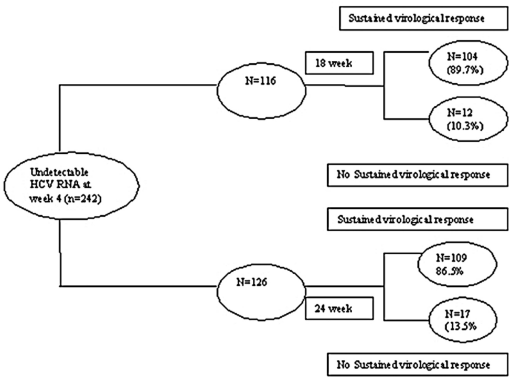
treatment response. In current study main objective is to assess Sustained virological response (SVR) rate with respect to 18 vs. 24 week treatment plan in RVR cases. In this retrospective study 316 seropositive genotype 3 HCV patients were selected who were receiving pegintron alfa 2a (180 μg/week) plus ribavirin (weight based) therapy. Treatment duration of these patients was individualized on the basis of week 4 virological response, defined as rapid virological response (RVR). Patients with RVR (Fig. 1) were randomized to 18 week (group A, $n = 116$) and 24 week (group B, $n = 126$). Patients with no RVR ($n = 74$) were allocated as group C and treated for 24 weeks. High SVR was observed in group A (89.7%) and group B (86.5%) as compared to group C (59.5%). High SVR was observed in both 18 vs. 24 weeks treatment (group A vs. group B) but the difference is non significant ($p = 0.31$). The difference in SVR rate in group B vs. C with 24 weeks treatment was found highly significant ($p < 0.001$). Data suggests that 18 weeks treatment of pegintron with weight based ribavirin is equally good in RVR achieving patients.

Figure 1: Predictability of sustained virologic response in 18 and 24 week treatment groups with rapid virological response after peginiron combination therapy



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Antiviral Activity of *Cymbopogon nardus* (L.) Rendle Fractions Against HSV-1

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An *in vitro* study was carried out to investigate the antiviral activity in four hexane fractions of *Cymbopogon nardus* (L.) Rendle towards HSV-1. Cytotoxicity assay was conducted towards Vero cell line using MTT assay to determine the fraction concentration that cause 50% cell death (CC_{50}). Plaque reduction and virus reduction assays were used to evaluate antiviral properties of test fractions. Two treatments were used to determine the fraction mode of action. Pre-treatment [(C + V) + A] was done by inoculating the virus (V) to the cells (C) before being treated with fraction (A). Post-treatment [(C + A) + V], involved inoculation of the cells with virus after treatment with fraction. Five different concentrations of fractions were used. HSV-1 dose was fixed at ~50 pfu/well. Cytotoxicity test showed 1.0 CC_{50} values for the four fractions ranged between 0.078 mg/ml and 0.240 mg/ml. Moderate antiviral activity was observed in both treatments, with 32.44% reduction showed by F117 in post-treatment while in pre-treatment assay, F147 showed a reduction of 42.93% (Fig. 1). Similar reduction was also observed in virus yield assay where F117 reduced virus yield by 39.43% and F147 reduced virus yield by 39.91% (Fig. 2). In conclusion, test fractions F117, F123, F147 and F166 of *C. nardus* were not cytotoxic and have moderate antiviral activity against HSV-1 as shown by plaque reduction assay. These fractions will be combined to test whether there will be an increase in antiviral activity.

Keywords: *Cymbopogon nardus*; HSV-1; Plaque reduction assay; Virus yield reduction assay

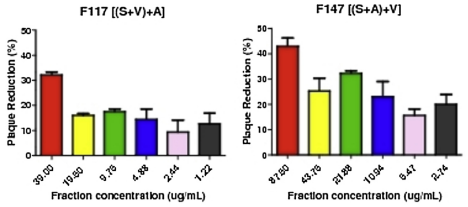

Figure 1: Percentage of plaque reduction in post-treatment [(S+V)+A] and pre-treatment assay [(S+A)+V].

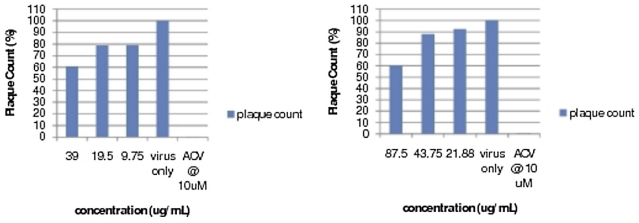

Figure 2: Effect of test fraction on virion production in virus yield reduction assay. Total virion was harvested after 48 hours incubation with test fraction and titrated to quantify total virion produced compared to control (total virion from infected cells without treatment). ACV plaque count was 0.25%.

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Amino Acid Substitutions At Residue 207 of Viral Capsid Protein 1 (VP1) Confer Pleconaril Resistance in Coxsackievirus B3 (CVB3)

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Pleconaril binds into a hydrophobic pocket within VP1 of entero- and rhinoviruses, stabilizes the viral capsid and prevents viral adsorption and/or uncoating. Previous studies showed that pleconaril susceptibility of CVB3 correlates with the amino acid in position 1092 of the hydrophobic pocket. In the present study, nine independently derived resistant variants of the clinical isolate CVB3 97927 were isolated under pleconaril treatment in HeLa cells. Pleconaril did not inhibit the plaque production of these plaque-picked and three times plaque-purified resistant isolates at the maximum noncytotoxic drug concentration. Sequence analysis of the genome region coding for the capsid proteins VP1, VP2, VP3 und VP4 revealed substitution of isoleucine by methionine at residue 92 of VP1 in three of these resistant isolates. Six others possess a single amino acid substitution at residue 207 of VP1 ($3 \times$ Ile1207 → Arg, $3 \times$ Ile1207 → Lys) that was not described until now. To localize this newly identified resistant mutation, structure models of VP1 were generated with the Swiss–PdbViewer. The results indicate that amino acid in position 1207 belongs to the GH-loop of VP1 and is located near to the heel of the foot-shaped pocket. This loop was previously shown to undergo conformational changes during drug binding. Furthermore, the virulence of the pleconaril-sensitive CVB3 97927 and 3 of its pleconaril-