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Printed at Saujanya Printing Press, D-47, Okhla Industrial Area, Phase-1, New Delhi - 110 020.

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International Journal of Food, Nutrition and Dietetics

Volume 1 Number 1 January - April 2013

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Impact of Socio-Economic Factors on Underweight of the Pre-Schoolers

Sowmya B.M.

Abstract

India is one of the fastest growing countries in terms of population and economics, sitting at a population of 1,139.96 million (2009)[1] and growing at 10-14% annually (from 2001-2007). [1]India's Gross Domestic Product growth was 9.0% from 2007 to 2008; [1]since Independence in 1947, its economic status has been classified as a low-income country with majority of the population at or below the poverty line. Though most of the population is still living below the National Poverty Line, its economic growth indicates new opportunities and a movement towards increase in the prevalence of chronic diseases which is observed in at high rates in developed countries such as United States, Canada and Australia. The combination of living in poverty and the recent economic growth of India have led to the co-emergence of two types of malnutrition: under nutrition and over nutrition.

Key Words: Over Nutrition: refers to an excessive intake of one or more nutrients, which creates a stress in the bodily functions. **Pre-term:** Babies born before the end of 37 weeks gestation (less than 259 days). **Post-term:** Babies born at 42 completed weeks or any time there after (294 days and over) of gestation. **Term:** Babies born from 37 completed weeks to less than 42 completed weeks (259-293 days) of gestation. **Under Nutrition:** (also known as Protein-Calorie malnutrition or energy-deficiency) is associated with exacerbation of health conditions, increased frailty, and decline in physical, cognitive, and affective function.

Introduction

Children are nature's gift and the fountain of life. They are our future and are a supremely important asset of life. Their nature is solicitude and it is our responsibility. The strength of nation lies in the health of citizens. Children are future citizens and their health is nation's wealth. There is a meaningful truth in saying that "Nation marches on the tiny feet of young children and no nation can flourish without due love and attention paid to its children". By promoting their health we will be strengthening the development of the family, nation and the world.

A good nutrition is essential for the growth of the children. Nutrient requirements are recommended dietary intakes by national and international experts are primarily need for healthy normal growth and development. According to United Nations declaration "the child shall enjoy the special protection and shall be given opportunities and facilities by law and order and by means to enable him to develop physically and mentally in a healthy and normal manner and in a condition of freedom of dignity". The child has all the right to enjoy the benefits of social security, housing, nutrition and medical care.

Realizing the importance of children, World Health Organization (WHO)[2] has declared the themes relating to children in the following years,

1951 – Health for your child and world's children.

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⁽Received on 20.12.2012; Accepted on 18.04.2013)

^{1979 –} A healthy child a sure future.

^{1984 -} Children's health tomorrow's wealth.

2005 – Make every mother and *child* count.

In the millennium declaration of September 2000,[3] member states of United Nations made a most passionate commitment to address the crippling and multiplying misery that grip many areas of the world. Governments set a date of 2015 by which they would meet the Millennium development goals. Among these, the first goal is to eradicate extreme poverty and hunger, which is measured by the prevalence of underweight children. The target is to halve the burden of under nutrition. The next important goal with regards to children is to achieve two third reductions in mortality and infant mortality by 2015.

Three quarter of children who die world wide of causes related to malnutrition are described by nutritionists as mildly to moderately malnourished and thus betray outward signs of problem to a casual observer.

In India, majority of people live in rural areas. The health status and health problems vary in rural children because of low standard of living, poor hygiene and inadequate sanitation. Several studies have been conducted throughout the country on various aspects like growth pattern, feeding practices, morbidities and nutritional deficiencies among pre-school children. But very less information is available regarding the nutritional status, in this part of nation and in our field practice area. The World Bank estimates that India is ranked 2nd in the world of number of children suffering from malnutrition, after Bangladesh (in1998),[4] where 47% of the children exhibit a degree of malnutrition. The prevalence of underweight children in India is among the highest in the world, and is merely double that of Sub-Saharan Africa with a dire consequences for morbidity, mortality, productivity and economic growth. The UN estimate that 2.1 million of Indian children die before reaching the age of 5 years. Every day, 1000 Indian children die because of diarrhea alone. According to the 1991 census of India it has around 150 million children, constituting 17.5% of India's population, who are below the age of 6 years. Child under nutrition

measured as a poor anthropometric status – is internationally recognized as an important public health indicator for monitoring nutritional status and health in population. Young children are most vulnerable to under nutrition and face the greatest risk of its adverse consequences. In addition, growth retardation in early childhood is associated with significant functional impairment in adult life [5] and reduces the work capacity [6] which in turn has an impact on economic productivity.

Determinants of childhood undernutrition

Under nutrition have several levels of determinants. Poverty is a strong underlying determinant that leads to household food insecurity, poor childcare, maternal under nutrition, unhealthy environments and poor health care. These factors then lead to the immediate determinants of childhood under nutrition, that is, low birth weight, inadequate dietary intake of nutrients and frequent infectious diseases. Low birth weight, primarily due to Intra Uterine Growth Retardation (IUGR) in developing countries, is a consequence of maternal under nutrition prior to and during pregnancy and subsequently contributes to under nutrition in infancy and childhood .[7] This is especially important in areas such as South Asia, where there is a very high prevalence of low birth weight. The diets of many children in developing countries are inadequate, and children in first two years of life are at risk.

In order to tackle the problem of under nutrition, the Government of India has taken up several initiatives and ICDS is one of them. Integrated Child Development Services (ICDS) scheme was launched in 1975 with the objectives of improving the nutritional health status of children in the age group of 0-6 years in addition to services to the mother. In Karnataka, there have been very few studies and no district level data bases are available on nutritional parameters. The Department of Women and Child Development, Government of Karnataka which is the nodal department viewed the need for taking a stack of the situation in the state using new "WHO Growth Standards". The new "WHO Growth Standards" sets the benchmark for growth and development of all children from birth to the age of five, which is age and gender specific. The significance of these standards is also to provide a unique link between physical growth, motor development and a strong evidence for the protection, promotion and support of the right of every child to develop his/her full potentials.

Child health problems

The problems facing the health worker in the developing world are vast and are nowhere more evident than in the field of child care. The main health problem encountered in the child population comprises the following:

- 1. Low birth weight
- 2. Malnutrition
- Infection and parasitoses
- 4. Accident and poisoning
- 5. Behavioral problems.

The birth weight of an infant is the single most important determinant of its chances of survival, healthy growth and development. There are two main group of low birth weight babies 1) those are prematurely (short gestation) and 2)those with foetal growth retardation. By international agreement low birth weight has been lined as a birth weight of less than 2.5 kg (up to & including 499 gms), the measurement being taken preferably with the first year of life. Apart from birth weight, babies can also be classified into three steps according to gestational age using word "preterm", "term", and "post-term".

The prevalence and distribution of malnutrition in a society have implications for public health outcomes and policy formulation. Body Mass Index (BMI), calculated as weight (kg)/height² (m), provides an indicator of nutritional status. Global Acute Malnutrition (GAM), or "Wasting", is defined as low weight for height, or the presence of edema. It can be moderate (MAM) or severe (SAM). Severe Acute Malnutrition characterizes children

with a very low weight for height, by visible severe wasting, or by the presence of edema.

Protein-Energy Malnutrition (PEM) has been identified as a major health and nutrition problem in India. It occurs particularly in children in the first five years of life. It is characterizes by low birth weight if the mother is malnourished, poor growth in children and high level of mortality in children between 12-24 months, and is estimated to be an underlying cause in 30% of death among children under age 5 years.

Statement of problem

"A study on impact of socio-economic factors on underweight of the pre-schoolers in Tumkur".

Objectives

- 1. To review the levels of under nutrition in the community based on ICMR standards using anthropometric data in Tumkur.
- To find out the different socio-economic factors affecting nutritional status of the pre-schoolers and to identify the problems of working and non-working mothers in feeding their children.

Hypothesis

H1-Socio-economic factors directly influence the nutritional status of the pre-schoolers.

H2-Work schedule of the mother affects the nutritional status of the child.

Research methodology

For accomplishing the objectives of the study, a house hold survey work was adopted. The study was conducted in Tumkur city. With a purposive sampling method, Sixty three children were selected (divided into children of working and non-working mothers). Self

		Worki	ng Women	Non-	Working Women
		No.	%	No.	0/ /0
	Pucca	10	25.64	8	33.33
Turna of houses	Semi-pucca	20	51.28	14	58.33
Type of house	Kutcha	9	23.07	2	8.33
	Nuclear	24	61.53	13	54.16
Type of family	Joint	8	20.51	6	25
Type of family	Extend ed	7	17.94	5	20.83
Source of	Tap	24	61.53	13	54.16
drin king	Tube well	12	30.76	6	25
water	Open well	3	7.69	5	20.83
	Open Space	20	51.28	16	66.66
Toilet facility	Toilet room	19	48.71	8	33.33
Economic da ha	APL	14	35.89	3	12.5
Economic status	BPL	25	64.10	21	87.5
	Illiterate	18	46.15	5	20.83
T des anti en 1 es el	Read & Write	10	25.64	14	58.33
Education level	Educated	11	28.20	5	20.83

Table1: Socio-Economic status of both Working and Non-Working Women

Table 2: Prevalence of underweight of the children according to BMI

	W	forking Women	Non-Working			
Sex	Count	Standard Deviation	Count	Standard Deviation		
Boys	12	2.44	10	2.27		
Girls	27	1.92	14	3.32		
Pooled	39	2.18	24	2.795		

administered knowledge questionnaire was found appropriate to assess the nutritional status of the children and the problems of working and non-working mothers in feeding their children in Tumkur.

Findings and Discussion

Household deprivation status (HDS) has strongly influenced the nutritional status of pre-school children. Household deprivation status of working and non-working women has 51.28 percent and 58.33 percent of semi pucca house respectively. While 61.53 percent of working and 54.16 percent of non-working women were of nuclear family. Whereas 17.94 percent of working and 20.83 percent nonworking women were of extended families. Joint families were found about 20.51 percent and 25 percent respectively in both working and non-working women. It was found that

Table 3: Feeding practice followed by working and non-working women

Variablas	Working women				Non-working women			
vanabies	Yes		No		Yes		No	
	No.	No. % J		9/6	No.	0/ 0/	No.	0/ /0
Colostrums feeding	36	92.30	3	7.69	24	100	-	-
Pre Lacteral feeding	9	23.07	- 30	76.92	7	29.16	17	70.83
Complimentary food	3-6 n	anths	7-9 months		9-12 months		Above 1year	
was introduced	No.	n/ ,0	No.	%	No.	0/ /0	No.	0/ ,0
Working women	9	23.17	18	46.15	-	-	12	30.76
Non working women	3	12.5	15	62.5	-	-	6	25

	Workin g women					Non-working women						
Type of food	Sometimes		Everyday		No		Sometimes		Everyday		No	
rypeoriood	No.	0/ /0	No.	a/ /0	N 0.	%	No	0/ ,0	No	0/ /0	No	0/ /0
Rice/ Millet	1	2.56	38	97.43	-	-	1	4.16	23	95.83	-	-
Roots & Tubers	34	87.17	5	12.82		-	24	100	-	-		-
Meat/Fish/Egg	35	89.74	2	5.12	2	5.12	21	87.5	2	8.33	1	4.16
Vegetables	9	23.07	- 30	76.92	-	-	6	25	18	75		-
Biscuits/Bread	13	33.33	26	66.66	-	-	8	33,33	16	66.66	-	-
Fruits	32	82.05	7	17.94	-	-	17	70.83	7	29.16	-	-

Table 4: Types of food given for children by working and non-working women

maximum percent of families' i.e. 61.53 percent and 54.16 percent respectively among working and non-working women had basic facilities like source of drinking water, while only 48.74 percent of families had toilet room facilities among working women and 33.33 percent among families with non-working women. When considered economical status of the families, maximum of 64.10 percent among working and 87.5 percent among nonworking women came under Below Poverty Line. In case of mother's literacy, about 28.20 percent among working and 20.83 percent among non-working women were highly educated. The household deprivation index is not a direct measure of economic condition of the household as the per capita income or expenditure or the standard of living index but a measure of the extent to which the household is deprived.

According to BMI criteria (weight for height), it was seen that prevalence of underweight among 27 girls and 12 boys of working women was not significant statistically. Among non-working women group, 14 girls and 10 boys were underweight and was not significant statistically. On the whole, the BMI of pre-school children of working women varied between the lowest being 9.73 and the highest being 16.99 and 5.42 being the lowest and 18.08 being the highest BMI among the children of non-working groups.

Children whose mothers have some education but have not completed middle school are much less likely to be stunted, wasted, or underweight than are children whose mothers are illiterate. Children whose mothers have completed middle school or higher education are even less likely to suffer malnutrition.

Maximum of 92.30 percent working women and 100 percent non-working women have feed colostrum a protein rich, watery and yellowish fluid that comes from the mammary gland during the first two or three days and differs from the regular milk. Very less percent of working women were not aware of the advantages of colostrum which contains B_{12} binding protein, antibodies against viral diseases such as small pox, polio, measles and influenza. Whereas 23.07 percent of working and 29.16 percent women feed their newborn with sweet water, or honey before feeding colostrum. Only after Exclusive Breast Feeding (EBF) for six months, 46.15 percent of working and 62.5 percent of non-working women introduced complimentary foods such as milk other than breast milk, juice of fresh fruits and soup from green leafy vegetables and solid supplements for their infant.

It is observed that 89.74 percent and 87.17 percent of working women feed their child with flesh foods and egg and roots and tubers sometimes. Whereas cent per cent and 87.5 percent of non-working women feed their child with flesh foods and roots and tubers sometimes. Many of working women feed their child with all type of foods everyday and in case of non-working women; the child was given all type of food every day except roots and tubers. Only very few families i.e. about 5 percent followed vegetarian food pattern among working and non-working women.

Recommendations

- The findings of the present study point to a need to improve access to nutritional health care and health education for pregnant women, pre-schoolers and their caregivers. Survey findings support the proposal of including nutrition education component for maternity care providers.
- Effective economic, social and political changes, food security, personal hygiene, maternal education, nutrition education program especially for mothers are few interventions and tools to bring about change in child health.

Conclusion

Childhood malnutrition levels are still alarmingly high around the world. According to the recent report, deaths of about 55% children under 5 years of age are due to malnourishment (UNICEF, 1994).[3] In 1995, almost one third of children under 5 in developing countries were estimated to be under weight (UNACC/SCN, 1997) [8] with half living in south Asia. The nutritional status of under five is a sensitive indicator of a country's health status as well as economic condition. The study concluded that the family's socio-economic factors and mother's education are the important determinants of underweight in pre-schoolers. The impact of underweight is multifarious. It has an all pervasive impact on physical well being and socio-economic condition of a nation. Prevalence of wasting and underweight were also remarkably high among low birth weight children. The educational level of mothers was positively related to the better nutritional status of children. Educated mothers are more conscious about their children's health. They tend to look after their children in a better way. This study finding also suggests that mother's education played a significant role in reducing

prevalence of underweight. Nutrition security is not just having enough food but it's the outcome of good health, a healthy environment and good care. India experienced a rapid economic boom between 1991 and 2007. However, this economic growth has not translated into improved nutritional status among young Indian children. The present study assessed the nutritional status of preschoolers using the anthropometric measures weight for height. The socio-economic variables were significantly associated with malnutrition in pre-schoolers.

Socio-economic-demographic factors, low maternal education, poor nutrition knowledge for mother and feeding practices for sick children seem to act mainly through prenatal factors, whereas other determinants seem to influence more directly the children's stunting and underweight. It is recommended that an improvement in societal infrastructure, better maternal education and nutrition are needed to address the child malnutrition issue.

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Effect of Cooking Methods on the Total Phenolic Content and Antioxidant Activity of the Commonly Consumed Pulses

Sumeet Kaur*, Balwinder Sadana**

Abstract

The effect of various cooking methods on the antioxidant activity of commonly consumed pulses was studied. Raw and processed pulses were analyzed for their antioxidant activity and total phenolic content. The antioxidant activity of the raw pulses ranged from 64.1to 95.3 % inhibition, with the highest value in red kidney beans and lowest in moth beans. Maximum retention of antioxidant activity was observed in microwave cooked pulses with mean retention of 89.3% whereas minimum retention was observed in germinated pulses with mean retention of 74.4%. Total phenolic content of the raw pulses varied from 52 to 313 mg/100g whereas the corresponding values in cooked pulses ranged from 45 to 263mg/100g. The reduction of total phenolic content might be due to the differences on distribution and content of phenolic compounds in the seed coat and cotyledon between tested seeds. The study recommended that microwave cooking was the suitable method for retaining maximum AOA in the pulses followed by pressure cooking.

Key words: Pulses; Cooking methods; DPPH; Antioxidant activity; Total phenolic content; Proximate composition.

Introduction

Pulses are also recognized as a food choice with significant potential health benefits. They represent an important source of protein for vegetarians and have a low glycemic index. Pulses contain complex carbohydrates (dietary fibres, resistant starch and oligosaccharides), protein with a good amino acid profile (high lysine), important vitamins and minerals (B vitamins, folates and iron) as well as antioxidants and polyphenols .[1] Pulses also enzyme inhibitors, contain lectins, oligosaccharides, polyphenols, phytates and saponins-also known as antinutritional factors

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(Received on 25.03.2013; Accepted on 18.04.2013)

(ANF's) - that affects the digestibility and bioavailability of nutrients in humans and animals. Pulses also contain tocopherols, flavonoids and isoflavones, all of which can act as antioxidants. The processing of pulses inhibits or reduces the activity of these compounds.[2]

Antioxidants are our first line of degree against free radicals damage and are critical for maintaining optimum health and wellbeing.[3] Antioxidants are the substances that delay or inhibit oxidative damage when present in small quantities compared to an oxidizable substrate. Therefore, antioxidants can help in increase prevention by effectively quenching free radicals or inhibiting damage caused by them.[4]

Although dry pulses do not contain any ascorbic acid, the germinated pulses and immature green pulses do contain ascorbic acid which also has antioxidant activity.[5] From the nutrition point of view, only thermally processed pulses are important since pulses are never eaten raw. A factor that is attributed for the less use of legumes is it being hard-tocook. A variety of processing methods have

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been practiced such as soaking, germination and cooking.[6] Soaking, boiling and steaming processes significantly affected the total phenolic contents and antioxidant activities. The food processing methods including soaking, pressure cooking, microwave cooking, germination and fermentation greatly influence nutritive values of legumes. Of these cooking and germination plays an important role as it influence the bioavailability and utilization of nutrients.[7]

However, how processing methods effects health promoting phenolic and antioxidant activities have not been studied. Little information is available in literature regarding the effect of cooking on changing the health promoting phenolic content and antioxidant activity of the commonly consumed legumes.

Materials and Methods

Procurement of samples

Five commonly consumed whole pulses namely; Bengal gram, black gram, green gram, moth beans and red kidney beans were purchased from the three markets of Ludhiana city namely Dugri Phase-I, Ghumar Mandi and Jawahar Nagar. Each pulse was collected from the three outlets of each market, the samples of which were pooled and considered as single sample of that pulse. Collected samples were thoroughly cleaned of dirt, insects and unwanted particles.

Cooking of pulses

Five hundred grams of each pulse was divided into five equal portions. One portion was retained as raw while others were cooked using cooking methods viz. soaking, germination, pressure cooking and microwave cooking. Each processing method was carried out using soaked pulses and time taken by cooking was also standardized.

Soaking

One hundred gram of each sample was

Table 1.1: Processing/ cooking time (min) of pulses

Pulses	Germination (h)	Pressure woking (min)	Microwave cooking (min)
Beng al gram whole	24	25	35
Black gram whole	24	15	25
Green gram whole	16	10	20
Moth beans	16	15	20
Red kidney beans	36	25	30

soaked in the tap water for 10 hours. Excess water was drained and soaked grains were dried in dehydrator at 60°C.

Germination and Steaming

The soaked grains were allowed to germinate at room temperature for specific time period varied for different pulses (Table 1.1). The germinated seeds were pressure steamed at 15 lbs for 2 mins.

Pressure Cooking

Soaked pulses were pressure cooked for specific time period for different pulses at 15lbs pressure (Table 1.1).

Microwave Cooking

Pulses were cooked in the microwave oven at 2450 MHz specific time period for different pulses (Table 1.1).

All the cooked samples were dried at 60±2°C till these were moisture free. Dried samples were powdered in an electric grinder. The powders were sieved to obtain homogenized powder and stored in airtight containers for further analysis.

Preparation of extracts for the determination of total phenolic content (TPC) and antioxidant activity:

One gram of finely ground sample was taken into a spout less beaker and 25ml of 70% methanol was added. The contents were refluxed for 2 hours and then filtered.

Pulses	Raw	Soaking	Gemination	Pressure	Microwave	CD at
			& steaming	cooked	cooked	5%
Bengal	153±5.5	142±6.4	140±10.4	73±4.4	63±0.8	19.9
gram whole						
Black gram	285±6	155±12.3	249±4.6	261±34.7	263±21.7	60.9
whole						
Green gram	313±4.8	192±15.4	235±6.8	246±37.7	247±3.0	588
whole						
Moth beans	52±3.5	125±26.7	87±15.3	134±6.8	45±28.7	60.3
Red kidney	302±17.9	243±20.0	167±27.2	253±3.0	232±4.1	55.4
bea ns						
Mean	221±51.2	171±21.01	176±30.1	193±16.9	170±47.6	-

Table 1.2: Effect of cooking method on TPC in pulses

Methanol was evaporated and the volume of the remaining water extract was made to 10ml. The extracts were used for the determination of TPC and antioxidant activity.

Estimation of total phenolic content

Total phenolic content was estimated by modified method given by Singleton *et al* (1999).[8] Methanolic extract (0.1ml) was dissolved in 6.5ml of distilled water. To this, 0.5 ml of Folin Phenol reagent was added and shaken thoroughly. After 5 min, 1 ml of saturated solution of sodium carbonate was added. The blue color developed was read after 1 hour at 760 nm against the blank. The blank was prepared from reagents and water only. The concentration of total phenols was read from the standard curve, prepared by using Gallic acid at the concentration ranging between $10-50\mu g/ml$.

Evaluation of antioxidant activity of whole pulses using DPPH method

Estimation was carried out by method given by Sreeramulu et al (2009).[9] 0.1ml of aliquot of sample extract was taken in a test tube and then 2.9ml of 0.05mM DPPH reagent was added and vortexed vigorously. The content was incubated in the dark for 30 min at room temperature. Discoloration of DPPH was

Pulses	Raw	Soaking	Germination & steaming	Pressure cooked	Microwave cooked	CD at 5%
Bengal gram whole	66.9±2.6	52.9±5.2	45.3±6.0	48.1±2.2	49.7±5.4	NS
Black gram whole	90.9±3.0	43.4±2.6	58.1±3.1	83.1±2.4	84.7±0.9	7.98*
Green gram whole	84.5±2.7	73.0±8.7	84.4±2.2	84.1±0.2	83.8±0.9	NS
Moth beans	64.1±12.9	46.6±3.6	28.5±3.1	63.9±7.1	48.9±10.6	NS
Red kidney beans	95.3±0.3	91.6±0.6	91.6±0.1	73.8±3.3	92.1±1.4	5.5*
Mean	80.38±6.3	61.56±9.1	61.64±11.8	70.64±6.7	71.82±9.3	-

Table 1.3: Effect of cooking method on AOA in pulses

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Pulses	Soaking	Germination &	Pressure	Microwave
	_	steaming	cooking	cooking
Bengal gram whole	79.1	67.7	71.9	74.2
Black gram whole	47.7	63.9	91.4	98.2
Green gram whole	86.4	99.9	99.5	99.2
Moth beans	72.7	44.5	99.7	76.3
Red kidney beans	96.1	96.1	77.4	96.6
Mean	76.6	74.4	87.8	89.3

Table 1.4: % retention of Antioxidant activity of raw and processed pulses

measured against blank at 517 nm.

Results and Discussion

Total phenolic content: The raw samples had 313.3±4.79 to 52±3.46 mg/100g of total phenolic content with the mean value of 221.1±51.2. The results found in present investigation is supported by Sreeramulu *at el* (2009)[9] who reported the range of 62.35-418.34 mg/100g with value of in 194.9±9.6 Bengal gram, 418.34±12.57 black gram dhal,62.35±4.31 green gram, 332.98±8.06 rajmah.

The processed samples had 142±6.4 to 243±20.03, 87.5±15.3 to 249.1±4.61, 73.3±4.38 to 260.8±34.7, 45±28.7 to 263.3±21.7 percent values for soaking, germination, pressure cooked and microwave cooked samples with 171.4±21.01, 175.8±30.12, mean of 193.4±16.95, 169.9±47.63 respectively. Among the raw pulses, moth beans had the lowest and green gram had the highest value. Among soaking, germination, pressure cooked and microwave cooked pulses, moth beans and red kidney beans, moth beans and black gram, Bengal gram and black gram, moth beans and black gram had the lowest and the highest value of total phenol content.

The trend showed that in comparison to raw samples there was significant decline in the total phenolic content in processed samples and among various cooking methods microwave cooked samples had the lowest content of total phenolic content followed with soaking, germination while the pressure cooked samples had the highest value. For processed value, Xu and Chang (2008a)[10] supported the trend that the total phenolic content of processed cool season food legumes was significantly reduced as compared to the raw samples. After soaking, the loss in TPC in peas and chickpeas decreased with soaking and pressure cooking lost more TPC than regular cooking.

The reduction in phenolic content during cooking may be due to binding of polyphenols with other organic substances and proteins, or form alterations in the chemical structure of polyphenols. The reduction of total phenolic content might be due to the differences on distribution and content of phenolic compounds in the seed coat and cotyledon between tested seeds. Boateng et al (2008)[11] also supported that although soaking and cooking dry beans have been shown to reduce the antinutrients such as trypsin inhibitor and reduce phytic acid; it can also increase the contents of tannins, catechins and polyphenols.

Dewanto et al (2002)[12] explained that thermal processing may release more bound phenolic acids from the breakdown of cellular constituents which might be the cause of increase in TPC of moth beans after processing. Siddhuraja and Becker (2006)[13] observed that total phenolic content were significantly (pd″0.05) higher after soaking and dry heating compared to cooking (boiling) of autoclaving of both raw and presoaked seeds. Further, an increase in phenolic content primarily depends on the type of legume and the preparation procedure used. Negi *et al* (2008)[14] observed a decrease in phenolic content during cooking (soaking, germination, pressure cooking and microwave cooking) which may be due to binding of polyphenols with other organic substances and proteins, or form alterations in the chemical structure of polyphenols.

Antioxidant activity

The antioxidant activity of the raw samples ranged from 64.1±12.9 to 95.3±0.28, with the highest value in red kidney beans and lowest in moth beans. The mean value of raw pulses was found to be 80.38±6.31. Among the raw samples, moth beans and red kidney beans had the lowest and highest value of antioxidant activity respectively. In processed samples, the antioxidant activity was observed as 43.4±2.65 to 91.6±0.57 in soaking, 28.5±3.05 to 91.6±0.15 in germination, 48.1±2.25 to 84.1±0.23 in pressure cooked and 48.9±10.56 to 92.1±1.38 in microwave cooked pulses with mean value 61.56±9.09, 61.64±11.8, 70.64±6.71, 71.82±9.3 respectively. In processed samples, black gram and red kidney beans, moth beans and red kidney beans, Bengal gram and green gram, moth and red kidney beans had the lowest and highest value in soaking, germination, pressure cooked and microwave cooked pulses respectively.

In comparison to raw pulses there was significant decrease antioxidant activity with soaking followed by germination, pressure cooking & microwave cooking. Xu and Chang (2008a) observed that after processing, the DPPH free radical scavenging capacities (DPPH values) of processed cool season food legumes were significantly reduced as compared to the respective original unprocessed cool season food legumes. [10]After boiling, free radical scavenging capacities of cool season food legumes were reduced about 60-70%. After steaming, free radical scavenging capacities of cool season food legumes were reduced. It was observed that maximum retention of antioxidant activity was observed in microwave cooked pulses with mean retention of 89.3% whereas minimum retention was observed in germinated pulses with mean retention of 74.4%.

The results are being supported by Xu B, Chang S K C (2008b)that the DPPH free radical scavenging capacities of processed beans was significantly reduced as compared to the respective unprocessed black beans.[15] Siddhuraja (2006)[13] stated that the stability of antioxidant products such as phenolics and flavonoids during heating may be due to the formation of milliards products such as hydroxymethylfurfuraldehyde (HMF) which produces high antioxidant activity.

Conclusion

Investigation revealed that pulses were an important source of antioxidant activity in commonly consumed diet.

Processing/cooking methods significantly decreased the total phenolic content and antioxidant activity in pulses. Maximum decrease in AOA was observed in soaked samples while the minimum decrement was observed in microwave cooked pulses. Microwave cooking was the suitable method for retaining the AOA in the pulses followed by pressure cooking. The maximum decrease in TPC was observed during microwave cooking in all pulses while least decrease was during pressure cooking followed by germination. It indicates that pressure cooking was the most suitable while microwave cooking was the least suitable for retention of TPC.

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Mycotoxin Detection in Food Products Based on Wheat Fractions

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Abstract

Study was undertaken to detect the presence of aflatoxin $B_1(AFB_1)$ in commonly consumed wheat fractions i.e. semolina, porridge and refined wheat flour. Forty pooled samples of wheat fractions were collected from various localities of Ludhiana city. Raw samples of wheat fractions were qualitatively and quantitatively analyzed for AFB₁, contamination by PMC and TLC methods. Out of 40 samples, 28 (70%) were found to be contaminated with AFB₁. Positive screened wheat fractions were used for preparing *idli*, *upma*, *porridge*, *kulcha* and *bhatura* using fermentation-steaming, shallow frying, microwave, baking and deep frying cooking methods. Cooked products were again analyzed for AFB₁. Maximum reduction of AFB₁ was shown by microwave cooking of porridge at 150°C for 10 min., with mean destruction of 76.1% while deep frying (280°C for 30 sec) of *bhatura* and shallow frying (120°C for 3 min) of *upma* showed minimum destruction of 43.2 and 40.8 % respectively.

Keywords: Wheat products; Aflatoxin B₁: Detoxification; Baking; Microwave cooking.

Mycotoxins are compounds that are produced by fungi and cause illness or even death when food and feed containing them are consumed.[1] The mycotoxigenic fungi involved in the human food chain belong mainly to three genera: Aspergillus, Fusarium and Penicillium. While Fusariuim sp. are commonly destructive plant pathogens producing mycotoxins before or immediately after post- harvesting, Penicillium and Aspergillus sp. are more common contaminants of commodities and foods during drying and subsequent storage. [2]Of more than hundred toxic fungal metabolites isolated and identified so far, aflatoxins are unique in having high toxicity and carcinogenicity as well as being resistant to degradation under normal food processing conditions. Diet is the major route

(Received on 10.04.2013; Accepted on 15.04.2013)

through which humans as well as animals are, exposed to aflatoxins. Exposure to aflatoxins in diet is considered an important risk factor for the development of primary hepatocellular carcinoma, particularly in individuals already exposed to hepatitis B.[3] Aflatoxins are also implicated with Indian childhood cirrhosis, Reye's Syndrome and Kwashiorkor .[4] The International Agency for Research on Cancer confirmed aflatoxins as a potential carcinogens .[5]

Cereals form the major bulk of all Indian diets. They are the major source of many nutrients .[6]Of all cereals, wheat occupies a unique position. Wheat (*Triticum aestivum*) has been shown to be naturally contaminated with different levels of aflatoxins during storage.[7] It was observed that level of aflatoxin contamination in grain sample was related with level of sanitation of mill storage places. A similar view had also been expressed by Kalyansundaram and Jayaraman 1996.[8] Wheat is milled to form semolina (suji/rava), porridge (dalia) and refined wheat flour (maida). Semolina is a coarsely ground endosperm and its chemical composition is similar to that of refined wheat flour. In India, it is also used in the preparation of large number of savoury and sweet preparations. Porridge is considered as breakfast cereal. It is

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made from wheat grain and requires cooking before serving. Scanty information is available regarding the presence of aflatoxins in commonly consumed food based on wheat fractions (i.e.semolina, porridge and refined wheat flour). Hence, the present study was undertaken to see the prevalence of aflatoxins in wheat fractions and further the effect of domestic processing on extent of detoxification of aflatoxins in cooked products.

Materials and Methods

A two stage sampling technique was used for selection of samples of wheat fractions i.e. semolina, porridge and refined wheat flour. First stage consisted of convenient sampling of 4 localities namely Ghumar Mandi, Chaura Bazaar, Khud Mahalla and Jawahar Camp. Second stage consisted of random selection of 40 samples (each weighing 1 kg/lot) of semolina, porridge and refined wheat flour. Samples were cleaned and stored in air tight containers at room temperature (37° C) and were further analyzed for aflatoxin B₁(AFB₁) using pressure mini column (PMC)[9] technique and thin layer chromatography (TLC) method. Positively screened wheat fraction samples were used for preparing *idli*, *upma*, *porridge*, *kulcha* and *bhatura* using various ingredients and processing methods given in Table 1.

Developed products were dried at $60 \pm 2^{\circ}$ C, ground to powder and analyzed for AFB₁ to see the impact of cooking methods on AFB₁ destruction. Extraction for quantitative estimation of aflatoxins in raw and cooked samples was done by the method of Romer (1975).[10] Spotting of extracts was done on TLC plates along with standards of AFB₁

Table 1 Ingredients and methods used for preparation of germinated supplementary products

Product	Description of Recipe	Ingredients	Amount	Temperature and time	Cooking method
Idli	Mix semolina with curd and salt to taste. Mixture was made to pouring consistency by using water. Mixture was kept in a hot place for 2 hour S for fermentation. Then adding half teaspoon of eno salt, mixture was put in idli pan and steamed for 12-15 minutes.	Semolina Curd Water Eno salt	60g 25g 50ml ½ tsp	30° C, 2 h 120° C, 20 min	Fermentation Steaming without pressure
Upma	Semolina was shallow 'fried for 3 minutes with 1 tbsp. Of oil till it turned brown in colour. Then water was added and brought to boil. Seasoning was added and cooked till mass was dried.	Semolina Oil Water	60g 15g 180ml	120° C, 3 min	Shallow frying
Porridge	Broken wheat with water was microwave cooked for 10 minutes at 150°C	Broken wheat Water	60g 360ml	150° C, 10 min	Microwave cooking
Kulcha	Refined wheat flour was mixed with 3% yeast, 0.8% salt, 5% each sugar and butter and 56% water in the mixing bowl for 8 min. to prep. are dough. The dough was divided into three portions and kept for proofing for 20 min. at 30°C. After 20 min. pined out the proofed balls. Balls were kept in greased baking tray and proofed them for \pm 20 min. Kulchas were baked at 240°C. for 10 min.	Refined wheat flour Compressed yeast Salt Sugar Butter Water	125g 3.75g 1g 6.25g 6.25g 70m1	30° C, 20 min 240° C, 10 min	Fermentation Baking
Bhatura	Refined wheat flour was mixed' with 3% yeast, 0.8% salt, 5% each sugar and butter and 56% water in the mixing bowl for 8 min. to prepare dough. The dough was divided into three portions and kept for proofing for 20 min. at 30°C. After 20 min. each portion was rolled into rounds of 8 inches diameter and deep fried in oil for 30 seconds at 280°C.	Refined wheat flour Compressed yeast Salt Sugar Butter Water	125g 3.75g 1g 6.25g 6.25g 70ml	30° C, 20 min 280° C, 10 min	Fermentation Deep frying

procured from Sigma Chemical Co. USA. Quantization of AFB₁was done by method of Coker et al (1984). [11]Aflatoxin levels in raw and cooked samples were detected using the following equation:

Where

S is the volume in μ l of mycotoxin standard of equal intensity to $Z \mu l$ of sample, Y is

Aflatoxin (ug/kg) =
$$\frac{SxYxV}{W \times Z}$$

concentration of mycotoxin standard in $\mu g/$ ml, Z is volume in μ l of sample extract required to give fluorescence intensity comparable to that of 'S' μ l of mycotoxin standards, V is volume (µl) of solvent required to dilute final extract and W is weight (g) of original sample contained in final extract.

Results and Discussion

Among the total 40 samples of wheat fractions, 28 samples (70%) were found to be contaminated with AFB1 though there was no visual mould in any of the positive samples. AFB₁ contamination was observed at the rate of 39, 32 and 29%, respectively in semolina, porridge and refined wheat flour fractions. The results of Table 2 are in confirmation with

Table 2: Screening of raw wheat fractions for aflatoxin B_1 (N=40)

Type of wheat	n	No. of positive	Level of
fraction	11	samples	contamination
Semolina	13	11(39)	++++(4)
			++(3)
			+(4)
Porridge	14	9(32)	++++(3)
			+++(1)
			++(2)
			+(3)
Refined wheat	13	8(29)	++++(4)
flour			++(3)
			+(1)
		28(70)	

N : Total no. of samples, n : No. of samples of each fraction Figure in parentheses [] represents % of positive samples Figure in parentheses () represents number of positive samples ++ 116-205 μg/kg + 26-115 µg/kg

+++ 206-295 μg/kg ++++ 296-385 µg/kg

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findings of Breckenridge et al (1986)[12] who reported aflatoxin contamination in commonly consumed cereals like rice and wheat above the permissible limit of 20 ppb (FDA) and 30 ppb prescribed by PFA Act (1954).[13] Wheat has been shown to be naturally contaminated with different levels of aflatoxins during storage .[7]

Semolina samples showed maximum incidence of contamination with moisture content ranging from 12.2-14.3 % with mean value of 13.1 %. The porridge samples had mean moisture content of 9.2% with range of 8.1-10.7% while the refined wheat flour samples had mean moisture content of 12.9% and ranged from 12.2-14.6%. The recommended safe moisture level for storage of wheat flour is 10%. [14] It was also reported that the most important factor in growth of AFB₁ production by Aspergillus flavus was the moisture and relative humidity surrounding the substrate. The safe moisture content of various food commodities at which fungus may not grow varies from one commodity to other therefore any increase in moisture content above the safe level for a particular commodity may support the growth of fungus. In the present investigation samples having moisture more than recommended safe level exhibited the presence of aflatoxin. Findings are also supported by Kumar et al 2002[15] who detected that 20% wheat flour samples above safe limit were positive to AFB₁

Out of 20 positively screened samples as semolina, porridge and refined wheat flour with 2+, 3+ and 4+ level of contamination, 12 samples were randomly selected for AFB₁ detection with both TLC and PMC methods (Table 3). Mean AFB₁ levels in semolina, porridge and refined wheat flour were 267.8+47.7 μg/kg, 272.6+30.9 μg/kg and 283.6+61.3 μ g/kg, respectively, detected by TLC method whereas the corresponding mean value of AFB₁ in semolina, porridge and refined wheat flour were 212.5+37.5 kg/kg, 225+22.4 μ g/kg and 225.0+35.4 μ g/kg as detected by PMC method.

Results of effect of processing on AFB, destruction (Table 4) revealed that

Type of wheat fraction	Sample no.	TLC, μg/kg	PMC, μg/kg±25	Per cent variation
Semolina	1	340.4	275	19.2
	3	357.9	275	23.2
	10	204.7	175	14.5
	12	168.0	125	25.6
	Mean ±S.E.	267.8 ± 47.7	212.5 ± 37.5	20.6 ±2.43
	t value	0.91 ^{NS}		
Porridge	2	198.9	175	12.0
	4	321.7	275	14.5
	7	311.3	225	27.7
	8	196.0	175	10.7
	11	335.0	275	17.9
	Mean ±S.E.	272.6 ± 30.9	225 ± 22.4	16.6 ± 3.0
	t value	1.24 ^{NS}		
Refined wheat flour	5	343.6	275	20.0
	6	160.9	125	22.3
	9	346.3	275	20.6
	Mean ±S.E.	283.6 ± 61.3	225 ± 35.4	21.0 ± 0.7
	t value	0.83 ^{NS}		

Table 3: TLC and PMC method used for aflatoxin B₁ (AFB₁) detection in raw samples

NS: Non significant

Table 4: Effect of cooking methods on destruction (%) of AFB₁ in cooked .products

Name of product	Cooking method	Initial AFB1 in raw sample, μg/kg	Residual AFB1 in cooked sample, μg/kg	% destruction
Idli	Fermentation-	267.8	95.7	64.2
	Steaming	207.0		
Upma	Shallow frying	267.8	171.1	40.8
Porridge	Microwave	272.6	61.8	76.1
Kulcha	Fermentation	283.6	84.4	65.3
	and Baking			
Bhatura	Fermentation	283.6	159.4	43.2
	and Deep frying	200.0	157.4	
t value 1-2	1.98 ^{NS}	2-4	0.23 ^{NS}	
1-3	3.12*	2-5	1.98 ^{NS}	
1-4	2.11**	3-4	4.21*	
1-5	0.64 ^{NS}	3-5	2.54*	
2-3	3.45*	4-5	2.22**	

*Significant at 5% level

** Significant at 10% level

NS Non-significant

Fermentation-steaming resulted in 64.2% destruction of AFB₁ in *idli* when fermented at 30°C for 2 h and then microwave cooking of porridge at 150°C for 10 min has significant impact on the AFB₁ destruction. The results indicated that baking of kulchas at 240°C for 10 min resulted in 65.3% destruction of AFB₁ The mean residual AFB₁ in cooked sample was

84.4±11.9 µg/kg. Saritha and Uma Reddy (1998)[16] showed 80% destruction of AFB₁ in bread made from refined wheat flour. The deep frying of bhaturas at 280°C for 30 sec resulted in 43.2% destruction of AFB₁. The mean residual AFB₁ in cooked sample was 159.4±31.7 µg/kg. Deep frying and shallow frying showed 43.2 and 40.8% destruction of AFB₁ respectively, which was minimum as compared to other methods. Thus the residual level of AFB₁ in cooked products in *idli*, upma, porridge, kulcha and bhatura was found to be 5-8 times higher in comparison to permissible limit of 30 μ g/kg (PFA 1954),[13] thus indicating that there is certain degree of exposure of population to the carcinogenic aflatoxin. Despite the fact that appreciable amounts of aflatoxins are lost during the process of heating, considerable quantities still remain to cause potential harmful effects on human health. So the present investigation warrants that cereal and cereal products should be tested for the presence of aflatoxins periodically.

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Nutritional and Medicinal Properties of Valerian (Valeriana Officinalis) Herb: A Review

Shirin Adel Pilerood*, Jamuna Prakash**

Abstract

Valerian (*Valeriana officinalis*) belonging to valerianaceae family is a well known herb and medicinal plant that has been widely used all over the world especially in Europe, China and Middle East. It is widely used as a sleep aid and sedative in many parts of the world but is also known to relax smooth muscle, hence used for treating stomach and intestine cramps. Alkaloids, terpenes, organic acids and its derivatives, valepotriates and flavones are the known pharmacologically active compounds found in valerian extract. In general, it is accepted that the valepotriates are the compounds responsible for the sedative activity of the Valerianaceae. The present article aims at reviewing the recent reports on its constituents, traditional use, clinical use and scientific verification of pharmacological actions of valerian.

Keywords: Active constituents; Sedative; Sleep aid; Antioxidant properties; Appetite; Food intake.

Introduction

Valerian (Valeriana officinalis) plant root is a herb which is used worldwide over centuries. It belongs to Valerianaceae family. There are 10 genera and about 300 species in the family Valerianaceae. The Valeriana genus is of the family Caprifoliaceae and approximately contains 200 species.[1] The Valerianaceae are typically distributed worldwide and consist of herbs, rarely shrubs, with opposite leaves, a sympetalous, spurred corolla, 1-4 stamens, and a tricarpellate, poorer ovary with 1 functional locule and a single, apical ovule, the fruit is an achene, with a pappus like calyx in some members. The economic uses include some cultivated ornamentals (e.g. *Centranthus*) and negligible edible, medicinal, or essential oil plants. The plant of Valeriana officinalis is native to Europe and Asia and in addition has naturalized in eastern North America. This tall

(Received on 11.04.2013; Accepted on 20.04.2013)

perennial has a preference in moist woodlands; it has been broadly cultivated in northern Europe. Most of the European supply is grown in Holland. Low lying, damp sandy humus with lime fertilizer is the way to cultivate Valerian. It harvests in the late fall and dries. *V. officinalis* is the species which is used in Europe. This genus contains more than 250 species. In traditional Chinese and Japanese medicine *V. fauriei* is used commonly.[2-5] *Valerian capensis* is other species which is used in African traditional medicine[6], *V. edulis* is used in Mexico and *V. wallichii* is used in India.[7]

History of use

The roots of *V. officinalis* known as valerian, since long are taken as sedative medicine in Europe. Valerian is an agent with mild sedative and sleep-promoting properties that is often used as a milder substitute or a possible alternate for stronger synthetic sedatives, such as the benzodiazepines, in the treatment of nervous states and anxiety-induced sleep disturbances.[8] Lesniewicz *et al*,[9] reported that valerian is tranquillizer for people with hyper-excitability and as a smooth-muscle relaxing agent to treat stomach and intestine cramp. Valerian is also a component of many herbal mixtures, which are widely used to treat sleeping disorders.[10] Nowadays, valerian

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extracts are available as dietary supplements, which primarily involve dried root or extracts from the root, formulated into tablets or soft gelatin capsules. Usually each dose contains approximately between 50 mg and 1 gram of dried root or extract. The use of these dietary supplements is widespread, with an estimated 210 and 125 million doses sold annually in the United States and in Europe respectively.[11] Though not supported by research, traditionally it has been recommended for epilepsy.[12] Restlessness, insomnia, nervousness, and tension are the present indications for valerian as reported by Tariq and Pulisetty.[13] It is suggested that the large doses when stopped, as most sleep aids, cause withdrawal symptoms.[14] Cohen and Toro[15] expressed that patients with liver disease are advised not to take valerian. Although it is shown to be a successful remedy for the reduction of anxiety, in some individuals some side effects like headaches and night terrors were reported.[16] He explained the reason may be due to the fact that some people lack a digestive conversion property required to effectively break down valerian. In these individuals, valerian can cause agitation.

Nutritional and Chemical constituent

Valerian was studied for its mineral content by Adamczyk and Jankiewicz[17] and they reported that valerian root contains 13.1 ppm copper, 75.1 ppm zinc and 16.8 ppm manganese. Adel Pilerood and Prakash[18] analyzed the chemical constituents of valerian as reported in Table 1. More than 150 chemical constituents were found in valerian of which many are physiologically active.[19] There is significant variation in the chemical constituents in plants from different sources and growing conditions, processing methods and storage conditions.[20] To guarantee the quality of the drug, producers have standardized production of the plant extracts.[21] Alkaloids, terpenes, organic acids and its derivatives, valepotriates and flavones are the known pharmacologically active compounds found in valerian extract. In

general, it is accepted that the valepotriates are the compounds responsible for the sedative activity of the Valerianaceae.[22] Alkaloids (0.01–0.05%), notably terpene alkaloids are present in valerian.[23] The main valerian alkaloids are actinidine, chatinine, valerianine, valerine, alpha-methyl pyrryl ketone and naphthyridin methyl ketone.[24-26] The structures of some valerian alkaloids are shown in Fig 1.

Actinidine

Actinidine (Ia) is a steam-volatile monoterpenoid pyridine alkaloid with a cyclopenta [c] pyridine skeleton present in the essential oil of valerian root[27] and *Actinidia polygama* (silver vine).[28] Actinidine is compound in valerian, which can attract cats.[22] Biosynthesis of actinidine results from

Figure 1: The structures of principal compounds present in volatile essential oil of *Valeriana officinalis*



lysine and quinolinic acid as precursors.[29] Actinidine is an alkaloid which is psychoactive which interferes with the gammaaminobutyric acid (GABA)-ergic metabolism; it is an agonist on benzodiazepine receptors and thus revealed an allosteric modulation of the GABA-receptor-proteins.[22]

Waliszewski[30] isolated Chatinine from valerian but its biological properties have not been studied. Alpha-methyl pyrryl ketone was studied in Germany as a central nervous system active compound in 1970.[31] Synthetic naphthyridinones similar in structure to natural naphthyridyl methyl ketone were introduced as potential drugs for the treatment of schizophrenia.[32-33] Since the pharmacological properties of valerian alkaloids have been studied separately only infrequently, it is difficult to say how these participate in the medical effects of V. officinalis.

Organic acids and Terpenes

Organic acids and terpenes are available in the volatile essential oil, which is 0.2-2.8% of the dry weight of the root. The essential oils are not only seen in the subrerranean parts of the plants but also in the aerial parts.[34] Terpenes are characterized chemically as monoterpenes and sesquiterpenes. Valeric, isovaleric, valerenic, isovalerenic and acetoxyvalerenic acids, bornyl acetate, bornyl isovalerenate, 1-pinene, 1-comphene, 1terpineol, valeranone and borneol, cryptofauronol are most considerable valerian organic compounds. It is suggested that some of the oil components pose sedative properties.

Isovaleric acid and bornyl isovalerate are two compounds which are mainly responsible for the characteristic aroma of valerian. Isovaleric acid and 3-methylbutanoic acid do not have significant pharmacological and toxicological properties and only share the drug's odor. However, it was found in 2007 that isovaleric acid decreases ATPase activity in the synaptic membranes of the cerebral cortex and it may be necessary in the pathophysiology of the neurological dysfunction of isovaleric acidemic patients.[35]

Valerenic acid (IIa) and its aldehyde valerenal (IIb) are monoterpenes which are pharmacologically active compound. Cavadas et al[36] recommended that valerian acts via GABA mechanisms. Other studies revealed binding of valerian extract to GABA receptors, but the functional effect of the binding was not demonstrated. Data from the study of Yuan et al[37] and Trauner et al[38] suggest that the pharmacological effects of valerian extract and valerenic acid are mediated through modulation of GABAA receptor function. By passive diffusion valerenic acid is known to penetrate into the central nervous system trans cellular.[39] Dietz et al[40] showed that valerenic acid is a partial agonist of the 5HT receptor with the strong binding affinity to the 5-HT (5a) receptor, but only weak binding affinity to the 5-HT(2b) and the serotonin transporter. In a study valerenic acid, acetylvalerenolic acid and valerenal served as inhibitors of NF- κ B at a concentration of 100 μg/ml. Acetylvalerenolic acid reduced NF-κB activity to 4%, while valerenic acid reduced NF-κB activity to 25%.[41] Valeranone (III) was tested as a medical drug in hyperkinetic

Constituents	Value	Constituents	Value
Moisture (g)	7.60±0.11	Ash (g)	8.97±0.30
Protein (g)	4.63±0.10	Phosphorous (mg)	328±1.00
Fat (g)	1.17 ± 0.08	Calcium (mg)	829±0.8
Insoluble fiber (%)	77.00±0.20	Iron (mg)	272.0±0.89
Soluble fiber (%)	7.3±0.10	Zinc (mg)	4.80±0.01
Carbohydrate (g) (By difference)	2.24±0.02	Copper (mg)	2.69±0.01
Vitamin C (mg)	44.90±0.40	Manganese (mg)	11.47±0.00
Total carotenoids (mg)	132.7±0.1	Chromium (µg)	249.0±0.01
Anthocyanin (mg)	ND	-	-

Table 1: Nutritional composition of Valerian

[Adapted from Ref. 18.]

behavior disorders.[42] In animal experiments its sedative, tranquilizing and antihypertensive properties were pharmacologically investigated but the activity of valeranone was found to be lesser than those of the standard substances used.[43] Thus, valerian may carry the sedative effects of anaesthetics and other medications that act on GABA receptors, and use of valerian before surgery may cause a valerian-anaesthetic interaction.

Valepotriates

Valepotriates are esterified iridoidmonoterpenes. Their name is derived from the valeriana-epoxy-triester, because these are triesters of polyhydroxycyclopenta-(c)-pyrans with carboxylic acids: acetic, valeric, isovaleric , α -isovaleroxy-isovaleric, β -methylvaleric, β acetoxy-isovaleric, β -hydroxyisovaleric and β acetoxy- β -methylvaleric acid.[44] It is a major component consisting of 50-80% active compounds. Valepotriates are divided into two classes: monoene and the diene derivatives. The principal diene valepotriates are valtrate, isovaltrate, 7-desisovaleroyl-7-acetylvaltrate and 7-homovaltrate, and the major monoene didrovaltrate derivatives are and isovaleroxyhydroxydidrovaltrate. The amount of valepotriates varies widely between species. In general the underground parts of plant contain higher amount of valepotriates than the other parts of the plant.[45] Valepotriates are unstable compounds: they are thermolabile and decompose quickly under acidic or alkaline conditions in water, as well as in alcoholic solutions. However in anhydrous methanol, and stored at 20°C, the diene valepotriates were found to be relatively stable. Dissolved in methanol or ethanol, with only a small amount of water and stored at room temperature, gives 90% decomposition within a few weeks.[46] The main decomposition products of the valepotriates are the yellowcoloured baldrinals.[47] Baldrinals are chemically reactive and may subsequently form polymers.[46]

In vitro antioxidant studies

Zheng and Wang[48], studied the antioxidant activity of selected herbs which were grown in the same place with similar conditions to avoid variations of oxygen radical absorbance capacity (ORAC) values because of ecological factors. Herbs (2.0 g) were extracted with 15 ml of phosphate buffer (75 mM, p^{H} 7.0) using a Polytron homogenizer (Brinkmann Instruments, Inc., Westbury, NY) for 1 min and were then centrifuged at 20000g for 20 min. The supernatant was used for the ORAC and total phenolic compound assay after suitable dilution with phosphate buffer (75 mM, p^{H} 7.0). They reported the total phenolic content of valerian as 1.78 mg of Gallic acid equivalent (GAE)/g of fresh weight and ORAC as 15.82 μ mol of TE/g of fresh weight.

Nutritional and Medicinal properties

The root and rhizome of the valerian plant (*Valeriana officinalis L.*) is used medicinally for its sedative properties with indications including nervous tension, insomnia, anxiety and stress.[49]

One study found that valerian could sedate the agitated person and stimulate the fatigued person, bringing about a balancing effect on the system.[50]

In an *in vivo* and *in vitro* investigation of valepotriates and valeranone on guinea-pig ileum smooth muscle preparations it was found that dihydrovalerate and valeranone were able to relax stimulated smooth muscle preparations with potency comparable to that of papaverine. Moreover, it was shown that these valeriana compounds cause smooth muscle relaxation through a musculotropic action, which is also known to be the case for papaverine.[51]

Hazelhoff[51], in his dissertation, showed that there is a significant reduction in the locomotor activity of mice when the valerian and *V. officinalis* extract was administered. The

effect of a mixture of valepotriates on the elevated plus-maze performance of diazepam withdrawn rats was evaluated by Andreatini and Leite.[52] The rats were chronically (28 days) treated with diazepam (doses increased up to 5.0 mg/kg) and to provide a withdrawal syndrome they were treated with a control solution for 3 days. Chronically vehicle-treated rats were used as control. The abstinent animals treated with the vehicle showed a significant reduction in the percentage of time spent in the open arms when compared with the control animals. Diazepam and valerian 12.0 mg/kg reversed this anxiogenic effect. They did found significant difference in valerian group than the other group.

Mechanism of action

Because of valerian's traditional use as a sedative, anti-convulsant, migraine treatment and pain reliever, most basic science research has been directed at the interaction of valerian constituents with the GABA neurotransmitter receptor system.[38] The mechanism of action of valerian in general and as a mild sedative in particular is not known.[53] Valerian extracts and some of its constituents, mainly valerenic acid, appear to have some affinity for the GABAA receptor, but the exact mechanism of action is not clear. Benke et al [54] described a specific binding site on GABAA receptors with nM affinity for two general constituents of valerian namely valerenic acid and valerenol. Both valerenic acid and valerenol increased the response to GABA at multiple types of recombinant GABAA receptors. A point mutation in the beta2 or beta3 subunit of recombinant receptors strongly decreased the drug response. In vivo, valerenic acid and valerenol have shown anxiolytic activity with high potencies in the elevated plus maze and the light/dark choice test in wild type mice. In beta3 point-mutated mice the anxiolytic activity of valerenic acid was found to be absent. Thus, neurons expressing beta3 containing GABAA receptors are a main cellular substrate for the anxiolytic action of valerian extracts.[54] Substances such as

valerenic acid and its derivatives acetoxyvalerenic acid and hydroxyvalerenic acid have to pass the blood-brain barrier and interact with this receptor in the brain. It was hypothesized that the investigated terpenes from *V. officinalis* can probably only cross through the blood-brain barrier by a still unknown transport system and not transcellularly by passive diffusion.[39]

Effect on appetite

It is also found that valerian increased the food intake and cause weight gain in adult wistar rat.[55] It has been shown that valerian increases release of GABA (gamma aminobutyric acid) and inhibits enzyme induced break down of GABA.[36,56-57] GABA has direct relationship with serotonin the secretion of which inhibits appetite and food intake.[58] It has been found that ghrelin hormone (hunger hormone) increases production and release of GABA[59], the adverse effect may accrue and increase ghrelin production and so improve the appetite. On the other hand Actinidine (Ia) which is a steam-volatile in the essential oil of valerian root[27], is psychoactive which interferes with the gamma-aminobutyric acid (GABA)-ergic metabolism; it is an agonist on benzodiazepine receptors and thus revealed an allosteric modulation of the GABA-receptorproteins.[22] The mechanism of action needs more study.

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Energy Metabolism in Diabetic Peripheral Neuropathy: Implications for Human Nutrition and Dietetics

Kumar Senthil P., MPT*; Adhikari Prabha, MD**; Jeganathan P.S., PhD***

Abstract

Diabetes mellitus is a well-known lifestyle-associated metabolic disorder leading to microvascular and macrovascular complications as a consequence of insulin resistance and chronic hyperglycemia. Diabetic peripheral neuropathy (DPN) is the leading microvascular complication of DM, with disabling neuropathic pain, sensory/motor deficits and associated psychosocial disturbances. This short communication was aimed at exploring the role of energy metabolism in the pathogenesis, diagnosis and therapy for DPN and found evidence for altered lipid metabolism, with persistent anerobic glycolysis in peripheral nerves of DPN and efficacy of alpha-lipoic acid supplementation in DPN.

Keywords: Metabolic neuropathy; Nutritional Endocrinology; Energy metabolism; Diet Neurology.

Diabetes mellitus is a well-known lifestyleassociated metabolic disorder leading to microvascular and macrovascular complications as a consequence of insulin resistance and chronic hyperglycemia. Diabetic peripheral neuropathy (DPN) is the leading microvascular complication of DM, with disabling neuropathic pain, sensory/motor deficits and associated psychosocial disturbances.

Pathogenetically, Vincent *et al*[1] presented evidence that both chronic and acute hyperglycemia cause oxidative stress in the peripheral nervous system that can promote the development of diabetic neuropathy. They said, "proteins that are damaged by oxidative stress have decreased biological activity leading to loss of energy metabolism, cell signaling, transport, and, ultimately, to cell

(Received on 01.03.2013; Accepted on 18.04.2013)

death."

Diagnostically, Low *et al*[2] examined the effect of ischemia on nerve conduction in experimental diabetic neuropathy (EDN) and related electrophysiological changes to nerve adenosine triphosphate (ATP), creatine phosphate (CP), and lactate under anoxic conditions and their findings suggested that the maintenance of nerve transmission in anoxic-ischemic states depended upon anaerobic metabolism and that resistance to ischemic conduction block (RICB) in EDN was due to anaerobic glycolysis maintained for a longer time than normal nerves.

Pande *et al*[3] examined changes in global gene expression in DPN and identified pathways that included lipid metabolism, carbohydrate metabolism, energy metabolism, and peroxisome proliferator-activated receptor signaling, apoptosis, and axon guidance, and the gene expression changes were consistent with structural changes of axonal degeneration.

Suzuki *et al*[4] studied 36 Type II (noninsulin-dependent) diabetic patients without occlusive arterial diseases in the lower extremities and 12 age-matched and sexmatched non-diabetic subjects to clarify the association between diabetic polyneuropathy and foot ulcers using 1H-and 31P-magnetic resonance spectroscopy and imaging, and

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their findings indicated that motor nerve dysfunction in diabetic patients was closely associated with impaired energy metabolism, fatty infiltration and increased intracellular p^H of plantar muscles and high frequency of foot ulcers.

Thurston *et al*[5] found increases in the nerve content of glucose, sorbitol, and fructose in alloxan-induced diabetic rats and proposed five major hypotheses to explain the pathogenesis of diabetic neuropathy: 1) hypoxia/ischemia, 2) hyperglycemic pseudohypoxia, 3) myo-inositol deficiency, 4) fructose and polyol accumulation and osmotic disequilibrium, and 5) nonenzymatic glycation of macromolecules by fructose and glucose.

Therapeutically, Kishiet *et al*[6] evaluated the effect of Alpha-lipoic acid on glucose uptake, nerve energy metabolism, the polyol pathway, and protein kinase C (PKC) activity in experimental diabetic neuropathy (EDN) induced by streptozotocin. Alpha-lipoic acid supplementation reversed the deficits in EDN, increasing endoneurial glucose, fructose, and sorbitol levels while myo-inositol was significantly reduced.

Stevens *et al*[7] reported the selective effects of administration of the antioxidant DL-alphalipoic acid (ALA) to streptozotocin-injected diabetic rats as follows: "ALA improved digital sensory but not sciatic-tibial motor NCV, corrected endoneurial nutritive but not composite nerve blood flow (NBF), increased the mitochondrial oxidative state without correcting nerve energy depletion, and enhanced the accumulation of polyol pathway intermediates without worsening myo-inositol or taurine depletion."

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