Acetylcholine esterase (AChE) enzyme is found mainly at neuromuscular junctions and cholinergic brain synapses, where its activity serves to terminate synaptic transmission. During neurotransmission, Ach is released from the nerve to synaptic cleft and binds to Ach receptors and post synaptic membrane, relying from the nerve. Ach E also located on the post synaptic membrane, terminates the signal transmission by hydrolysing Ach. The liberated choline is taken up again by the pre synaptic nerve and Ach E synthesized by combining with Acetyl-Co A through the action of choline acetyle transferase (Purves et al., 2008; Ohanka, 2012).

AChE activity is one of the most frequently used indicators to verify OP effects (Aguiar et al., 2004). The inhibition of ChE activity serves as a reliable biomarker both of exposure and effect of Ops (Chambers et al., 2002; Vioque-Fernandez et al., 2009). Inhibition of ACh E is responsible for the degradation of acetylcholine will result excessive stimulation of cholinergic nerves. This will result in tremors, convulsions and finally the death of aquatic organism (Baxter and Barker, 1998). Many factors seem to involved in affecting the ACh E activity caused by OPs such as length of exposure period and concentrations (Uncer et al., 2006).

Inhibition of AChE that is responsible for the degradation of acetylcholine will result in excessive stimulation of cholinergic nerves. This will result in tremors, convulsion and finally the death of the aquatic organism (Baxter and Barker, 1998). Several factors seem to be involved in affecting the AChE activity caused be OPs such as length of exposure concentrations (Uncer et al., 2006). Inhibition of AChE, impairs cholinergic nerve impulses and may result in death of organisms (Salles et al., 2006).
Inhibition of AChE was accompanied by an increase in acetylcholine levels (Brzezinski and Ludwicki, 1973). This condition can lead to increase of cateholamines which can affect the activity of enzymes involved in glycogenolysis and glycogen synthesis. Continuous stress may affect the synthesis site of AChE or decrease the levels of excess AChE. Mortality of fish may be due to inhibition of other enzymes, especially those taking part in carbohydrate and protein metabolisms. The inhibitory effect on AChE activity indicates that insecticides might interfere in vital processes like energy metabolism of nerve cells (Ansari et al., 1987). Consequently, inhibition of AChE leads to paralysis and death.

Results

Table-4 and Figure-7, shows the activity of AChE (µM of Ach hydrolised/mg protein/hr) in the different tissues like brain, gill, liver and muscle of C. carpio at different exposure periods of quinolphos toxicity. It was observed that, AChE activities in the tissues were significantly increased at day 1 and significant decrease at day 7 and day 15. Then significantly increase at day 21 and recovered to near control value at day 30.

Discussion

Decrease in AchE activity and increase in Ach levels is due to the inhibition of AchE and consequent accumulation of Ach. Acetyl cholinesterase is an enzyme that modulates the amount of neurotransmitter substance at neuron junctions. The inhibition of AchE and elevation in Ach content may be due to the decreased ionic composition in the liver exposed for 5 and 10 day which is in accordance with the earlier reports, thus supporting the findings of current investigation. Increase in AchE activity and decreased Ach content in 15 day may be due to rapid detoxification of quinalphos as liver is a major site for detoxication, assuming that pesticide
concentration is within the threshold limit. This nominal concentration might be rapidly
detoxified leaving less for inhibitory activities. These results reflect that organisms are adopting
with the sublethal concentration which seems to be strategic and adaptive.

The AChE activity is vital to normal behavior and muscular function in animals and
represents a prime target on which some can exert a detrimental effect. Inhibition of the AChE
activity results in a build up of acetylcholine causing prolonged excitatory postsynaptic potential.
This results in repeated, uncontrolled firing of neurons leading to hyperstimulation of the nerve
or muscle fibres, which leads paralysis, and eventual death.

Elevated levels of acetylcholine were observed in cyprinus carpio exposed to
technical grade cypermethrin (Reddy and Philip, 1994). During monocrotophos poisoning, the
Ach levels were elevated in different brain regions of albino rat (Venkataswamy, 1991). The
elevation of acetylcholine (Ach) was also observed in different tissues of albino mice treated
with monocrotophos and biocide azadirachtin (Sivaiah, 2006). Described that the inhibitory activity
of AChE and in different tissues of albino mice treated with monocrotophos.

The principal biological role of acetyl cholinesterase is believed to be the
termination of impulse transmission by hydrolysis of the neurotransmitter, acetylcholine to acetic
acid and choline (Aidley, 1971; Barnard, 1974; Nachmansohn and Neumann, 1975). Because
organophosphates are highly hydrophobic in nature, they may interact at the hydrophobic
aromatic surface region, thus reducing ACh binding space and leading to the reduction in AChE
activity (Valeeswara rao and Jagannatha rao, 1995).
Alka Gupta et al., (1999) observed that the acetylcholinesterase enzyme activity was decreased in rat brain and blood after quinolphos exposure. The AChE levels were also decreased in different brain regions during imidaclorpid toxicity (Kishandar, 2007).

Synthetic pyrethroids poisoning in rats exhibited altered cholinergic functioning in the form of significant decrease in cholinergic receptor binding and inhibition in acetyl cholinesterase activity (Sinha et al., 2006). The brain possesses a remarkable capacity to regulate and to compensate perturbances in the levels of neurotransmitters (Robinson et al., 1986). This ability of the brain to compensate is also reflected in the recovery of behaviour, before the recovery of the enzyme activity is complete (Bignami et al., 1972; Jovic, 1974). The different areas of brain are known to sub serve different functions and any change in the cholinergic system of these areas is reflected in the behavior.

However at day 30 the inhibitory activity was increased and came nearer to normal level. Thus, the fish *Cyprinus carpio* fairly recovered from the inhibitory activity in the tissues of fish. The concomitant recovery in AChE activity at day 30 might be due to active metabolism of quinolphos which is being removed from the site of action and thus enabling the enzymes to resume unhindered hydrolysis of Ach. Similar reports were also observed by Coppage and Duke (1972) in fish brain exposed to malathion. The above evidences are in support of inhibition of acetylcholinesterase (AChE) during quinolphos toxicity.

Thus the results obtained in the present study shows that the pesticide quinolphos is interfering with the nervous system of the fish *Cyprinus carpio* by inhibiting the enzyme acetylcholinesterase.

**Succinate Dehydrogenase (SDH), Lactate Dehydrogenase (LDH), Pyruvate Activity**
Cells function largely because of the action of enzymes. Life is a dynamic process that involves constant changes in chemical composition. These changes are regulated by catalytic reactions, which are regulated by enzymes. The use of enzymes in the diagnosis of disease is one of the important benefits derived from the intensive research in biochemistry since the 1940's. Enzymes have provided the basis for the field of clinical chemistry. It is, however, only within the recent past few decades that interest in diagnostic enzymology has multiplied. The phenomenon of catalysis makes possible biochemical reactions necessary for all life processes. Catalysis is defined as the acceleration of a chemical reaction by some substance which itself undergoes no permanent chemical change. The catalysts of biochemical reactions are enzymes and are responsible for bringing about almost all of the chemical reactions in living organisms.

The mitochondria contain the biochemical machinery for aerobic cellular respiration, the process by which sugars, fatty acids, and amino acids are broken down to carbon dioxide and water, with some of their chemical energy captured as adenosine triphosphate (ATP). A key series of reactions in cell respiration is the Krebs (citric acid) cycle, a complex pathway involving some nine enzymes and numerous metabolic intermediates. One of the best studied enzymes in the Krebs cycle is succinate dehydrogenase (SDH), which catalyzes the oxidation of succinate to fumarate.

Lactate dehydrogenase transfers hydrogen using NAD$^+$ as hydrogen acceptor thus catalyzing the oxidation of L-lactate to pyruvate. LDH activity is present in all the cells of the body predominantly in cytoplasm of the cell (Raised serum lactate dehydrogenase associated with gangrenous small bowel volvulus: a case report. Indian Journal of Clinical Biochemistry, 2003, 18 (2) 6-7 Uday Kumar, Anand Sharan and Shaheena Kamal).
Several reports available on the effects of pesticides on different fresh water animals indicating noticeable changes in the activities of SDH (Swami et al., 1983; Suneetha, 2012); LDH (Philip, 1984; David, 1995; Hymavathi, 2001; Suneetha, 2012); SDH and LDH (Sastry and Siddiqui, 1982). Recently Suneetha (2012) has reported that a decrease in the SDH activity in the gill, liver and muscle of the fresh water fish *Labeo rohita* after exposing lethal and sub lethal concentration of two pesticides, endosulfan and fenvalerate for 24 hrs and 15 days.

Sambasiva rao et al., (1984) reported that SDH activity was found to be inhibited in the muscle, gill liver of fish *Channa striatus* subjected to commercial and technical grade phenothoiate inferring depressed tissue oxidative metabolism under pesticide exposure. Radhaiah and Jayantha rao (1990) observed in *Tilapia mossambica* decrease in the level of SDH while elevated levels of LDH activity under fenvalerate intoxication. (Sivaprasad rao and Ramana rao,1979), several workers reported that organophosphate insecticides caused ultra structural changes in mitochondria, endoplasmic reticulum etc., and inhibited enzyme of TCA cycle is of prime importance and an alteration or inhibition of oxidative enzymes pose a red threat to the survival of animals during pesticide exposure.

**Results**

The activities of succinate dehydrogenase (SDH) (Table-5 and Figure-8) and lactate dehydrogenase (LDH) (µM formazone/mg protein/hr) (Table-6 and Figure-9), the levels of pyruvate (Table-7 and Figure-10) (mg/gm wet wt) in the gill, liver and muscle of the fish at the respective exposure periods of sub lethal concentrations of quinolphos, besides controls. From the data, it was observed that SDH, LDH and Pyruvate activities initially increased significantly
at day 1 in all tissues, followed by decrease of it at days 7, 15. Then at day 21 significant increased in the activity and finally at day 30 it recovers to nearer to the control values.

Discussion

The edible fresh water fishes constitute one of the major sources of nutritious food for humans. Fish are largely being used for the assessment of the quality of aquatic environment and as such can serve as bioindicators of environmental pollution. Among the aquatic species, the fish are the major targets of toxicants contamination. The biochemical parameters in fish are valid for physiopathological evolution and sensitive for detecting potential adverse effects and relatively early events of pollutants, like pesticide damage (almedia et al., 2012; Matos et al., 2007; Osman et al., 2010).
Sastry and Siddiqui (1982) reported increased in LDH activity of liver and decreased in SDH activity of liver in Channa punctatus exposed to sublethal concentrations of sevin. Ghoshes (1987) reported that there was increased LDH activity of in liver and muscle of Clarias batrachus when exposed to sublethal concentrations of Tara 909, Suquin and Croton 36.

Several reports are available on the decreased in SDH activity with the increase in LDH activity accompanied in the organs of the fresh water animals exposed to pesticide toxicity (Koundinya and Ramamurthy, 1978; Siva Prasad Rao and Ramana Rao, 1979; Dayananda Reddy, 1980; Bhagyalakshmi et al., 1982).

Suneetha, (2012) observed a decreased of SDH activity in the gill, liver and muscle of the fresh water fish Labeo rohita after exposing to lethal and sublethal concentrations of two pesticides like endosulfan and fenovalarate for 24hr and 15 days.

Decrease in SDH activity may be due to damage to the mitochondrial structural integrity as reported by Bergen (1971) and Satya pradad (1963). Devaraj et al., (1993) reported the decreased tissue respiration suggested hypoxic condition which reduced the conversion of succinate to fumarate by inhibiting succinate dehydrogenase activity in all regions of the fish brain.

Stress is an energy demanding process and animal mobilizes energy substrates to cope with stress metabolically(vijayan et al.,1997). Changes in the activity of enzymes like succinate dehydrogenase and lactate dehydrogenase are sensitive to environmental pollutants like pesticides(Devi,2003).
In the present investigation the oxidative enzymes like succinate dehydrogenase showed reduction and elevation in non oxidative enzyme like LDH in their activity in all the osmoregulatory (gill) and non osmoregulatory (liver and muscle) tissues of the fish cyprinus carpio which indicates the oxidative metabolism in the fish exposed to quinolphos toxicity.