VIRAL DISEASES OF LEGUMINOUS CROPS

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Abstract

In this paper viral diseases of leguminours crops and symptomatology is reviewed. Among them Cucumber mosaic, Common mosaic, Peanut Mottle, Cowpea mild mottle, Golden mosaic, Alfalfa mosaic, Sunn-hemp mosaic, Dolichos enation mosaic, Peanut stunt, Dolichos yellow mosaic, Blackgram mottle, Urdbean leaf crinkle, Cowpea severe mosaic, Southern bean mosaic, Bean common mosaic, Mung bean mosaic, Bean yellow mosaic, Tobacco ringspot, Tomato spotted wilt, leaf curl, Necrotic mosaic, Mosaic, and Clover yellow vein mosaic diseases are responsible for loss of leguminous crops. Detection of plant viruses and disease management (management of virus diseases and integrated management) possible through improved resistance and genetic engineering. Some transgenic legumes are resistant to Bean dwarf mosaic virus, Pea seed borne mosaic potyvirus and Alfalfa mosaic viruses.

Introduction

Pulses are considered to be the important source of dietary protein to predominantly vegetarian population of India. However, over the three decades the production remained almost static i.e., between 10 to 14 m tones per annum. In recent years, much emphasis has been directed towards increased cultivation of legume crops. Since, intensive cultivation practices often create new and more severe plant disease problems, it is essential to know the various diseases of these crops and the ways to control them. *Leguminosae* (*Fabaceae*), a large family of dicotyledonous plants, commonly called the pea family, consists of approxt 18000 species. The fruit is typically a pod or legume. Many of the Papilionoideae are important food crops, e.g. *Phaseolus*, *Vicia*, *Pisum sativum* (Pea), *Lens culinaris* (Lentil) and *Arachis hypogea* (Peanut). Others such as *Trifolium* (Clovers) and *Medicago sativa* (Lucerne) are used for forage. Viral diseases are shown to be one of the many factors responsible for loss of leguminous crops **(Table.1)**. Natural infection by Tospovirus of cucurbitaceous and fabaceous vegetable crops in India has been studied by Jain *et al.* (2007). A distinct begomovirus causing dolichos yellow mosaic disease in India has been studied by Maruti *et al.* (2006), Dolichos yellow mosaic virus belongs to a distinct lineage of old world begomoviruses of which biological and molecular properties were described by Maruthi *et al.* (2006). Cloning restriction mapping and phylogenetic relationship of genome components of MYMIV from *Lablab purpureus* has been studied by Singh *et al.* (2005). Yellow mosaic virus infecting soybean in northern India is distinct from the species infecting soybean in southern and western India was studied by Usharani *et al.* (2004). Current status of begomoviruses in the Indian subcontinent has been studied by Narayan Rishi (2004). Development of a specific detection technique for *cowpea golden mosaic virus* was reported by Roy *et al.* (2004). Two newly described begomoviruses of *Macroptilium lathyroides* and common bean was studied by Idris *et al.* (2003). A quantitative method to screen common bean plants for resistance to bean *common mosaic necrosis virus* and genetic

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characterization of their differential reactions among host group (Three common bean cultivars to NL-3K strain of bean *common mosaic necrosis virus*) was studied by Strausbaugh *et al.* (2003). Response of urdbean genotypes to powdery mildews, leaf curl, and yellow mosaic disease was evaluated by Barhate *et al.* (2003) Molecular characterization of the rep (Replication initiator protein) protein encoded by black gram isolate of *Indian mungbean yellow mosaic virus* was studied by Pant *et al.* (2001). A dosage- dependent Allele from bean conferring hypersensitive resistance or spreading vascular necrosis in response to the *Potyvirus bean common mosaic virus* was studied by Collmer *et al.* (2000). Identification of variants of *mungbean yellow mosaic virus* by host reaction through nucleic acid spot hybridization was studied by Biswas and varma (2000) and sources of resistance to *bean common mosaic virus* in French bean was studied by Dhar and Gurha (1998).

Symptomatology

Bean yellow mosaic virus (BYMV), a member of the potyvirus group is a flexuous rod, about 750 x 12 nm. Its dilution end point is 10^{-3} to 10^{-4} and the thermal inactivation point varies between 50 and 62 °c depending on the strain. Longevity in vitro varies from 1 to 4 days at room temperature. BYMV and its diseases in Soyabean are found through out Asia, Brazil, and in certain areas of the USA and the former USSR. Early symptoms include vein clearing along the small, branching veins of young leaves. Later, a conspicuous yellowing mottling of the entire leaf develops. Rusty, necrotic spots appear in the yellow areas as the leaves mature. Some strains produce severe mottling and crinkling of the leaves. *Soyabean mosaic virus* (SbMV) and at least 14 closely related potyviruses are reported from soybean (Jain *et al.,* 1992, Qusus *et al.,* 1995). Symptom severity depends on host genotype, virus strain, plant age and environmental conditions. Seedlings arising from infected seeds are spindly, with rugose or crinkled unifoliolate leaves, which may be mottled or curl longitudinally downward. Subsequent leaflets are chlorotic, severely stunted, mottled and rugose. Plants infected early in the season are stunted, have shortened petioles and internodes and often show browning of stems and petioles. Leaves are reduced in size; the youngest show the most severe symptoms (Pacumbaba, 1995). Typically, infected plants mature conspicuously later than uninfected ones and remain green while most other plants have become defoliated and dried.

Bean golden mosaic is caused by *bean golden mosaic virus* (BGMV) belonging to Geminivirus group (Goodman and Bird, 1978). The virion was first purified by Galvez and Castano (1976) and the disease was first reported from southern Brazil in 1961. BGMV has been recorded widely from Mexico, Central America, the Caribbean, Venezuela, Colombia and Argentina (Galvez and Morales, 1989b, and CIAT, 1990). Bean golden mosaic is known variously as bean yellow mottle, bean golden yellow mosaic, bean double- yellow mosaic and 'mosaico dorado' (Galvez and Morales, 1989b). Under dense vector population susceptible genotypes of *P. vulgaris* develop a brilliant golden yellow coloration, starting in the veins of the first trifoliolate leaves within two weeks of sowing. Following exposure to viruliferous whiteflies (*Bemisia tabaci* Genn.), small yellow dots appear near leaf veins about four days later. Young leaves of diseased plants usually become rolled and cupped. Severely affected plants become stunted with bleached leaves, pods often exhibit blotching and seed may be discolored as well as reduced in size and number. Less susceptible cultivars develop less intense symptoms, with a tendency toward remission (Galvez and Morales,

1989b).

Blackeye cowpea mosaic virus (BICMV) and *cowpea aphid borne mosaic virus* (CABMV) are two potyviruses that are pathogenic to cowpea. BICMV was first reported in the USA by Anderson (1955) and CAMV was reported a decade later from Europe and Africa (Bock and conti, 1974). Other potyvirus including pea nut mottle (Demski *et al.,* 1983), cowpea rugose mosaic, cowpea green vein banding and *cowpea severe mottle viruses* have also been reported from naturally infected cowpeas (dos Santos *et al.,* 1981). CAMV is a closely related but distinct virus within the BCMV subgroup of potyviruses (Khan *et al.,* 1993; Mink *et al.,* 1994). Natural infection of cowpea with BICMV or CAMV produces various symptoms such as mottling, interveinal chlorosis, green vein banding, leaf distortion, blistering and stunting of plant (Bock and Conti, 1974). Alfalfa mosaic is caused by *alfalfa mosaic virus* (AMV) and is classified under alfamo virus group. Virus particles are bacilliform with three different lengths; they are readily transmitted both by sap inoculation and nonpersistently by aphids to a wide range of host plants (Bos and Jaspars, 1971). AMV has been noted by Hampton *et al.* (1978) as causing malformation of leaves and mosaic of both red and white clovers. Akita (1981b) described symptoms on red clover as yellow mosaic and leaf wrinkling, while some symptomless plants had a latent infection. Fletcher (1983) reported that leaves of infected subterranean clover plants in New Zealand were smaller than normal and displayed vein- banding and interveinal yellowing.

Bean leaf roll virus (BLRV) is a member of the large group of yellowing viruses, the luteoviruses. The luteovirus group appears to comprise a continuum of serologically related viruses (Waterhouse *et al.,* 1988) and other members of this group, which have been reported to cause yield losses in faba bean, include subterranean clover red leaf virus (SCRLV) in New Zealand (Wilson and Close, 1973) and Australia (Johnstone, 1978), beet western yellows virus (BWYV) in the USA (Duffus, 1964) and chickpea stunt virus (CpSv). BLRV on faba bean produces symptoms of upward leaf rolling and thickening, accompanied by interveinal chlorotic yellowing (Cockbain, 1983). Early infection can suppress flowering and pod set.

Pea enation mosaic virus (PEMV) is the only member in its group and shares no known serological relationship with any other plant virus. The infectious genome is packaged in two isometric spherical nucleoprotein particles. The coat protein molecular weight is 21 KDa. The genome is composed of two ssRNA species consisting of 570 nucleotides (RNA1) and 4253 nucleotides (RNA2) (Demler and de Zoeten, 1994). A third small RNA (RNA3) is occasionally observed and is considered to be satellite RNA. PEMV was first identified by Osborn (1953) from fababean. Diagnostic symptoms in pea include translucent flecks or 'windows', together with vein – clearing and malformation in leaves and stipules. Plants are usually severely stunted and distorted. Pods are typically deformed severely and produce characteristic out growths or proliferations on its surface. The virus causes death of plants in susceptible cultivars or when plants are infected at an early stage. PsbMV was first discovered in Europe (Musil, 1966). The virus was reported shortly after in the United States (Stevenson and Hagedorn, 1969) and described by Hampton (1969) as 'Pea fizzle top virus. The virus is seed borne in pea, lentil and faba bean. Common symptoms include epinasty or down ward leaf rolling, mild chlorosis, vein clearing, mosaic, a general stunting of the plant, terminal resetting (a result of the reduction in internodal

growth) and deformed pods that fail to set. Mid season pea cultivars typically display more severe resetting symptoms than the early cultivars (Hampton and Baggett, 1970). *Pea streak carlaviruses* (PeSV) virions are slightly flexuous rod –shaped. Examination of purified virions of the PeSV – Walla Walla strain however, revealed three distinct particle sizes of 640 nm, 140nm and 95 nm in length (Larsen *et al.,* 1993). Veerisetty and Brakke (1977) reported that PeSV and alfalfa latent virus (ALV) were two distinct viruses based on coat protein molecular weight and comparative sizes of their RNAs. The capsid protein of PeSV with a molecular weight of 28 KDa encapsidates ss RNA of 8.1 kb as resolved in glyoxal – denaturing gels (Larsen *et al.,* 1993). Pea streak carlavirus (PeSV) was first reported on peas in Virginia by Zaumeyer in 1938 and thereafter in Wisconsin pea fields by Hagedorn and Walker (1949a). Symptoms in peas are characterized by purple to brown necrotic streaks on stems and petioles, brown necrotic lesions on leaves, and wilting of the plant. Symptoms of affected pods include brown necrotic lesions often associated with sunken areas. Several strains of PeSV exist and have been described as PeSV- Walla Walla (Larsen *et al.,* 1993), PeSV – central Ferry (Kaiser *et al.,* 1993) and alfalfa latent virus (Veerisetty and Brakke 1978). Particles of Red clover vein mosaic carlavirus (RCVMV) are slightly flexuous rods (Varma, 1970). encapsidating a single stranded RNA species with a length of 7.05 Kilobases as determined by glyoxal denaturing agarose gels (Larsen *et al.,* 1996b). The virus coat protein has an apparent molecular weight of 32-33.5 KDa (Veerisetty and Brakke, 1977; Larsen *et al.,* 1996b).

RCVMV was first described in red clover by Osborn in 1937. Hagedorn and Walker (1949b) later described the virus in pea as 'Wisconsin pea stunt' by which it is still often referred. Symptoms include marked vein clearing accompanied by a mosaic in pea leaves. Diagnostic symptoms in field infected plants include severe stunting, pronounced shortening of internodes resulting in resetting of leaves, lack of apical dominance and proliferation of axillary buds. Pod formation is severely affected when plants are infected before flower set thus reducing yields, moreover plant death can occur if infected at an early stage of growth (Hagedorn, 1984).

Bean yellow mosaic virus (BYMV) is a member of the potyvirus group. Synonyms include bean virus 2, and pea mosaic (or pea common mosaic) virus (PMV), it infects a wide range of legumes including soybean, fababean, clovers and a number of non- legume species. In general, plants infected with BYMV have considerably reduced growth and a very few survive to produce viable seed. The virus is transmitted through seed and can survive in stored seed upto five years (Gladstones, 1970). Plants infected with BYMV initially show yellow mottling of leaves, followed by the formation of many small leaves near the top of the plant and curling over of the stem into the form of a Shepherd's crook (Gondran *et al.,* 1994).

Broad bean true mosaic (BBTMV) and Broad bean strain (BBSV) viruses are members of the comovirus group. The two viruses are unrelated serologically and ELISA can be used for their identification. Foliar symptoms on faba bean are very similar for both the viruses, with chlorotic mottling in patches on the leaves and some times leaf deformation, although some leaves on infected plant may appear normal (Gibbs *et al.,* 1968). Apical dieback may occur in cooler conditions BBTMV and BBSV have been reported from Europe, North Africa and Asia. Bos *et al.* (1988) regarded BBTMV and BBSV as economically important viruses of faba bean, with frequent seed transmission. Pea early browning, primarily a disease of pea is locally important

in Netherlands and England (Boulton, 1996). *Pea early browning tobravirus* (PEBV) can also infect faba bean. PEBV is almost always symptom less on faba bean (Cockbain *et al.,* 1983) although virus concentration in plants may be high. The broad bean yellow band virus (BBYMV) serotype of PEBV (Russo *et al.,* 1984), however, can produce yellow vein – banding, rings and line patterns on the pods, although symptom less infection with BBYBV can also occur.

Pea seed borne mosaic potyvirus (PSbMV) causes a disease of prime importance in pea but the virus can affect faba beans as well. Three pathotypes (strains) have been reported from pea: P-1, L-1 and P-4, all of these can infect faba bean, but pathotype L-1 is the only one, which can infect lentil (*Lens culinaris*). Foliar symptoms of PSbMV in faba bean are vein- clearing and mosaic, particularly on younger leaves and are quite similar to those of BYMV. Fagbola *et al.,* (1996) noted varying severity of symptoms, including stunting and severe leaf, flower and pod distortion. Sterility mosaic is the most important disease of Pigeon pea in India and Nepal (Reddy *et al.,* 1990b). Recent studies at the Scottish Crops Research Institute (SCRI), Invergowrie, UK, on the similar reversion disease of black currant have detected a virus as the causal agent (A. T. Jones, SCRI, Invergowrie, UK, 1996, Personal Communication). The disease was first reported from Pusa in the state of Bihar, India, more than 65 years ago by Alam (1931) who gave the first detailed description of the disease. In the field, sterility mosaic can be easily identified as patches of bushy, pale green plants without flowers or pods (Reddy *et al.,* 1990 b). The leaves of infected plants are small with light and dark green mosaic. Mosaic symptoms initially appear as vein – clearing on young leaves. Strains of sterility mosaic prevalent in Bihar state of India and in Nepal cause severe internodal shortening of the branches and clustering of leaves which some times become filiform, the disease is transmitted by mite *Aceria cajani*. Cucumber mosaic is caused by *Cucumber mosaic virus* (CMV), which is a member of the cucumovirus group and like BYMV, is transmitted by aphids. According to Jones and McLean (1989) the initial symptoms of aphid transmitted CMV are not unlike those of BYMV. Further, unlike BYMV infected plants, CMV infected plants set and produce seeds but majority of the seedlings arising from infected seed die shortly after emergence or within 6-8 weeks post emergence. Plants that survive are stunted and have down curled leaflets.

Clover yellow vein mosaic is caused by *Clover yellow mosaic virus* (CYVV) of the potyvirus group, it infects several species in the *Leguminosae*, particularly *Trifolium Spp.* Hampton *et al.,* (1978), in a survey, observed mottling or mosaic when CYVV was artificially inoculated into white clover, but none when inoculated on to red clover. White clover mosaic is caused by White clover mosaic virus (WCMV) of the potexvirus group. Symptoms of WCMV on white clover have been described by Gibbs *et al.,* (1966) as chlorotic rings, patches and flecks which later develops into brown necrotic flecking. Carr (1984) described the typical symptoms on white, red, alsike and crimson clovers in Britain as light green striping or flecking of the leaves between the veins, although sometimes the infected white clover remains asymptomatic. Johnstone and McLean (1987) observed that WCMV causes systemic chlorotic mottle and vein- clearing in subterranean clover. Red clover necrotic mosaic is caused by red clover necrotic mosaic virus (RCNMV), which is classified in the dianthovirus group. RCNMV has been identified from USA (Edwardson and Christie, 1986), Canada (Rao and Hiruki, 1985), Czehoslovakia, Poland and Sweden (Musil *et al.,* 1983) and Britain (Prame and Harkness, 1987), where it was first isolated only in 1971. In red

clover, RCNMV causes veinal chlorosis, often followed by severe necrosis and deformation. The plants become weakened, stunted and may even die (Gilmour and Pemberton, 1976; Bowen and Plumb, 1979), however, if it survive, the symptoms tend to fade over the summer months (Carr, 1984). Subterranean clover red leaf is caused by Soyabean dwarf luteovirus (SDV), which is transmitted through aphid. Symptoms on subterranean clover are observed as intense reddening of the leaflets that develops progressively from the leaflet margins (Johnstone and McLean, 1987). These symptoms resemble those attributable to manganese deficiency and indeed the plants have only about half the manganese content as compared to healthy plants.

Detection of Plant Viruses

Diagnosis remains difficult for plant virus diseases. It has long been far from simple to demonstrate the presence of viruses. Viruses cannot be seen with the naked eye nor with hand lens or light microscope. The tools commonly used by plant pathologists for examining diseased plant for plant infecting fungi and bacteria are:

1. Identification of plant species and genotype infected.
2. Study of symptomatology
   a. External
   b. Internal (Inclusion bodies).
3. Study of transmissible nature by Sap inoculation, Transmission by vectors such as insects, mites, nematodes etc. Tissue grafting.
4. Establishment of pure culture and determination of host range of virus (es).
5. Determine physical properties of the virus (es) Longevity in vitro, Dilution end point and Thermal inactivation point.
6. Electron microscopy to determine particle shape and size of virus (es).
7. A – Detection by serological methods (Precipitin test, gel diffusion test, ELISA, Western blotting, Dot immuno binding assay).
   B – Detection by molecular biological methods (PCR based detection, Nucleic acid spot hybridization).
8. Proof of pathogenicity (Koch's postulates) by inoculation with purified virus preparation. Manuals for identifying disease are produced by the international centers of the consultative group on International Agricultural Research (CGIAR), including Centro Internacional de Agricultura Tropical (CIAT), International Center for Agricultural Research in the Dry Areas (ICARDA), International Crops Research Institute for the Semi – Arid Tropics (ICRISAT) and International Institute of Tropical Agriculture (IITA), on common bean, tropical pasture legumes, lentil, faba bean, ground nut, chick pea, pigeon pea and cowpea, these are widely available especially in developing countries.

Management of Viral Diseases

There is no cure for virus diseased plants and viruses cannot usually be eliminated from plants once they infect them. Since, replication of the virus is intimately associated with host metabolism any chemical interference will affect host metabolism and likely be phytotoxic. Some

chemicals, such as benzimidazole compounds (Carbendazim) or cytokinin – like substance may reduce symptom severity or decrease virus multiplication but cannot eliminate virus from infected plants. Viral diseases must be prevented only with proper prophylactic measures. The aim is to opt for indirect virus control by interfering with virus ecology so as to stop or delay the onset of virus incidence and to decrease the rate of progress of viral disease. Control of viral disease is much more difficult than of those caused by other pathogens, because of the complex disease cycle (fig.1), efficient transmission and lack of viricides (Varma, 1993).

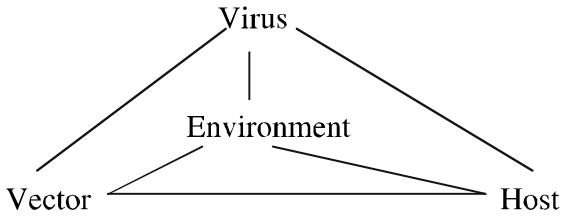

Fig. - 1 Virus disease cycle

The effective management of plant viral diseases demands integration of management practices, such as avoidance of sources of infection, host resistance, cultural practices (such as planting date, rouging infected plants early in the season) and minimal insecticide sprays to control the insect vector, have been effective in reducing virus incidence in legume crops. Measures for avoidance of infection with aphid borne viruses include growing crops at a distance away from known early sources of infection such as clover fields; even so, isolation may not always be effective where the vectors are particularly active (Bos *et al.,* 1988). It is difficult to isolate crops from infection by viruses such as BLRV and pea enation mosaic virus, which persist over long distances in their aphid vectors. It may be possible, especially for seed multiplication to grow crops in areas such as higher ground where the vectors may be less active or arrive at later stages of crop maturity. Sowing earlier in the spring when possible may allow the crop to be more mature when the most damaging period of vector activity occurs. PEBV may be avoided by not growing susceptible crops on land where the virus is known to be present.

Strict statutory seed certification schemes, such as that successfully used for many years in The Netherlands to control the spread of PEBV in pea (Boulton, 1996), and replicase mediate resistance to pea seed – borne mosaic virus (Jones *et al.,* 1998). Transformation of peas to produce agriculturally important insect resistance traits has also been reported (Chrispeels *et al.,* 1998; Charity *et al.,* 1999; Morton *et al.,* 2000). Madappattuparambil *et al.,* (2008) studied transgenic peanut (*Arachis hypogea* L.) plants expressing crylEc and Rice chitinase cDNA (Chi11) exihibit resistance against insect pest *Spodoptera litura* and fungal pathogen *Phaeoisariopsis personata.* Even so, the production and use of seed carrying little or no virus is desirable. Seed lots, or preferable sample plants grown from seed, which better indicate the actual rate of transmission can be screened for infection by ELISA. Those involved with distribution of seed for commercial, experimental or breeding purposes should also ensure that only healthy seed is exchanged or acquired. Production of clean seed is a principal objective in the management of many legume diseases and is often an integral part of disease management strategies. Management of seed borne pathogens of legumes is possibly more important than in other crops including cereals. The best way to avoid seed

borne infection is to identify locations or seasons for production of healthy seed, so avoiding high-risk areas or periods.

Table - 1 Viral Diseases of some important leguminous crops

Disease	Pathogen	Genome	Distribution/ Importance	References
Cucumber mosaic	Cucumber mosaic cucumovirus	ssRNA	USA	Harter (1938), Aderson (1955)
Common mosaic	Bean Common Mosaic Potyvirus	ssRNA	Wide spread	Morales and Bos (1988)
Peanut Mottle	Peanut Mottle Potyvirus	ssRNA	East Africa	Bock *et al.* (1978)
Cowpea mild mottle	Cowpea mild mottle carlavirus	ssRNA	Nigeria	Rossel and Thottappilly (1985)
Golden mosaic	Bean golden mosaic geminivirus	ssDNA	Latin America	Goodman and Bird (1978), Williams (1976)
	Lima bean golden mosaic geminivirus	ssDNA	Nigeria	Vetten and Allen (1983)
Alfalfa mosaic	Alfalfa mosaic alfamovirus	ssRNA	Sudan	Nour and Nour (1962)
Sunn-hemp mosaic, Dolichos enation mosaic	Sunn-hemp mosaic tobamovirus	ssRNA	India	Kassanis and Varma (1975)
Peanut stunt	Peanut stunt cucumovirus	ssRNA	Sudan	Ahmed and Mills (1985)
Dolichos yellow mosaic	Dolichos yellow mosaic geminivirus	ssDNA	India	Capoor and Varma (1950), Harrison *et al.* (1991)
Urdbean leaf crinkle	?	ssRNA	India	Williams *et al.* (1968), Beniwal *et al.* (1980)
Cowpea severe mosaic	Cowpea severe mosaic comovirus	ssRNA	Trinidad	Dale (1949)
Southern bean mosaic	Southern bean mosaic sobemovirus	ssRNA	India	Tremaine and Hamilton (1983)
Alfalfa mosaic	Alfalfa mosaic alfamovirus	ssRNA	Iran, probably widespread	Kaiser (1979), Jaspars and Bos (1980)
Cucumber mosaic	Cucumber mosaic cucumovirus	ssRNA	Widespread?	Purivirojkul and Poehlman (1977)
Bean common mosaic,? Mung bean mosaic	Bean common mosaic potyvirus	ssRNA	Iran and India, probably widespread	Kaiser and Mossahebi (1974)

Continue

Bean yellow mosaic	Bean yellow mosaic potyvirus	ssRNA	Indonesia	Iwaki and Auzay (1978)
Tobacco ringspot	Tobacco ringspot	ssRNA	Sri Lanka	Vignarajah (1978)
	nepovirus			
Tomato spotted wilt, leaf curl	Tomato spotted wilt tospovirus	ssRNA	India	Ghanekar *et al.* (1979)
Cowpea mild mottle	Cowpea mild mottle carlavirus	ssRNA	Tanzania	Mink and Keswani (1987)
Mungbean yellow mosaic	Mungbean yellow mosaic geminivirus	ssDNA	Widespread in South Asia	Honda *et al.* (1983), Harrison *et al.* (1991)
Peanut mottle	Peanut mottle potyvirus	ssRNA	East Africa	Bock *et al.* (1978)
Ringspot	Cucumber mosaic cucumovirus	ssRNA	West Africa, Fiji	Fauquet *et al.* (1979), Brunt and Phillips (1981), Rossel and Thottappilly (1985)
Necrotic mosaic	Cowpea mild mottle carlavirus	ssRNA	Ivory Coast	Fauquet *et al.* (1979), Rossel and Thottappilly (1985)
Mosaic	Cowpea mosaic comovirus, Cowpea severe mosaic comovirus	ssRNA	East Africa Brazil	Kitajima *et al.* (1979), Allen (1983)

Reference. The Pathology of food and pasture Legumes. Edited by D.J. Allen and J.M. Lenne, ICRISAT, International crops Research, CAB International, 1998.London, UK. ? Represents not available

Weeds are very important reservoirs of viruses belonging to groups like cucumo – poty, gemini and tospoviruses, which have a wide host range. Removal of weeds in an around field reduce the incidence of viral diseases. Insecticides are effective in preventing infection and spread with in a crop of luteoviruses such as BLRV since the vector is deterred or killed before virus is transmitted to the host phloem cells. They are less effective when viruses are transmitted in the non – persistent manner. Spread with in the crop of non – persistent viruses in epidemic situations, nevertheless, may be significantly limited by insecticide use. For most viral diseases, resistant lines have been developed by conventional breeding and along with judicious insecticide sprays to control the vector population which helps in management of the disease. Some examples include K-134 in groundnut against bud necrosis virus, for the white fly transmitted geminiviruses like TOLCV, CLCUV, ICMV and yellow mosaic virus in legumes.

In order to avoid development of insecticide resistance among the insect vectors and to reduce insecticide load on population and environment, biological control should be advocated. Unfortunately, biological control of vectors has not received the attention it deserves. Identification of proper and effective bioagent (Parasites and Predators) is a prerequisite for successful execution of biocontrol programme on vectors. The best and simplest approach for avoiding damage by viruses is the use of cultivars with genetic resistance if available, so that the crop becomes less vulnerable to virus. With the advances in the development of regeneration systems of legume

crops (eg. Pea, chickpea fababean, *Medicago* etc.), there is good potential for producing transgenic legumes **(Table 2)** through genetic engineering to reduce losses. Based on five seasons screening

Table - 2 Some examples of Transgenic Legume resistant to virus

Crop/ Virus	Sources of Transgene	Level of resistance	References
Bean dwarf mosaic virus (BDMV)	BV1 or of BDMV	Delayed infection	Yu Ming *et al.,* 2000
Pea seed borne mosaic potyvirus	N1b gene (Viral replicase gene)		Jones *et al.,* 1998
Alfalfa mosaic virus	Coat protein	Partial resistant	Grant *et al.,* 1998

ruder field and artificial conditions cowpea genotypes, Arka Garima, BC-244002, KLS-10, cowpea–263 have been identified as resistant sources to cowpea golden mosaic geminivirus (Chakraborty *et al.,* 1999). Azuki bean has a reproducible and efficient *Agrobacterium*-mediated transformation system (Yamada *et al.,* 2001; El-Shemy *et al.,* 2002). The majority of legume transformation studies have favored the use of *Agrobacterium tumefaciens* to generate transgenic soybeans (Hinchee *et al.,* 1988; Chee *et al.,* 1989), chickpeas (Fontana *et al.,* 1993) and pea (Puonti-Kaerlas *et al.,* 1990, 1992; De Kathen and Jacobsen 1990; Davies *et al.,* 1993; Schroeder *et al.,* 1993; Shade *et al.,* 1994; Zubko *et al.,* 1990). Breeding for resistance to faba bean viruses has been reviewed by Cockbain (1983), Bos *et al.,* (1988) and Makkouk *et al.,* (1993). However, there are few good sources of resistance and breeding programmes are not as advanced in faba bean as in other cool season crop legumes, particularly pea.

Advances in understanding and managing the major diseases of the most economically important crops including soybeans, groundnut and common bean have been relatively rapid. The availability of large germplasm collections and use of multilocational testing have contributed to progress in breeding for resistance. Molecular genetic and plant transformation technologies have made it possible to use novel approaches for developing plants resistant to specific viruses through transfer of alien genes, particularly of viral origin, commonly known as parasite derived resistance (Sanford and Johnson, 1985) includes use of novel viral genes viz. coat protein, replicase, movement, DNA copies of satellite RNAs that reduce symptom expression, defective interfering molecules, ribozymes or antisense RNA. Progress in transformation of large-seeded legumes has been extensively reviewed (Christou, 1997; Nagl *et al.,* 1997; Trick *et al.,* 1997), and more recent progress is presented in **(Table. 3).** There are several approaches that have been used to engineer plants resistant to viruses. The first method aims at introducing genes encoding viral coat protein (coat protein- mediated transgenic resistance to viruses such as TSWV, BPMV, BPMV and PEMV have been obtained in Peanut, Soybean, Bean and Pea crops respectively, The transgenic plants appears to block a receptor in the plant cell that is required for uncoating the viral nucleic acid, or to interfere with viral replication or expression of viral genes. Transformed Pea *(Pisum sativum* L.) lines were produced with two chimeric gene construct encoding the coat protein (CP) of Alfalfa mosaic virus (AMV) strain NZ1, where resistance is manifested by delay in symptom development or escape from infection altogether. A second approach to engineering virus resistance in plant has centered on satellite RNAs. The third approach aims at using antisense virus RNA to govern

Table - 3 Summary of legume transformation systems yielding transformed plants that transmitted the transgenic genotype to progeny reported since or in addition to Atkins and Smith (1997) and Babaoglu *et al.,* (2000).

Species, Genotype	**DNA Delivery**	**Explant**	**Selection**		**Citation**
			Marker	**Agent**	
Red clover (*Trifolium pratense*) NEWRC germplasm	At (EHA101, A208)	Petiole pieces (O)	*nptII*	Kan	Quesenberry *et al.* (1996)
Pigeon pea (*Cajanus cajan* L. Millsp.) N Hyderabad	At (GV2260)	Embryonic axis (O,C)	*nptII*	Kan	Lawrence and Koundal (2001)
	AT (EHA105)	Embryonic axes and cotyledonary nodes (O)	*nptII*	Kan	Satyavathi *et al.* (2003)
Chickpea (*Cicer arietinum*) PG1/PG12/Chafa/Turkey	At (C58C1/ EHA101)	Embryonic axis (O)	*pat*, *nptII*	PPT, Kan	Krishnamurthy *et al.* (2000)
Guar (*Cyamopsis tetragonoloba*) Lewis/ Santa Cruz	At (LBA4404)	Cotyledons (O)	*nptII*	Kan	Joersbo *et al.* (1999)
Jack	EHA105	Immature cotyledon (E)	hpt	Hyg	Yan *et al.* (2000)
BR-16/DokoPC/BR-19/Conquista	MB	Embryonic axis (O)	*ahas*	imazapyr	Aragão *et al.* (2000)
Bert	EHA101	Cotyledonary node (O)	*hph*	Hyg	Olhoft *et al.* (2003)

Continue

Lupin (*Lupinus angustifolius*) Unicrop/Merrit	At (AgL0)	Axillary shoot embryonic	*bar*	PPT	Pigeaire *et al.* (1997)
Lentil (*Lens culinaris* Medik) Laird/CDC599–23	MB	Cotyledonary node (O)	*als*	Chlorsulfuron	Gulati *et al.* (2002)
Bean (*Phaseolus vulgaris*) Olathe/Carioca	MB	Embryonic axes (O)	*bar*	PPT	Aragão *et al.* (2002)
Pea (*Pisum sativum*) 94-A26/ Bolero/Hadlee/ Crown/ Courier/89T46.UK	At (AGL1)	Immature cotyledons (O)	*nptII*	Kan	Grant *et al.* (1998)
Laser, Heiga	At (EHA105; C58C1/LBA4404)	Cotyledons (O)	*nptII, bar*	Kan, PPT	Nadolska-Orczyk and Orczyk (2002)
Mung bean (*Vigna radiata* L. Wilczek) K-851	At (LBA4404)	Cotyledonary node (O)	*nptII*	Kan	Jaiwal *et al.* (2001)
Fava bean (*Vicia faba*) Mythos	At (EHA101 and 105)	Epicotyls (O,C) Internodal stem	*nptII*	Kan	Böttinger *et al.* (2001)
Azuki bean (*Vigna angularis* Willd. Ohwi/Ohashi) Beni-dainagon	At (EHA105)	Elongated epicotyls (O,C)	*nptII*	Kan	Yamada *et al.* (2001)

Genotype, DNA delivery system, explant, selectable marker gene and agent, and citation are presented. N, Not identified; At, A. tumefaciens; Ar, A. rhizogenes; MB, microprojectile bombardment. Agrobacterium strain and tissue culture type: O, organogenesis; E, embryogenesis; and C, callus are indicated in parentheses.

transgenic resistance. Considerable progress has been made towards the successful management of important diseases of most legume crops through the search for host resistance. The durability of resistance against many of the diseases of legumes remains inadequately tested, in part because of the relatively recent development of resistant cultivars and perhaps partly because of the protective effects of the complex cropping systems in which most legumes are grown. There are a number of causes of site differential interactions where one of which is the 'breakdown' of race specific resistance (Allen, 1983). If race non- specific resistance tends to be qualitative trait,

then it may be highly significant as technologies are now available for genetic marking for characters under quantitative control (Edwards, 1992, Dudley, 1993). Recent advances in marker technology have paved the way to a new revolution in our ability to manipulate quantitative traits in crop improvement. Combined disease resistance is required in most legume production systems. This has proved relatively easy to attain in some cases like range of viruses in cowpea, and rosette of groundnut. A case of true multiple resistances conferred by the R3/I gene in common bean has been mentioned. The conserved sequences among genes for disease resistance cloned from widely different plant hosts (Kanazin *et al.,* 1996) seem likely to be useful in identifying evolutionarily related genes in legumes including soyabean. The importance of combined disease resistance in pigeon pea for resource poor farmers can't be overemphasized, recently, the line ICPL 87119, which is resistant to both wilt and sterility mosaic, has been released as 'Asha' (meaning 'hope') for general cultivation in India (Reddy *et al.,* 1990). Pea has been successfully transformed using immature cotyledons (Grant *et al.,* 1995) and a similar method is showing some success in chickpea. However, other legumes such as faba beans are proving to be very difficult to transform, particularly because of the lack of success in regenerating plants in tissue culture. Providing efficient transformation and regeneration systems are developed, the genetic engineering approach will be promising for developing cultivars with resistance to viruses using viral coat protein genes, like the work in progress on several viruses of groundnut (McDonald *et al.,* 1980*).*

Integrated Management

In any integrated program, host plant resistance should be the basic component since it influences other management practices. Management of legume diseases should address the cropping system as a whole, if full advantage is to be taken of available control measures. Other components include the adjustment of sowing date, use of cultivars of different duration, crop rotation, intercropping, cultivation and landform, plant population and spacing patterns. Interactions between different diseases, abiotic stresses such as drought and unfavorable temperatures also must be considered. The economic and socio – economic aspects of integrated management packages should be examined when these packages are being field-tested. Our ultimate aim must be the development of safe, economic and durable management strategies for a range of farm situations, this will probably be achieved only through a combination of measures into an integrated management system including cultural practices, crop and varietal mixtures and in some systems also chemicals, as well as host plant resistance. More recently, investigation of farmers management of common bean diseases in the Great Lakes region of Africa revealed that local strategies were based on microclimate regulation (through sowing density, time of sowing, choice of soil type, foliage reduction, weeding and staking), genetic diversity (use of species and varietal mixtures) and sanitation (seed selection and removal of debris) (Trutmann *et al.*, 1993). In these systems, it was concluded that enhanced disease management should be possible through improved resistance while maintaining variability, but emphasis should be given to technologies, which does not decrease the existing management flexibility. Genetic engineering for plant virus resistance is a rapidly growing approach to develop virus resistant genotypes. The use of virus genes has proven to be a versatile and broadly applicable strategy for achieving resistance and field experiments to date are very promising.

References

Akita, S., (1981b): Bulletin of the National Grassland Research Institute, 20, 93-102.

Alam, M., (1931): Administration report of the Botanical section 1930-31, Department of Agriculture, Bihar and Orissa, India, pp 42-65.

Allen, D.J., (1983): John Wiley & Sons, Chichester, UK,413 pp.

Anderson, C.W., (1955): Plant Disease Reporter, 39, 349-352.

Atkins CA, and Smith P.M.C., (1997): Springer-Verlag, Berlin, pp 283–304.

Babaoglu M, Davey MR, and Power, J.B., (2000): AgBiotechNet 2000, Vol 2, June ABN 050. http://www.agbiotechnet.com/reviews/june00/html/Babaoglu.htm.

Barhate, B.G., Bendre, N.J., Patil, J.V. and Kute, N.S., (2003): Legume Research, 26(2), 140-142.

Biswas, K.K., and Varma, A., (2000): Indian Phytopathology, 53, 134-141.

Bock, K.R. and Conti, M., (1974): CMI /AAB Descriptions of Plant Viruses No. 134, Common wealth Agricultural Bureaux, Farnham Royal, UK.

Bos, L. and Jaspars, E.M.J., (1971): CMI Descriptions of Plant Viruses No. 46, 4 pp.

Bos, L., Hampton, R.O. and Makkouk, K.M., (1988): Kluwer Academic Publishers, Dordrecht, The Netherlands, pp. 591-615.

Boulton, R.E., (1996): Plant Pathology, 45, 13-28.

Carr, A.J.H., (1984): British Crop Protection Council, Corydon, UK, pp. 72-80. Chakraborty, S., Singh, M., Kalloo, G., Banerjee, M.K. and Pandey, P.K., (1999): Annals of Applied Biology 134 Supplement, pp. 40-41.

Charity, J.A., Anderson, M.A., Bittisnich, D.J., Whitecross, M., and Higgins, T.J.V., (1999): Mol. Breed, 5, 357–365.

Chrispeels, M.J., M.F.G. Desa, and T.J.V. Higgins., (1998): Seed Sci. Res, 8, 257–263. Christou P (1997): Field Crop Res., 53, 83–97.

CIAT, (1990): Bean Program Centro Internacional de Agricultura Tropical, Cali, Colombia.

Cockbain, A.J., (1983): The Faba bean (Vicia faba L.) Butterworths, London, UK. Pp. 421-462.

Cockbain, A.J., Woods, R.D. and Calilung, V.C.J., (1983): Annals of Applied Biology, 102, 495-499.

Collmer, C.W., Marston, M. F., Taylor, J.C. and John, Molly, (2000): Molecular Plant- Microbe Interactions, 13(11); 1266-1270.

Davies DR, Hamilton J. and Mullineaux P.M., (1993): Plant Cell Rep., 12, 180–183.

De Kathen, A. and Jacobsen, H.J., (1990): Plant Cell Rep., 9, 276– 279.

Demler, S.A and de Zoeten, G.A., (1994): Webster, R.G. and Granoff, A. (eds) Encyclopedia of Virology Academic Press, San Diego, USA, pp. 1083-1089.

Demski, J.W., Alexander, A.T., Stefani, M.A. and Kuhn, C.W., (1983): Plant Disease, 67, 267- 269.

Dos Santos, A.A., Lin, M.T. and Kitajima, E.W., (1981): Phytopathology, 71, 890 (Abstr.).

Dudley, J.W., (1993): Crop Science, 33, 660-668.

Duffus, J.E., (1964): Phytopathology, 54, 736-738.

Edwards, M., (1992): Field Crops Research, 29, 241-260.

Edwardson, J.R. and Christie, R.G. (eds), (1986): Viruses infecting forage legumes. Vol. 1, Florida Agricultural Experiment Stations Monograph Series No. 14, 1-246.

El-Shemy HA, Khalafalla MM, Wakasa K. and Ishimoto M., (2002): Cell Mol. Biol. Lett., 7, 709-719.

Fagbola, O., Boulton, R.E., Jellis, G.J. and Dhillon, N.P.S., (1996): Plant varieties and seeds, 9, 43-51.

Fletcher, J.D., (1983): Journal of Agricultural Research, 28, 279- 282.

Fontana GS, Santini L, Caretto S, Frugis G. and Mariotti D., (1993): Plant Cell Rep., 12, 194–198.

Frame, J. and Harkness, R.D., (1987): Grass and Forage Science, 42, 213-223.

Galvez, G.E. and Castano M.J., (1976): Turrialba, 26, 205 – 207.

Galvez, G.E. and Morales, F.J., (1989b): Centro Internacional de Agricultura Tropical, Cali, Colombia, pp. 379-406.

Gibbs, A. J., Guissani- Belli, G. and Smith, H.G., (1968): Annals of Applied Biology, 61, 99-107.

Gibbs, A.J., Anupam Varma and Woods, R.D., (1966): Annals of Applied Biology, 58, 231-240.

Gilmour, J. and Pemberton, A. W., (1976): Disease Notice (Plant Pathology Laboratory, Harpenden, UK) No. 16, 1 P.

Gladstones, J.S., (1970): Field crop Abstracts, 23, 123-148.

Gondran, J., Bournoville, R. and Duthion, C., (1994): INRA, Versailles, 48 pp.

Goodman R.M. and Bird, J., (1978): CMI/AAB Descriptions of plant viruses No. 192, Common Wealth Agricultrural Bureaux, Farnham Royal, UK.

Grant, J., Pither, J.M., Fifield, W., Cooper, P. and Timmerman, V.G., (1998): European Association of Grain Legume Research, 372-373.

Grant, J.E. Cooper, P.A., McAra, A.E. and Frew, T.J., (1995): Plant Cell Reports, 15, 254-258.

Hampton, R., Beczner, L., Hagedorn, D., Bos, L., Inouye, T., Barnett, O., Musil, M. and Meiners, J., (1978): Phytopathology, 68, 989-997.

Hampton, R.O. and Baggett, J.R., (1970): Plant Disease reporter, 54, 355-358.

Hampton, R.O., (1969): Phytopathology, 59, 1029.

Hederow, Z.G., (1980): Tagungsberichte der Academics der Landwirtschaftswissenschaften der DDR, Berlin 184, 361-366.

Hinchee MAW, Connor-Ward DV, Newell CA, McDonnell RE, Sa-to SJ, Gasser CS, Fischhoff, DA, Re DB, Fraley, RT. And Horsch, R.B., (1988): Biotechnology, 6, 915–922.

Hogedron, D.J., (ed.) (1984): The American Phytopathological Society, St. Paul, Minnesota, USA, 57 pp.

Idris, A.M., Hiebert, E., Bird, J. and Brown, J.K., (2003): Phytopathology, 93(7); 774-783.

Jain, R.K., Bag, S., Umamaneswaran, K. and Mandal, B., (2007): J. Phytopathology, 155, 22-25.

Jain, R.K., McKern, N.M., Tolin, S.A., Hill, J.H., Barnett, O.W., Tosic, M., Ford, R.E., Beachy, R.N., Yu, M.H., Ward, C.W. and Shukla, D.D., (1992): Phytopathology, 82, 294-299.

Johnstone, G.R., (1978): Australian Journal of Agricultural Research, 29, 1003-1010. Johnstone, G.R. and McLean, G.D., (1987): Annals of Applied Biology, 110, 421-440. Jones, A., L., Johansen, I, E., Bean, S.J., Bach, 1. and Manle, A.J., (1998): J. Gen. Virol., 79, 3129-3137.

Jones, R.A.C. and McLean, G.D., (1989): Annals of Applied Biology, 114, 609-637. Kaiser, W.J., Klein, R.E., Larsen, R.C. and Wyatt, S.D., (1993): Plant Disease, 77, 922-926.

Kanazin, V. Marek, L.F. and Shoemaker, R.C., (1996): Proceedings of National Academy of Sciences, USA 93, 11746-11750.

Khan, J.A., Lohuis, D., Goldbach, R. and Dijkstra, J., (1993): Journal of General Virology, 74, 2243-2249.

Larsen, R.C., Kaiser, W.J. and Wyatt, S.D., (1996b): Plant Disease, 80, 709.

Larsen, R.C., Wyatt, S.D. and Kaiser, W.J., (1993): Phytopathology, 83, 1374 (Abstr.).

Madappattuparambil, Ravindran Beena, Tuli, Rakesh, Gupta, Aparna Dutta, Kirti, Pulugurtha Bharadwaja, (2008): Transgenic Plant Journal, Vol. 2, No. 2, pp.,157-164.

Makkouk, K.M., Bos, L., Horn, N.M. and Srinivasa Rao, B., (1993): John Wiley and Sons, Chichester, UK pp. 179- 192.

Maruthi, M.N., Manjunatha, B., Rekha, A.R., Govindappa, M.R., Colvin, J. and Muniyappa V., (2006): Annals of Applied Biology, 1-9.

Maruthi, M.N., Rekha, A.R., Govindappa, M.R., Colvin, J. and Muniyappa V., (2006): Plant Pathology, 55, 290.

Mc Donald, D. and Raheja, A.K., (1980): Advances in Legume Science, Royal Botanic Gardens, Kew, UK, pp. 501-514.

Mink, G.I., Vetten, H.J., Ward, C.W. Berger, P.H., Morals, F.J., Myers, J.M., Silbernagel, M.J. and Barnett, O.W., (1994): Archives of virology, 139, 231-235.

Morton, R.L., Schroeder, H.E., Bateman, K.S., Chrispeels, M.J. Armstrong, E. and Higgins, T.J.V., (2000): Proc. Acad. Sci., (USA) 97, 3820-3825.

Musil, M., (1966): Biologia (Bratislavia), 21, 133-138.

Musil, M., Micinski, B., Leskova, O. and Pruszynska, M., (1983): Zeszyty Probleowe Postepow Nauk Rolniczych, 291, 227-232.

Nagl, W. Ignacimuthu, S. and Becker, J., (1997): J Plant Physiol., 150, 625–644.

Narayan Rishi, (2004): Indian Phytopathology, 57(4), 396-407.

Osborn, H.T., (1935): Phytopathology, 25, 160-177.

Pacumbaba, R.P., (1995): Plant Disease, 79, 193-195.

Pant, V., Gupta, D., Roychoudhury, N., Malathi, V.G., Varma, A. and Mukherjee, S.K., (2001): Journal of General Virology, 82, 2559-2567.

Powell- Abel, P., Nelson, R.S., De, B., Hoffmann, N., Rogers, S.G., Fraley, R.T. and Beachy, R.N., (1986): Science, 232, 738-743.

Puonti-Kaerlas J, Eriksson T, and Engstr_M.P., (1990): Theor. Appl. Genet, 80, 246– 252.

Puonti-Kaerlas J, Eriksson T, and Engstr_M.P., (1992): Theor. Appl. Genet, 84, 443–450. Qusus, S., Cramer, C.L., Saghaimaroof, M.A. and Tolin, S.A., (1995): Phytopathology, 85, 632.

Rao, A.L.N. and Hiruki, C., (1985): Plant Disease, 69, 959-961.

Reddy, M. V., Sharma, S.B. and Nene, Y.L., (1990b): CAB International, Wallingford, UK, pp. 303-347.

Roy, Anirban and Malathi, V.G., (2004): J. Plant Biochemistry and Biotechnology, 13, 131-133.

Russo, M., Gallitelli, D. Vovlas, C. and Savino, V., (1984): Annals of Applied Biology, 105, 223-230.

Sanford, J.C. and Johnson, S.A., (1985): J. Theor. Biol., 11B, 395-405.

Schroeder HE, Schotz AH, Wardley-Richardson T, Spencer D, and Hig-gins T.J.V., (1993): Plant Physiol., 101, 751–757.

Shade R.E., Schroeder H.E., Pueyo J.J., Tabe L.M., Murdock L.L., Higgins T.J.V. and Chrispeels M.J., (1994): Biotechnology, 12, 793–796.

Singh, Sudheer, Kumar, Chakraborty, S.,Singh, Ashok Kumar, Pandey, P.K., (2005): Bioresource Technology, 1-8.

Steverson, W.R. and Hagedorn, D.J., (1969): Phytopathology, 59, 1051-1052 (Abstr.). Strausbaugh, C. A., Miklas, P.N., Singh, S.P., Myers, J.R., and Forster, R.L., (2003): Phytopathology, 93(6), 683-690.

Strausbaugh, C. A., Myers, J.R., Forster, R.L. and Mcclean, P.E., (2003): Phytopathology, 93(11), 1430-1436.

Tanimura, Y., Matskawa, I. and Banba, H., (1985): Bulletin of Hokkaido Prefectural Agricultural Experiment station, 5, 85-93.

Thurston, H.D., (1992): West View Press, Boulder, Colorado, USA.

Trick HN, Dinkins RD, Santarem ER, Di R, Samoylov V, Meurer C, Walker D, Parrott WA, Finer JJ, and Collins G.B., (1997): Plant issue Cult Biotechnol., 3, 9–26.

Trutmann, P., Voss, J. and Fairhead, J., (1993): International Journal of Pest management, 39, 334-342.

Usharani, K.S., Surendranath, B., Haq., Q.M.R. and Malathi, V.G., (2004): Current Sci., 80(6), 845-850.

Varma, A., (1993): Crop protection and sustainable Agriculture, Wiley, Chichester (Ciba Foundation Symposium 177) p. 140-157.

Veerisetty, V. and Brakke, M.K., (1977): Virology, 83, 226-231.

Veerisetty, V. and Brakke, M.K., (1978): Phytopathology, 68, 59-64.

Vishwa Dhar, and Gurha, S.N., (1998): Indian Jr. of Pulses Research, 11(1), 138-139.

Waterhouse, P.M., Gildow, F.E. and Johnstone, G.R., (1988): CMI/AAB Descriptions of plant viruses No. 339, Commonwealth Agricultural Bureaux, Farnham Royal, UK.

Whitehead, R. (eds), (1996): CAB International, Wallingford, UK.

Williams, R.J., (1976): Plant Disease Reporter, 60, 853-857.

Wilson, J. and Close, R.C., (1973): New Zealand Jr. of Agricultural Research, 16, 305-310.

Yamada T, Teraishi M, Hattori K and Ishimoto M., (2001): Plant Cell Tissue Organ Cult., 64, 47-54.

Yu Ming, H., Sanders, R., Ursin, V.M. and Gilbertson, R.L., (2000): Molecular Plant Microbe Interactions, 13, 297-308.

Zaumeyer, W.J., (1938): Journal of Agricultural Research, 56, 747-772.

Zubko EI, Kuchuk NV, Tumanova LG, Vikonskaya NA. And Gleba Y.Y., (1990): Biopolymers Cells, 3, 80–84.

PLANT-INSECT MUTUALISTIC ASSOCIATIONS AND THEIR EXPLOITATION BY ROBBERS AND CHEATERS

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Abstract

This study reviews existing research findings on the diversity of plant insect mutualistic associations and the use of deceit in the exploitation of such associations. A multitude of plant-insect mutualistic associations, including plant-insect pollinators, ant- myrmecochore mutualisms, digestive mutualism of carnivorous plants with insects and protective mutualism between ants and myrmecophytes are disrupted by the exploiting plant / insect species. While insect robbers / cheaters deprive the plants of resources without providing any beneficial services, many plants use deceit to obtain services from insects without providing them the desired resource, in return. The review reveals that a variety of partner choice mechanisms at the levels of morphology, physiology and behaviour are used by the exploited species to impose host sanctions and to filter out the robbers / cheaters so as to avoid costly association with exploiters and to interact and invest predominantly with the cooperators. It is, therefore, abundantly clear that elucidation of 'cooperation and conflict' between plants and their insect associates is possible only by considering mutualistic associations as part of the complex network involving multispecies guilds and multitrophic interactions.

Keywords

Cooperation, deceit, ant plants, plant-insect pollinators, extrafloral nectar, myrmecophytes, myrmecochory.

Introduction

Plant-insect mutualistic associations (involving cooperative interactions) are widespread in nature. These include i) plant-insect pollinator mutualism (Kremen *et al.*, 2002), ii) mutualism between seed-dispersing ants and myrmecochores (ant-dispersed plants), the seeds of which are dispersal by ants (myrmecochory) (Beattie, 1985), iii) digestive mutualism between carnivorous plants and insects (Anderson and Midgley, 2002), iv) and, protective mutualism between ants and myrmecophytes (plants which offer shelter in the form of domatia and food in the form of Beltian bodies and/or extrafloral nectar (EFN), to the ants) (Janzen, 1966; Heil and McKey, 2003). While bees (also wasps, butterflies, flies and beetles) are significantly implicated in pollination, ants play the dominant role in seed dispersal and myrmecophyte protection. However, the mutualistic associations are frequently riddled by instances of conflicts between the two partners which resultantly shift the nature of the association from mutualistic to exploitative (Doebeli and Knowlton, 1998; Bronstein, 2001; Sachs and Simms, 2006; Douglas, 2008). Thus, many insects exploit plants by utilising food (pollen/nectar/Beltian bodies) and /or domatia (as shelter) and not providing pollination / seed dispersal / herbivore protection service(s) to the host plant, in return. At the same time a variety of plants are documented to use deceit in order to avail the

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services of insects. There are also instances of exploitation of plants by one of the interacting insect partners which changes the trophic behaviour of the insect mutualist.

The present review aims at providing a brief synthesis of recent information on mutualistic plant-insect associations and the prevalence of deceit in such associations. At the same time, the mechanisms used by the exploited partner to reduce exploitation and to increase cooperation are highlighted to give an insight in the 'cooperation and conflict' involved in such associations. A cheater is an individual of a partner species that receives the benefit of mutualism but does not reciprocate (Mainero and del Rio, 1988). Hereafter, the term cheater would be used for an exploiter irrespective of whether it has/does not have mutualistic ancestors since phylogenetic information is not available for a large majority of exploiting species.

Plant-insect pollinator mutualism

More than 75% of crop species depend on pollination by animals (Kremen *et al.*, 2002; Klein *et al.*, 2007). Animal pollinators include birds, bats and insects. Insect pollination is known as entomophily (Gullan and Cranston, 2004). Pollinating insects obtain nectar and/or pollen, or may utilise the flowers for laying eggs. They thus obtain nutritive or reproductive benefits while aiding sexual reproduction of the flowering plants. Most pollinators visit the flowers for obtaining nectar and pollen. However, in some cases the objective of the floral visit is mating and oviposition, the most well known being *Ficus* (Moraceae) (Janzen, 1979) and *Yucca* (Agavaceae) (Baker, 1986).

Anthophilous (flower-visiting) taxa include bees, wasps and ants (Hymenoptera), butterflies and moths (Lepidoptera), flies (Diptera) beetles (Coleoptera) and thrips (Thysanoptera) (Gullan and Cranston, 2004).

Hymenopteran insects

Melittophily (bee pollination) is well known. Honey bees especially *Apis mellifera* is an important pollinator of crop worldwide though stingless bees (*Melipona* spp.), bumble bees (*Bombus* spp.) and many solitary bees also play significant role in crop pollination (Klein *et al.,* 2009). Pollination by wasps (sphecophily) is reported in some plants such as those belonging to the genus *Eucomis* (Hyacinthaceae) and African milkweed *Xysmalobium orbiculare* which are specialized for pollinated by pompilid wasps (Shuttleworth and Johnson, 2008). Mutualism between figs (*Ficus* spp.) and fig wasps (Chlocloids: Agaonideae) is a classical example of species-specific plant-pollinator association. Each species of fig is associated with a unique species of fig wasp. Figs and their wasps depend upon each other to reproduce. Fig inflorescence is in the form of an urn-shaped receptacle, the syconium (which has tiny flowers inside). It has a small hole through which fig wasps enter. Inside the syconium the wasps lay their eggs in the fig's ovules. Thus, each maturing larva galls and feeds on the developing seeds. The larvae destroy the developing fig seeds but when the adult wasps finally emerge from the fig, they pick up pollen and take it to another tree.

Ants are reported as pollinators only in some cases (Peakall and Beattie, 1989; Peakall *et al.,* 1991; Bosch *et al.,* 1997; Gómez, 2000; Schürch *et al.,* 2000). It is suggested that to minimize the interference caused by ants to insect pollinators (Ghazoul, 2001) and/or to avoid the negative impact of the extrafloral nectary visiting ants on pollen function (Beattie *et al.*, 1985) insect-pollinated plants have evolved various strategies to avoid ant visits to the flowers. Ants are,

however, found to be effective pollinators of some orchids (Peakall, 1989; Sugiura *et al.,* 2006). A guild of ant species are also recorded as dominant pollinators of *Cytinus hypocistis* (Cytinaceae), a Mediterranean root holoparasite (de Vega *et al.,* 2009).

Non-hymenopteran insect pollinators

Lepidopteran pollinators

Butterflies are diurnal and use vision to locate brightly coloured but odourless flowers while moth being nocturnal use odour (usually sweet) to find the flowers which are usually white or pale-coloured. While butterfly pollination (psychophily) occurs in flowers exhibiting diurnal anthesis, moth pollination (phalaenophily) occurs in nocturnal anthesis flowers. Butterflies pollinated flowers are usually arranged in clusters and provide a landing platform (such as those belonging to compositae). Moths are hover-feeders so moth pollinated flowers are characterized by deep tubular structures. Some plants such as the *Lantana camara* ((Schemske, 1976) and wild carnation, *Dianthus carthusianorum* (Bloch *et al.,* 2006) are predominantly pollinated by diurnal Lepidoptera. The cabbage white butterfly (*Pieris rapae*), notorious as a pest of cabbage also happens to be a pollinator of many wild and cultivated plants (Conner *et al.,* 1995). The Karner blue butterfly, *Lycaeides melissa samuelis*, the larvae of which feed only on wild lupine leaves is endangered pollinator. The monarch butterfly, *Danaus plexippus* is a pollinator of common milkweed, *Asclepias syriaca.* Obligate pollination mutualism between yuccas and yucca moths is extensively documented (Pellmyr and Leebens-Mack, 1999; Pellmyr and Leebens-Mack, 2000). Yuccas are pollinated exclusively by yucca moths, whose larvae in turn consume some of the developing yucca seeds. Female yucca moths possess unique tentacular mouthparts that are used to actively pollinate host flowers where they oviposit (Pellmyr and Krenn, 2002). The sticky pollen is scrapped off the anthers and then compacted with the help of tentacles and occasionally the forelegs legs. It is then placed on the concave posterioventral surface of the head. Next, the moth searches flowering yucca plants where she oviposits into or near the pistel. After oviposition she uses the apical portion of the tentacles to transfer a small portion of pollen load on to the floral stigma.

Dipteran and coleopteran pollinators

Myophily (fly pollination) is exhibited by large flies belonging to the families Bombyliidae, Syrphidae, Anthomyiidae, Tachinidae, Calliphoridae, and Muscidae as well as small nematoceran fly pollinators (Kevan and Baker, 1983). Flies are documented as regular visitors and pollinators of a variety of flowering plants including mango, tea, cashew, cacao and onions (Kearns, 2001 and references therein). *Aristolochia*, the most diverse genus of the family Aristolochiaceae is found to be pollinated exclusively by flies belonging to at least five different families.

Beetle pollination or cantharophily is reported in the *Magnoliaceae, Annonaceae, Eupomatiaceae* and *Calycanthaceae* (Gottsberger, 1989). Bimodal pollination by wasps and beetles has been reported in African milkweed *Xysmalobium undulatum* (Shuttleworth and Johnson, 2008). Thrips which feed on nectar and pollen were earlier regarded as minor pollinators but are now documented to be important pollinators of a number of flowering plants (Moog *et al.,* 2002; Williams *et al.,* 2001).

Nectar and/pollen robbing insects and adaptive strategies by plants to prevent robbing

Nectar robbers deprive the plants of nectar without playing a role in pollination. Nectar robbery has been defined as the consumption of nectar from flowers without contacting the sexual parts of the flower by physically damaging the flower in a way that is not done by legitimate pollinators (Inouye, 1983). Floral robbers are commonly viewed as acting as plant antagonists. They have been placed into three categories on the basis of the resource robbed and occurrence /absence of floral damage caused by the robbing insect (Barrows *et al.,* 1976). These include: i) nectar-foraging which restricts the robbing insects' visits to individual flowers thereby reducing outcrossing (Heinrich and Raven, 1972), ii) nectar-foraging-perforating (which bites holes in corollas through which nectar is imbibed, though it does not necessarily restrict the visits to individual flowers, and iii) pollen-foraging, where the insect robber collects pollen and, restricts its visits to individual flowers (Barrows *et al.,* 1976). Nectar-foraging-perforating robbers not only deplete nectar from flowers but also facilitate theft by secondary robbers which use the holes made by the former to access nectar (Hawkins, 1961). Some short-tongued bumblebees utilise the holes left by the hole-biting *Bombus terrestris* for nectar collection (Stout *et al.,* 2000).

Nectar robbers include the stingless bee, *Trigona* (*Trigona*) *fulviventris* (Barrows, 1976) bumble bees, *Bombus* spp. (Inouye, 1983; Zhang *et al.,* 2009) carpenter bees, *Xylocopa* spp. (Dedej and Delaplane, 2004) and ants (Norment, 1988.). Nectar robbing reduces the nectar standing crop and also diminishes the visits by legitimate pollinators. Primary robbers can cause even legitimate pollinators such as honey bees to access nectar by making visits through holes such as those made by *Xylocopa* spp. and thus act as secondary nectar thieves (Delaplane and Mayer, 2000). Nectar robbing may cause floral changes. The sugar concentration of nectar available to other pollinators also changes in some cases (Pleasants, 1983). Other pollinators including humming birds are known to avoid nectar robbed plants (Irwin, 2000).

Plants have evolved a wide variety of strategies to avoid being robbed. These include physical mechanisms in the form of longer corolla tubes (Lara and Ornelas, 2001), thicker corolla tissue (Inouye, 1983) and floral nectar barriers (Coombs and Peter, 2009). Chemical mechanisms include production of floral repellents (Agarwal and Rastogi, 2008a) including volatile scents (Junker and Blüthgen, 2008) and nectar unpalatable to potential robbers (Shuttleworth and Johnson, 2009a). One hypothesis put forward is that plants may be selected to provide their nectar reward in many small flowers to keep away large nectar robbers (Pettersson, 1999). In this way the plants may discourage visits by large hovering foragers since it may be too costly for them to collect the scattered nectar. Moreover, the plant can direct this energy in the production of more number of flowers.

Plant resource exploiting insects and adaptive strategies of the host plants

There is a possibility of cheating by the fig wasps by using too many ovules. To prevent seed production from ceasing and thereby causing extinction of mutualism, fig trees must prevent

the wasps from ovipositing in all flowers. Fig trees use a number of strategies to prevent wasps from ovipositing in all flowers and reduce galling. In the hot summer, wasps have shorter life spans and therefore, fail to oviposit in many flowers. During winter although the longer lifespans permit the wasps to exploit more number of flowers interference competition among the competing wasp foundresses reduces the proportion of flowers that are galled. It is found that the fig trees encourage the entry of multiple foundresses by delaying ostiole closure of the syconia during the low temperature conditions of winter (Wang *et al.,* 2009).

Cheating by wasps is also prevented since there is another player in this game in the form of parasitic wasps which lay eggs in the outer ovules. To escape being parasitized fig wasps only use the seeds closest to the center of the fruit. Thus the parasitic wasps help to prevent cheating by the fig wasps and ensure cooperation between the pollinating wasps and the fig plants.

Deceitful plants

Many orchids exploit existing plant-pollinator relationships and use deception to accomplish pollination. Most common mechanisms include: i) 'generalized food deception' (Steiner, 1998; Brodmann *et al.,* 2008), ii) Batesian floral mimicry, where the flowers resemble rewarding plant species (Johnson *et al.,* 2003), or even mimics pollinating insects (Schiestl, 2005) iii) sexual deceit, where the flowers mimic female insect mating signals, especially their pheromones (Ayasse *et al.,* 2000; Schiestl *et al.,* 2003) and or shape and colour patterns (Paxton and Tengö 2001) and are pollinated by the lured male insects, which often try to copulate with the flower. The flowers of orchid *Ophrys sphegodes* 'double dupes' the males of the bee, *Andrena nigroaenea.* After attracting them by production of odour similar to that produced by the female sex pheromones the pollinated flowers repel the pseudocopulating males by production of chemical compounds similar to those produced by impregnated females (Paxton and Tengö 2001). The orchid *Leporella fimbriata* Lindle, exploits the winged male *Myrmecia urens* Lowne ants, which are observed to pseudocopulate with the flowers (Peakall, 1989). In a rare strategy, the deceptive orchid, *Disa nivea* successfully exploits a reciprocally specialized mutualism between a nectar-producing plant (*Zaluzianskya microsiphon*) and its long-proboscid fly pollinator (*Prosoeca ganglbaueri*). *Disa nivea* is a rare southern African orchid known only from habitats that support large populations of *Z. microsiphon*, which the exploiting species closely resembles in both general morphology and floral spectral reflectance. The orchids *Epipactis helleborine* and *E. purpurata* show chemical mimicry to attract wasp pollinators (Brodmann *et al.,* 2008). They emit green-leaf volatiles which are attractive to prey-hunting foragers of the social wasps *Vespula germanica* and *V. vulgaris.*

The perianth of the zygomorphic flowers of *Aristolochia* has three sepals which together form a tubular calyx. The basal part of this forms a chamber (utricle) around the fused styles, stigmas, and anthers. The utricle is connected to a tube which ends as an expanded limb, which is often colorful and presumably attracts the pollinators (Brantjes, 1980). The flies being saprophagus the plants apparently use deceit to lure the flies for pollination (Endress, 1994; Vogel, 1990)

though in a few species the flies oviposit and develop on the decomposing flowers (Sakai, 2002). Immediately after opening the flowers use odour to attract the flies and trap them inside the utricle. On the second day, the anthers dehisce and the flies are released with the pollen load.

Ant-plant mutualistic interactions

Seed dispersal mutualisms

About 70 plant families are known to have ant-dispersed (myrmecochorous) seeds (Beattie, 1985). The seeds of myrmecophores bear a lipid-rich appendage known as the elaiosome. Since the fatty acid composition of elaiosomes is similar to that of insect hemolymph (Hughes *et al.,* 1994), it is attractive to ants. Thus the elaiosome is suggested to function as a dead-insect analogue. Ants and the myrmecochorous seeds have a reciprocal association. The ants benefit by getting a lipid-rich reward resource while the plant propagule gets dispersed in refuse heaps or in nest chambers (Handel, 1976). Additionally the seeds get a nutrient-rich microsite for germination.

Although it is often generalized mutualism recent studies indicate the occurrence of keystone ant mutualists (Ness *et al.,* 2009). It was demonstrated that *Aphaenogaster* ants (predominantly *A. rudis*) collect about 74 % of the myrmecochorous seeds in eastern North American forests.

Seed predators/ robbers

Ants which remove seeds but destroy them act as seed predators since they kill the potential plants. Recent studies demonstrate that the red imported fire ant, *Solenopsis invicta*, is attracted to and destroys many ant-dispersed seeds (Zettler *et al.,* 2001). The ants consume elaiosomes and either scarify or destroy the seeds thereby preventing seed dispersal by native ant species. Thus they act as seed robbers.

Digestive mutualistic insect-carnivorous plant associations

Carnivorous plant, *Roridula gorgonias* uses sticky traps to capture insect but lacks digestive enzymes for the digestion. It has digestive mutualism with hemipterans, *Pameridea* sp. (Hemiptera: Miridae). The hemipteran feeds on the captured prey and then defecates on the leaves of the carnivorous plant which absorbs the nitrogen directly from the leaves. Hemipterans are also the major pollinators of *Roridula* and are responsible for up to 65% of seed set in *R. dentata* (unpublished data as given in Anderson and Midgley, 2002). The combination of nutritional and pollination benefits of this mutualism indicates that hemipterans may be crucial for plant survival. Pitcher plant, *Nepenthes* suffers from putrifaction on capturing excess prey, but the specialist ants living on theses plants reduce the incidence of putrification by upto 12 fold by removing excess prey and thus are regarded as carnivorous plant mutualists (Clarke and Kitching, 1995).

Cheaters involved in digestive mutualistic associations

Many insects act as cheaters by taking the reward offered by the plants but not providing digestive services to the carnivorous plants. Thus the cheaters exploit the mutualistic association. *Roridula dentata* is associated with a unique species of hemipteran (*Pameridea marlothi*) (Anderson

and Midgley, 2002). Here, a specialist spider (*Synaema marlothi* Dahl., Thomsidae) which is also associated exclusively with *R. dentate* acts as a cheater. It reportedly decreases the numbers of hemipteran individuals inhabiting *Roridula* plants and also decreases efficiency of indirect prey digestion by up to 30%.

Ant-plant protective mutualism

Many plants are involved in 'biological warfare' since they attract and reward ants (by providing food and/or shelter) to deter or kill insect herbivores (reviewed by Bronstein *et al.*, 2006). A large number of studies have documented the obligate associations of ants with myrmecophytes (Janzen, 1966; Beattie, 1989; Bronstein, 1998; Heil and McKey, 2003). Ants provide protection against herbivores and in some cases from competition with other plants (Hölldobler and Wilson, 1995 and references therein). Ants are also involved in facultative protective associations with annual (Agarwal and Rastogi, 2008b, 2009) and perennial, extrafloral nectary-bearing plants (Gaume *et al.,* 1997).

Ants as exploiters and adaptive strategies of the ant-plants to avoid exploitation

While the mutualistic ant, *Pseudomyrmex ferrugineus* protects the Acacia, the myrmecophytes is exploited by the ant, *Pseudomyrmex gracilis* which colonises the plant thus preventing the colonization of the mutualist (Clement *et al.,* 2008). The exploiting ant species not only has adverse effect on the growth of the myrmecophyte but also does not defend it. Ants belonging to genus *Allomerus* though providing protection from insect herbivores to the host plants are also castration parasites since they destroy flowers on the branches they inhabit (Yu, 2001; Izzo and Vasconcelos, 2002). However, its host plant, *Hirtella myrmecophila* (Chrysobalanaceae) drops domatia from older leaves, to minimize the effects of cheating by *A. octoarticulatus* (Izzo and Vasconcelos, 2002).

Acacia ant-plants defended by the symbiotic *Pseudomyrmex* ant mutualists secrete sucrose-free extrafloral nectar which excludes exploiters (Kautz, 2009). Experiments reveal that the mutualist workers avoid sucrose due to the absence of the sucrose-cleaving enzyme invertase while the exploiters and generalists with no particular affinity toward *Acacia* myrmecophytes prefer sucrose. Thus *Acacia* plants are specialized with respect to their ant partners and the digestive specializations prevent mutualists from becoming cheaters. The extrafloral nectary-bearing sponge gourd plants, *Luffa cylindrica,* involved in facultative association with a guild of liquid sugar loving aggressive ants species prevents the exploitation of floral nectar (and also deterrence of pollinators) by means of floral repellents (Agarwal and Rastogi, 2008a).

In the Ant-acacia mutualism, *Acacia mayana* while guarded by the ant *Pseudomyrmex ferrugineus* also harbours the generalist ant, *Camponotus planatus*. *C. planatus* appears to be a parasite of the *Acacia–Pseudomyrmex* mutualism since it occupies the swollen thorns on the host tree, and harvests nectar from extrafloral leaf nectaries but does not protect it from insect herbivores. However *C. planatus* is unable to harvest the second trophic reward in the form of protein-rich food (Beltian) bodies produced by the tree for its *Pseudomyrmex* ant-guards (Raine *et al.,* 2004). The specialised

larval adaptations needed to use Beltian bodies as brood food, is absent in the parasitic ant species. Thus this resource is apparently more resistant to exploitation by generalists than extrafloral nectar.

Conclusion

Plant-insect mutualistic associations generally regarded as pair-wise associations are actually rather complex involving other species which influence the associations. Thus these associations should be regarded as part of the complex network involving multispecies guilds and multitrophic interactions (Stanton, 2003). Attempts are being made to elucidate the ecological and evolutionary dynamics of mutualisms in the context of exploitation (Jones *et al.,* 2009). Recent studies focus on how mutualists use various mechanisms to target rewards preferentially at mutualists (Pellmyr and Huth, 1994; West *et al.,* 2002; Edwards *et al.,* 2006; Bever *et al.,* 2009). Extra-floral and floral nectar composition serves to attract and fulfill the physiological requirements of mutualists while deterring and protecting the resource from robbers and exploiters (González-Teuber and Heil, 2009). In *Acacia–Pseudomyrmex* system a study of 23 *A. cornigera* and 24 *A. hindsii* plants (all of which were inhabited by *P. ferrugineus*) showed that high reward hosts produce significantly more extrafloral nectar, food bodies, and nesting space than low-reward hosts (Heil *et al.,* 2009). Moreover, exploiter ants were recorded to be less dependent on the host-derived rewards and were able to colonize many of the low-reward hosts.

Finally it must be pointed that the evolution of cooperation in fact is itself a puzzle which poses a challenge to evolutionary biologists since natural selection should favour cheaters who can receive benefits without reciprocating the services (Foster and Wenseleers, 2006). Partner choice mechanisms are fundamental to the evolution of cooperation since these permit individuals to invest in cooperating partners and avoid cheaters (Edwards, 2009). A variety of partner choice mechanisms have been implicated in the evolution of mutualism. These include i) host sanctions - selection imposed by hosts rewarding cooperation or punishing less cooperative behaviour (Kiers *et al.,* 2003), ii) sensory traps which are signal mimics that exploit the adaptive, neural responses of signal receivers to elicit out-of-context behaviours (Edwards and Yu, 2007) and, iii) filters - mechanisms which allow individuals to prevent or to avoid costly association with parasites, allowing mutualists to solely interact with and invest in cooperators (Yu, 2001). Paradoxically, occurrence of cheaters may facilitate the maintenance of partner choice (Foster and Kokko, 2006). Thus cheaters may be instrumental in stabilising cooperation in mutualistic associations. It appears that cheating persists when the cheaters are able to avoid host sanctions (Denison and Kiers, 2004). Punishment has been documented to enforce cooperation and prevent cheating in both social insects and human societies (Henrichet *et al.,* 2006; Wenseleers and Ratnieks, 2006). Recent evidences thus suggest that failure to punish leads to cheating in plant- insect mutualism also (Edwards *et al.,* 2009). Since the exploited partner suffers a loss on account of nectar/pollen robbing and deceitful exploitation of its resources (without getting benefit in return) further investigations need to focus on the fitness consequences on the exploited species taking into account the network of interacting species.

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References

Agarwal VM and Rastogi N, 2008a. Role of floral repellents in the regulation of flower visits of extrafloral nectary-visiting ants in an Indian crop plant. *Ecological Entomology*, 33**:** 59–65.

Agarwal VM and Rastogi N, 2008b. Deterrent effect of extra-floral nectary visiting ants on *Raphidopalpa foveicollis*, a major insect pest of sponge gourd, *Luffa cylindrica. Entomologia Experimentalis et Applicata,* 128**:** 303–311.

Agarwal VM and Rastogi N, 2009. Spatiotemporal dynamics and plant-part preference patterns of the plant-visiting ants and the insect herbivores of the sponge gourd plants. *Journal of Asia-Pacific Entomology, Elsevier*, 12: 57-66.

Anderson B and Midgley JJ, 2002. It takes two to tango but three is a tangle: mutualists and cheaters on the carnivorous plant *Roridula. Oecologia,* 132:369–373.

Anderson, B, Steven Johanson SD and Carbuttc. Exploitation of a specialized mutulism by a deceptive orchid. *American Journal of Botany,* 92:1342-1349.

Ayasse M, Schiestl FP, Paulus HF, Löfstedt C, Hansson BS, Ibarra F and Francke W, 2000. Evolution of reproductive strategies in the sexually deceptive orchid *Ophrys sphegodes*: How does flower-specific variation of odor signals influence reproductive success? *Evolution*, 54(6):1995–2006.

Baker HG, 1986. Yuccas and yucca moths: a historical commentary. *Annals of the Missouri Botanical Garden,* 73: 556–564.

Barrows EM, 1976. Nectar Robbing and Pollination of *Lantana camara* (Verbenaceae) *Biotropica,* 8(2): 132-135.

Barrows EM, Chabot MR, Michener CD, and Synder TP, 1976. Foraging and mating behavior in *Perdita texana* (Hymenoptera: Andrendae) *Journal of the Kansas Entomological Society*, 49: 275–278.

Beattie AJ, 1985. The evolutionary ecology of ant–plant mutualisms. Cambridge University Press, U.K.

Bever JD, Richardson SC, Lawrence BM, Holmes J and Watson M, 2009. Preferential allocation to beneficial symbiont with spatial structure maintains mycorrhizal mutualism. *Ecology Letters*, 12: 13–21.

Bloch D, Werdenberg N and Erhard A, 2006. Pollination crisis in the butterfly-pollinated wild carnation *Dianthus carthusianorum*? *New Phytologist* 169: 699–706.

Bosch J, Retana J and Cerda X, 1997. Flowering phenology, floral traits and pollinator composition in a herbaceous Mediterranean plant community. *Oecologia*, 109, 583–591.

Brantjes NBM, 1980. Flower morphology of *Aristolochia* species andconsequences for pollination. *Acta Botanica Neerlandica* 29: 212–213.

Brodmann J, Twele R, Francke W, Hölzler G, Zhang Q and Ayasse M, 2008. Orchids mimic green-leaf volatiles to attract prey-hunting wasps for pollination. *Current Biology*, 18 (10): 740-744.

Coombs G and Peter CI, 2009. Do floral traits of *Strelitzia reginae* limit nectar theft by sunbirds? *South African Journal of Botany,* 75: 751–756.

Conner JK, Davis R and Rush S, 1995. The effect of wild radish floral morphology on pollination efficiency by four taxa of pollinators. *Oecologia,* 104(2): 234-245.

Endress PK, 1994. Diversity and evolutionary biology of tropical flowers. Cambridge University Press, Cambridge, UK.

Brantjes NBM, 1980. Flower morphology of *Aristolochia* species and consequences for pollination. *Acta Botanica Neerlandica* 29: 212–213.

Bronstein JL, 1998. The contribution of ant plant protection studies to our understanding of mutualism. *Biotropica*, 30:150–161.

Bronstein JL, 2001. The exploitation of mutualisms. *Ecology Letters,* 4: 277–287.

Bronstein JL, Alarcon R and Geber M, 2006. The evolution of plant– insect mutualisms. *New Phytologist,* 172: 412–428.

Clarke CM and Kitching RL, 1995. Swimming ants and pitcher plants: a unique ant-plant interaction from Borneo. *Journal of Tropical Biology,* 11: 589–602.

Clement LW, Köppen SC, Brand WA and Heil M, 2008. Strategies of a parasite of the ant-Acacia mutualism. *Behavioral Ecology and Sociobiology,* 62(6): 953-962.

Dedej S and Delaplane KS, 2004. Nectar-robbing carpenter bees reduce seed-setting capability of honey bees (Hymenoptera: Apidae) in rabbiteye blueberry, *Vaccinium ashei* ,'Climax'. *Environmental Entomology*, 33: 100-106.

de Vega, C, Arista M, Ortiz PL, Herrera CM and Talavera, S, 2009. The ant-pollination system of *Cytinus hypocistis* (Cytinaceae), a Mediterranean root holoparasite. *Annals of Botany,* 103: 1065–1075.

Delaplane KS and Mayer DF, 2000. Crop pollination by bees. CABI Publishing, Oxon, United Kingdom.

Denison RF and Kiers ET, 2004. Lifestyle alternatives for rhizobia: mutualism, parasitism, and forgoing symbiosis. *FEMS Microbiology Letters*, 237: 187–193

Douglas AE, 2008. Conflict, cheats and the persistence of symbioses. *New Phytologist,*177: 849–858.

Doebeli M and Knowlton N, 1998. The evolution of interspecific mutualisms. *Proceedings of the National Academy of Sciences USA,* 95: 8676–8680.

Dolling WR and Palmer JM, 1991. *Pameridea* (Hemiptera: Miridae): predaceous bugs specific to the highly viscid plant genus *Roridula. Systematic Entomology,* 16: 319 - 328.

Edwards DP, 2009.The roles of tolerance in the evolution, maintenance and breakdown of mutualis. *Naturwissenschaften*, 9610): 1137-1145.

Edwards DP, Hassall M, Sutherland WJ and Yu DW, 2006. Selection for protection in an ant-plant mutualism: host sanctions, host modularity, and the principal agent game. *Proceedings of the*

Royal Society of London B, 273: 595–602.

Edwards DP and Yu DW, 2007. The roles of sensory traps in the origin, maintenance, and breakdown of mutualism. *Behavioral Ecology and Sociobiology,* 61: 1321–1327.

Edwards DP, Ansell FA, Woodcock P, Tom M. Fayle TM, Chey VK and Hamer KC 2009. Can the failure to punish promote cheating in mutualism? *Oikos,* doi: 10.1111/j.1600-0706.2009.17591.x

Ellis AG and Midgley JJ, 1996. A new plant-animal mutualism involving a plant with sticky leaves and a resident hemipteran. *Oecologia*, 106: 478–481.

Endress PK, 1994. Diversity and evolutionary biology of tropical flowers. Cambridge University Press, Cambridge, UK.

Fiala B and Maschwitz U, 1992. Domatia as most important adaptations in the evolution of myrmecop hytes in the Paleotropical tree genus *Macaranga* (Euphorbiaceae). *Plant systematics and evolution,*180: 53–64.

Foster, KR, Kokko, H, 2006. Cheating can stabilize cooperation in mutualisms. *Proceedings of the Royal Society of London B,* 273: 2233–2239.

Foster KR and Wenseleers T, 2006. A general model for the evolution of mutualisms. *Journal of Evolutionary Biology,* 19:1283–1293.

Ghazoul J, 2001. Can floral repellants pre-empt potential ant-plant conflicts? Ecology *Letters*, 4: 295-299.

Gaume L, McKey D and Anstett MC, 1997. Benefits conferred by 'timid' ants: active anti-herbivore protection of the rainforest tree *Leonardoxa africana* by the minute ant *Petalomyrmex phylax*. *Oecologia,* 112**:** 209–216.

Gómez JM, 2000. Effectiveness of ants as pollinators of *Lobularia maritima*: effects on main sequential fitness components of the host plant. *Oecologia*, 122: 90–97.

Gómez JM, Zamora R. Hódar JA and García D, 1996. Experimental study of pollination by ants in Mediterranean high mountain and arid habitats. *Oecologia*, 105: 236–242.

González-Teuber M and Heil M, 2009. Nectar chemistry is tailored for both attraction of mutualists and protection from exploiters. *Plant Signaling and Behavior* 4(9): 809-813.

Gottsberger G, 1989. Comments on flower evolution and beetle pollination in the genera *Annona* and *Rolh'nia (Annonaceae). Plant Systematics and evolution,* 167: 189- 194.

Gullan PJ and Cranston P, 2004. The Insects: An Outline of Entomology**,** Wiley-Blackwell, pp.528.

Handel SN, 1976. Dispersal ecology of *Carex pedunculata* (Cyperaceae), a new North American myrmecochore. *American Journal of Botany* 63**:** 1071–1079.

Hawkins RP, 1961. Observations on the pollination of red clover by bees. *Annals of Applied Biology*, 49: 55–65.

Heil M and McKey D, 2003. Protective ant–plant interactions as model systems in ecological and evolutionary research. *Annual Review of Ecology, Evolution and Systematics,* 34: 425–453.

Heil M, González-Teuber M, Lars W, Clement LW, Kautz S, Verhaagh M and Silva Bueno JC,

2009. Divergent investment strategies of Acacia myrmecophytes and the coexistence of mutualists and exploiters. *Proceedings of the National Academy of Sciences,* 106 (43): 18091–18096.

Henrich J, McElreath R, Barr A, Ensminger J, Barrett C, Bolyanatz A, Cardenas, CJ, Gurven M, Gwako E, Henrich N, Lesorogol C, Marlowe F, Tracer D and Ziker J, 2006. Costly punishment across human societies. *Science*, 312: 1767-1770.

Heinrich B and Raven PH, 1972 Energetics and pollination ecology. *Science,* 176**:** 597–602.

Hölldobler B and Wilson EO 1994. Journey to the ants. The Belknap Press of the Harvard University Press, Cambridge, MA, pp. 228.

Hughes L, Westoby M and Jurado E, 1994. Convergence of elaiosomes and insect prey—evidence from ant foraging behavior and fatty-acid composition. *Functional Ecology***,** 8: 358–365.

Inouye DW, 1983. The ecology of nectar robbing. In: The biology of nectaries. Bentley B and Elias T, (eds.) Columbia University Press, New York.

Irwin RE, 2000. Hummingbird avoidance of nectar-robbed plants: spatial location or visual cues. *Oikos,* 91: 499-506.

Izzo, TJ and Vasconcelos HL, 2002. Cheating the cheater: domatia loss minimizes the effects of ant castration in an Amazonian ant-plant. *Oecologia,* 133: 200-205.

Janzen DH 1966. Coevolution of mutualism between ants and acacias in Central America. *Evolution*, 20: 249–275.

Janzen DH, 1979. How to be a fig. *Annual Review of Ecology and Systematics,* 10: 13–51.

Johnson SD, Alexandersson R and Linder HP, 2003.Experimental and phylogenetic evidence for floral mimicry in a guild of fly-pollinated plants. *Biological Journal of the Linnean Society,* 80: 289–304.

Jones EI, Ferrière R and Bronstein JL, 2009. Eco-Evolutionary dynamics of mutualists and exploiters. *The American Naturalist,* 174: 780–794.

Junker RR and Blüthgen N, 2008. Floral scents repel potentially nectar-thieving ants. *Evolutionary Ecology Research*, 10: 295–308.

Kautz S, Lumbsch HT, Ward PS and Heil M, 2009. How to prevent cheating: a digestive specialization ties mutualistic plant-ants to their ant-plant partners. *Evolution*, 63(4): 839-853.

Kearns CA, 2001. North American dipteran pollinators: assessing their value and conservation status. *Conservation Ecology* 5(1): 5. [online] URL: http://www.consecol.org/vol5/iss1/art5/

Kevan PG and Baker HG, 1983. Insects as flower visitors and pollinators. *Annual Review of Entomology,* 28: 407-445.

Kiers ET, Rousseau RA, Stuart A, West SA and Denison RF, 2003. Host sanctions and the legume – *Rhizobium* mutualism. *Nature,* 425: 78-21.

Klein A, Vaissière BE, Cane JH, Steffan-Dewenter I., Cunningham SA, Kremen C and Tscharntke T, 2007. Importance of pollinators in changing landscapes for world crop. *Proceedings of the National Academy of Sciences USA*, 274: 303–313.

Kremen C, Williams NM and Thorp RW, 2002. Crop pollination from native bees at risk from

agricultural intensification. *Proceedings of the National Academy of Sciences* USA, 99: 16 812–16 816.

Mainero JS and del Rio CM 1985. Cheating and taking advantage in mutualistic associations. In: The biology of mutualism. Boucher DH, (ed.) Croom Helm, London, pp 192- 216.

Moog U, Fiala B, Federle W and Maschwitz U, 2002.Thrips pollination of the dioecious ant plant *Macaranga hullettii* (Euphorbiaceae) in Southeast Asia. *American Journal of Botany,* Vol. 89 (1) 50-59.

Ness JH, Morin DF and Giladi I, 2009. Uncommon specialization in a mutualism between a temperate herbaceous plant guild and an ant: are *Aphaenogaster* ants keystone mutualists? *Oikos,* doi: 10.1111/j.1600-0706.2009.17430.x,

Norment CJ, 1988. The effect of nectar-thieving ants on the reproductive success of *Frasera speciosa* (Gentianaceae). *American Midland Naturalist,* 120: 331-336.

Paxton RJ and Tengö J, 2001. Doubly duped males: the sweet and sour of the orchid's bouquet. *Trends in Ecology and Evolution,* 6, (4): 167-169.

Peakall R, 1989. The unique pollination of *Leporella fimbriata* (Orchidaceae): pollination by pseudocopulating male ants (*Myrmecia urens,* Formicidae). *Plant Systematics and Evolution,* 167: 137-148.

Peakall R and Beattie AJ, 1989. Pollination of the orchid *Microtis parviflora*by flightless worker ants. *Functional Ecology,* 3: 515-522.

Peakall R, Handel S N and Beattie AJ, 1991. The evidence for and importance of ant pollination. In: 'Ant-Plant Interactions', C R. Huxley and Cutler D, (eds.) Oxford University Press, Oxford. *Invited contribution,* pp. 421-429.

Peakall R, Angus CJ and Beattie AJ, 1990. The significance of ant and plant traits for ant pollination in *Leporella fimbriata. Oecologia,* 84: 457-460.

Pellmyr O and Huth CJ, 1994. Evolutionary stability of mutualism between yuccas and yucca moths. *Nature*, 372: 257–260.

Pellmyr O and Krenn HW, 2002. Origin of a complex key innovation in an obligate plant-insect mutualism. *Proceedings of the National Academy of Sciences*, 99: 5498-5502.

Pellmyr O and Leebens-Mack J, 1999. Forty million years of mutualism: evidence for an Eocene origin of the yucca-yucca moth association. *Proceedings of the National Academy of Sciences,* 96: 9178-9183.

Pellmyr O and Leebens-Mack J, 2000. Reversal of mutualism as a mechanism for adaptive radiation in yucca moths. *American Naturalist*, 156: S62-S76.

Pettersson MW, 1999. Can selection to escape nectar thieving force plants to portion nectar in many flowers? *Acta Oecologica,* 20 (1): 67-69.

Pleasants JM, 1983. Nectar production in *Ipomopsis aggregate* (Polemoniaceae). *American Journal of Botany*, 70: 1468-1475.

Raine NE, Gammans N, Macfadyen IJ, Scrivner GK and Stone GN, 2004. Guards and thieves: antagonistic interactions between two ant species coexisting on the same ant-plant. *Ecological Entomology*, 29: 345–352.

Sachs JL and Simms EL, 2006. Pathways to mutualism breakdown. *Trends in Ecology and Evolution,* 21: 585–592.

Sakai S, 2002. Aristolochia spp. (Aristolochiaceae) pollinated by flies breeding on decomposing flowers in Panama. *American Journal of Botany,* 89(3): 527–534.

Schemske DW, 1976. Pollination Specificity in Lantana camara and Lantanatrifolia (Verbenaceae). *Biotropica,* 8: 260-264.

Schiestl FP, 2005. On the success of a swindle: pollination by deception in orchids. *Naturwissenschaften,* 92(6): 255-264.

Schiestl FP, Peakall R, Mant JG, Ibarra F, Schulz C, Franke S and Francke W, 2003. The chemistry of sexual deception in an orchid-wasp pollination system. *Science,* 302:437–438

Schurch S, Pfunder M and Roy BA, 2000. Effects of ants on the reproductive success of Euphorbia cyparissias and associated pathogenic rust fungi. *Oikos,* 88: 6-12.

Stanton ML, 2003. Interacting guilds: moving beyond the pairwise perspective on mutualisms. *The American Naturalist,* 162(4 Suppl): S10-23.

Steiner KE, 1998. The evolution of beetle pollination in a South African orchid. *American Journal of Botany,* 85: 1180–1193.

Stout J C, Allen J A and Goulson D, 2000. Nectar robbing,forager efficiency and seed set: bumblebees foraging on the self-incompatible plant Linaria vulgaris (Scrophulariaceae). *Acta Oecologica,* 21: 277-283.

Shuttleworth A and Johnson SD, 2008. Bimodal pollination by wasps and beetles in the African milkweed Xysmalobium undulatum. *Biotropica,* 40:568–574.

Shuttleworth A and Johnson S, 2009a. Specialized pollination in the African milkweed Xysmalobium orbiculare: a key role for floral scent in the attraction of spider-hunting wasps. *Plant Systematics and Evolution,* 280(1-2): 37-44.

Shuttleworth A, Johnson SD 2009b A key role for floral scent in a wasp-pollination system in Eucomis (Hyacinthaceae). *Annals of Botany,* 103: 715–725

Sugiura N, Miyazaki S and Nagaishi S, 2006. A supplementary contribution of ants in the pollination of an orchid, Epipactis thunbergii, usually pollinated by hover flies. *Plant Systematics and Evolution,* 258: 17-26.

Vogel S, 1990. The role of scent glands in pollination. Amerind Publishing, New Delhi, India.

West SA, Kiers ET, Pen I and Denison RF, 2002. Sanctions and mutualism stability: when should less beneficial mutualists be tolerated? *Journal of Evolutionary Biology,* 15: 830–837.

Yu DW, 2001. Parasites of mutualisms. *Biological Journal of the Linnean Society,* 72: 529–546.

Wang RQ, Ridley J, Sun BF, Zheng Q, Dunn DW, Cook J, Shi L, Zhang YP and Yu DW, 2009. Interference competition and high temperatures reduce the virulence of fig wasps and stabilize a fig-wasp mutualism. *PLoS ONE,* 4 (11) e7802.

Wenseleers T and Ratnieks FLW, 2006. Enforced altruism in insect societies. *Nature,* 444: 50.

Williams G, Adam P, and Mound LA, 2001. Thrips (Thysanoptera) pollination in Australian subtropical rainforests, with particular reference to pollination of Wilkiea huegeliana (Monimiaceae). *Journal of Natural History,* 35: 1-21.

Zettler JA, Spira TP and Allen CR, 2001. Ant–seed mutualisms: can red imported fire ants sour the relationship? *Biological Conservation,* 101: 249–253.

Zhang YW, Qian Y, Zhao JM and Guo YH, 2009 Differential effects of nectar robbing by the same bumble-bee species on three sympatric Corydalis species with varied mating systems. *Annals of Botany,* 104(1): 33-39.

JOURNAL OF SCIENTIFIC RESEARCH

BANARAS HINDU UNIVERSITY

Section - C

Physical Science

तत् त्वं पूषन् अपावृणु सत्यधर्माय दृष्टये

NAKED-EYE ANALYTE SENSING THROUGH STRATEGICALLY DESIGNED CHEMOSENSORS

K. K. Upadhyay,*[a] Virendra Kumar,[a] Priyanka Rai,[a] Santosh Srivastava,[b] K. K. Singh[a] and Shubhra Singh[a]

Introduction

The basis of human civilization on this planet is based upon social interaction and mutual recognition. For mutual recognition some sort of mutual relations are must. This macroscopic behavior of living beings is also exhibited in its microscopic form in the realm of atoms and molecules. For executing any function at the level of atoms and molecules mutual interaction is must. Chemists specially supramolecular chemists have been trying to understand the details of chemical processes in terms of recognition of chemical species through strategically designed chemosensors also known as chemoreceptors.[1] These chemoreceptors are also being used as dosimeters for the quantitative estimation of a particular analyte.[2] The first naked eye chemoreceptor was synthesized by Park and Simmons in 1968 for chloride ion.[3] Since then a variety of colorimetric receptors have been synthesized.[4] These colorimetric receptors are actually molecules incorporating electron rich and deficient pockets simultaneously within the same molecular framework hence capable to produce charge transfer absorption spectra on their UV-visible scanning. This type of charge transfer from donor to acceptor within the same molecule on absorption of UV-visible light by the receptor is known as intramolecular charge transfer (ICT).[5] If an analyte is able to modulate this ICT of the receptor in a characteristic way which is not possible by any other analyte in the same way then that particular analyte is recognized by the receptor through a naked eye change in color of the receptor.[6] That is why molecules with capability of exhibiting ICT may be used as a probe for the naked eye sensing of those analytes which are able to bind with these probes in a selective way. The receptors having binding centres on donor and acceptor both are able to function as ditopic receptors. On the other hand if it possesses binding center on only one then it is known as monotopic one. In recent years there has been an upsurge in the tailor made design and synthesis of such receptors which are highly specific for a particular analyte. Peoples are doing recognition of some tedious analytes like chiral molecules. The recognition of analytes finds applications in many fields like synthetic organic chemistry,[7] catalysis,[8] kinetics[9] and in abetting environmental pollution[10] etc.

How naked eye receptors work:

A naked-eye receptor must have potential to exhibit intramolecular charge transfer (ICT) spectrum on its scanning through UV-visible light. Hence it must have the electron rich and deficient pockets within the same species. At the same time it must have some suitable centers on donor/ acceptor or both so that its ICT may be modulated by a suitable analyte resulting into naked-eye change. This modulation of ICT may be either in terms of $\in_{max}$ or λ_{max} leading to naked-eye change in the receptor. In the light of wide distribution of ions from biological system

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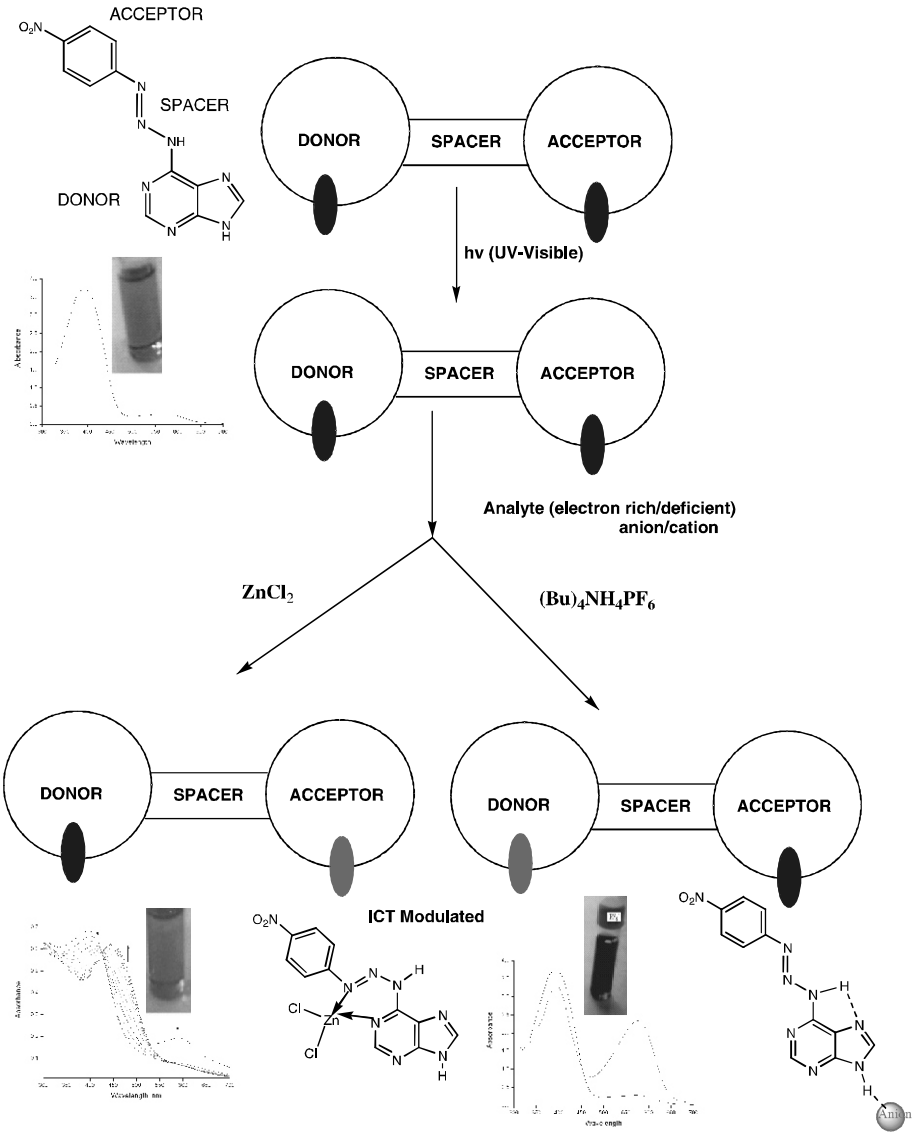
to material ones the ions (cations and anions) constitute a very important class of analyte.[11] For last few years we have been involved in design, synthesis and evaluation of suitable naked-eye receptors which are appropriate binder of cations and anions.[12-14] Either of these species may bind with donor or acceptor pockets of the receptor. If the cation binds with the donor pocket than it reduces the flow of electron from donor to acceptor increasing HOMO-LUMO gap which results into hypsochromic or blue shift while there will be bathochromic shift if the cation binds with acceptor pocket of the receptor leading to decrease in the HOMO-LUMO gap. These shifts will be in vice-versa order if the anion is the analyte.



Scheme - 1 Showing modulation in ICT after interaction of cation/ anion with a particular receptor (*p*-nitrophenyltriazenyl purine; PNTP)

On the other hand the modulation of ICT in terms of $\in_{max}$ may be understood in terms of different extent of approach of equilibrium on the right hand side which may further be understood in terms of different binding ability of receptor with different analytes leading to different binding constants.

$$RH + X^- \rightleftharpoons [R\text{-----}H\text{------}X] \rightleftharpoons R^- + HX$$

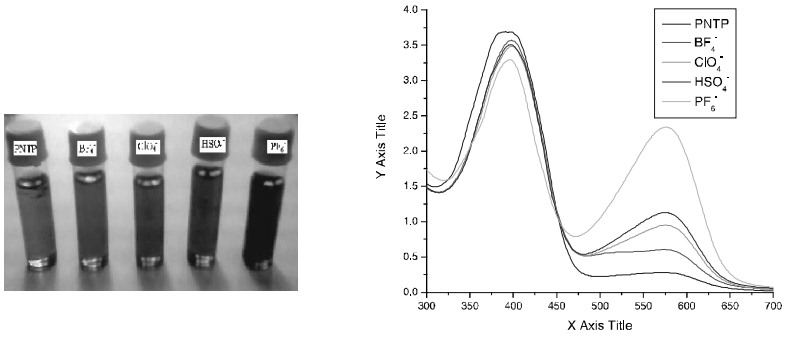


Figure-1 Color changes of PNTP upon interaction with anions and UV-Visible changes of PNTP upon addition of anions

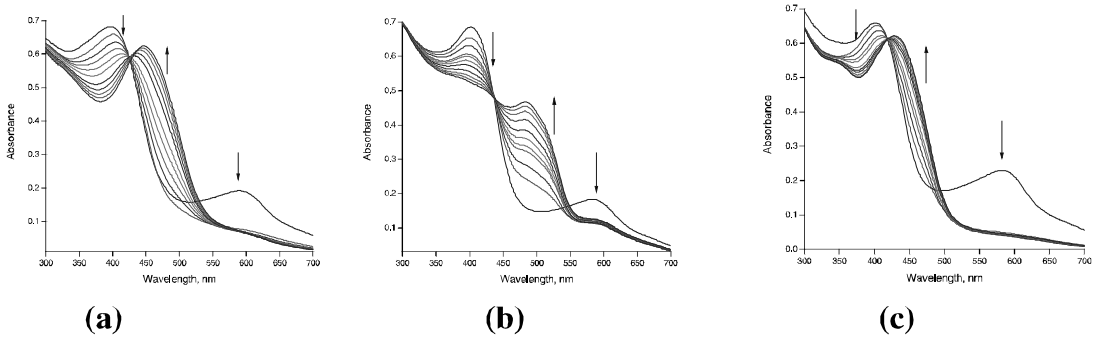


Figure - 2 Titration Curves of receptor 1 with d^{10} metal ions: (a) Zn^{II} (b) Cd^{II} (c) Hg^{II}

An entirely new type of sensing through which we came across recently is the solvent assisted sensing of Hg^{II} where a particular ICT which was not observed in the nujol mull of the receptor but was observed in a polar solvent where X=O (X=C/ S) like DMSO, DMF or acetone. This solvent assisted peak was modulated by cation or anion.[12-14] In the cation one we did naked-eye sensing of an obnoxious metal ion like Hg^{II} at the milimolar level in DMSO/aq. DMSO through strategically designed receptors by the coupling of diazonium salts of a few sulfonamides over a series of active methylene compounds like ethylacetoaceatate, diethyl malonate, acetyl acetone etc. The experimental observations of Hg^{II} naked-eye sensing with one such receptor involving ethylacetoacetate as the active methylene compound (OSPBE)[12] has been presented below.

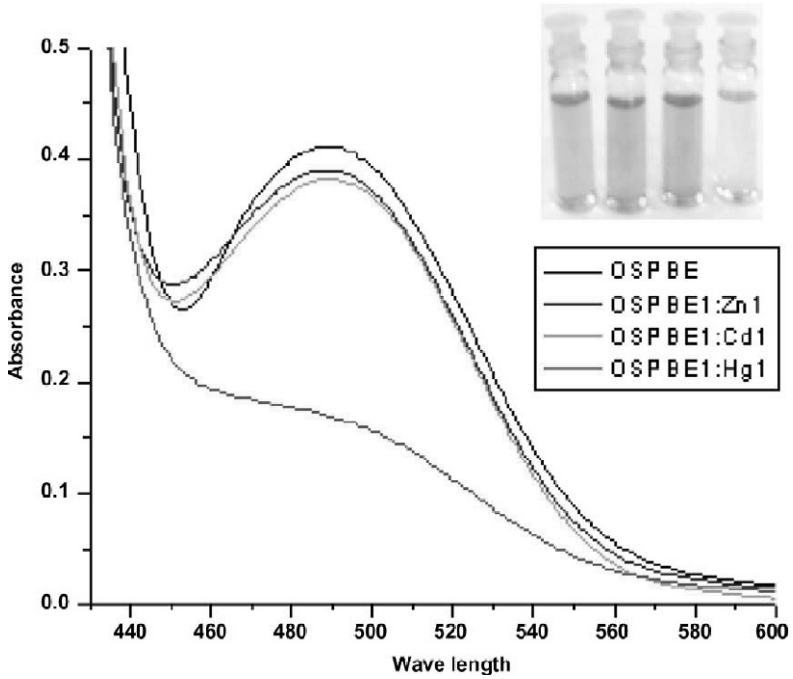


Figure - 3 Absorption spectra of 1x10^{-3} M DMSO solution of OSPBE on addition of 1 equiv. of Zn^{II}, Cd^{II} and Hg^{II} (420–600 nm)

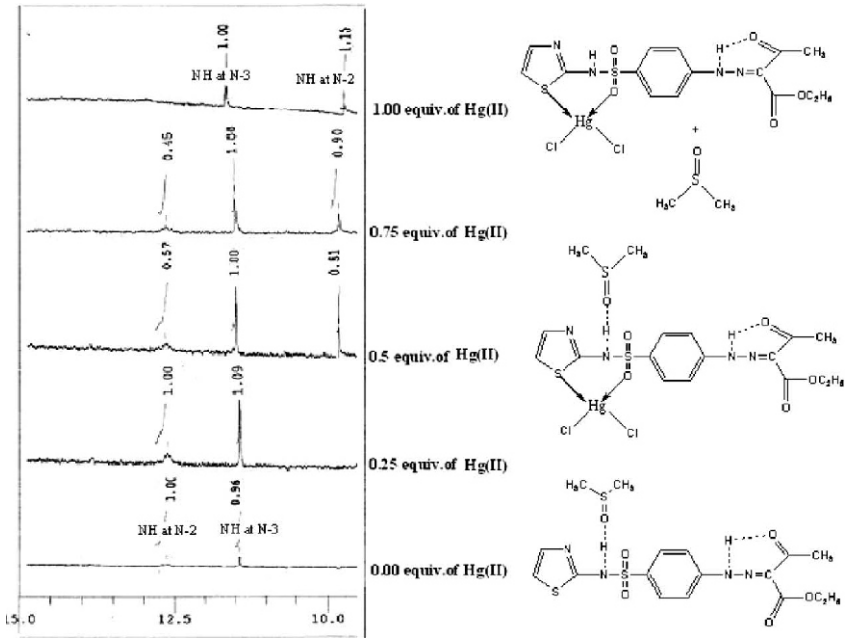


Figure - 4 Truncated ^{1}H NMR spectra (9.5–15.0 ä ppm) of OSPBE on the concomitant addition of 0–1 equiv. of Hg^{II} as its chloride salt to the 1x10^{-3} M DMSO-d_6 solution of OSPBE.

The receptor OSPBE selectively binds with Hg^{II} as its chloride salt among the d^{10} metal ions and produces olive green color. As it can be seen that receptor's absorption peak in the form of a broad band at 489 nm gets vanished on binding with Hg^{II}. The 1H NMR titration spectra shown above narrates the entire process of binding of Hg^{II} with receptor i.e. OSPBE.

Conclusion

Recognition of analytes through purposely designed receptors is an emerging area for last few decades. Many receptors having different potential towards the recognition of analytes have been synthesized by different groups. One of the major bottle neck in this field is the solubility of receptors in aqueous medium. Most of the receptors which have been synthesized and used are in non-aqueous medium like DMSO, acetonitrile etc. That is why the real use of these naked eye receptors has not been percolated to common man for day to day uses. Hence it is the urgent need of hours to do suitable changes at suitable places in preexisting receptors so that they may become water soluble and could be exploited towards analyte sensing particularly for cations and anions to its full potential.

References

1. Examples of chemoreceptors: **(a)** T. Gunnlaugsson, M. Glynn, G.M. Tocci (née Hussey), P.E. Kruger, F.M. Pfeffer, *Coord. Chem. Rev.* **2006**, 250, 3094. **(b)** R. Martinez-Manez, F. Sancenon, *Chem. Rev.* **2003**, 103, 4419. **(c)** K. Ghosh, A.R. Sarkar, G. Masanta, *Tetrahedron Lett.* **2007**, 48, 8725.
2. (a) C. Liu, X. Qian, G. Sun, L. Zhao and Z. Li, *New J. Chem.,* **2008**, 32, 472. (b)
3. 1968 park: C. H. Park, H. E. Simmons, J. Am. Chem. Soc. **1968**, 90, 2431.
4. colorimetric receptors
5. J. Shao, H. Lin, Huakuan Lin, *Talanta* **2008**, 77, 273.
6. ICT based selective receptor S. H. Mashraqui, S. Sundaram and A. C. Bhasikuttan, *Tetrahedron* **2007**, 63, 1680.
7. P. D. Beer, P. A. Gale, *Angew. Chem., Int. Ed.* **2001**, 40, 486.
8. J. L. Sessler, J. M. Davis, *Acc. Chem. Res. 2001*, 34, 989
9. A. Bianchi, K. B. James, E. G. Espana, Supramolecular Chemistry of Anions, Wiley-VCH, New York, 1997.
10. M. Grunder, J. F. Dozol, Z. Asfari, J. Vicens, J. Radioanal. Nucl. Chem. 1999, 241, 59.
11. (a) N. T. V. Le, D. R. Richardson, *Biochim. Biophys. Acta* **2002**, 1603, 31. **(b)** D. H. Saavedra, H. Zhou. J. M. McCord, *Biomed. Pharmacother.* **2005**, 59, 204. **(c)** A. F. Parisi, B.L. Vallee, *Am. J. Clin. Nutr.* **1969**, 22, 1222. (d) R. Uauy, M. Olivares, M. Gonzalez, *Am. J. Clin. Nutr.* **1998,** 67, 952. **(e)** E. D. J. Harris, *Trace Elem. Exp. Med.* **2001**, 14, 207.
12. K.K. Upadhyay, S. Upadhyay, K. Kumar, R. Prasad, *J. Mol. Struct.* **2009**, 927, 60.
13. K. K. Upadhyay, A. Kumar, S. Upadhyay, R. K. Mishra, P. K. Roychoudhuary, *Chem. Lett.* **2008**, 37, 186.
14. K. K. Upadhyay, A. Kumar, R. K. Mishra, R. Prasad, *Bull. Chem. Soc. Jpn.* **2009**, 82, 813.

EFFICIENT SYNTHESIS OF SUBSTITUTED PHTHALEIN DYES USING MICROWAVE IRRADIATION TECHNIQUE

Vandana Srivastava* and Shweta Srivastava

Abstract

Some substituted phthalein dyes have been synthesized by acid catalysed condensation of the various phenols with phthalic anhydride through a novel route of microwave irradiation technique under solvent free condition in high yields within minutes. The method described here is a good, safe, clean, economical and environmentally friendly sovent free and fast reaction conditions are the important features of this procedure and it is an alternative to the classical procedures.

Introduction

Phthaleins are an important class of organic compounds which have wide range applications, such as analytical reagents viz. pH indicators in chemistry and colorants i.e. dye dilution methods to determine kidney or liver function. A large number of phenolphthalein derivatives exhibit cathartic activity and some have been used in pharmaceutical preparations[1,2]. Many methods are described in literature for the synthesis of phthaleins or xanthenes dyes. Simple fusion of both components[3-6] and sometimes combined with azeotropic removal of water[7] or fusion in the presence of catalysts[8-13] such as $POCl_3$, PPA, $POCl_3$-$HClO_4$, $ZnCl_2$ and methane sulphonic acid. The inherent drawbacks of all described synthetic methods are long heating time (upto 10-15hrs) and at defined temperature in the presence of acidic catalyst necessary for high yield reactions. However, the synthesis of these phthaleins through conventional method are facilitated by long heating in the presence of solvent with low yield has received special attention[14,15] to develop more facile and rapid procedure for the synthesis of phthaleins of various phenols.

In recent years microwave heating has gained popularity in organic synthesis. Microwave irradiation is a non-conventional energy source which generates rapid intense heating of polar substances with consequent significant reductions in reaction time from hours to minutes, and give cleaner reactions that are easier to work up, and in many cases increases the yields than those from conventional heating methods[16-20]. Here we describe the application of solvent- free microwave methodology to the synthesis of phthaleins of various phenols.

Results and Discussion

In the course of our effords to produce phthaleins, a mixture of phthalic anhydride (1) and phenols (2) with catalyst conc. H_2SO_4 was irradiated in microwave oven for a specific time(Scheme 1). After usual work up it offered the pure compound (3). Synthesis of phthalein dyes using conc. H_2SO_4 catalysts under microwave irradiations is rapid. The reaction takes place in a single step in this step condensation reaction takes place which results the formation of the product. Phthaleins themselves are believed to prefer the lactone form. Accordingly, the high conversion is attainable only if the free acid formed during the reaction is immediately transformed

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corrosion problems. However, the reaction did not proceed in the absence of any catalyst. The reactants phthalic anhydride and phenol (1:2), the ratio is stiochiometric due to this the purification of the reaction mixture is simple as the separation of excess phenol by time- consuming steam distillation can be avoided.

Some of the known compounds (3a-f) have been reported by conventional method. Here we have repeated their preparation under microwave irradiation. The results obtained are summarised in Table 1.

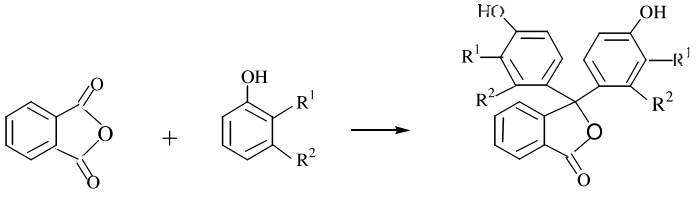
Table - 1 Yield and time of compounds 3a-f

Compound	%Yield			Time (Min.)	M.P. (^{0}C)
	Catalyst				
	Conc. H_2SO_4	Anhy. $ZnCl_2$	Anhy. $AlCl_3$		
3a	84	65	62	2	256-258
3b	72	60	52	2	223-225
3c	67	57	49	4	198-200
3d	80	61	57	4	210-212
3e	74	60	48	2	238-240
3f	81	66	54	3	196-197

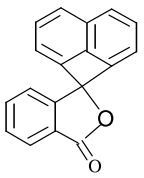
*IR[7]: 3200-3450 (ν OH), 1740-1780 (ν C=O), 1590, 1500, 1470

Table 2: Colour of Phthalein Dyes in NaOH Solution

Compound	3a	3b	3c	3d	3e	3f
Colour of Dye in NaOH Solution	Pink	Red	Bluish purple	Blue (alizarin)	Prussian blue	Faint green



3a. $R^1 = R^2 = H$
3b. $R^1 = CH_3$, $R^2 = H$
3c. $R^1 = H$, $R^2 = CH_3$
3d.. $R^1 = OH$, $R^2 = H$
3e R^1, R^2 = Benzo



3f : 2 = β-naphthol

Scheme 1 :Synthesis of phthaleins in Microwave

Conclusion

It may be concluded that microwave synthesis of the phthalein dyes is the method of choiceIt may be concluded that microwave synthesis of the phthalein dyes is the method of choice, especially for thermostable compounds. This reaction condition provides an efficient method for the rapid access of medicinally important class of organic compounds and can be used as an alternative to the existing procedure.

Experimental

IR spectra were recorded as neat samples on a Perkin-Elmer Spectrum 100 FT-IR spectrophotometer. The microwave irradiated reactions (MWI) were made in a domestic microwave oven (LG, 900W at 2450 MHz). Analytical thin layer chromatography was performed using E. Merck silica gel G. Visualization was accomplished with UV light as well as iodine vapour.

Synthesis of phthaleins (3a-f) of various phenols:

Phthalic anhydride was intimately mixed with various phenols in (1:2) molar proportion. The reaction mixture was irradiated in a microwave oven at 360W for 2-5 minutes in the presence of various catalyst mainly, conc. H_2SO_4, anhy. $AlCl_3$, anhy. $ZnCl_2$. The completion of the reaction was monitored by TLC on silica gel using toluene-ethyl acetate as a solvent system. The reaction mixture was allowed to attain room temperature. The product thus obtained was washed thoroughly with water to remove excess of the phenols. The condensation product was then dissolved in (10%) aq. NaOH and filtered. Phthalein dyes (3a-f) were precipitated from the filtrate by gradual addition of dilute HCl with stirring. Pure phthaleins were obtained by recrystallisation from ethanol.

References

1. Davis, E. G., White, E. C., and Robert, *R.,* (1918): *J. Urology,* 2, 277.
2. Schraufstatter, S., (1950): *Z. Naturforsch,* 5B 190; Loewe, *H.,* (1948): *J.Pharmacol. Exptl. Therap.,* 94, 228.
3. Antonovich, V. P., Ibragimov, G.I., Grekova, I.M.,and Nazarenko,V. A., (1977): *Zh. Anal. Khim,* 32, 876.
4. Kroupa, J., (1990): Czech 250342, *Chem. Abstr.*, 111, 59553.
5. Vodak, Z., and Leminger, O., (1957): Czech 84119, *Chem. Abstr.,* 51, 2867.
6. Leminger, O., and Vodak, Z., (1956): Czech 83863, *Chem. Abstr.,* 50, 16124.
7. Leminger, O., and Vodak, Z., (1956): Czech 83987, *Chem. Abstr.,* 50, 16123.
8. Kosheleva, G. N., and Mikhailov, (1957): G. I., U.S.S.R. 104125, *Chem. Abstr.*, 51, 4217.
9. Hruskova, V., Belusa, J., Kaminska, Z., and Jelinek, (1983): J., Czech 197993, *Chem Abstr.*, 98, 88969.

10.Mikhailov, G. I., Mezentseva, N. C., and Boldyreva, (1969): A. P., U.S.S.R. 232277, *Chem. Abstr.*, 70, 96415.

11.Yarovenko, E. Ya., Kondashova, M. F., Gaivoronskaya, T. G., and Mikhailov, (1968): G. I., U.S.S.R.187191, *Chem Abstr.,* 68, 59300.

12.Sun, W. C., Gee, K. R., Klaubert, D. H., Haugland, R. P., (1997): J. Org. Chem., 62, 6469-6475.

13.Yuichiro, U., Jiao, G. S., Burgess, K., (2004): Practical Synthetic Procedures, 31, 2591-2593.

14.Vishnoi, N. K., (1979): Advanced Practical Organic Chemistry, p.365- 375, Vikas Publishing House PVT LTD.

15.Mann, F. G., and Saunders, B. C., Practical Organic Chemistry, p. 239, Fourth Edition, Longman Group Limitted U. K.

16. Kappe, C. O., (2004): *Angew. Chem. Int. Ed.,* 43, 6250.

17. Varma, S., (1999): *Green Chemistry,* 1, 43.

18. Borah, R., Kiltie, D. J., and Sarma, J. C., (2002): *Indian J. Chem*., 41B, 1032.

19. Kidwai, M., Dave, B., and Venkataramanan, R., (2002): *Indian J. Chem.,* 41B, 2414.

20. Patel, V. M. and Kishor Desai, R., (2004): *Arkivoc,* (i), 123.

21. Kukriti, V. and Chamoli, R. P., *Dyes and Pigments*, (1996): 32, 15-24. *Abstr*., 70, 96415.

ON UNORTHODOX APPROACH TO GENERATING MEASURES OF INFORMATION

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Abstract

A new method for obtaining measures of information is given and used to obtain related measures of information i.e., measure of inaccuracy, measure of directed divergence and measure of entropy.

Key Words

Measure of inaccuracy, Directed Divergence, Entropy

Introduction

Let $P = (p_1, p_2, ...p_n)$ and $Q = (q_1, q_2, ...q_n)$ be two non degenerate complete probability distributions such that

$$p_i, q_i > 0 \forall i = 1,2,.....n \quad \text{..... (1)}$$

and

$$\sum_{i=1}^{n} p_i = 1 = \sum_{i=1}^{n} q_i \quad \text{..... (2)}$$

Then the following measures of inaccuracy [4], measures of directed divergence and measures of entropy [6] respectively are well known in literature respectively:

$$I(P:Q) = -\sum_{i=1}^{n} p_i \ln q_i \quad \text{..... (3)}$$

$$D(P:Q) = -\sum_{=i=1}^{n} p_{i1} \ln \frac{p_i}{q_i} \quad \text{..... (4)}$$

$$and\ S(P) = -\sum_{i=1}^{n} p_i \ln p_i \quad \text{..... (5)}$$

Kapur [3] gave a new approach to generate measures of inaccuracy, measures of directed divergence and measures of entrop. He has considered the function.

$$I^*(P:Q) = -\sum_{i=1}^{n} f(p_i) g(q_i) \quad \text{..... (6)}$$

where (i), $f(p_i)$ is a positive continuous function.

(ii) g(q) is a convex function which is so chosen that

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(a) $I^*(P:Q)$ *is minimum subject to (2) when* $q_i = p_i$ *for each I so that the minimum value of* $I^*(P:Q)$ *is* $I^*(P:P)$

(b) $I^*(P:Q)$ *is a concave function of p, p ..... pn*

The condition (a) will be satisfied if

$f(p)(P_i)=A$ *(a constant)* ..... (7)

so that $g'(p_i)=\frac{A}{f(p_i)}$ ..... (8)

So $g(p_i)=\int_c^{p_i}\frac{A}{f(p_1)}dp_1$ ..... (9)

$g''(p_i)=\frac{Af'(p_i)}{f^2(p_i)}$ ..... (10)

Since we want $g(p_i)$ to be convex so $Af'(p_i) < 0$. The minimum value of I(P:Q) is

$$\sum_{i=1}^{n} f(p_i)\int_c^{q_i}\frac{A}{f(p_i)}dp_i \quad \text{..... (11)}$$

If $I^*(P:P)$ is a concave function of $p_1, p_2, \ldots\ldots\ldots p_n$ and vanishes for degenerate distribution it can be used as a measure of entropy since it is clearly a permutationally symmetric function of $p_1, p_2, \ldots\ldots\ldots p_n$.

Let us consider the following cases (Kapur [2]) :

Case I: When $f(p_i)=p_i$ ..... (12)

then $I(P:Q)=-\sum_{i=1}^{n} p_1 \ln q_i$ ..... (13)

which is Kerridge's [4] measure of inaccuracy and

$I(P:P)=-\sum_{i=1}^{n} p_i \ln p_i$ ..... (14)

is Shannon's [6] measure of entropy.

Case II: If $f(p_i)=p_i^a$, then ..... (15)

$$I(P:Q)=\frac{\sum_{i=1}^{n} p_i^a\left(q_i^{1-a}-1\right)}{\alpha-1} \quad \text{..... (16)}$$

and $$I(P:P)=\frac{\sum_{i=1}^{n} p_i^a-1}{\alpha-1} \quad \text{..... (17)}$$

Which is Havrda-Charvat's [1] measure of entropy.

Case III: If $f(p_i)=p_i+p_i^2$ ..... (18)

then $$I(P:Q)=\sum_{i=1}^{n}(p_i+p_i^2)\ln\left(\frac{q_i+1}{q_i}\right) \quad \text{..... (19)}$$

and $$I(P:P)=\sum_{i=1}^{n}(p_i+p_i^2)\ln\left(\frac{p_i+1}{p_i}\right) \quad \text{..... (20)}$$

This also gives a measure of directed divergence

$$D^*(P:Q)=\sum_{i=1}^{n}(p_i+p_i^2)\ln\left(\frac{q_i+1}{p_i+1}\frac{p_i}{q_i}\right) \quad \text{..... (21)}$$

2. Main Result:

Let $f(p_i)=p_i+ap_i^2$ ..... (22)

then $$g'(p_i)=\frac{A}{p_i(1+ap_i)}=A\left[\frac{1}{p_i}-\frac{a}{1+ap_i}\right] \quad \text{..... (23)}$$

Hence $$g(p_i)=A\ln\left(\frac{p_i}{1+ap_i}\right)+B \quad \text{..... (24)}$$

Also $$g''(p_i)=-\frac{A(1+2ap_i)}{p_i^2(1+ap_i)^2} \quad \text{..... (25)}$$

Thus for g (p_i) to be convex $A<0$ *and* $a\geq-\frac{1}{2}$

Now $$g(p_i)=A\ln\left(\frac{1+ap_i}{p_i}\right)+B, A>0 \quad \text{..... (26)}$$

So $$I(P:Q)\sum_{i=1}^{n}(p_1+ap_1^2)\left\{A\ln\left(\frac{1+aq_1}{q_1}\right)+B\right\} \quad \text{..... (27)}$$

and $$I(P:P)\sum_{i=1}^{n}A(p_i+ap_i^2)\ln\left(\frac{1+ap_i}{p_i}\right)+\sum_{i=1}^{n}BP_i+\sum_{i=1}^{n}Bap_i^2 \quad \text{..... (28)}$$

I(P:P) will be a measure of entropy if it vanishes for any degenerate distribution

$\Delta_r = (0,0,.....,1,.....,0)$

i.e. $A(1+a)\ln(1+a) + B(1+a) = 0$ ..... (29)

i.e. $B = -A\ln(1+a)$ ..... (30)

Thus $I(P:P) = A\left[\sum_{i=1}^{n}\left(p_i + ap_i^2\right)\ln\left(\frac{1+ap_i}{p_i}\right) - \ln(1+a)\right]$ ..... (31)

is a valid measure of entropy when A > 0 and $a \geq \frac{1}{2}$

Particular Cases:

(i) When a = 0 and A = 1, then

$$I(P:P) = \sum_{i=1}^{n} p_i \ln p_i \quad \text{..... (32)}$$

which is Shannon's [6] measure of entropy,

I(P:P) a measure of entropy suffers from an infirmity that it does not vanish for any degenerate probability distribution = (0, 0, …., 1, …, 0)

(ii) When $A = 1, 1(P:P)$

$$= \sum_{i=1}^{n}\left(p_i + ap_i^2\right)\left\{\ln\left(\frac{1+ap_i}{p_i}\right) - \ln(1+a)\right\} \quad \text{..... (33)}$$

is a valid measure of entropy,

Measure of inaccuracy will be

$$I(P:Q) = \sum_{i=1}^{n} p_i(1+ap_i)\left\{\ln\left(\frac{1+aq_i}{q_i}\right) - \ln(1+a)\right\} \quad \text{..... (34)}$$

and measure of directed divergence will be

$D_a(P:Q) = I(P:Q) - I(P:P)$

$$= \sum_{i=1}^{n} p_i(1+ap_i)\ln\left\{\frac{1+aq_i}{1+ap_i}\frac{q_i}{p_i}\right\}$$

When $a = 0$, $D_a(P:Q) = D(P:Q)$ given by Kullback and Leibler [5]

When $a = 1, D_a(P:Q) = D(P:Q)$ given by (21)

When $a = \frac{1}{\lambda}, I(P:Q) = \sum_{i=1}^{n} p_i\left(\frac{\lambda + p_i}{\lambda}\right)\ln\left\{\frac{\lambda + q_i}{p_i(\lambda+1)}\right\}$

Which is Ferrari's measure of entropy and corresponding measure of inaccuracy is

$$I(P:Q)=\sum_{i=1}^{n} p_i\left(\frac{\lambda+p_i}{\lambda}\right)ln\left\{\frac{\lambda+q_i}{q_i(\lambda+1)}\right\}$$

and directed divergence is

$$D(P:Q)=\sum_{i=1}^{n} p_1\left(\frac{\lambda+p_i}{\lambda}\right)ln\left\{\frac{\lambda+q_1}{\lambda+p_i}\frac{p_i}{q_i}\right\}$$

References

1. J.H. Havrda and F. Charvat (1967): "Quantification methods of classification processes, concept of structural entropy", Kybernetika 3, 30-35.
2. J.N. Kapur (1994) : "Measures of information and their application", Wiley Eastern and Wiley.
3. J.N. Kapur (2000): "Geometry of probability spaces", MSTS, New Delhi.
4. D.E.F. Kerridge (1961): "Inaccuracy and inference", J. Roy, Soc. 23A, 184-194.
5. S. Kullback and R.A. Leibler (1951): "On information and Sufficiency", An. Math. State, 22, 77-89.
6. C.E. Shannon (1948): "The Mathematical Theory of Communication: Bell. Syst. Tech. Journ. 27, 423-467.

TRANSMISSION OF INFECTIOUS DISEASES BY DROPLET INFECTION AND THEIR CONTROL BY AGE SPECIFIC IMMUNIZATION: A DELAY MODEL

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Abstract

Infectious diseases of childhood that spread mostly by droplet infection are Chickenpox, Measles, Diphtheria and Whooping cough. In order to study the spread of these respiratory diseases, a delay mathematical model has been proposed and analyzed using stability theory. In the proposed model the underlying population has been divided into two subpopulations consisting of infants and juveniles. For the control of the disease it has been assumed in the model that only infants are vaccinated at a constant rate. Since in the target population, age distribution is considered, a delay in maturation rate has been incorporated in the model. The model has been analyzed by conducting the linear and non-linear stability analysis of the disease free and endemic equilibrium points. On the basis of the asymptotic long term analysis, criteria for the spread and control of the disease have been derived.

Keywords

Maturation delay, Disease frees equilibrium, Endemic equilibrium, Stability, Vaccination, Droplet infection.

Introduction

Infectious diseases of childhood that spread mostly by droplet infection are Chickenpox, Measles, Diphtheria and Whooping cough. Droplet infection is direct projection of a spray of droplets of saliva and naso-pharyngel secretions during coughing, sneezing or speaking and spitting in to surrounding atmosphere. The expelled droplets may impinge directly upon the conjunctiva, oro-respiratory mucosa or skin of a close contact.

In the above-mentioned infectious diseases these droplets which contain millions of bacteria and viruses can be the source of infection to others. When a healthy susceptible person comes within the range of these infected droplets, he is likely to inhale some of them and acquire infection.

All these above-mentioned diseases occur primarily among children under the age of 10 years and one attack generally confers life-long immunity. In all the four diseases referred above immunity after vaccination is long-lasting. In order to study the dynamics of these kinds of disease, age dependent epidemic mathematical models have to be constructed. Some age-dependent epidemic models have been studied mainly by Hethcote [4], Anderson and May [1] and Bussenberg and Castillo-Chavez [2]. Tchuenche et al [7] have studied global behaviour of an SIR epidemiological model with time delay. Jin Z and Ma Z [5] have studied the stability of an SIR epidemic model

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with time delay. Shujing et al [3] have studied impulsive vaccination of an SEIRS model with time delay. In these models age specific vaccination and maturation delay have not been considered. Only recently Misra et. al [6] have studied effects of age based vaccination on the dynamics of delay epidemic model.

In view of the above a delay mathematical model has been considered in this paper to study the transmission of infectious diseases by droplet infection and their control by age specific immunization.

In the formulation of the proposed mathematical model the underlying population has been divided in to two age groups consisting of infants and juveniles because only the populations of these two age groups are being affected by the disease under consideration. For controlling of the disease it has been assumed that only infants are vaccinated at a constant rate as this is being observed in some of the vaccination policies. Since in the target population age distribution is considered, a delay in maturation rate has been also incorporated to make the model more realistic. With these assumptions the mathematical model has been constructed which is being given by the following system of non-linear ordinary differential equations.

Mathematical model 1

$$\frac{dS_1}{dt} = \Lambda - \beta_1 S_1 P - (d+\mu)S_1 - mS_1(t-T) \tag{1}$$

$$\frac{dI_1}{dt} = \beta_1 S_1 P - (d+\gamma_1)I_1 - mI_1(t-T) \tag{2}$$

$$\frac{dR_1}{dt} = \gamma_1 I_1 + \mu S_1 - dR_1 - mR_1(t-T) \tag{3}$$

$$\frac{dS_2}{dt} = -\beta_2 S_2 P + mS_1(t-T) - dS_2 \tag{4}$$

$$\frac{dI_2}{dt} = \beta_2 S_2 P - (d+\gamma_2)I_2 + mI_1(t-T) \tag{5}$$

$$\frac{dR_2}{dt} = \gamma_2 I_2 - dR_2 + mR_1(t-T) \tag{6}$$

$$\frac{dP}{dt} = w(I_1 + I_2) - \delta P \tag{7}$$

with the initial conditions

$$\begin{aligned} &S_1(0) = S_{10} > 0 && I_1(0) = I_{10} > 0 && R_1(0) = 0 \\ &S_2(0) = S_{20} > 0 && I_2(0) = I_{20} > 0 && R_2(0) = 0 \\ &P(0) = P_0 > 0 \end{aligned} \tag{8}$$

where,

S_1 = Susceptible class consisting of infants

S_2 = Susceptible class consisting of juveniles

I_1 = Infective class consisting of infants

I_2 = Infective class consisting of juveniles

R_1 = Removed class consisting of infants

R_2 = Removed class consisting of juveniles

P = Infective pathogen or infectious agents

Λ = Recruitment rate.

d = Natural death rate of humans.

β_i = Transmission rates of infection for i = 1,2

γ_i = Removal rates for i = 1,2

α = Death rate of pathogen.

μ = Vaccination rate

w = The rate at which infective individual produces pathogen.

m = Maturation rate

T = Maturation delay

Equilibrium points:

The two equilibrium points of the model are:

(i) Disease free equilibrium point

$E_0(\bar{S}_1, \bar{I}_1, \bar{R}_1, \bar{S}_2, \bar{I}_2, \bar{R}_2, \bar{P})$, where

$$\bar{S}_1 = \Lambda/(d+\mu+m),\ \bar{I}_1 = 0,\ \bar{R}_1 = \mu\Lambda/(d+m)(d+m+\mu)$$

$$\bar{S}_2 = m\Lambda/d(d+\mu+m),\ \bar{I}_2 = 0,\ \bar{R}_2 = m\mu\Lambda/d(d+m)(d+m+\mu)\ \&\ \bar{P} = 0$$

(ii) Endemic equilibrium point

$E_1(S_1^*, I_1^*, R_1^*, S_1^*, I_1^*, R_1^*, P^*)$, where

$$S_1^* = \Lambda/(d+m+\mu+\beta_1 P^*),\ I_1^* = \beta_1 S_1^* P^*/(\gamma_1+d+m),$$

$$R_1^* = (\mu S_1^* + \gamma_1 I_1^*)/(d+m),\ S_2^* = mS_1^*/(\beta_2 P^* + d),$$

$$I_2^* = (\beta_2 S_2^* P^* + mI_1^*)/(\gamma_2+d),\ R_2^* = (mR_1^* + \gamma_2 I_2^*)/d\ \&$$

$$P^* = \left\{ -q \pm \sqrt{q^2 - 4pr} \right\} / 2p \text{ , provided } q^2 > 4pr$$

where ,

$$p = \delta(\gamma_2 + d)(\gamma_1 + d + m)\beta_1\beta_2,$$

$$q = \delta(\gamma_2 + d)(\gamma_1 + d + m)\{(d + m + \mu)\beta_2 + d\beta_1\} - w\Lambda(\gamma_2 + d + m)\beta_1\beta_2$$

$$r = \delta(\gamma_2 + d)(\gamma_1 + d + m)(d + m + \mu)d - w\Lambda\{(\gamma_2 + d + m)\beta_1 d + (\gamma_1 + d + m)\}\beta_2 m$$

Before analyzing the main model we will present a brief discussion of the following mathematical models (a) and (b) which are special cases of the main model. In the model (a) vaccination and delay has not been considered and in model (b) vaccination has been considered at a constant rates but delay has not been taken into account.

Sub Model (a):

$$\frac{dS_1}{dt} = \Lambda - \beta_1 S_1 P - (d + m)S_1$$

$$\frac{dI_1}{dt} = \beta_1 S_1 P - (d + \gamma_1 + m)I_1$$

$$\frac{dR_1}{dt} = \gamma_1 I_1 - (d + m)R_1$$

$$\frac{dS_2}{dt} = -\beta_2 S_2 P + mS_1 - dS_2$$

$$\frac{dI_2}{dt} = \beta_2 S_2 P - (d + \gamma_2)I_2 + mI_1$$

$$\frac{dR_2}{dt} = \gamma_2 I_2 - dR_2 + mR_1$$

$$\frac{dP}{dt} = w(I_1 + I_2) - \delta P$$

With respect to the above model (a) we put the following results:

(i) The disease free equilibrium point is linearly stable if following conditions are satisfied.

$$\begin{aligned}
&2\mathrm{d} + \mathrm{m} - \beta_1 \bar{S}_1 > 0 \\
&2d + \gamma_1 + m - \beta_1 \bar{S}_1 - w > 0 \\
&2d - \gamma_1 + m > 0 \\
&2d - \beta_2 \bar{S}_2 - m > 0 \\
&2d + \gamma_2 - \beta_2 \bar{S}_2 - m - w > 0 \\
&2d - \gamma_2 - m > 0 \\
&\delta - w - \beta_1 \bar{S}_1 - \beta_2 \bar{S}_2 > 0
\end{aligned} \tag{9}$$

(ii) The endemic equilibrium point is linearly stable if following conditions are satisfied.

$$
\begin{aligned}
&2d + m + \beta_1 P^* - \beta_1 S_1^* > 0 \\
&2d + \gamma_1 + m - \beta_1 P^* - \beta_1 S_1^* - w > 0 \\
&2d - \gamma_1 + m > 0 \\
&2d - \beta_2 P^* - \beta_2 S_2^* - m > 0 \\
&2d + \gamma_2 - \beta_2 P^* - \beta_2 S_2^* - m - w > 0 \\
&2d - \gamma_2 - m > 0 \\
&\delta - w - \beta_1 S_1^* - \beta_2 S_2^* > 0
\end{aligned}
\tag{10}
$$

(iii) The disease free equilibrium point is non- linearly stable in the region given by

$$
\begin{aligned}
&D = \{(S_1, I_1, R_1, S_2, I_2, R_2, P) : 0 < S_1 + I_1 + R_1 \le \alpha_1, 0 < S_2 + I_2 + R_2 \le \alpha_2 \ \& \ P > 0 \\
&\text{where, } \alpha_1 = \Lambda/(d + m) \ \& \ \alpha_2 = m\Lambda / d(d + m)\}
\end{aligned}
$$

if following conditions are satisfied

$$
\begin{aligned}
&2d + m - \beta_1 \Lambda/(d + m) > 0 \\
&2d + \gamma_1 + m - \beta_1 \Lambda/(d + m) - w > 0 \\
&2d - \gamma_1 + m > 0 \\
&2d - \beta_2 m\Lambda / d(d + m) - m > 0 \\
&2d + \gamma_2 - \beta_2 m\Lambda / d(d + m) - m - w > 0 \\
&2d - \gamma_2 - m > 0 \\
&\delta - w - (\beta_2 m / d + \beta_1)\Lambda/(d + m) > 0
\end{aligned}
\tag{11}
$$

(iv) The endemic equilibrium point is non-linearly stable in the region D if following conditions are satisfied.

$$
\begin{aligned}
&2d + m + \beta_1 P^* - \beta_1 \Lambda/(d + m) > 0 \\
&2d + \gamma_1 + m - \beta_1 P^* - \beta_1 \Lambda/(d + m) - w > 0 \\
&2d - \gamma_1 + m > 0 \\
&2d + \beta_2 P^* - \beta_2 m\Lambda / d(d + m) - m > 0 \\
&2d + \gamma_2 - \beta_2 P^* - \beta_2 m\Lambda / d(d + m) - m - w > 0 \\
&2d - \gamma_2 - m > 0 \\
&\delta - w - (\beta_2 m / d + \beta_1)\Lambda/(d + m) > 0
\end{aligned}
\tag{12}
$$

Sub Model (b):

$$\frac{dS_1}{dt} = \Lambda - \beta_1 S_1 P - (d + m + \mu) S_1$$

$$\frac{dI_1}{dt} = \beta_1 S_1 P - (d + \gamma_1 + m) I_1$$

$$\frac{dR_1}{dt} = \gamma_1 I_1 + \mu S_1 - (d+m)R_1$$
$$\frac{dS_2}{dt} = -\beta_2 S_2 P + mS_1 - dS_2$$
$$\frac{dI_2}{dt} = \beta_2 S_2 P - (d+\gamma_2)I_2 + mI_1$$
$$\frac{dR_2}{dt} = \gamma_2 I_2 - dR_2 + mR_1$$
$$\frac{dP}{dt} = w(I_1 + I_2) - \delta P$$

With respect to the above model (b) we put the following results:

(i) The disease free equilibrium point is linearly stable if following conditions are satisfied.

$$\begin{aligned}
&2\mathrm{d} + \mathrm{m} - \beta_1 \overline{\mathrm{S}}_1 > 0 \\
&2d + \gamma_1 + m - \beta_1 \overline{S}_1 - w > 0 \\
&2\mathrm{d} - \gamma_1 - \mu + \mathrm{m} > 0 \\
&2d - \beta_2 \overline{S}_2 - m > 0 \\
&2d + \gamma_2 - \beta_2 \overline{S}_2 - m - w > 0 \\
&2d - \gamma_2 - m > 0 \\
&\delta - w - \beta_1 \overline{S}_1 - \beta_2 \overline{S}_2 > 0
\end{aligned} \tag{13}$$

(ii) The endemic equilibrium point is linearly stable if following conditions are satisfied.

$$\begin{aligned}
&2d + m + \beta_1 P^* - \beta_1 S_1^* > 0 \\
&2d + \gamma_1 + m - \beta_1 P^* - \beta_1 S_1^* - w > 0 \\
&2\mathrm{d} - \gamma_1 - \mu + \mathrm{m} > 0 \\
&2d - \beta_2 P^* - \beta_2 S_2^* - m > 0 \\
&2d + \gamma_2 - \beta_2 P^* - \beta_2 S_2^* - m - w > 0 \\
&2d - \gamma_2 - m > 0 \\
&\delta - w - \beta_1 S_1^* - \beta_2 S_2^* > 0
\end{aligned} \tag{14}$$

(iii) The disease free equilibrium point is non- linearly stable in the region given by

$$\mathrm{D} = \{(\mathrm{S}_1, \mathrm{I}_1, \mathrm{R}_1, \mathrm{S}_2, \mathrm{I}_2, \mathrm{R}_2, \mathrm{P}) : 0 < \mathrm{S}_1 + \mathrm{I}_1 + \mathrm{R}_1 \le \alpha_1, 0 < \mathrm{S}_2 + \mathrm{I}_2 + \mathrm{R}_2 \le \alpha_2 \ \& \ \mathrm{P} > 0$$
$$\text{where, } \alpha_1 = \Lambda/(\mathrm{d}+\mathrm{m}) \ \& \ \alpha_2 = \mathrm{m}\Lambda/\mathrm{d}(\mathrm{d}+\mathrm{m})\}$$

if following conditions are satisfied.

$$2d + m - \beta_1 \Lambda/(d+m) > 0$$

$$
\begin{aligned}
&2d+\gamma_1+m-\beta_1\Lambda/(d+m)-w>0\\
&2\mathrm{d}-\gamma_1-\mu+\mathrm{m}>0\\
&2d-\beta_2 m\Lambda/d(d+m)-m>0\\
&2d+\gamma_2-\beta_2 m\Lambda/d(d+m)-m-w>0\\
&2d-\gamma_2-m>0\\
&\delta-w-(\beta_2 m/d+\beta_1)\Lambda/(d+m)>0
\end{aligned}
\tag{15}
$$

(iv) The endemic equilibrium point is non-linearly stable in the region D if the following conditions are satisfied.

$$
\begin{aligned}
&2d+m+\beta_1 P^*-\beta_1\Lambda/(d+m)>0\\
&2d+\gamma_1+m-\beta_1 P^*-\beta_1\Lambda/(d+m)-w>0\\
&2\mathrm{d}-\gamma_1-\mu+\mathrm{m}>0\\
&2d+\beta_2 P^*-\beta_2 m\Lambda/d(d+m)-m>0\\
&2d+\gamma_2-\beta_2 P^*-\beta_2 m\Lambda/d(d+m)-m-w>0\\
&2d-\gamma_2-m>0\\
&\delta-w-(\beta_2 m/d+\beta_1)\Lambda/(d+m)>0
\end{aligned}
\tag{16}
$$

Now we analyse the main model given by (1) to (7)

2. Linear Stability Analysis:

2.1 Linear Stability Analysis of the Disease Free Equilibrium Point E_0

Consider the following transformation about the equilibrium E_0.

$$
\begin{aligned}
&S_1(t)=\bar{S}_1+n_1(t),\quad I_1(t)=n_2(t),\quad R_1(t)=\bar{R}_1+n_3(t),\\
&S_2(t)=\bar{S}_2+n_4(t),\quad I_2(t)=n_5(t),\quad R_2(t)=\bar{R}_2+n_6(t),\&\\
&P(t)=n_7(t)
\end{aligned}
$$

Using the above transformation in equations (1) to (7) we get

$$\frac{dn_1}{dt}=-(d+\mu)n_1(t)-mn_1(t-T)-\beta_1\{\bar{S}_1+n_1(t)\}n_7(t) \tag{17}$$

$$\frac{dn_2}{dt}=-(\gamma_1+d)n_2(t)-mn_2(t-T)+\beta_1\{\bar{S}_1+n_1(t)\}n_7(t) \tag{18}$$

$$\frac{dn_3}{dt}=\mu n_1(t)+\gamma_1 n_2(t)-dn_3(t)-mn_3(t-T) \tag{19}$$

$$\frac{dn_4}{dt}=mn_1(t-T)-dn_4(t)-\beta_2\{\bar{S}_2+n_4(t)\}n_7(t) \tag{20}$$

$$\frac{dn_5}{dt}=mn_2(t-T)-(\gamma_2+d)n_5(t)+\beta_2\{\bar{S}_2+n_4(t)\}n_7(t) \tag{21}$$

$$\frac{dn_6}{dt} = mn_3(t-T) + \gamma_2 n_5(t) - dn_6(t) \tag{22}$$

$$\frac{dn_7}{dt} = w\{n_2(t) + n_5(t)\} - \delta n_7(t) \tag{23}$$

Which after linearization becomes as:

$$\frac{dn_1}{dt} = -(d+\mu)n_1(t) - mn_1(t-T) - \beta_1 \bar{S}_1 n_7(t) \tag{24}$$

$$\frac{dn_2}{dt} = -(\gamma_1 + d)n_2(t) - mn_2(t-T) + \beta_1 \bar{S}_1 n_7(t) \tag{25}$$

$$\frac{dn_3}{dt} = \mu n_1(t) + \gamma_1 n_2(t) - dn_3(t) - mn_3(t-T) \tag{26}$$

$$\frac{dn_4}{dt} = mn_1(t-T) - dn_4(t) - \beta_2 \bar{S}_2 n_7(t) \tag{27}$$

$$\frac{dn_5}{dt} = mn_2(t-T) - (\gamma_2 + d)n_5(t) + \beta_2 \bar{S}_2 n_7(t) \tag{28}$$

$$\frac{dn_6}{dt} = mn_3(t-T) + \gamma_2 n_5(t) - dn_6(t) \tag{29}$$

$$\frac{dn_7}{dt} = w\{n_2(t) + n_5(t)\} - \delta n_7(t) \tag{30}$$

Now for the linear stability analysis of E_0 we proceed as follows:

$$\text{Let, } V_1 = \left[n_1(t) - \int_{t-T}^{t} mn_1(s)ds \right]^2 + (d+\mu+m+\beta_1\bar{S}_1) \int_{t-T}^{t} \int_{s}^{t} mn_1^2(u)duds \tag{31}$$

Differentiating (31) with respect to t and using the inequality $(a^2 + b^2) \geq \pm 2ab$, we get

$$\frac{dV_1}{dt} \leq \{2(mT-1)(d+\mu+m) + (mT+1)\beta_1\bar{S}_1\}n_1^2(t) + (mT+1)\beta_1\bar{S}_1 n_7^2(t) \tag{32}$$

$$\text{Let, } V_2 = \left[n_2(t) - \int_{t-T}^{t} mn_2(s)ds \right]^2 + (d+\gamma_1+m+\beta_1\bar{S}_1) \int_{t-T}^{t} \int_{s}^{t} mn_2^2(u)duds \tag{33}$$

Differentiating (33) with respect to t and using the inequality $(a^2 + b^2) \geq \pm 2ab$, we get

$$\frac{dV_2}{dt} \leq \{2(mT-1)(d+\gamma_1+m) + (mT+1)\beta_1\bar{S}_1\}n_2^2(t) + (mT+1)\beta_1\bar{S}_1 n_7^2(t) \tag{34}$$

$$\text{Let, } V_3 = \left[n_3(t) - \int_{t-T}^{t} mn_3(s)ds \right]^2 + (d+\gamma_1+m+\mu) \int_{t-T}^{t} \int_{s}^{t} mn_3^2(u)duds \tag{35}$$

Differentiating (35) with respect to t and using the inequality $(a^2 + b^2) \geq \pm 2ab$, we get

$$\frac{dV_3}{dt} \le \{2(mT-1)(d+m)+(mT+1)(\mu+\gamma_1)\}n_3^2(t)+(mT+1)\{\mu n_1^2(t)+\gamma_1 n_2^2(t)\} \tag{36}$$

Let, $V_4 = n_4^2(t) + \int_{t-T}^{t} mn_1^2(s)ds$ (37)

Differentiating (37) with respect to t and using the inequality $(a^2+b^2) \ge \pm 2ab$, we have

$$\frac{dV_4}{dt} \le mn_1^2(t) + (m-2d+\beta_2\bar{S}_2)n_4^2(t) + \beta_2\bar{S}_2 n_7^2(t) \tag{38}$$

Let, $V_5 = n_5^2(t) + \int_{t-T}^{t} mn_2^2(s)ds$ (39)

Differentiating (39) with respect to t and using the inequality $(a^2+b^2) \ge \pm 2ab$, we have

$$\frac{dV_5}{dt} \le mn_2^2(t) + (m-2\gamma_2-2d+\beta_2\bar{S}_2)n_5^2(t) + \beta_2\bar{S}_2 n_7^2(t) \tag{40}$$

Let, $V_6 = n_6^2(t) + \int_{t-T}^{t} mn_3^2(s)ds$ (41)

Differentiating (41) with respect to t and using the inequality $(a^2+b^2) \ge \pm 2ab$, we have

$$\frac{dV_6}{dt} \le mn_3^2(t) + (m+\gamma_2-2d)n_6^2(t) + \gamma_2 n_5^2(t) \tag{42}$$

Let, $V_7 = n_7^2(t)$ (43)

Differentiating (43) with respect to t and using the inequality $(a^2+b^2) \ge \pm 2ab$, we have

$$\frac{dV_7}{dt} \le w\{n_2^2(t)+n_5^2(t)\} + 2(w-\delta)n_7^2(t) \tag{44}$$

Now, we define a Lyapunov functional

$$V = V_1 + V_2 + V_3 + V_4 + V_5 + V_6 + V_7 \tag{45}$$

Then, using the above results we get

$$\begin{aligned}\frac{dV}{dt} \le -[&\{2(1-mT)(d+\mu+m)-(1+mT)(\beta_1\bar{S}_1+\mu)-m\}n_1^2(t)\\ &+\{2(1-mT)(\gamma_1+d+m)-(1+mT)(\beta_1\bar{S}_1+\gamma_1)-m-w\}n_2^2(t)\\ &+\{2(1-mT)(d+m)-(1+mT)(\mu+\gamma_1)-m\}n_3^2(t)\\ &+\{2d-\beta_2\bar{S}_2-m\}n_4^2(t)+\{2d+\gamma_2-\beta_2\bar{S}_2-m-w\}n_5^2(t)\\ &+\{2d-\gamma_2-m\}n_6^2(t)+2\{(\delta-w)-(mT+1)\beta_1\bar{S}_1-\beta_2\bar{S}_2\}n_7^2(t)]\end{aligned} \tag{46}$$

Thus, the disease free equilibrium point E_0 is linearly stable if following conditions are satisfied:

$$\begin{aligned}
&2(1-mT)(d+\mu+m)-(1+mT)(\beta_1\bar{S}_1+\mu)-m>0\\
&2(1-mT)(\gamma_1+d+m)-(1+mT)(\beta_1\bar{S}_1+\gamma_1)-m-w>0\\
&2(1-mT)(d+m)-(1+mT)(\mu+\gamma_1)-m>0\\
&2d-\beta_2\bar{S}_2-m>0\\
&2d+\gamma_2-\beta_2\bar{S}_2-m-w>0\\
&2d-\gamma_2-m>0\\
&\delta-w-(mT+1)\beta_1\bar{S}_1-\beta_2\bar{S}_2>0
\end{aligned} \tag{47}$$

4.2 Linear Stability Analysis of the Endemic Equilibrium Point E_1

Consider the following transformation about the equilibrium E_1

$$\begin{aligned}
&S_1(t)=S_1^*+x_1(t),\quad I_1(t)=I_1^*+x_2(t),\quad R_1(t)=R_1^*+x_3(t)\\
&S_2(t)=S_2^*+x_4(t),\quad I_2(t)=I_2^*+x_5(t),\quad R_2(t)=R_2^*+x_6(t)\\
&P(t)=P^*+x_7(t)
\end{aligned}$$

Using the above transformation in equations (1) to (7) we get

$$\frac{dx_1}{dt}=-(\beta_1P^*+d+\mu)x_1(t)-mx_1(t-T)-\beta_1\{S_1^*+x_1(t)\}x_7(t) \tag{48}$$

$$\frac{dx_2}{dt}=\beta_1P^*x_1(t)-(\gamma_1+d)x_2(t)-mx_2(t-T)+\beta_1\{S_1^*+x_1(t)\}x_7(t) \tag{49}$$

$$\frac{dx_3}{dt}=\mu x_1(t)+\gamma_1x_2(t)-dx_3(t)-mx_3(t-T) \tag{50}$$

$$\frac{dx_4}{dt}=mx_1(t-T)-(\beta_2P^*+d)x_4(t)-\beta_2\{S_2^*+x_4(t)\}x_7(t) \tag{51}$$

$$\frac{dx_5}{dt}=mx_2(t-T)+\beta_2P^*x_4(t)-(\gamma_2+d)x_5(t)+\beta_2\{S_2^*+x_4(t)\}x_7(t) \tag{52}$$

$$\frac{dx_6}{dt}=mx_3(t-T)+\gamma_2x_5(t)-dx_6(t) \tag{53}$$

$$\frac{dx_7}{dt}=w\{x_2(t)+x_5(t)\}-\delta x_7(t) \tag{54}$$

Which after linearization becomes as:

$$\frac{dx_1}{dt}=-(\beta_1P^*+d+\mu)x_1(t)-mx_1(t-T)-\beta_1S_1^*x_7(t) \tag{55}$$

$$\frac{dx_2}{dt}=\beta_1P^*x_1(t)-(\gamma_1+d)x_2(t)-mx_2(t-T)+\beta_1S_1^*x_7(t) \tag{57}$$

$$\frac{dx_3}{dt}=\mu x_1(t)+\gamma_1x_2(t)-dx_3(t)-mx_3(t-T) \tag{58}$$

$$\frac{dx_4}{dt} = mx_1(t-T) - (\beta_2 P^* + d)x_4(t) - \beta_2 S_2^* x_7(t) \tag{59}$$

$$\frac{dx_5}{dt} = mx_2(t-T) + \beta_2 P^* x_4(t) - (\gamma_2 + d)x_5(t) + \beta_2 S_2^* x_7(t) \tag{60}$$

$$\frac{dx_6}{dt} = mx_3(t-T) + \gamma_2 x_5(t) - dx_6(t) \tag{61}$$

$$\frac{dx_7}{dt} = w\{x_2(t) + x_5(t)\} - \delta x_7(t) \tag{62}$$

Now for the linear stability analysis of E_1 we proceed as follows:

Let,

$$U_1 = \left[x_1(t) - \int_{t-T}^{t} mx_1(s)ds \right]^2 + (d + \mu + m + \beta_1 P^* + \beta_1 S_1^*) \int_{t-T}^{t} \int_{s}^{t} mx_1^2(u)duds \tag{63}$$

$$U_2 = \left[x_2(t) - \int_{t-T}^{t} mx_2(s)ds \right]^2 + (d + \gamma_1 + m + \beta_1 P^* + \beta_1 S_1^*) \int_{t-T}^{t} \int_{s}^{t} mx_2^2(u)duds \tag{64}$$

$$U_3 = \left[x_3(t) - \int_{t-T}^{t} mx_3(s)ds \right]^2 + (d + \mu + m + \gamma_1) \int_{t-T}^{t} \int_{s}^{t} mx_3^2(u)duds \tag{65}$$

$$U_4 = x_4^2(t) + \int_{t-T}^{t} mx_1^2(s)ds \tag{66}$$

$$U_5 = x_5^2(t) + \int_{t-T}^{t} mx_2^2(s)ds \tag{67}$$

$$U_6 = x_6^2(t) + \int_{t-T}^{t} mx_3^2(s)ds \tag{68}$$

$$U_7 = x_7^2(t) \tag{69}$$

Differentiating (63) to (69) with respect to t and using the inequality $(a^2 + b^2) \geq \pm 2ab$, we get,

$$\frac{dU_1}{dt} \leq \{2(mT-1)(d + \mu + m + \beta_1 P^*) + (mT+1)\beta_1 S_1^*\} x_1^2(t) + \beta_1 S_1^* (mT+1) x_7^2(t) \tag{70}$$

$$\frac{dU_2}{dt} \leq (mT+1)\beta_1 P^* x_1^2(t) + \{(2mT-1)(d + \gamma_1 + m) + (mT+1)(\beta_1 P^* + \beta_1 S_1^*)\} x_2^2(t) + \beta_1 S_1^* (mT+1) x_7^2(t) \tag{71}$$

$$\frac{dU_3}{dt} \leq (mT+1)\mu x_1^2(t) + (mT+1)\gamma_1 x_2^2(t) + \{(mT+1)(\mu + \gamma_1) + 2(mT-1)(d+m)\} x_3^2(t) \tag{72}$$

$$\frac{dU_4}{dt} \le mx_1^2(t) + \{m - 2(\beta_2 P^* + d) + \beta_2 S_2^*\}x_4^2(t) + \beta_2 S_2^* x_7^2(t) \quad (73)$$

$$\frac{dU_5}{dt} \le mx_2^2(t) + \beta_2 P^* x_4^2(t) + \{m - 2\gamma_2 - 2d + \beta_2 P^* + \beta_2 S_2^*\}x_5^2(t) + \beta_2 S_2^* x_7^2(t) \quad (74)$$

$$\frac{dU_6}{dt} \le mx_3^2(t) + \gamma_2 x_5^2(t) + (m + \gamma_2 - 2d)x_6^2(t) \quad (75)$$

$$\frac{dU_7}{dt} \le w\{x_2^2(t) + x_5^2(t)\} + 2(w - \delta)x_7^2(t) \quad (76)$$

Now, we define a Lyapunov functional

$$U = U_1 + U_2 + U_3 + U_4 + U_5 + U_6 + U_7$$

Then, using the above results we get

$$\begin{aligned}\frac{dU}{dt} \le -[&\{2(1-mT)(d+m+\mu+\beta_1 P^*) - (1+mT)(\beta_1 S_1^* + \beta_1 P^* + \mu) - m\}x_1^2(t) \\ &+ \{2(1-mT)(\gamma_1 + d + m) - (1+mT)(\beta_1 P^* + \beta_1 S_1^* + \gamma_1) - m - w\}x_2^2(t) \\ &+ \{2(1-mT)(d+m) - (1+mT)(\mu+\gamma_1) - m\}x_3^2(t) \\ &+ \{2d + \beta_2 P^* - \beta_2 S_2^* - m\}x_4^2(t) + \{2d + \gamma_2 - \beta_2 P^* - \beta_2 S_2^* - m - w\}x_5^2(t) \\ &+ \{2d - \gamma_2 - m\}x_6^2(t) + 2\{\delta - w - (mT+1)\beta_1 S_1^* - \beta_2 S_2^*\}x_7^2(t)] \end{aligned} \quad (77)$$

Thus, the endemic equilibrium point E_1 is linearly stable if following conditions are satisfied:

$$\begin{aligned}&2(1-mT)(d+m+\mu+\beta_1 P^*) - (1+mT)(\beta_1 S_1^* + \beta_1 P^* + \mu) - m > 0 \\ &2(1-mT)(\gamma_1 + d + m) - (1+mT)(\beta_1 P^* + \beta_1 S_1^* + \gamma_1) - m - w > 0 \\ &2(1-mT)(d+m) - (1+mT)(\mu+\gamma_1) - m > 0 \\ &2d + \beta_2 P^* - \beta_2 S_2^* - m > 0 \\ &2d + \gamma_2 - \beta_2 P^* - \beta_2 S_2^* - m - w > 0 \\ &2d - \gamma_2 - m > 0 \\ &\delta - w - (mT+1)\beta_1 S_1^* - \beta_2 S_2^* > 0\end{aligned} \quad (78)$$

3. Non-Linear Stability Analysis

3.1 Non-Linear Stability Analysis of the disease free equilibrium point E_0

First we construct a region D as follows:

$$D = \{(S_1, I_1, R_1, S_2, I_2, R_2, P) : 0 < S_1 + I_1 + R_1 \le \alpha_1, 0 < S_2 + I_2 + R_2 \le \alpha_2 \ \& \ P > 0 \quad \text{where,}$$
$$\alpha_1 = \Lambda/(d+m) \ \& \ \alpha_2 = m\Lambda/d(d+m)\}$$

Let W_{11} is a positive definite function given by

$$\begin{aligned}W_{11} = &\left[n_1(t) - \int_{t-T}^{t} mn_1(s)ds\right]^2 + \left[n_2(t) - \int_{t-T}^{t} mn_2(s)ds\right]^2 + \left[n_3(t) - \int_{t-T}^{t} mn_3(s)ds\right]^2 \\ &+ n_4^2(t) + n_5^2(t) + n_6^2(t) + n_7^2(t)\end{aligned} \quad (79)$$

Differentiating (79) with respect to t and using the inequality $(a^2 + b^2) \geq \pm 2ab$ in the region D we get

$$\frac{dW_{11}}{dt} \leq \{(mT-2)(d+\mu+m)+\beta_1\alpha_1+(mT+1)\}\mu n_1^2(t)$$
$$+\{(mT-2)(d+\gamma_1+m)+\beta_1\alpha_1+(mT+1)\gamma_1+w\}n_2^2(t)$$
$$\{(mT-2)(d+m)+\mu+\gamma_1\}n_3^2(t)+\{-2d+m+\beta_2\alpha_2\}n_4^2(t)$$
$$+\{-2d-\gamma_2+\beta_2\alpha_2+m\}n_5^2(t)+\{-2d+\gamma_2+m\}n_6^2(t)$$
$$+2\{(w-\delta)+(mT+1)\beta_1\alpha_1+\beta_2\alpha_2\}n_7^2(t)$$
$$+(d+\mu+m+\beta_1\alpha_1)\int_{t-T}^{t}mn_1^2(s)ds+(d+\gamma_1+m+\beta_1\alpha_1)\int_{t-T}^{t}mn_2^2(s)ds$$
$$+(d+\mu+\gamma_1+m)\int_{t-T}^{t}mn_3^2(s)ds+mn_1^2(t-T)+mn_2^2(t-T)+mn_3^2(t-T) \quad (80)$$

Let, $W_{12} = (d+\mu+m+\beta_1\alpha_1)\int_{t-T}^{t}\int_{s}^{t}mn_1^2(u)duds+(d+\gamma_1+m+\beta_1\alpha_1)\int_{t-T}^{t}\int_{s}^{t}mn_2^2(u)duds$

$$+(d+\mu+\gamma_1+m)\int_{t-T}^{t}\int_{s}^{t}mn_3^2(u)duds+\int_{t-T}^{t}mn_1^2(s)ds+\int_{t-T}^{t}mn_2^2(s)ds+\int_{t-T}^{t}mn_3^2(s)ds \quad (81)$$

Differentiating (81) with respect to t we have,

$$\frac{dW_{12}}{dt} = (d+\mu+m+\beta_1\alpha_1)mTn_1^2(t)-(d+\mu+m+\beta_1\alpha_1)\int_{t-T}^{t}mn_1^2(s)ds$$
$$+(d+\gamma_1+m+\beta_1\alpha_1)mTn_2^2(t)-(d+\gamma_1+m+\beta_1\alpha_1)\int_{t-T}^{t}mn_2^2(s)ds$$
$$+(d+\mu+m+\gamma_1)mTn_3^2(t)-(d+\mu+m+\gamma_1)\int_{t-T}^{t}mn_3^2(s)ds$$
$$+mn_1^2(t)-mn_1^2(t-T)+mn_2^2(t)-mn_2^2(t-T)+mn_3^2(t)-mn_3^2(t-T) \quad (82)$$

Now, we define a Lyapunov functional

$$W_1 = W_{11}+W_{12} \quad (83)$$

Then from (80) and (83) we get,

$$\frac{dW_1}{dt} \leq -[\{2(1-mT)(d+\mu+m)-(1+mT)(\beta_1\alpha_1+\mu)-m\}n_1^2(t)$$
$$+\{2(1-mT)(d+\gamma_1+m)-(1+mT)(\beta_1\alpha_1+\gamma_1)-m-w\}n_2^2(t)$$

$$+\{2(1-mT)(d+m)-(1+mT)(\gamma_1+\mu)-m\}n_3^2(t)$$
$$+\{2d-m-\beta_2\alpha_2\}n_4^2(t)+\{2d+\gamma_2-\beta_2\alpha_2-m-w\}n_5^2(t)$$
$$+\{2d-\gamma_2-m\}n_6^2(t)+2\{(\delta-w)-(mT+1)\beta_1\alpha_1-\beta_2\alpha_2\}n_7^2(t)] \quad (84)$$

Thus, disease free equilibrium point E_0 is non-linearly stable in the region D if following conditions are satisfied:

$$\begin{aligned}
&2(1-mT)(d+\mu+m)-(1+mT)\{\beta_1\Lambda/(d+m)+\mu\}-m>0\\
&2(1-mT)(d+\gamma_1+m)-(1+mT)\{\beta_1\Lambda/(d+m)+\gamma_1\}-m-w>0\\
&2(1-mT)(d+m)-(1+mT)(\gamma_1+\mu)-m>0\\
&2d-m-\beta_2 m\Lambda/d(d+m)>0\\
&2d+\gamma_2-\beta_2 m\Lambda/d(d+m)-m-w>0\\
&2d-\gamma_2-m>0\\
&\delta-w-\{(mT+1)\beta_1+\beta_2 m/d\}\Lambda/(d+m)>0
\end{aligned} \quad (85)$$

3.2 Non-Linear Stability Analysis of equilibrium point E_1

Let W_{21} is a positive definite function given by

$$W_{21}=\left[x_1(t)-\int_{t-T}^{t}mx_1(s)ds\right]^2+\left[x_2(t)-\int_{t-T}^{t}mx_2(s)ds\right]^2+\left[x_3(t)-\int_{t-T}^{t}mx_3(s)ds\right]^2$$
$$+x_4^2(t)+x_5^2(t)+x_6^2(t)+x_7^2(t) \quad (86)$$

Differentiating (86) with respect to t and using the inequality $(a^2+b^2)\geq\pm 2ab$ then in the region D we get,

$$\begin{aligned}
\frac{dW_{21}}{dt}\leq{}&\{(mT-2)(\beta_1P^*+d+\mu+m)+(mT+1)(\beta_1P^*+\mu)+\beta_1\alpha_1\}x_1^2(t)\\
&+\{(mT-2)(d+\gamma_1+m)+\gamma_1(mT+1)+\beta_1P^*+\beta_1\alpha_1+w\}x_2^2(t)\\
&+\{(mT-2)(d+m)+\gamma_1+\mu\}x_3^2(t)+\{-2d+m+\beta_2\alpha_2-\beta_2P^*\}x_4^2(t)\\
&+\{-2d+m+\beta_2\alpha_2+\beta_2P^*-\gamma_2+w\}x_5^2(t)+\{-2d+m+\gamma_2\}x_6^2(t)\\
&+2\{(w-\delta)+(mT+1)\beta_1\alpha_1+\beta_2\alpha_2\}x_7^2(t)\\
&+(\beta_1P^*+d+\mu+m+\beta_1\alpha_1)\int_{t-T}^{t}mx_1^2(s)ds+(\beta_1P^*+d+\gamma_1+m+\beta_1\alpha_1)\int_{t-T}^{t}mx_2^2(s)ds\\
&+(\mu+d+\gamma_1+m)\int_{t-T}^{t}mx_3^2(s)ds+mx_1^2(t-T)+mx_2^2(t-T)+mx_3^2(t-T)
\end{aligned} \quad (87)$$

Let, $W_{22} = (\beta_1 P^* + d + \mu + m + \beta_1\alpha_1)\int_{t-T}^{t}\int_{s}^{t} mx_1^2(u)duds + \int_{t-T}^{T} mx_1^2(s)ds$

$$+ (\beta_1 P^* + d + \gamma_1 + m + \beta_1\alpha_1)\int_{t-T}^{t}\int_{s}^{t} mx_2^2(u)duds + \int_{t-T}^{T} mx_2^2(s)ds$$

$$+ (\mu + d + \gamma_1 + m)\int_{t-T}^{t}\int_{s}^{t} mx_3^2(u)duds + \int_{t-T}^{T} mx_3^2(s)ds \quad (88)$$

Differentiating (88) with respect to t ,we have

$$\frac{dW_{22}}{dt} = (\beta_1 P^* + d + \mu + m + \beta_1\alpha_1)\left[mTx_1^2(t) - \int_{t-T}^{T} mx_1^2(s)ds \right]$$

$$+ (\beta_1 P^* + d + \gamma_1 + m + \beta_1\alpha_1)\left[mTx_2^2(t) - \int_{t-T}^{T} mx_2^2(s)ds \right]$$

$$+ (\gamma_1 + d + \mu + m)\left[mTx_3^2(t) - \int_{t-T}^{T} mx_3^2(s)ds \right]$$

$$+ x_1^2(t) + x_2^2(t) + x_3^2(t) - x_1^2(t-T) - x_2^2(t-T) - x_3^2(t-T) \quad (89)$$

Now, we define a Lyapunov functional

$$W_2 = W_{21} + W_{22} \quad (90)$$

Then from (88) and (90) we get,

$$\frac{dW_2}{dt} \le -[\{2(1-mT)(\beta_1 P^* + d + \mu + m) - (1+mT)(\beta_1 P^* + \beta_1\alpha_1 + \mu) - m\}x_1^2(t)$$

$$+ \{2(1-mT)(\gamma_1 + d + m) - (1+mT)(\beta_1 P^* + \beta_1\alpha_1 + \gamma_1) - m - w\}x_2^2(t)$$

$$+ \{2(1-mT)(d+m) - (1+mT)(\gamma_1 + \mu) - m\}x_3^2(t)$$

$$+ \{2d + \beta_2 P^* - \beta_2\alpha_2 - m\}x_4^2(t) + \{2d + \gamma_2 - \beta_2 P^* - \beta_2\alpha_2 - m - w\}x_5^2(t)$$

$$+ \{2d - m - \gamma_2\}x_6^2(t) + 2\{\delta - w - \beta_2\alpha_2 - (1+mT)\beta_1\alpha_1\}x_7^2(t) \quad (91)$$

Thus endemic equilibrium point E_1 is non-linearly stable in the region D if following conditions are satisfied.

$$2(1-mT)(\beta_1 P^* + d + \mu + m) - (1+mT)(\beta_1 P^* + \beta_1\Lambda/(d+m) + \mu) - m > 0$$

$$2(1-mT)(\gamma_1 + d + m) - (1+mT)(\beta_1 P^* + \beta_1\Lambda/(d+m) + \gamma_1) - m - w > 0$$

$$2(1-mT)(d+m) - (1+mT)(\gamma_1 + \mu) - m > 0$$

$$2d + \beta_2 P^* - \beta_2 m\Lambda / d(d+m) - m > 0 \quad (92)$$

$$2d + \gamma_2 - \beta_2 P^* - \beta_2 m\Lambda / d(d+m) - m - w > 0$$

$$2d - m - \gamma_2 > 0$$

$$\delta - w - \{\beta_2 m / d + (1+mT)\beta_1\}\Lambda/(d+m) > 0$$

Discussion

Here we have analyzed an ordinary differential equation model with time delay for transmission of infectious diseases by droplet infection. By conducting the linear and non-linear stability analysis of the disease free and endemic equilibrium points, it has been shown that disease free and endemic equilibrium points are stable under the conditions involving disease related parameters and time delay. It can be observed from the conditions of stability that if T becomes zero then the stability conditions given for model-1 coincide with the conditions given for model 1(b) which is a special case of model-1. From the values of equilibrium points it may be observed that if the rate of vaccination increases then equilibrium level of disease will decrease. From the stability conditions of model-1 it may be observed that the delay in maturation may lead to the instability of the otherwise stable equilibrium points for large time-delay.

References

1. R.M. Anderson, and R.M. May, *Age Related Change in the Rate of Disease Transmission on Implications for the Design of Vaccination Programs*, J. Hyg., **94** (1985), 365-436.
2. S. Busenberg and C. Castillo-Chavez, *A General Solution of the Problem of Mixing of Subpopulations and its Application to Risk and Age Structured Epidemic Models for the Spread of AIDS,* IMA J. of Math. Med. and Biol., **8** (1991), 1-29.
3. Shujing Gao, Lansnn Chen & Z. Teng, *Impulsive Vaccination of an SEIRS Model with Time Delay and Varying Total Population Size*, Bulletin of Mathematical Biology, **69** (2007), 731-745.
4. H.W. Hethcote, *Optimal Ages of Vaccination for Measles*, Mathematical Biosciences. **89** (1988), 29-52.
5. Z. Jin and , Z. Ma, *The Stability of an SIR Epidemic Model with Time Delays*, J. Mathematical Bioscience and Engineering, **3** (2006), 101-109.
6. O.P.Misra, Y.N. Meitei, V.K. Chaturvedi and S.K.S. Rathore, *Effect of age-based vaccination policy on the dynamics of delay epidemic models*, Mathematical Biology, Recent trends, Anamaya Publishers, New Delhi (2006), 297-304.
7. J.M.Tchnenche, A. Nwagwo and R. Levins, *Global behaviour of SIR Epidemic model with time delay*, Mathematical Methods in the Applied Sciences (Wiley Intersciences), **30** (2007), 733-749.

BIS (BENZOTRIAZOL-1-YL) METHANETHIONE: NON-TOXIC VERSATILE THIOPHOSGENE EQUIVALENT

Archana Singh, Raju R. Kale, Bhuwan B. Mishra, V. Tripathi, and Vinod K. Tiwari*

Introduction

Benzotriazole methodology offers a well known versatile synthetic tool in organic synthesis and has gained extensive popularity since past few decades.[1-2] Nowadays benzoriazole mediated methodology magnificently used in construction of diverse range of biologically active molecules and heterocyclic skeleton. Benzotriazole easily introduce into a chemical moiety by a vast variety of reaction and easily removed at the end of reaction sequence without affecting the other functional group. It significantly influence the activity of other parts of molecule better in comparison to many other groups like phenyl, vinyl etc.

Activation of benzotriazole with other groups

The synthesis of furthermore functionalized benzotriazole is considered to become important subject for synthetic and biological researches since they are expected to exhibit novel properties applicable to such fields. Comparison of benzotriazole activation with other groups is shown below in **Figure 1.**

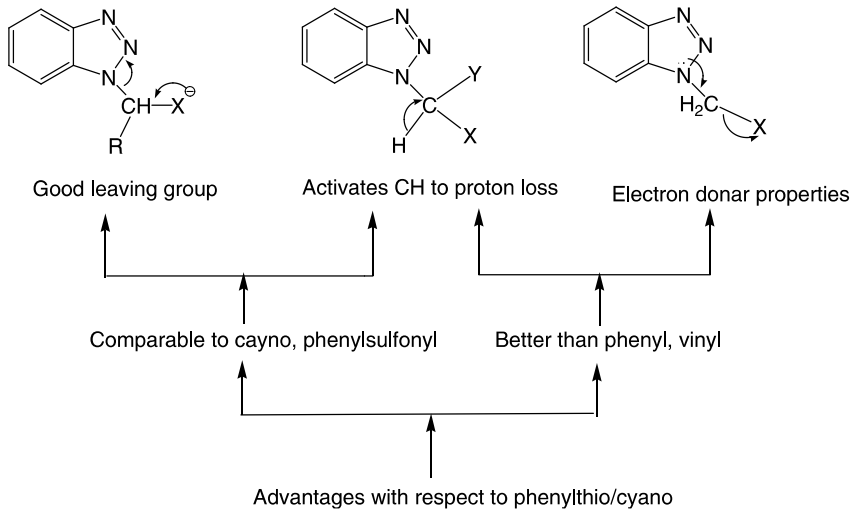

Figure - 1 Activation through benzotriazole

In comparison to many other substituents including halogen, benzotriazole is well known to acts as a leaving group as well as the stability of benzotriazole intermediate/synthons makes it more attractive synthons for organic chemist. Compounds with a benzotriazolyl group R to an amino or ether functionality (X, NR_2, OR) are stable, nonvolatile, easily prepared, and versatile, while their halogen analogues are physiologically dangerous and often too reactive to be conveniently used as reagents.[3-9]

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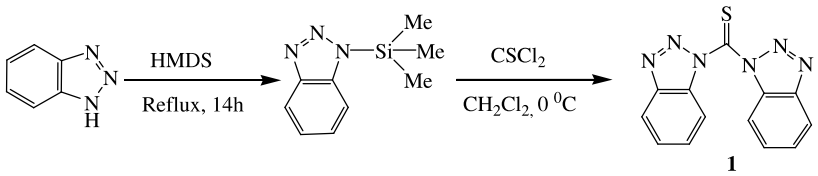
Benzotriazole based synthetic auxiliary also offers many advantages particularly being inexpensive, non-toxic, and have sufficient stability.[2] Very recently with the help of TGA, DTA , DSC analyses confirm that benzotriazole is significantly more stable than 1,2,3-triazole.[10] These properties make *N*-substituted derivatives of benzotriazole sufficiently stable as synthetic auxiliary possess both electron donating and electron withdrawing properties and due to this it show very interesting reaction with the compounds containing heteroatoms especially O, N and S. In past few decades, benzotriazole draws enormous attention and explored in organic synthesis as a new synthetic methodology, catalyst in several reactions such as Baylis Hillman[11] and various coupling reactions and recently as light-activatable DNA cleaving agents.[12]

All these advantage and inertness with stability of benzotriazole ring system always attracts increasing interest of synthetic chemist for the implementation of new methods on this moiety to provide new horizon to synthetic chemistry by preparing many more new Bt mediated synthons which can replace old, conventional and multistep preparative method by new, convenient and simple methodology for the synthesis of useful drugs, biologically active compounds, and many natural product analogues.

Bis **(benzotriazol-1-yl) methanethione**

Bis(benzotriazol-1-yl)methanethione was obtained as yellow crystal (m.p. 170-171 ^{0}C) starting from benzotriazole in high yield (**Scheme 1**).[13] It can also be obtained directly from benzotriazole reacting with thiophosgene.



Scheme - 1

Synthetic applications of bis-(benzotrizol-1-yl) methanethione

Bis(benzotriazolyl) methanethione is a reagent derived from benzotriazole, now proved itself a very effective thiophosgene equivalent in thioacylation[14] and in synthesis of different chemically and biologically active compounds. Compared to thiophosgene,[15] bis-(benzotriazolyl) methanethione is found to more advantageous not because of its less toxicity but also due to its high stability that can be stored for years at room temperature and crystalline solid nature which make it easy to handle during the course of reaction (**Figure 2**).

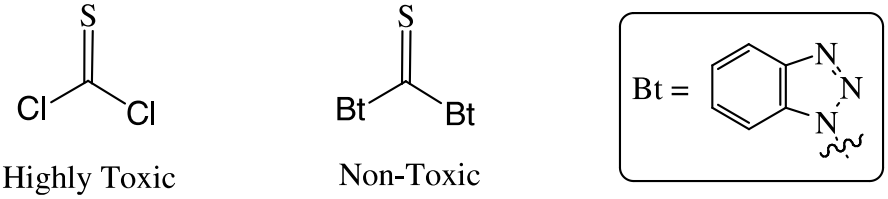


Figure - 2 Structure of Bis(benzotriazolyl)methanethione

It also acts as a precursor in synthetic chemistry for the preparation of many useful reagents, which take part in the synthesis of a vast range of valuable organic molecules like trisubstitued guanidines, N-hydroxy, N-amino guanidines, *â*-enamino thioic acid derivatives, thiosemicarbazide and N- hydroxythiourea. Some representative reactions and reagents derived from *bis*(benzotriazol-1-yl) methanethione are presentated in **Figure 3**.

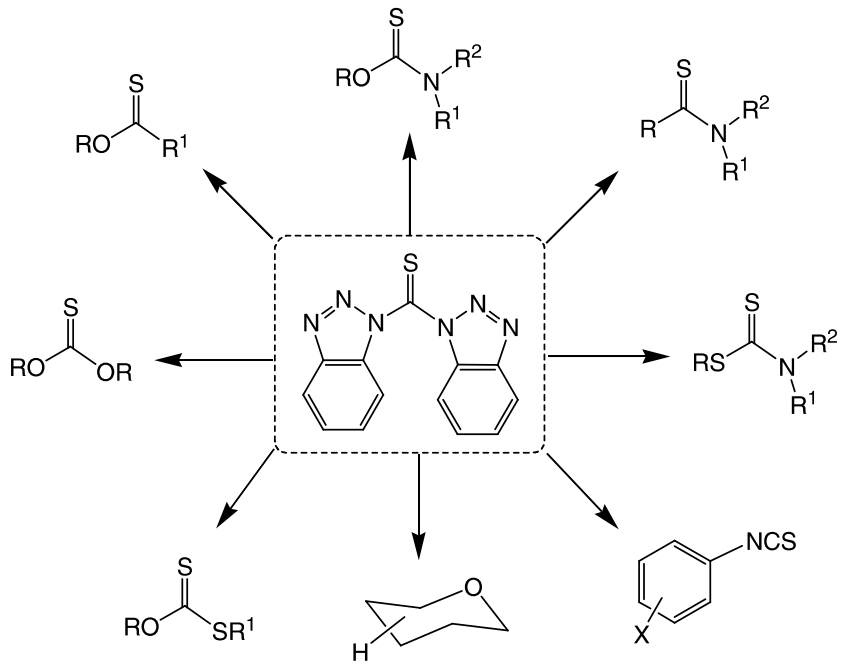
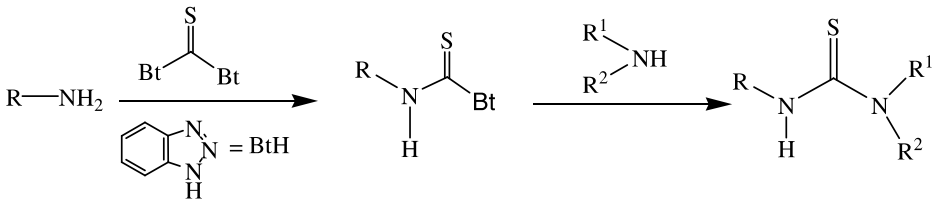


Figure - 3 *Bis*-(benzotriazol-1-yl) methanethione mediated reagents

Preparation of symmetrical/unsymmetrical thioureas

Thioureas are important organic compounds of considerable use in medicinal chemistry due its biological activity as antibacterial, antimicrobial, fungicidal, herbicidal, rodenticidal etc.[16.] Thioureas are also most important synthetic building block for synthesis of five and six membered heterocycles.[17]

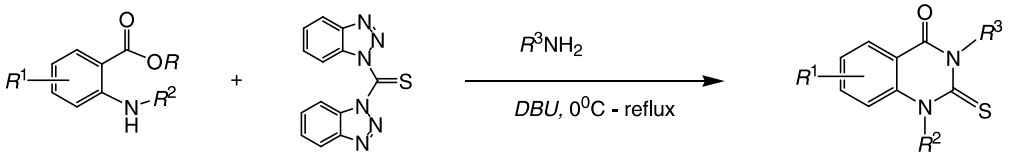
Bis (benzotrizol-1-yl) methanethione has been successfully utilized as isothiocynate equivalent for the efficient synthesis of secondary and tertiary thioureas in high to excellent yield (**Scheme 2**).[18]



Scheme - 2

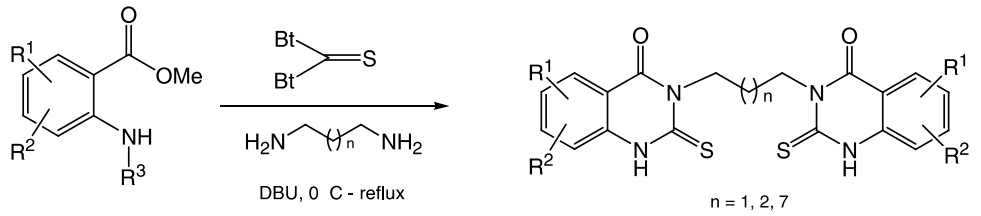
Synthesis of quinazolinone derivatives

Methaqualone (**Figure 3**), the most popular quinazolinone drug synthesized in 1951 for its antimalarial effect,[19] is currently being used for the assessment of the abuse liability of sedative hypnotic drugs.[20] In addition of associated with the diverse range of pharmacological activities, quinazolinone skeleton is frequently encountered as building block for hundreds of alkaloid.[21] The preparative methods leading to these molecules mainly include the reaction of substituted anthranilic acids or its functional derivatives with isothiocyanates, thioureas, excess of refluxing formamide, imidates, methyl N-aryldithiocarbamates, ammonium aryldithiocarbamates, amine and CS_2 in basic medium, RNHCOOEt and imidazole, amine and sodium cyanate, $CSCl_2$ either in presence of NEt_3 or hydrazine, polymer supported $FeCl_3$, orthoesters, and amines under solvent free conditions, anilines, CS_2,



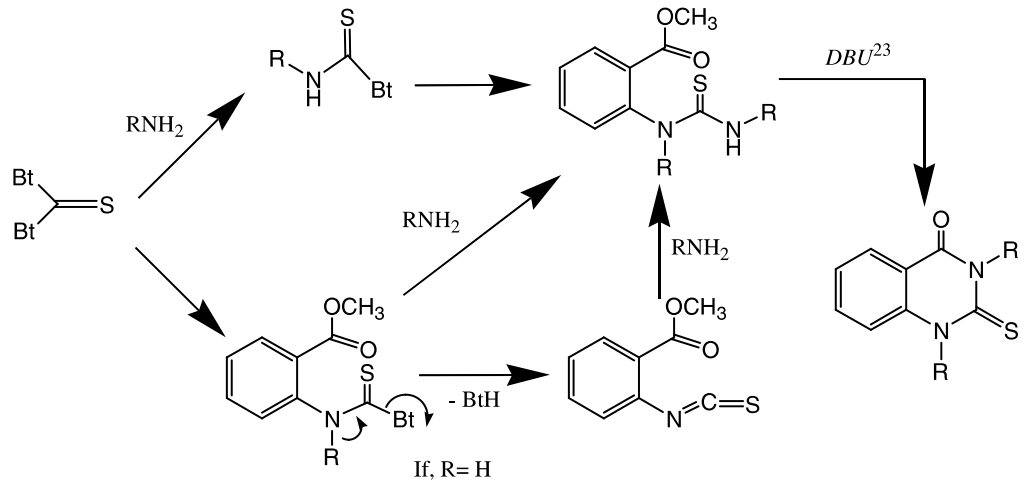
Scheme - 3

Similar reaction of methyl anthranilate with diamines and *bis*-(benzotriazol-1-yl)methanethione in presence of *DBU* using anhydrous dichloromethane as solvent afforded the desired quianzolinone derivative in good yield (**Scheme 4**).[23]



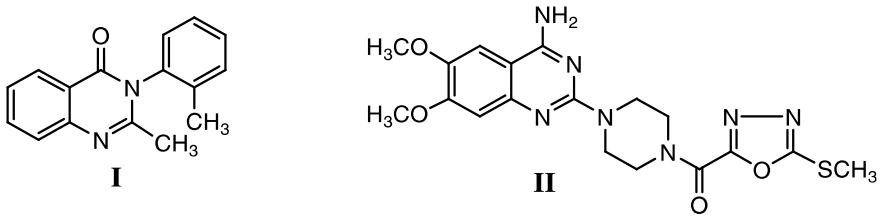
Scheme - 4

In our one-pot addition-cyclization, yields are high particularly with N^1-unsubstituted anthranilic esters; however with N^1-substituted one, e.g. NCH_3, the reaction yield was comparatively low, *i.e.* in general with N^1-unsubstituted derivatives, the cyclization was facile. The reason can easily be understood as depicted in **Scheme 5**. The mechanism proposed for the reaction involves the addition of amine to *bis*-(benzotriazol-1-yl)methanethione *via* two different route A and B resulting in the formation of thiocarbamoylbenzotriazoles (that on elimination of BtH, in case of N^1 -unsubstitution resulted in isothiocyanates) that on addition of anthranilic ester yielded uncyclized thiourea. Further cyclization proceeds in a similar reported way,[24] which involves abstraction of a proton from the terminal amido functionality by *DBU* giving a thioureidyl anion that results in thioquinazolinones through cyclative amidation.



Scheme - 5

Because of interesting metabolic profile and ability to engage in hydrogen bonding, [1,3,4]-oxadiazoles and thiadiazole are attractive pharmacophores that commonly utilized as scaffold in medicinal chemistry. 2-Amino- [1,3,4]-oxadiazoles itself have demonstrated broad spectrum of biological activity including muscle relaxants and antiomitotics. Tiodazosin, a hybrid of quinazoline and [1,3,4]-oxadiazole heterocycles has been marketed as antihypertensive agents (Figure 4).[25] Compound having thiazole ring also possessing a wide spectrum of biological activity.



I **II**

Figure - 4 Structure of Methaqualone (**I**) and Tiodazosin (**II**)

In recent years microwave assisted chemistry has become an emerging tool for the synthesis of diverse range of molecules for medicinal interest over the conventional method. In this relevance, we turned our attention to *bis*-(benzotriazolyl)-methanethione and amidine base *DABCO*, and developed a simple and direct one-pot procedure that was compatible with other heterocyclic ring and substituents. Thus, *MW* irradiation of methyl anthranilate, *bis*-(benzotriazol-1-yl)methanethione, and 5-Phenyl-[1,3,4] thiadiazol-2-yl-amine in presence of *DABCO* as catalyst afforded the compound in good yield, but that could not be isolated free from *DABCO* after elution with 20% EtOAc:*n*-hexane through silica gel column (**Figure 5**).

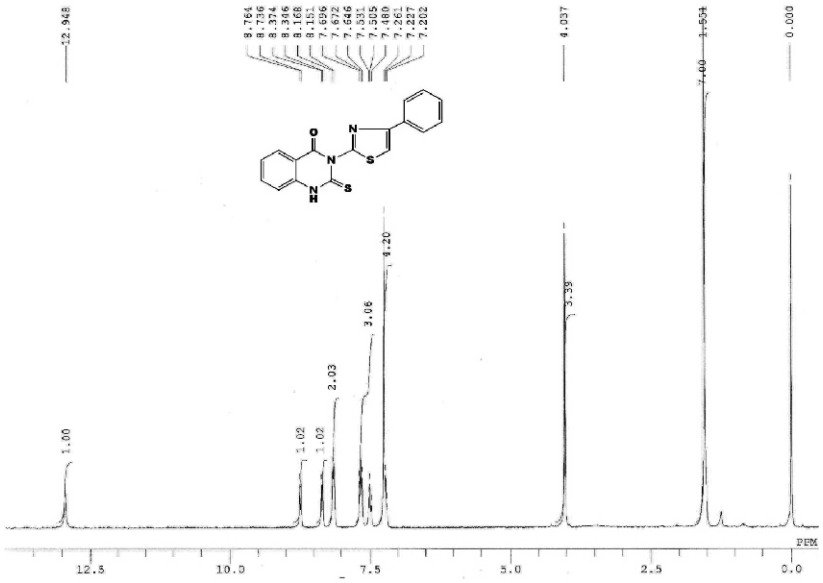
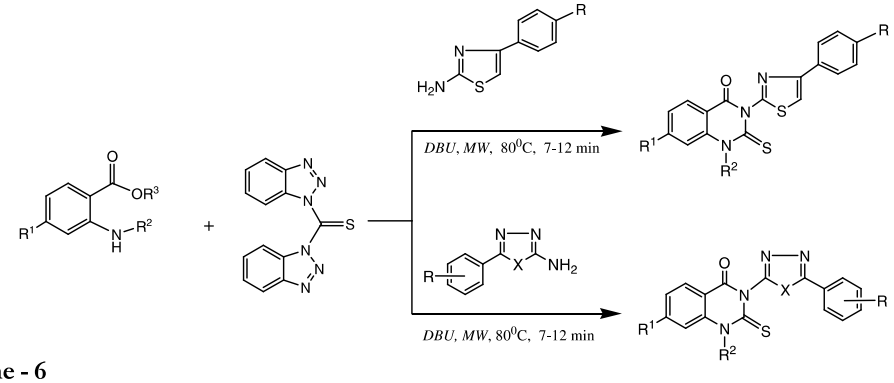


Figure - 5 ^{1}H NMR of synthesized quinazolinone (*DABCO* catalyzed reaction)

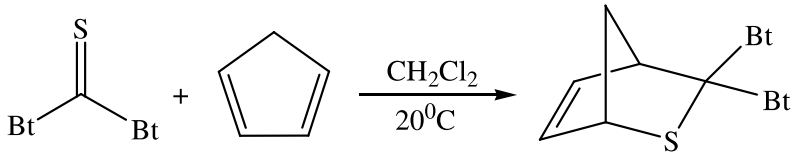
We reported a microwave-assisted simple, convenient, and high yielding synthetic methodology for the diverse thioquinozolinone by the amidine base catalyzed one-pot reaction of anthranilate ester, *bis*-(benzotriazolyl)-methanethione, and heterocyclic amines containing thiazole, [1,3,4]-oxadiazoles and [1,3,4]- thiadiazole heterocycles (**Scheme 6**). [26]



Scheme - 6

Diels-Alder Addition of bis(benzotriazole-1-yl)methanethione

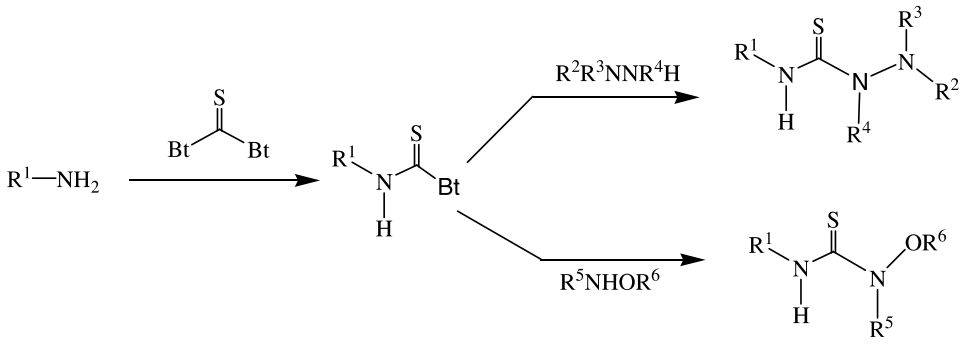
Diels-Alder Addition of bis(benzotriazole-1-yl)methanethione to cyclopentadiene provides the moisture-stable crystalline adduct in excellent yield, which can be store at room temperature for several months without any deterioration like its thiophasgene analogue which are unstable and decompose to black tar if not kept at dry ice temperature.[27] This crystalline adduct is a very useful precursor for the synthesis of *cis*-3, 5-fused mercapto esters.



Scheme - 7

Synthesis of Thiosemicarbazides and *N*-hydroxythioureas

Thiosemicarbazides possess a wide range of interesting and important biochemical and pharmaceutical properties. Thiosemicarbazides act as a building block for the synthesis of diverse range of heterocycles possessing a broad spectrum of biological activities.[28] *S*-methyl-*N*-hydroxyisothiourea is known to inhibit nitrous oxide syntheses (NOS).[29] By using bis(benzotriazole-1-yl)methanethione as a precursor with reaction of the appropriate hydrazine and corresponding hydroxylamine, we can also synthesized Thiosemicarbazides and *N*-hydroxythioureas of diverse substitution patterns in excellent yields respectively **(Scheme 8)**.[30]

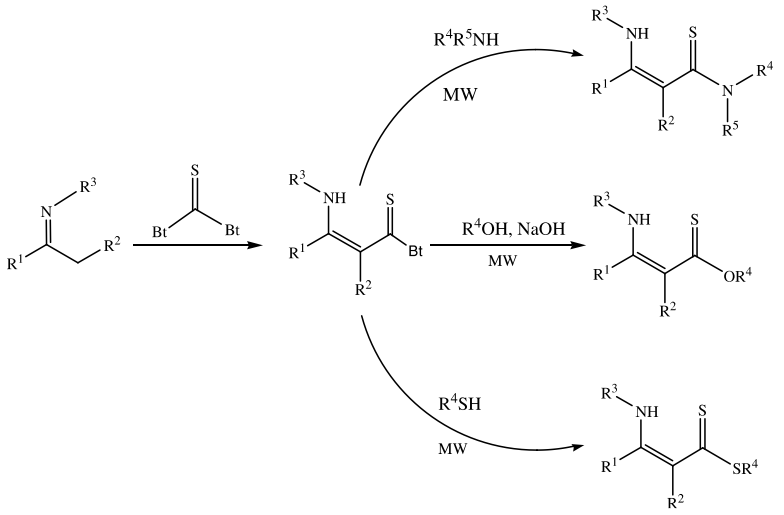


Scheme - 8

Synthesis of *â*-enamino thioic acid derivatives

â-Enaminothioic acids are important synthetic building block for the synthesis of diverse heterocycles like pyrazole, 4-aminoquinolines, dihydrothiopyrans, thiazoline, thiazolin-4-one, 1,3-thiazolin-4-one, 6H-1,3-thiazines, *â*-keto thioic acid derivatives, as well as useful precursors for liquid crystals.[31-33]

Reaction of bis(benzotriazole-1-yl)methanethione **(1)** with imines gives air stable benzotriazolyl *â*-enaminothione and this benzotriazolyl â-enaminothione which proved their utilization as a very useful reagent for simple and efficient preparation of â-enamino thioic acid derivatives (thioamides, thioesters and dithioesters) in considerable yields *via* microwave mediated nucleophilic substitution of the benzotriazolyl moiety. Reaction of benzotriazolyl *â*-enaminothione with secondary amines gave *â*-enamino thioamide, with alcohols or thiols in the presence of sodium or potassium hydroxide give thioesters and dithioesters, respectively (**Scheme 9**).[34]

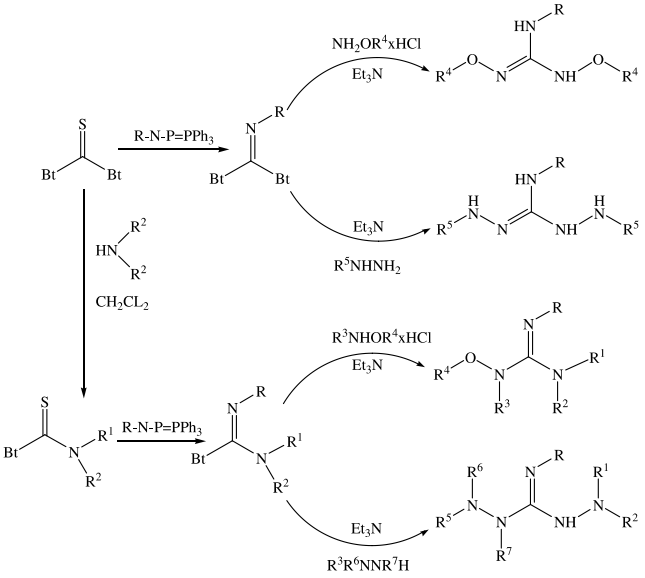


Scheme - 9

Synthesis of di-*N*-hydroxy and *N*-aminoguanidines

Guanidines are not only synthetically important unit combines *p*-donor and *p*-acceptor nitrogens but also biologically important structural units which show anti-tumor properties in Guanidino-containing drugs such as MIBG and MGBG.[35] So guanidine containing drug's provide a new hope for the treatment of many metabolic diseases, cancer, cardiovascular diseases, and diabetes.[36]

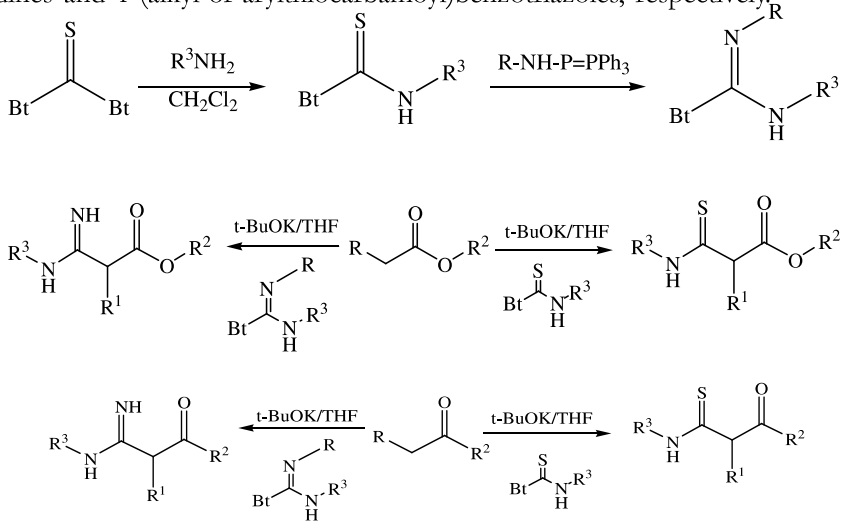
Reagents of classes (bis-benzotriazol-1-yl-methylene)amines**,** benzotriazole-1-carboxamidines and benzotriazole-1-carboximidamides that can readily react with diverse hydroxylamine and hydrazine giving mono, symmetrical di-*N*-hydroxy- and *N*-aminoguanidines with different substitution patterns in good yields can be synthesized by using bis(benzotriazole-1-yl)methanethione.[37]



Scheme - 10

C-Aminoimidoylated and *C*-thiocarbamoylated

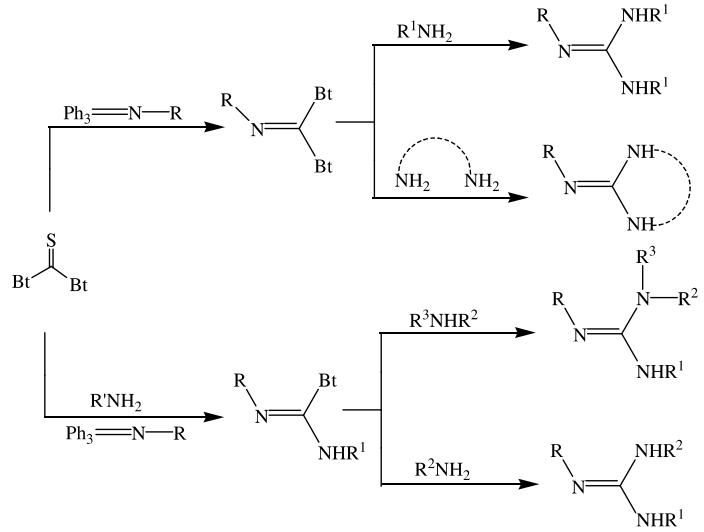
Esters, sulfones, and ketones were *C*-aminoimidoylated and *C*-thiocarbamoylated with the class of reagents obtained from bis(benzotriazole-1-yl)methanethione, benzotriazole-1-carboxamidines and 1-(alkyl-or-arylthiocarbamoyl)benzotriazoles, respectively.[38]



Scheme 11

Synthesis of 1, 2, 3, trisubstituted guanidine

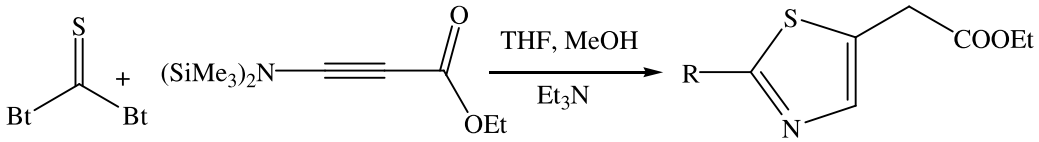
Several pharmacologically significant natural products containing guanidine skeleton have been isolated from plants and other microorganisms.[39] Katritzky *et al* reported a high yielding synthesis of acyclic and cyclic 1,2,3, trisubstituted guanidine with a different method for the guanylation of various primary and secondary amines by the use of new class of reagents (bis-benzotriazol-1-yl-methylene)amines and benzotriazole-1-carboxamidines and these reagents are prepared by bis(benzotriazole-1-yl)methanethione (**Scheme 12**).[40]



Scheme 12

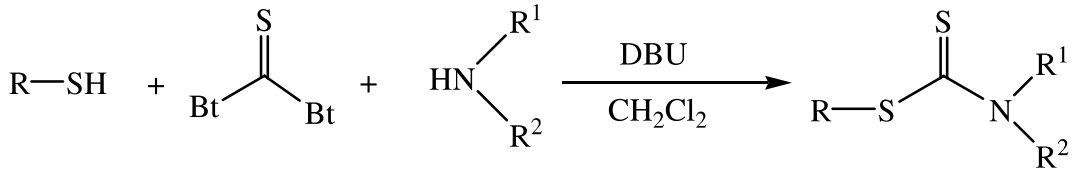
Synthesis of thiazole derivatives

Thiazole nucleus not only plays a very important role in metabolism, but also known for a diverse range of pharmacological effects. Sasmal et al have demonstrated a bis(benzotriazole-1-yl)methanethione mediated one-pot efficient methodology for the synthesis of thiozol ring *via* N-desilylation, thioacylation followed by cycloisomerisation in an intramolecular thia-Michael fashion (**Scheme 13**).[41]



Scheme 13

Bt-mediated synthesis of Dithiocarbamates: Organic dithiocarbamates (*DTC*s) have received much attention by synthetic and medicinal chemists due to their interesting chemistry and diverse pharmacological properties. The *DTC* framework is ubiquitously found in a variety of biologically active molecules and it gained importance as building blocks, combinatorial scaffold, as well as intermediates in organic synthesis to develop new active chemical entities (NCE's). [42] In spite of the growing interest in applications of these compounds, preparative methods available for their synthesis are still limited. Most of the synthetic methods are associated with one or the other limitations including low availability of starting material, employment of harsh reaction conditions, high reaction temperatures, long reaction times, low yields, and more over required two or more steps. Recently we have developed a convenient and high yielding method for the synthesis of diverse dithiocarbamates having various substituents including alkyl, aryl, heteroaryl, and alkylaryl at the thiol chain or at the amine chain or at both thiol and amine chains by the one-pot reaction of mercaptans, amines, and *bis*-(benzotriazolyl)-methanethione in presence of amidine base under mild reaction conditions (Scheme **14**).[43]



Scheme 14

The above described methodology has been successfully applied for the synthesis of N/S glycosyl dithiocarbamates.[44] Structures of some representative glycosyl dithiocarbamates synthesized by our reported one-pot benzotriazole mediated methodology are given below:

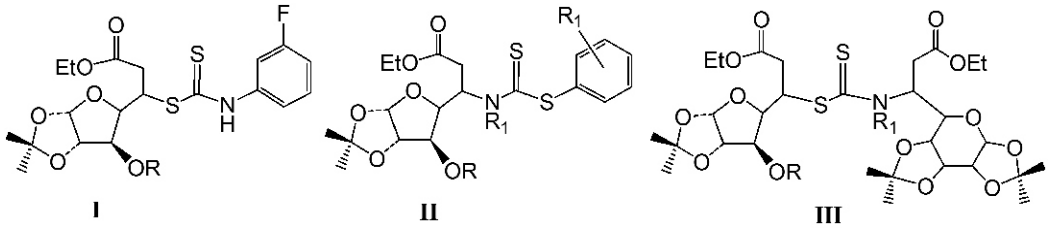
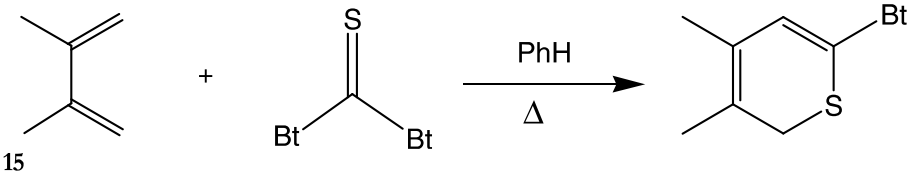


Figure - 6 Some representative glycosyl DTC

Bis(benzotriazole-1-yl)methanethione mediated cycloaddition

It is found that thiocarbonyl transfer reagents undergo ready cycloaddition with a variety of systems gives stable crystalline solid rather then unstable adduct formed by its parent thiophosgene.[45] Bis(benzotriazole-1-yl)methanethione on reaction with 2, 3 dimethyl-1, 3,-butadiene gave 3,4-dimethyl-6-(benzotriazol-1-yl)-2H-thiapyran.[46]



Scheme 15

Conclusion

Present review is focused on preparation and vast synthetic applications of bis(benzotriazole-1-yl)methanethione, that has been shown advantageous over thiophosgene as being more effective equivalent in numerous important chemical reactions and more over due to non-toxic nature of benzotriazole containing molecules. It plays significant role in several chemical reactions particularly for the synthesis of thiourea, *N*- hydroxythiourea, thiosemicarbazide, triazoles, thiozoles, guanidines, *N*-hydroxy-*N*-amino guanidines, *â*-enamino thioic acids, diverse dithiocarbamates, and quinazolinones etc. This synthetic auxiliary may serve as a synthetic key, structural, and functional tool for future synthetic organic chemistry.

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References

1. AR Katritzky, X Lan, JZ Yang, OV Denisko, *Chem. Rev.*, **1998**, *98*, 409.
2. AR Katritzky, K Manju, SK Singh, NK Meher, *Tetrahedron*, **2005**, *61*, 2555.
3. AR Katritzky, *Il. Farmaco.*, **1988**, *12*, 1175.
4. AR Katritzky, S Rachwal, GJ Hitchings, *Tetrahedron*, **1991**, *47*, 2683.
5. AR Katritzky, *Bull. Soc. Chim. Belg.*, **1992**, *101*, 409.

6. AR Katritzky, Z Yang, DJ Cundy, *Aldrichimica Acta*, **1994**, *27*, 31.
7. AR Katritzky, X Lan, *Chem. Soc. Rev.*, **1994**, *23*, 363.
8. AR Katritzky, X Lan, WQ Fan, *Synthesis*, **1994**, 445.
9. AR Katritzky, DC Oniciu, *Bull. Soc. Chim. Belg.*, **1996**, *105*, 635.
10. AR Katritzky, ZQ Wang, M Tsikolia, CD Hall, M. Carman, *Tetrahedron Letter*, **2006**, *43*, 7653.
11. AR Katritzky, MS Kim, K Widyan, *ARKIVOC*, **2008**, *(iii)*, 91.
12. PA Wender, SMC Touami, UC Philip, *J. Am. Chem. Soc.*, **1996**, *118*, 6522.
13. RE Orth, SJ Soedigdo, *Heterocycl. Chem.*, **1965**, *2*, 486.
14. AR Katritzky, RM Witek, VR Garcia, PP Mohapatra, JW Rogers, J Cusido, AAA Abdel-Fattah, PJ Steel, *J. Org. Chem.*, **2005**, *70*, 7866.
15. S Sharma, *Synthesis*, **1978**, 804.
16. J Smith, JL Liras, SE Schneider, EV Anslyn, *J. Org. Chem.*, **1996,** *61*, 8811; b) DC Schroeder, *Chem. Rev.*, **1955**, *55*, 181.
17. F Duus, *In Comprehensive Organic Chemistry*; Jones, N. J., Ed.; Pergamon Press: Oxford, **1979**, *3*, 465.
18. AR Katritzky, S Ledoux, RM Witek, SK Nair, *J. Org Chem.*, **2004**, *69*, 2976.
19. IK Kacker, SH Zaheer, *J. Ind. Chem. Soc.*, **1951**, *28*, 344.
20. AH Amin, DR Mehta, SS Samarth, *Prog. Drug. Res.*, **1970**, *14*, 218.
21. SB Mhaske, NP Argade, *Tetrahedron*, **2006**, *62*, 9787.
22. VK Tiwari, DD Singh, HA Hussain, BB Mishra, A Singh, *Monatsh. Chem.,* **2008**, *139*, 43.
23. VK Tiwari, HA Hussain, BB Mishra, DD Singh, VJ Tripathi, *Med. Chem. Res.*, **2006**, *15*, 325.
24. N Tewari, RC Mishra, VK Tiwari, RP Tripathi, *Synlett.,* **2002**, *11*, 1779.
25. a) RA Partyka, RR Crenshaw, U.S. Patent 4001238, **1977**; (b) S Vardan, S Mookherjee, R Eich, *Clin. Pharm. Ther.* **1983**, *34*, 290.
26. VK Tiwari, RR Kale, BB Mishra, A Singh, *ARKIVOC*, **2008**, *(xiv)*, 27.
27. C Larsen, DN Harpp, *J. Org. Chem.*, **1980**, *45*, 3713.
28. (a) JC Kappel, TS Yokum, GJ Barany, *Comb. Chem.*, **2004**, *6*, 746; (b) CB Chapleo, PL Myers, ACB Smith, MR Stillings, IF Tulloch, DS Walter, *J. Med. Chem.*, **1988**, *31*, 7; (d) G Clifton, SR Bryant, CG Skinner, *J. Med. Chem.*, **1970**, *13*, 377.
29. K Ichimori, DJ Stuehr, *J. Med. Chem.*, **1999**, *42*, 1842.
30. AR Katritzky, NM Khashab, AV Gromova, *ARKIVOC*, **2006**, *(iii)*, 226.
31. H Al-Badri, N Collignon, J Maddalino, S Masson, *Tetrahedron*, **2000**, *56*, 3909.
32. S Coen, B Ragonnet, C Vieillescazes, J Roggero, *Heterocycles*, **1985**, *23*, 1225.
33. C Cativiela, JL Serrano, MM Zurbano, *J. Org. Chem.*, **1995**, *60*, 3074.
34. AR Katritzky, H Tao, K Kirichenko, *ARKIVOC*, **2007**, *(x)*, 142.

35. S Ekelund, P Nygren, R Larsson, *Biochem. Pharm.*, **2001**, *61*, 1183.

36. AR Katritzky, V Rogovoy, *ARKIVOC,* **2005**, *(iv)*, 49.

37. AR Katritzky, NM Khashab, S Bobrov, M Yoshioka, *J. Org. Chem.*, **2006**, *71*, 6753.

38. AR Katritzky, NM Khashab, DN Haase, M Yoshioka, I Ghiviriga, PJ Steel, *J Org. Chem.*, **2007**, *72* ,67.

39. (a) RGS Berlinck, *Nat. Prod. Rep.*, **1999**, *16*, 339; b) L Heys, CG Moore, PJ Murphy, *J. Chem. Soc. Rev.*, **2000**, *29*, 57.

40. AR Katritzky, NM Khashab, S Bobrov, *Helvetica Chimica Acta*, **2005,** *88*, 1664.

41. PK Sasmal, S Sridhar, J Iqbal, *Tetrahedron Letters*, **2006,** *47*, 8661.

42. (a) AK Mukherjee, R Ashare, *Chem. Rev.*, **1991,** 91, 1; (b) M Dhooghe, N De Kime, *Tetrahedron*, **2006,** *62*, 513.

43. VK Tiwari, A Singh, HA Hussain, BB Mishra, VJ Tripathi, *Monatsh. Chem.*, **2007**, *138*, 653.

44. A Singh, RR Kale, VK Tiwari, *Trends Carbohydrate Res.*, **2009**, *1*, 80.

45. HJ Reich, JE Trend, *J. Org. Chem.*, **1973,** *38*, 2637.

46. DN Harpp, JG Macdonald, C Larsen, *Can. J. Chem.*, **1985**, *63*, 951.

RELATIONSHIP BETWEEN WAVE FUNCTIONS OF TWO-DIMENSIONAL HYDROGEN ATOM IN PARABOLIC AND POLAR COORDINATES

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Abstract

We show, for the two-dimensional hydrogen atom, the relationship between its wave functions in polar and parabolic coordinates.

Keywords

Two-dimensional hydrogen atom; polar and parabolic coordinates

Introduction.

The Schrödinger equation for bounded states of the hydrogen atom in two dimensions:

$$-\frac{\hbar^2}{2M}\nabla^2\psi-\frac{\tilde{k}}{r}\psi=E\psi \tag{1}$$

has the following normalized wave functions in polar coordinates (r,φ) [1,2]:

$$\psi_{lm}(r,\varphi)=\frac{2p_0}{\hbar}(-i)^m\left[\frac{(l-|m|)!}{2\pi(2l+1)(l+|m|)!}\right]^{1/2}e^{-\frac{p_0 r}{\hbar}}\cdot\left(\frac{2p_0 r}{\hbar}\right)^{|m|}L_{l-|m|}^{2|m|}\left(\frac{2p_0 r}{\hbar}\right)e^{im\varphi}\ , \tag{2}$$

where $p_0=\sqrt{-2ME}=\dfrac{2M\tilde{K}}{\hbar(2l+1)}$ $,l=0,1,...;m=0,\pm1,...,\pm l$ *and* L_b^a are the associated Laguerre polynomials [3-5].

In parabolic coordinates (*u,v*) defined by:

$$x=\frac{1}{2}\left(u^2-v^2\right)\quad,\quad y=uv\quad, \tag{3}$$

The normalized solutions of (1) are given by [1,2]:

$$\tilde{\psi}_{lq}(u,v)=\frac{p_0\,i^{l-q}e^{-\frac{p_0}{2\hbar}(u^2+v^2)}H_{l+q}\left(\sqrt{\frac{p_0}{\hbar}}\,u\right)H_{l-q}\left(\sqrt{\frac{p_0}{\hbar}}\,v\right)}{\hbar\left[2^{2l-1}\pi\,(2l+1)(l+q)!(l-q)!\right]^{1/2}} \tag{4}$$

where $q=0,\pm1,...,\pm l$ and the H_n are the Hermite polynomials [3-5].

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The problem is to express (2) in terms of (4), which it is resolved in [6] employing non-trivial relations from group theory, with the following answer:

$$\psi_{l\,m}(r,\varphi) = i^m \sum_{q=-l}^{l} (-1)^q\, d^{\,l}_{qm}\,(-\frac{\pi}{2})\,\tilde{\psi}_{l\,q}(u,v) \tag{5}$$

such that [6,7]

$$d^{\,l}_{qm}(\theta) = \left[(l+m)!(l-m)!(l+q)!(l-q)!\right]^{1/2} \cdot \sum_{k=0}^{l} \frac{(-1)^k\,(Sen\frac{\theta}{2})^{m-q+2k}\,(Cos\frac{\theta}{2})^{2l-m+q-2k}}{k!(l+q-k)!(l-m-k)!(m-q+k)!} \tag{6}$$

In the next Sec. we shall show –for special values of *m*- expressions between ψ and $\tilde{\psi}$ (alternative ones to (5)) which can be obtained without using group theory. In fact, it is sufficient to use known relations for the Laguerre and Hermite polynomials; our procedure accepts easy generalization to arbitrary values of parameter *m*.

Relationship between polar and parabolic wave functions..

When we search for writing ψ_{lm} in terms of $\tilde{\psi}_{lq}$ it results that the identity [5]:

$$L_a\left(\xi^{\,2}+\eta^{\,2}\right) = \frac{(-1)^a}{2^{2a}} \sum_{k=0}^{a} \frac{H_{2k}(\xi)\,H_{2a-2k}(\eta)}{k!(a-k)!} \tag{7}$$

is basic in our process, which we illustrate in two cases:

a).- $\underline{m=0}$.

From (2) we have the following solutions for arbitrary l:

$$\psi_{l0} = \frac{p_0}{\hbar}\left[\frac{2}{\pi(2l+1)}\right]^{1/2} e^{-\frac{p_0 r}{\hbar}}\, L_l\left(\frac{2p_0 r}{\hbar}\right), \tag{8}$$

there we put $r = \frac{1}{2}\left(u^2+v^2\right)$, then we employ (7) with $a=l$, $\xi = \sqrt{\frac{p_0}{\hbar}}\,u$, $\eta = \sqrt{\frac{p_0}{\hbar}}\,v$ and we remember (4) to deduce the following expression ($\Gamma(z)$ denotes the gamma function) :

$$\psi_{\,l0} = \frac{(-1)^l}{2^{2l}} \sum_{q=-l}^{l} \frac{\sqrt{(l+q)!(l-q)!}}{\Gamma\left(\frac{l+q}{2}+1\right)\Gamma\left(\frac{l-q}{2}+1\right)}\, Cos\left[\frac{(q-l)\pi}{2}\right]\tilde{\psi}_{\,l\,q}\ , \tag{9}$$

much more simple in computations than the corresponding relation obtained from (5) for $m=0$; in (9) it is clear that $\Gamma\left(\frac{l\pm q}{2}+1\right) = \left(\frac{l\pm q}{2}\right)!$ when $\left(\frac{l\pm q}{2}\right)$ is an integer.

For example, (9) implies that $\psi_{\,00} = \tilde{\psi}_{\,00}$ and :

$$\psi_{10}=\frac{1}{\sqrt{2}}\left(\tilde{\psi}_{1-1}-\tilde{\psi}_{11}\right)\quad,\quad \psi_{20}=\frac{1}{2}\left[\sqrt{\frac{3}{2}}\left(\tilde{\psi}_{22}+\tilde{\psi}_{2-2}\right)-\tilde{\psi}_{20}\right]\quad,$$

$$\psi_{30}=\frac{1}{4}\left[\sqrt{5}\left(\tilde{\psi}_{3-3}-\tilde{\psi}_{33}\right)+\sqrt{3}\left(\tilde{\psi}_{31}-\tilde{\psi}_{3-1}\right)\right]\quad,\ \text{etc.} \tag{10}$$

in accordance with (5).

b).- $\underline{m=1}$.

The equation (2) gives us the wave functions :

$$\psi_{l1}=-2i\left(\frac{p_0}{\hbar}\right)^2\left[\frac{2}{\pi(2l+1)(l+1)l}\right]^{1/2} r\, e^{-\frac{p_0 r}{\hbar}} L^2_{l-1}\left(\frac{2p_0 r}{\hbar}\right) e^{\,i\varphi} \tag{11}$$

where we employ $e^{i\varphi}=\frac{1}{2r}(u+iv)^2$ and the same expressions for r, ξ, η as used in ψ_{l0}, resulting thus that:

$$\psi_{l1}=-i\frac{p_0}{\hbar}\left[\frac{2}{\pi(2l+1)(l+1)l}\right]^{1/2} e^{-\frac{p_0 r}{\hbar}}(\xi+i\eta)^2 L^2_{l-1}\left(\xi^2+\eta^2\right) \tag{12}$$

On the other hand, by repeated partial differentiation of (7) with respect to ξ and/or η for $a = l + 1$, and the use of the known properties [5]:

$$\frac{d}{dz}H_n(z)=2n\cdot H_{n-1}(z)\,,\ \frac{d}{dz}L^a_n(z)=-L^{a+1}_{n-1}(z) \tag{13}$$

It is easy to show the interesting identity:

$$(\xi+i\eta)^2 L^2_{l-1}\left(\xi^2+\eta^2\right)=\frac{(-1)^l}{2^{2l}}\sum_{q=0}^{l}\frac{1}{q!(l-q)!}\left[(l-2q)\cdot H_{2q}(\xi)H_{2l-2q}(\eta)-2i(l-q)H_{2q+1}(\xi)H_{2l-2q-1}(\eta)\right] \tag{14}$$

which jointly with (4) and (12) lead to the expansion:

$$\psi_{l1}=\frac{i(-1)^l}{2^l\sqrt{l(l+1)}}\sum_{q=-l}^{l}\sqrt{(l+q)!(l-q)!}\cdot\left[\frac{q\,Cos\left(\frac{q-l}{2}\pi\right)}{\Gamma\left(\frac{l+q}{2}+1\right)\Gamma\left(\frac{l-q}{2}+1\right)}+\frac{2(-1)^{q-l}\,Sen\left(\frac{q-l}{2}\pi\right)}{\Gamma\left(\frac{l+q-1}{2}+1\right)\Gamma\left(\frac{l-q-1}{2}+1\right)}\right]\tilde{\psi}_{lq} \tag{15}$$

which is more economical – in calculations - than (5) for *m=1*. Then (15) implies:

$$\psi_{11}=-i\left[\tfrac{1}{2}\left(\tilde{\psi}_{1-1}+\tilde{\psi}_{11}\right)+\tfrac{1}{\sqrt{2}}\tilde{\psi}_{10}\right],\qquad \psi_{21}=+\tfrac{i}{2}\left(\tilde{\psi}_{22}-\tilde{\psi}_{2-2}+\tilde{\psi}_{21}-\tilde{\psi}_{2-1}\right),\ \text{etc.} \tag{16}$$

Similarly, with (7) for $a = l+2$ we can obtain an expression for $(\xi+i\eta)^4\ L^4_{l-2}(\xi^2+\eta^2)$ and then to

deduce ψ_{l2} in terms of the $\tilde{\psi}_{lq}$, and so on; therefore, our method admits application for any value of m. From (2) we have that $\psi_{l-m} = \overline{\psi}_{lm}$, implying that it is only necessary to develop expressions for $m \geq 0$

References

1. Ch. S. Johnson Jr. and L.G. Pedersen, Problems and solutions in quantum Chemistry and Physics, Dover NY (1986)
2. A.T. Fromhold Jr., Quantum mechanics for applied Physics and Engineering, Dover NY (1991)
3. J. López-Bonilla, A. Lucas-Bravo and S. Vidal-Beltrán, Proc. Pakistan Acad. Sci. 42 , No.1 (2005) 63-65
4. C. Lanczos, Linear differential operators, D. Van Nostrand, London (1961)
5. N.N. Lebedev, Special functions and their applications, Dover, NY (1972)
6. G.F. Torres del Castillo and A. López-Villanueva, Rev. Mex. Fís. 47, No.2 (2001) 123-127
7. J. D. Talman, Special functions: A Group theoretic approach, W.A. Benjamin Inc. NY (1968)

ASCENT AND DESCENT OF PRODUCT AND SUM OF TWO COMPOSITION OPERATORS ON ℓ^p SPACES

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Abstract

Let l^p $(1 \leq p \leq \infty)$ be the Banach space of all p-summable sequences (bounded sequences for $p = \infty$) of complex numbers under the standard p-norm on it and $C\phi$ be a composition operator on l^p induced by a function ϕ on N into itself. In this paper we discuss the ascent and descent of Product and Sum of two composition operators on l^p spaces.

Introduction

Let X denote an arbitrary vector space and T be a linear operator on X. Let D(T), N(T) and R(T) denote domain, kernel and range of T respectively. Let N denote the set of natural numbers. The following statements and definitions are relevant and instructive in our context.

Theorem 1.1. $N(T^n) \subseteq N(T^{n+1})$; $n = 0, 1, 2, \ldots$. If $N(T^k) = N(T^{k+1})$ for some k, then $N(T^k) = N(T^n)$ when $n \geq k$.

Definition 1.1. If there is some integer $n \geq 0$ such that $N(T^n) = N(T^{n+1})$, the smallest such integer is called the ascent of T and is denoted by a(T). If no such integer exists we say that $a(T) = \infty$.

Theorem 1.2. $R(T^{n+1}) \subseteq R(T^n)$; $n = 0, 1, 2 \ldots$. If $R(T^{k+1}) = R(T^k)$ for some k, then $R(T^n) = R(T^k)$ when $n \geq k$.

Definition 1.2. If there is some integer $n \geq 0$ such that $R(T^{n+1}) = R(T^n)$, the smallest such integer is called the descent of T and is denoted by d(T). If no such integer exists we say that $d(T) = \infty$.

Theorem 1.3. If a(T) is finite and d(T) = 0, then a(T) = 0.

Theorem 1.4. If a(T) and d(T) are both finite, then necessarily $a(T) \leq d(T)$.

Theorem 1.5. If D(T) = X and the ascent and descent of T are both finite, they are equal.

Composition operators on l^p spaces

The composition operator $C\phi$ on l^p induced by a function ϕ on N into itself is defined by $C_\phi(f) = f.\phi$ for all $f \in l^p$. It is well known that a necessary and sufficient condition for a function ϕ on N into itself to induce a composition operator on l^p is that the set $\{ |\phi^{-1}(n)| : n \}$ is bounded. Here $|\phi^{-1}(n)|$ denotes the number of elements in $\phi^{-1}(n)$; (see [20] and [24]).

The study of ascent and descent of an operator has been done as a part of spectral properties of an operator (see [1], [2], [5] and [6]). Since composition operators provide diverse and illuminating examples of operators which leads to useful insight into structure theory of operators, it is desirable to study ascent and descent of these operators and their sum and product.

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Examples 2

We now give different examples of product of two composition operators which is given below:

Example 2.1. Let ϕ be a self-map on defined as follows.

$$\phi(n)=\begin{cases}1, & if \quad n=1.2\\ n-1, & if \quad n>2\end{cases}$$

Here a(Cϕ) = 0 and d(Cϕ) = ∞. Clearly CϕCφ ≠ CφCϕ

Let φ be a self-map on **N** defined as follows.

$$\varphi(n)=\begin{cases}n, & if\ n\ is\ odd\\ n-1, & if\ n\ is\ even.\end{cases}$$

Here a(Cφ) = 1 = d(Cφ). Clearly Cϕ Cφ ≠ CφCϕ.

But a(Cϕ Cφ) = 1 = d(Cϕ Cφ) and a(CφCϕ) = 1 = d(CφCϕ).

Ascent and Descent of Sum and Product of two Composition Operators

Example 2.2. Let ϕ be a self-map on **N** defined as follows.

$$\phi(n)=\begin{cases}n, & if\ n\ is\ odd\\ n-1, & if\ n\ is\ even.\end{cases}$$

Here a(Cϕ) = 1= d(Cϕ).

Let φbe a self-map on **N** defined as follows.

$$\varphi(n)=\begin{cases}1, & if \quad n=1.2\\ 2, & if\ n=3\\ n+1 & if \quad n>3.\end{cases}$$

Here a(Cφ) = ∞ = d(Cφ). Clearly CϕCφ ≠ CφCϕ

But a(CϕCφ) = 2 = d(CϕCφ) and a(CφCϕ) =1= d(CφCϕ).

Example 2.3. Let ϕ be a self-map on N defined as follows.

$$\phi(n)=\begin{cases}1, & if \quad n=1.2\\ 2, & if \quad n=3\\ n+1, & if \quad n\in\{5.7.9.....\}\\ n-1, & if \quad n\in\{4.6.8.....).\end{cases}$$

Here a(Cϕ) = 3 = d(Cϕ).

Let φbe a self-map on **N** defined as follows.

$$\varphi(n) = n + 2, \text{ if } n \text{ is odd}$$

and

$$\varphi(2n\text{-}2) = \varphi(n) = n, \text{ if } n \in \{2,4,6,8,10.....\}.$$

Here a(Cφ) = ∞ = d(Cφ). Clearly CϕCφ ≠ CφCϕ

But a(CϕCφ) = 1 = d(CϕCφ) and a(CφCϕ) =1= d(CφCϕ).

Example 2.4. Let ϕ be a self-map on N defined as follows.

$$\phi(n) = \begin{cases} n, & \text{if } n \text{ is odd} \\ n-1, & \text{if } n \text{ is even.} \end{cases}$$

Here a(Cϕ) = 1 = d(Cϕ).

Let φ be a self-map on N defined as follows.

$$\phi(n) = \begin{cases} n, & \text{if } n \text{ is odd} \\ n-1, & \text{if } n \text{ is even.} \end{cases}$$

Here a(Cφ) = 0 = d(Cφ). Clearly CϕCφ≠ CφCϕ

But a(CϕCφ) = 1 = d(CϕCφ) and a(CφCϕ) =1= d(CφCϕ).

Example 2.5. Let ϕ be a self-map on N defined as follows.

$$\phi \text{ (s)} = \text{s -1,if s} \in \{2,3,.....,n\}$$

and

$$\phi \text{ (t)} = \text{t, if t} \in \mathbf{N}\text{- } \{2,3,.....,n\}.$$

Here a(Cϕ) = 0 = d(Cϕ).

Let φbe a self-map on N defined as follows.

$$\varphi(t) = t, \ \forall t \in N$$

Here a(Cφ) = 0 = d(Cφ). Clearly CϕCφ = CφCϕ

But a(CϕCφ) = 0 = d(CϕCφ) and a(CφCϕ) = 0 = d(CφCϕ).

3. RESULTS

In this section we give a characterization of product of two composition operators on l^p spaces.

Theorem 3.1. Let Cϕ and Cφ be two composition operators on l^p spaces and CϕCφ= CφCϕ. Then the following results hold.

(i) a(CϕCφ) ≤ Max {a(Cϕ), a(Cφ)} (ii) d(CϕCφ) ≤ Max {d(Cϕ), d(Cφ)}.

Proof. (i) Case-I: If a(Cϕ) = ∞ or a(Cφ) = ∞. Then result (i) is obviously true.

Case-II: If a(Cϕ) = m < ∞ and a(Cφ) = n < ∞. This implies that $R(\phi^m) = R(\phi^{m+1})$ and $R(\varphi^n) = R(\varphi^{n+1})$. Now suppose n > m, Max {m, n} = n. We claim that $N((C\phi C\varphi)^{n+1}) = N((C\phi C\varphi)^n)$. Let f ∈ $N((C\phi C\varphi)^{n+1})$. This implies that $(C\phi C\varphi)^{n+1}(f) = 0$. Since CϕCφ = CφCϕ.

then $(C\phi^{n+1} C\varphi^{n+1})(f) = 0$. This implies that $f(\varphi^{n+1}\phi^{n+1}) = 0$. Since $R(\phi^m) = R(\phi^{m+1})$ and $R(\varphi^n) = R(\varphi^{n+1})$. Hence $f(\varphi^n \phi^n) = 0$. Therefore $(C\phi C\varphi)^n (f) = 0$. Thus $f \in N((C\phi C\varphi)^n)$. Thus

$$N((C\phi C\varphi)^{n+1}) \subseteq N((C\phi C\varphi)^n). \tag{1}$$

Since it is obvious that

$$N((C\phi C\varphi)^n) \subseteq N((C\phi C\varphi)^{n+1}). \tag{2}$$

Combining equations (1) and (2), we get

$$N((C\phi C\varphi)^{n+1}) = N((C\phi C\varphi)^n).$$

Hence $a(C\phi C\varphi) \leq n = \text{Max}\{a(C\phi), a(C\varphi)\}$.

(ii) Case-I: If $d(C\phi) = \infty$ or $d(C\varphi) = \infty$. Then result (ii) is obviously true.
Case-II: If $d(C\phi) = m < \infty$ and $d(C\varphi) = n < \infty$. This implies that $\phi : R(\phi^m) \to R(\phi^m)$ is injective and $\varphi : R(\varphi^m) \to R(\varphi^m)$ is injective. Since $C\phi C\varphi = C\varphi C\phi$ then $\phi : R(\phi^{m+i}) \to R(\phi^{m+i})$ is injective and $\varphi : R(\varphi^{m+i}) \to R(\varphi^{m+i})$ is injective for each $i \geq 1$. Now suppose $n > m$, $\text{Max}\{m, n\} = n$. We claim that $(\phi\varphi) : R((\phi\varphi)^n) \to R((\phi\varphi)^n)$ is injective. Let $k_1 \in R(\phi^n)$ and $k_2 \in R(\varphi^n)$ such that $k_1 \neq k_2$. It is given that $\phi : R(\phi^n) \to R(\phi^n)$ is injective and $\varphi : R(\varphi^n) \to R(\varphi^n)$ is injective. This implies that $\phi(k_1) \neq \phi(k_2)$ and $\varphi(k_1) \neq \varphi(k_2)$. Thus $(\phi\varphi)(k_1) \neq (\phi\varphi)(k_2)$. Therefore $(\phi\varphi) : R((\phi\varphi)^n) \to R((\phi\varphi)^n)$ is injective.

Hence $d(C\phi C\varphi) \leq n = \text{Max}\{a(C\phi), a(C\varphi)\}$. □

Theorem 3.2. $a(C\phi C\varphi) = \infty$ if and only if there exists a sequence of distinct integers $\{n_k\}$ such that $n_k \notin R((\varphi\phi)^k)$ but $n_k \in R((\varphi\phi)^{k-1})$ for each $k \geq 1$.

Proof. $C\phi C\varphi = C\phi.\varphi$ is a composition operator induced by $\varphi.\phi$ Now by theorem 3.1in [5] follows that $a(C\phi C\varphi) = \infty$ if and only if there exists a sequence of distinct integers $\{nk\}$ such that $n_k \notin R((\varphi.\phi)^k)$ but $n_k \in R((\varphi.\phi)^{k-1})$ for each $k \geq 1$. □

Theorem 3.3. $d(C\phi C\varphi) = \infty$ if and only if $(\varphi.\phi) : R((\varphi.\phi)^k) \to R((\varphi.\phi)^k)$ is not one-to-one for all $k \geq 0$.

Proof. $C\phi C\varphi = C\varphi.\phi$ is a composition operator induced by $\varphi.\phi$. Now by theorem 3.2 in [5] follows that $d(C\phi C\varphi) = \infty$ if and only if $(\varphi.\phi) : R((\varphi.\phi)^k) \to R((\varphi.\phi)^k)$ is not one-to-one for all $k \geq 0$. □

Theorem 3.4. $a_e(C\phi C\varphi) = \infty$ if and only if there exist a sequence $\{E_k\}^{\infty}k=1$ of subsets of **N** such that each E_k is infinite, $E_k \subseteq R((\varphi.\phi)^{k-1})$ and $R((\varphi.\phi)^k) \cap E_k = \phi$ for each $k \in \mathbf{N}$. □

Proof. $C\phi C\varphi = C\varphi.\phi$ is a composition operator induced by $\varphi.\phi$. Now by theorem 3.1in [6] follows that $a(C\phi C\varphi) = \infty$ if and only if there exist a sequence $\{E_k\}^{\infty}{}_{k=1}$ of subsets of **N** such that each E_k is infinite, $E_k \subseteq R((\varphi.\phi)^{k-1})$ and $R((\varphi.\phi)^k) \cap E_k = \phi$ for each $k \in \mathbf{N}$. □

Theorem 3.5. $d_e(C\phi C\varphi) = \infty$ if and only if for each $k \geq 0$; $|\phi^{-1}(n)| > 1$ for infinitely many $n \in R((\varphi.\phi)^k)$.

Proof. $C\phi C\varphi = C\varphi.\phi$ is a composition operator induced by $\varphi.\phi$. Now by theorem 3.2 in [6] follows that $de(C\phi C\varphi) = \infty$ if and only if for each $k \geq 0$; $|\phi^{-1}(n)| > 1$ for infinitely many $n \in R((\varphi.\phi)^k)$. □

4. Example

We now give different examples of sum of two composition operators which is given below :

Example 4.1. Let ϕ be a self-map on **N** defined as follows.

$$\phi(1) = 1 = \phi(2)$$

and

$$\phi(n) = n, \forall n \geq 3.$$

Here $a(C\phi) = 1 = d(C\phi)$.

Let φ be a self-map on **N** defined as follows.

$$\varphi(3) = 3 = \varphi(4)$$

and

$$\varphi(n) = n, \text{ if } n \in \mathbf{N} - \{3, 4\}.$$

Here $a(C\varphi) = 1 = d(C\varphi)$.

But $a(C\phi + C\varphi) = 0 = d(C\phi + C\varphi)$.

Example 4.2. Let ϕ be a self-map on **N** defined as follows.

$$\phi(1) = 1 = \phi(2)$$

and

$$\phi(n) = n-1, \forall n \geq 3.$$

Here $a(C\phi) = 0$ and $d(C\phi) = \infty$.

Let φ be a self-map on $\mathbf{N}$ defined as follows.

$$\varphi(n) \begin{cases} n, & \text{if } n \text{ is odd} \\ n-1, & \text{if } n \text{ is even.} \end{cases}$$

Here $a(C\varphi) = 1 = d(C\varphi)$.

But $a(C\phi + C\varphi) = 0$ and $d(C\phi + C\varphi) = \infty$.

Example 4.3. Let ϕ be a self-map on $\mathbf{N}$ defined as follows.

$$\phi(n) \begin{cases} n \quad 1, & \text{if } n \text{ is odd} \\ n-1, & \text{if } n \text{ is even.} \end{cases}$$

Here $a(C\phi) = 0 = d(C\phi)$.

Let φ be a self-map on $\mathbf{N}$ defined as follows.

$$\varphi(n) \begin{cases} n, & \text{if } n \text{ is odd} \\ n-1, & \text{if } n \text{ is even.} \end{cases}$$

Here $a(C\varphi) = 1 = d(C\varphi)$.

But $a(C\phi + C\varphi) = 0 = d(C\phi + C\varphi)$.

Example 4.4. Let ϕ be a self-map on $\mathbf{N}$ defined as follows.

$\phi(n) = 2n-1$, for each natural number.

Here $a(C\phi) = \infty$ and $d(C\phi) = 0$.

Let φ be a self-map on defined as follows.

$\varphi(n) = 2n$, for each natural number.

Here $a(C\varphi) = \infty$ and $d(C\varphi) = 0$.

But $a(C\phi + C\varphi) = \infty$ and $d(C\phi + C\varphi) = 0$.

Example 4.5 Let ϕ be a self-map on $\mathbf{N}$ defined as follows.

$$\phi(1) = 1 = \phi(2)$$

and

$$\phi(n) = n, n \forall \geq 3.$$

Here $a(C\phi) = 1 = d(C\phi)$.

Let φ be a self-map on $\mathbf{N}$ defined as follows.

$$\varphi(n) \begin{cases} n, & \text{if } n \text{ is odd} \\ n-1, & \text{if } n \text{ is even.} \end{cases}$$

Here a(Cφ) = 1 = d(Cφ).
But a(Cϕ + Cφ) = 1 = d(Cϕ + Cφ).

Example 4.6 Let ϕ be a self-map on **N** defined as follows.

$$\phi(n) \quad \begin{matrix} n \quad 1, & \text{if } n \text{ is odd} \\ n-1, & \text{if } n \text{ is even.} \end{matrix}$$

Here a(Cφ) = 0 = d(Cφ).
Let φ be a self-map on **N** defined as follows.

$$\varphi(n) = n+1, \text{ for each natural number.}$$

Here a(Cφ) = ∞ and d(Cφ) = 0.
But a(Cϕ + Cφ) = 0 = d(Cϕ + Cφ).

5. RESULTS

In this section we study ascent and descent of sum of two composition operators on l^P spaces.

Theorem 5.1. If ϕ or φ is an injective self-map on **N** then d(Cϕ+Cφ) = 0.
Converse is not true.

Proof. If ϕ is injective then R(Cϕ) = l^P. Now R(Cϕ + Cφ) = R(Cϕ) + R(Cφ) = l^P + R(Cφ) = l^P. Thus d(Cϕ+Cφ) = 0. The following example shows that the converse is not true. □

Example 5.1. Let ϕ be a self-map on **N** defined as follows.

$$\phi(1) = 1 = \phi(2)$$

and

$$\phi(n) = n, \text{ for each } n \geq 3.$$

Let φ be a self-map on **N** defined as follows.

$$\varphi(3) = 3 = \varphi(4)$$

and

$$\varphi(n) = n, \text{ if } n \in \mathbf{N} \{3, 4\}.$$

Then d(Cϕ + Cφ)= 0.

Ascent and Descent of Sum and Product of two Composition Operators

Theorem 5.2. If ϕ and φ be any two self-maps on **N** into itself and let Cϕ+φ denote the composition operator induced by ϕ + φ. Then a(Cϕ+φ) = ∞.

Proof. Let $n_1 = 1$. For $k \geq 2$, let n_k denote the smallest element of $R((\phi+\varphi)^{k-1})$.
Then clearly we get a sequence $\{n_k\}_{k=1}^{\infty}$ such that $n_k \in R((\phi + \varphi)^{k-1}))$ but $n_k \notin R((\phi+\varphi)^k)$. Hence by theorem 3.2 of [5] it follows that a(Cϕ+φ) = ∞.

Theorem 5.3. $d(C_{\phi+\varphi}) = \infty$ if and only if $(\phi + \varphi) : R((\phi + \varphi)^k) \to R((\phi + \varphi)^k)$ is not one-to-one for all $k \geq 0$.

Proof. Follows from theorem 3.2 of [5]. □

Remark 5.1. Let ϕ and φ are both injective neither of them is surjective. If $\phi \neq \varphi$ then clearly $a(C_\varphi + C_\varphi) = \infty$.

References

[1] Aupetit B.; Primer on Spectral Theory,Springer-Verlag,New-York 1991.

[2] Burgos M., Kaidi A., Mbekhta M., Oudghiri M.; The Descent Spectrum and Perturbations,J. Operator Theory 56(2006), 259-271.

[3] Carlson J.W.;The Spectra and Commutants of Some Weighted Composition Operators, Trans.Amer.math.Soc.317 (1990), 631-654.

[4] Carlson J.W.; Hyponormal and Quasinormal Weighted Composition Operators on l^2, Rocky Mountain J.Math.20 (1990), 399-407.

[5] Chandra H.,Kumar P.; Ascent and Descent of Composition Operators On l^p Spaces, Demon stratio Mathematica XLIII, No.1 (2010),161-165.

[6] Chandra H.,Kumar P.; Essential Ascent and Essential Descent of a Linear Operator and a Composition Operator,preprint.

[7] Grabiner S.; Uniform Ascent and Descent of Bounded Operators,J.Math. Soc.Japan 34(1982),317-337.

[8] Halmos P.R.,A Hilbert Space Problem Book,Van Nostrand,Princeton,N.J.,1967.

[9] Komal,B.S.,and singh R.K.,Composition Operators on l^p and its Adjoint, Proc.Amer.Math.Soc.70 (1978), 21-25.

[10] Kelley,R.L.;Weighted Shifts on Hilbert Space,Dissertation,University of Michigan,Ann Ar bor,1966.

[11] Kumar A.,Singh R.K.;Multiplication Operators and Composition Operators with Closed RangesBull.Austral.Math.Soc.16 (1977), 247-252.

[12] Kumar,D.C.,Weighted Composition Operators,Thesis University of Jammu,1985.

[13] Kumar R.; Ascent and Descent of Weighted Composition Operators On Lp spaces,Matmatick Vesnik 60(2008),47-51.

[14] Kaashoek M.A.; Ascent,Descent,Nullity and Defect: A Note On a Paper by A.E.Taylor Math.Ann ; 172(1967),105-115.

[15] Kaashoek M.A.,Lay D.C.; Ascent,Descent,and Commuting Perturbations,Trans. Amer. Math. Soc. 169(1972),35-47.

[16] Lay D.C.; Spectral Analysis Using Ascent,Descent,Nullity and Defect ; Math. Ann. 184(1970),197-214.

[17] Lal N., Tripathi G.P.; Composition Operators on l^2 of the form Normal Plus Compact,J.Indian.Math.Soc. 72(2005), 221-226.

[18] Mbekhta M.; Ascent,Descent et Spectre Essential Quasi-Fredholm, Rend. Circ. Math. Palermo(1997),175-196.

[19] Mbekhta M., Muller V.; On the Axiomatic Threory of Spectrum II, Studia Math. (1996), 129 147.

[20] Nordgren E.A.; Composition Operators on Hilbert Spaces,J.Math. Soc.Japan 34(1982), 317 337.

[21] Nordgren E.A.; Composition Operators,Canada. J.Math.20 (1968), 442-449.

[22] Parrott S.K.,Weighted Translation Operators,Thesis,University of Michigan,Ann Arbor,1965.

[23] Shields A.L.; Weighted Shift Operators and Analytic Function Theory,Topics in Operator Theory(C.Pearcy,ed.),Math.Surveys, no.(13),Amer. Math.Soc.,Providence,R.I., 1974,49-128.

[24] Singh L.; A Study of Composition Operators on l2 ,Thesis, Banaras Hindu University 1987.

[25] Tripathi G.P.;A Study of Composition Operators and Elementray Operators,Thesis, Banaras Hindu University 2004.

[26] Taylor A.E., Lay D.C.; Introduction to Functional Analysis, John- Wiley, New York Chichester-Brisbane 1980.

[27] Whitley R.; Normal and Quasinormal Composition Operators,Proc.Amer.Math.Soc. 70(1978), 114-118.

SOME METHODS OF CONSTRUCTIONS OF REGULAR BLOCK DESIGNS

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Abstract

Some methods of construction of regular group divisible designs are described which lead to some new series of designs. In particular, some new non-isomorphic solutions for certain Group divisible (GD) designs are presented.

AMS Subject classifications

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Keywords and Phrases

Regular GD design; BIB design; non-isomorphic.

Introductions

Group divisible (GD) designs constitute the largest, simplest and perhaps the most important type of two-associate partially balanced incomplete block designs. A GD design is an arrangement of $v = mn$ treatments in b blocks each of size $k<v$ distinct treatments; each treatment is replicated r times and the set of treatments can be partitioned in $m \geq 2$ groups of $n \geq 2$ treatments each, any two distinct treatments (θ, φ) occurring together in λ_1 blocks if they belong to the same group and in λ_2 blocks if they belong to different groups. Furthermore, if $r-\lambda_1=0$, the GD design is said to be singular, if $r-\lambda_1>0$ and $rk-v\lambda_2=0$ it is called semi-regular(SR); and if $r-\lambda_1>0$ and $rk-v\lambda_2>0$, it is called regular(R).

For the GD designs, it holds that

$$(rk-\upsilon\lambda_2)-(\tau-\lambda_1)=n(\lambda_1-\lambda_2) \qquad \text{..... (1.1)}$$

Where $rk-\upsilon\lambda_2(=\theta_1, say)$ and $r-\lambda_1(=\theta_2, say)$ are eigen-values of NN' other than rk, with the respective multiplicities (m-1) and m (n-1), and then $\theta_1 \geq 0$ and $\theta_2 \geq 0$. Note that N is the incidence matrix of GD designs.

The regular type means that all the eigen values of NN' are positive. For a regular block design, Fisher's inequality, i.e. the number of blocks being bounded below by the number of treatments always holds.

It follows from (1.1) that if $|\theta_1-\theta_2|=1$ then the GD design does not exist. Hence $|\theta_1-\theta_2| \geq 2$ Furthermore, if $|\theta_1-\theta_2|$ is a prime, then $\lambda_2=\lambda_1 \pm 1$. Note that in an SGD design $\lambda_2<\lambda_1$ and in an SRGD design $\lambda_2>\lambda_1$. It seems that smaller values of eigen values θ_1 and θ_2 yield more efficient GD designs in the sense of providing good estimates for certain functions of treatment effects depending on association in treatment structure.

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Clalworthy (1973) gave a table of 209 regular GD designs. Since, then Freeman (1976), Kageyama and Tanaka (1981), Dey(1977), Bhagwandas and Parihar (1980,1982), Dey and Nigam (1985), Bhagwandas et al. (1895), Sinha and Kageyama (1986), and Sinha (1987) have given several methods of constructing GD designs.

In this paper, we describe some new methods of construction of GD design, thereafter some new series of GD designs are obtained. In particular, non-isomorphic solutions of certain GD designs with $r,k \leq 10$ are reported.

THEORAM 2.1. If N denotes the incidence matrix of a BIB design $(v = 2k, b, r, k, \lambda)$, then the incidence structure

$$S = \begin{bmatrix} 0 & 0 & N & N & N & J\text{-}N & N & N \\ N & N & 0 & 0 & N & N & N & J\text{-}N \\ N & J\text{-}N & N & N & 0 & 0 & J\text{-}N & J\text{-}N \\ J\text{-}N & J\text{-}N & J\text{-}N & N & N & N & 0 & 0 \end{bmatrix} \quad \text{..... (2.1)}$$

is the incidence matrix of a regular GD design with parameters

$$v^* = 4v,\ b^* = 8b,\ r^* = 6r,\ k^* = 3k,\ \lambda_1^* = 6\lambda,\ \lambda_2^* = 2r,$$

Proof. Clearly S is of order $v^* \times b^*$ and has row (column) sum $r^*(k^*)$. Further, let $P = SS'$ (S' begin the transpose of S). Then we have,

$$P = I_4 \otimes (A - B) + J_4 \otimes B, \quad \text{..... (2.2)}$$

Where $A = 5NN' + (J - N)(J - N)'$,

$$= 6(R - \lambda) I_v + 6\lambda J_v \quad \text{..... (2.3)}$$

$$B = 2NN' + (J - N)N' + N(J - N)',$$

$$= 2rJ_v \quad \text{..... (2.4)}$$

From (2.3) and (2.4), it follows that $\lambda^*_1 = 6\lambda$ and $\lambda^*_2 = 6r$. The mathematical form of P shows that S is the incidence matrix of a GD design. Since for resulting GD design it holds that $r^* - \lambda^*_1 > 0$ and $r^*k^* - v^*\lambda^*_2 > 0$; hence this is a regular GD design.

The applications of Theorem 2.1, Consider N, the incidence matrix of BIBD (2,2,1,1,0), we obtain regular GD design with parameters $v^* = 8$, $v^* = 16$, $r^* = 6$, $k^* = 3$, $\lambda^*_1 = 0$, $\lambda^*_2 = 2$, $m = 4$, $n = 2$. The same design is listed as R55 in Clatworthy (1973). The solution obtain by us is new non-isomorphic to that given in Clatworthy (1973), in both the solutions the block intersections numbers are different. In our solution the blocks are as follows:

(3,5,8), (4,6,7), (3,6,8), (4,5,7), (1,5,8), (2,6,7),

(1,5,7), (2,6,8), (1,3,7), (2,4,8), (2,3,7), (1,4.8),

(1,3,6), (2,4,5), (1,4,6), (2,3,5).

Remark 2.1.

The complementary design of RGD design $v^* = 8, v^* = 16, r^* = 6, k^* = 3, \lambda^*_1 = 0, \lambda^*_1 = 2, m = 4, n = 2$. has the parameters $v^* = 8, v^* = 16, r^* = 10, k^* = 5, \lambda^*_1 = 4, \lambda^*_2 = 6, m = 4, n = 2$. This is R136 in in Clatworthy(1973), but the present solution is non-isomorphic to the reported solution in Clatworthy(1973) in terms of block structure. The blocks in the present solution are as follows:

(1,2,4,6,7),	(1,2,3,5,8),	(1,2,4,5,7),	(1,2,3,6,8),
(2,3,4,6,7),	(1,3,4,5,8),	(2,3,4,6,8),	(1,3,4,5,7),
(2,4,5,6,8),	(1,3,5,6,7),	(1,4,5,6,8),	(2,3,5,6,7),
(2,4,5,7,8),	(1,3,6,7,8),	(2,3,5,7,8),	(1,4,6,7,8),

THEOREM 2.2. If N be the incidence matrix of a BIB design with parameters (v, b, r, k, λ) and $v = 2k$, then

S=

N	N	N	N	N	N	N	N	N	N	0	0	0	0	0
N	N	N	J-N	J-N	J-N	0	0	0	0	N	N	N	N	0
N	N	0	N	0	0	J-N	J-N	J-N	0	N	J-N	J-N	0	N
N	0	J-N	0	J-N	0	N	N	0	J-N	J-N	J-N	0	N	N
0	0	N	J-N	0	J-N	N	O	J-N	N	J-N	O	J-N	J-N	N
0	N	0	0	N	J-N	0	N	J-N	J-N	0	J-N	N	J-N	J-N

Yield a regular GD design with parameters

$$v^* = 6v, b^* = 15b, r^* = 10r, k^* = 4k, \lambda^*_1 = 10\lambda, \lambda^*_2 = 3r, m = 6, n = v$$

Proof : Obvious.

As an illustration, Consider N, the incidence matrix of BIBD (2, 2, 1, 1, 0) in Theorem 2.2, we obtain regular GD design with parameters $v^* = 12, b^* = 30, r^* = 10, k^* = 4, \lambda^*_1 = 10\lambda, \lambda^*_2 = 3, m = 6, n = v$ The same RGD is reported by Freelman (1976) as R110b. Solutions obtained by us is non-isomorphic to that reported in Freeman (1976). The blocks in our solutions are as follows:

(1,3,5,7),	(2,4,6,8),	(1,3,5,11),	(2,4,6,12),	(1,3,8,9),
(2,4,7,10),	(1,4,5,10),	(2,3,6,9),	(1,4,8,11),	(2,3,7,11),
(1,4,10,12),	(2,3,9,11),	(1,6,7,9),	(2,5,8,10),	(1,6,10,12),

(2,5,9,11),	(1,8,9,12),	(2,7,10,11),	(3,5,8,10),	(4,6,7,9),
(3,6,8,12),	(4,5,7,11),	(3,6,10,11),	(4,5,9,12),	(3,7,10,12),
(4,8,9,11),	(5,7,9,12),	(6,8,10,11),	(1,6,7,11),	(2,5,8,12),

THEOREM 2.3.

The existence of a BIB design with $v = 2k$, implies the existence of a regular GD design with parameters

$$v^* = 7v, b^* = 14b,\ r^* = 8r, k^* = 4k, \lambda_1^* = 8\lambda,\ \lambda_2^* = 2r,\ m = 7,\ n = v$$

Proof : We start with the BIB Design $v = 2k,\ b = 2r,\ r,\ k,\ \lambda$. Now we form the incidence structure S by arranging N the incidence matrix of BIB Design, J-N, the complementary structure of N, and o the null matrix as

S =

N	N	N	N	N	N	N	N	0	0	0	0	0	0
N	0	0	J-N	N	J-N	0	0	0	J-N	N	0	N	N
N	0	N	0	J-N	0	J-N	0	N	0	J-N	N	0	N
0	0	J-N	N	0	0	0	J-N	J-N	N	0	N	N	0
0	N	J-N	0	0	J-N	N	0	N	N	O	0	J-N	N
0	N	0	J-N	0	0	J-N	N	0	N	N	N	0	J-N
0	N	0	0	J-N	N	0	J-N	N	0	N	J-N	N	0

is the incidence matrix of the required regular GD design.

As an illustration, Consider N as the incidence matrix of BIBD (2, 2, 1, 1, 0) in Theorem 2.3, the resulting regular GD design has the parameters

$$v^* = 14,\ b^* = 28,\ r^* = 8,\ k^* = 4,\ \lambda_1^* = 0,\ \lambda_2^* = 2;\ m = 7,\ n = 2, \qquad \ldots\ldots\ldots\ldots\ldots(2.5)$$

This is deign R_{113} in Clatworthy (1973). Solution obtained by us is new non-isomorphic to the solution reported in Clatworthy (1973). In our solutions the blocks are as follows:

(1,3,5,7),	(2,4,6,8),	(1,9,11,13),	(2,10,12,14),	(2,6,7,9),	(1,4,7,12),
(2,3,8,11)	(1,3,6,14),	(2,4,5,13),	(1,4,10,13),	(2,3,9,14),	(1,6,9,12)
(2,5,10,11),	(1,8,11,14),	(2,7,12,13),	(5,8,9,13),	(6,7,10,14),	(4,7,9,11),
(3,8,10,12),	(3,6,16,13),	(4,5,12,14),	(5,7,11,14),	(6,8,12,13),	(3,7,10,13),
(4,8,9,14),	(3, 5,9,12),	(4,6,10,11)			

Corollary 2.1: If the incidence structure S is written in the form as

$$S = \begin{array}{|cccccccccccccc|}
\hline
N & N & N & N & N & N & N & N & 0 & 0 & 0 & 0 & 0 & 0 \\
N & N & J\text{-}N & 0 & 0 & J\text{-}N & 0 & 0 & 0 & N & N & 0 & N & N \\
N & N & 0 & J\text{-}N & 0 & 0 & J\text{-}N & 0 & N & 0 & J\text{-}N & N & 0 & J\text{-}N \\
N & N & 0 & 0 & J\text{-}N & 0 & 0 & J\text{-}N & J\text{-}N & J\text{-}N & 0 & J\text{-}N & J\text{-}N & 0 \\
0 & 0 & 0 & N & J\text{-}N & N & 0 & J\text{-}N & N & 0 & N & 0 & N & J\text{-}N \\
0 & 0 & N & 0 & J\text{-}N & N & J\text{-}N & 0 & J\text{-}N & N & 0 & N & 0 & N \\
0 & 0 & N & J\text{-}N & 0 & 0 & N & J\text{-}N & 0 & N & J\text{-}N & J\text{-}N & N & 0 \\
\hline
\end{array} \quad \ldots(2.6)$$

Then S in the incidence matrix of a regular GD design with the parameters

$$v^* = 7v,\ b^* = 14b,\ r^* = 8\Omega,\ k^* = 4k,\ \lambda_1^* = 8\lambda,\ \lambda_2^* = 8r\ ;\ m = 7,\ n = v.$$

Remark 2.2:

If in S given in (2.6) take N, the incidence matrix of a BIBD (2, 2, 1, 1, 0), we get regular GD design with the same parameters given in (2.6). But this solution is new non-isomorphic to both solutions reported in Clatworthy (1973) and that in (2.5) with respect to distributions of block intersections numbers. The blocks for new non-isomorphic solution are written as follows:

(1,3,5,7), (2,4,6,8), (1,3,5,7), (2,4,6,8), (1,4,11,13), (2,3,12,14),

(1,6,9,14), (2,5,10,13), (1,8,10,12), (2,7,9,11), (1,4,9,11), (2,3,10,12),

(1,6,12,143, (2,5,11,14), (1,8,10,14), (2,7,9,13), (5,8,9,12), (6,7,10,11),

(3,8,11,13), (4,7,12,14), (3,6,9,14), (4,5,10,13), (5,8,11,14), (6,7,12,13),

(3,8,9,13), (4,7,10,14), (3,6,10,11), (4,5,9,12).

The method described in theorem 2.1, 2.2 and 2.3 are useful for the combinatorial constructions of regular GD designs, but they may produce deigns with relatively large parameter values. In the range r, k $\leq$ 10 of much practical value, some examples are here taken up.

References

Bhagwandas and Parihar, J, S. (1980): Some New Group Divisible Deigns. J. Statist. Plan. Infer. 4, 321-323.

Bhagwandas and Parihar, J, S. (1982): Some New Series of Regular Group Divisible deigns, Comm. Statist Theory Meth. 11, 761-768.

Bhagwandas, Bomerjee, S and Kageyama, S. (1985): Patterned Constructions of Partially Balanced Incomplete Block Designs. Comm. Statist. A- Theory Methods, 14, 1259-1267.

Clatworthy, W. H. (1973): Tables of two-associate-class partially balanced designs, NBS Applied Mathematics Series, 63, U. S. Department of Commerce, National Bureau of Standards, Washington, D. C.

Dey, A. (1997): Constructions of group divisible designs. Biometrika, 64, 647-649.

Dey,A. and Nigam, A.K. (1985): Construction of group divisible designs . J. Ind. Soc. Agric. Statist. 37, 163-166.

Freeman, G.H. (1976): A cycle method of constructing regular group divisible incomplete block designs. Biometrika, 63, 555-558.

Kageyama,S. and Tanaka, T. (1981): Some families of group divisible designs. J. Statist. Plan. Inference, 5, 231-241.

Sinha, K.(1987): A method of construction of regular group divisible designs. Biometrika, 4, 443-444.

Sinha,K. and kageyama, S. (1986): pairwise balanced semi-regular and regular group divisible designs. Bull. Inform. cybernet., 22, 55-57.

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