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Differential Effects of Protein Kinase C Inhibitors on Nuclear and Cytoplasmic *DMPK* Transcript Dynamics in Myotonic Dystrophy Type 1 Cells

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Abstract

The major molecular basis for myotonic dystrophy type 1 disorder is the occurrence of a mutation leading to expanded CTG triplet units in the 3'-untranslated region of Dystrophia Myotonica Protein Kinase (*DMPK*) gene, which leads to the formation of foci in the nuclei of these cells. An assay based on the *DMPK* transcript (*Bpm I* polymorphism) was developed to test the effect of compounds of protein Kinase C inhibitors- hypericin and Ro 31-8220- to determine whether either inhibitor affected the transport of DMPK transcripts from nucleus to the cytoplasm or reduced the proportion of the transcript in both fibroblast and myoblast cells. The results from this study showed that the protein kinase C inhibitors were not able to affect the relocation of mutant *DMPK* transcripts from the nucleus to the cytoplasm in both DM1 fibroblasts and myoblasts, but Ro 31-8220 significantly reduced mutant transcripts in the nuclear fraction of DM1 fibroblasts. In the nuclei of fibroblasts, Ro 31-8220 treatment showed a 38.5±5.4% proportion of mutant transcript, hypericin treatment exhibited 54.2±3.5% mutant transcript, while those of DMSO treatment and untreated were 43.7±3.0% and 51.4±0.8%, respectively. For cytoplasmic fractions of DM1 fibroblasts, Ro 31-8220-treated cells indicated 94.4±1.7% normal transcript, hypericin treatment showed 91.6±1.0%, with DMSO-treated and untreated cells having 90.0±1.9% ad 89.5±2.3%, respectively. In the DM1 myoblasts nuclear fraction, 61.8±1.7% mutant transcript was observed in Ro 31-8820- treated cells, 67.5±2.8% mutant transcript occurred in hypericin treatment, while DMSO treatment and untreated cells had 52.4±1.7% and 48.7±0.8%, respectively. In myoblast cytoplasmic fractions, hypericin and Ro 31-8220 showed 90.6±2.7% and 80.8±0.3% normal transcripts, respectively, with DMSO treatment showing 81.1±0.5% normal transcript, while the untreated showed 80.0±5.7% normal transcript. This study has discovered the role of Ro 31-8220 in reducing the proportion of mutant transcripts I nuclear fraction while increasing the levels of the normal transcripts was increased in the cytoplasmic fraction of DM1 cells. Taken together, results from this study suggest that Ro 31-8220 may have therapeutic potential for reducing mutant *DMPK* transcript levels with potential therapeutic value.

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1.0 Introduction

One of the molecular hallmarks of myotonic dystrophy is the expansion of non-coding repeat sequences of expanded repeat CTG units of Dystrophia Myotonica Protein Kinase gene (*DMPK)* for myotonic dystrophy type 1 (DM1) (Brook *et al*,. 1992; Fu et al., 1992; Mahadevan et al., 1992) or noncoding CCTG expanded repeat units in intron 1 of Zinc Finger protein 9 gene (*ZNF9*) which was previously referred to as Cellular Nucleic Acid Binding Protein gene (*CNBP*) for myotonic dystrophy type 2 (DM2) (Liquori *et al*., 2001). The mutation in the repeat units leads to the expansion of the repeat units out of its normal range, and mutation has also been a driving force in determining susceptibility to infections as well (Udosen & Nya, 2017; Udosen, 2019). The noncoding repeat units lead to the production of mutant transcripts, which are toxic and interact with splicing protein factors such as muscleblind proteins to entrap them in the nucleus of DM cells, resulting in the formation of foci, a major feature associated with the disease condition (Davis *et al*., 1997; Fardaei *et al*., 2001; Mankodi *et al*., 2001; Fardaei *et al*., 2002; Taneja *et al*., 1995). As a consequence, one of the major splicing factors (muscleblind proteins) is prevented from performing its cellular functions of regulated alternative splicing of a subset of genes, thereby inducing abnormal splicing events which contribute to the symptoms associated with the disease condition (Philips *et al*., 1998; Mankodi *et al*., 2002; Savkur *et al*., 2001; Kimura *et al*., 2005; Lin *et al*., 2006).

For the DM condition, a defect in the regulation of alternative splicing takes place in a few splicing conditions which are usually regulated in a developmental manner, but in the disorder, it cannot express the adult isoform mRNA in adult individual tissues (Lin *et al*., 2006). The regulation of alternatively spliced transcripts could be total or partial (Ranum & Cooper, 2006). Consequently, fetal splice variants continue to be expressed in adult tissues and protein that is functionally inactive and irrelevant in adult individual tissues, thereby inducing DM symptoms.

The misregulation of alternative splicing is one of the molecular features, along with nuclear foci formation, which is currently being used as a potential target for therapy. Alternative splicing abnormality associated with DM was exploited in a study showing that Protein Kinase C inhibitors effected the reversal of misregulated splicing in a cell-based splicing assay model (Udosen *et al*., 2023). Previous studies demonstrated that Protein Kinase C inhibitors disrupt nuclear foci, but it remains unknown whether these compounds directly influence mutant *DMPK* transcript abundance or intracellular localization. The use of mutant *DMPK* transcript as molecular target for therapy has not been explored hence it informs the basis for its application in this study. In this study, two protein Kinase C inhibitors, namely-hypericin and Ro 31-8220, were utilised to assess their effects on nuclear foci in DM cells as well as their ability to influence the proportional amounts of *DMPK* transcripts in both the nuclear and cytoplasmic fractions.

2.0 Materials and Methods

2.1 Cell Culture

The fibroblast cell line used in this study was a human DM1 patient fibroblast cell line informative for *Bpm 1* polymorphism, which had been previously telomerised and were obtained from Professor David Brook's laboratory in the University of Nottingham, United Kingdom. The fibroblast cells contained an inducible MyoD, which in the presence of doxycycline, induces the fibroblasts to differentiate into myoblasts (Udosen *et al*., 2023). Sub-culturing of cells in flasks was carried out at 37^{o}C incubation by utilising 2.0ml of 0.25% Trypsin-EDTA for 5-10 minutes under 5% CO_2. The protocols were carried out in a laminar workflow safety cabinet operating under sterilised environmental conditions (Udosen *et*

al., 2023; Udosen *et al*., 2026). The study with cell line was performed in line with the University of Nottingham research guidelines and ethics.

2.2 Compound Treatment

The compounds utilised for this study were the protein kinase C inhibitors, namely hypericin and Ro 31-8220, along with dimethyl sulphoxide (DMSO) as controls. During a pilot study using 96-well plates, concentrations of 100 µM, 48 µM, 8 µM and 1.6 µM were utilised and optimised for application to T-75 flasks with 48 µM as the optimal concentration. Compound treatments' replicates were performed in triplicate for 48 hours.

2.3 Dystrophia myotonica protein kinase (*DMPK*) transcript analysis

Separate fractions of nuclear and cytoplasmic RNA were extracted from cultured cells based using a minor modification of the previous protocols (Hamshere *et al.*,1997). Firstly, cells were washed with freeze-cold PBS, followed by the addition 1ml of 0.65% Nonidet P-40 (NP-40). NP-40 was added to the cells in the flask for about 1-2 minutes in order to disrupt the cell membrane to release its cytoplasmic contents, so that its nuclear membrane will still remain intact, which gives a grainy appearance under the microscope. The flask contents were drained off into a new tube as cytoplasmic extracts. The nuclei of the cells were dislodged by the addition of 2mls of NP-40 to effect its removal from the flask into a tube, and the nuclear extracts were subsequently centrifuged to pellet them. The supernatant was removed, and 400µl of DEPC H_2O was used to resuspend the nuclear pellet, followed by the introduction of 100µl of 0.5M Tris HCl, 0.05M EDTA and 2.5% (v/v) SDS to the nuclear extracts. For the isolation and purification of cytoplasmic extracts, 250 µL of the mixture was added. Both nuclear and cytoplasmic RNA fractions were purified using two equal volumes of phenol/chloroform extraction mixtures. The RNA was immediately precipitated by centrifugation in isopropanol consisting of 0.3 M sodium acetate at pH 5.3, in equivalent volumes. The RNA pellets were washed using 75% ethanol, centrifuged for isolation of supernatants. The RNA pellet was air-dried for 15-20 minutes at room temperature, followed by its dissolution in 40µl of DEPC H_2O, prior to its estimation and measurement by nanodrop. RNA samples obtained from DM1 fibroblasts were subjected to DNase1 treatment before cDNA synthesis by reverse transcription utilising New England Biolabs protocols prior to PCR for *Bpm1* assay. For the *Bpm I* assay of *DMPK* transcript, 1/20th of cDNA was used for PCR with N11 (5'CACTGTCGGACATTCGG-GAAGGTGC3'), the forward oligonucleotide primer, while 133 (5'GCTTGCACGTGTGGC-TCAAGCAGCTG3') was the reverse oligonucleotide primer. Amplification was carried out by minor modifications of the protocols from Hamshere et al. (1997). The PCR comprised of denaturation of DNA at a temperature of 95ºC for a duration of 5 minutes, followed by repeated cycles of 95ºC for a duration of 30 seconds; annealing of oligonucleotide primers for 1 minute at 58ºC; and extension at 72ºC for a duration of 1 minute. It was subsequently followed by the final extension at 72ºC for 5 minutes. The PCR product was heated to 98ºC for 1 minute, then cooled to 4ºC for 10 minutes to induce homoduplex formation overnight, followed by *Bpm I* digestion. The final products of the restriction enzyme were run on 3% agarose gels and visualised by the Gel Documentation System. For quantification of the relative levels of expression of normal and mutant DMPK alleles, an N11 primer with a fluorescent label was employed in amplification of DMPK at 22-26 cycles, and the resulting products were restricted overnight with *BpmI* prior to analysis on the Genescan genetic analyser.

Statistical analysis was performed using a t-test in Graphpad prism software with p= 0.05 used as the basis for estimation of the level of significance.

3.0 Results

In this study, the effect of the Protein Kinase C inhibitors (hypericin and Ro 31-8220) on mutant *DMPK* transcripts was assessed to determine whether they removed it from the nuclei of DM1 cells or reduced its proportion.

In *DMPK* analysis, *Bpm1* restriction of the amplicon gave 154bp and 138bp for normal and mutant transcript, respectively. In order to determine if mutant transcripts were released from the nucleus following compound treatment, it was observed that both PKC inhibitors did not release the mutant transcripts into the cytoplasm, showing that the transcripts were still trapped in the nucleus (Figure 1).

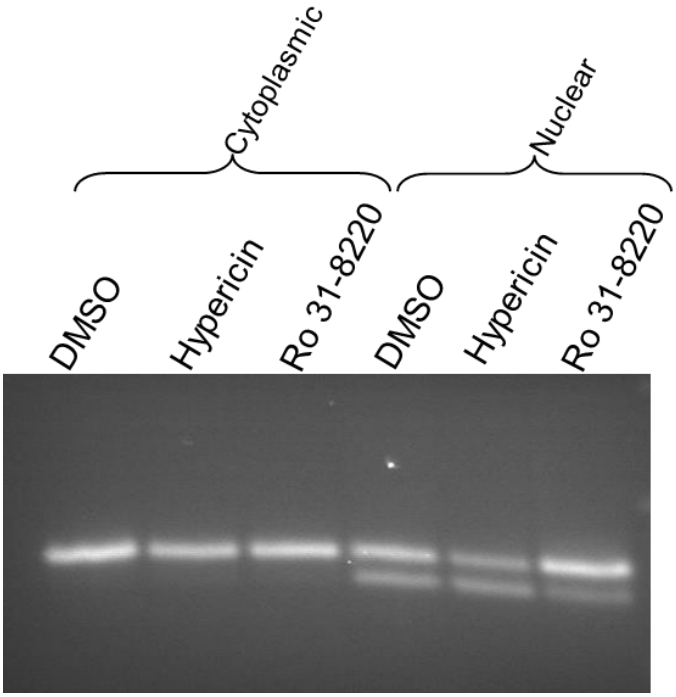

Figure 1: Protein Kinase C inhibitor treatments of DM1 fibroblast showing no effect on translocation of mutant DMPK transcript (138bp) from the nucleus to the cytoplasm, while the normal transcript (154bp) occurs in both the nucleus and cytoplasm

Subsequently, compound treatments in DM1 fibroblast cells were assessed for their effect on the *DMPK* transcripts to determine if they decreased or increased the proportion of the mutant transcripts in the nuclear fraction. It was observed that protein kinase C inhibitor treatment altered the ratio of normal transcripts relative to mutated *DMPK* transcripts. In the nuclear fraction, Ro31-8220 had 38.5±5.4% mutant transcripts compared to 54.2±3.5% for hypericin treatment, while DMSO and untreated exhibited mutant transcripts of 43.7±3.0% and 51.4±0.8%, respectively (Figure 2). From the results above, Ro31-8220 significantly reduced the proportion of mutant nuclear transcripts relative to untreated controls.

When the DM1 fibroblast was induced by the presence of doxycycline to differentiate into myoblasts and subjected to compound treatments, the PKC inhibitors treatment also confirmed the non- release of mutant *DMPK* transcripts into the cytoplasm (Figures 4 and 5). It was observed that the proportion of repeat expanded mutant transcripts in Ro31-8220-treated myoblasts was 61.8±1.7%, hypericin-treated myoblasts showed 67.5±2.8% mutant *DMPK* transcripts, while that of DMSO and untreated myoblasts showed 52.4±1.7% and 48.7±0.8% proportion of the mutant transcript, respectively Figures 2 and 3). It was observed that both Ro31-8220 and hypericin-treated myoblasts had more mutant transcripts than controls. Statistically, Ro31-8220 treatment was

significantly different from DMSO treatment (p=0.0.003) and untreated myoblasts (p=0.0003). In addition, hypericin treatment was significantly greater than DMSO treatment (p= 0.001) and untreated myoblast cells (p= 0.0004). The effect of Ro31-8220 treatment was statistically significant (p= 0.01), whereas that of hypericin treatment was not significant (p= 0.25) compared to untreated cells.

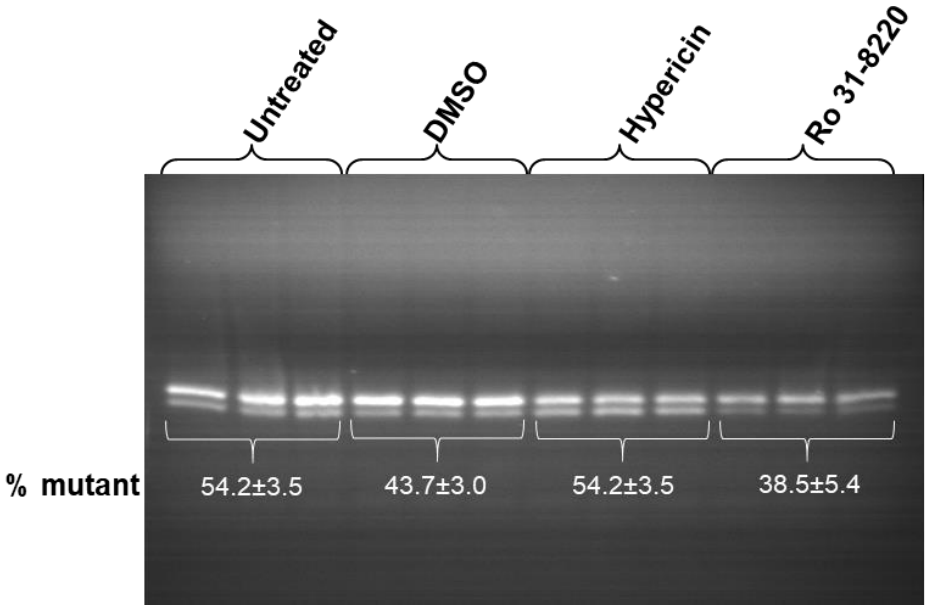

Figure 2: Gel resolution of both normal and mutant *DMPK* transcripts from the nuclear fractions of DM1 fibroblasts across different treatments. The experimental values are expressed as mean ± standard deviation

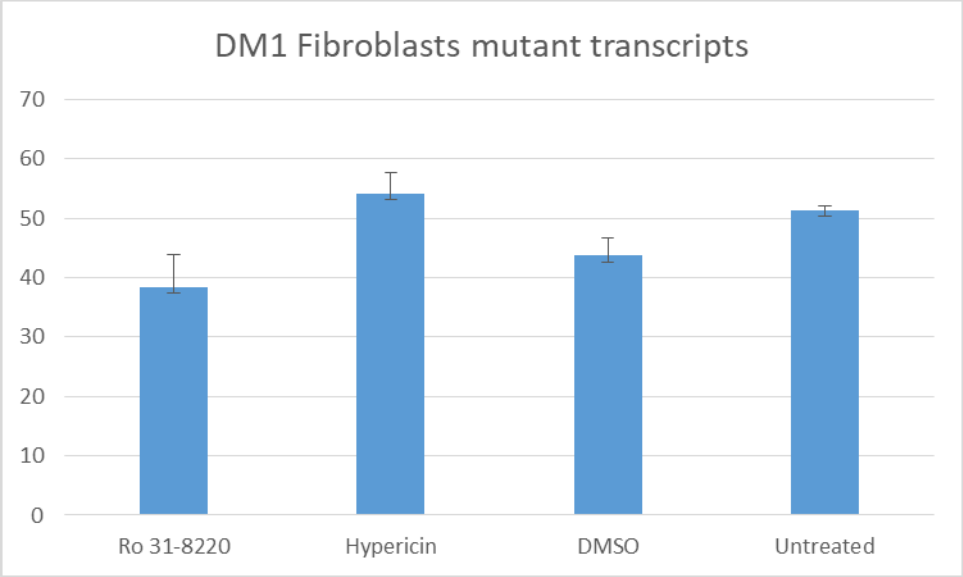

Figure 3: The relative proportions of mutants DMPK transcripts in different treatments of DM1 fibroblasts

When the cytoplasmic fractions of DM1 fibroblasts and DM1 myoblasts were analysed for the proportions of normal *DMPK* transcripts, it was observed that in DM1 fibroblasts, Ro31-8220 and hypericin treatments showed 94.4±1.7% and 91.6±1.0% respectively, while that of DMSO was 90.0±1.9%, with untreated exhibiting 89.5±2.3% proportion (Figures 6 and 7). Ro31-8220 treatment increased the relative proportion of the normal DMPK transcripts in the cytoplasm of DM1 fibroblast in comparison with controls. Statistically, Ro31-8220 was significant when compared to controls (p= 0.04 for both DMSO and untreated), while that of hypericin was not significant (p= 0.27 for DMSO, p= 0.22 for untreated).

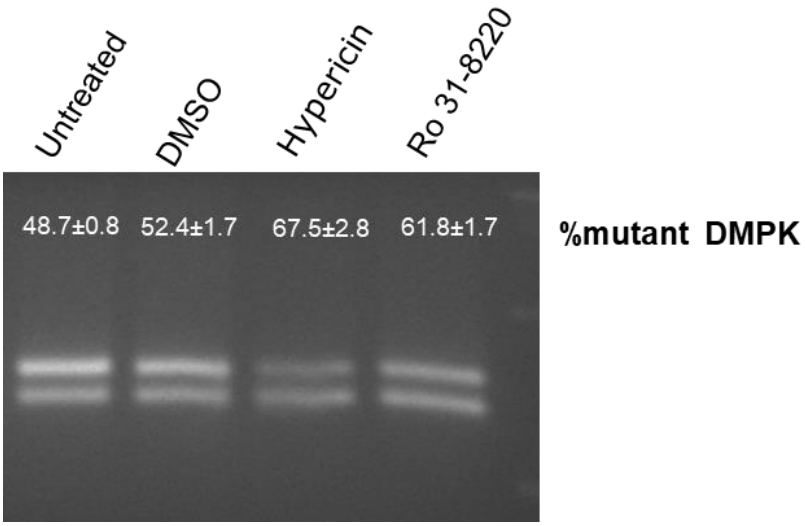

Figure 4: Gel resolution of normal (154bp) and mutant *DMPK* transcripts (138bp) from the nuclear fractions of different treatments in DM1 myoblasts. Experimental values are expressed as mean ± standard deviation

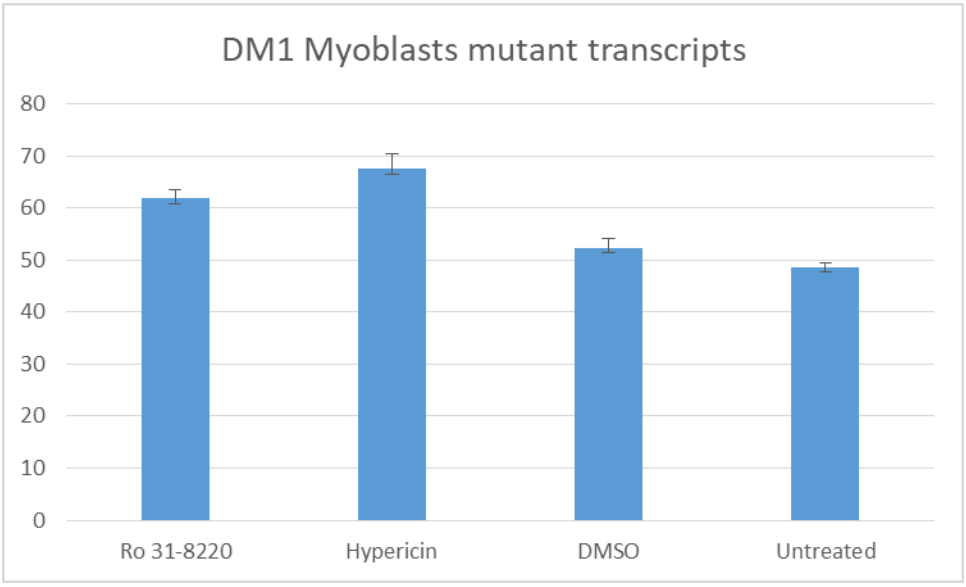

Figure 5: Mutant *DMPK* transcript proportions from the nuclear fractions of different treatments of DM1 myoblasts

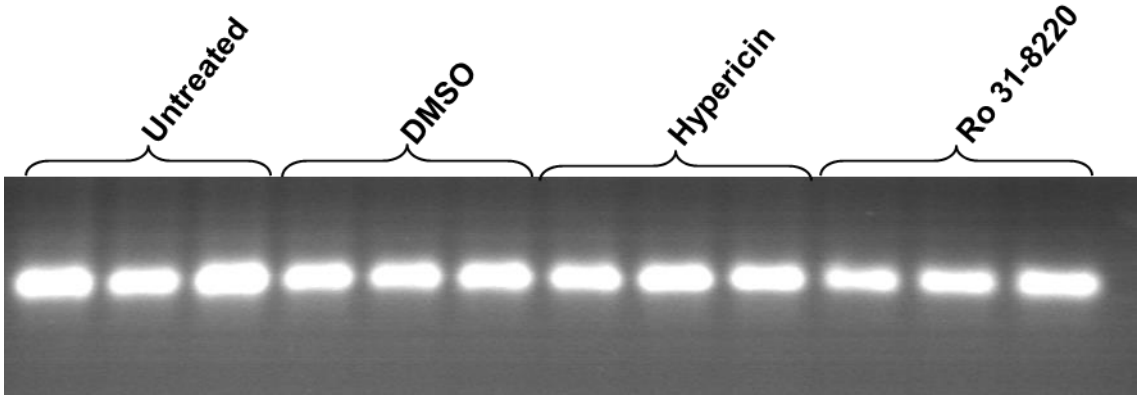

Figure 6: Cytoplasmic fraction of DM1 fibroblasts showing only the occurrence of normal *DMPK* transcripts across the different treatments

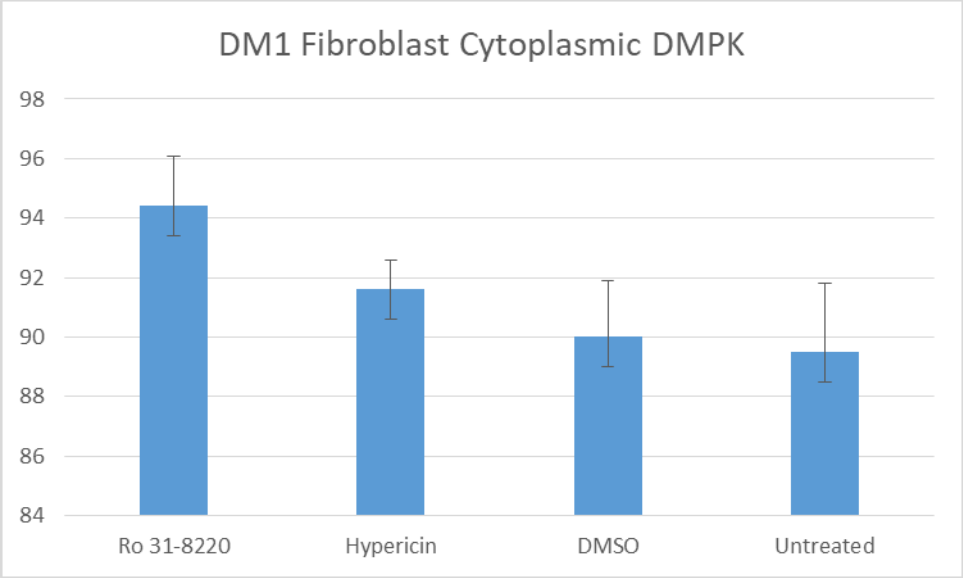

Figure 7: The relative proportions of the normal *DMPK* transcript from the cytoplasmic portions of different treatments of DM1 fibroblasts

In DM1 myoblasts, the proportion of normal *DMPK* transcript was 82.8±0.3% for Ro31-8220 treatment, 90.6±2.7% for hypericin treatment, while that for DMSO and untreated was 81.1±0.5% and 80.0±5.7%, respectively (Figures 8 and 9). Statistical analysis showed that Ro31-8220 was significant compared with DMSO (p= 0.007) but was insignificant in comparison with untreated (p= 0.44). However, hypericin treatment showed a significant difference compared with controls (p= 0.004 for DMSO and p= 0.04 for untreated). Both Ro31-8220 and hypericin significantly increased the relative proportion of the normal DMPK transcripts in the cytoplasmic fraction of DM1 myoblasts.

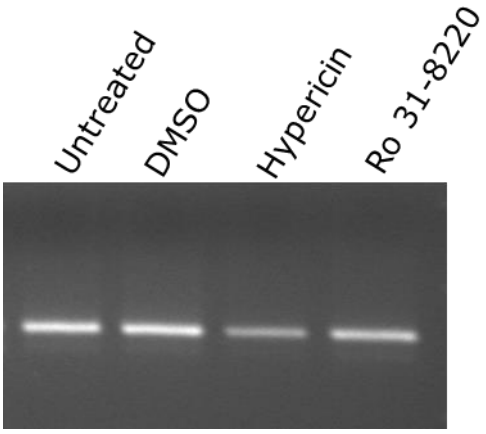

Figure 8: Cytoplasmic *DMPK* transcript showing the normal transcripts (154bp) on agarose gel

4.0 Discussion

Mutations in the *DMPK* gene are responsible for myotonic dystrophy type 1, an inherited neuromuscular disorder which contribute to altered cellular function and mutations also contribute to susceptibility to infections (Udosen & Nya, 2017; Udosen, 2019). In myotonic dystrophy type 1, the mutation cause association of muscleblind proteins with expanded CUG repeats to form foci. From a previous study, both hypericin and Ro 31-8220 were observed to disrupt muscleblind protein-trinucleotide repeat interactions that form nuclear foci in DM1 fibroblast and myoblast cells, leading to clearance of foci in these cells (Ketley *et al*., 2014). This justified their use in this study to assess their effect on one of the biomarkers of the disorder. From this study, it was observed

that the proportion of the mutant *DMPK* transcripts containing the expanded CUG repeats that cause the myotonic dystrophy condition was higher in the DM1 myoblasts treated with the protein kinase C inhibitors than in the DM1 fibroblasts. This could be due to the fact that the average number of nuclear foci per cell, which differs between myoblasts and fibroblasts with myoblasts showing an average of 5 foci per cell in contrast to fibroblast which exhibit an average of four foci per cell which is in tandem with a previous study (Holt *et al*., 2009). The treatments with protein kinase C inhibitors effected a greater reduction in the proportion of the nuclear mutant DMPK transcripts in fibroblast in comparison to myoblast cells with Ro 31-8220, showing a substantial effect in the reduction of the transcript. It is possible that Ro 31-8220 could specifically affect the degradation of the mutant *DMPK* transcript containing expanded CUG repeats thereby resulting in its reduction in the nuclei. Additionally, it could be suggested that hypericin had no effect on Protein Kinase C signaling pathways nor did it have any effect on expanded CUG RNA in the cell model under study.

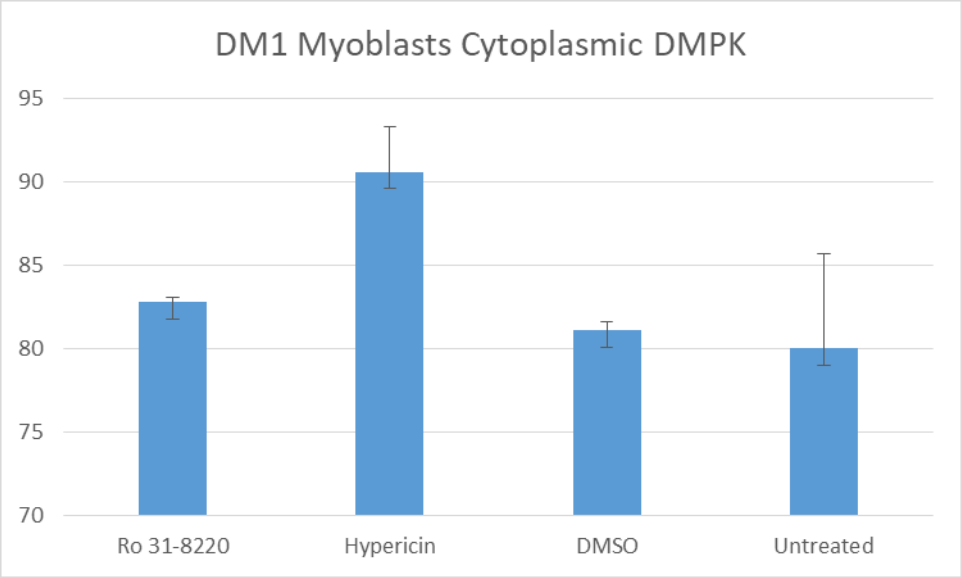

Figure 9: The proportion of normal *DMPK* transcripts from the cytoplasmic fraction of different treatments of DM1 myoblasts

When the effect of the compounds was assessed on the proportion of the normal *DMPK* transcripts in the cytoplasmic cellular fractions, it was discovered that both protein kinase C inhibitors had an effect in increasing the percentage proportions of the normal transcripts in the cytoplasms of both fibroblast and myoblast cells in relation to the controls, with hypericin having a greater effect. This seems to suggest that both protein kinase C inhibitors could have possible effects in ameliorating effects of the disease through increasing the proportions of the normal transcripts in the cytoplasms of DM1 cells. However, this study is limited due to the cell model hence it would need further validation using a mice model of the disease. Additionally, it should be noted that cellular metabolism and physiology may differ between fibroblasts and myoblasts which could account for the differential effects of the protein kinase C inhibitors between these cell lines. Indeed, these protein kinase C inhibitors were observed from a previous study to affect splicing abnormalities in myotonic dystrophy type 2 (DM2) cell line (Udosen, 2020) as well as DM1 cellular splicing assay (Udosen *et al*., 2023). This study extends application of protein kinase C inhibitors on investigating its action on the distribution of the mutant DMPK transcript. In conclusion, this study suggests that Ro 31-8220

could be a possible therapeutic agent based on its effect on *DMPK* transcripts in both cytoplasmic and nuclear fractions of DM1 cells.

In conclusion, the protein kinase C inhibitors, Ro 31-8220, effected a reduction in mutant transcript in the nucleus of DM1 fibroblasts while hypericin increased the proportion of the wild transcripts in the cytoplasm of both DM1 fibroblasts and myoblasts.

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