Some other similarity measures such correlation coefficient is also used to measure the target drug sensitivity results based on $evalU_{\lambda}$ At final stage of the work selected genomic characterization matrix G is analyzed to measure the drug and anti drug sensitivity results.

4. Results and Discussion

In order to measure the performance accuracy of the proposed and existing schema in this work the following publicly available datasets will be used such as CCLE, COSMIC, Cell Miner it consists of large number of dataset samples with genomic characterizations, which is implemented in Matlab environment. The experimentation results of the proposed and existing schema is measured based on the 10-fold cross validation with under different values λ_1 and λ_2 . MSE is also measured between the values for training and testing samples. The results in Table 2 illustrates that ISP, IRM and IRM-Elastic Net, PSO-HMM performs significantly than earlier methods , since the proposed PSO-HMM methods genomic characterizations based drug sensitivity prediction is done.

Table 2: Pearson's Correlation Coefficients between Observed and Predicted Sensitivities Values Using 10 Fold Cross-Validation of CCLE Data Set

Erotinib sensitivity correlation coefficients						
	CCLE	RF	ISP	IRM	IRM-EN	PSO-HMM
V=0.5	0.35	0.43	0.700	0.728	0.786	0.856
V=0.75	0.35	0.43	0.714	0.731	0.748	0.856
V=1	0.35	0.45	0.713	0.734	0.754	0.864
V=1.5	0.34	0.44	0.712	0.746	0.798	0.847
AZD0530 sensitivity correlation coefficients						
	CCLE	RF	ISP	IRM	IRM-EN	PSO-HMM
V=0.5	0.21	0.29	0.5197	0.587	0.621	0.789
V=0.75	0.21	0.29	0.5217	0.5898	0.6134	0.794
V=1	0.21	0.29	0.5234	0.5964	0.6147	0.799
V=1.5	0.21	0.29	0.4978	0.5941	0.6241	0.783

Sequential floating forward search (SFFS) is proposed in this work to perform the feature selection and predicative is done based on the selected features. The cost function is generated using PSO-HMM with number of iterations is specified to reach the target anti drug sensitivity results with 15 targets of Leave one out error analysis shown in Table 3.

Table 3: Leave one out error analysis for Mouse Embryonal Rhabdomyosarcoma

Drug name	*Error*	*Predicated value*	*Actual value*
Alisertib	0.5124	0.1356	1
Crizotinib	0.0123	0.8971	0.8479
GANT61	0.1568	0.1548	0.6589
BMS-754807	0.0045	0.5471	0.8471
BIX 01294	0.0056	0.4213	0.9874
Pelitinib	0	0	0
Obatoclax	0.05468	0.6154	0.894
TrichostatinA	0.0546	0.4568	0.7148
Cediranib	0.1546	0.2146	0.6589
Dasatinib	0.02356	0.7785	0.7894
Panobinostat	0.01457	0.6489	0.54689
Vorinostat	0.0145	0.5487	0.6987
SNS-032	0.0004	0.4568	0.8974
Carfilzomib	0.4568	0.2546	0.8906
AZD-8931	0	0	0
BEZ235	0.0045	0.6546	0.8478
cilengitide	0.01548	0.7154	0.8416

5. Conclusion and future work

The main aim and objective of this paper is to propose novel PSO-HMM methods for prediction of drug sensitivity especially for anti cancer prediction. The Particle Swarm Optimization (PSO) seems helpful and appropriate technique intended for Hidden Markov Models (HMM) training. The proposed PSO-HMM is applied for Anti-Cancer Drug Sensitivity based on the calculation of likelihood. The proposed PSO-HMM prediction makes use of well-designed and genomic information of training drugs. The proposed PSO-HMM is experimented to 60 drugs from embryonal rhabdomyosarcoma tumor cell and achieves higher prediction accuracy when compare to existing methods. The prediction results of the proposed PSO-HMM are evaluated using $evalU_{\lambda}$ among forecast and real investigational compassion. The present work will be extended to apply other dataset Cancer Genome Atlas (TCGA) which is collected from http://cancergenome.nih.gov/ and experimented using matlab environment. In the future; the other methods such as Linear PSO or pareto-ranking methods will be also used for anticancer drug sensitivity. Prospect investigate determination necessitate make use of the structure designed for creation of grouping treatment through diverse optimization goals such as circumvent struggle through targeting many self-governing pathways.

References

[1] L. Chin, J.M Andersen, P.A Futreal, "Cancer genomics: from discovery science to personalized medicine," Nature Medicine (17), pp. 297–303, 2011.
[2] S. Gua, G. Yinb, J.J Lee, "Bayesian two-step Lasso strategy for biomarker selection in personalized medicine development for time-to-event endpoints," Contemp Clin Trials (36),pp.642–650,2013.
[3] R .Tibshirani , "Regression shrinkage and selection via the lasso," J R Stat Soc Series B (28), pp.267–288,1996.
[4] H. Zou, T. Hastie , "Regularization and variable selection via the elastic net," J R Stat Soc Series B (67),pp.301–320,2005.
[5] L. Wang, X. Chen, B. Zhang, "Statistical analysis of patient-specific pathway activities via mixed models, " J Biom Biostat (8), pp. 7313, 2013.
[6] T. Shimamura, S. Imoto, Y. Shimada, Y. Hosono, A. Niida, "A novel network profiling analysis reveals system changes in epithelial-mesenchymal transition," PLoS ONE 6:e20804 , 2011.
[7] Richmond , Y. Su, "Mouse xenograft models vs gem models for human cancer therapeutics," Disease Models Mech (1), pp. 78–82, 2008.

[8] A. Hopkins, J. Mason, J. Overington, "Can we rationally design promiscuous drugs?," Current Opinion Struct. Biol (16), pp. 127–136, 2006.
[9] Z. A. Knight , K. M. Shokat, "Features of selective kinase inhibitors," Chem. Biol (12), pp. 621–637, 2005.
[10] B. J. Druker, "Translation of the Philadelphia chromosome into therapy for CML," Blood, 112, pp. 4808–4817, 2008.
[11] D. Marbach et al, "Wisdom of crowds for robust gene network inference," Nat. Methods 9, pp.796–804 , 2012.
[12] G. Damia, M. D'Incalci, "Contemporary pre-clinical development of anticancer agents--what are the optimal preclinical models?", Eur J Cancer 45, pp.2768-81,2009.
[13] R. H. Shoemaker , "The NCI60 human tumour cell line anticancer drug screen," Nature reviews (6), pp.813-23,2006.
[14] S. V. Sharma, D. A. Haber, J. Settleman, "Cell line-based platforms to evaluate the therapeutic efficacy of candidate anticancer agents," Nature reviews (10), pp.241-53, 2010.
[15] J. Barretina, G. Caponigro, et.al , "The Cancer Cell Line Encyclopedia enables predictive modelling of anticancer drug sensitivity," Nature, (483), pp.603–607,2010.
[16] M. L. Sos, K. Michel et.al , "Predicting drug susceptibility of non-small cell lung cancers based on genetic lesions," J. Clin. Investigation (119), pp. 1727–1740, 2009.
[17] J. E. Staunton, D. K. Slonim, H. A. Coller, P. Tamayo, M. J. Angelo, J. Park, U. Scherf, J. K. Lee, W. O. Reinhold, J. N. Weinstein, J. P. Mesirov, E. S. Lander, and T. R. Golub, "Chemosensitivity prediction by transcriptional profiling," in Proc. Nat. Acad. Sci. USA, pp. 10787–10792, 2001.
[18] J. K. Lee , D. M. Havaleshko, H. Cho, John N. Weinstein , E. P. Kaldjian, J. Karpovich, A. Grimshaw, and D. Theodorescu, "A strategy for predicting the chemosensitivity of human cancers and its application to drug discovery," Proc. Nat. Acad. Sci. USA, pp. 13086–13091, 2007.
[19] M. Garnett, M. J. Garnett, E. J. Edelman et. Al, "Systematic identification of genomic markers of drug sensitivity in cancer cells," Nature (483), pp. 570–575, 2012.
[20] Park H, Konishi S, Robust solution path for high dimensional sparse regression modeling. Commun Stat Simul Comput, In press, 2014.
[21] X.Hu, R.C.Eberhart , " Solving constrained nonlinear optimization problems with particle swarm optimization," In: Proceedings of the Sixth World Multi conference on Systems, Cybernetics and Informatics, 2002.
[22] L. Tothfalusi ,L. Endrenyi, "Estimation of CMAX and TMAX in populations after single and multiple drug administrations," J Pharmacokinet Pharmacodyn (30), pp. 363–385, 2003.