and have very strong publication history in this field. Although it is an extend research to the author’s previous study, the manuscript should be taken as independent study as much as possible. The comments from mine is also based on this idea that I hope the readers can understand the manuscript easily without frequently checking tens of previous research. One of the advantage of the current study is that the authors made full use of different dataset and information, any unclear explanation will make the reader have lots of confusion. I expect the author could provide an easy-understand and easy-reproducible manuscript in next version.

1. In the introduction section, 64 distinct genome-wide significant loci was mentioned with two author’s previous studies. I suggest the authors to remove the exact number or provide the SNPs list as the supplementary tables. Too many such situations were occurred in the manuscript, be clear explained or not to mention will be recommended. For the supplementary Table 1, the genomic assembly of hg19/GRCH37 should be mentioned. Meanwhile, rs-id and allele frequency in case and control are highly recommended for the independence of the current study.
2. I checked the study: Tsoi et al. 2017, PMID: 28537254 mentioned by author and didn’t find any comparisons between H3k27ac and other chromatin marks. I am not sure whether I didn’t find it or else by other reasons. The study (PMID: 28537254) do conducted a regulatory elements enrichment analysis in which H3K27ac was taken as predictor for regulatory elements. I didn’t find any other information about comparisons between H3k27ac and other chromatin marks at least in the method section of PMID: 28537254.
3. In Table 1, how many loci or markers were enrolled for the analysis? Why use cytoband to index the table not the specific regions since cytoband is obviously large then 500K which was applied for the analysis in the manuscript. I guess ‘min p-value’ indicates multiple SNPs located in the corresponding loci, right? What’s the linkage disequilibrium between these SNPs with eQTL? I suggest the authors to note the release version or date when collecting pharmgkb and drugbank so that the reader can repeat the analysis. Finally, it is very important to show the exact drug-gene or drug-SNPs in Table 1 or to show the LD between GWAS-SNPs with these drug-gene or drug-SNPs.
4. Figure 1a, what’s the role of eQTL data was not explicit. It looks they are used in the supplementary section and Table 1. However, I am not sure the function of the eQTL in the analysis. Figure 1d: GEO2R is tools not statistic method. the exact statistical method should be mentioned, paired t-test or other method? I can still very confuse to Figure 1c. How these drugs and genes are connected? Is there any SNPs or genes or drugs were ignored since they are not connected.