Results
RESULTS

Body Weight, Lung Weight and Tumor Incidence

Fig. 1, 2 and Table 1 shows the body weight, lung weight and tumor incidence respectively of different groups of mice that were sacrificed at the end of the study. The final body weight of the B(a)P administered animals (group 2) was found to be significantly (p<0.05) lowered and the lung weight, tumor incidence was significantly increased (p<0.05) than that of untreated control animals (group 1). Nobiletin increased (p<0.05) the final body weight and significantly decreased lung weight and tumor incidence in group 3 animals when compared with group 2 lung cancer animals. There seems to be no significant difference between nobiletin alone treated animals (group 4) and control animals (group 1).

Serum Tumor Marker Enzymes

Fig. 3 depicts the levels of the tumor marker CEA in serum of control and experimental animals. CEA level were found to be significantly increased (p<0.05) in B(a)P-induced lung cancer bearing group 2 animals whereas their levels were significantly lowered on group 3 treatment with nobiletin when compared with group 2 lung cancer animals. There is no significant difference between nobiletin alone treated animals (group 4) and control animals (group 1).

Marker Enzymes

Fig. 4 represents the activities of marker enzymes in lung tissue of control and experimental groups. The activities of marker enzymes ADA, AHH, γ-GT, 5’NT and LDH were found to be significantly (p<0.05) increased in lung cancer bearing animals (group 2) when compared to control animals of group1. Upon treatment the activities of these enzymes were reversed to near normalcy in nobiletin treated animals (group 3). when
compared with group 2 lung cancer animals. However no significant difference was observed between the nobiletin alone treated (group 4) and control (group 1) animals.

**Tissue and Serum Lipid Peroxidation**

The levels of lipid peroxidation in lung and serum of control and experimental animals are depicted in fig.5. There found to be an increase in LPO in group 2 (p<0.05) cancer bearing animals group 2 when compared with control animals group 1. Nobiletin treatment resulted in significant decrease in the level of LPO in group 3 (p<0.05) animals when compare with group 2 animals. However the nobiletin alone treated group 4 animals when compared with group 1 control animals did not show any significant difference in the LPO levels.

**Antioxidants (Enzymic And Non Enzymic)**

Table 2 antioxidants such represent the cellular enzymic as SOD, CAT, GPx, GR and GST in serum and lung of the various experimental groups. A highly significant (p<0.05) reduction in the activity of enzymic antioxidants in the tumor bearing animals (group 2) was observed. When compared with control animals group1. These adverse changes were reversed to near normal values in nobiletin treated animals group 3 animals when compared to lung cancer animal group2. However, the nobiletin alone treated animals (group 4) did not show any significant changes when compared with control animals (group 1).

Fig .7 represent the non-enzymatic antioxidants (GSH, vitamin E and vitamin C) in serum and lung of the various experimental groups. A highly significant (p<0.05) reduction in the activity of non-enzymatic antioxidants in the tumor bearing animals (group 2) was observed. When compared with control animals group1. These adverse changes were reversed to near normal values in nobiletin treated animals group 3 animals when compared
to lung cancer animal group 2. However, the nobiletin alone treated animals (group 4) did not show any significant changes when compared with control animals (group 1).

**Histopathological Studies**

Fig. 6 shows the histological analysis of lung section of control and experimental groups. Lung from control (group 1) animals revealed normal architecture with small uniform nuclei (plate 1). Lung cancer bearing animals (group 2) revealed loss of architecture, alveolar damage as seen from hyperchromatic and irregular nuclei in the cells of alveolar wall (plate 2). Cancer bearing animals treated with nobiletin (group 3) exhibited reduced alveolar damage with near normal architecture (plate 3). Nobiletin treated animals (group 4) showed no appreciable change of histopathological abnormalities as that of control animals (plate 4).

**Xenobiotic Metabolising Enzymes (Phase I And Phase II)**

Fig. 8 & Fig. 9 depicts the phase I enzyme activities in lung both control and experimental animals. The levels of Cyt.P450, Cyt.b5, NADPH Cyt. P450 and Epoxide hydrolase were significantly increased in group 2 (p<0.05) cancer bearing animals when compared to group 1 animals. Nobiletin treatment resulted in significant decrease in the activities of these enzymes in group 3 (p<0.05) animals when compared to group 2 animals. There seems to be no significant difference between nobiletin alone treated animals (group 4) and control animals (group 1).

Fig. 10 represent the phase II detoxification enzyme activities in lung of control and experimental animals. The levels of GST, UDP-GT, DTD were decreased in group 2 (p<0.05) cancer bearing animals when compared to group 1 animals. Nobiletin treatment resulted in significant increase in the activities of these enzymes in group 3 (p<0.05).
animals when compared to group 2 animals. There seems to be no significant difference between nobiletin alone treated animals (group 4) and control animals (group 1).

**Mitochondrial Studies**

**TCA Cycle and Electron Transport Complexes**

Fig.11 represent the activities of TCA cycle (major) enzymes such as alpha-KDH, ICDH, SDH and MDH in the lung of control and experimental group of animals. The activities of these enzymes were found to be significantly (p<0.05) decreased in cancer induced group 2 animals when compared with the control animals (Group 1). Supplementation of nobiletin to group 3 animals caused a significant (p<0.05) increase in the activities of the TCA cycle enzymes when compared with lung cancer bearing group2 animals.

Fig.12 represent the activities of electron transport chain complex enzymes in the lung of control and experimental group of animals. The activities of all the four complexes were found to be significantly (p<0.05) decreased in cancer induced group 2 animals when compared with the control animals (Group 1). Cancer bearing animals treated with nobiletin group 3 showed a significant (p<0.05) increase in these enzyme activities when compared with the cancer bearing group 2 animals.

**Lysosomal Enzymes**

Fig.13 represent the effect of nobiletin on the activities of lysosomal proteases, cathepsin-D and cathepsin-B, in the serum and lung of control and experimental group of animals, respectively. Significant (p< 0.05) increase in the activities of lysosomal proteases, cathepsin-D and cathepsin-B, was noticed in animals having lung cancer (Group 2). Supplementation with nobiletin significantly (p<0.05) restored the activities of these
lysosomal enzymes to normalcy in Group 3 animals when compared to lung cancer bearing group2 animals.

The activities of serum and lung lysosomal enzymes, β-D-glucosidase, β-D-galactosidase, β-D-glucuronidase, β-D-N-acetylglucosaminidase and acid phosphatase, is shown in table 3. The activities of these enzymes were found to be significantly (p<0.05) increased in the tumor-bearing animals (Group 2). Upon nobiletin treatment, the activities of these enzymes markedly (p< 0.05) returned to normalcy in Group 4 animals when compared to lung cancer bearing group2 animals.

**Studies on Cell Proliferation: Proliferating Cell Nuclear Antigen (PCNA)**

Fig. 14 represent the immunohistochemical staining for PCNA in the lung of control and experimental group of animals. B(a)P induced group 2 (plate 2) showed a significant increase in the number on PCNA positive nuclei when compared with group 1 normal control animals(plate 1), while nobiletin treatment significantly decreased the number of PCNA positive nuclei (plate 4) when compared with B(a)P induced animals. Nobiletin alone treated animal group 4 (plate 3)did not show any change in the PCNA levels.

Fig. 15 represent the immunohistochemical staining for COX-2 in the lung of control and experimental group of animals. B(a)P induced group 2 (plate 2) showed a significant increase in the protein expression when compared with group 1 normal control animals (plate 1), while nobiletin treatment significantly decreased the protein expression (plate 4) when compared with B(a)P induced animals. Nobiletin alone treated animal group 4 (plate 3) did not show any change in the COX-2 protein expression levels.

**Histochemical analysis of mast cell by toluidine blue staining**

Fig. 16 represent the histochemical staining for mast cells by toluidine blue method in the lung of control and experimental group of animals. Tumor induced group 2 animals
showed significant increase in the number of mast cells (plate 2) (can be termed as mast cell density) when compared with group 1 normal control animals (plate 1). Whereas nobiletin alone treated group 3 animals (plate 3) did not show any significant change Whereas nobiletin treated group 4 animals showed decrease in mast cell density (plate 4) when compared group 2 tumor induced animals.

**Nobiletin on COX-2, MMP-2 and MMP-9 Expression**

Fig. 17 represent the immunoblots and quantitative results for COX-2 in the control and experimental group of animals. Tumor induced (group 2) animals showed a significant increase in the levels of these COX-2 when compared with normal control (group 1) animals, whereas nobiletin treated (group 4) animals shows significantly decreased the levels of this marker when compared with tumor induced (group 2) animals.

Fig. 18 and fig. 19 represent the immunoblots and quantitative results for MMP-2 and MMP-9 in the control and experimental group of animals. Tumor induced (group 2) animals showed a significant increase in the levels of these MMP-2 and MMP-9 when compared with normal control (group 1) animals, whereas nobiletin treated (group 4) animals shows significantly decreased the levels of protein expression when compared with tumor induced (group 2) animals.

Fig. 20 represent the protein and mRNA expression for PCNA in the lung of control and experimental group of animals. B(a)P induced group 2 (plate 2) showed a significant increase in the protein expression when compared with group 1 normal control animals (plate 1), while nobiletin treatment significantly decreased the protein expression (plate 4) when compared with B(a)P induced animals. Nobiletin alone treated animal group 4 (plate 3) did not show any change in the PCNA protein expression levels.

**Pro-apoptotic and anti-apoptotic proteins**
Fig. 21 and fig. 22 shows the immunobloting and densitometric analysis of p53, caspase 3, Bcl-2 and Bax in lung of control and experimental group of animals. B(a)P induced animals of group 2 showed significant increase in the levels of Bcl-2 with subsequent significant decrease the expression levels of p53, Bax and caspase 3 when compared with group 1 normal control animals. Nobiletin treated group 4 showed significant decrease expression in the level of anti apoptotic protein Bcl-2 with concomitant increase in the levels of proapoptotic proteins p53, Bax, caspase 3.

**Nobiletin on TNF-α and IL-6 Protein Expressions**

Fig. 23 represents the immunoblotting and densitometric analysis to confirm the protein expression levels of TNF-α and IL-6 in control and experimental groups in mice. B(a)P induced group 2 animals showed there was a significant (p<0.05) increase in expression levels of TNF-α and IL-6. Nobiletin treatment significantly (p<0.01) decreased the levels of these above proteins in group 3 animals when compared with group 2 cancer bearing animals. Nobiletin alone treated group 4 animals showed similar expression to group 1 normal control animals.

**Nobiletin Induces DNA Fragmentation**

Fig. 24 represents the DNA Fragmentation by agarose gel electrophoresis in the lung of control and experimental groups of mice. Lane M shows the molecular marker of DNA. Lane 1 and Lane 4 depicts the control (group 1) and nobiletin alone treated (group 4) shows the normal DNA content. Lane 3 depicts the nobiletin treated group 3 animals showed increased DNA Fragmentation suggesting apoptosis when compared to tumor induced group 2 animals that showed reduced DNA fragmentation (Lane 2).
Figure 1: Effect of Nobiletin on body weight changes in the control and experimental animals

The values are expressed as Mean ± SD for six animals. Group I (control) was compared with Group II (B(a)P). The statistical significant levels were

#p<0.001, *p<0.01, $p<0.005

Figure 2: Effect of Nobiletin on Lung weight changes in the control and experimental animals

The values are expressed as Mean ± SD for six animals. Group I (control) was compared with Group II (B(a)P). The statistical significant levels were

#p<0.001, *p<0.01, $p<0.005
Figure 3: Effect of Nobiletin on carcinoembryogenic antigen in the control and experimental animals

The values are expressed as Mean ± SD for six animals. Group I (control) was compared with Group II (B(a)P). The statistical significant levels were #p<0.001, *p<0.01, $p<0.005.

Figure 4: Effect of Nobiletin on Lung marker enzymes in the control and experimental animals

The values are expressed as Mean ± SD for six animals. Group I (control) was compared with Group II (B(a)P). The statistical significant levels were #p<0.001, *p<0.01, $p<0.005.
Figure 5: Effect of Nobiletin on Lipid peroxidation of Lung and Serum in the control and experimental animals

The values are expressed as Mean ± SD for six animals. Group I (control) was compared with Group II (B(a)P). The statistical significant levels were #p<0.001, *p<0.01, $p<0.005

Figure 7: Effect of Nobiletin on Non enzymic antioxidants enzymes in the control and experimental animals

The values are expressed as Mean ± SD for six animals. Group I (control) was compared with Group II (B(a)P). The statistical significant levels were #p<0.001, *p<0.01, $p<0.005
FIGURE 6  HISTOPATHOLOGICAL STUDIES IN THE LUNG OF CONTROL AND EXPERIMENTAL GROUP OF ANIMALS (%E, 400X)

Plates 1-4 represents the lung section of group 1-4 of experimental animals.
Plate 1: Control animals showing normal architecture
Plate 2: B(a)P induced lung cancer animal showing proliferation of alveolar cells with excess staining for glycoconjugate indicating proliferation
Plate 3: B(a)P along with Nobiletin treated animals show near normal lung architecture with decreased staining for glycoconjugates
Plate 4: Nobiletin alone treated did not show any significant changes
Histopathological report of Mice Lung

**Group I:** Control animals show lung parenchyma with bronchi and bronchioles lined by normal epithelial cells.

**Impression:** Normal Appearance.

**Group II:** Benzo(a)pyrene treated animalsshow lung parenchyma with a neoplasm composed of malignant epithelial cells arranged in glandular pattern and solid sheets. The adjoining bronchioles show epithelial proliferation and dysplasia. The parenchyma shows chronic non-specific inflammatory cell infiltration.

**Impression:** Adenoma Carcinoma of bronchi

**Group III:** Lung cancer induced animals treated with Nobiletin show lung parenchyma with marked chronic non-specific inflammatory reaction. There is moderate degree of fibrosis and sclerosis of blood vessels. There is no evidence of residual tumor.

**Impression:** Moderate Interstitial fibrosis with chronic non-specific inflammation and Sclerosis of blood vessels.

**Group IV:** Nobiletin alone treated animals showing normal appearance. The interstitium appears mildly thickened with scanty inflammatory reaction.

**Impression:** Normal Appearance
Figure 8: Effect of Nobiletin on Lung microsomal phase I metabolizing enzymes in the control and experimental animals

The values are expressed as Mean ± SD for six animals. Group I (control) was compared with Group -II(B(a)P). The statistical significant levels were #p<0.001, *p<0.01,$p<0.005

Figure 9: Effect of Nobiletin on Lung microsomal phase II metabolizing enzymes in the control and experimental animals

The values are expressed as Mean ± SD for six animals. Group I (control) was compared with Group -II(B(a)P). The statistical significant levels were #p<0.001, *p<0.01,$p<0.005
Figure 10: Effect of Nobiletin on Lung microsomal phase II metabolizing enzymes in the control and experimental animals

The values are expressed as Mean ± SD for six animals. Group I (control) was compared with Group -II(B(a)P). The statistical significant levels were #p<0.001, *p<0.01,$p<0.005

Figure 11: Effect of Nobiletin on mitochondrial enzymes in the control and experimental animals

Each value is expressed as mean ± SD for six mice in each group. alpha-KG - mmoles of potassium ferrocyanide liberated/min/mg protein, ICDH - nmols of a-KG liberated/min/mg protein, SDH - mmoles of succinate oxidised/min/mg protein, MDH - nmols of NADH oxidised/min/mg protein: a compared with group -I; b compared with group -II ; c compared with group – III. Group I (control) was compared with Group -II(B(a)P). The statistical significant levels were #p<0.001, *p<0.01,$p<0.005
Figure 12: Effect of Nobiletin on of electron transport chain complex enzymes in the lung of the control and experimental animals

Each value is expressed as mean ± SD for six mice in each group. Units - Complex I - nmoles of NADH oxidised / min / mg protein, Complex II - nmoles of 2,6- dichloroindophenol reduced / min / mg protein, Complex III - nmoles of cytochrome C reduced / min / mg protein, Complex IV - nmoles of cytochrome C oxidised / min / mg protein. a compared with group I; b compared with group - II; c compared with group –III. The statistical significant levels were #p<0.001, *p<0.01,$p<0.005

Figure 13: Effect of Nobiletin on Lung Cathepsin D & B in the control and experimental animals

The values are expressed as Mean ± SD for six animals. Group I (control) was compared with Group -II(B(a)P). The statistical significant levels were #p<0.001, *p<0.01,$p<0.005
Table 1 Body weight, lung weight and tumor incidence in control and experimental animals.

<table>
<thead>
<tr>
<th>Particulars</th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>Group 4</th>
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</thead>
<tbody>
<tr>
<td>Body weight (g)</td>
<td>34.2±3.4</td>
<td>18±2.21 <em>a</em></td>
<td>25.2±2.81 <em>b</em></td>
<td>32±3.20</td>
</tr>
<tr>
<td>Lung weight (mg)</td>
<td>279±27.9</td>
<td>346±37.6 <em>a</em></td>
<td>301.5±30.7 <em>b</em></td>
<td>266.5±26.5</td>
</tr>
<tr>
<td>Number of animals</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>examined</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tumor incidence</td>
<td>0</td>
<td>6</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>No of tumor incidence</td>
<td>0</td>
<td>3.98±0.38 <em>a</em></td>
<td>1.28±0.41 <em>b</em></td>
<td>0</td>
</tr>
<tr>
<td>/ mice</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

Result are expressed as mean ± S.D (n=6)

Statistical significance at *p<0.05, *group 2 compared with group 1, *group 2 compared with group 3.
Table 2 Activities of enzymic antioxidants in the serum of control and experimental animals.

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>Group 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOD</td>
<td>6.30±0.62</td>
<td>4.62±0.35</td>
<td>5.97±0.43</td>
<td>6.28±0.59</td>
</tr>
<tr>
<td>CAT</td>
<td>264±36.2</td>
<td>135±25.2</td>
<td>167±22.0</td>
<td>259±34.8</td>
</tr>
<tr>
<td>GPx</td>
<td>53.70±5.62</td>
<td>35.31±3.80</td>
<td>43.86±5.32</td>
<td>52.75±5.48</td>
</tr>
<tr>
<td>G6PD</td>
<td>224±25.5</td>
<td>167±15.4</td>
<td>220±29.9</td>
<td>220±25.4</td>
</tr>
</tbody>
</table>

Results are expressed as mean ± S.D (n=6)

Statistical significance at p<0.05, a group 2 compared with group 1. b group 3 compared with group 2.

Units: SOD- units/min/mg protein; CAT-µmoles of H₂O₂ consumed/min/mg protein; GPx-µmoles of GSH oxidized/min/mg protein; GR- µmoles NADPH oxidized/min/mg protein; GST-nmol of CDNB-GSH conjugate formed/min/mg protein; G6PD-nmol of NADPH formed /min/mg protein.
Figure: 14 Immunohistochemical analysis of PCNA in the lung of control and experimental group of animals (400x)

Plate 1-4: Represent the lung section of group 1 to 4 animals of experimental animals.

Plate 1: Control group 1 animals
Plate 2: B(a)P induced group 2 animals arrows showing the positive nuclei
Plate 3: B(a)P with Nobiletin treated group 3 animals showing reduced number of positive nuclei.
Plate 4: Nobiletin treated group 4 animals showing normal architecture
Figure: 15 Immunohistochemical analysis of Cox in the lung of control and experimental group of animals (400x)

Plate 1-4: Represent the lung section of group 1 to 4 animals of experimental Animals.
Plate 1: Control group 1 animals
Plate 2: B(a)P induced group 2 animals arrows showing the positive nuclei
Plate 3: B(a)P with Nobiletin treated group 3 animals showing reduced number of positive nuclei.
Plate 4: Nobiletin treated group 4 animals showing normal architecture
Figure: 16 Immunohistochemical analysis of mast cell toluidine blue in the lung of control and experimental group of animals (40x)

Plate 1-4: Represent the lung section of group 1 to 4 animals of experimental animals. Arrows indicate mast cell.
Figure 17: Effect of Nobiletin on protein and mRNA level of Cox-2 in control and experimental animals

Western blot

COX-2 (70 Kda)

β-actin (42KDA)

RT-PCR

M- Ladder
L1- Control
L2- B(a)P Induced
L3- B(a)P+Nobiletin
L4- Nobiletin alone

Fig. 17 shows the effect of Nobiletin on the protein expression and mRNA expression of COX-2, dependent cell proliferation in lung tissues of the experimental lung cancer bearing mice by Western blotting and RT-PCR respectively. Each bar represents the mean ± SEM of three independent observations. A is compared with control; b is compared with B(a)P induced lung cancer. Significance at *p<0.05 level using Student’s-Newman-Keuls test.
Figure: 18 Effect of Nobiletin on Protein and gene expression of MMP-2 in control and experimental animals

Western blot

<table>
<thead>
<tr>
<th>L- Ladder</th>
<th>L1- Control</th>
<th>L2- B(a)P Induced</th>
<th>L3- B(a)P+Nobiletin</th>
<th>L4- Nobiletin alone</th>
</tr>
</thead>
</table>

MMP-2 (72 KDA)  
β-actin (42KDA)

Relative intensity of MMP-2/GAPDH

RT-PCR

MMP-2 (146bp)  
GAPDH (282 bp)

Fig:18 shows the effect of Nobiletin on the protein expression and mRNA expression of MMP-2, dependent cell proliferation in lung tissues of the experimental lung cancer bearing mice by Western blotting and RT-PCR respectively. Each bar represents the mean ± SEM of three independent observations. A is compared with control; b is compared with B(a)P induced lung cancer. Significance at *p<0.05 level using Student’s-Newman-Keuls test.
Figure 19 Effect of Nobiletin on protein and mRNA level of MMP-9 in control and experimental animals

Fig. 19 shows the effect of Nobiletin on the protein expression and mRNA expression of MMP-9, dependent cell proliferation in lung tissues of the experimental lung cancer bearing mice by Western blotting and RT-PCR respectively. Each bar represents the mean ± SEM of three independent observations. 

L: Ladder  
L1: Control  
L2: B(a)P Induced  
L3: B(a)P+Nobiletin  
L4: Nobiletin alone  

A is compared with control; b is compared with B(a)P induced lung cancer. Significance at *p<0.05 level using Student’s-Newman-Keuls test.
Figure:20 Effect of Nobiletin on Protein and mRNA level of PCNA in control and experimental animals

Western blot

PCNA (36KDA)

β-actin (42KDA)

Relative intensity of Cox-2/GAPDH

L- Ladder
L1- Control
L2- B(a)P Induced
L3- B(a)P+Nobiletin
L4- Nobiletin alone

Fig.20 shows the effect of Nobiletin on the protein expression and mRNA expression of MMP-9, dependent cell proliferation in lung tissues of the experimental lung cancer bearing mice by Western blotting and RT-PCR respectively. Each bar represents the mean ± SEM of three independent observations. A is compared with control; b is compared with B(a)P induced lung cancer. Significance at *p<0.05 level using Student’s-Newman-Keuls test.
Figure:21 Effect of Nobiletin on Proteins p53 and Caspase-3 Levels in control and experimental animals

Fig:21 shows the effect of Nobiletin on the proteins (Bax and Bcl-2) expression. Each bar represents the mean ± SEM of three independent observations. a is compared with control; b is compared with B(a)P induced breast cancer. Significance at *p<0.05 level using Student’s-Newman-Keuls test.
Figure 22: Effect of Nobiletin on Proteins Bax and Bcl-2 Levels in control and experimental animals

![Image showing protein bands for Bax and Bcl-2](image)

**Figure 23** shows the effect of Nobiletin on the proteins (Bax and Bcl-2) expression. Each bar represents the mean ± SEM of three independent observations. a is compared with control; b is compared with B(a)P induced breast cancer. Significance at *p<0.05 level using Student’s-Newman-Keuls test.
Figure: 23 Effect of Nobiletin on Proteins TNF-α and IL-6 Levels in control and experimental animals.

Fig: 17 shows the effect of Nobiletin on the proteins (TNF-α and IL-6) expression. Each bar represents the mean ± SEM of three independent observations. a is compared with control; b is compared with B(a)P-induced breast cancer. Significance at *p<0.05 level using Student’s-Newman-Keuls test.
Figure: 24 DNA FRAGMENTATION
DNA damage of Lung Assessed by Agarose Gel Electrophoresis

L- Ladder
L1- Control
L2- B(a)P Induced
L3- B(a)P+Nobiletin
L4- Nobiletin alone