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ANTI-ERYPTOTIC ROLE OF QUERCETIN: A NOVEL THERAPEUTIC APPROACH TO ATTENUATES HYPEROSMOLARITY-INDUCED OXIDATIVE DAMAGE IN ERYTHROCYTES

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Abstract

Quercetin, a dietary flavonoid, found in abundance in many food sources like vegetables, tea and fruits and has widely been studied due to its anti-inflammatory, antioxidative and cytoprotective properties. It has strong ability to scavenge free radicals helping in reduction of oxidative stress in cellular systems. In human erythrocytes it helps to reduce hyperosmotically induced oxidative stress that are the main triggers of eryptosis, programmed death of erythrocytes, mainly characterized by blebbing, cell shrinkage and PS exposure. In this study the antioxidative effect of quercetin was studied by using different therapeutic doses cited in literature on nucleated cells, resulted in increased activity of antioxidant defense system and reduced oxidative stress. This was confirmed by performing antioxidant enzyme assay (SOD, GPx and CAT). Mean cell volume (MCV) was evaluated by using hematology analyzer. As elevated intracellular Ca^{2+} is the main trigger of eryptosis hence the role of Ca^{2+} in eryptosis induction was confirmed by using amlodipine, a Ca^{2+} channel blocker. Hemolysis % was estimated to find out the cytotoxicity of quercetin. The findings of this study confirmed that the therapeutic doses of quercetin (25, 50 and 75μM) exhibited anti-eryptotic potential via mitigating the oxidative stress and subsequent Ca^{2+} entry, ultimately reduction in eryptosis.

Introduction

Quercetin, a dietary flavonoid, has widely been studied due to its anti-inflammatory, antioxidative and cytoprotective properties (1). It has strong ability to scavenge free radicals and can chelate transition metals due to its polyphenolic structure thereby helping in reduction of oxidative stress in cellular systems (2). Erythrocytes are highly vulnerable to oxidative stress and hyperosmotic shock due to their continuous motion in blood vessels as they are exposed to oxygen tension and due to autoxidation of hemoglobin (3). Although erythrocytes are devoid of mitochondria and nuclei, can still be able to maintain a delicate balance of membrane integrity, ionic balance, and redox homeostasis (4). Any disruption in this balance particularly under pathological conditions can lead to eryptosis, a programmed erythrocytes death, which is characterized by membrane blebbing, shrinkage of cell, externalization of phosphatidylserine (PS) (5, 6). Elevated level of reactive oxygen species (ROS) may lead to raised oxidative stress. Oxidative stress and hyperosmotic shock cause the activation of calcium sensitive channels which

lead to the influx of Ca^{2+} to the interior of cell (7). Higher intracellular Ca^{2+} leads to the activation of calpain, a cysteine protease, and scramblase, responsible for externalization of PS to the outer surface of cell membrane serving as an eat-me signal for macrophages (5, 8). Oxidative stress also inactivates flippase, responsible for maintaining the cell membrane symmetry by flipping back the PS to interior of cell membrane, all these circumstances lead to the distortion of cellular symmetry leading to PS exposure and increased eryptosis (9). Although eryptosis is a physiological phenomenon but its excessive activation may lead to the uncontrolled clearance of erythrocytes from circulation which leads to anemia, impaired oxygen delivery and resulting in systemic inflammation in wide-ranging pathological conditions like cardiovascular disease, diabetes, kidney disease (10). Quercetin plays an essential role in conserving the antioxidant defense system of erythrocytes by maintain the level of glutathione and reducing lipid peroxidation, consequently strengthening erythrocytes membranes (11). Due to its antioxidant properties quercetin helps in inhibition of Ca^{2+} influx thereby preventing the activation of calpain that is involved in destabilizing the cytoskeleton and promote excessive eryptosis (12, 13). It also has role in modulating intracellular signaling pathways like MAPK cascades and protein kinase C involved in regulation of eryptosis (14). These multidimensional effects proposed that quercetin functions both as an antioxidant and an active regulator of eryptosis. This research article systematically assesses quercetin role in erythrocytes survival, with focus on molecular mechanism, impact on eryptosis and potential therapeutic applications in oxidative stress-related diseases. By clarifying these aspects this study helps in seeking how natural compounds help in hematological health and in identification of novel approaches for mitigating premature clearance of erythrocytes from circulation.

Methodology

For experiments to be performed screened and fresh blood was collected from blood banks of Faisalabad, Pakistan by preapproval of Directorate of Graduate Studies (DGS). The study was also approved by the Institutional Bioethics Committee (IBC), University of Agriculture, Faisalabad, Pakistan. Erythrocytes from whole blood were collected after centrifuging the blood. After centrifugation, isolated erythrocytes were stored in an incubator for 48 hours having hematocrit at 0.4% in ringer solution at 37 °C. ringer solution provides an isotonic environment to the cells (15). Different therapeutic doses of quercetin are then added to the isolated erythrocytes.

Oxidative stress measurement

Antioxidant enzyme assay (Catalase, Superoxide dismutase, Glutathione peroxidase) was performed to determine the level of oxidative stress experienced by erythrocytes before and after the addition of phytochemical, quercetin. Determination of hemolysis was done centrifuging the incubated samples (3 minutes at 3000 rpm, room temperature), and the plasma (supernatant) was collected. 100% hemolysis is the absorption of erythrocytes that are disintegrated in the distilled water (16). Superoxide dismutase activity was measured by using protocol method that uses a reaction mixture including methionine, riboflavin and phosphate buffer in specific concentrations. All these reaction mixtures are taken into 96-well plate and absorbance was taken at 560nm in an ELISA plate reader (17, 18). Glutathione peroxidase was evaluated by the addition of 0.1 mL enzyme extract, 40 mM H_2O_2, and 20 mM guaiacol at pH of 5 in a 96-well plate and absorbance was taken at 470nm in an ELISA plate reader (19). Catalase activity was evaluated by adding the reaction mixture comprising of H_2O_2 5.9 nM, 0.1 mL enzyme extract and 50 mM phosphate buffer at pH 7 in 96-well plate and then the activity was measured by taking absorbance at 240 nm in an ELISA plate reader (17, 20).

Cell size measurement

Measurement of cell size of treated (with effective dose of quercetin, 50 µM) and untreated cells were measured through mean cell volume (MCV) along with the aid of automated hematology analyzer (21).

Confirmation of the role of Calcium (Ca^{2+})

To explore the effect of Ca^{2+} on eryptosis, a calcium channel blocker like amlodipine, along with quercetin, was used to treat erythrocytes. This was done by treating erythrocytes with amlodipine (10 µM) in isotonic ringer solution. MCV was done to check the volume change in erythrocytes, after 24 hours. This allowed to investigate the potential anti-eryptotic properties of these substances (18).

Hemolysis measurement

For the confirmation of anti-hemolytic effect of quercetin, erythrocytes samples were centrifuged for 3 minutes at 37 °C. At 405nm wavelength hemoglobin level in supernatant was measured. 100% hemolysis was considered by the absorbance of RBCs lysed in d.H_2O (22).

Statistical analysis

For statistical analysis all results were interpreted as mean ± SEM (standard error of mean). One-way ANOVA (Analysis of variance) and Tukey's test as post-test were performed. GraphPad InStat was used for this analysis (17, 23).

Results

The purpose of this study was to analyze the anti-eryptotic effect of quercetin by implementing the oxidative stress (by sucrose) that is one of the eryptosis-stimulating mechanisms. To validate this, hemolysis measurement, oxidative stress measurement, cell size measurement, and role of Ca^{2+} in inhibition of eryptosis were all evaluated. The findings of this research are shown in graphs, featuring mean values ±SEM with clear indication of statistical significance. Investigation of hemolysis was done with the exposure of sucrose at different concentrations (350 mM, 450 mM, 550 mM) to erythrocytes. The supernatant was collected and poured on 96-well ELISA plate and the percentage of hemolysis was observed via an ELISA plate reader/spectrophotometer.

Oxidative stress measurement

To determine the consequence of sucrose against oxidative stress, antioxidant defense was evaluated by the ELISA plate reader.

Superoxide dismutase

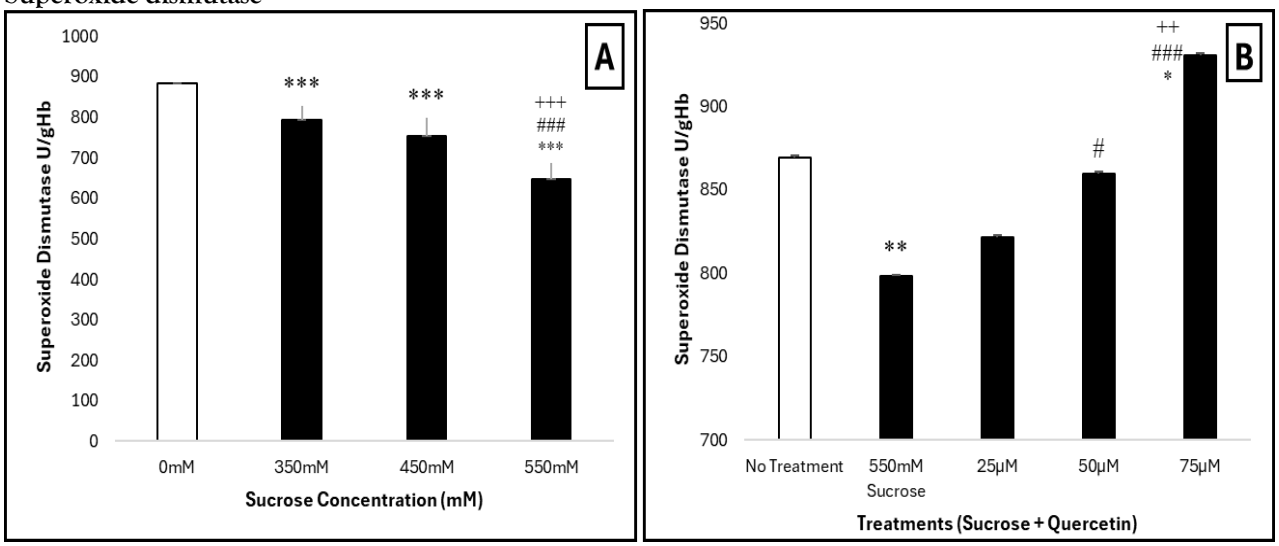

Figure 1 Superoxide dismutase activity in the presence and absence of quercetin

Arithmetic means ±SEM (n=12) were estimated. **Figure 1 (A)** shows erythrocytes samples treated with sucrose and incubated for 48 hours with a ringer solution. Erythrocytes in the absence of sucrose (control) are shown with white bar and in the presence of sucrose (350mM, 450mM, 550mM) are shown with black bars respectively. The graph was plotted between hemolysis % (y-axis) and different sucrose concentrations (x-axis). The obvious variation between treated and non-treated samples is shown by ***($p<0.01$), ***($p<0.01$), ***($p<0.01$) and the significant difference between treatment A and C was shown by $^{\#\#\#}$($p<0.01$), treatment B and C was shown by $^{+++}$($p<0.01$). The results showed that there was a decrease in superoxide dismutase activity due to an increase in sucrose concentration. Sucrose disrupted the normal redox balance that causes the decrease in SOD's activity. In **Figure 1 (B)** different therapeutic concentrations of quercetin were added showing a remarkable increase in enzymes activity. Significant difference between non-treated and treated samples were shown as **($p<0.01$), *($p<0.05$) and between treatments $^{\#}$($p<0.05$), $^{\#\#\#}$($p<0.001$).

Glutathione peroxidase:

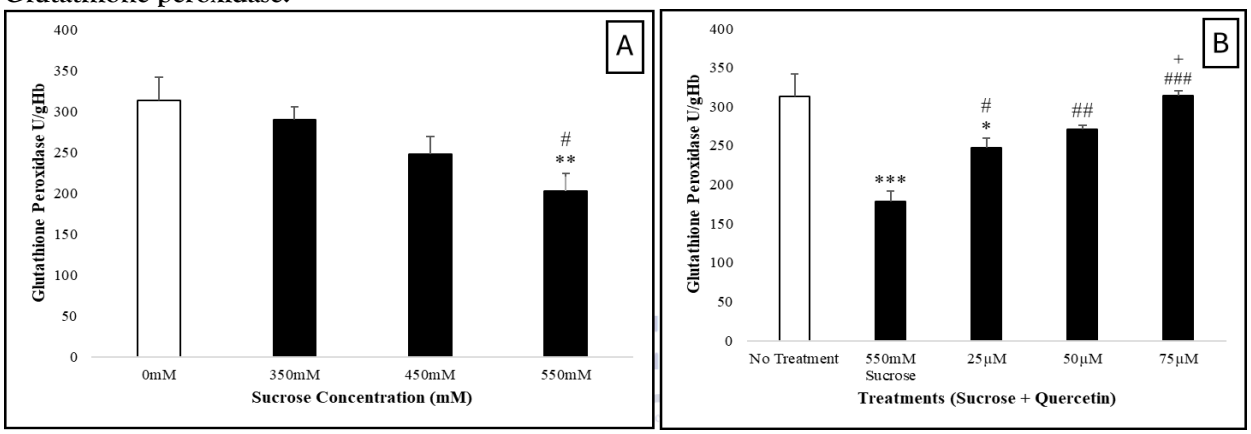

Figure 2 Superoxide dismutase activity in the presence and absence of quercetin

Arithmetic means ±SEM (n=12) were estimated. In **Figure 2 (A)** erythrocytes were treated with sucrose and incubated for 48 hours with a ringer solution. Erythrocytes in the absence of sucrose (control) is shown with white bar and in the presence of sucrose (350mM, 450mM, 550mM) is shown with black bars respectively. Graph was as for superoxide dismutase activity. The obvious distinction between control and treatment in enzyme activity showed by **($p<0.01$) and within treatments $^{\#}$($p<0.05$). The results showed that there was decrease in glutathione peroxidase activity due to increased sucrose concentration. While in **Figure 2 (B)** Arithmetic means ± SEM (n=12) were estimated. Erythrocytes were treated with sucrose and quercetin and incubated for 48 hours with a ringer solution. Erythrocytes in the absence of sucrose (control -) is shown with a white bar and in the presence of sucrose 550mM (control+) is shown with a black bar and erythrocytes in the presence of sucrose and quercetin (25µM, 50µM, and 75µM) is also shown with black bars respectively. Graph was plotted between catalase activity (y-axis) and different quercetin concentrations (x-axis). The obvious distinction between control and treatment in enzyme activity showed by ***($p<0.001$) and *($p<0.01$) and variation between the treatment A and other samples shown by $^{\#}$($p<0.01$), $^{\#\#}$($p<0.01$), $^{\#\#\#}$($p<0.01$). significant variation between treatment B and D was shown by $^{+}$($p<0.05$).

Catalase

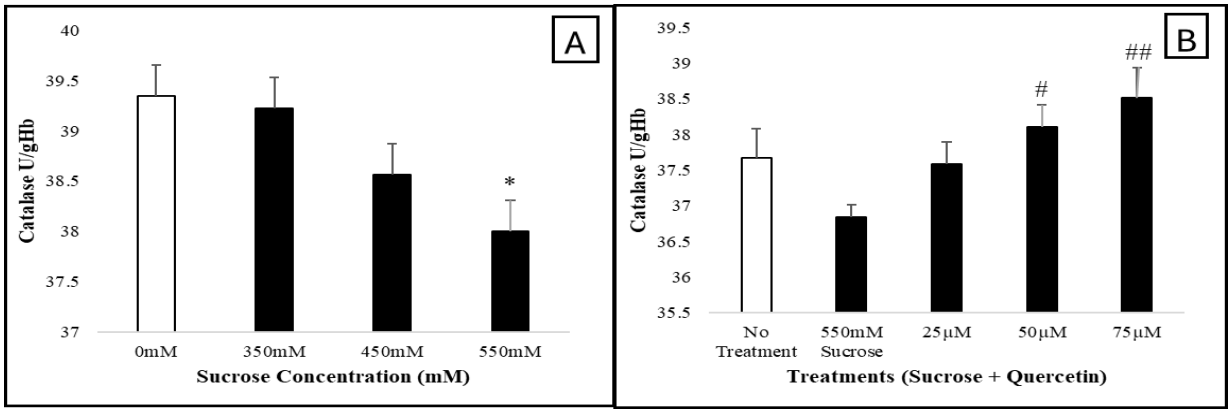

Figure 3 Superoxide dismutase activity in the presence and absence of quercetin

Arithmetic means ±SEM (n=12) were estimated. **Figure 3 (A)** shows erythrocytes treated with sucrose and incubated for 48 hours with a ringer solution. Erythrocytes in the absence of sucrose (control) is shown with white bar and in the presence of sucrose (350mM, 450mM, 550mM) is shown with black bars respectively. Graph is plotted between hemolysis % (y-axis) and different sucrose concentrations (x-axis). The obvious distinction between control and treatment in enzyme activity showed by *($p<0.05$). The results showed that there was decrease in catalase activity due to increase sucrose concentration. Whereas in **Figure 3 (B)** arithmetic means ± SEM (n=12) were estimated. Erythrocytes were treated with sucrose and quercetin and incubated for 48 hours with a ringer solution. Erythrocytes in the absence of sucrose (control -) are shown with a white bar and in the presence of sucrose 550mM (control+) are shown with black and erythrocytes in the presence of sucrose and quercetin (25µM, 50µM, and 75µM) was also shown with black bars respectively. Graph is plotted between catalase activity (y-axis) and different quercetin concentrations (x-axis). The obvious distinction between treatment A and other treatments in enzyme activity showed by #($p<0.05$) and ##($p<0.01$). The results showed that there was an increase in catalase activity due to increase in quercetin concentration.

Cell size measurement

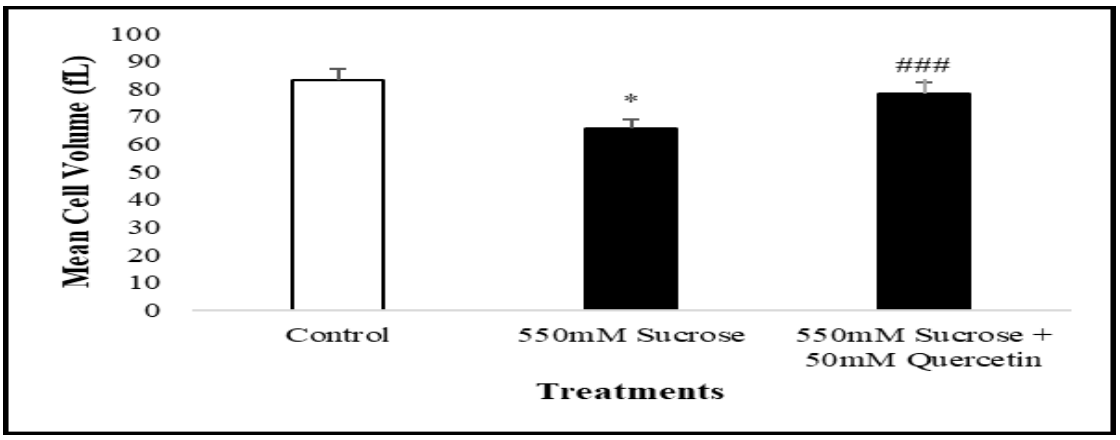

Figure 4; Effect of quercetin on erythrocytes mean cell volume was evaluated. Bars show the

mean ± SEM (n = 12) values. Isolated erythrocytes were incubated for 48 without quercetin and sucrose (white bar) and with sucrose and sucrose along with quercetin (black bars). Significant difference between control and sample containing sucrose was*(p< 0.05) and between sample containing sucrose and sucrose along with quercetin was ###(p<0.001). the results showed cell size returned to normal size (80-100) upon addition of quercetin.

Determination of role of Ca^{2+}

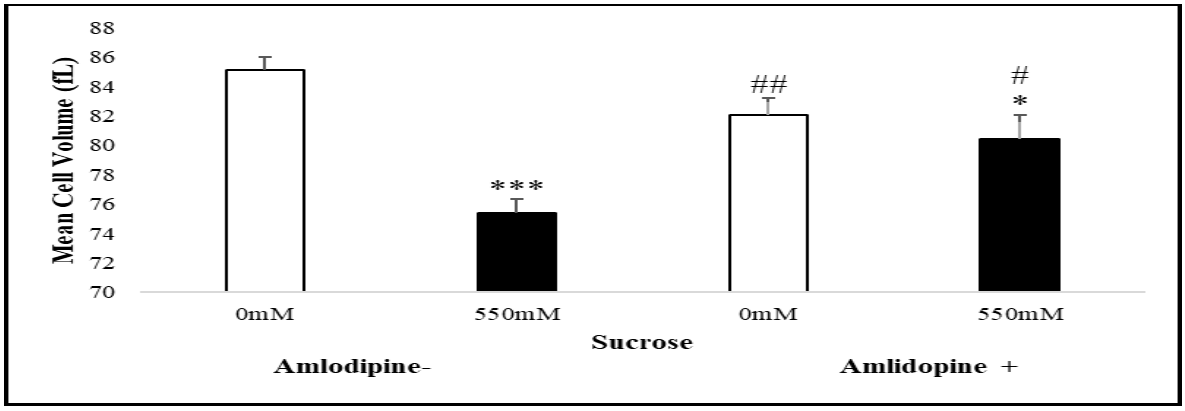

Figure 5: Arithmetic means ± SEM (n = 12) were estimated. Erythrocytes treated with 550mM

Sucrose, and amlodipine and incubated for 24 hours. Graph was plotted between 550mM sucrose (at X-axis) and MCV (at Y-axis). Untreated erythrocytes (0mM) were shown in the white bar and sucrose treated erythrocytes with the black bar. The left side of graph shows sample that are not treated with amlodipine while the right side of graph shows samples treated with amlodipine. The remarkable variation between amlodipine untreated samples was shown by ***(P<0.001), *(P<0.05). The variation between amlodipine treated and untreated samples was shown by ** P<0.01. The results show that cell volume decreases on treatment withs sucrose while there is no significant change in cell volume when treated with sucrose in the presence of amlodipine.

Cytotoxic Assay

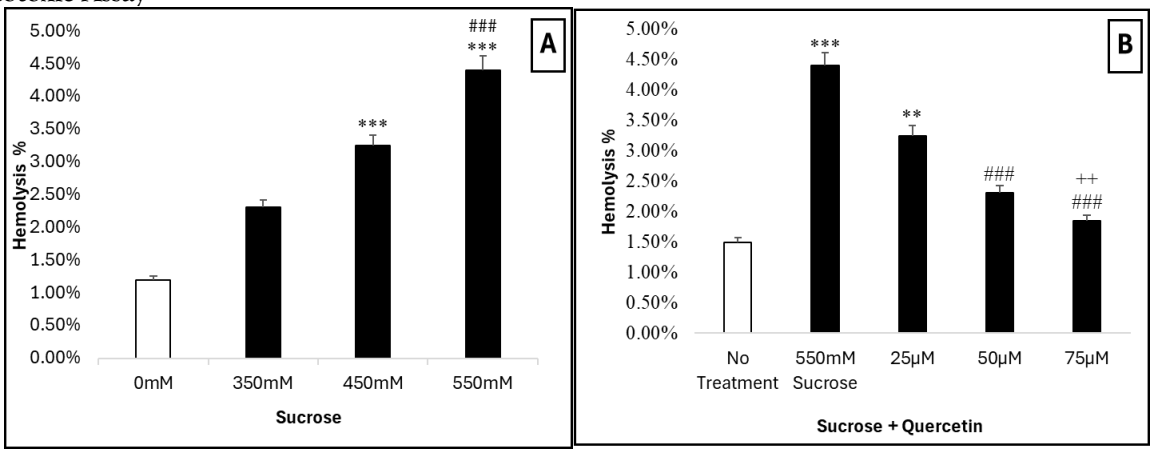

Figure 6: Hemolytic activity in the presence and absence of Quercetin.

Arithmetic means ± SEM (n=12) were estimated. **Figure 6 (A)** shows increase in oxidative stress by increasing sucrose concentration from 0mM to 350mM, 450mM and 550mM. The graph between hemolysis percentage (y-axis) and different sucrose concentrations (x-axis) was plotted. White bar indicates untreated sample/control while the black bars indicate samples treated with different concentrations of sucrose (350mM, 450mM, 550mM). Sucrose causes increase in oxidative stress in erythrocytes which is one of major triggers of eryptosis. The hemolysis percentage of the treated cells was determined. The significant variation in treated and non-treated sample was shown by **($p<0.01$) and $^{\#}$($p<0.05$), where * indicates the significance between non-treated and treated samples while # indicates the significance of treatment B with other treatments. The results showed that hemolysis percentage increases with an increase in sucrose concertation. In **Figure 6 (B)** erythrocytes were treated with quercetin, an antioxidant and anti-inflammatory compound. It will hinder the process of hemolysis as it scavenges free radicals and thus helps in the protection of erythrocytes from oxidative stress. Arithmetic means ±SEM (n=12) were estimated. In fig.B the graph between hemolysis % (y-axis) and different concentrations of quercetin (x- axis) was plotted. White bar shows non-treated sample while black bars indicate samples treated with different therapeutic concentrations of quercetin (25µM, 50µM, and 75µM). Notable variations in quercetin treated and non-treated samples are shown by ***($p<0.001$), *($p<0.05$). The comparison of treatment A with others were showed with significance of $^{\#}$($p<0.05$), $^{\#\#}$($p<0.01$), $^{\#\#\#}$($p<0.001$) and comparison of treatment B with other treatments was shown as $^{+}$($p<0.05$). The results have shown that by increasing the concentration of quercetin, the hemolysis percentage decreased.

Discussion

The proposed research was designed to confirm the antioxidative, anti-hemolytic and anti-eryptotic effects of quercetin on human erythrocytes. Erythrocytes exhibit high percentage of hemolysis when treated, indicating disruption of cellular membrane under hyperosmotic shock and oxidative stress (24). These results confirmed the role of quercetin in mitigating the sucrose stimulated eryptosis. This effect was countered using quercetin in a dose-dependent manner. Therapeutic doses of quercetin (25 µM, 50 µM, 75 µM) were used which have already been reported in literature on nucleated cells (25) quercetin showed best results at 50µM (26, 27) . Quercetin, an antioxidant helps in scavenging free radicals helps in reducing oxidative stress in cellular systems and strengthen the body's natural antioxidant defense system (28). Oxidative stress and hyperosmotic shock are the main triggers of eryptosis (29). Erythrocytes were exposed to sucrose causing hyperosmotic shock (30), forcing the outward flow of water out of the cell resulting in cell shrinkage. This results in destabilization of membrane symmetry causing ionic imbalance specifically via elevated intracellular Ca^{2+} (31). The elevated Ca^{2+} results in activation of phospholipases and proteases that weakens the cytoskeleton. Similarly auto-oxidation of hemoglobin generates reactive oxygen species that accumulate and results in generation of oxidative stress (32) causing lipid peroxidation, oxidation of protein and reduction in deformability. These molecular changes contribute to the induction of eryptosis, the programmed erythrocytes death (33). Hence sucrose-induced hyperosmotic stress not only impairs erythrocyte integrity but also enhance eryptotic pathways, linking osmotic imbalance directly to oxidative damage and premature erythrocytes clearance. The antioxidative effect of quercetin was evaluated by comparing the erythrocytes which were treated with quercetin and not treated with quercetin. Erythrocytes exposed to sucrose showed higher oxidative stress as compared to those treated with therapeutic dose of quercetin (11, 34). This was confirmed by antioxidant enzyme assays. The level of SOD, POD and CAT was significantly declined by reduction in in ROS production. Cell size in erythrocytes is specifically regulated via

specialized channels including Ca^{2+} sensitive channels (35). Results showed a decrease in mean corpuscular volume (MCV) as quercetin prevents the shrinkage of erythrocytes. Role of Ca^{2+} in inducing eryptosis was also explored by the introduction of amlodipine, a Ca^{2+} channel blocker. The results showed a significant decrease in MCV as it showed amlodipine effectively blocked the Ca^{2+} channels (36), these are the channels which become open due to high level of sucrose leading to elevated intracellular Ca^{2+}, one of the hallmarks of eryptosis. Hence quercetin is a highly potent phytochemical having a strong ability to protect erythrocytes from oxidative stress. It can help in reducing the level of ROS and has a cytoprotective role ultimately help in mitigating oxidative stress and prevents cellular damage (11, 37). Membrane integrity, maintenance, biconcave shape and cytoskeletal stability of erythrocytes are maintained by redox homeostasis provided by quercetin. The dual functionality of quercetin i.e., scavenging of free radicals and upregulating the natural antioxidant defense system (SOD, POD, CAT) contributes not only to prolonging erythrocytes lifespan but also mitigates the onset of premature eryptosis, a process implicated in diabetes and various metabolic disorders.

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Informed Consent: Not applicable.

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Author Contribution:
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Marium Ghaffar: Write up and Proof reading

Rumaisa Fatima: Review and Proof reading
Afifah Anwar: Review and Proof reading

Aroosa Komal: Review and Proof reading

Mehwish Fatima: Review and Proof reading

Dr. Kashif Jilani: Supervision, Principal investigator, Proof reading.

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Data availability: Data supporting the findings can be provided upon reasonable request.

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